ARTHRITIS

AND ALLIED CONDITIONS

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TO

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PROFESSOR OF MEDICINE

IN THE UNIVERSITY OF PENNSYLVANIA

WITH GRATEFUL APPRECIATION

PREFACE

The author appreciates very much the privilege of being permitted to write this third edition of "Arthritis and Allied Conditions," in so short a period after the appearance of the second edition. Because of the generous assistance of numerous colleagues and friends, it has been possible to include in this third edition 13 additional chapters and more than 100 new photographs. Extensive changes have been made in the text so that the subject matter is brought up to the minute; practically every page has been revised or rewritten in an effort to exclude antedated material and to include all newer important developments.

New chapters have been added on such subjects as penicillin, psychogenic factors in rheumatic diseases, rheumatic manifestations of tropical diseases, recent advances in arthritis and allied conditions, common mistakes in arthritis and allied conditions, Dupuytren's contracture, diagnostic digest of the average arthritic problem for the general practitioner, effect of climate upon arthritis, occupational therapy, and differential diagnosis of rheumatoid arthritis for the practitioner.

An attempt has been made to bring to the general practitioner the essential points in the diagnosis, differential diagnosis and treatment of arthritis and allied conditions. As in previous editions, summaries have been placed in box form so that they are easily available for those individuals with limited time for reading. Diagnostic features and therapy have been written primarily for the general practitioner so that he can personally diagnose and care for the great majority of his patients with arthritis and allied conditions.

Many of the chapters have been practically rewritten. The sections on gold therapy, sulfonamides and penicillin include the important recent developments in these fields.

The chapter on massage has been enlarged and greatly improved by the addition of more than 20 photographs which portray the technics of the various forms of massage.

A feature of the third edition is the new introductory chapter which presents a diagnostic digest of the average arthritic problem for the general practitioner. In addition, there is a new chapter on the treatment of the average arthritic problem by the general practitioner.

The sections on orthopedic care, roentgen diagnosis and therapy, and

physical therapy, have been expanded because of the extreme interest of orthopedists, roentgenologists and physical therapists in the problems associated with arthritis and allied conditions.

The subjects of rheumatoid arthritis and degenerative joint disease have been discussed in detail including the symptoms, differential diagnosis, treatment, etiology and pathology. In all controversial subjects, such as massive doses of vitamin D, gold therapy, roentgen therapy, etc., the opinions of rheumatologists, as well as the experience of the author at the Arthritic Clinic of the University of Pennsylvania, have been summarized. The value of occupational therapy, physical therapy, massage, exercises, transfusions, diet, endocrine products, climate, psychogenic factors, iontophoresis of histamine or choline derivatives, removal of focal infections and such questionable therapy as chaulmoogra oil, sulfur, bee venom, snake venom, foreign protein therapy, fever therapy, vaccines, etc., are discussed in detail. A full discussion is given of the advantages of spa therapy together with a detailed account of spas in this country.

Chapters are also included on specific infectious forms of arthritis such as gonococcal, pneumococcal, tuberculous, syphilitic and other specific forms of arthritis, as well as important details on rheumatic fever, spondylitis, fibrositis, gout, intermittent hydrarthrosis, traumatic arthritis and other conditions.

The etiology and treatment of the painful shoulder and bursitis is discussed in detail as are painful feet and backache. A discussion is also included of herniated intervertebral disks and other causes of sciatic pain.

A section is included on the organization of an arthritic clinic and on typical case summaries, as well as on tumors of joints and related structures.

The section on joint manifestations of tropical diseases should be of particular interest to many physicians in the Armed Forces.

The chapter on penicillin presents the summary of present investigations in this field and includes the details of penicillin therapy, diseases amenable to therapy, dosage of penicillin, mode of administration, complications, etc. The chapter on sulfonamides similarly brings this field up to date.

An interesting added feature is the chapter on Recent Advances in Arthritis and Allied Conditions and the new chapter on Mistakes in the Diagnosis and Handling of Arthritic Patients.

Several hundred pages of new material have been added to the book. This is not reflected in the total size of the book inasmuch as wartime regulations have required conservation of paper and a larger page size was used which makes possible the expansion of the text by approximately 15 per cent without increasing the size of the volume.

Every effort has been made to produce an index which is as complete as possible so that the practitioner can find any desired subject promptly.

The author wishes to thank his many friends and colleagues for their assistance and suggestions and to express his grateful appreciation and cordial thanks for the many kindly reviews, suggestions and constructive criticisms offered by physicians at home and abroad; most of these suggestions have been embodied in this third edition. Among these, thanks are particularly due to Dr. Philip Hench of Rochester, Minn., Dr. Paul Holbrook of Tucson, and Dr. Walter Bauer of Boston (who have been directing the program of rheumatic diseases in the Army), as well as to Dr. Loring Swaim of Boston, Dr. O. H. Perry Pepper, Professor of Medicine at the University of Pennsylvania, and to my father, Dr. Julius H. Comroe of York, Pa. Dr. Eugene P. Pendergrass, Professor of Radiology at the University of Pennsylvania, rendered valuable assistance with the section on radiology and also put at our disposal his entire *x*-ray collection. Dr. Julia Morgan, Associate Professor of Medicine at Shantung Christian University in Tsinanfu, Shantung, China, furnished most of the material for the chapter on Tropical Diseases. Many other individuals have assisted in the preparation of various chapters: Dr. Josef Nylin and Dr. George Morris Piersol in the section on massage, exercises and physical therapy: Drs. Paul Colonna, Irvin Stein and Roy Peck in the sections on orthopedic care and splinting of joints; and A. M. Rechtman and Mr. Morris Wax in the section on painful feet.

New photographs were made by Mr. Reuben Goldberg and drawings by Miss Edna Hill. As in previous editions, the tolerance, assistance and encouragement of my wife have again made this task more pleasant; her aid with the manuscript was invaluable. The firm of Lea & Febiger, and particularly Mr. Victor Boland, made it possible to include in this volume all important material from medical periodicals appearing up to two weeks prior to actual publication of this volume.

B. I. C.

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ARTHRITIS

CHAPTER I

DIAGNOSTIC DIGEST OF THE AVERAGE ARTHRITIC PROBLEM

The busy general practitioner can diagnose correctly the great majority of patients who come to him presenting joint symptoms. Knowledge of a few pointers, in addition to a complete history and physical examination, blood count, urine and sedimentation rate—all of which can be done easily by the general practitioner—should enable him to arrive at a correct diagnosis very quickly in most patients with joint disease.

If the practicing physician will record a detailed history and perform a complete physical examination, most cases of joint disease can be diagnosed rather readily. In some cases, one requires one or more special laboratory tests or in some instances trial with specific medication to make the diagnosis. Occasionally, only the passage of time and careful and repeated observations will permit a final diagnosis; this, however, is the exception rather than the rule.[1]

It is often comparatively easy to differentiate various types of arthritis by certain clinical and pathologic features; at times, the identity of the articular disorder may be determined only after the most careful and painstaking study. Unfortunately, the etiology of all of the various types of arthritis is not known—resulting in some difficulty in classifying the disorders.

Most cases of arthritis may be placed into one or more of the following five groups:

1. Infectious cases caused by specific microörganisms.
2. Cases probably infectious but in which the etiology is still unproved.
3. Degenerative joint disease (also called osteo-arthritis or hypertrophic arthritis).
4. Traumatic joint disease.
5. Gout and gouty arthritis.

Intermittent hydrarthrosis and tabetic joints are much more uncommon.[3]

The specific organisms most commonly responsible for an infectious arthritis include the tubercle bacillus, gonococcus, hemolytic streptococcus, staphylococcus, Treponema pallidum as well as many others encountered less frequently. The arthritis of rheumatic fever and of rheumatoid arthritis is probably infectious but the exact etiology of both of these is still unknown (although many individuals believe that rheumatic fever is closely associated with infection by the hemolytic streptococcus).[3]

Traumatic joint disease may result from injury to the ligaments, cartilage or synovial membrane.

One may also have disturbance of joints secondary to abnormal postural strain, lesions of bone, primary or metastatic tumors, functional or psychogenic causes or associated with loose bodies.

Much can be learned by a carefully taken history, and among the many valuable pointers to remember in this connection are:

1. *Gout is very rare in females;* approximately 98 per cent of cases are found in males. Thus, the general practitioner should not strongly consider gout as a diagnostic probability in women, unless a typical gouty picture presents itself.
2. The *most common forms of joint disease* are rheumatoid arthritis, degenerative joint disease (hypertrophic arthritis, osteo-arthritis) and fibrositis.
3. The *typical signpost of degenerative joint disease* is the presence of *Heberden's nodes*—swellings found at the terminal interphalangeal joints of the fingers.
4. *Mixed forms of joint disease* are frequently present: *i. e.*, a mixture of rheumatoid arthritis together with degenerative joint disease; this picture is often difficult to appraise.
5. *Always inquire concerning trauma to the involved joint, especially if only one joint is involved.* Traumatic joint disease may result from sudden trauma or repeated slight trauma (occupational, recreational, postural, etc.) over a period of weeks, months or years.
6. *Always inquire as to a recent attack of gonorrhea* (usually from ten to twenty days prior to the onset of joint symptoms)—especially in patients with an *acute polyarticular arthritis.*
7. Investigate any *previous attacks of rheumatic fever;* in *90 per cent of cases* of rheumatic fever, *the first attack of the disease occurs before the age of fifteen.* Rheumatic fever is rare under three years of age, but first attacks have been reported in infants.
8. A *sore throat or acute tonsillitis preceding* the onset of acute polyarthritis *suggests rheumatic fever* (since less than 10 per cent of patients with rheumatoid arthritis have a sudden onset, simulating rheumatic fever).
9. *Obtain the age of the patient; marked degenerative joint disease is uncommon under* the age of *forty* (except when due to trauma or if an artificial menopause has occurred). *Rheumatoid arthritis* may occur at any age but the onset is *usually between twenty and forty years.*
10. Does the *arthritic process* seem to *flit* from one joint to another every four or five days? This always suggests rheumatic fever.
11. Do *salicylates* in adequate dosage *relieve the pain* and result in marked improvement in the joint picture? If so, this again suggests rheumatic fever.
12. Is there any history of an *undulating type of fever* with remissions and exacerbations—suggestive of undulant fever— *Brucellosis*?

Which Joints Have Been Involved?

(*a*) Involvement of the **terminal interphalangeal joints** usually indicates degenerative joint disease.
(*b*) Repeated involvement of the **great toe** joints suggests **gout.**
(*c*) **Symmetrical involvement** of the **metacarpophalangeal joints** or of the **proximal interphalangeal joints** usually indicates rheumatoid arthritis.
(*d*) Symptoms centering about one **shoulder** should suggest first the probability of a **subdeltoid bursitis**; about 80 per cent of patients complaining mainly of shoulder discomfort will be found to have subdeltoid bursitis.

13. Is there a sudden onset of joint pain, swelling and tenderness, which persists for several weeks and then entirely disappears, leaving the patient without any joint symptoms in the interim? If there is a recurrence of such attacks—at first every eight to eighteen months—and later becoming more frequent, it is strongly suggestive of *gout.*
14. Does the *joint swelling recur at regular intervals* (from seven to twenty-one days) involving the same joint or joints (most often the knees)? This would suggest *intermittent hydrarthrosis,* especially if there is an abrupt onset of an increasing effusion (usually into a knee) with pain and limitation of motion—with slow disappearance of the fluid within several days.
15. Is *marked sweating* associated with the joint involvement? This symptom may be found in *rheumatic fever, undulant fever* or at times in *rheumatoid arthritis.* Inquire concerning chills and fever—and suspect the possibility of *malaria.*

Have Skin Lesions Been Present, Along With the Joint Involvement?

If so, consider (1) *Lupus erythematosus* (particularly if there is the characteristic butterfly eruption on the cheeks and bridge of the nose). (2) *Psoriasis* (with its typical rash—and an arthritic picture resembling rheumatoid arthritis except that the *terminal* interphalangeal joints may be involved). (3) *Haverhill fever*—with a rubella-like or morbilliform rash chiefly on the extremities, and a polyarthritis. (4) The rash of *syphilis* or the primary sore of *lymphopathia venereum* with polyarticular involvement. (5) *Scarlet fever,* which is occasionally complicated by a migratory polyarthritis. (6) *Rheumatic fever.* (7) Joint phenomenon often associated with *erythema nodosum* and *erythema multiforme exudativum.* (8) *Drug intoxication,* including sulfonamides. (9) *Serum sickness.* (10) Various types of *purpura.* (11) *Gonococcal* infection. (12) Xanthomatous deposits in subcutaneous tissues and joints. (13) Tuberculosis. (14) Joint pains may occur in yaws. (15) Contagious exanthemata (as measles or German measles). (16) Subacute bacterial endocarditis (with petechiæ). (17) *Boeck's sarcoid.* (18) *Dermatomyositis.* (19) *Scleroderma.* (20) Leprosy. (21) *Gout* (in the presence of tophi). (22) *Bacteremia,* including meningococcemia (with petechiæ). (23) Mycotic infections, including histoplasmosis. (24) Ochronosis.

16. Are *tophi* present (most often on the helices of the ears and in the knuckles); they are easily overlooked unless *gout* is suspected. Tophi rarely appear before the tenth year of the disease, except in severe cases. Do not wait until tophi are present to diagnose gout.
17. Have recurrent bouts of *diarrhea* been a feature? Arthritis occurs in about 4 per cent of cases of chronic ulcerative colitis and is more apt to accompany subsequent exacerbations of the colitis than the primary attack.
18. Has there been a *slow insidious onset of monarticular arthritis?* If so, suspect tuberculosis, and especially if there is a family history of tuberculosis or a previous personal history of pleurisy with effusion, suppurative cervical adenitis or fistula *in ano*. Tuberculosis of the spine (Pott's disease) is the most common form of tuberculous arthritis, while tuberculosis of the knee is the commonest type of joint tuberculosis in adults.

Always Suspect Gout[2]

If joint symptoms are associated with a chronic nephritis or renal colic.
If joint symptoms occur in patients with polycythemia, leukemia or after injections of liver extract, salyrgan, insulin, gynergen, thiamin chloride, decholin or after the use of a ketogenic diet.
If acute joint symptoms develop without trauma within a few minutes or hours.
If there is a **family history of gout.**
If *acute joint symptoms* have *followed an operative procedure in males.*
If there is a history of **olecranon bursitis** or *Achilles tendinitis.*
If sudden joint symptoms appear during early morning hours.
If acute arthritis occurs in men over forty, in the absence of a history of gonorrhea or acute trauma.
If acute joint symptoms follow slight trauma, or excessive holiday food or drink.
If joint symptoms recur in the spring or fall.
If joint symptoms occur after hunting and fishing expeditions.

19. Have any *petechia* been noted? A non-suppurating arthritis may occur in subacute bacterial endocarditis; the inflammation usually subsides in a few days. Various forms of septicemia (especially meningococcal) commonly manifest petechiæ; any form of septicemia may produce joint involvement.
20. Has there been any history of *dietary deficiency*—especially *vitamin deficiency?* There is no proof that any common form of arthritis develops as a direct result of a particular vitamin deficiency; however, pain near the joints may result from the subperiosteal hemorrhages so common in scurvy. Muscular pains and calf tenderness are common manifestations of deficiency of portions of the B complex.
21. Is there a history of *joint disease without heat, pain or tenderness?* If so, suspect the presence of a *Charcot joint* (which may occur in tabes dorsalis, syringomyelia and occasionally in paresis, psoriasis, leprosy, poliomyelitis, compression and destruction of areas of the spinal cord or following peripheral nerve section).

22. If the *pupils* are *rigid* and *knee jerks* are *absent,* suspect that the joint involvement is syphilitic.
23. Is there any history of *hypothyroidism* or *hyperthyroidism?* Joint pains may occur in either of these conditions.
24. Is the patient being treated for *syphilis?*
25. Have there been any *recent injections* of *serum?* The joint swellings of serum sickness may mimic rheumatic fever.
26. Has there been any acute or chronic *strain of the low back?* Acute or chronic strain of the low back, frequently due to faulty posture and muscular insufficiency, is *probably the commonest cause of backache.*

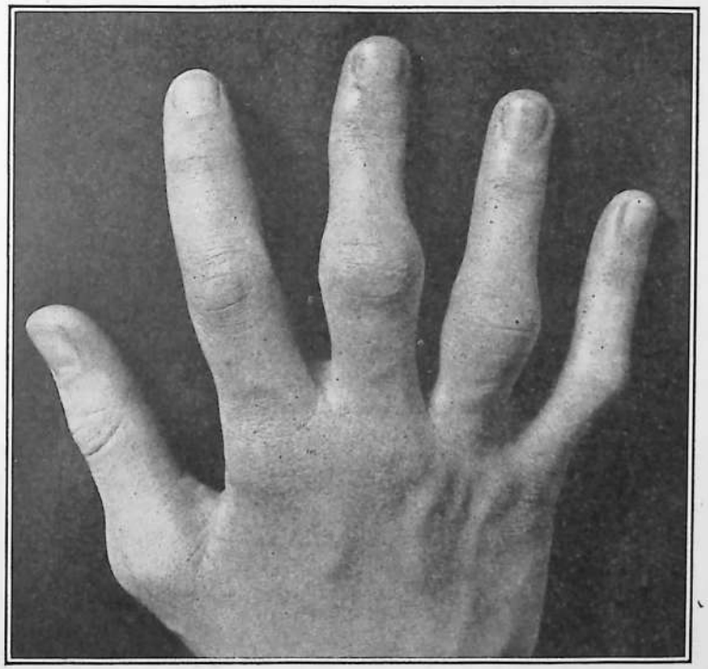

FIG. 1.—Rheumatoid arthritis. Note the fusiform swellings and deformities in the proximal interphalangeal joints of this young woman.

27. If *backache* is the presenting symptom, is this *relieved* by rest in bed on a firm mattress or by the application of heat? Pain in the spine due to neoplasm may be aggravated by immobilization; severe pain may accompany metastatic malignancy in the spine long before the lesion may be seen by *x*-ray.
28. Is the arthritis associated with a marked *loss of weight?* Marked loss of weight is common in rheumatoid arthritis but does not occur in uncomplicated degenerative joint disease, fibrositis or gout.

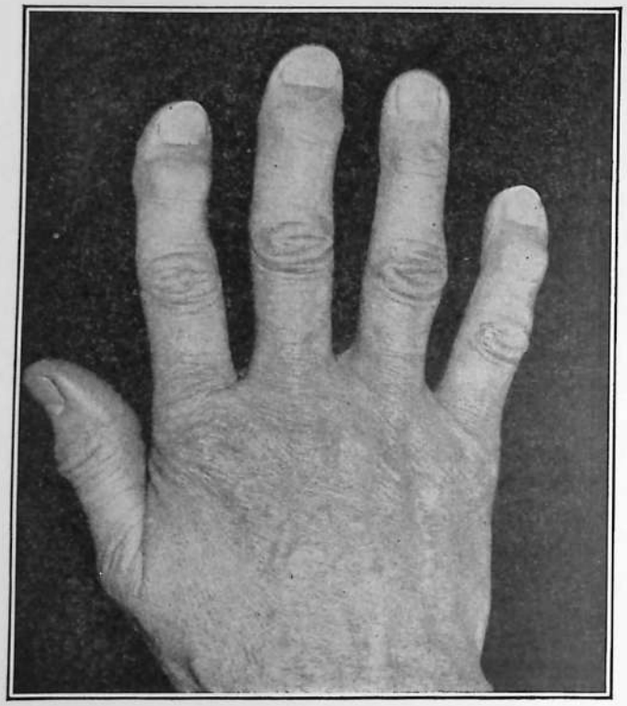

FIG. 2.—Degenerative joint disease of the hand. Heberden's nodes seen at the distal interphalangeal joints (in a woman aged fifty-eight years).

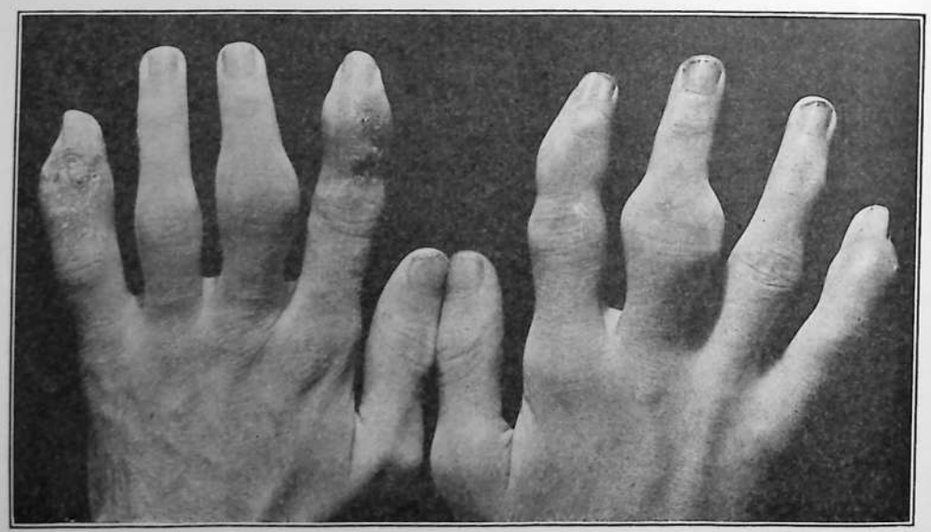

FIG. 3.—Gout. Note asymmetrical swellings and drainage from ulcerated tophi.

Importance of Physical Findings in the Arthritic

1. The presence of **tophi** clinches the diagnosis of *gout.* These are found most often on the helices of the ear and on the knuckles, but have been reported externally around various joints, in the cartilage of the eyelids and nose, as well as in the heels, subcutaneous tissues or muscles, Achilles tendon, patella, etc. They are *painless*, except when they are the site of an acute attack or interfere with motion. They may appear white, cream colored or yellow. Tophi should be opened and some of the contents examined microscopically, when the characteristic needle shaped crystals of monosodium urate will be seen.
2. The presence of **Heberden's nodes** is diagnostic of degenerative joint disease (osteo-arthritis; hypertrophic arthritis). These are small cartilaginous or bony masses situated usually at the proximal end of the terminal phalanges. They may affect any of the fingers, but are more common in the index and middle fingers. One or all of the digits may be involved; the thumbs are affected infrequently. They are immovable and are attached to the underlying bone. They may be tender on pressure, especially if the nodes develop rapidly.
3. **Fusiform, spindle shaped swellings,** especially involving the proximal interphalangeal joints of the fingers, with a tendency to symmetrical involvement are characteristic of *rheumatoid arthritis.* They often progress gradually from the peripheral joints toward the body, with the joints of the hands and feet most often involved at the onset.
4. The presence of **subcutaneous nodules,** most commonly found on the dorsal surface of the forearm 1 to 2 inches below the elbow joints, is suggestive of rheumatic fever or rheumatoid arthritis. They may be found elsewhere, especially over extensor surfaces and over the spine or occiput, in bedridden cases. The nodules of rheumatoid arthritis are usually larger and persist longer than the corresponding lesions of rheumatic fever, and they vary in size from tiny seedlike particles to small walnuts. They may be attached to tendon sheaths or bursal walls, but the skin overlying them is usually freely movable. They are not usually tender. The presence of such nodules is often an indication of a poor prognosis. They are found in from 5 to 25 per cent of patients with rheumatoid arthritis or rheumatic fever.
5. *Lymphadenopathy* of a moderate degree is common in rheumatoid arthritis, especially in children.
6. The *tip of the spleen* can be felt in about 10 per cent of cases of rheumatoid arthritis.
7. *Muscular atrophy* may be marked in advanced cases of rheumatoid arthritis, with or without muscular twitchings. However, it does not occur in uncomplicated degenerative joint disease or gout.
8. *Cold, clammy hands,* with a *smooth, shiny atrophic* appearance of the *skin,* is often noted in the extremities of individuals with rheumatoid arthritis. In severe cases, bronzing of the skin over the face and extremities and patches of vitiligo may be seen. *Psoriasis is found in approximately 3 per cent of patients with rheumatoid arthritis.*
9. *Fever* may be present in rheumatic fever, rheumatoid arthritis, the acute stages of gout, brucellosis, any form of specific infectious arthritis (gonorrheal, pneumococcal, meningococcal, etc.), but this is absent in uncomplicated degenerative joint disease, and in the gouty individual in the free intervals between attacks.
10. The presence of an *olecranon bursitis* should lead one to suspect gout.

Importance of Physical Findings in the Arthritic (*Continued*)

11. *Evidence of syphilis* must be diligently sought: reflex changes, pupillary abnormalities, penile scar, rash, mucous patches, Romberg sign, etc.
12. Pain on elevation of the arm and inability to cross the forearm behind the back should suggest *subdeltoid bursitis*, especially if the symptoms are confined to the shoulder and arm.
13. The presence of a *cardiac valvular lesion*, previous history of rheumatic fever, and a migratory form of joint involvement, responding to salicylates in adequate dosage, should suggest rheumatic fever.
14. A positive tourniquet test would make one consider scurvy or purpura.
15. Evidence of poor posture; weak feet; spinal and other deformities must be sought. The joint pains may be merely an evidence of strain from the forementioned causes.
16. The finding of a *pulmonary lesion* suggestive of *tuberculosis* should naturally raise the suspicion of a tuberculous etiology of the joint pathology.
17. Sluggish, thick speech, weak reflexes, bradycardia, puffiness of the hands and feet without any evidence of pitting on pressure, a dull expression and a hypotension indicate that the joint pains and puffiness may be due to *hypothyroidism*. Similarly, symptoms of *hyperthyroidism* must be investigated (rapid pulse, goiter, exophthalmos, sweating, etc.) as this condition may also produce discomfort in the neighborhood of joints.
18. The presence of any *purpuric spots* may suggest the diagnosis of purpura hemorrhagica.
19. *Serum sickness* may be suspected when one notes urticaria, fever, enlarged lymph nodes, swollen painful joints and a history of the administration of serum from seven to fourteen days previously.
20. *Hysteria* may mimic any disease. Diagnostic pointers include its sudden development after emotional shock, variability of symptoms, presence of glove or stocking type of anesthesia, etc.
21. *Painful inflammatory lesions in muscles* may occasionally lead to a mistaken diagnosis of arthritis. These may be due to trichiniasis when one will obtain the history of eating poorly cooked pork, sausage, hamburg, etc.; gastrointestinal symptoms preceding the muscular pains; edema of the face—especially of the eyelids; painful, tender muscles; fever and an eosinophilia.
22. The finding of a *stiff, poker spine* in a young adult (usually male, between twenty and thirty years), alone or with involvement of hips, shoulders or peripheral joints, frequently associated with low back pain, spasm of the paravertebral muscles and shifting rheumatic pains—usually indicates a *rheumatoid arthritis of the spine.*

USEFUL LABORATORY TESTS IN THE DIFFERENTIAL DIAGNOSIS OF ARTHRITIS

1. *Sedimentation rate: Normal figures* are obtained in uncomplicated degenerative joint disease (osteo-arthritis, hypertrophic arthritis), in most cases of fibrositis, in the intervals between acute attacks of gout and in subdeltoid bursitis. *Increased sedimentation rates* are usually found in the active stage of rheumatic fever, rheumatoid arthritis, during acute attacks of gout, gonorrheal arthritis and other forms of specific infectious arthritis, rheumatoid arthritis of the spine and in tuberculous arthritis.
2. *Blood uric acid: The only rheumatic disease in which the blood uric acid is usually elevated during an acute attack is gout.* (Elevation of the blood uric acid may occur in a variety of pathologic states including acute and chronic glomerulonephritis, cardiac decompensation, leukemia, polycythemia, lead poisoning, some acute febrile illnesses, etc.)
3. *Blood Wassermann test* should be done in all patients with joint symptoms—to rule out syphilis. In many states, this will be done free of charge, if necessary, by the city or state laboratory. *Physicians should make greater use of these facilities.*
4. *Blood count* and *urinalysis* should be done in all patients with arthritic symptoms. Joint pains may be an early sign of *leukemia*, especially in children. *Diabetes mellitus* may mimic the loss of weight, tiredness and pains in the extremities which occur in rheumatoid arthritis.
5. *Basal metabolic rate* is useful in establishing the diagnosis of myxedema or hyperthyroidism.
6. *Roentgen examination* of several of the involved joints may be of assistance in diagnosis and prognosis. These investigations may occasionally reveal evidence of unsuspected pathology—such as metastatic carcinoma.
7. *Examination of synovial fluid*, especially its cell count and the differential pattern of the leukocytes, may prove useful. In acute infectious forms of arthritis, the total leukocyte count and the percentage of polys are greatly increased in this fluid; this is also true, to a lesser degree, in rheumatoid arthritis and rheumatic fever. In traumatic arthritis, the cell count is only slightly increased.
8. *Miscellaneous laboratory examinations* sometimes of value: smear of urethral or cervical discharge for Gc; Gc complement fixation test; agglutination test for Brucellosis; examination of prostatic secretion for leukocytes; *x*-ray of the teeth, paranasal sinuses and gall bladder for focal infection; biliary drainage or cholecystography for evidence of a diseased gall bladder; electrocardiogram—which may occasionally swing the balance in favor of rheumatic fever; stool culture for B. dysentery; synovial fluid culture or biopsy for tuberculosis; blood culture; gastric analysis; blood cholesterol (increased in hypothyroidism); Frei test (positive in lymphopathia venereum); tuberculin test; blood calcium (elevated in hyperparathyroidism).

EXAMINATION OF THE AVERAGE ARTHRITIC PATIENT BY THE BUSY GENERAL PRACTITIONER

As the patient walks into the office, the observing physician can quickly get a rough estimate of the degree of disability, joint deformity, number of joints involved, state of nutrition, pain on motion, age and sex of the patient.

1. *If the patient is a female,* the general practitioner could *discard* the probability of **gout.**

2. If there is marked disability, joint deformity, multiple joint involvement, muscular atrophy, evidence of loss of weight, signs of anemia in a patient (more often a female) between the ages of twenty and forty-five, the patient probably would have **rheumatoid arthritis.** If the history shows that the process has come on gradually, over a period of months or years, with fusiform swelling of some of the proximal interphalangeal joints of the hands, and if the sedimentation rate is increased, the chances are at least nine out of ten that the original impression of rheumatoid arthritis was correct.

3. If the patient complains only of a painful shoulder, of fairly recent onset (hours, days or several weeks), with some radiation of this pain down the arm, in the region of the deltoid muscle, or even to the elbow or hand, one is most likely dealing with a **subdeltoid bursitis.** In addition, there will be marked tenderness over the subdeltoid bursa at the shoulder; the patient will be unable to raise the arm over the head without marked pain, and will be unable to cross the forearm behind the back without great discomfort. He will probably also complain of difficulty in brushing his teeth, combing his hair, or of getting into his coat or overcoat because of pain in the shoulder. *X*-ray examination will reveal evidence of this disorder in only 50 per cent of the cases—*i. e.*, in those where calcification may be found. Thus, *a negative x-ray of the shoulder should not rule out subdeltoid bursitis.*

4. The patient with **gout** usually will not walk into the office, unless he does so between the acute attacks of joint discomfort. One will seldom miss the diagnosis of gout if he remembers the characteristic clinical picture, since one does not require laboratory tests to make the diagnosis in most cases.

The physician is usually called to the patient's home if the latter is having an acute attack of gout in a weight-bearing joint. *The history is typical.* The patient, most often a *male,* usually *past* the age of *thirty-five* (although occasionally cases have been reported in children), develops *sudden* joint pain, redness and swelling—most often with involvement of a *great toe* (although any joint in the body may be involved). The *first attack* of gout is usually *monarticular.* Although attacks may begin in the early hours of the morning, symptoms are usually first detected when the individual puts his foot on the floor, after awakening. *In only about 5 per cent of cases is the disease polyarticular at its onset.* The foot in gout is usually warm and red. The attack lasts from *three to eleven* or more days and the **symptoms then disappear completely.** Another attack may occur in from twelve to eighteen months, varying in severity, and attacking joints either previously involved, or others. Further attacks usually come at closer intervals and the disease becomes more severe. The first few attacks may be afebrile, but subsequent bouts may be accompanied by a temperature of 101° to 102° F. **This sudden onset of attacks of pain with absolute disappearance of all joint phenomena between attacks is almost pathognomonic of gout.** One must also remember that *tophi* occur in only about 50 per cent of all cases of gout, and that they rarely appear before

the tenth year of the disease, except in very severe cases. *Increased blood uric acid* is seen, at some time, in about 75 per cent of cases. *Roentgen-rays* are of little diagnostic value early in the disease, and bony erosions are exhibited in only about one-third of the cases, even in the later stages. *If the patient with acute gout is given adequate doses of colchicine, acute pain will be relieved within ten to twelve hours in most cases.*

5. The older patient with **degenerative joint disease** is a man or woman, usually past forty-five years, who appears in the office with no great discomfort, but who is greatly distressed about the small knobs forming near the terminal joints of many fingers, or the slight stiffness and creaking of the knees or neck. The individual with an uncomplicated degenerative joint disease will have lost no weight, will have an approximately normal blood count, a normal sedimentation rate, and will, to all intents and purposes, show no signs of systemic illness (unless complicating diseases are present). There will be neither marked muscular atrophy nor any marked deformities or ankylosis.

The knoblike protrusions at the terminal interphalangeal joints are the tell-tale *Heberden's nodes,* the ear-marks of degenerative joint disease, and one may detect slight deformity and deviations of the terminal phalanges. There will not be any fusiform swelling of the proximal interphalangeal joints, and the metacarpophalangeal joints will be essentially normal. There will be no increase in normal synovial fluid in any of the joints. Other favorite sites of degenerative joint disease include the knees, cervical and lumbar spine and hip joints. In the knees, the disease may cause some aching and stiffness and actual pain (but without any degree of joint swelling) if there is marked involvement of the patella or strategically situated osteophytes pressing upon nerve fibers. Degenerative changes in the cervical spine may cause stiffness and limitation of motion in the neck. Intervertebral disks may be thinned, especially in the cervical and lumbar region, and marginal osteophytes may form, producing a radiculitis which results in pains radiating down the arms or legs. The main symptoms of degenerative joint disease of the spine include localized or radicular pain, stiffness, limitation of motion and muscular spasm. Coughing, sneezing or straining at stool may result in pain radiating along the nerve trunks which are subjected to irritation by the arthritic lesions, as these nerves leave the intervertebral foramina. If these changes involve the lumbar spine, the pain may be localized, or may radiate down the thighs or legs.

If the process involves the hip joint (formerly called malum coxæ senile or morbus coxæ senilis), the most disabling variety of degenerative joint disease results. Fortunately, this is not one of the more common clinical sites of this entity. In most cases, the onset is between forty-five and sixty-four years. It is unilateral in about two-thirds of the cases and is more frequently observed in men than in women, especially in laborers and athletes. The cartilage over the head of the femur and the acetabulum in the weight-bearing areas becomes thinned and the head of the femur may become flattened. The acetabulum usually becomes enlarged

and deepened, and there is sclerosis of bone and the formation of cystlike cavities in the subjacent bone. The symptoms usually begin insidiously, with pain in the hip, groin or knee. The hip becomes stiff and there is pain and a limp on motion, and relief with rest. *The pain of hip joint disease is often referred to the groin or knee.* Roentgen examination will disclose the general loss of the joint space and flattening of the head of the femur, at times resulting in a mushroom appearance over the neck of the bone.

6. The patient with **rheumatoid arthritis of the spine** *may pass undiagnosed for long periods, even years.* For this reason, the *general practitioner must be aware of the early signs and symptoms of this disease.* If it is treated in its early stages by proper roentgen and other therapy, over 90 per cent of these individuals can return to work, with very marked improvement in their joint and general condition.

In the early stages of rheumatoid spondylitis, the patient may complain of shifting rheumatic pains for days or weeks, appearing intermittently over a course of months or years. *Later, pain appears in the low back or sacro-iliac region.* The discomfort is often more marked at night, especially several hours after retiring. Relief is obtained by getting up and moving about, or by massage. These patients are usually stiff on awakening in the morning, and improve after exercise. Pain in the back is often brought on by stubbing the toes, jarring heavily on the heels, walking down stairs, deep breathing, coughing or sneezing. With progression of the disease, the patient may complain of pain along the entire spine, stiffness, and later, rigidity, leading to the poker spine.

Pain and spasm are the chief causes of limitation of motion in the early stages; later, calcification in the ligamentous structures leads to complete rigidity. The early symptoms are frequently insignificant and are easily overlooked or misinterpreted. Referred pain is almost always present at some stage of the disease, and varies according to the segment of the spine involved and the severity of the process. It may simulate the pain of renal calculus, tabes dorsalis, pleurisy, etc., and may radiate down the arms, legs or around the chest or abdomen. Its intensity may vary from those of a mild nature to severe lancinating stabs.

Rheumatoid spondylitis occurs *predominantly in males* (from 4 to 1 up to 10 to 1 in some series). Most of these cases appear between twenty and thirty years of age.

Most of these patients will show obliteration of the sacro-iliac joints roentgenologically very early in the disease; bilateral involvement is frequent, with increased density and narrowing of the joint space, and often, ankylosis antedating involvement of the spine. It is good medical practice to perform a sedimentation rate and a roentgen examination of the pelvis and lumbar spine in all patients with persistent rheumatic pains in the limbs, without definite signs of specific rheumatic infection. A rapid sedimentation rate together with loss of definition of the sacro-iliac joints, and possibly in addition, osteoporosis of the ilium, sacrum or lumbar vertebral bodies, suggest the presence of a rheumatoid spondylitis. Only

rarely does one find rheumatoid spondylitis without involvement of the sacro-iliac joint.

7. The onset of **rheumatic fever** occurs before the age of fifteen in 90 per cent of cases (although cases may be found at any age). It is very rare under the age of three. No one clinical sign or laboratory test is pathognomonic of this disease. The attack frequently follows a sore throat or acute tonsillitis. Larger joints are most often affected first. The knees, ankles, shoulders and wrists are most often involved but no joint has seemed to escape. In the average case, several joints are affected simultaneously or successively. One of the striking features of rheumatic fever is the tendency of the inflammatory process to flit from joint to joint, often subsiding and entirely disappearing in one joint as another is affected. Frequently, symmetrical large joints are involved simultaneously, and the same joint may be involved several times before the infection has been overcome. The affected joint is hot, swollen, red and tender and motion usually produces marked pain.

The *fever* is often high in those patients with severe joint manifestations ranging from 100° to 104° F. It is irregular and atypical and persists for a variable period of time. When an antipyretic is used (salicylates), the temperature usually falls slowly by lysis, with subsidence of the joint phenomena. In untreated cases, the fever persists for about a month. Copious *sweating* occurs together with the fever; this may so drench the patient that the bed-clothes become soaked with perspiration. The pulse is usually markedly elevated and remains high, even after the temperature falls to normal in some cases. Rheumatic fever may closely simulate rheumatoid arthritis and only the passage of time and subsequent developments permit a correct diagnosis.

In children, the onset of the disease is often much more insidious than in adults. Symptoms in children may include fatigue, malaise, growing pains, intermittent fever, tachycardia, pallor, anemia, night-sweats, epistaxis, chorea, subcutaneous nodules and cardiac murmurs. In some children the only evidence of the disease may be vague muscular pains, abdominal discomfort, epistaxis and a general feeling of sickness. The affected joints are somewhat painful, but there is little swelling. Children often show no redness, swelling or heat in the joints. Stiffness is more marked than tenderness. Growing pains may be a manifestation of this disease in children; these vary greatly but may consist of vague, dragging, fleeting pains in muscles and occasionally in joints, frequently accompanied by easy fatigability, loss of weight and anorexia.

Permanant joint damage practically never occurs in rheumatic fever. The response of these patients to salicylates is dramatic with relief of joint pain and lowering of the temperature to normal (if sufficient dosage is used). Involvement of the pleura or lungs is not uncommon and rheumatic peritonitis may occur. *X*-ray examinations of the joints are usually negative in rheumatic fever except for soft tissue swelling.

In children, important signs of activity of rheumatic fever include pallor, failure to gain weight, rapid sedimentation rate, leukocytosis, fever,

continuance of symptoms (joint pains, sore throat, etc.) and the presence of cardiac murmurs.

Recurrences are so numerous in rheumatic fever that they are almost the rule. Exacerbations of the disease are frequently preceded by upper respiratory infections. The cardiac damage in rheumatic fever is permanent and is often progressive and usually increases with subsequent attacks. In general, greater cardiac damage occurs in younger individuals with rheumatic fever than in older patients. An active stage of the disease may be present with a normal temperature.

In *summary, the common symptoms of rheumatic fever* include a preceding sore throat or tonsillitis; a migratory, symmetrical polyarthritis in adults (but joint symptoms may be minimal or absent in children); fever 101° to 104° F. (oral), irregular—but often between 99° and 101°; excessive perspiration during the acute attacks; tachycardia out of proportion to the degree of fever; occasional presence of subcutaneous nodules—varying in size from a pin-head to an almond—but usually about the size of a split pea; their most common location is about the elbows, fingers, knees, palms, neck, wrists, ankles, carpometacarpal joints, toes, occiput or iliac crests. They are usually firm and painless and are usually not attached to the skin. The presence of such nodules is considered to be indicative of a serious infection. Somewhat similar nodules may occur in rheumatoid arthritis so that their presence is not pathognomonic of rheumatic fever. Cardiac involvement, to some extent, occurs in practically every case of rheumatic fever and migratory, pulmonary hemorrhagic solidification and hematuria may occur. Over 10 per cent of patients with rheumatic fever manifest cutaneous lesions, which are frequently overlooked. These include erythema nodosum, erythema multiforme, small purpuric, papular or urticarial lesions and erythema annulare rheumaticum (the latter being a transitory rash appearing subsequent to the endocarditis and characterized by pale red or bluish macular rings, semicircles or polygons from 1 to 3 cm. in diameter; these lesions are noted most often on the chest, abdomen, back and thorax). The rheumatic erythemas appear and disappear irregularly with wide fluctuations in intensity.

Chorea is the initial manifestation of the rheumatic process in about 25 per cent of rheumatic children. Recurrences of chorea past the age of twenty are very uncommon.

For the busy practitioner an increase in the rapidity of the sedimentation rate is the best index of persisting infection. The negative agglutination reaction with hemolytic streptococci, and the increased antistreptolysin titer in rheumatic fever, are not of practical value to the practitioner.

8. The patient with **gonorrheal arthritis** will usually give a history (if searched for) of gonorrhea, with the onset of joint symptoms usually within ten to twenty days after primary infection. Males predominate by three to one. Gonorrheal arthritis is usually a polyarthritis at the onset—the most frequently involved joints being the knees, ankles and wrists, in order. Tenosynovitis occurs frequently about the wrists and ankles in these patients. Occasionally there is an associated conjunctivitis and

iridocyclitis. The gonococcus complement fixation of the blood is positive in over 80 per cent of cases (if properly performed); this may remain negative for the first four weeks of the disease. Gonococci may be isolated from joint fluid (by special cultural methods) in some cases.

It is not generally known that gonorrheal arthritis is polyarticular at its onset in about 80 per cent of the cases. Although this disease may occur at any age, it is most common between twenty and forty. The acute joint infection may later develop into a stubborn monarthritis and persist for weeks or months if not properly treated. Any stage of joint involvement may be encountered, from a mild arthralgia to severe purulent arthritis with bony ankylosis.

Most patients with gonorrheal arthritis have a fever, which may be slight or may range to 104° F. There are no pathognomonic roentgen signs of gonorrheal arthritis.

A negative gonococcus complement fixation test does not rule out gonorrhea as this is negative in about 20 per cent of the cases. The test may remain positive for several years following clinical recovery, although a negative test is usually obtained within one-half to one and a half years following clinical recovery.

Tenosynovitis is more frequent in patients with gonococcic arthritis than in any other type of arthritis and is a valuable diagnostic sign. Occasionally the tenosynovitis is present without evidence of arthritis. The most commonly affected tendon sheaths are those of the extensors of the hand and wrist.

Coniunctivitis is more common in gonorrheal arthritis than in other forms of joint disease. The most common is a catarrhal conjunctivitis with a sterile exudate but occasionally one observes an iridocyclitis.

Spur formation on the os calcis is usually not the result of gonorrhea. Gonorrheal spurs are uncommon, having been seen in less than 10 per cent of cases of gonorrheal arthritis. *Gonorrheal spondylitis* is also *uncommon* and contrary to the popular conception, the sternoclavicular joint is actually involved in less than 3 per cent of cases of gonorrheal arthritis, and the temporomandibular in only 2 per cent.

The response of gonorrheal arthritis to sulfonamides is dramatic. These should be begun as soon as the diagnosis is suspected. *Penicillin* may be used if prompt response to sulfonamides is not obtained.

9. **Tuberculous arthritis** is usually a slow, insidious process involving usually only one joint; several joints are involved occasionally. Joints affected in order of frequency are the spine, knee and hip (and less often the elbow, ankle, shoulder, wrist and rarely the temporomandibular joint). There is a history of preceding trauma in about 50 per cent.

Tuberculous arthritis is most common in the first fourteen years of life, but may occur at any age. Tuberculous arthritis has been reported appearing in men over fifty years of age.

The presence of a considerable amount of joint fluid, or arthritic involvement of four or more joints is evidence against a tuberculous arthritis.

In almost 50 per cent of cases, there is an associated visceral tuberculous

lesion (lungs, lymph nodes) and frequently one can obtain a family history of tuberculosis or a previous personal history of pleurisy with effusion, suppurative cervical adenitis or fistula *in ano*. Tuberculous arthritis occurs in about 4 per cent of all cases of tuberculosis.

Active early tuberculosis may be present in bones and joints with negative roentgen findings. Tuberculosis of the knee is the commonest type of joint tuberculosis in adults. A low grade tuberculous arthritis may persist in a knee for years with very little discomfort. Pain is usually the first symptom; this is usually insidious in onset. In about 20 per cent of cases, swelling of the knee is the only initial symptom. Local heat is usually present and there is often gross limitation of motion. In most cases, the patient's physical condition is good. One of the most valuable laboratory aids in the diagnosis of early cases (which is often extremely difficult) is bacterial examination of the pus from an abscess.

Tuberculosis of the spine is the most common form of tuberculous arthritis; it is uncommon in adults but is seen most often between the ages of two and ten. The lesions occur most commonly in the bodies of the vertebræ. Collapse of the anterior borders of the vertebral bodies results in angulation of the spine with shortening of the trunk. Cold abscess formation and paralysis are the two most common complications.

Tuberculous tenosynovitis is rare. The diagnosis is made by the finding of a gradually developing painless or slightly tender mass (most often on the volar aspect of the hand) with partial stiffness and inability to completely flex or extend the fingers. In the later stages, finger motion occasionally produces creaking or grating due to the accumulation of fibrinous deposits within the tendon sheaths.

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CHAPTER II

STUDY OF THE ARTHRITIC PATIENT

Arthritis is one of our oldest and most neglected diseases. The earliest known example of multiple arthritis in a fossil vertebrate is in a skeleton of a platecarpus (a large swimming reptile), which lived about 100,000,000 years ago. This now resides in the Museum of Natural History in the University of Kansas.[10] Chronic arthritis of the spine was present in the Ape Man of 2,000,000 years ago as well as in our ancestors, the Java and Lansing men of 500,000 years ago, and Egyptian mummies dating to 8000 B.C.[43] The Romans built extensive baths throughout their empire because of this disease.

Rheumatic diseases are the commonest cause of chronic illness in this country and rank second in causing temporary and permanent disability.[57] It has been estimated that nearly 7,000,000 people (at least 1 in every 20 persons) in the United States have some form of rheumatic disease.[24] Rheumatism is more common than the total number of cases of tuberculosis, diabetes, cancer and heart disease combined.

Arthritis surpasses injury from accidents from the standpoint of days lost from work. The rheumatic diseases disable ten times as many persons per year as does diabetes or tuberculosis and seven times as many as cancer. As a cause of days lost from work from chronic disorders in this country, mental and nervous diseases rank first and rheumatism second. Rheumatic diseases may affect individuals in any economic group but more than half of the total number of days lost from work were in those families with incomes of less than $1000 per year.

Among chronic diseases, rheumatism cripples in the largest number and kills in the smallest. Because of its ability to cripple without killing, it should be put at the head of all other chronic diseases as of preëminent medical, economic and social importance. Unfortunately, a large percentage of individuals affected by the rheumatic diseases receive no medical treatment or treat themselves.[16]

Chronic rheumatic diseases lead to more crippling, higher economic loss and greater suffering than do any other medical disorders. In England, chronic rheumatic diseases cause one-sixth of the total invalidism of the insured population.[15]

Baillou (1538–1616) was the first to use the term rheumatism to designate a form of acute arthritis (as distinguished from gout); he also was the first to make rheumatism a clinical entity.

It has been estimated that on a given day in this country about 1 person in each 1000 has a rheumatic or allied disease.[59] More than 320,000 otherwise able individuals are rendered unemployable in the United States for an entire year by rheumatic diseases. Recent National Health Surveys have shown that the rheumatic diseases were the sixth greatest cause of

disability and were third among those disabling persons for a year or more. Rheumatic diseases were twice as common among those with a family income less than $1000 per year as among those with $3000 or more and three and a half times as common among individuals on relief as in the higher income group.[8,9]

ESTIMATED ANNUAL NUMBER OF DAYS LOST FROM WORK OR OTHER USUAL PURSUITS BY REASON OF SPECIFIC CHRONIC DISEASES (UNITED STATES, 1937)

Disease	Annual number of days lost
Nervous and mental diseases	132,500,000
Rheumatism	97,200,000
Heart disease	95,200,000
Arteriosclerosis and high blood pressure	56,900,000
Tuberculosis, all forms	41,400,000
Cancer and other tumors	36,300,000
Nephritis and other kidney diseases	28,400,000
Diseases of the female organs	26,800,000
Hay fever and asthma	21,900,000
Diseases of gall bladder and liver	20,000,000
Diabetes mellitus	19,200,000
Ulcers of the stomach and duodenum	13,600,000
Hernia	13,600,000
Chronic diseases of the skin	10,300,000
Anemia	8,500,000
Diseases of the bladder and urethra	8,200,000
Chronic bronchitis	8,000,000
Chronic appendicitis	7,600,000
Goiter and other thyroid diseases	7,600,000
Other diseases of the circulatory system	6,600,000
Sinusitis	6,000,000
Varicose veins	5,900,000
Chronic tonsillitis and other throat infections	5,400,000
Hemorrhoids	5,100,000
Chronic pleurisy	4,200,000
Chronic diseases of the eye	4,200,000
Chronic diseases of the ear	3,300,000
Diseases of the prostate and male genito-urinary organs	3,300,000

From Preliminary Reports, the National Health Survey, Sickness and Medical Care Series Bulletin No. 6, U. S. Public Health Service, Washington, D. C., 1938.[41]

PREVALENCE OF CERTAIN CHRONIC DISEASES IN THE UNITED STATES[41]

	Total cases
1. Rheumatism	6,850,000
2. Heart diseases	3,700,000
3. Cancer and other tumors	930,000
4. Tuberculosis—all forms	680,000
5. Diabetes mellitus	660,000

The term rheumatism is derived from the Greek word meaning "to flow," designating an evil humor which was thought to descend from the brain and produce pains in the joints and other parts of the body.[28] This is now used to include arthritis, gout, neuralgia, lumbago and neuritis and its use is therefore to be discouraged inasmuch as it frequently leads to misunderstandings. The three most common forms of rheumatic disease are rheumatoid arthritis, degenerative joint disease and primary fibrositis.

A complete study of the patient with joint symptoms is essential. A

correct diagnosis must be made as soon as possible and all additional complicating factors should be learned at once. Too often the physician may diagnose incorrectly a type of arthritis without a complete study, and may learn several months or years later that his case of "rheumatoid arthritis" is actually one of gout, hyperthyroidism, or, perhaps, the articular complications of scleroderma. Although a complete study of each patient consumes time, it is productive of the best end results.

Classification of Arthritis[31]

Arthritis due to **infection.** *Specify joint and organism when known,* as:
- Of spine due to tuberculosis.
- Of knee due to gonococcic infection.
- Of wrist due to pneumococcic infection.
- Of hip due to syphilis.
- Arthritis due to gonococci, tubercle bacilli, pneumococci, Treponema pallida, typhoid and paratyphoid bacilli, meningococci, staphylococci, streptococci (and arthritis occasionally associated with dengue, dysentery, chronic ulcerative colitis, leprosy, malaria, Brucellosis, scarlet fever, yaws, and infections with B. coli, B. diphtheriæ or B. influenzæ); some forms of tenosynovitis.

Arthritis due to **rheumatic fever.** *Record* rheumatic fever *as primary diagnosis invariably.*

Arthritis, rheumatoid, of.....*Specify* as:
- Arthritis, rheumatoid, multiple (synonyms: proliferative arthritis, atrophic arthritis, chronic infectious arthritis, Still's disease).
- Arthritis, rheumatoid, atypical (focal infection arthritis).
- Arthritis, rheumatoid, of spine (synonyms: rheumatoid spondylitis, atrophic spondylitis, Marie-Strümpell disease, spondylose rhizomelique, infectious spondylitis, spondylitis ankylopoietica, von Bechterew's disease).

Arthritis due to direct **trauma** of.....*Specify joint and trauma,* as:
- Of knee due to contusion.
- Of elbow due to habitual dislocation.
- This includes traumatic forms of synovitis and tenosynovitis, arthritis, fibrositis, bursitis, sprains, strains, internal derangements, fractures into joints, loose bodies and damage to cartilages, etc.

Neurogenic arthropathy (Charcot joint) (associated with tabes dorsalis, syringomyelia, cord trauma, nerve injuries or leprosy). *Specify joint,* as:
- Tabetic arthropathy of knee.

Arthritis due to **gout.** Specify joint. Record gout as primary diagnosis invariably.

New growths of joints. *Specify joint and neoplasm,* as:
- Synovioma of knee, cyst; xanthoma; hemangioma; giant cell tumors; synovioma.
- Primary or secondary.

Degenerative joint disease, multiple due to unknown cause; synonyms: osteo-arthritis (hypertrophic arthritis, degenerative arthritis, chronic senescent arthritis).

Mixed forms of arthritis (especially rheumatoid arthritis and degenerative joint disease).

Hydrarthrosis, intermittent, of.....*Specify joint.*

Periarticular fibrositis. *Specify joint.*

Disease in which arthritis, arthropathy or arthralgia is frequently associated (diagnose disease, list joint manifestation as symptom):

Acromegaly.
Acute disseminated lupus erythematosus.
Cyst of meniscus of knee.
Dermatomyositis.
Drug intoxication. *Specify drug when known.*
Erythema multiforme exudativum.
Erythema nodosum.
Hemophilia.
Hysteria.
Ochronosis.
Osteochondritis dissecans.
Osteochondromatosis.
Periarteritis nodosa.
Psoriasis.
Raynaud's disease.
Reiter's disease.
Scleroderma.
Serum sickness.
Purpura, various types.

Other Disturbances of Joints

I. Disturbances of the joints secondary to abnormal postural strain.
II. Disturbances of the joints secondary to lesions of bone.
III. Disturbances of the joints associated with loose bodies.
IV. Disturbances of the joints secondary to functional or psychogenic causes.

Of the 7,000,000 sufferers from rheumatic disease, more than 3,000,000 actually have arthritis;[53] 130,000 of these are completely disabled and another 800,000 are partially disabled and lose an average of five months' work each year. Over 50 per cent of the disability is in persons under forty-five years of age.[44]

The lack of facilities for proper care of arthritic patients in this country is appalling. In a recent survey, it was shown that there were not more than a total of 200 free hospital beds in the entire country specifically available for arthritic patients as compared with more than 100,000 free beds for the care of tuberculous patients.[53] Furthermore, although $100,000,000 is available for research work in tuberculosis, less than $10,000,000 is available for similar projects in arthritis. These facts must be emphasized to the general public so that adequate facilities and money is made available at once for the proper care and study of arthritic patients.

Special hospitals must be established for the treatment of these crippling diseases in an effort to lighten the economic and social burden which they carry. We do not have in this country adequate facilities for early diagnosis, research, proper therapy, hospitalization or undergraduate or postgraduate medical education in the rheumatic diseases.

It was estimated that there would be 60,000 cases of chronic arthritis per year in World War I in the United States Army alone—with its size at the end of that war. Approximately 35,000 ex-service men from World War I are now disabled by arthritis; these individuals receive an annual compensation of $10,000,000.[31]

Rheumatic diseases include the common affections of joints, muscles and related structures.

Plan of Study for the Arthritic Patient

Complete, Detailed History.—*This is the most important single factor in diagnosis.*
Complete, *detailed physical examination.*
Red and white *blood count,* hemoglobin determination and differential blood count.
Urinalysis: specific gravity, albumin, sugar and microscopic.
Blood *Wassermann* test.
Blood *sedimentation rate.*
Blood *uric acid* determination.
Roentgen examination of at least one of the affected joints, especially of the hands or feet, if these are involved.
The above studies are done routinely in every arthritic patient.

Additional Examinations Often Requested:

Examination of synovial fluid (total nucleated cells, differential count, red blood cells, culture, smear, appearance, clotting, protein concentration, sugar content, viscosity).
Gonococcus complement fixation test.
Cervical smear and culture for gonococci; thorough pelvic examination or gynecologic consultation.
Blood agglutination test, skin test and opsonophagocytic index for Brucellosis.
At least two examinations of prostatic secretion for leukocytes.
Complete dental *x*-ray (even though edentulous); one-third of edentulous patients have unextracted roots.
Thorough examination of tonsils, paranasal sinuses (by transillumination, nasopharyngoscope and roentgen-ray if necessary), and search for excessive lymphoid tissue in the pharynx.
Biliary drainage and cholecystogram if symptoms of gall bladder disease are present.
Basal metabolic rate (if symptoms suggest hyper- or hypothyroidism).
Careful examination of patient from orthopedic viewpoint: posture, weak feet, evidence of low back strain, etc.
Electrocardiogram in occasional cases if diagnosis of rheumatic fever is questioned.
Special examinations sometimes requested: culture of stool for B. dysentery and streptococci; biopsy of synovia for TB.; nose and throat culture for hemolytic streptococci; blood culture; antistreptolysin titer; agglutination test for hemolytic streptococci group A; glucose tolerance curve; gastric acidity; gastrointestinal roentgen-ray and barium enema; blood cholesterol; Frei test; smears of blood for malaria.

In soldiers, it has been estimated that the order of frequency in rheumatic diseases will be about as follows: most frequent—fibrositis (muscular rheumatism); then, in order, rheumatic fever, rheumatoid arthritis, gonorrheal arthritis, septic arthritis, degenerative joint disease, gout, tuberculous arthritis and other less frequent conditions. Among the American Army of over 4,000,000 soldiers in World War I there were reported only

Arthritic Record

History

P.M.H.: arthritis; rheumatic fever; growing pains; heart disease; scarlet fever; syphilis; gonorrhea; allergy; diabetes; Brucellosis; hypo- or hyperthyroidism; anemia; psoriasis; dysentery; polycythemia; neuritis; leukemia; leprosy; hemophilia; lymphogranuloma venereum; previous injections.
Preceding operations: tonsils; teeth; sinuses; others.
Associated diseases:
Onset of joint pains—date—
Frequency of attacks:
Recurrence of joint flare-up with interval of complete freedom from attacks.
Joint first involved:
Other joints involved, in order:
Joints involved at present, in order of distress to patient:
Nodules or tophi.
Occupations:
Trauma: Strain:
Fatigue—mental; physical
Fever: Loss of weight: Sweating:
Emotional shock.
Recent upper respiratory tract infection: Exposure to cold:
Menopause:
Diet: alcohol:
Previous therapy and response:
rest: splints: diet:
salicylates: colchicine:
Foci: removed:
Physical therapy:
Injections: gold; sulphur; bee venom; vaccine; histamine; foreign protein (typhoid vaccine, aolan, intramuscular blood); chaulmoogra oil; others.
Transfusions:
Vitamin therapy:
Response to pregnancy:
Response to jaundice:
Response to intercurrent infection:
Other drugs.

Consultations

Orthopedic:
Nose and Throat: Allergic:
Dental: *Physical therapy:*
Roentgen-ray (diagnostic and therapeutic)
Gynecologic:
Genito-urinary:

Laboratory Results

RBC *Hb.*
WBC *Differential:*
Polys. Lymphs. Eosinoph.
Basoph. Monocytes
Others:
Urine: Sp. gr.: Alb.:
Sugar *Micr.:*
Blood Wassermann test:
Sedimentation rate—one hour:
Blood *uric acid:*
Gc. complement fixation test:
Synovial fluid examination:
Agglut. for B. abortus:

B.M.R.
Blood cholesterol:
Gastric analysis:
Biliary drainage:
Stool culture:
E.C.G.
Agglutination for hem. strep.:
Antistreptolysin titer:
Glucose tolerance:
Blood calcium: Blood phosphorus:
Tuberculin test:
Nose and throat culture:
Blood culture:
Joint biopsy?
Frei test:

Diagnosis:

Therapy

Rest
Splinting of affected joints
Diet—vitamins
Foci of infection
Exercises
Physical therapy; occupational therapy; orthopedic care
Gold
Iron—transfusions
Protection from cold and dampness
Analgesics
Symptomatic
Special climate

FIG. 4.—Arthritic record chart, using Jansen ciphers.

82 cases of gouty arthritis, 188 cases of tuberculous arthritis, 2671 cases of tenosynovitis, 7895 instances of gonorrheal arthritis as compared with 33,613 instances of rheumatoid arthritis and degenerative joint disease,

24,770 cases of acute articular rheumatism (probably rheumatic fever) and 12,093 instances of fibrositis (muscular rheumatism).

Important Points in History Taking in Arthritis

Mode of onset: Joints involved; progression of joint affection; remissions. Complete freedom from joint symptoms between attacks?
History of acute or chronic *trauma;* occupation; physical or mental strain.
Fatigue—mental or physical; loss of weight.
Relation of onset to acute upper respiratory infection, pharyngitis or tonsillitis; relation to menopause; previous arthritis, rheumatic fever, heart disease, "growing pains," scarlet fever, injections of liver extract, insulin or ergotamine tartrate; pregnancy.
History of recent attack of *gonorrhea.*
History of *allergy* (hay fever, asthma, urticaria, food allergy, eczema).
Response to therapy (salicylates, colchicine, surgical procedures).
Associated diseases: diabetes mellitus, psoriasis, hemophilia, anemia, neuritis, Brucellosis, dysentery, hypothyroidism, hyperthyroidism, syphilis, polycythemia, leukemia, leprosy, lymphogranuloma venereum.
Fever: presence, type, duration.
Sample diet: deficiency? Alcoholism?
Recent operations; recent injection of serum.

Important Points in Physical Examination

Joints affected; amount of swelling, tenderness, redness, deformity, limitation of motion.
Presence of: fusiform swellings of proximal interphalangeal or metacarpophalangeal joints; Heberden's nodes; subcutaneous nodules; tophi; olecranon bursitis.
Evidence of syphilis: Romberg sign, pupils, reflex changes, penile scar, rash, mucous patches, etc.
Evidence of cervical rib.
Signs of subdeltoid bursitis (pain on elevation of arm; inability to cross forearm behind back).
Tip of *spleen* may be palpable in chronic rheumatoid arthritis.
Generalized *adenopathy* not uncommon in chronic rheumatoid arthritis.
Cardiac lesion (size of heart; rhythm; tone of cardiac sounds; murmurs). Clubbed fingers.
Tourniquet test for scurvy or purpura.
Signs of leprosy (nodules; areas of anesthesia).
Marked arteriosclerosis. Evidence of herpes zoster.
Foci of infection (tonsils, teeth, paranasal sinuses, lymphoid tissue in pharynx, prostate, cervix, etc.).
Evidence of poor posture; weak feet; spinal deformities.
Tenderness over spine (localized especially at lumbosacral or sacro-iliac joints), limitation of motion of spine (on bending).

In the British Expeditionary Forces (1940), 26 per cent of all medical patients admitted to several General Hospitals had rheumatic disorders, the most common of which was fibrositis (70 per cent), with 15 per cent of those affected having rheumatic fever and 9 per cent having rheumatoid arthritis; only 6 per cent had degenerative joint disease—usually traumatic.

Military factors which may be expected to cause an increase in the incidence of rheumatoid arthritis include emotional disturbances, exposure to dampness, prolonged chilling, exposure to respiratory infections, etc.[25]

RHEUMATOID ARTHRITIS

Rheumatoid arthritis is a *generalized disease* with definite constitutional manifestations. *None of the many theories of etiology* (endocrine, infectious, metabolic, neurogenic or circulatory) *has been generally accepted.*

There is *no single pattern of development* of the disease; a variety of clinical forms may be noted. Although the *onset* is most common between the ages of twenty and forty-five, this is not limited to any age group and the disease may begin in infancy or senility. *Females* are affected much more frequently than males except in rheumatoid arthritis of the spine (where males outnumber females about 10 to 1).

In most instances of rheumatoid arthritis, the *onset is insidious,* although this may be explosive (or any intermediate variation). For weeks, months or years, the only symptoms are *prodromes* such as lassitude, loss of weight, weakness, fatigue, exhaustion, cold, sweaty hands and feet, numbness and tingling in the extremities or symptoms referable to the nervous system.

Small or large joints may be affected with the proximal interphalangeal and metacarpophalangeal joints of the hands being involved most often. At the onset, migratory joint involvement may be observed, but in most instances, the joint involvement becomes permanent. The affected joints become warm, swollen, tender and later show limited motion and deformity. Only one or a few joints may be involved in the early stages but sooner or later the *symmetrical, polyarticular distribution* so typical of rheumatoid arthritis makes its appearance.[51]

Other prominent features include *muscular weakness* and *atrophy.* In some areas, *subcutaneous nodules* are found in as many as 20 per cent of cases (being instances of inflammation of extra-articular fibrous tissue). The *lymph nodes* may be enlarged in from 20 to 50 per cent of cases and *splenomegaly* is seen in some cases. Less frequent findings include atrophy and pigmentation of the skin, *cardiac findings* (often similar to those of rheumatic fever), pleuritis, uveitis, scleritis, etc.

Laboratory findings include an increased *sedimentation rate* in the active stage of the disease; frequently a shift to the left in the Schilling or filament-non-filament counts; *agglutination by the patient's serum of strains of hemolytic streptococci* (Lancefield Group A) in about half of the cases (although the test is seldom positive in individuals whose rheumatoid arthritis is of less than a year's duration); low antistreptolysin and anti-

fibrinolysin titers (as contrast with elevated values in the serum of patients with rheumatic fever); hypochromic anemia in severe cases with variable leukocyte counts—somewhat elevated often in the early stages and lowered in some long-standing cases; and increased leukocytes in the synovial fluid and somewhat characteristic findings on biopsy of the subcutaneous nodules. *Roentgen findings* may be of inestimable value, but one may observe very little in the roentgenogram early in the disease, except soft tissue swelling and possibly slight decalcification. The roentgenogram must be interpreted carefully, and one must consider the joints involved, the stage of the disease and the severity of the process.[51]

DISEASES MISTAKEN FOR RHEUMATOID ARTHRITIS

This list does not include degenerative joint disease, gonorrheal and other specific forms of infectious arthritis, traumatic arthritis and other primary joint disturbances, but comprises numerous diseases not always thought of as producing joint pains.

Periarticular fibrositis
Rheumatic fever
Syphilis
Subdeltoid bursitis
Gout
Weak, painful, pronated feet
Peripheral neuritis (associated with alcoholism, diabetes mellitus, primary anemia, chronic arsenic poisoning)
Serum sickness
Hypertrophic pulmonary osteo-arthropathy
Tuberculosis (pulmonary)
Psoriatic arthritis
Myxedema
Hyperthyroidism
Malingering
Hemophilia
Arthritic purpura
Leukemia
Hysteria
Cervical rib and scalenus anticus syndrome
Raynaud's syndrome
Scleroderma
Acrosclerosis
Calcinosis
Parkinsonism
Angina pectoris
Lipomas
Osteochondritis dissecans
Osteomyelitis
Herpes zoster
Polycythemia
Intermittent claudication
Drug intoxication
Deficiency diseases: scurvy, beriberi, rickets
Hyperparathyroidism
Paget's disease
Trichiniasis
Erythralgia
Infantile paralysis
Periarteritis nodosa
Malignant disease of joints
Leprosy
Lupus erythematosus
Acromegaly
Ochronosis
Subacute bacterial endocarditis
Coronary thrombosis
Boeck's sarcoid (Lupus Pernio)
Dermatomyositis
Milkman's disease
Arachnodactyly

POINTS USEFUL IN DIFFERENTIAL DIAGNOSIS

Periarticular Fibrositis:

Onset of pain, aching, stiffness or soreness in any area where fibrous tissue is present (usually with absence of swelling and other physical findings).
Pain and stiffness generally worse on awakening or after periods of inactivity such as attending church, movies or sitting in a chair; relief of discomfort by mild exercise.
Joint tenderness mild or absent; usually no objective stiffness of joints.
Usually no muscular atrophy or constitutional phenomena.
Sedimentation rate usually normal.
No bony changes seen by roentgen-ray.
Good prognosis so far as deformity is concerned.

FIG. 5.—Chronic rheumatoid arthritis of knees in male aged fifty-five years.

Rheumatic Fever:

Onset under fifteen years in majority of cases.
Sudden onset of multiple joint pains, swelling and redness usually with high fever and often accompanied by profuse sweating.
Often preceded by sore throat or upper respiratory infection.
Characteristic flitting from joint to joint.
Usually dramatic response to salicylates (given in adequate doses)

Cardiac findings frequently present.
No bony changes on roentgen examination.
No permanent joint changes.
Not uncommon among soldiers in camps in temperate climates.
Antistreptolysin titer increased during active stage of the disease.
Electrocardiographic changes if serial pictures are made.

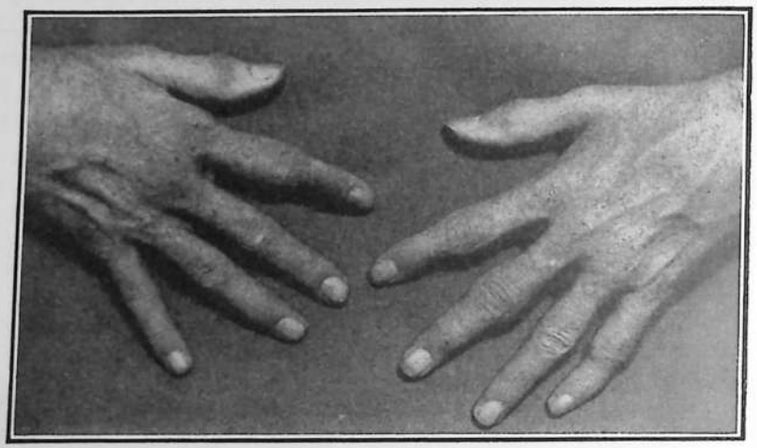

FIG. 6.—Rheumatoid arthritis. Note fusiform swellings of proximal interphalangeal joints.

Syphilis:

Arthritis may occur in early, late or congenital syphilis.

Joint syphilis may include arthralgia in early syphilis, hydrarthrosis (in early, recurrent or heredosyphilis), polyarthritis and synovitis, gummatous osteo-arthritis and Charcot joints.[26]

Syphilitic arthralgia often produces nocturnal pain (as do gout and metastatic malignancy).

Clutton's joints (syphilitic hydrarthroses) are the articular manifestations of congenital syphilis. These are usually found as painless, symmetrical non-inflammatory effusions in childhood; occasionally, they are monarticular. There is usually no local redness or heat and practically no constitutional reaction. Clutton's joints are rarely seen in adults. They are especially common in the knees, elbows and ankles, but may attack any joint, producing stiffness on extreme flexion and extension, because of the effusion. Other stigmata of congenital syphilis aid in diagnosis. The articular lesions clear completely even without specific treatment.

Syphilitic polyarthritis may accompany early syphilis, involves symmetrical larger joints, producing only slight discomfort or constitutional disturbance in proportion to degree of involvement. There is little tendency to flit from joint to joint; large symmetrical joints are usually involved.

Gummatous osteo-arthritis is usually asymmetrical involving chiefly the knees and elbows, producing large tense joints, only slight pain or constitutional reaction, very little decrease in mobility and very rarely ankylosis.

Syphilitic gummatous infiltration of suprapatellar or olecranon *bursæ* may occur; this may break down or become hard and fibrotic.

Charcot joints may (contrary to previous teaching) show pain at some time in their course; diagnosed by "the loss of contours, the projecting knobs, the bag-of-bones feel and the extraordinary hypermobility, permitting in some cases the most amazing distortions without the slightest distress."[55] Rigid pupils and absent knee jerks are of greater diagnostic importance in tabetic arthropathy than are blood and spinal fluid tests.[54] Symptomless hyperextensibility of joints (especially fingers and thumbs) is also a feature of a congenital mesenchymal dysplasia known as **Ehlers-Danlos[56,61] syndrome** (in which additional findings include hyperelasticity of the skin, and friability of the skin and its blood-vessels with formation of papyraceous scars). Joint disease without heat, pain or tenderness should always make one consider neuro-arthropathy.[4]

A blood Wassermann test should be done on all patients with bone, joint or bursal lesions.

The *blood Wassermann test may be negative in 75 per cent and spinal fluid Wassermann negative in over 50 per cent of patients with Charcot joints;*[55] however, neurologic signs of tabes are practically always present.

The *main collateral aids in the diagnosis of syphilis of the skeletal system include*—blood Wassermann test, roentgen findings, and careful analysis of skin lesions and scars. Syphilitic skin lesions are present in almost 50 per cent of patients with osseous syphilis.

In cases of *chronic irregularly recurring bilateral hydrarthrosis, suspect syphilis.*

Always include an examination for neurologic signs of syphilis (pupillary reactions, tendon reflexes, lightning pains, Romberg sign, bladder signs, etc.).

Syphilitic joint lesions are usually bilateral, not very painful, but of long standing.

Syphilitic dactylitis may produce fusiform swellings of the fingers (and occasionally of the toes); this is common in congenital syphilis.[33]

Generalized aches and pains may result from meningeal involvement in syphilis.

Spondylitis due to syphilis is not a rarity and severe root pains may follow spinal deformities subsequent to a destructive syphilitic process.

"Subdeltoid Bursitis" Syndrome:

Commonest cause of pain at the shoulder.

May cause severe pain in the shoulder region, radiating at times into the neck, arm, forearm and even to the finger tips.

Produces marked limitation of motion of the shoulder; extreme pain on abduction, or in attempts to place the forearm across the back.

Tenderness in the region of the greater tuberosity of the humerus.
Usually relieved by procaine injection or roentgen therapy in acute cases; some improve promptly without treatment.

Gout:

Sudden attacks of pain, swelling and redness in joints with intervals of complete freedom from pain.
Males involved in 98 per cent of cases.
Elevation of blood uric acid.
Finding of tophi, with demonstration of monosodium urate crystals; tophi may not appear until late in the disease.
Relief of symptoms with *adequate* dosage of colchicine.
Frequent involvement of the big toe.
Occurrence at times as an acute post-operative arthritis.
Occurrence of arthritis in a patient with polycythemia, leukemia, pernicious anemia (after liver therapy), chronic nephritis, renal colic, etc.
Frequent association with olecranon bursitis.

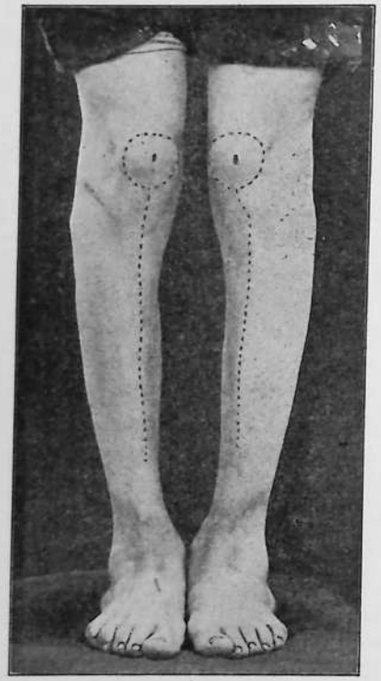

FIG. 7.—Weak feet, showing the inward rotation of the legs when the feet are placed side by side, indicating an attitude of persistent abduction. (Whitman.)

Foot Strain (Weak Feet; Flat Feet):

Foot strain frequently leads to pain in the knees and lower back from faulty posture and strain of ligaments.
In many cases, pain thought to be due to arthritis of the lower spine or knees actually results from disorders of the feet.
The two common signs of disorder of the feet are: Pronated, unbalanced posture, and the formation of callus on the sole behind the second and third toes.[39]
Foot pain is usually due to (1) local pressure; (2) strain of muscles or ligaments; or (3) local inflammatory changes.[58]
Foot strain is probably the commonest cause of pain in the legs and feet; strain develops readily in pronated, weak feet.
Foot strain is usually related directly to the amount of walking and standing performed by the individual.
Pain due to foot strain is usually not present while in bed or on arising; pain due to arthritis may appear at any time.
Remember, however, that weakening and flattening of the feet are often early signs of rheumatoid arthritis (associated with swelling, tenderness, pain also at rest, etc.).

Peripheral Neuritis:

Neuritis may produce sharp burning pain in the distal portion of the extremities, chills, fever, general malaise, exaggerated tendon reflexes (in an early stage), tingling and numbness in the hands and feet, muscular tenderness, superficial hyperesthesia, muscular atrophy (or even paralysis), and later, gradual disappearance of deep reflexes, etc.
Claw hand or talipes equinus may follow multiple neuritis.
Involvement in neuritis is usually symmetrical and bilateral.
So-called *alcoholic neuritis* (*usually due to lack of vitamin* B_1) *causes 90 per cent of all neuritis,*[22] and is more frequent in women than in men.
Neuritic pains are often mistaken for early rheumatoid arthritis or fibrositis.
Neuritis may be caused by: lead, arsenic, bismuth, mercury, Jamaica ginger, carbon bisulfide, carbon monoxide, ether, etc.
Disease entities often associated with neuritis are: diabetes mellitus, vitamin deficiencies, leukemia, diphtheria and chronic secondary anemia.
Deformity of the hands produced by neuritis is occasionally incorrectly diagnosed as arthritis.
Diabetic neuritis is rare under the age of forty.
Chronic arsenic poisoning may be associated with painful joints in the extremities.[33]
Brachial neuritis may simulate subdeltoid bursitis or arthritis of the shoulder joint.

Serum Sickness:

May mimic rheumatic fever.
Occurs most commonly from seven to fourteen days after administration of serum.
Characterized by fever, enlargement of lymph nodes, edema of the skin, urticaria and swollen, painful joints.
No permanent joint damage results.
Symptoms often ameliorated by epinephrine HCl 1:1000 solution, 0.3 to 0.6 cc. (5 to 10 m.) given hypodermically; may be repeated every hour for several doses if necessary. Improvement also follows use of ephedrin sulfate, 25 to 45 mg. (gr. $\frac{3}{8}$ to $\frac{3}{4}$) three times a day by mouth, or histaminase (2 tablets orally three times a day).[12]

Hypertrophic Pulmonary Osteo-arthropathy:[11]

Hypertrophic pulmonary osteo-arthropathy was first described in 1889 by Bamberger[3] as an ossifying periostitis. Pierre Marie described a series of cases the following year.[37]
It was observed by Hippocrates; however, the causative factor of this condition remains problematic. It has been suggested recently that the widespread bone and soft tissue involvement in this syndrome may result from dyspituitarism.[20]
Usually considered to be secondary to chronic suppuration in the lungs, intrathoracic tumors,[60] congenital and valvular heart disease, chronic lesions of the liver and intestinal tract, polycythemia vera or hypothyroidism; no cause found in some cases.

Usual findings: clubbed fingers and/or toes and other soft tissue changes associated with subperiosteal symmetrical proliferation of bone, usually affecting the bones of the arms or legs, but also observed in the clavicle, ribs and pelvis.[49]

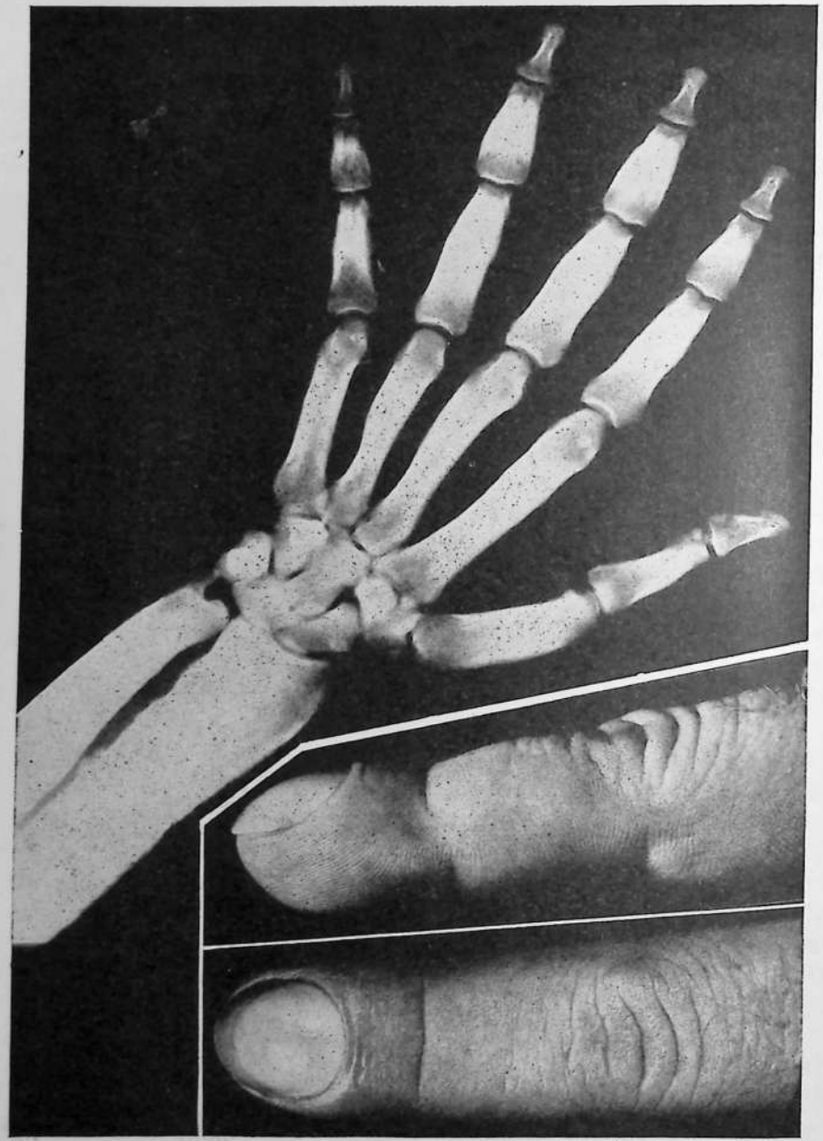

FIG. 8.—Hippocratic finger and roentgenogram of hand showing the characteristic features of hypertrophic osteo-arthropathy. (Campbell *et al.*, Proc. Mayo Clin.)

In some cases, sclerosis and thickening of the cortex.
Curving of the nails (parrot beaking).

Atrophy of the cancellous tufts with elongation of the terminal phalanges.[34]
Non-pitting edema of the distal portions of the extremities.
Joint lesions in about one-third of the cases; periarticular involvement, synovitis, erosion of articular surfaces or ankylosis.
No single hypothesis adequately explains the syndrome.

FIG. 9.—Psoriasis arthropathica. (Dawson, Nelson's Loose Leaf Living Medicine.)

Pulmonary Tuberculosis:
The loss of weight, general malaise, low grade long continued fever, secondary anemia and anorexia of pulmonary tuberculosis may closely simulate the general symptoms of rheumatoid arthritis.

Psoriatic Arthritis:
Association of psoriasis and rheumatoid arthritis in an individual (see rheumatoid arthritis). Considered by some to be a distinct entity.

Myxedema:
Often accompanied by "neuralgia" in various parts of the body. Diagnosis is suggested by obesity, coarse features, harsh dry skin, loss of hair, non-pitting edema, bradycardia, decreased tendon reflexes and lowered basal metabolic rate.

Hyperthyroidism:
May resemble rheumatoid arthritis: loss of weight, weakness, low grade fever, tachycardia, general aches and pains.
Usually readily distinguished by B.M.R., mass in neck, exophthalmos, roentgenograms, response to rest and iodides, etc.

Malingering:

For insurance or other purposes this must be differentiated from arthritis; the malingerer, and the hysterical individual, will show: normal sedimentation rate and negative roentgen-ray findings.

Hemophilia:

Hemorrhage into joints is not uncommon; diagnosis is suggested by a history of previous severe hemorrhages; hereditary tendency; male sex; prolonged clotting time of blood.

Arthritic Purpura (Schönlein's disease).[50]

Joint involvement may be part of the general purpuric manifestations, producing pain, swelling and tenderness in one or multiple joints.

Leukemia:

Often mistaken for rheumatic fever in young children; diagnosed by finding an abnormal leukocyte pattern; may require sternal marrow biopsy (see rheumatic fever).

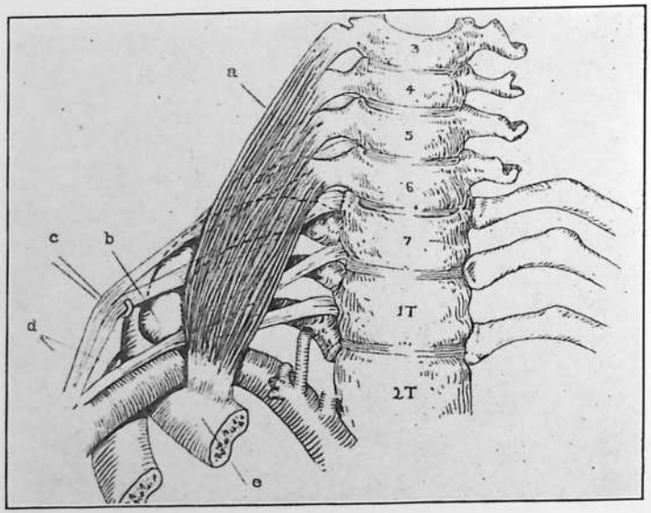

FIG. 10.—Scalenus anticus and cervical rib: *a*, scalenus anticus; *b*, cervical rib; *c* and *d*, portions of the brachial plexus; *e*, first rib. (Bickman, Operative Surgery, courtesy of W. B. Saunders Company.)

Hysteria:

May mimic any known disease. Diagnostic pointers include sudden development after emotional shock, symptoms incompatible with an organic lesion, variability of the symptoms, presence of glove or stocking type of anesthesia; normal sedimentation rate; negative roentgen findings.

Cervical Rib and Scalenus Anticus Syndrome:

May cause neuralgic pain, paresthesias, or anesthesia extending from the neck to the hand.

Decreased amplitude or absence of radial pulse on the affected side.

Increased pain on downward pressure on the shoulder of the affected side or on rotation of the head toward the affected side.

Occasional presence of a bruit over the clavicle.

Simulates arthritis of the shoulder joint or cervical spine; produces symptoms by irritation of the cords of the brachial plexus from stretching of these over the sharp edge of the cervical rib, or by compression of the brachial plexus by the scalenus anticus muscle.

Temporary relief is often provided by injection of procaine hydrochloride into the scalenus anticus muscle.

Average age of appearance of symptoms—thirty years.

	Rheumatoid arthritis	Buerger's disease	Scleroderma	Raynaud's disease
Age of onset	20-50 most often	20-50	20-55	20-45
Sex incidence	2 or 3 females to 1 male	95% *males*	Equal	95% *females*
Pulses	Present	1 or more —*absent*	Present	Present
Vascular tone	High	Low or high	High	High
Color changes	Variable	Pallor or cyanosis	May have blanching	White, then cyanotic, then red. *Bilateral; symmetrical.*
Roentgen findings	*Typical:* narrowed joint space; demineralization of bones; fusiform joint swelling, etc.	Negative	May have *calcification in periarticular soft tissues and absorption of terminal phalanges* in advanced cases	Negative
Sedimentation rate	*Rapid*	Normal	Normal	Normal
Clinical response to heat	Improved	No change	Questionable	Improved
Dissociated vascular response in digits	*Present*	No dissociation	No dissociation	No dissociation
Joint changes—clinically	*Fusiform joint swellings*	None	May have atrophic changes and periarticular soft tissue calcification	Occasional subjective sense of swelling; usually no objective swelling
Pigmentation of skin	May occur late	None	May occur	None
Pain	*Present*	Present	*Usually none* unless ulcers form	Present
Tight, leathery skin	Skin may be taut	Absent	*Present*	Absent
Loss of hair in involved extremities	None	None	*Usually marked*	None

Raynaud's Syndrome:

Much more common in females than males.

Characterized by attacks of coldness and ischemia of one or more fingers (or much less commonly, toes, ears, cheeks, etc.), with numbness, stiffness and actual pain. It is usually symmetrical and may last for minutes or days; it is commonest in winter. With subsidence of the attack, the affected parts usually become red, hot and the seat of severe tingling pain.

Essential pathology: vasospasm.

Symptoms often simulate early rheumatoid arthritis.

Scleroderma:

This uncommon disease is characterized by circumscribed or diffuse induration and atrophy of skin and subcutaneous tissue. The skin becomes hard and immobile, later becoming thin and parchment like.

Stiffness and deformity may result from shrinking of periarticular tissues (a sort of periarticular fibrosis); this may cause confusion with rheumatoid arthritis.

This is a systemic disease involving the collagenous tissues throughout the body, including the collagenous tissues of the blood-vessels themselves.

It may render the patient helpless from structural changes with very little or no pain.

Any set of bizarre symptoms may arise, varying with the tissues involved; any organ in the body (including the brain, heart, esophagus, etc.) may be affected.

The extremities show the most marked changes at the tips.

Motion becomes limited by the leathery tightening of the skin.

The skin becomes smooth and shiny and hair disappears from the involved areas.

Brownish pigmentation may appear with deposition of calcium in the pigmented skin (especially over the joints, such as the hands and elbows).

Changes may occur quickly (within a few months) and cover the body. Morphea type may have lesions localized to small plaques which may produce distortion.

The terminal phalanges may become atrophic.

This may be associated with Raynaud's disease.

The skin becomes stretched so tightly that wrinkles disappear.

Localized or generalized immobility may result.

The extremities or jaw may become fixed and motionless due to contractures and muscular atrophy.

Sedimentation rate is normal.

Acrosclerosis.[27,42]

A process of shrinkage and contraction of the skin seen symmetrically and conjointly on the hands, fingers and face, resulting in shorter, sharper phalanges and finally mutilation and clawlike contractures of the hands.

Arthralgia and dysphagia are not uncommon.

Affects chiefly females during adolescence or adult life.

Diagnosed by the history of vasospastic disease and skin changes.

Resorption of bone beginning in the distal phalanges and extending to the proximal phalanges.

May involve mucous membranes.

Spontaneous partial involution of the scleroderma may occur; prognosis as regards life is favorable.

Interstitial Calcinosis:

Development of hard, small, calcified nodules, often in the vicinity of joints; calcium deposits in the skin, subcutaneous tissues or deep seated interstitial connective tissues.

Lesions are most common in the fingers, elbows, knees and buttocks.[13]

Grayish chalky material may exude from the lesions if the overlying skin breaks down.

The sedimentation rate is normal as is the blood count, uric acid and blood calcium. Studies of the calcium metabolism reveal no abnormality.

The calcified areas are not connected with bone. There is no demineralization of bone and no arthritic changes (by roentgenogram). The generalized form, calcinosis universalis, is most often seen in children and has a poor prognosis.

Treatment is unsatisfactory. In some instances, it has been claimed that rapid resorption of the calcium deposits with improvement in the clinical picture may follow hemithyroidectomy and parathyroidectomy;[46] others found little influence on the clinical course after such an operation.[10]

FIG. 11.—*Calcinosis circumscripta* (chalk gout) in a male aged sixty-two years. There is no demineralization of the bones. Deposits of calcium of various sizes were seen along the shafts of all of the long bones; these were near joints in most cases.

Calcinosis is characterized by deposition of calcium in the skin and subcutaneous tissues. The diffuse or universal type may involve large portions of the surface of the body and in some cases also the interstitial tissues of tendons, muscles and nerve sheaths. The circumscribed variety is usually restricted in its distribution to the articular regions of the phalanges and the extensor portions of the knees and elbows.

Vasospastic phenomena are common in calcinosis, and scleroderma and sclerodactylia are present in about 40 per cent of these patients. Although the contractures and deposits about the joint may limit motion, the bones and joints are not usually involved.

Parkinson's Disease (Paralysis agitans):

Charcot first pointed out the resemblance between Parkinsonism and chronic rheumatoid arthritis, especially the similar deformities of the hands and feet in the two disorders and the muscular rigidity, tremor and shiny appearance of the face which may occur in both.

1. Occasionally misdiagnosed rheumatoid arthritis because of the semi-flexion of the arms and wrists and stiffness and flexion of the head and body.[45]
2. Patients are usually between fifty and seventy, although the onset is occasionally earlier in life.
3. Characterized by a rhythmic tremor, mostly in the hands, with a pill-rolling movement of the hands. The tremor appears at rest and is lost by movement (although it is constantly present in advanced cases).
4. Handwriting appears small.
5. The tremor may extend to the legs, lips, chin and tongue.
6. There appears rigidity of the muscles of the neck, head, spine, extremities and face.
7. A masklike facies appears.
8. Winking is infrequent.
9. It is difficult for the patient to straighten up; he is often bent forward with his head bent forward on the neck, the upper part of the trunk slightly flexed, the forearm semi-flexed and the hand in constant pill-rolling motion.
10. All motions are slow.
11. There is difficulty in getting started in walking because of the peculiar posture—a propulsive gait.
12. The patient turns *en masse*—the body remaining stiff and straight as a poker (sometimes leading to the mistaken diagnosis of rheumatoid arthritis of the spine).
13. The patient may have a drawing dull pain in the shoulders as an early sign, as well as muscular weakness.

Angina Pectoris:

Pain in angina pectoris occasionally radiates to the left elbow; if precordial symptoms are not marked, this may lead one to suspect localized arthritis in the left elbow.

The correct diagnosis is suggested by: relief of pain by nitroglycerin or amyl nitrite; brief duration of the pain; absence of objective signs of joint disease; negative roentgen examination of joints. Fibrositis (as pleurodynia) may cause pain which simulates angina pectoris.

Lipomas:

May cause pains in the extremities due to accumulation of fatty tissue; juxta-articular fatty masses may be painful. Joint roentgen-rays are negative; sedimentation rate is normal.

Osteochondritis Dissecans:

Occurs most often in the knee. Symptoms are due to the formation of loose bodies.

Pain in the joint.

Sensation of catching or actual locking of the joint.

Typical roentgen findings (hiatus on the bone with loose bodies in the joint).

Osteomyelitis:

Pain, swelling and tenderness over the affected area of bone; joint roentgen-ray is usually negative unless the process has extended to the joint.

Herpes Zoster:

May simulate the root pains of spondylitis; diagnosis is possible only when the characteristic vesicular rash has appeared; relief of pain in some cases by roentgen therapy, injections of pituitrin subcutaneously or iodides (intravenously).

Polycythemia Vera:

Joint pains and limitation of motion may occur.[14]

Gout occasionally appears in these individuals.

Neuralgic pains in the extremities and muscular spasms may occur.

Diagnosed by the marked increase in erythrocytes and hemoglobin, red cyanosis, enlarged spleen, etc.

Intermittent Claudication:

Cramplike pains in the lower extremities are occasionally mistaken for arthritis.

Paroxysms of pain in the legs or feet while walking; relief by rest.

Pulsation of the dorsalis pedis or posterior tibial arteries is usually weak or may be absent.

No joint changes.

Drug Intoxication:

Arthralgia and joint swelling and pain occasionally follows sulfonamide administration (*sulfatoxic arthritis*).

In these cases, joint symptoms usually disappear within forty-eight hours after sulfonamides are discontinued.

Arthritic symptoms occasionally follow the administration of some drugs (as arsenic, bismuth, mercury, gold, barbiturates).

Deficiency Diseases:

Scurvy: lack of vitamin C may result in hemorrhages in or about the joints, producing painful joint swellings.

Scurvy is usually diagnosed by the spongy, readily bleeding gums; general tendency to hemorrhages; reduced capillary resistance; subperiosteal hemorrhages; history of diet inadequate in vitamin C; anemia; ready response to vitamin C (ascorbic acid) in adequate doses.

Occurs most commonly under the age of two; arthritis of any form is very rare at this age.

Tenderness is usually in the shafts of bones (in scurvy) rather than of the joints.

Scurvy may occur in adults.

Vitamin B_1 (thiamin) deficiency.

May produce pains and weakness in the extremities. (See discussion of multiple neuritis.)

Responds to adequate vitamin B_1 (thiamin chloride) therapy.

Rickets.

Occurs most often under two years of age.

Characterized by pallor, muscular atrophy, swellings in the ends of bones at the epiphyseal line, generalized decalcification of the skeleton.

Occasionally confused with arthritis because of disinclination to move the limbs (from hyperesthesia or tenderness).

Characteristic roentgen-ray findings.

Fairly rapid response to vitamin D therapy.

Hyperparathyroidism:

Pains in the bones and joints not uncommon; diagnosis is suggested by rarefaction in bones, increased blood calcium and phosphatase, low blood phosphorus, increased urinary calcium and phosphorus, possible finding of parathyroid tumor.

Paget's Disease:

May cause pains of the extremities and cramps in the leg muscles; occasionally mistaken for arthritis.

Usually readily diagnosed by the increase in size of the head, marked bowing of legs, and pathognomonic roentgen findings.

Occasionally involves only one bone.

No actual changes in joints unless there is an associated degenerative joint disease (osteo-arthritis) which is not uncommon; pains are chiefly in the bones.

Trichiniasis:

Painful inflammatory lesions in muscles occasionally lead to a mistaken diagnosis of arthritis.

History of eating poorly cooked pork, sausage, hamburg, etc.

Gastrointestinal symptoms (nausea, vomiting, abdominal pain and diarrhea) preceding muscular pains.

Edema of the face, especially of the eyelids.

Painful, tender, and at times, swollen muscles.

Normal white blood count or leukocytosis of moderate degree and almost invariably an eosinophilia (after the second day).

Fever (may be marked).

Larvæ are often found by examination of muscle tissue.

Positive reaction to intracutaneous injection of trichinella antigen (after the third day of the disease); antigen obtainable from National Institute of Health, U. S. Public Health Service, Washington, D. C.

Immediate and complete recovery may follow administration of tetrachlorethylene (2.5 to 3 cc. in adults) followed by an ounce of magnesium sulfate two hours later.[1]

Erythralgia (formerly called erythromelalgia):

Characterized by burning pain in the extremities (especially the feet); redness of the skin; discoloration of the limbs; swelling, sensation of warmth and throbbing of the arteries.

Late in the disease, fixation and contractures may occur.

Infantile Paralysis (acute anterior poliomyelitis):

Usually produces pains in muscles and joints of the affected limbs; diagnosis made by history of an epidemic, lumbar puncture findings, and the occurrence of muscular weakness or paralysis.

Periarteritis Nodosa:

May cause pain in joints, muscles or along peripheral nerves.[35] Twenty-seven per cent of cases show arthritis.

Very rarely diagnosed before death; only 26 of 101 cases described in the English literature were diagnosed antemortem.[23]

Occurs chiefly in adult males (70 per cent).

Symptoms are variable and include: crampy abdominal pains, vomiting, diarrhea, pain in muscles and along peripheral nerves, pain in joints, rash (erythematous, purpuric or maculopapular), evidence of nephritis (albuminuria, hematuria, edema), moderate degree of fever and marked polymorphonuclear leukocytosis and eosinophilia (in about 12 per cent).[30]

Most suggestive finding: nodules in subcutaneous tissues along the course of medium sized arteries; these nodules are usually painless and non-pulsating.

Articular and neuromyositic symptoms may appear early and may be the first or second symptom noted; any joint may be involved.[7]

Articular symptoms usually fade into the background as the syndrome progresses.

Diagnosis is confirmed by muscle biopsy; negative biopsy findings do not exclude the disease.

Usually fatal within a few weeks or months.

Malignancy of Joints:

Uncommon; diagnosed by roentgen-ray.

May be benign (osteochondroma, xanthoma, fibroma, lipoma, angioma) or malignant (synovioma); primary or metastatic.

Leprosy:

May cause paresthesias or areas of anesthesia in the extremities; claw hand may simulate arthritic contractures.

Lupus Erythematosus:[47,48,52]

Erythematous butterfly rash on the face.

In chronic lupus erythematosus, about 20 per cent have joint symptoms.

Joint symptoms cannot be differentiated from early subacute infectious arthritis and are not diagnostic of lupus erythematosus. Diagnosis depends upon recognition of the characteristic skin lesions.

Articular symptoms are rarely diagnosed properly until the skin lesions have appeared.

Acute disseminated lupus may show arthralgia and polyarthritis in over 60 per cent of cases, together with nephritis, endocarditis, pleuritis, prolonged fever, anemia and polyserositis.

FIG. 12.—Neural leprosy (claw hand); aged seventy years; duration thirteen years. Jamaican negro. (Howard Fox.)

FIG. 13.—Lupus erythematosus of face and hands.

Acromegaly:

Tufting (or proliferation) at the tips of the distal phalanges.
Fingers and palms become broadened.
Hands look puffy and feel stiff.
Coarse facial features with thick lips, large nose and prominent head.
Prognathism (protruding jaw) from overgrowth of the mandible; mastication may be impaired.
Degenerative joint changes, especially involving the spine.
Enlargement of the sella turcica in 90 per cent of cases
Headaches, weakness.
Visual changes.
Backache, neuralgic pains and paresthesias may occur.

Ochronosis:

Cartilage and fibrous tissue may become darkened.
Degenerative cartilaginous changes may occur, with degenerative changes in larger joints.
Bluish discoloration of the cartilages of the ear.
Black flecks or streaks in the scleræ.

Pigmentation of the skin of the face.
Discoloration of the tendons of the hands and feet.
Alkaptonuria in some cases (the urine turning brown on standing or after addition of alkali).

Subacute Bacterial Endocarditis:

Rarely has associated arthritis from septic emboli lodging in or around a joint.
Never suppurates.
Inflammatory reaction usually subsides in several days.
Diagnosed by history of previous rheumatic fever, cardiac valvular lesion, loss of weight, fever, petechiæ, microscopic hematuria, sweating and positive blood culture for Streptococcus viridans.

Coronary Thrombosis:

Occasionally mistaken[26] for acute subdeltoid bursitis or cervical spondylitis because of radiation of pain to the shoulder.[2,6,17,18,29] Electrocardiographic findings are pathognomonic; chest leads may be required.

Kehl reported 6 cases of Dupuytren's contracture following coronary occlusion.[32] The changes in the palms were typical of various stages of Dupuytren's contracture. The etiology and pathogenesis were not understood; it was assumed that irritation of the sympathetic ganglia may assume an important etiologic rôle. The patients noticed pain, stiffness and swelling of the hand, with numbness, tingling, livid discoloration and abnormal skin temperature locally.

Johnson noted disabling changes in the hands resembling sclerodactylia following myocardial infarction in 39 of 178 consecutive cases. In 38 of these, pain occurred in both hands; 34 had pain in one or both shoulders; 22 developed contracture of the palmar fascia and the same number had residual stiffness of the shoulders.[29]

Boeck's Sarcoid:

Boeck's sarcoid (also called sarcoidosis, lupus pernio, etc.) is a chronic (usually) benign, infectious disease of unknown etiology involving the skin, lymph nodes, salivary glands, eyes, lungs and bones (particularly of the hands and feet). No race is exempt but there is an apparent predilection of the disease for the negro race in this country.

Fever is unusual, despite even marked pulmonary involvement. The general malaise, night sweats, insidious onset and *arthralgia* may simulate various forms of arthritis.

About half of these patients show *skin lesions* (as small, firm, cutaneous nodules or diffusely thickened and infiltrated skin over the nose, ears and face). There is no itching or pain associated with the eruption.

Symmetrical *nodules* may appear *at the phalangeal joints* of the fingers, imparting a *knotty appearance* to the hands and fingers. The skin may scale over these projections. Although the fingers may feel stiff or tight, they are not tender or painful.

Many of these patients have a generalized *lymphadenopathy* with firm, discrete, non-tender nodes. The *spleen* and *liver* may be enlarged from sarcoid involvement of these organs.

Uveoparotid fever is an expression of sarcoidosis.

Laboratory findings in sarcoidosis include a slight hypochromic anemia, normal or slightly diminished leukocytes (with an increase in monocytes and eosinophils), *increased sedimentation rate,* negative blood Wassermann reaction, *increased blood calcium and phosphatase, increased plasma proteins* due to the increase in globulin (often with reversal of the albumin globulin ratio). On *roentgen examination* of the fingers, the medullary portions of the phalanges and metacarpals may show rarefaction, with small punched out areas appearing later in the small bones of the hands and feet (simulating gout or rheumatoid arthritis).

Most of these patients recover spontaneously.

Dermatomyositis:[36]

Arthritis of a few or many joints is occasionally present.

Etiology is unknown; there is a parenchymatous and interstitial myositis, involving a large part of the entire musculature of the body.

Symptoms include general malaise, headache, heaviness in the extremities, muscular pains, tiring easily after walking a few steps and finally complete incapability of muscular function; the patient lies in bed, unable to move his limbs, as though in a cast.

Characteristic pathologic changes occur in the skin and skeletal muscles with a perivascular lymphocytic inflammatory reaction.

The onset is more often subacute with dermatitis, muscular aching, weakness and a low grade fever. Within several months, the symptoms of both the *myositis* and *dermatitis* are usually apparent. The dermatitis is very variable and may simulate pellagra, toxic erythema, urticaria or light sensitivity. The dermatitis may disappear leaving the skin apparently normal or pigmented, or it may persist.

The involved *muscles* are usually *weak, tender* and *painful.* In the acute stages of the disease, the muscles may be painful even at rest. There is *no demonstrable intra-articular disease* although fibrous contractures and muscular atrophy may limit the range of motion.

Although relapses and remissions often occur over periods of months or years, the disease may end fatally in several weeks. In most instances, a *skin and muscle biopsy* are needed to confirm the diagnosis. It is often difficult to distinguish this disease from lupus erythematosus and scleroderma. Prognosis is poor and treatment is symptomatic.

Milkman's Syndrome (Multiple spontaneous idiopathic symmetrical fractures):

1. Involves the entire skeleton, flat and tubular bones.
2. Origin is in the cortex of the bone; it slowly encircles the entire bone, extending into the marrow cavity.
3. Symmetry of involvement of bones; this may be symmetrical as to direction and location of the lesions.
4. Progression of the process with periods of intermissions and exacerbations.
5. Insidious onset and unknown etiology.

6. May produce disturbance of gait—a waddling type.
7. May cause pain in the lower back and extremities.
8. Localized pain appears in the individual bones before there is any physical or roentgen evidence of the disease.
9. Crepitus is not found.
10. There is tenderness over the skeletal tissues.
11. *X*-ray examination shows bands or zones of increased radioparency. The bone appears as if the entire bony structure were erased at the point of involvement. These zones vary from a few mm. to 1 cm. in width. The bone above and below the transparency looks normal except for a thinning of the cortex and increased striations of the spongiosa and a moderate deficiency of the calcium content. The edges of the bones appear irregular. The transparent zones have lost all of their calcium and are composed of structureless supportive or ground substance.
12. The area of calcium deficiency begins in the cortex and gradually increases, extending across the bone transversely to its long axis.
13. There is no bending or bowing of bones and no deformity until late in the disease when complete separation, displacement and overlapping of the fragment may occur.
14. The transparent zones increase in number and width.
15. There is no attempt at repair and no callus formation; the fragment ends remain irregular and jagged.
16. The blood uric acid, urea nitrogen, calcium and phosphorus are normal.
17. The condition is usually misdiagnosed rheumatoid arthritis or sacroiliac sprain, neurosis or osteomalacia. Roentgen examination suggests the possibilities of fragilitas ossium, late rickets, multiple myeloma, parathyroid disturbance and hunger osteopathy.

Arachnodactyly (Spider fingers):[19,21]

A familial and congenital syndrome characterized by relaxation of ligaments permitting marked hypermobility of joints of the extremities (often with secondary contractures), generalized muscular hypodevelopment, abnormally long gracile fingers and toes resulting in spider-like resemblance, decreased amount of subcutaneous fat producing an appearance of emaciation and accentuating the appearance of length of the extremities.

Fingers capable of being distorted into grotesque positions due to marked hypermobility of joints because of relaxation of ligaments.

About 50 per cent of cases also have deformities of joints, especially of the feet; kyphosis; scoliosis due to inadequate musculature and ligamentous relaxation; high arched palate; prominent ears; marked sexual underdevelopment and other less important findings.

Most commonly found in children: tall, underweight and with disproportionately long extremities.

Commonly have flat feet, hammer toe and club foot.

Transmitted hereditarily as Mendelian dominant.

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CHAPTER III

THE SEDIMENTATION RATE

THIS test is very useful in the diagnosis and prognosis of some forms of arthritis. It is not a specific diagnostic test for any one disease.[29] *The sedimentation rate is consistently normal in healthy individuals (except during pregnancy).* It is increased markedly (as a rule) in acute rheumatic fever and rheumatoid arthritis, and is usually normal in uncomplicated cases of degenerative joint disease. The erythrocyte sedimentation rate is almost always elevated during the active stage of rheumatoid arthritis, varying from 30 to 125 mm. per hour by the Westergren method.[24]

The rate is increased in over 80 per cent of patients with chronic low grade infections (such as sinusitis, nasopharyngitis, etc.). Thus, in a patient with chronic sinusitis or chronic bronchitis, the sedimentation rate may be elevated. If the patient has concomitant degenerative joint disease, the physician must not be misled into thinking that the joint disease must be of an inflammatory nature merely because the sedimentation rate is increased. In other words, the sedimentation rate is merely an additional instrument which is useful in assisting in diagnosis and prognosis and in following the course of the process, and must not be relied upon solely, but only in conjunction with other factors.

The general practitioner has not yet appreciated the importance and simplicity of this examination.[13,42] It requires less apparatus, effort and time than the performance of a blood count and can be done by any physician in the office or at the bedside. There is nothing complicated in the procedure and, for practicing physicians, the results may be read directly without correction factors.

Most cases of rheumatoid arthritis show a greatly increased sedimentation rate; this is usually proportional to the extent and severity of the disease. As the disease subsides, the rate usually becomes slower and finally reaches normal in the inactive or "burnt-out" stage of the disease. The rate is usually between 30 and 100 mm. an hour (normal 8 to 10) by the Westergren technic. Practically all observers agree that this test is sufficiently reliable to give information of definite clinical value. It is especially useful in distinguishing fibrositis and degenerative joint disease from rheumatoid arthritis and rheumatic fever; in the two former disorders, the sedimentation rate is practically normal in most instances.[26]

For research work, the corrected sedimentation rate has been considered to be the most reliable single test for determining activity in rheumatoid arthritis.[1,35]

The Rourke-Ernstene and the Westergren methods are the two most commonly employed.[31] The Rourke-Ernstene technic is somewhat too complicated for the general practitioner. It is generally agreed that the Rourke-Ernstene method is the most reliable, but this is also the most

time-consuming and complex. The normal range for this method is 0.05 to 0.4 mm. per minute; an extremely accelerated rate may be as high as 2 to 2.5 mm. per minute.[30] The simplified sedimentation correction chart devised by Singer and Ordway will probably be of great value to laboratories and research workers. A recent clinical evaluation of the blood sedimentation test as a routine diagnostic procedure in more than 2000 cases showed this test to be of definite value when performed by a simple method (such as the Westergren technic) and without correction for anemia.[2] Some workers have concluded that anemia has little to do with the phenomenon of blood sedimentation.[11] The Westergren method is probably in more common use than others.

Physicians have shied away from this test because (1) some have thought that it required special costly apparatus; (2) some have been bothered by correction factors which have been advocated; (3) others have not known the normal values or the indications for this examination; and (4) physicians have been confused by the multiple technics described.[20]

Ham and Curtis have discussed in detail the influence of technical, erythrocyte and plasma factors and the quantitative comparison of five of the most commonly used sedimentation methods.[22] They have summarized these technics as follows:

1. *Rourke-Ernstene:* 3 cc. venous blood mixed with 0.013 cc. of 15 per cent heparin placed in special tube to height of 100 mm. corrected for erythrocyte concentration. Normal: 0.05 to 0.4 mm. per minute.
2. *Linzenmeier:* 1.5 cc. venous blood mixed with 0.4 cc. of 5 per cent sodium citrate solution; placed in special tube (5 mm. diameter) to height of 50 mm. Reading as minutes required for erythrocytes to fall to 18 mm. mark. No correction. No longer used to great extent in this country.
3. *Westergren:* Described later.
4. *Cutler:* Described later.
5. *Wintrobe-Landsberg:* Blood collected as in "1": normal reading 0 to 9 mm. in men and 0 to 20 mm. per hour in women.

Red blood cells in plasma or serum settle because their density is greater than that of the medium. The erythrocytes form aggregates or rouleaux of varying sizes; the sedimentation rate increases in general in proportion to the size of these rouleaux. The physico-chemical basis of the reaction involved in the sedimentation rate is uncertain. *The erythrocyte sedimentation rate is a rough index of the concentration of plasma fibrinogen,* except in those instances when the levels of serum globulins or lipids are abnormally elevated or when there is an advanced macrocytic or microcytic change in the red blood cells. The frequent correlation of sedimentation rate with plasma fibrinogen in infectious disease permits the former to serve as a rough index of the concentration of fibrinogen. Although the correlation between the sedimentation rate and the plasma protein fractions is usually very close, considerable deviations exist in some cases so that the sedimentation rate is not an accurate measure of any constituent of the blood.[22] Others state that the sedimentation rate depends upon the cell volume percentage,[19] fibrin percentage in plasma, temperature, or

the presence of a foreign protein in the circulation[25] (which may be the protein of fetal catabolism, of bacteria or of necrotic tissue).

Ham and Curtis have shown that numerous factors may influence the sedimentation rate. These include the height of the column of blood, internal diameter of the sedimentation tube, anticoagulant used, effect of standing, room temperature, inclination of the tube, and erythrocyte concentration and size.

Because of their simplicity, the *technics of Cutler or Westergren* are suggested for the general practitioner.

CUTLER TECHNIC

Add 4.5 cc. of blood to 0.5 cc. of 3 per cent citrate solution in a Cutler 5 cc. tube. This is inverted gently several times to permit mixing.

The tube is then placed in a *strictly vertical position.* It may rest in modeling clay or putty or in a rack.

Readings are made (by patient or physician) every five minutes (for an hour) of the distance to which the erythrocytes have sedimented; if it is impractical to read the levels at five minute intervals, one reading may be made at the end of an hour.

These are charted as shown in Figure 14.[10,11]

With this technic the corpuscles will not fall more than 8 mm. in an hour (for normal males) and 10 mm. (for normal females).

If the smaller Cutler tube (5 mm. internal diameter) is used, 1.8 cc. of venous blood is mixed with 0.2 cc. of 3 per cent sodium citrate. When this is placed in the small tube, the top of the column of blood reaches 50 mm. in height.[9]

Caution.—In *pregnancy*[14,15] the sedimentation rate may be increased; this is the only known physiologic process in which the sedimentation rate is rapid.

Correction for Anemia.—It has been thought, in general, that the sedimentation rate bears an inverse proportion to the volume percentage of the red blood cells and that the greater the anemia, the more rapid will be the sedimentation rate.[33] *Cutler and his associates feel that anemia per se does not cause a rapid sedimentation rate.*[11] They find the sedimentation rate normal even when pronounced anemia is present (if there is no associated infection or neoplasm). They claim that rapid settling of blood is due to the formation of large aggregates of erythrocytes or rouleaux and that the ability of the erythrocytes to form aggregates is a function which is specific for the particular plasma involved. Hospitals or clinics should correct the sedimentation rate for the degree of anemia present.[18,33,34,37] Cutler and his associates do not believe that it is possible to devise a formula for correction of anemia. Others have stated that if the sedimentation rate is but slightly increased and the patient is markedly anemic, the increase in sedimentation rate is (probably) accounted for by the anemia. If there is a marked degree of anemia associated with infection, the sedimentation rate may become rapid from this cause alone and in such cases

a rapid sedimentation rate is not a true index of the degree of activity of the disease.

The test is of little value if congestive heart failure is present, as this may delay the sedimentation rate (and often bring it within normal limits).[43]

In *allergic individuals, the sedimentation rate is often slower than normal;* this does not apply to serum disease and urticaria in which the sedimentation rate is usually increased.[27]

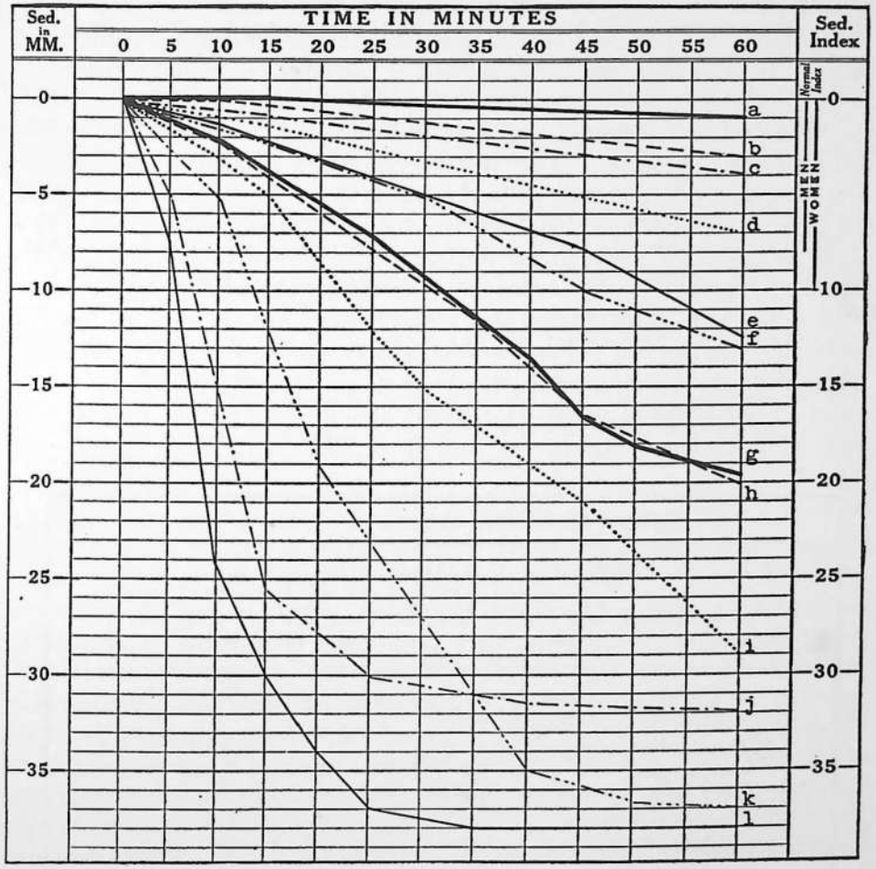

FIG. 14.—Sedimentation chart. *a* and *d*, healthy males; *b*, sickle cell anemia (R.B.C., 2,550,000); *c*, secondary anemia (R.B.C., 3,860,000); *e*, bleeding peptic ulcer (R.B.C., 1,360,000); *f*, coronary artery disease; *g*, primary pernicious anemia (R.B.C., 2,320,000); *h*, postoperative (nephropexy), (R.B.C., 5,220,000); *i*, carcinoma of the stomach (R.B.C., 2,180,000); *j*, lobar pneumonia; *k*, degenerated bleeding uterine fibroid (R.B.C., 1,860,000); *l*, carcinoma of lung (R.B.C., 2,400,000). (Cutler, Am. Jour. Med. Sci.)

The sedimentation rate is often prolonged (abnormally low) also in some virus infections and in certain cases of Brucellosis. In severe hepatic disease, the abnormally elevated sedimentation rate may decrease to normal coincident with a decrease in the plasma fibrinogen from above to below normal.

WESTERGREN TECHNIC[39–41]

Apparatus Required.—Sterile syringe (2 or 5 cc.), sterile Wassermann needle, alcohol sponge, solution of 3.8 per cent sodium citrate in distilled water, a special Westergren pipet at least 200 mm. long (which may be procured at most surgical supply houses at very low cost), a rack in which the pipet may be held in a vertical position and a small amount of plasticine or mercury into which one end of the pipet is placed to prevent leakage of the contained blood.

Technic.—One-half cc. of the sodium citrate solution is placed in a small (6 to 10 cc.) glass tube; to this is added 2 cc. of blood drawn by venipuncture with as little stasis as possible (without the use of a tourniquet if this can be done). The tube is inverted several times so that clotting does not occur. The Westergren pipet is filled to the 200 mm. mark and placed in a *strictly vertical position* in a rack, its lower end dipping into plasticine or mercury so that the blood will remain in the tube. This is kept at room temperature for an hour. At the end of this time the distance to which the erythrocytes have settled is measured (*i. e.*, the height of the column of clear plasma above the red cells); this, reported in millimeters, represents the sedimentation rate in one hour.

Acceleration of sedimentation rate may occur if the tube is not maintained in the vertical position; a slight inclination from the vertical may result in marked increase in the velocity of settling.

Clinical Application of the Sedimentation Test

In general, the sedimentation rate becomes rapid in the presence of inflammation, tissue damage (as in neoplasms), severe trauma, local and general infections, anemia and pregnancy.

Moderate abnormalities have been found in leukemia, hyperthyroidism, liver diseases with jaundice, coronary occlusion and thrombosis in general, acute and chronic nephritis, starvation, acute intoxication, etc.[22]

Although the sedimentation rate is usually normal during the first twenty-four hours of an acute infection (even though fever and leukocytosis are present), it is abnormal in most chronic infections.

Irregular fluctuations, as may be seen in leukocyte counts, do not often occur.

When the sedimentation rate indicates an active process, this returns to normal gradually as the patient improves; the test is of prognostic value and serves as an index of the relative severity of infection and tissue damage (especially in rheumatic fever, rheumatoid arthritis, tuberculosis, pelvic inflammatory disease, acute coronary occlusion, pneumonia, Hodgkin's disease, acute cholecystitis, etc.).

The sedimentation rate is normal in many diseases; if it is increased, however, it is certain that some pathologic process is present (unless the patient is pregnant). **A normal sedimentation rate does not always rule out disease.**

Normal Figures.—With the Westergren technic, the normal range of settling is 1 to 3 mm. per hour in men and from 4 to 7 mm. per hour in women. Figures of from 8 to 15 mm. denote a slightly accelerated rate; from 15 to 40, a moderately accelerated rate, and from 80 to 110, an extremely accelerated rate.

More recently, a micro-sedimeter has been placed upon the market, which utilizes only 1 drop of blood. This apparatus is of value where venipuncture cannot be done.

Rogatz described a **simple micrometric apparatus** for determining the sedimentation rate of the erythrocytes (the **Landau microsedimentation apparatus**—available from the Clay-Adams Co., Inc., 44 East 23rd Street, New York City). The apparatus and technic are reduced to a simple form, requiring only 0.05 cc. of blood. If this test is proven to be reliable, it may fulfill the requirements for an ideal method for the general practitioner.[32]

Lee has devised a "*Sedimentometer*" which photographs the sedimentation tube onto a moving light-sensitive surface.[28] This gives a continuous photosedimentation curve similar to those plotted on the Cutler graphs.

APPLICATION OF SEDIMENTATION TEST IN ARTHRITIS

One of the important applications of this test in arthritis is in differentiating rheumatic fever and rheumatoid arthritis on the one hand from degenerative joint disease (osteo-arthritis) and fibrositis on the other. Most investigators agree that in active rheumatic fever and in most cases of active rheumatoid arthritis the sedimentation rate is rapid, while in osteo-arthritis this is usually normal.[6,23,35] However, Price and Leichentritt have observed patients with clinically active arthritis with normal sedimentation rates while in other instances, the sedimentation rate remained persistently elevated for weeks after a complete subsidence of the acute process had occurred. In fibrositis, the sedimentation rate may be elevated occasionally but is within normal limits in the majority of cases. A return of the sedimentation rate toward a normal value is an indication of improvement; progression in the opposite direction suggests that the disease has become worse.[38] The sedimentation rate does not fluctuate from day to day as may occur with the white blood count, but improves gradually as the disease subsides. In one series of 100 cases of rheumatoid arthritis sedimentation rates (Westergren technic) were more than 25 mm. in 91 of these patients and the mean value was 71.6 mm. in one hour.[36] Most workers agree that the sedimentation rate is increased in practically all cases of active rheumatoid arthritis. However, the sedimentation rate is occasionally normal in the early months of the more insidious cases or in the first few weeks of some of the acute cases.[16]

In 100 cases of fibrositis, a sedimentation rate over 25 was observed in only 11 patients and the mean value was 11.6 mm. in one hour.

Many observers have found that the blood sedimentation rate is normal in chorea.[7] When other manifestations of rheumatic fever are present

along with chorea, the rate is usually fast. A fast sedimentation rate in an individual with chorea should be considered as confirmatory evidence of active rheumatic fever. Conversely, the presence of a normal sedimentation rate in uncomplicated chorea is strong evidence against its rheumatic origin.[8]

In pure chorea, in the absence of a history or other manifestation of rheumatic fever, the incidence of rheumatic heart disease is low.[32]

However, all of Bauer's 70 choreic patients had rapid sedimentation rates on admission; he believed that careful study will reclassify supposed choreics with normal sedimentation rates into other disease groups.[5] This is contrary to the findings of others who reported normal sedimentation rates in choreics without other rheumatic symptoms.[3,4]

It has been found that, for practical purposes, physically induced fever used therapeutically does not affect the sedimentation rate. An accurate determination of the sedimentation rate can be made at any time during or after a fever session.[17]

The sedimentation rate is increased in the active stages of gout so that it is of no value in differentiating this disease from infectious arthritis. Haden and Kinell found that the sedimentation rate in gout varied with the activity of the disease, being lower in acute subsiding cases than in acute cases and reaching its lowest level in inactive cases and its highest level in chronic progressive gout. Eighty-seven per cent of all gouty patients showed an increase in the sedimentation rate.[21]

In a series of 500 observations upon 220 patients, Dawson and his colleagues, using the Westergren method, found the following:[12]

The sedimentation rate was greatly elevated in active cases of rheumatoid arthritis, usually attaining values exceeding 30 mm. in one hour.

In rheumatoid arthritis, this paralleled to an extraordinary degree the extent and severity of the arthritic process.

Exacerbations of the disease were almost always attended by an increase, and remissions by a decrease of the sedimentation rate.

The sedimentation rate tended to return to normal in old, long-continued and arrested cases.

The sedimentation rate, although as a rule slightly elevated, rarely reached values greater than 30 mm. in cases of osteo-arthritis; in uncomplicated cases of degenerative joint disease, the sedimentation rate was usually normal.

Patients with "myositis," "fibrositis," "neuritis," etc., had a normal or only slightly elevated rate.

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CHAPTER IV

THE SYNOVIAL FLUID

THE joint cavity is really an enlarged tissue space lined by modified connective tissue. The synovial membrane is merely a group of connective tissue cells which form the inner wall of the joint capsule.

Joints normally contain a small amount of fluid which moistens and lubricates the joint surfaces. This is known as synovial fluid. The term synovia, from the Latin *syn* (with) and the Greek *ovon* (egg), describes well the sticky viscid nature of most synovial fluids. These are usually thin and glairy, resembling the white of an egg. Normal synovial fluid in human beings is clear, transparent, viscid, pale yellow and sticky. Only a few cc. are normally present in a joint, even at autopsy.

Kling believes that normal synovial fluid is not a degeneration product or a transudate but that it is elaborated by special cells and that its functions are lubrication and protection of the surfaces of the joint against acid metabolites.[30] Cherry and Ghormley state that synovial fluid is probably a diffusate, with added proteins.[17] Ropes and Bauer[38] agree that synovial fluid is probably a dialysate of blood plasma. The high alkalinity of normal synovial fluid and its lubricating and protective function depend upon its mucin.

Synovial membrane is a connective tissue structure with various sized villi on its inner aspect projecting into the joint cavity. It is well supplied with blood and lymph vessels and with nerves. Diffusible substances of small molecular size in homogeneous solution are removed from joints through the subsynovial blood capillaries rather than through the lymphatics. Passive exercise, by increasing intra-articular pressure and joint blood flow, markedly accelerates this rate of absorption. Absorption is markedly increased from acutely inflamed joints (both during rest and exercise). Diffusible dyes injected into the joints are also absorbed chiefly by capillaries and only to a slight extent by lymphatics.

Albumin and globulin in the synovial fluid are due to slight permeability of capillaries to these. Proteins are removed from joints mainly through the lymphatics. Synovial membrane is more permeable than most true membranes. Living bacteria enter the synovial fluid more readily than the urine or spinal fluid.[3]

Because of the marked increase in the amount of synovial fluid in some joints which are the seat of an arthritic process, aspiration is sometimes desirable. It is often possible to obtain further information concerning the type of arthritis by a study of this fluid. *In several thousand aspirations of joint effusions in all types of arthritis, Kling has never observed any ill effects;* these included aspiration of septic and tuberculous joints. Aspiration of joint exudate should be done carefully with strict asepsis; if performed in this manner, it is without danger. In rheumatoid arthritis,

the joint fluid is sterile. The finding of pathogenic microörganisms suggests a specific bacterial arthritis.

Chemical examination of synovial fluid in arthritis for total protein or non-protein nitrogen yields very little information of practical value in differential diagnosis. The amount of sugar in synovial fluid varies with the level of the blood sugar at the time of aspiration of the joint, the number of leukocytes present in the fluid and the presence of organisms. It is the same as blood sugar—80 to 110 mg. per cent, if fasting specimens are taken; the blood sugar level declines sooner after a meal than synovial fluid sugar.[23] In general, a smaller amount of sugar is found in synovial fluid when bacteria and a high cell count are present. Inasmuch as non-infected fluids also show low sugar values if the leukocyte count is high, these determinations cannot be used to differentiate between infected and non-infected synovial fluids. The non-protein nitrogen value can be correlated only with its level in the blood so that this assumes no diagnostic significance. Urea, uric acid and non-protein nitrogen diffuse completely through the synovial membrane; in general, the non-electrolytic distribution is consistent with that in blood plasma dialysate.

Normal Synovial Fluid

Synovial fluid is similar to plasma dialysate and is an example of tissue fluid in general.
Total leukocyte count: 200 cells or less per c.mm. (not affected by cytology of peripheral blood).
Average differential pattern.[3]
- Monocytes, 48 per cent.
- Lymphocytes, 25 per cent.
- Polymorphonuclears, 7 per cent.
- Clasmatocytes, 10 per cent.
- Unclassified phagocytes, 5 per cent.
- Synovial cells, 3 per cent.
- Unclassified cells, 2 per cent.
- Usually no eosinophils or basophils.

Erythrocytes are not normally present in synovial fluid.
Culture—negative.
Total protein—less than 2.5 per cent,[20] albumin/globulin—4; this varies greatly.
Non-protein nitrogen—same as blood—20 to 40 mg. per cent; fibrinogen, none. (Normal synovial fluid will not clot.)
No cholesterol or fatty acids.
Glucose—lower in synovial fluid than blood.
Mucin—0.14 to 0.85 per cent. Mucin is responsible for the osmotic pressure.
pH—7.4.
Specific gravity—1.008 to 1.015, average 1.010.
Viscosity—average—150 (varies from 2.8 to 400).

Coggeshall and his associates studied the synovial fluid aspirated at death from 29 normal human knee joints. The average cell count was 63 per c.mm. There were 63 per cent mononuclear phagocytes, 25 per

cent lymphocytes, 6 per cent polymorphonuclear leukocytes, 4 per cent synovial cells and 2 per cent unidentified cells.[18]

By examinations of the synovial fluid in human beings and in cattle, the following normal characteristics may be summarized:[1,21,30,36,40]

A transudate in the joints (such as may be present in congestive heart failure with peripheral edema or nephrosis) shows a rather low total protein content (usually not over 2.5 gm. per cent); in the presence of an exudation into the joint space, the total protein content usually rises above 2.5 gm. per cent. This increased amount of protein need not be accompanied by infection of the synovial fluid.

The functions of the synovial fluid are:

1. To lubricate the joint surfaces and permit motion of cartilaginous surfaces on each other with a negligible amount of friction.
2. To nourish some of the joint structures: Articular cartilage and possibly the superficial cells of the synovial membrane.
3. To bind acid (by means of the synovial mucin).

The function and importance of *mucin* in synovial fluid were not well understood until recently. Mucin is a glycoprotein, probably both an albumin and a globulin. It is similar to mucin of the subcutaneous tissue.

The synovial membrane does not form a closed cavity but invests the joint space in the manner of a sleeve. Its metabolism is similar to that of other adult tissue.[15] Crystalloid and diffusible substances are absorbed from the joint cavity into the venous circulation by the capillaries.[19] Adkins and Davies found that true solutions entered the capillaries and lymphatics with ease (most of the substance being removed in the blood stream). The globulin molecule could be removed by both routes, but above this sized particle, removal ceased by both routes.[1] This absorption is promoted by movement or massage. No openings have been found in the synovial membrane; the removal of particulate matter from the joint space is accomplished by phagocytosis and lymphatic transportation. The numerous lymphatic vessels which have been demonstrated under the synovial lining in normal joints are decreased in number or entirely absent in rheumatoid arthritis.[32]

There are no pathognomonic features of the chemical composition of synovial fluid in various forms of joint disease, except possibly the increase in bilirubin following trauma to a joint. Carbon dioxide, lactic acid, non-protein nitrogen and other substances have been found in joint fluid in about the same concentration as in normal blood.[16]

The decrease in glucose in joints of patients with rheumatoid arthritis is due at least in part to delayed appearance of the sugar.

The distribution of electrolytes is in general agreement with that expected from the Gibbs-Donnan theory of membrane equilibrium.[3] The protein content of synovial fluid is complex, consisting of a mucinous protein, serum albumin and globulin. The synovial membrane is somewhat permeable to protein molecules such as albumin and globulin.

The amount of protein entering the joint space varies with the amount

of inflammation. The proportion of globulin increases as the membrane becomes more permeable.

Egg albumin has been found in the knee joints of rabbits five minutes after its intravenous injection.[3] It (a polysaccharide protein combination) is the chief component of normal synovial fluid and the one upon which most of its physiologic action depends.[29,30] The mucin in synovial fluid is responsible for the main physical, biological and chemical properties of this fluid, although the real significance of this mucin is not well recognized. It provides most of the viscosity and lubrication and aids in the regulation of distribution of substances between blood and synovial fluid. Mucin has a base binding power ten times that of serum albumin. It is thought that mucin protects the articular cartilage from acid metabolic products which occur in inflammatory effusions by maintaining a high degree of alkalinity.[28] Ropes and Bauer are of the opinion that the mucin is formed in part by connective tissue cells around the joint and that it is added as fluid passes through the connective tissue around the joint.[37] Cherry and Ghormley believe that mucin in synovial fluid is a result of cellular disintegration, and not of secretion.[17] Kling states that mucin is the result of special cell activity, and not of degeneration.[30]

The *ferments* which have been found *in normal synovial fluid* include protease, lipase and amylase.[34] By means of these, fibrin, tissue débris or other products of inflammatory or traumatic reactions are digested. Dehydrogenases (enzymes that remove hydrogen from tissue metabolites) have been shown to be present in synovial membrane and cartilage.[7]

A fluid film is formed by the synovial fluid between the ends of the bones which can stand a pressure of 900 pounds per square inch.

The importance of proper lubrication of joints was recently demonstrated when it was found that the coefficient of friction in the stifle joints of horses (corresponding to the knee joint of man) was fourteen times as much in dry joints as in joints kept lubricated.[22] After several hours of mechanical motion in lubricated joints, no damage to the joint surface was observed; in dry joints, marked heat and grating were noted and the bony ends were worn down.

Protein is absorbed slowly from a joint. Proteins leave joints only by way of the lymphatics. Massage and exercise increase the rate of removal of protein from joints. Specific antibodies found in the blood may be carried over into the joint fluid.

Infectious joint fluids have decreased mucin due to an accelerated breakdown of mucin. The amount of mucin in traumatic joints is normal.

Joint fluid from patients with traumatic arthritis, degenerative joint disease, Charcot joints or osteochondromatosis is usually clear, does not clot and has a total leukocyte count below 1000, with less than 500 polymorphonuclear leukocytes per c.mm. In this group the protein concentration is less than 5.2 gm. per 100 cc.; the mucin precipitate (with acetic acid) is ropy and the solution is clear. There is only a moderate reduction of viscosity in the fluids of these patients and the serum fluid fasting sugar difference is less than 10 mg. per 100 cc.

If one aspirates a joint fluid which is turbid, clots, has a low viscosity, a total leukocyte count above 3000 with the polymorphonuclear cells above 500 per c.mm., a protein content more than 5 gm. per 100 cc. and a serum fluid fasting sugar difference of more than 20 mg. per 100 cc., he is dealing with fluid from a patient with rheumatoid arthritis or a specific infectious arthritis.[2]

Cytologic Technic for Synovial Fluid.—Using a white cell counting pipet, the synovial fluid is diluted 1:10 with a solution of physiologic saline to which methyl violet has been added (to make a 0.005 per cent solution). Both the leukocytes and the erythrocytes are counted in the same area. Differential counts are made by staining fixed smears (direct from fresh synovial fluid without centrifuging) with Giemsa or with any other blood stain used in the laboratory. If desired, the fresh undiluted fluid may be stained by supravital means using a mixture of neutral red and Janus green. If cell counts are low, one may centrifuge the joint fluid and stain a smear of the sediment to facilitate the differential counts.

Leukocyte counts per c.mm. joint fluid may be classified roughly as follows:

Low (up to 1000).
Slightly increased 1000 to 5000.
Markedly increased 5000 to 10,000.
High (over 10,000) as in acute infections and septic types of arthritis.

The variations of findings in synovial fluid are as follows:

Rheumatic Fever.—Preponderance of high cell counts.

Differential count: In the early stages there is an increase of polys (averaging 90 per cent); later there may occur many large phagocytic cells.[33]

Rheumatoid Arthritis.—Preponderance of high cell counts.

Differential count: Usually a marked predominance of polys (about 50 to 90 per cent).

Degenerative Joint Disease.—Low or slightly increased cell counts.

Differential count: Usually a predominance of lymphocytes (50 to 60 per cent); polys not more than 15 per cent; increased numbers of synovial lining cells in some cases.

Gonorrheal Arthritis.—Usually very high cell counts.

Differential count: Usually a predominance of polys, averaging about 90 per cent of the cells.

Gonococcus complement fixation test done with synovial fluid—positive in about 75 per cent of cases. This is seldom positive unless a positive reaction is also obtained with the patient's blood. The incidence of positive reactions with blood is somewhat higher than with synovial fluid so that for practical purposes this examination should be done only with the patient's blood; only confirmatory data are usually obtained by performing the test with synovial fluid.

Anaërobic culture of synovial fluid will reveal gonococci more frequently than will a smear of this material. Gonococci tend to localize and survive in areas supplied with large amounts of mucin (urethra, cervix, conjunctiva, joints).[24]

Charcot Joints.—Cell count usually low or slightly increased.

Differential count: Predominance of polys (from 25 to 70 per cent).

Tuberculous Arthritis.—Cell count usually high.

Differential count: Predominance of polys (from 60 to 90 per cent).

Guinea pig inoculation of the synovial fluid may be done.

Miscellaneous Types of Joint Disease.—*Intermittent hydrarthrosis:* Cell count slightly increased; predominance of polys (50 to 70 per cent).

Traumatic arthritis: Cell count slightly increased, rarely more than 1000 per c.mm.;[4] polys may vary from 10 to 90 per cent. *Icterus index above 5 units is highly indicative of traumatic etiology (with rare exceptions).*[31] Red blood cells are often present; they may have frank intra-articular hemorrhage.

Syphilitic arthritis: Cells slightly increased; polys or lymphocytes may predominate.

Acute streptococcic arthritis: Cell count very high with from 90 to 100 per cent of polys.

Scurvy: Total cell count normal in one case with a predominance of monocytes.

Joint effusion with *cardiac failure:* Cell count low; polys and lymphocytes about equal (approximately 25 per cent each).

Method of Aspirating Joint Fluid.—The *surface over the joint is cleansed* with soap and water, iodine and alcohol in order. *Sterile towels* are draped in such a manner that the *field* is *aseptic.* The procedure should be considered as a surgical one and strict asepsis should be observed throughout; contamination of the joint cavity must be guarded against. The skin overlying the area of puncture, subcutaneous tissues, deeper tissues and joint capsule are *infiltrated with 2 per cent procaine hydrochloride.* It is preferable to use a *short beveled lumbar puncture type of needle in the aspiration of fluid from joints;* the stylet should not be removed until the joint space has been entered. *A large bore needle* (15 or 16 gage) is advised; this will not become clogged by thick exudative material. The *joint fluid may be removed by suction with a 50 cc. syringe.* If desired, air may be reinjected (Caution! be sure that the needle is not in a blood-vessel). Kling prefers to enter the joint cavity from its most prominent and lowest point. In the case of the knee joint, he usually aspirates the suprapatellar bursa, entering this about 2 inches above the patella.

If desired, one may use an aspirating syringe with a side-cock adapter, as much fluid as desired can be removed from the joint without detaching the syringe from the needle.[8] This method has the advantage that if it is desirable to inject air for visualization of the joint space or for therapeutic purposes, it may be done by withdrawing air through the side tip into the syringe and then injecting this into the joint cavity.[35,39] Other forms of apparatus for inflation of joints have been described.[5,6] Various authors[9–14] have discussed the technic and value of *arthroscopy,* joint biopsy and visualization of joints by injection of drugs.[25–27]

If *samples* are desired *for chemical analysis,* these should be placed in a tube containing sodium oxalate crystals so that clotting will not occur.

Gonococcus complement fixation tests or Wassermann tests may be performed upon this fluid but will not usually be positive unless the reaction of the peripheral blood is also positive. *A practical routine examination of joint fluid should include:*

Appearance; quantity; turbidity; reaction; specific gravity; clot formation.
Total cell count.
Differential count.
Gram stain of centrifuged sediment.
Protein concentration.
Culture in anaërobic jar if gonorrhea is suspected and routine culture for other bacteria.
Guinea pig inoculation if tuberculosis is suspected.
Icterus index (if traumatic arthritis is suspected).
Viscosity and sugar content are performed routinely by some.

The *significance of certain features of examination of joint effusion* as stressed by Kling is as follows:[29]

Bilirubin: Most inflammatory effusions—under 5 units; traumatic effusions—over 5 units.

Viscosity: High in chronic arthritis; low in transudate or acute inflammation.

Reaction (pH): Acid in septic arthritis; repeatedly alkaline reaction indicates favorable prognosis.

Differential count: Acute stages (regardless of etiology)—predominance of neutrophils; lymphocytes and monocytes increased in chronic stage.

Quantity of fluid: Large in acute and severe inflammation; small in mild, chronic arthritis.

Little special information can be obtained by chemical studies on the synovial fluid, which cannot be learned from an examination of the patient's blood. This applies also to agglutination tests, complement fixation and Wassermann tests. Done with synovial fluid occasionally, however, these tests may be positive using joint fluid while similar tests on the patient's blood are negative.

If experimental studies are being undertaken in this field, one should remember that the total number of nucleated cells in synovial fluid may increase postmortem but that there is very little change in individual cell percentages.[40] The cells found in the synovial fluid are not affected by variations in the cytology of the blood to any great degree. For those persons seeking further information in this field, one should consult the treatise on the synovial membrane and the synovial fluid by Dr. David H. Kling,[30] of Los Angeles, and the excellent summary by Bauer and his coworkers on the physiology of articular structures.[3]

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CHAPTER V

ETIOLOGY OF RHEUMATOID (ATROPHIC) ARTHRITIS

The cause of rheumatoid arthritis is still unknown. Precipitating factors include fatigue, exposure to cold, acute infections, trauma, emotional strain, climatic changes, etc.

Most of the evidence presented to date suggests that rheumatoid arthritis is at least in part an infectious disease; in favor of this are the fever, tachycardia, rapid sedimentation rate, leukocytosis, lymphadenopathy, occasional splenomegaly, inflamed articular structures, high titer of serum agglutinins to the Streptococcus hemolyticus and serum precipitins to the carbohydrate and protein fractions of this organism.[56,69] However, it has frequently been pointed out that some of the clinical features which suggest that rheumatoid arthritis is an infectious disease are also exhibited by gouty arthritis, which is considered to be a metabolic disorder. Proponents of the bacterial theory of etiology suggest direct bacterial invasion of the joint structures, the emanation of bacterial toxins from an infected focus or the reaction of bacterial allergens with hypersensitive joint tissues.[26] Although many organisms have been incriminated, none has yet been found to be causative.

Some of the theories concerning its etiology include: (1) focal infection; (2) bacterial allergy; (3) virus infection; (4) vitamin deficiency; (5) metabolic disorders: carbohydrate; protein; food allergy; calcium; sulfur; (6) traumatic; (7) circulatory disturbance; (8) neurogenic; (9) endocrine.

The *infectious theory* presupposes infection with bacteria, protozoa or viruses.[33] There is little evidence that the latter two play any important rôle in the etiology of this disease. Most clinicians treating arthritic patients believe that bacterial infection, most likely streptococci (especially the hemolytic group) is in some way of etiologic importance.

It is probable, although unproven, that some as yet unidentified microbe plays a rôle in its etiology. Many other factors may contribute or prepare the soil for such an infection; these include heredity, poor environment, trauma (whether accidental, occupational, recreational, etc.), endocrine imbalance, dietary and vitamin deficiency, associated diseases, psychic shock, exposure to cold, worry, etc.

Recent experimental work by Angevine, Rothbard and Cecil suggests that bacterial allergy may play a definite rôle in the etiology of rheumatoid arthritis.[10] It was found that a smaller number of intravenously injected bacteria were required to produce arthritis in rabbits which had received previous intravenous injections with heat-killed streptococci than in normal or intradermally immunized animals. In other experiments, these workers injected repeated small amounts of heat-killed streptococci or nucleoprotein fraction into the right knee joints of rabbits. When these animals were later injected intravenously with live cultures of the same

bacteria, the previously injected joints were more susceptible to infection than other joints injected with heat-killed staphylococci or horse serum.

Although focal infection has been incriminated for more than thirty years as a cause of rheumatoid arthritis, *definite experimental evidence on this point is difficult to obtain.* At the present time, the pendulum is swinging away from complete acceptance of the theory of focal infection in rheumatoid arthritis.[11,67,78]

One cannot say that a focus of infection is responsible for rheumatoid arthritis. In some cases, no foci can be found, or if discovered, their removal may not result in improvement of the arthritis; in others, improvement may occur without removal of foci. Foci of infection should be viewed as "portals of entry" of infection, *i. e.*, organisms or toxins may gain entrance to the blood stream and then to the joints through such portals, but there is not necessarily a constant or even intermittent dissemination of toxic material from such foci. A detailed report on the etiology of rheumatoid arthritis has been presented by the editorial committee of the American Rheumatism Association.[44,46,47]

Direct evidence concerning the possible infectious nature[54] *of rheumatoid arthritis* has been obtained from cultures of blood,[41,44] foci of infection,[6,49] lymph nodes,[9] stools,[8,86] joint tissues and fluids, and from studies of tropism and cataphoretic velocity of bacteria isolated from these; indirect evidence is derived from skin tests to various bacteria,[14–16] and examinations for agglutinins,[57,69,71] precipitins, antifibrinolysins, antistreptolysins and complement fixation reactions.

Blood cultures in rheumatoid arthritis have varied from "consistently negative"[24] to 69 per cent positive.[47] (Streptococci have been found[42] in 6 per cent of more than 5000 routine blood cultures in various diseases, including pernicious anemia, leukemia, colitis, meningococcus meningitis, pyelonephritis, rheumatic fever, rheumatoid arthritis and chronic rheumatic heart disease). Most workers at present find that blood cultures are negative in patients with rheumatoid arthritis.

In various studies, from 50 to 90 per cent of patients with rheumatoid arthritis possess agglutinins to hemolytic streptococci (usually in high titer).[1,46,95]

Recent investigators find that only about 40 to 50 per cent of patients with rheumatoid arthritis show a significant agglutination of strains of Lancefield's group a hemolytic streptococci with their serum.[55] Antistreptolysins are present only in early cases.[27] More than 75 per cent of patients[38,93] with rheumatoid arthritis show positive skin reactions to hemolytic streptococci; however, no one specific bacterium has been found to be the cause of chronic arthritis.[48] Isolation of streptococci from joint fluid has been reported[39,40] and denied.[28] Angevine and his associates made extensive cultural studies on special media, including the chorioallantoic membrane and inoculation into mice on 85 patients with rheumatoid arthritis and 21 with rheumatic fever. No organism of significance was isolated consistently.[2]

Angevine[1] has reported careful cultural studies of blood, synovial fluid

and tissue and subcutaneous nodules, observing all possible precautions including dust-free, air-filtered rooms, wearing of masks by operators, use of rubber bulbs on pipets, etc. In 83 patients with rheumatoid arthritis, he was consistently unable to isolate any organism of significance. In addition, he made special cultural studies for the presence of pleuropneumonia-like organisms in synovial fluid and tissues and in subcutaneous nodules of 13 patients with rheumatoid arthritis, but found none. Results were also negative in 22 patients with rheumatic fever similarly studied. Preston studied the fluid aspirated from the joints of 19 patients with typical rheumatoid arthritis; he did not find any organism related to the pleuropneumonia group.

Other generally accepted factors concerning rheumatoid arthritis are as follows:[26] (1) Rheumatoid arthritis is a disease of temperate climates, the incidence of the disease diminishing as we reach the equator; there is said to be little rheumatoid arthritis in the tropics.[13] (2) There is a definite tendency for more than one member of a family to be affected. (3) Although most cases occur in the thin, asthenic, flat-chested type, rheumatoid arthritis may occur in individuals of the thick, barrel shaped type. (4) More than 80 per cent of cases of rheumatoid arthritis begin between twenty and fifty years of age, with a peak at thirty-five years. (5) Females are much more frequently affected (3:1) than males. (6) Race exerts little influence upon its occurrence except that whites are affected more frequently than negroes. (7) Other determining influences include poor home environment, poor hygiene, hardships, trauma, nervous shocks, repeated pregnancies, poverty, grief and family worry.[17] The rôle which may be played by the paraneuritic nodules found by Freund, Leichentritt and others is as yet unknown. These small nodules, found around nerve tissue and in muscles, are thought to be the result of some systemic disturbance because of their widespread occurrence.

More recently, others have postulated that spasm of small peripheral veins may play a rôle in the etiology of rheumatoid arthritis.

Another theory offered recently is that rheumatoid arthritis is due to a derangement of the hyaluronic acid metabolism. Hyaluronic acid is a mucopolysaccharide found in synovial and subcutaneous tissues. According to this hypothesis, arteriolar spasm results in the delivery of an enzyme to cells engaged in the synthesis of hyaluronic acid. Perversion of this synthesis is said to give rise to a complex antigenic carbohydrate (which arouses bacterial agglutinins) and to an irritant protein, which latter is unable to escape from the locality and produces the clinical manifestations.

Under certain experimental conditions, *overdosage* with *desoxycorticosterone acetate elicits a polyarthritis* resembling histologically that seen in acute rheumatic fever. These joint lesions were *more readily produced* by *desoxycorticosterone acetate in adrenalectomized or thyroidectomized* than in intact *rats*, especially if they were exposed to cold. These joint lesions are often accompanied by the appearance of Aschoff nodules in the heart, and at times also by periarteritis nodosa. The marked similarity between these experimentally produced lesions and the manifestations of rheumatic fever

were interpreted as indicating that the adrenal cortex may play an important rôle in the pathogenesis of rheumatic fever and possibly rheumatoid arthritis in man.

Selye and his associates showed that arthritic lesions rarely developed in the intact rat treated only with desoxycorticosterone acetate, but that such *lesions developed with great frequency* in similarly treated animals if the thyroid or adrenals were removed, and especially so if **the animals were kept in cold surroundings.** It was shown that unilateral nephrectomy and sodium chloride treatment increased the sensitivity of rats to many overdosage effects of desoxycorticosterone acetate.

Albino rats were sensitized by ablation of the left kidney five days before injections were begun; 1 per cent aqueous sodium chloride solution was given instead of drinking water, beginning on the day of operation. Ten of these animals were then bilaterally adrenalectomized and an equal number were thyroidectomized. Each animal was then given 2 mg. of finely ground desoxycorticosterone acetate crystals subcutaneously twice a day in an aqueous suspension containing 20 mg. per cc. *Signs of arthritis were seen first on the fourteenth day of desoxycorticosterone acetate treatment;* at that time, 6 animals in the adrenalectomized group and 2 in the thyroidectomized group showed hyperemia and swelling of the tarsal joint region of one or both hind feet; prominent swelling of other joints was usually absent. The swollen areas were tender and the swelling tended to appear and reappear irregularly. In several animals of the control group (without surgical intervention), injections of desoxycorticosterone acetate were followed by the appearance of arthritic manifestations. Autopsy findings revealed macroscopic evidence of nephrosclerosis, periarteritis nodosa or rheumatic nodules in the heart in almost all animals of all of these groups.

Another group of rats was sensitized by ablation of the left kidney and was then given 1 per cent sodium chloride solution to drink; adrenalectomies were then performed. These animals developed no arthritis in fourteen days. They were then placed outdoors for twenty-four hours on a cool, windy winter day; at the end of this twenty-four hours, 4 of the 6 rats had enormous tarsal swellings. This led these investigators to assume that temperature changes play an important rôle in the pathogenesis of the arthritis.

Arthritides have been found to be common in women who do cleaning work which requires spending a good deal of time with their hands in cold water.[63]

Although the causative agent in these experiments seemed to be the desoxycorticosterone acetate, various factors were effective as *predisposing agents* in the production of the joint lesions—namely *exposure to cold, thyroidectomy* or *adrenalectomy*—all of which greatly facilitated the production of the arthritis. The detrimental effect of the adrenals and thyroid on thermoregulation may be involved in this production of arthritis experimentally.

Selye reviewed some of the literature on the *relation of endocrine disturbances to arthritis.* Various workers have commented upon the frequency

of rheumatoid conditions in hypothyroidism[31,59,66] and its occasional occurrence in Addison's disease[23] and status thymicolymphaticus.[74]

Joint lesions have also been reported often associated with hyperfunction of the adrenals or thyroid,[29,52,70,92] as well as in Cushing's syndrome.[62] The *arthralgias of the menopause* are also well known, as well as the development of true rheumatoid arthritis at this time.[53,65,73] Most observers up until now have thought that the occurrence of rheumatoid arthritis in a woman at the menopause was a coincidental phenomenon and that the menopause was not usually etiologically related. Joint lesions may also appear during or following the production of an artificial menopause.[51,91] Intermittent hydrarthrosis has also been reported with reaccumulations of fluid in the joint cavity with direct time relation to the sexual cycle.[5,79,83] In this connection, the disappearance of many of the manifestations of rheumatoid arthritis during pregnancy and its reappearance following delivery—are worthy of mention.[45]

Various investigators have speculated on the rôle *of the pituitary* in the production of some cases of arthritis.[12,32,37,42,50,61,76] The hypertrophic joint changes (osteo-arthritis) associated with acromegaly are well known; this usually has a predilection for joints of the spinal column. Joint lesions have also been reported in individuals with hypopituitary dwarfism,[93] diabetes insipidus[43] and dystrophia adiposa-genitalis;[84] however, it is difficult to formulate the possible etiologic rôle of the pituitary gland in arthritis. The possible rôles of bacterial allergy, endocrine disturbances, sulfur, diet, vitamin deficiency, and trauma have been made the subject of separate sections.

There is no proof that lack of any specific food is a causative factor in rheumatoid arthritis; however, the frequency of evidence of deficiency states in patients with rheumatoid arthritis suggests that this condition may increase the food requirements, especially of the vitamin B complex.[3]

Evidence in favor of the various metabolic theories is unconvincing. The majority now believe that any alteration in carbohydrate metabolism is not related causally to the arthritic process. Sulfur metabolism has been shown to be normal in patients with rheumatoid arthritis. Although calcium and phosphorus excretion is sometimes increased in these patients, it is no greater than that which follows disuse and normal values are found for fasting serum calcium, phosphorus and phosphatase.

The vitamin C deficiency seen in patients with rheumatoid arthritis is found in many other chronic disorders; administration of large doses of ascorbic acid does not alter the course of the disease.

The cause of rheumatoid arthritis is not fully explained by the abnormal peripheral circulation. No food allergy has been shown to be causally related to this disorder.[55]

The incidence of rheumatic disease falls off definitely and sharply as one approaches warmer climates. Northern European spas have large numbers of patients with rheumatoid arthritis while those in southern Europe have very few.[89]

In Great Britain, it is generally believed that cold and damp conditions,

clay soils and seaside localities are all potentially unsuitable for the rheumatic patient; however, Scandinavian countries usually place their sanatoria for the treatment of rheumatism by the seaside. Local patriotism may have some influence on opinions in this matter.[89]

In Germany and Scandinavia rheumatic fever and rheumatoid arthritis are regarded as having a common etiology and the development of rheumatoid arthritis in a patient with previous rheumatic fever is not unusual.[89] The incidence of rheumatic heart disease in patients with rheumatoid arthritis is discussed later.

The *eclectic theory* attempts to explain the origin of rheumatoid arthritis from a dysfunction or imbalance of the nervous,[90] peripheral vascular and gastrointestinal systems. Some of the findings which have been mentioned as the basis for a possible etiologic relationship between disturbances of the peripheral vascular system and rheumatoid arthritis include increased sweating, cold hands and feet, decrease in capillary circulation in the nail beds, symptomatic improvement following vasodilating procedures, and delayed removal of sugar from the blood.[69] However, Myers has noted that no vasoconstriction is found in the periarticular tissues of involved joints. Furthermore, it has been pointed out that rheumatoid arthritis is infrequent in vascular diseases such as Raynaud's disease, scleroderma, thrombo-angiitis obliterans and arteriosclerotic occlusions.[56] Also, in opposition to this theory it has been shown[7,68] that gastric anacidity (if corrected for age) is no more common in arthritis than in control patients; colonic abnormalities,[4,72] if present, cannot be proved to be a causative factor in rheumatoid arthritis; and *no constant deviations are found in the gastrointestinal tract in patients with rheumatoid arthritis.*[25]

Vasoconstriction of smaller blood-vessels, especially those of muscles, has been observed in arthritic patients.[75] Fletcher[35] has stated that the majority of these patients inherit an asthenic body build, with an unstable nervous and vasomotor system, together with lack of intestinal tone and that some unknown toxic agent has engrafted an arthritis onto this soil.[35] Others did not consider circulatory changes of etiologic importance inasmuch as the slow blood flow and decreased surface temperature did not consistently parallel the activity or extension of the arthritis.[58,60] Some investigators have noted that some very ill arthritic patients have warm hands.[30]

Sabin produced in mice a progressive, proliferative polyarthritis which clinically and pathologically resembles human rheumatoid arthritis.[81] Arthritis developed in practically 100 per cent of mice when 0.5 cc. of a twenty-four hour culture of a filtrable pleuropneumonia-like microörganism was given intravenously (or 1 cc. intraperitoneally). Swelling of the joints appeared as early as four or five days after the injection. Fusiform swellings of isolated digits occurred frequently and new joints were involved as others receded. A chronic, progressive arthritis occurred in one or more joints and ankylosis of the knees was not uncommon. These organisms were cultivated from the chronically affected joints as long as seventy days after intravenous injection. The mice appeared in good

health except for the arthritis; none died from the infection. Pathologic changes were confined to the joints and included proliferation of synovial membrane, capsule, perichondrium and connective tissue, and probably of the endosteum and the epiphyseal marrow directly beneath. Sabin previously isolated these organisms from the brain of a normal mouse.[82] In studying infectious arthritis of rodents due to pleuropneumonia type of organisms,[34] it has been found that gold compounds have protected against the inoculated disease and arsenicals have had some prophylactic action, but not as much as gold. None of the sulfonamide derivatives had any effect on this process. Swift and Brown[87] cultivated pleuropneumonia-like microörganisms from arthritic exudate of a patient with rheumatic fever and from an excised erythema nodosum nodule, but later reported that these microörganisms probably originated from the mice inoculated with the exudate; they were unable to grow these germs directly from the exudates or tissues of patients with rheumatic fever.[88] Freyberg could not obtain cultures of pleuropneumonia-like organisms from synovial fluid, blood, subcutaneous nodules or synovial tissues of patients with rheumatoid arthritis.[36]

Arthritis is a frequent complication of other diseases in animals known to be caused by the organisms of bovine pleuropneumonia and the mastitis of sheep and goats. Preston[77] isolated pleuropneumonia-like organisms from spontaneous arthritis in rats; he was unable to obtain any organism related to the pleuropneumonia group from fluid aspirated from the joints of 19 patients with typical rheumatoid arthritis.

Rothbard has produced arthritis in albino rats by group A hemolytic streptococci.[80] Collier and Esseveld have discovered a spontaneous infectious polyarthritis in wild and domestic rats in Java.[18,19] This shows features of parallelism with chronic rheumatoid arthritis in man and may yield information of practical importance.[20] A fulminating type of polyarthritis could be produced in normal rats by scarification or by plantar, intraperitoneal, intrapleural or subcutaneous injection of the arthritic exudates.

Collier and Staverman described a spontaneous arthritis of rats (hypertrophic osteo-arthritis) which might attack all joints in varying degree. The hypertrophic changes are so great that they resemble the changes seen in tabes and syringomyelia.

These workers inoculated into series of rats pericardial fluid, blood and joint fluid respectively. In the second transmission (passage), there developed a clinical and roentgen picture which was indistinguishable from that seen in spontaneous polyarthritis of rats. It was possible to inoculate rats from one patient with joint exudate and twice with citrated blood; after a long incubation period the rats developed a disease clinically identical with spontaneous arthritis of rats.[21]

Arthritis in swine has been shown to be due to an organism, the Erysipelothrix rhusiopathiæ.[22] This form of arthritis bears some relationship to the human form of the disease, but studies in this field are at present rather inconclusive.

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CHAPTER VI

PATHOLOGY OF RHEUMATOID (ATROPHIC) ARTHRITIS

THE *joint cavity* is really a large tissue space surrounded by modified connective tissue (synovial membrane or capsule on the lateral aspects) and by hyaline cartilage above and below. This cavity is derived embryologically from a mesenchymal cleft. The stability of most joints is due mainly to the support of surrounding ligaments aided by tendons, joint capsules and muscles; due to the pull of tendons, much of the joint cavity is normally collapsed. Intra-articular pressure has been estimated to vary between −2 and −12 cm. of water.

The *synovial membrane* is not a true membrane but a connective tissue structure with a smooth glistening inner aspect, especially where it lies over tendons and ligaments, and a dull surface at other areas.[3] The intima of the synovialis is firmly adherent over tendons and menisci and extends for a short distance on to the margins of the articular cartilage. The outer layer is fibrous and blends with the perichondrium and periosteum. Although these two layers can be distinguished microscopically, there is no definite line of demarcation. The synovial membrane is well equipped with blood and lymphatic vessels, is sensitive to stimuli and contains sensory nerves; the rich lymphatic network drains into the deep lymphatic vessels. Various sized villi project from the synovial surface into the joint cavity.

The synovial tissue has a high resistance to tear and is very pliable, permitting it to withstand severe stresses of joint motion. It is more permeable than true membranes; living bacteria can enter the synovial fluid more readily than the urine or spinal fluid. It is also somewhat permeable to protein molecules, such as albumin and globulin.

The redundant synovial membrane, surrounding the joint space as a sleeve, is attached to the margins of the articular cartilage near the epiphyseal line; it covers practically all of the joint except the cartilage. The synovial membrane has marked powers of regeneration, even if destroyed in large portions. Following chemical, mechanical or bacterial injury, this tissue proliferates extensively. The firm fibrous tissue comprising the joint capsule adheres to and encloses it.

The articular ends of bones are composed of a cancellous interior and a thin coating of compact bone; vascular connective tissue is present only in the cancellous bone.

The bones are bound together by the joint capsule which consists of an outer fibrous and an inner synovial layer.[25]

Hyaline cartilage usually covers the articular surfaces of bone. This is bluish white in color with a ground glass appearance, and is rich in matrix and relatively poor in cells. The number of cellular elements decreases with age. The lateral margins of the articular cartilage are covered by a

delicate extension of the synovial membrane. Human adult cartilage is avascular and contains no lymphatics or nerve tissue.

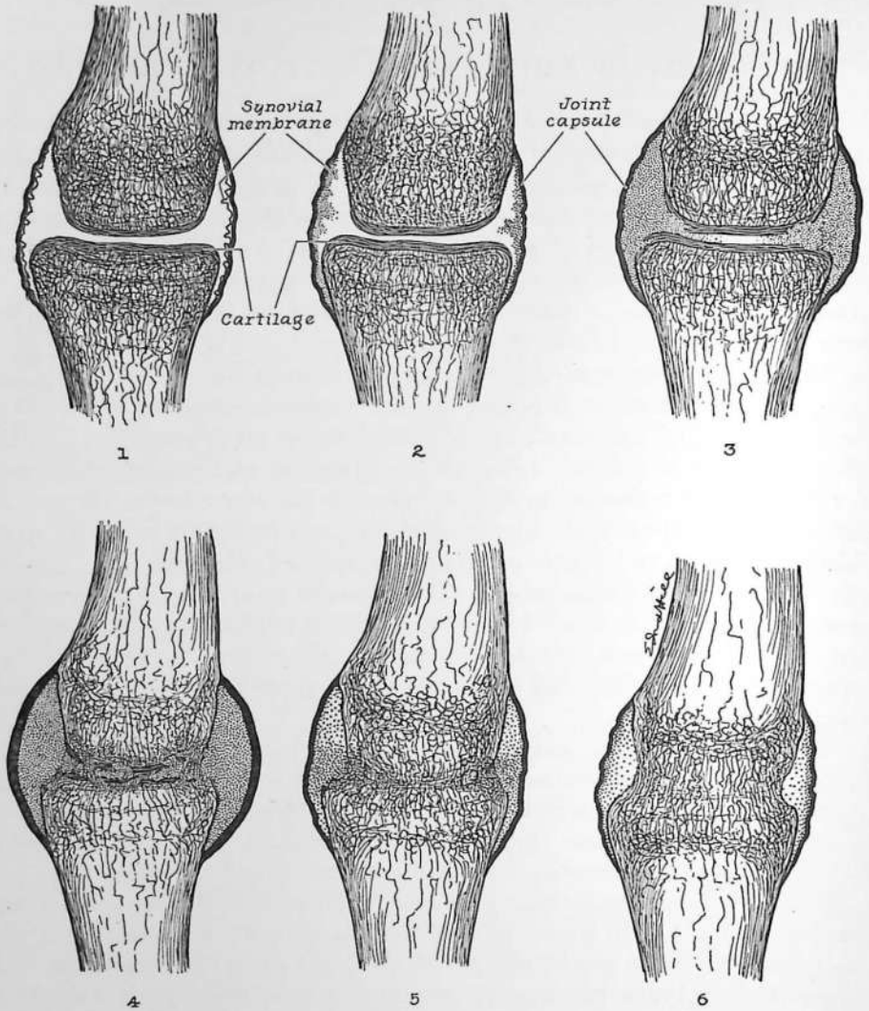

FIG. 15.—**Pathology of rheumatoid arthritis.** *1*, Normal joint, with joint space shown abnormally distended in order to portray synovial membrane and its reflection. *2*, Proliferation and inflammation of subsynovial tissues; outpouring of fluid into the joint: synovitis. *3*, Further distention of the joint with fluid, stretching the capsule; ingrowth of synovial tissue with destruction of cartilage. Decalcification of bone. *4*, Hypertrophied, inflamed synovial tissue covers cartilage as a pannus, destroying large areas of cartilage. Further distention of the joint; further demineralization of bone. Thickening of joint capsule. *5*, Almost complete destruction of joint cartilage; beginning bony ankylosis. Decrease in joint swelling. *6*, Bony ankylosis. Some hypertrophied synovial tissue remains. Joint swelling practically disappeared.

Most of the articular cartilage is nourished by synovial fluid. This may take place along the intercellular system of fibrils as a physical process. Bony loose bodies in joints become necrotic while free bits of cartilage

remain viable in joints for weeks. The numerous small capillaries in the delicate extension of synovial membrane covering the lateral portion of the articular cartilage may account for the better nutrition and healing ability of this portion than of the central zone.[15] The deeper parts of cartilage may receive some nourishment from subarticular vessels. Degenerative changes begin earlier in the central area than in the lateral margins, probably due to the difference in nutrition of the two parts.

Cartilage has a very limited reparative ability. Lesions produced in cartilage in animals show only slight proliferation after many weeks. If the cartilaginous wound extends into subchondral bone or lies near the synovial membrane or perichondrium, pannus may rapidly fill in the defect.[3]

Articular cartilage contains from 67 to 77 per cent of water (as compared with an average of 44 per cent water in bone). The former is practically completely elastic. This is an important quality because it receives all of the blows and jolts given to the skeleton.

Cartilage possesses a sluggish respiration, decreasing with age corresponding to the decrease in the number of its cellular elements. Glycolysis of the synovial membrane and cartilage is approximately the same as that of other adult tissues.[8] Articular cartilage contains glycogen, collagen, chondroitin sulfuric acid, lactic acid and calcium salts; its exact chemical composition is not accurately known.

It is protected against acid metabolites by the acid binding ability of mucin and by the antitryptic quality of synovial fluid against the trypsin elaborated by leukocytes in purulent effusions.

Endocrine factors may play a rôle in the growth of cartilage. In young pigs, injections of anterior pituitary extract have produced hypertrophy and hyperplasia in the transitional zone and later liquefaction and ulceration of the articular cartilage; in adult animals, a predominance of degenerative changes was noted. Gonadectomy in immature subjects produced similar sequelæ.[3] The growth and differentiation of euhyaline cartilage in normal young guinea pigs was enhanced by thyroid substance given orally for from two to thirty days.

In some instances, tendons are intimately associated with the joint capsule and may replace the fibrous layer. Bursæ often communicate with a joint cavity; in other instances they are distinct and lie between tendons and bony prominences. Histologically, they resemble synovial tissue. There is a negative pressure of from 60 to 70 mm. of water within normal joints, resting or in motion. When intra-articular effusions develop the intra-articular pressure becomes positive, as high as +70 to +100 mm. of water (and even to +700 mm. occasionally).[21]

Whillis has studied the development of the interphalangeal joints of the feet in a series of human embryos from 30 to 125 mm. in length and has supplemented this with a study of rat embryos.

Inasmuch as *rheumatoid arthritis is a systemic disease, pathologic changes occur in the skeleton and other systems.* It has been shown recently that this *disorder affects primarily* the colloidal, jelly-like material in the interstices

between the cells and fibers of mesenchymal origin, *i. e.*, the *ground substance of the mesenchyme.*

Bennett studied 101 autopsied cases of rheumatic fever and 48 cases of rheumatoid arthritis. In the rheumatic fever subjects, knee joints known to have been afflicted were examined in 11; in 3 instances, pathologic changes were found. A non-specific synovitis was found in 2.

Among the 48 rheumatoid arthritic subjects examined *at postmortem*, the *earliest alterations seen were* hyperemia and diffuse lymphocytic and plasma cell infiltration of the subsynovial tissues, often associated with the formation of lymphoid follicles. The thickened and redundant synovial villi occasionally showed areas of fibrinoid degeneration. Regardless of the duration of the arthritis, there was little or no fibrinous exudate on the synovial lining. *With progression of the disease,* the chronic inflammatory and proliferative changes in the synovial tissues extended from the perichondrial margins on to the articular surfaces. Joint cartilage then became thinned, disrupted and later, partially or completely destroyed. The tendon sheaths showed similar inflammatory changes. There were varying stages of fibrous and bony ankylosis.

Atrophy of bone was present in all cases and localized bony destruction was often noted in the subchondral cancellous portions or shafts; these lesions were usually the result of direct extension of inflamed connective tissue through the eroded articular surfaces or growths along blood-vessels through the bony cortex near the articular margins.

It was interesting that the *terminal interphalangeal joints were frequently involved.*

Subcutaneous nodules were seen in about 20 per cent of cases in both rheumatic fever and rheumatoid arthritis. Eleven showed amyloid infiltration.[6]

Important contributions to the pathology of rheumatoid arthritis have been made by Pommer,[33] Nichols and Richardson,[30] Strangeways,[35] Allison[1] and Ghormley,[18] Hoffa and Wollenberg,[22] Timbrell Fisher,[13,14] Klinge and Grzimek[26] and others.[31]

The *initial change in rheumatoid arthritis* is an inflammation of the synovial membrane.[32] This may become from 0.5 to 2.5 cm. thick; variations of 2 cm. in thickness may occur in the same specimen.[18] Ghormley[19] believes that the main pathologic picture in rheumatoid arthritis is proliferation of fibrous tissue and the formation of focal collection of lymphocytes in the synovial membrane and adjacent bone marrow.[22] These lesions are present in both the early and late stages of the disease and are not related to the duration of the pathologic process. The collections are usually not perivascular (as can be determined when the tissues are stained with Perdreau stain).[20] Others found these lesions to be perivascular in some instances.[31]

Fluid soon accumulates within the joint cavity. As the synovial membrane hypertrophies, it advances and spreads over the cartilaginous ends of the bones, beginning at the margins of the joint, forming what is commonly known as *pannus.* This pannus usually appears as soft, friable,

grayish red tissue which is firmly attached to the underlying articular surface. The thickness, size or number of villi of the synovial membrane are not definite criteria of the duration of the arthritic process. The pannus produces irregular destruction of the articular cartilage, probably from proliferation of fibroblasts which choke out the cartilage cells. *Simultaneously,* there occurs a *proliferation of connective tissue within the marrow spaces,* forming granulation tissue which destroys cartilaginous tissue from the bony side. As the process advances, *joint cartilage may be destroyed completely* and the ends of bones joined by fibrous or bony adhesions (*ankylosis*). The capsule of the joint becomes thickened and the surrounding muscles undergo spasm and atrophy, resulting in dislocation and deformities.

Pathology of Rheumatoid Arthritis

Synovial membrane affected first (as an inflammation of subsynovial tissues), inflammatory congestion of the synovial membrane (swollen, red or purple, pits on pressure).[23]

Round cell infiltration and at times, polymorphonuclear infiltration of the synovialis in some of the more acute cases.[34]

Effusion into the joint (thin and mucinous, or thick and grumous).[10]

Proliferation of synovial membrane resulting in a spreading layer of granulation tissue **(pannus)** over the articular cartilage.

Changes in the **joint cartilage**; dull, red, roughened.

Destruction of cartilage, beginning in the peripheral portions of the joint.

Simultaneous proliferation of connective tissue of marrow spaces,[27] resulting in granulation tissue growing toward the joint cartilage from the marrow spaces, destroying the cartilage from below.

Destruction of bony trabecu'æ by osteoclasts producing decalcification of bone; lessened density of articular ends of bones an early sign.

Thickening of the synovial membrane and joint capsule; capsule may become dense and fibrous.

Villi of granulation tissue may project into the joint cavity from the synovial membrane, pannus or marrow cavity;[30] these may become dense fibrous tissue, or cartilaginous or bony structures and may form loose bodies in the joint.[11]

Intra-articular adhesions; **ankylosis** (fibrous or bony).

Associated changes: Muscular spasm and contractures, dislocations and deformities, muscular wasting, subcutaneous nodules.

The pathologic histology of *subcutaneous nodules* in rheumatoid arthritis is considered by some[10,12,29] to be practically identical with that of the nodules of rheumatic fever; others feel that the nodules are different.[7,9]

Occasional associated findings: cardiac lesions, conjunctivitis, psoriasis, atrophy of the skin, amyloidosis, calcification of blood-vessels.

In rheumatoid arthritis there is an *infiltration of the subsynovial tissues* with lymphocytes and plasma cells with many mononuclear cells and with a few polymorphonuclear cells. This infiltration of the subsynovial tissues occurs without destruction of the intimal lining of the joint and without the appearance of as much fibrin on the surface as in specific infectious

arthritis; known pyogenic bacteria produce an exudate on the surface with organization beneath this.

As the rheumatoid arthritis progresses, there is an *increase in number and size of the synovial villi* and dense *collections of lymphoid cells* appear in the subsynovial tissues; this is not specific for rheumatoid arthritis as these collections appear in other inflammatory joint conditions.

Associated with this, one notes a *proliferation of connective tissue at the margins of the joints*—a vascularized and moderately inflamed connective tissue which extends from the soft tissue margins and erodes into and replaces the articular cartilage in an irregular fashion. Adhesions may develop between the joint surfaces. Under low power of the microscope, one notes a vascularized connective tissue lying over the cartilage, with replacement of some of the articular cartilage, cupping of the cartilage and extension of the proliferating connective tissue into the subchondral spaces.

In the *articular capsule,* one sees *areas of degeneration and necrosis* of the dense fibrous tissue, resembling subcutaneous nodules. One often sees nodules in the dense tissues of the capsule.

In the subchondral bone marrow, there develops a marked *bony atrophy.* The *cortical bone becomes thin* and narrowed and the trabecular bone is narrowed producing a bony atrophy or *osteoporosis,* much of which is probably associated with atrophy of disuse.

There are a number of *associated changes in the marrow spaces.* One often sees in the marrow collections of lymphocytes and plasma cells around a blood-vessel. These may aid in destroying the articular cartilage from beneath; however, much of the cartilaginous destruction is from invasion from the superficial tissues; very rarely, this invasion begins beneath the cartilage, especially at the margins of the joints.[4]

The *cartilage* is *gradually destroyed* by an extension of the inflammatory process so that the *ends of the bones* may become *denuded.* If these two surfaces are in contact, bony ankylosis may result.

Inflammatory changes, similar to those found in the synovial membrane, may be found *in* the *tendon sheaths,* and extending into the *tendons* and the *articular capsule. Subluxations* may then occur.

Although *this inflammatory reaction* has been reported more frequently in the proximal interphalangeal joints, it *may occur in the terminal interphalangeal joints* and may go on to ankylosis in these; this is especially true in fatal cases.

The *skin becomes atrophied* and one may occasionally find small groups of inflammatory cells in the corium. The *muscles* are *markedly atrophic* and show a tremendous *fatty replacement* with a diminution in the number and size of the muscle fibers. No specific changes are found in most cases; the findings could be secondary to muscular atrophy, from disuse.

Thus, *the process in rheumatoid arthritis begins as a low grade inflammation in the subsynovial tissues, spreads to the margins of the perichondrium and then to the articular surfaces.* The pathology of *Still's disease* is indistinguishable from rheumatoid arthritis in adults.

The *nodules in rheumatoid arthritis* may show 1 or more areas of necrosis with complete degeneration of the tissue with a sharp margin of the zone

of proliferating cells, which latter is surrounded by a zone of vascular connective tissue (infiltrated with lymphoid and plasma cells). One occasionally sees a thrombosed blood-vessel at the margin suggesting a violent vascular reaction. In some instances, complete liquefaction occurs in areas; this may be visible to the naked eye. At the margin of the zone of necrosis, there is a zone of palisade cells (with some mitotic figures indicating a rapid proliferative response). Areas in some of the necrotic centers may contain collagen. The nodules are prone to persist for months or two or more years, and activity may still be present after two years, with the injurious agent remaining within the necrotic tissue producing a stimulation of cells outside.[4]

These nodules *may be associated with lesions in the eye,* such as inflammations of the sclera, producing the so-called scleromalacia perforans—with its central necrotic tissue and surrounding areas as in the nodules.

The *nodules of rheumatic fever* are less permanent, often recur in crops and in large numbers, disappear quickly and on microscopic examination appear less sharply circumscribed, with much spacing between the connective tissue fibers and an irregularly latticed boundary, without the necrosis of rheumatoid arthritis; there is more fibrin and more inflammatory cell infiltration in the nodules of rheumatic fever with an edematous interstitial exudative response—a pronounced proliferation of vascular endothelium of the nearby capillaries. One rarely sees actual palisading of the cells at the margins of the nodule in rheumatic fever; we have an exudate superimposed on a somewhat altered collagen. At times, there may occur a marked proliferative response and tissue necrosis in small foci, with occasionally small areas of necrosis. The presence of many mitotic figures indicates an active proliferative response.

The nodules of rheumatic fever may be seen in individuals from six to thirty-two (averaging fourteen years); vary in size from 3 x 3 mm. to 20 x 30 mm. and are distributed over bony prominences, especially over the elbow, wrists, hands, ankles, feet, etc. The nodules in rheumatoid arthritis occur in older people, averaging forty-eight years (from eleven to eighty years), are somewhat larger—from 5 x 3 mm. to 30 x 40 mm. but are distributed in similar areas.

Occasionally a *gouty tophus may resemble a nodule of rheumatoid arthritis,* even including the palisading of cells.

The nodules of rheumatoid arthritis are similar to those of granuloma annulare, tuberculous and luetic lesions.

The pathologic changes are so different from those of specific infectious arthritis that one wonders how any known bacterial agent could produce it. The chronicity of rheumatoid arthritis is very unlike any specific infectious process. The association of inflammatory and proliferative change in synovial tissues (joint or tendon sheath) or changes in the synovial mucin (failure to coagulate when treated with acetic acid) suggests the possibility of some relationship between the mucinous bearing tissues and the disease. The inflamed tissue may not become quiescent for years, suggesting that the type of tissue plays an important part in the process.

Bennett examined microscopically the joint tissues in more than 1000

cases of joint disease, including 155 cases of rheumatoid arthritis. He concluded that it is difficult, if not at times impossible, to distinguish different forms of arthritis on the basis of synovial tissue changes alone. The chronic inflammatory reaction which is usually seen in the synovia of rheumatoid arthritis often has no diagnostic features. Focal collection of lymphoid cells with or without germinal centers are frequently seen in rheumatoid arthritis but are not limited to this disease, nor are fibrinoid deposits on or within the synovial tissues. In most instances, from the findings in the synovial tissue alone, one can only return a diagnosis of chronic synovitis on these tissues removed from patients with rheumatoid arthritis. Small or medium sized multinucleated giant cells have been observed frequently in fixed tissue sections of synovia of patients with rheumatoid arthritis.[5]

Leichtentritt and his associates examined carefully the peripheral nerves of 8 autopsied cases of rheumatoid arthritis and found in 7, small nodules in the perineurium which they considered specific for rheumatoid arthritis; they suggested the term rheumarthritic nodulous perineuritis.[28] These nodules were usually found in the perineurium, and rarely in the epineurium (but not in the endoneurium). They appeared to the naked eye as bluish, round or oval shaped lesions when stained with hematoxylin-eosin; they varied in size from 0.14 to 0.2 mm. in diameter.

Microscopically, they showed a peripheral ring of lymphocytes and plasma cells with a middle zone or epithelioid-like cells and an inner pale and acellular center.

Similar lesions were not found in a study of 100 cases without rheumatoid arthritis.

Freund stressed the likelihood of involvement of the nervous system in rheumatoid arthritis because of the pain along the course of nerves, sweating, glossy skin, muscular atrophy and hyperactive reflexes.[17] Bauer and Morrison found 8 cases of rheumatoid arthritis that showed peripheral nerve changes similar to those described by Leichtentritt and also noted other changes in the spinal cord.[2]

Freund also found *similar nodules in muscles* (such as the gastrocnemius) in patients with rheumatoid arthritis. Some of the nodules found in the muscles were lying close to a small nerve fiber. Here and there muscular fibers appeared hyalinized and degenerated. In every muscle which he has biopsied in patients with rheumatoid arthritis, Freund found these nodules present. Freund considered these paraneuritic nodules found along the course of nerves and in muscles to be specific for rheumatoid arthritis, and to be the cause of many symptoms such as paresthesias, pain, etc., from pressure and irritation of the peripheral nerves.

The pathology of these small **paraneuritic nodules** is identical to that of the subcutaneous nodules of rheumatoid arthritis except that the latter are conglomerations of many of these small nodules.

In 9 cases which were studied, Freund never failed to find these nodules. The earlier the case, the more likely one is to find active nodules. In later cases, there is considerable fibrosis in these. In older cases, one may examine a number of blocks of nerve tissue without finding the nodules

and suddenly come across these. Freund found these nodules in patients with no subcutaneous nodules.

Freund also examined the brain and spinal cord in patients with rheumatoid arthritis and found no important abnormalities; the ganglia likewise were negative.

The joint lesions vary with the severity and stage of the disease. *Early in the disease,* gross examination shows either a normal or slightly increased amount of synovial fluid with little or no evidence of synovitis or a marked periarticular swelling with edema. If the disease remains confined largely to the periarticular structures (as occurs occasionally), the ensuing fibrosis of these tissues may produce considerable impairment of joint function without any marked intra-articular change, although intra-articular changes are present in most cases.

Intra-articular changes are present in most cases. The *synovial lining becomes thickened early* in the disease, and is edematous and grayish or pinkish red. The synovial villi are increased in size and number and there is an increase in the synovial fluid. The *articular cartilage* loses its normal luster and may present superficial ulcerations. With progression of the process, the cartilage *becomes grayish white,* softer and may present larger areas of ulceration. With further proliferation of the inflamed synovial tissue, this extends onto the articular cartilage (as a *pannus*—inflammatory granulation tissue) and may completely cover the opposing articular cartilages, smothering and actually invading them. *Fibrous ankylosis* may occur or the invasion may be so extensive that the articular cartilages are destroyed, leading in some cases to bony ankylosis. The invading pannus may penetrate from the articular surface and produce proliferating connective tissue in the subchondral marrow spaces.

On gross examination of a severely involved joint, one may see all articular structures adhering to each other because of the pannus formation and adhesions and fibrous tissue ankylosis. The surface of the articular cartilage may be altered greatly or there may no longer be any articular cartilage visible. In the later stage of the process, the bone marrow of the adjoining bones becomes more vascular, the trabeculæ fewer in number and the bony cortex of the adjoining bones is thinned.[24]

In the joint with active rheumatoid arthritis, the inflammation of the synovial tissue is seen on microscopic examination. One finds increased vascularity, cellular infiltration (which may be perivascular) and marked proliferation of the synovial cells. One of the features of the disease is the focal collection of lymphocytes in the synovial tissue and at times in the bone marrow. In the early stages of the disease, the microscopic changes are variable.[24]

Other visceral changes that have been encountered occasionally include involvement of the pleura and pericardium (with an adherent pericarditis as a rare finding). Involvement of fibrous, muscle and nerve tissue is frequent. The finding at necropsy of *rheumatic heart disease in some patients with rheumatoid arthritis* raises the question of the relationship between these two diseases and whether cardiac damage may possibly

occur as an occasional feature of pure rheumatoid arthritis; this problem will require further study.[24]

Fox[16] described in detail lesions found in the collections of several museums and from the Philadelphia Zoölogic Society. He analyzed over 1700 skeletons and autopsies of wild animals and noted joint changes corresponding to chronic arthritis in man, even in material in its proper wild habitat when killed. The disease was easily discovered in hyenas and gorillas. The most conspicuous arthritis bearers were macrosomic: gorillas, felines, bears, hyenas, bovines, cervines and wild swine.

In 1749 skeletons, 77 were accepted as arthritic. The arthritis was seen best in the spinal column, but also occurred extensively in the appendicular skeleton. Small bodied animals such as rodents, bats, etc., had no arthritis. There was no apparent relationship to habits, diet or focal infection Examples were found which were similar to osteo- and to rheumatoid arthritis in man.

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CHAPTER VII

SYMPTOMS OF RHEUMATOID (ATROPHIC) ARTHRITIS

There is no single pattern of development of the disease; a variety of clinical forms may be observed. Although the onset is most common between the ages of twenty and forty-five, this is not limited to any age group and the disease may begin in infancy or senility. Women are affected much more frequently than men except in rheumatoid arthritis of the spine (where males outnumber females 10 to 1).[47]

Some cases of rheumatoid arthritis follow an upper respiratory infection while in others, precipitating influences seem to include severe nervous shocks, emotional strain and stress, psychic trauma and exposure to cold and dampness.

Most cases of rheumatoid arthritis begin insidiously although acute fulminating cases are seen occasionally. For weeks or months before actual joint involvement becomes apparent, the patient may complain of tiredness, lack of pep, general malaise, fatigue, numbness and tingling in the extremities, gradual loss of weight (which may be marked), exhaustion, vasomotor disturbances and general debility. Sooner or later, one or more joints—most often the *proximal interphalangeal joints of the fingers—gradually become warm, painful and swollen.*

Only one or a few joints may be involved in the early stages but sooner or later the symmetrical polyarticular distribution so typical of rheumatoid arthritis makes its appearance.

Sooner or later, the *joint shows a spindle shaped appearance* due to the thickening of the periarticular tissues and the atrophy of the surrounding muscles. The thickening and edema of the periarticular structures is evident on palpation. The patient complains of *pain, swelling* and *limitation of motion. During the early stages, this swelling may subside in part and reappear* in a few weeks or months but in most instances, once the actual swelling has appeared, it remains or increases until the disease becomes arrested or quiescent.[36]

The *joint involvement* is *often distributed symmetrically* and there is a tendency for progression of the process from the peripheral joints toward the trunk. It is interesting that while the proximal interphalangeal joints are involved very frequently, one rarely sees clinical evidence of damage in the distal interphalangeal joints in rheumatoid arthritis. Recent pathologic studies have shown, however, that these latter joints are involved in some cases. In order of *frequency of joint involvement in rheumatoid arthritis* are the proximal interphalangeal joints of the fingers, metacarpophalangeal joints, toes, wrists, knees, elbows, ankles, shoulders and hips. *Any joint in the body may become involved* in the process.

In the average case, there is little systemic reaction other than a low grade fever and tachycardia with some loss of weight during the early

stages of the process; however, in a small percentage of cases, one may see repeated mild (or occasionally severe) attacks of multiple joint involvement with fever, tachycardia and leukocytosis and swelling, redness and pain in the affected joints. *In fulminating cases,* we have seen spiking temperatures up to 103° or 104° F. which may be maintained for weeks. In some of these acute episodes, the white blood count may be elevated to 15,000 or 20,000 (although the white blood count is usually within normal limits in the majority of instances of rheumatoid arthritis—or even lowered in long standing chronic cases). *It is extremely difficult in the occasional acute fulminating case to differentiate the disease from rheumatic fever in the early stages.*[36]

Other features include muscular weakness and atrophy. In some instances, one may find subcutaneous nodules (as often as 20 per cent of cases in some areas); lymphadenopathy (of a moderate degree in from 20 to 50 per cent of instances); splenomegaly (usually in less than 10 per cent); atrophy and pigmentation of the skin; cardiac findings (frequently similar to those found in rheumatic fever); pleuritis; and ocular involvement.

Laboratory Findings in Rheumatoid Arthritis

Sedimentation test—moderately to markedly increased in most cases.
Blood count—slight to moderate secondary anemia (reduced hemoglobin rather than red cell count[16]—simple hypochromic anemia); white blood count normal or slightly decreased in chronic cases—slight to moderate leukocytosis in acute cases; polymorphonuclear leukocytes may be increased with shift to the left.
Blood uric acid—normal.
Roentgen-rays—described later.
Agglutination test to hemolytic streptococci—positive in a titer of 1:160 or more after several months to a year in the majority of cases; negative in most children and adolescents. Titers generally fall toward normal as improvement occurs.
Blood proteins—total proteins—normal; plasma fibrinogen and globulin—increased; albumin—decreased.[33]
Gastric acidity—tendency toward increased incidence of achlorhydria.
Blood cholesterol—may be decreased slightly,[26] but usually normal.[9]
Serum calcium and phosphorus—normal.
Basal metabolic rate—usually normal.
Blood cultures—variable results;[29] usually negative.

Important laboratory findings include the increased *sedimentation rate* during the active stage of the disease and the *roentgen findings,* which must always be interpreted in the light of the articular distribution, severity and duration of the disease. Other laboratory tests, except the blood count, urinalysis and Wassermann, are usually of little value to the general practitioner. Although the patient's serum agglutinates strains of hemolytic streptococci (Lancefield Group A) in about half of the cases, this test is seldom positive unless the disease is of at least a year's duration. The negative findings in most instances in respect to antistreptolysin and

antifibrinolysin titers are of academic interest only insofar as the practitioner is concerned. A shift to the left in the filament-non-filament count is often encountered, as is a moderate degree of hypochromic anemia. Histologic examination of the subcutaneous nodules requires an expert trained in this field to differentiate these from the nodules of rheumatic fever. Synovial fluid findings are also of some value, at times, when the diagnosis is in doubt.

Rheumatoid arthritis is a constitutional disease with symptoms affecting chiefly joints and muscles. The joint pathology is primarily a synovitis but sooner or later, there is involvement of the epiphyseal bone and bone marrow, cartilage, fibrous tissue of the joint capsule and the adjacent muscles so that in reality, rheumatoid arthritis comprises synovitis, chondritis, osteitis, epiphyseal non-suppurative osteomyelitis and an associated localized or diffuse periarticular and intramuscular fibrositis.[35]

In the typical rheumatoid arthritis, the *onset* is *usually insidious,* with *involvement first* in most instances *of the small joints,* with slow progression to larger, less peripheral symmetrical joints. These persons are often visceroptotic with marked vasomotor instability. The joints are usually not red but are swollen and sore, with little heat.

In Cecil's series of 245 cases of rheumatoid arthritis, it was interesting that 64 per cent showed a positive agglutination reaction to the Streptococcus hemolyticus and 7.6 per cent had one or more subcutaneous nodules. In 12 patients (5 per cent), rheumatoid arthritis was diagnosed (and treated) in the presence of a normal sedimentation rate. It was found that females were affected twice as commonly as males. In 40 per cent of cases, the disease began between the ages of twenty and forty years, while the process began between forty and sixty years in 43 per cent of cases. Four per cent of the cases had psoriasis (with characteristic changes in the finger nails and terminal phalanges[10]). Rheumatic heart disease was found in only 8 instances.

Symptoms of Rheumatoid (Atrophic) Arthritis

Sex incidence—2 or 3 females to 1 male.[32]
Race—whites more often affected than negroes.
Location—more common in the temperate zones than in the tropics.
Climate—more frequent in cold, damp climate than in dry areas.
Season—symptoms begin most often in the spring and fall, during changeable weather.[30]
Type of patient affected—often thin, asthenic.
Age of onset—usually between twenty and forty; may occur at any age, but children comprise only 1 to 3 per cent of these patients.[30]
Prodromes—anorexia; loss of weight; low grade fever; tachycardia; hypotension; sweating of hands and feet; nervousness; chilliness; fatigue; weakness; numbness, tingling, stinging and burning of fingers and toes,[4,34] which may feel "dead" (sometimes blue and sometimes white).
Onset—usually insidious; acute in less than 10 per cent of cases in adults (often acute in children).

Symptoms of Rheumatoid (Atrophic) Arthritis—(*Continued*)

Joint involvement—tendency to symmetrical involvement, with joint pains swelling, stiffness and progressive muscular atrophy; asymmetrical joint involvement at the onset in 30 per cent.[5]

Progression of joint involvement—often from peripheral joints toward the body; joints of the hands and feet are most often involved at the onset.

Frequency of joint involvement—proximal interphalangeal (fingers), metacarpophalangeal, toes, wrist, knee, elbow, ankle, shoulder, hip, temporomandibular, sternoclavicular, etc.[20]

Characteristic joint swelling—fusiform, spindle shaped swelling especially when the fingers are involved.

Pain—often fibrositic at the onset; aggravated by movement; not rapidly shifting from joint to joint; due to occurrence of muscular spasm and contractures in later stages.

Stiffness—usually present, especially after periods of rest; may become generalized.

Fever—slight, low grade; may be marked (101° to 104° F.) in exacerbations (especially in children).

Tachycardia—usually present, even without fever; elevated out of proportion to the temperature.

Loss of weight—moderate to marked; from 10 to 50 per cent of the body weight may be lost in a few months.[28]

Subcutaneous nodules—found in from 5 to 25 per cent of typical cases,[10,38] especially on dorsal surface of forearm 1 to 2 inches below the elbow joints, or over the spine or occiput in bedridden patients.[18]

Muscular atrophy—marked in some cases, with or without muscular twitchings.

Secondary anemia—frequent.

Mental depression—marked in most cases.

Skin changes—cold and clammy hands; smooth, shiny, atrophic appearance often noted in the extremities; skin over the palms and soles is often a deep pink color. The similarity between the cyanotic palms of some patients with rheumatoid a thritis and of patients with cirrhosis of the liver raises the question of hepatic function in rheumatoid arthritis.[49] In severe cases, bronzing of the skin over the face and extremities and patches of vitiligo are seen frequently. Association with psoriasis in approximately 3 per cent.

Generalized lymphadenopathy and slight **splenomegaly** occur in some cases.

The **familial incidence** of rheumatoid arthritis has been reported as high as 30 per cent in one Italian series; 2 boys and 2 girls were affected in one of the families. It is not uncommon to find rheumatoid arthritis in siblings.[44]

Uncommon associations—opera glass hand (*la main en lorgnette* of Marie and Leri), scleromalacia perforans and diabetes with resistance to insulin.[31]

ONSET AND PRODROMAL SYMPTOMS

The onset is insidious in most cases. Prodromes are usually present in most patients with rheumatoid arthritis although some notice the articular manifestations without any preceding fatigue, emotional upsets, transient articular or muscular pains, numbness and coldness of the extremities

(dead fingers), nervous irritability, fatigue, loss of weight or low grade fever.[35]

Dawson noted that *in 8 per cent of his patients, the disease began as an acute polyarthritis* which simulated rheumatic fever. There are many variations in the onset and course of rheumatoid arthritis, and the diagnostic difficulties are often great. *In some cases, the onset and clinical picture resemble rheumatic fever so closely that the true nature of the disease*

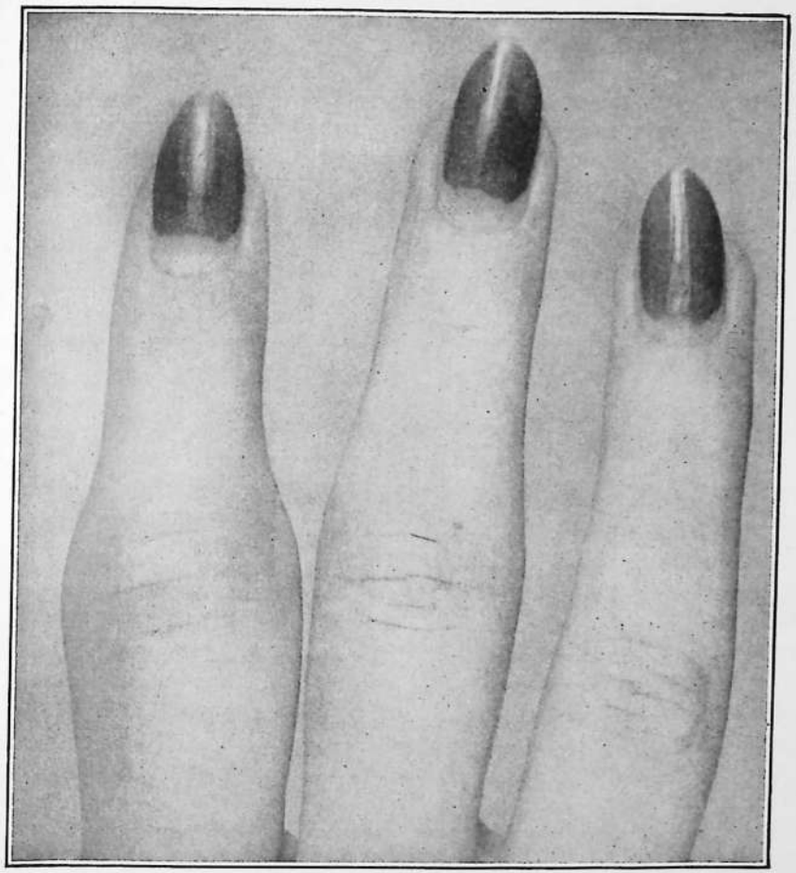

FIG. 16.—Second, third and fourth fingers in a young woman with *rheumatoid arthritis;* this illustrates the marked fusiform (spindle shaped) swelling which often occurs in the proximal interphalangeal joints. It is seen best in the finger on the extreme left.

is revealed only after the passage of considerable time; the persistence of the joint lesions and their resistance to treatment suggest rheumatoid arthritis.

Occasionally a single joint may be attacked from the onset and the spread of the disease to other joints may be insidious. *Ready fatigue of body and mind are considered to be almost constant precursors* of atrophic arthritis. Osgood[41] has warned that "a thin, ptotic, constipated, asthenic twenty to thirty year old member of such a family (with a tendency to

rheumatism) who has been burning either the mental or physical candle at both ends may well be warned that atrophic arthritis is very likely in the offing, whether or not she has had intermittent joint swelling and discomfort." Fatigue and nervous irritability are frequent precursors of rheumatoid arthritis.

The *early course of rheumatoid arthritis is characterized by remissions and relapses.* In the earliest stage of rheumatoid arthritis, the patient may notice only some tenderness and puffiness of several small or large joints

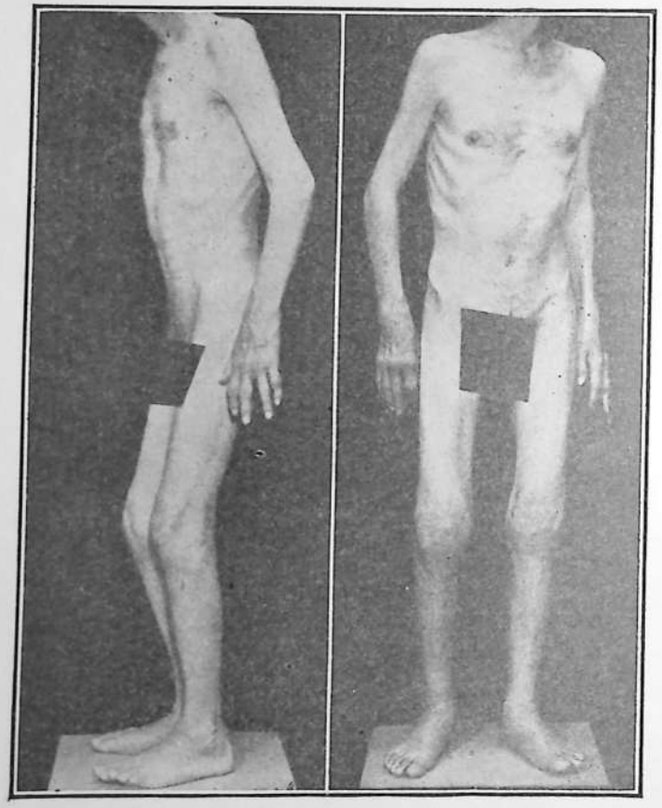

FIG. 17.—Emaciation of patient with rheumatoid arthritis. Note swollen knees and spindle shaped swellings of fingers.

with aching and stiffness and painful motion of the affected joints. At this time, *x*-rays of the affected joints are usually negative except for the presence in some of soft tissue swelling; the sedimentation rate may be normal in a few at this early period, but *a normal sedimentation rate should be viewed with suspicion as most patients with demonstrable rheumatoid arthritis have an accelerated rate.* However, a normal sedimentation rate does not rule out rheumatoid arthritis. At the beginning of the disease, there may be few constitutional symptoms.[35]

Slight stiffness and migratory joint pains may precede any definite inflammatory changes in the joints. The joints first affected are most commonly the proximal interphalangeal and the metacarpophalangeal joints. Motion of the affected joints results in pain.

As the disease progresses, *joint pain* and *swelling increase* and *muscular stiffness* becomes more marked. *Muscular atrophy* appears and there is *limitation of motion* of the affected joints. The sedimentation rate becomes definitely elevated, and the *patient gradually loses weight, becomes anemic* and has very little appetite. With progressions of the arthritis, the definite *fusiform swelling* makes its appearance about the joints affected (particularly the proximal interphalangeal joints); this swelling is due to overgrowth of the synovia, thickening of the joint capsule and an outpouring of fluid into the joint. The hands are cold and clammy and the skin covering these parts becomes atrophied, smooth and shiny.

Muscles in the vicinity of the affected joints become more atrophied and *flexion deformities* may occur from muscular spasm and lack of proper splinting of the affected joints. The *constitutional symptoms* become more marked, with fever, tachycardia, sweating and further loss of weight and appetite. The patient becomes somewhat incapacitated.

In an *advanced stage* of rheumatoid arthritis, all of the constitutional symptoms become more marked and some of the affected joints show definite *flexion contractures* (unless properly splinted earlier); partial or complete *ankylosis* may have occurred in some joints. If the disease continues, it may eventually burn itself out so that the sedimentation rate slowly returns toward normal and the patient's appetite and weight may gradually return to some extent. Signs of inflammation at the joints disappear and the patient is left with her articular deformities and residual thickenings of various joints.[35]

Fever.—*A low grade febrile reaction and tachycardia are usually present.* Some patients with rheumatoid arthritis may have no fever.[19] Although the temperature usually remains below 100° F., a recurring fever as high as 104° F. (without other obvious causes) has been reported, especially in children. *Low grade fever may persist for months* and may recur following an intermission. Fever may not be observed by the patient; in some individuals, the febrile period can be predicted by the gradual appearance of a sense of general malaise appearing from one-half to two hours before the onset of fever, and often persisting for several hours after the cessation of clinical febrile reaction. Fevers of 100° to 101° F., persisting for weeks or months, may be encountered. *Tachycardia* is usually present in rheumatoid arthritis, even in the absence of fever; it may continue for months after evidence of joint activity has disappeared.

PROGRESS OF THE DISEASE

The initial pain of rheumatoid arthritis is often generalized and fibrositic in nature, later becoming localized to the joints. *The severity of the pain is no index as to the ultimate prognosis.* Although joint pain is usually

relieved by rest and aggravated by motion, in some cases the pain is at its worst when the patient is in bed. In the early stages, migratory joint phenomena may render the diagnosis from rheumatic fever difficult. However, the pains do not shift from joint to joint as rapidly as in rheumatic fever. In the later stages, pain is frequently due to muscular spasm or contracture.

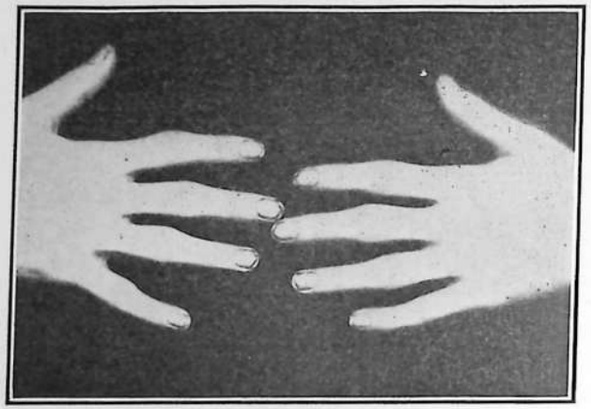

FIG. 18.—Typical swelling and spindling in rheumatoid arthritis. (Young, Jour. Bone and Joint Surg.)

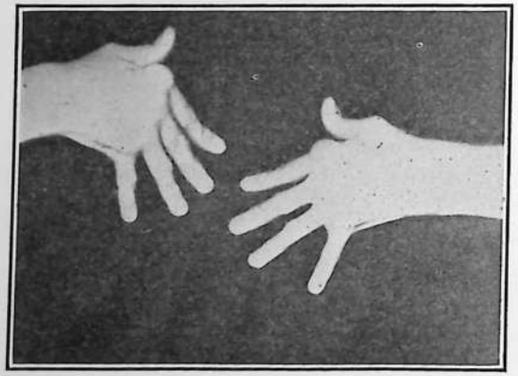

FIG. 19.—Ulnar deviation and deformity in rheumatoid arthritis. (Young, Jour. Bone and Joint Surg.)

Monarticular involvement is unusual. *Any joint of the body may be involved.* The joints first affected in most cases are the hands, especially the proximal interphalangeal and the metacarpophalangeal joints of the ring fingers. Severe involvement of the wrists may also appear at an early stage, followed by arthritis of the knees and ankles; the hips are affected infrequently. At the beginning and again later in the disease, stiffness of the neck and jaw may appear. Marked destructive changes in the jaw are uncommon. Following a period of prodromal joint pains

and stiffness, the joints may become permanently involved and the process passes into a subacute or chronic form.

Pain, stiffness, limitation of motion and *swelling are the main symptoms* noted by the patient. Pain may be increased preceding a storm and may disappear when the rain begins.[15] Swelling results from thickening of the joint capsule and synovium and, together with the muscular atrophy, produces the fusiform appearance. The swelling may appear much greater than is actually the case because of the surrounding muscular atrophy. The stiffness is usually worse after sleep or periods of rest. Effusions may

FIG. 20.—Rheumatoid arthritis of the knees in a middle aged male.

be present in bursæ in the neighborhood of involved joints. The normal contour of the joints is restored only in exceptional instances following a subsidence of activity in this disease.

Rheumatic pain is often increased in connection with atmospheric changes. This is noted especially with a fall of barometric pressure, as occurs before a thunderstorm.[40] Rheumatic patients have thus become known as weather prophets. It has been suggested that such pains may be caused directly by the increased sensitivity of the sensory nerves, arising from variation of the electrical ion-concentration of the air in a negative direction.[25]

It has also been shown that there may be enormous shifts in blood volume, amounting to as high as 40 per cent, in the same city.[7] In Philadelphia, this is maximal in April and minimal in October. It has been suggested that the changes in blood volume may be the cause of symptomatic variations with weather in arthritic patients.

The patient with fibrositis is especially susceptible to changes in temperature; he frequently develops stiffness in the neck immediately following a draft on that area. Such stiffening from cold may be due to changes in superficial tissues, capsules, ligaments and tendons.

The Course of Rheumatoid Arthritis.—The course usually runs over a period of years. *The progress of the disease may be interrupted temporarily or permanently at any stage so that no patient with rheumatoid arthritis should be considered as beyond aid.* On the other hand, *cure must not be promised in rheumatoid arthritis inasmuch as most individuals, even though markedly improved, will still manifest more or less joint involvement from time to time.*

Accurate statistics on the natural course of rheumatoid arthritis are not available. The experience of a large group of arthritic experts has shown that less than 25 per cent "recover," about 25 per cent become progressively worse and in the remainder, the disease becomes quiescent if the individual improves. One must remember these rough statistics when attempting to evaluate any new form of therapy in this disease.[36] Short emphasized that *53 per cent of 300 arthritic patients treated in the Massachusetts General Hospital during the past ten years showed more or less satisfactory improvement with only general therapy.*

One of the most pronounced general features of rheumatoid arthritis is the *muscular atrophy*, although the general nutrition of these individuals suffers severely. There is usually involvement to a greater or less degree of the connective tissue structures throughout the body and profound cachexia occasionally develops despite all efforts to avoid this.[36] All of the muscles of the extremities may become atrophied; this muscular atrophy is especially prominent in the small muscles of the hands.

One of the early manifestations of some cases of rheumatoid arthritis is marked wasting between the thumb and forefinger, and between the fourth and fifth metacarpals. The muscular atrophy found in arthritis can be produced reflexly in animals following injection of an irritant into the joints. Wasting of muscles may also proceed from disuse. In the early cases, an increase in tendon reflexes is noted.

Many cases of rheumatoid arthritis progress to a chronic condition; contractures tend to develop as the periarticular swelling gradually subsides. These *contractures*, unless properly attended to, result in the distressing deformities that are most apparent in the hands, knees, wrists and elbows. In the hands, the usual deformities that develop, if the process is unchecked, include flexion of the proximal interphalangeal joints with atrophy of the interossei and thickening of the metacarpophalangeal and proximal interphalangeal joints and flexion of the terminal interphalangeal joints. Finally, ulnar deviation at the metacarpophalangeal joints renders the hands almost useless. Fibrous and/or bony

ankylosis may occur together with subluxation or actual dislocation of these joints. Another very distressing deformity which must be avoided is fixation of the knees in partial flexion.[36]

Various degrees of remissions and relapses may occur with variable durations; although remissions may persist for years (occasionally), the usual disease picture is progression with time until partial or complete incapacitation occurs. With repeated attacks, the joints are injured further and more stiffness may ensue. Muscular spasm and atrophy appear unless measures are undertaken to promote physiologic rest of the part and to utilize the effects of heat, massage and exercise when indicated. *In about 10 per cent of patients, the course of the disease is progressively downhill despite the therapy or the lack of the same.* In the late stages of the disease, the characteristics presented by such patients are emaciation, muscular atrophy, joint deformity, contractures and ankylosis.

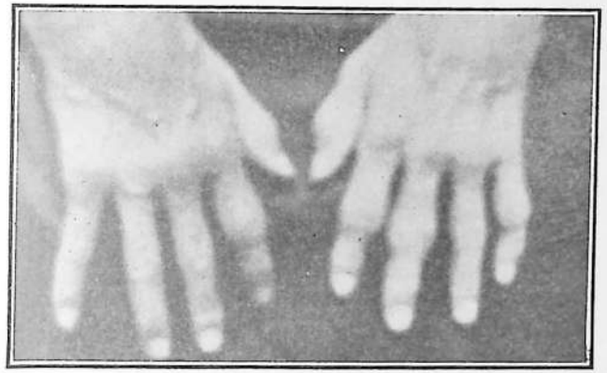

FIG. 21.—Rheumatoid arthritis: involvement of metacarpophalangeal and proximal interphalangeal joints.

In most instances, the disease becomes arrested or quiescent during its course so that the patient can be taught to live and carry on an occupation with his arthritis without great difficulty. In a small percentage of patients, despite all known treatment, the disease progresses to helpless crippling and invalidism with profound cachexia. The physician must always remember that some patients with rheumatoid arthritis recover more or less completely from the effects of their disease, and *the prognosis is not nearly as unfavorable as many general practitioners have believed in the past.*

It is difficult and often almost impossible to predict the course of events in a particular patient. Those patients with a more acute onset seem to have a much more favorable outlook.

Unfavorable prognostic signs include a slow, insidious onset with involvement of fresh joints from time to time, a persistently high sedimentation rate, marked muscular atrophy, subcutaneous nodules, marked persistent loss of weight, persistent tachycardia and marked depression of spirits.

A severe relapse may occur at any time in even the most favorable case and on the other hand, even the most severe form of the disease may suddenly become quiescent.[36]

Disturbances of growth may follow chronic arthritis in children.[61] They result chiefly from early ossification of the epiphyses. Persistence of infantile proportions in the hands or feet is most common; others are shortness of a portion of, or a whole extremity, symmetrical shortening of the limbs, and rarely, dwarfism.

Atypical Rheumatoid Arthritis.—This is much less common than typical rheumatoid arthritis. About 15 per cent of patients with rheumatoid arthritis do *not* present the typical onset with an insidious beginning, symmetrical joint involvement and the usual constitutional and vasomotor symptoms. *In about 5 per cent* of patients or more, *rheumatoid arthritis may remain atypical for months.* In some of these latter, the patient may apparently recover completely from his first attack and remain well for several months or years; he may suffer a number of similar attacks before his disease becomes chronic. These *atypical cases occasionally present monarticular joint involvement* although this is usually polyarticular, and at times, migratory.[6]

In this disorder, prodromes and the asthenic habitus of typical rheumatoid arthritis are usually lacking. Atypical or focal infection arthritis (sometimes called infective or secondary rheumatoid arthritis) usually appears acutely or subacutely and may follow a dental abscess, pharyngitis, tonsillitis or peritonsillar abscess or influenza.[30] Small or large joints may be involved symmetrically with rapid progress, but frequent remissions of the disease. The joints are usually warm, red and swollen and the patient is febrile and presents other signs of toxemia. It is often polyarticular and migratory, although monarticular arthritis is not uncommon.

If there is a definite history of close relationship between the presence of a focus of infection with an arthritis following this within a short period, this particular class of patient may be definitely benefited by proper removal of the focal infection. In general, the *more acute the onset of an arthritic picture, the better the prognosis.*[35] Many of these cases respond well to rest, splinting and analgesia.

MISCELLANEOUS FINDINGS

Loss of Weight.—From 10 to 50 per cent of the body weight may be lost in a few months. Some individuals may lose 100 pounds during the course of six months. In more than 50 per cent of these patients, a moderate loss of weight occurs (from 10 to 20 per cent of body weight) unless the caloric intake can be maintained at a high level.

Subcutaneous Nodules.—Subcutaneous nodules may appear in from 5 to 25 per cent of patients with rheumatoid arthritis.[12,13,21,24,36] This percentage undoubtedly varies tremendously in various areas. In Philadelphia, we do not see these nodules in more than 5 per cent of cases, even though they are searched for most diligently. Such nodules are usually

larger and persist longer than the corresponding lesions of rheumatic fever. Dawson has found them to vary in size from tiny seedlike particles to small walnuts. These nodules may persist for months or years. Millet seed nodules have been described by Vincent Coates of Bath. More than 100 nodules of different sizes have been observed in a single patient.[39] They may be attached to tendon sheaths or bursal walls but the skin

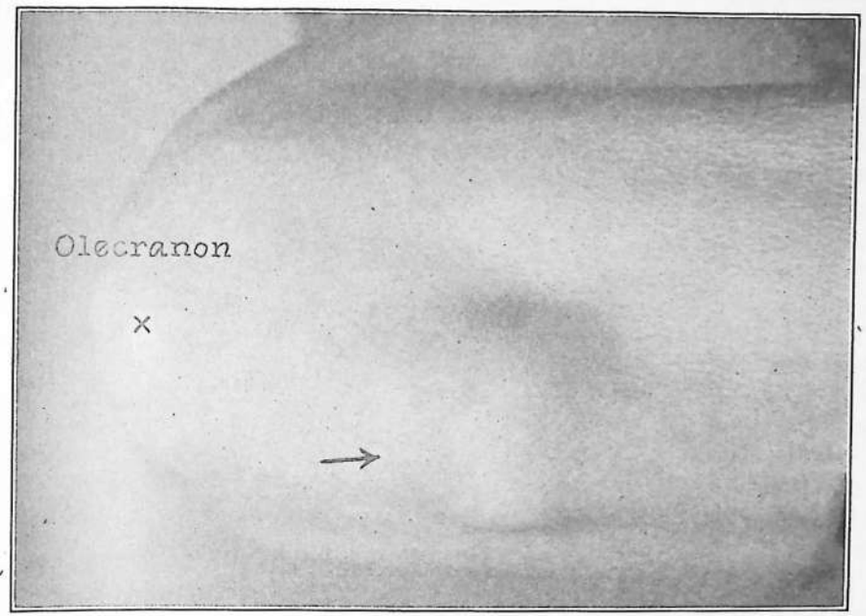

FIG. 22.—Subcutaneous nodule on extensor surface of forearm below the elbow joint

overlying them is usually freely movable. Traumatic influences probably determine the sites of their occurrence; continued trauma may result in increase in size of the nodule to such an extent that it may resemble a bursa.

The commonest site for subcutaneous nodules is the extensor surface of the forearm about 1 or 2 inches below the elbow joint. They may also occur in the olecranon bursa, over the patella, scapula, spinous processes, sacrum, scalp, along the tendons of the fingers and toes, and in the eye (scleromalacia perforans.[4]) These nodules are painless unless exposed to marked pressure because of their position. *The presence of such nodules is often an indication of a poor prognosis.*

The histologic similarity of the subcutaneous nodules of rheumatic fever and rheumatoid arthritis has been discussed by various writers,[11,14,23,27] while others (Bauer *et al.*) have pointed out dissimilarities between the nodules.

In a recent excellent study of large numbers of subcutaneous nodules removed from patients with rheumatoid arthritis and from those with rheumatic fever over a period of ten years, it was found that the nodules in both diseases were similar in that each consisted of a central zone of

necrosis, an intermediate zone of proliferating cellular tissue and a peripheral zone of inflammatory tissue.[8] However, in the rheumatic fever nodules the central zones showed predominantly a "fibrinoid" change as contrasted with a predominance of degeneration and necrosis in those of patients with rheumatoid arthritis.

The "fibrinoid" change in the rheumatic fever nodules was characterized by swollen, hyalinized, widely separated collagen bundles with persistence of some of the fibrillar components of connective tissue, which indicated that there had been an exudation of fibrin and plasma into and between the collagen fibrils.[8] The changes in these nodules have been interpreted as due chiefly to injury of small blood-vessels followed by exudation of plasma and blood cells into the connective tissue. Small focal lesions similar to the myocardial Aschoff nodules were also seen.

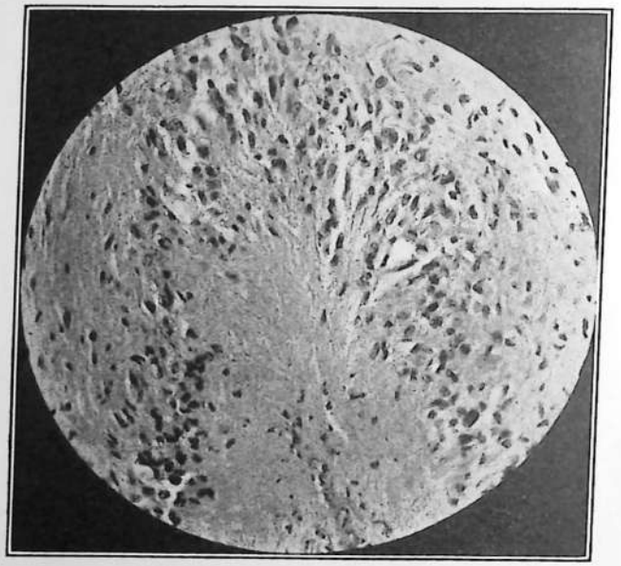

FIG. 23.—Subcutaneous nodule of rheumatoid arthritis; microscopic examination. (Dawson and Boots, New England Jour. Med.)

The nodules of rheumatoid arthritis revealed a predominance of marked proliferation and degeneration of connective tissue with a complete absence of Aschoff-like nodules; exudation was not prominent in these lesions. "Fibrinoid" change was not often seen. The central areas of necrosis were usually well marked with almost complete disintegration of all previously existing connective tissue.

It was concluded that inasmuch as the rheumatic fever nodules are chiefly an exudative process, they may appear and resolve very quickly, leaving little residuum. The longer duration and larger average size of the nodules of rheumatoid arthritis is probably due to the continued proliferation and necrosis.[17] Dawson has studied nodules which persisted for many months in rheumatic fever and found them surprisingly similar to

nodules of corresponding duration in rheumatoid arthritis. He also noted that the early and acute lesions as may occasionally occur in fulminating rheumatoid arthritis were similar to the nodules of rheumatic fever.[22] Copeman feels that too much attention has been given to the subcutaneous nodules of rheumatoid arthritis. In England he has seldom found these directly around joints as may occur in rheumatic fever.

Changes in the Skin.—*Cold and clammy hands* are common in patients with rheumatoid arthritis but one occasionally encounters the "*hot house palms*"—with warm moist hands. In some patients, there is a characteristic redness of the thenar and hypothenar eminences.[36] *Bronzing of the skin* over the face and extremities has been noted in some of the severe cases Vitiliginous patches have also been observed.

Psoriasis and rheumatoid arthritis is discussed in a separate section.

In chronic rheumatoid arthritis, the *nails may become dry, brittle and may develop longitudinal striations* of the nail plate with or without the formation of fissures (*onychorrhexis*). Subungual hyperkeratosis may appear and in some instances, there is a gradual separation of the nail plate from its bed, beginning at the free edge and progressing slowly toward the root (*onycholysis*); the entire nail may be lost. In some long-standing cases, the nails of the toes may become elongated, raised, thickened, green or black in color and may become twisted like a hook or spiral with an irregular and opaque surface; it may resemble a claw or a horn, and the texture of the nail resembles very much that of the horn of an animal (*onychogryphosis*). Pitting of the nails has been observed in some cases of rheumatic fever.[42]

RHEUMATOID ARTHRITIS IN CHILDREN (STILL'S DISEASE)

Rheumatoid arthritis in children, called *Still's disease,* is uncommon. This entity was described by Still in 1897 as a form of chronic joint disease characterized by fever, swelling of the joints and enlargement of the spleen and lymph nodes. Of his 22 patients, girls were more frequently affected than boys (1.5 to 1); the onset of the disease was usually insidious. The disease usually appeared before the second dentition. The earliest occurrence was at fifteen months. Fever did not seem to be related to the joint symptoms and was continuous at a constant low level or was intermittent. Profuse sweating and anemia were common. The discrete, hard, nontender lymph nodes, seldom larger than a hazel-nut, were related to the involved joints. The most frequently *involved joints* were the knee, wrist and cervical spine. *Muscular wasting* was present in most cases.[1]

Autopsies were performed on three of the patients; the synovial membranes were thickened and obliterative fibrous pericarditis and fibrous pleural adhesions were present in all 3 cases.

Atkinson *reviewed the literature* in 1939 and collected *118 cases* of Still's disease.[2] Schlesinger also reviewed these cases.[46] It was found that there was some increased prevalence in boys in this group.

Only 14 autopsies are recorded in Still's disease. The essential *findings*

at autopsy included: enlargement of the cervical, axillary and inguinal nodes in almost all cases, with other nodes enlarged occasionally; enlargement of the spleen in 12 cases with amyloid degeneration of the spleen in 5; fibrinous pleurisy or pericarditis in 3 cases; fibrous pleural or pericardial adhesions in 9 and peritoneal adhesions in 3.[43,48] The *heart was often enlarged* and small, dark, hard verrucæ were found along the line of closure of the tricuspid valve in 2 cases with sclerosis of the pulmonary valve in 1. Acute myocarditis was found in 1 case and chronic myocarditis in another. Renal amyloidosis was found in 4 with hyaline deposits in the glomerular tufts in another. Albuminuria occurred in these and in 2 others. Four of the patients died in uremia.

In 6 cases, partial or complete ankylosis of several joints was found; the degree of cartilaginous destruction varied with the duration of the disease.

Angevine studied 6 children with rheumatoid arthritis; all of these have improved under conservative therapy. These patients showed a moderate elevation of temperature with pain, swelling and stiffness of the joints early in the disease. In his cases, the spleen and lymph nodes were not enlarged. Fusiform swellings of the fingers developed as in adult cases. Subcutaneous nodules were very rare. In long-standing cases, extreme muscular wasting and fibrous ankylosis may occur together with extensive demineralization of bone. In children, *deformities appeared somewhat sooner* and *seemed to be more crippling* than in adults.[1]

Histologic sections of synovial membrane from relatively early cases showed proliferation of the synovial cells with an extensive chronic inflammatory process beneath them; this inflammation was characterized by lymphocytes, plasma cells and polymorphonuclear leukocytes, often in focal collections.

Treatment of rheumatoid arthritis in children follows the same general pattern as in adults. Good results have been reported in a few cases following injection of gold salts. Angevine thought that more attention should be paid to the psychic factors associated with the home, school and social environment; fortunately, these children have less apprehension than adults because they do not realize the serious possibilities associated with the outcome. It has been suggested that it is better to err on the side of early activity rather than on prolonged rest in bed because of the importance of preventing muscular wasting. *Great stress must be placed on the prevention of deformity in these individuals.*

FRACTURES IN CHRONIC ARTHRITIS

Baer reviewed 1625 case records at the Robert Breck Brigham Hospital in Boston to determine the frequency of fractures in arthritis, and their effect upon systemic and local disease process.

He concluded that *fractures are infrequent complications of chronic arthritis,* with healing taking place normally; one can expect a firm union in these cases. Fractures had no bearing on the activity of the disease *per se;* they did not produce an exacerbation of a quiescent arthritis.

With the marked bony atrophy which occurs in rheumatoid arthritis, simple trauma is sufficient to produce fracture. If proper precautions are used, ankylosis or limitation of motion of joints need not occur.

In degenerative joint disease, the fractures were usually caused by major trauma which would have been sufficient to produce fractures in normal bones.

Approximately 1 per cent of the 1625 *arthritic patients exhibited fractures;* this is in contrast to the frequency of dislocations in these individuals.

Baer did not classify *"infractions"* as fractures in this survey. By infractions is meant the disintegration which is frequently seen at the bone ends, due to atrophy and cystic degeneration; in the *x*-ray, one often sees a fracture line at the ends of the bones where the disintegration has occurred.

Very slight trauma may lead to fracture in the patient with advanced rheumatoid arthritis. In one instance, weight bearing alone was the only force operative, while in another, muscular effort resulted in fracture. *Manipulation of a joint* under anesthesia may also result in fracture; otherwise, major trauma was not a factor. Although one would theoretically expect that these individuals would frequently exhibit fractures because of the marked atrophy of bone with demineralization and cystic degeneration, these patients lead a guarded existence for the main part so that they do not often encounter the forces required to produce a fracture.[3]

The *site of the fracture* in the patients with rheumatoid arthritis was in the femur in 9 instances; in the humerus in 3; tibia, 2; and once each in the vertebra, fibula, radius and ulnar styloid. A simple fracture of the shaft was most common, occurring 9 times; comminuted fractures of the shaft were seen twice, and impacted fractures at the ends of the bones and of the vertebræ 3 times. Greenstick fractures occurred once.

In 5 instances, fracture resulted from surgical manipulation of a joint under anesthesia. In those with rheumatoid arthritis, union occurred in all cases except one, where union was delayed. *Immobilization of the limb in plaster did not lead to ankylosis* or permanent decrease in the range of joint motion in fractures of a limb in which one or more joints had been involved in the arthritis.[3]

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CHAPTER VIII

VISCERAL LESIONS ASSOCIATED WITH RHEUMATOID ARTHRITIS

Visceral disturbances include:

Enlargement of the spleen.
Lymphadenopathy.
Cardiac involvement.
Amyloidosis.
Lesions of the eye.
Gastro-intestinal disturbances.
Neurologic changes.

In a period of twenty-five years, necropsies were performed in only 30 patients with rheumatoid arthritis at the Mayo Clinic. The *causes of death* in these patients were divided into three groups:

1. Those not related to the arthritis—such as coronary occlusion, pulmonary suppuration, accidental death and death from carcinoma.

2. Death from therapy undertaken for the arthritis, including death from reaction to typhoid vaccine, cinchophen hepatitis, fat embolism following manipulation of joints and pulmonary embolism following application of casts.

3. Death from visceral diseases which were probably a part of the rheumatoid disease, as rheumatic heart disease, amyloid degeneration or prolonged exhausting diarrhea.[66]

Baggenstoss and Rosenberg studied at necropsy the *anatomic changes in the viscera of 30 patients with rheumatoid arthritis.*[4] The mean age at death was 37.6 years, the youngest being nine and the oldest eighty-one years; 11 patients were under the age of forty. Sixteen showed evidence of *rheumatic heart disease;* other cardiac lesions were found in 8. They observed no characteristic hepatic lesion which could be ascribed to rheumatoid arthritis. *Alterations of the liver* included atrophy, hypertrophy, chronic passive congestion, fatty change, central necrosis, amyloid deposits, subacute yellow atrophy, healed miliary tuberculosis and the serous hepatitis of Rossle and Eppinger. The *lymph nodes and spleen* which were enlarged occasionally during life showed non-specific degenerative and inflammatory effects such as degeneration of lymph follicles, amyloid deposits, hypertrophy of the spleen, proliferation of reticulo-endotbelial tissues, suppurative lymphadenitis and chronic passive congestion.

Nineteen of their patients showed a low grade non-specific *glomerulonephritis.* Among other renal lesions found were amyloid degeneration, nephrolithiasis with acute pyelitis, subacute or chronic interstitial nephritis and dissecting aneurysm of the right renal artery. Other organs showed various lesions of minor importance.

In another series of 61 autopsies in rheumatoid arthritis the causes of

death included bronchopneumonia in 24, septicemia and pyemia in 9, lobar pneumonia in 9, tuberculosis in 7, carcinoma in 3, amyloid disease in 3, cardiac decompensation in 3 and uremia in 3.[23]

Splenic Enlargement.—This is usually slight so that the tip is felt just below the costal margin in about 7 to 10 per cent of cases. Occasionally, as in the group of cases described by Felty, the spleen may extend halfway to the umbilicus. Cases of arthritis with splenomegaly and adenopathy were described by Chauffard in 1896 and by Still in 1897; in 3 of the cases described by Still there was no splenomegaly.[71] This is more common in children than in adults.[73]

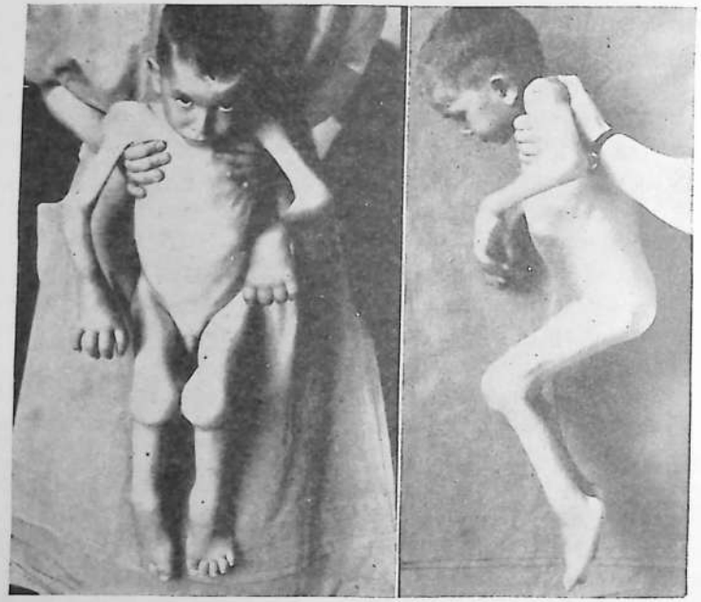

FIG. 24.—Rheumatoid arthritis (Still's disease) in a child, aged six years. (Lewin, Orthopedic Surgery for Nurses, courtesy of W. B. Saunders Company.)

Felty's Syndrome.—Felty, in 1924, reported a series of 5 patients with chronic arthritis, marked leukopenia and splenomegaly. Others have described similar cases.[51] An analysis of the cases reported in the literature discloses the following:[16,77]

Occurrence equally in men and women between the ages of forty-five and sixty-five.

Features: chronic arthritis, leukopenia, splenomegaly, and usually a marked loss of weight, averaging 40 pounds.

Intermittent febrile periods (usually under 101° F.)

Chronic arthritis with acute exacerbations, followed by stiffness and limitation of motion.

Definite splenic enlargement (which may extend to the umbilicus),

Liver palpable in 20 per cent of cases.

Slight generalized lymphadenopathy in 50 per cent of cases.

Average white blood count (upon admission) 2500 (with a range of from 800 to 4200); average differential count: 50 per cent polymorphonuclear cells, 40 per cent lymphocytes, and from 1 to 12 per cent of eosinophils.

Red blood cell count variable from 3,000,000 to 4,800,000 with a corresponding fall in the hemoglobin content.

Platelets, bleeding and clotting time normal.

Similar to Still's disease, except that the latter usually occurs in children, and has a leukocytosis and a crippling form of arthritis.

Pigmentation of the skin, confined chiefly to exposed surfaces, in 60 per cent.

Calf muscle biopsies from patients with Felty's syndrome showed the same pathology as those in patients with chronic rheumatoid arthritis.[18]

Occasionally, improvement (both general and arthritic) following splenectomy.

Several authors have reported beneficial results following splenectomy in these patients;[17,36] in others, splenectomy has not directly altered the course of the arthritis.[70] There seems to be no good evidence in favor of this procedure in these patients.

Bauer and his associates reviewed the literature on this subject and concluded that *this syndrome is but rarely encountered as an expression of rheumatoid arthritis.*[73] They were unable to find a single pathologically verified case in the literature in which an extraneous cause of the splenomegaly and leukopenia could be definitely eliminated. They pointed out the inadvisability of using the term "Felty's syndrome" and again suggested that the syndrome be more accurately named "rheumatoid arthritis," with associated splenomegaly and leukopenia. It was readily evident that 11 out of 14 cases reported in the literature as examples of Felty's syndrome strongly suggested the possibility that the associated accompanying disease was responsible for the splenomegaly and leukopenia. Many writers fail to exclude some concurrent disease which may be responsible for the leukopenia and the enlarged spleen and fail to realize that leukopenia and splenomegaly may be manifestations of rheumatoid arthritis.

Chauffard and Ramond[11] first mentioned in 1896 the occurrence of adenopathy and splenomegaly in adults with rheumatoid arthritis; Still noted this in children the following year. Since that time, the reported incidence of splenomegaly in rheumatoid arthritis has varied from 6 to 23 per cent.[12,26,56,76]

Lymphadenopathy has been reported in from 19 to 96 per cent of patients with rheumatoid arthritis, varying, evidently with differences in criteria for denoting enlargement and the assiduity in the search.[12,26,76]

Slight or moderate leukopenia is not unusual in rheumatoid arthritis, but severe neutropenia is uncommon.[21] White blood counts below 2000 occur in less than 0.5 per cent of patients with rheumatoid arthritis.[14]

In 293 unselected cases of rheumatoid arthritis, Bauer and his associates

found splenomegaly in 7 per cent, lymphadenopathy in 31 per cent and leukopenia consistently under 5000 in only 1 per cent.

It has been suggested that when one observes a patient with the triad of rheumatoid arthritis, leukopenia and splenomegaly, he should consider the possibilities of cirrhosis of the liver, healed acute yellow atrophy (or other chronic liver disease), pernicious anemia, amyloid disease, leukemia, agranulocytosis, hemolytic anemia, malignant lymphoma and acute and chronic infections—as well as periarteritis nodosa, dermatomyositis and disseminated lupus erythematosus.[73]

Lymphadenopathy.—*A moderate degree of lymphadenopathy is common in rheumatoid arthritis*, especially in children (Still's disease). Some observers have noted this in from 19 to 96 per cent of cases.[12,26,73,76] Large axillary and inguinal nodes may be found. Lymphoid hyperplasia is less common in adults with rheumatoid arthritis.[15]

Cardiac Involvement.—In a postmortem study of 23 patients with rheumatoid arthritis, Bayles[6] observed that in 6 there were changes in the heart valve leaflets and myocardium similar to those of rheumatic fever. Baggenstoss and Rosenberg[2] found at autopsy cardiac lesions indistinguishable from those of rheumatic fever in 14 of 25 patients with chronic infectious arthritis.

Monroe and Walcott found valvular heart disease in only 3 per cent of 142 patients with rheumatoid arthritis.[59] In another series of 50 cases of "acute rheumatoid arthritis," no clinical or electrocardiographic evidence of heart disease was found in a single instance.[54]

In a series of 69 children with rheumatoid arthritis (Still's disease) studied for a period of years, only 1 showed clinical evidence of carditis during life.[15] Field reported a child with rheumatic fever and rheumatoid arthritis.[22]

Hench suggests that clinicians may be missing associated lesions of rheumatic carditis in patients with rheumatoid arthritis.[40] In some series, 11 per cent of patients with rheumatoid arthritis will give a history of coincidental rheumatic fever, if carefully questioned.[55]

In 100 consecutive cases of typical rheumatoid arthritis, Dawson found unequivocal signs of mitral stenosis in 7 per cent; in all of these the arthritis had its onset before the age of thirty. He later noted that 35 per cent of 40 patients with rheumatoid arthritis were found to have evidence of rheumatic heart disease by clinical examination. Dawson feels that rheumatic fever and rheumatoid arthritis bear some relationship to each other, but that it is futile to speculate on the precise nature of this relationship inasmuch as the etiology of neither disease has as yet been definitely established.[20]

Other workers had noted the association of cardiac lesions with rheumatoid arthritis some time ago;[9,28] this relationship was mentioned by Todd in 1853 and later by Still who noted cardiac lesions (1896) in chronic arthritis of children—now known as Still's disease. In 12 of his cases that came to autopsy, 5 had an adherent pericardium and 1 had a thickened mitral valve.[57,71]

Garrod stated that the heart is not usually damaged in rheumatoid arthritis and that if valvular insufficiency was present, one should assume a previous attack of rheumatic fever as its cause. Others felt that in the usual case of rheumatoid arthritis with its insidious onset, cardiac involvement did not occur, but that when the disease began with an abrupt onset with an acute febrile attack, the heart was then often implicated.

Baggenstoss and Rosenberg reviewed the literature on the association of cardiac lesions with rheumatoid arthritis and noted that the clinical incidence of cardiac lesions varied from 4 to 40 per cent. There have been few pathologic studies of the heart in cases in which rheumatoid arthritis occurred. Their investigation was based on the necropsy study in 25 cases of rheumatoid arthritis (in which there was a history of rheumatic fever in only 2 cases). In 56 per cent, cardiac lesions identical with those of rheumatic fever were seen. Non-rheumatic cardiac lesions were present in 24 per cent. In 10 of the 14 cases in which rheumatic cardiac lesions were found, there was histologic evidence of an active and progressive inflammatory process at the time of death; in half of these cases, the heart disease was thought to be an important factor in causing death. Signs or symptoms of heart disease were present during life in only 7 of the 14 instances associated with rheumatic cardiac lesions. They concluded that this high incidence of rheumatic cardiac lesions was suggestive of a relationship between rheumatic fever and rheumatoid arthritis.

The rheumatic cardiac lesions found at autopsy involved the mitral valve alone in 6 cases, the mitral and aortic valve in 3, the mitral, aortic, tricuspid and pulmonary valves in 1; the pericardium in 5; the myocardium in 12; the left auricle in 3; the root of the aorta in 3 and the coronary arteries in 3.

Fingerman and Andrus examined the clinical and available pathologic material from 61 patients who died with chronic rheumatoid arthritis. In *31 per cent*, they found *lesions which were indistinguishable from those found in the rheumatic heart.* Six of these 19 cases with rheumatic cardiac lesions had chronic passive congestion of the liver. Only 3 of the patients showed the characteristic syndrome of Felty (chronic arthritis, enlarged spleen and leukopenia). *In 21 per cent, amyloidosis* was found, involving one or several organs. In 8 of the cases, glomerulitis was found.[23]

Nineteen of the 61 cases had fibrous adhesive pericarditis, aortic, mitral or tricuspid valvular involvement and combinations thereof of a rheumatic nature. In most instances, the lesions were old healed valve defects with varying degrees of deformity with thickening and retraction of the leaflets, thickening and shortening of the chordæ tendinæ and fusion of the commissures. The mitral valve was affected in 16 of 19 persons with rheumatic cardiac lesions; in one instance, this was in the acute stage. The aortic valve was involved in 11. Fibrous adhesive pericarditis was present in 7. In one instance, Aschoff nodules were seen in the myocardium.

The anatomic observations of Baggenstoss and Rosenberg support the clinical findings that the cardiac lesions in patients with rheumatoid arthritis are not as severe or as widespread as those in the hearts of young

persons with the rheumatic fever. Hench suggested that the cardiac lesions associated with rheumatoid arthritis may go unrecognized by the physician and the patient because individuals with rheumatoid arthritis usually lead a relatively restricted life with very little exercise; thus the cardiac lesions may have a greater opportunity to heal and less tendency to progress than among patients with rheumatic fever who later resumed moderately full activity.

Another explanation for the lack of severity of the cardiac lesions in the rheumatoid arthritic is that the age of onset of rheumatoid arthritis is usually later than that of rheumatic fever. In 90 per cent of Dawson's 257 cases of rheumatic fever the onset occurred before the age of fifteen, while in 90 per cent of 202 patients with chronic rheumatoid arthritis, the onset was after fifteen years of age. In Baggenstoss and Rosenberg's series, the average age at onset of the rheumatoid arthritis was 33.9 years. Most individuals believe that the heart is more vulnerable to rheumatic infection in younger than in older persons.

Inasmuch as most arthritic patients have definite lesions in the skeletal muscles leading to muscular soreness, tenderness and atrophy, Hench raised the question as to whether similar changes might not take place in the cardiac muscle.

However, Bennett concluded that there was such marked difference in the anatomic changes in rheumatic fever and rheumatoid arthritis that one must infer that the pathogenesis is different, and that these two diseases are separate and distinct entities.

Pericarditis with effusion has been reported occasionally in rheumatoid arthritis in children as well as adhesive pericarditis in adults.[44,57,71]

In many hundreds of tracings taken on a series of patients with rheumatoid arthritis, none showed any electrocardiographic change commonly considered to be indicative of myocardial damage:[53] *i. e., RST* abnormality, *T* wave inversion, auricular fibrillation, auricular flutter, auriculoventricular block of more than 0.21 second or block with dropped beats. It has been suggested that *in a patient where it is difficult to differentiate between rheumatic fever and rheumatoid arthritis, the absence of electrocardiographic evidences of myocardial involvement would argue somewhat in favor of the diagnosis of rheumatoid arthritis.*

Amyloidosis.—Amyloidosis is common as a complication of tuberculosis and other diseases in which chronic suppuration occurs: pulmonary abscess, osteomyelitis and pyelonephritis. Amyloid deposits have been seen occasionally in Hodgkin's disease, leukemia, myeloma and cirrhosis of the liver as well as in infectious, non-suppurative diseases such as malaria, syphilis, lymphogranuloma and rheumatoid arthritis.[69]

There are only a few case reports of the finding of amyloid disease in rheumatoid arthritis.[4,37,48,60,65,69]

Several workers have described the occurrence of amyloidosis in Still's disease (rheumatoid arthritis of children).[45,64]

In one individual with chronic arthritis for fourteen years, autopsy revealed extensive amyloid involvement of the bones, joints and other structures.[48]

In Solomon's patient with rheumatoid arthritis for nine years, amyloidosis of the spleen, liver, kidneys and adrenals was found.

The high incidence of *amyloid disease* in rheumatoid arthritis is interesting. Thirteen of 61 autopsied cases showed this disorder. The spleen was most commonly involved (11 cases), followed in order by the kidneys (9 cases), liver (9 cases), adrenals (8 cases) and pancreas and thyroid (1 case each). In 3 of the cases, there was also an active pulmonary tuberculosis, which may have been a contributing etiologic factor in the development of the amyloidosis. Although amyloid disease is a well-recognized sequel to chronic suppurative processes and chronic, non-suppurative inflammations, the literature previously had not contained frequent mention of this association in rheumatoid arthritis.[23]

Iritis and Other Lesions of the Eyes.—Iritis has been found in from 2.5 to 4.5 per cent of patients with rheumatoid arthritis.[8]

The iritis is often intractable. Iritis and scleromalacia perforans have been observed occasionally in rheumatoid arthritis as an integral part of the disease picture.[46,67] Rheumatic iridocyclitis and retinochoroiditis have been noted not infrequently during the course of a polyarthritis in children.[30,34] The ocular lesion may appear before, during or after the arthritis. The course of the iridocyclitis may be chronic, producing a ribbon-like keratitis. Bandlike degeneration of the cornea has been reported associated with chronic arthritis in infants and children.[49,78,79] Blepharitis is not uncommon in patients with chronic rheumatoid arthritis.

Scleromalacia Perforans.—This was first described by van der Hoeve. It is uncommon. The chief finding is the appearance of defects in the scleræ which may coalesce so that large gaps may appear in the scleræ in which the uvea lies uncovered or covered only by conjunctiva. Only 14 instances of this disorder were found in the literature up to 1938. Verhoeff and King noted that the microscopic findings in the scleræ in these cases resembled the structure of the subcutaneous nodules of arthritic patients.[74,75] More than two-thirds of the 15 cases of scleromalacia perforans reported to date have occurred in patients with rheumatoid arthritis. It should be remembered that the bones, joints and sclera are all of mesodermal origin.[42]

Angevine and Rothbard have discussed the mechanism by which bacteria become localized in the tissues of the eyes and joints.[1] In rabbits injected with hemolytic streptococci, histologic sections from the eyes and joints have shown that the synovial villi and ciliary processes were usually and most often the primary sites of inflammation when arthritis or cyclitis occurred. These bacteria were demonstrable first in the vessels of the synovial villi or of the ciliary processes bringing on a localized synovitis or iridocyclitis. This helps to explain why iritis and arthritis may both occur associated with infectious diseases.

Gastrointestinal Disturbances.—It is very unlikely that achlorhydria is of primary significance in this disease inasmuch as it occurs so seldom.[39,58] Autopsies in patients with rheumatoid arthritis have not disclosed gastritis.

Keefer[47] did not find abnormalities such as gastric anacidity, carbohydrate indigestion, colonic distention or stasis more often in rheumatoid arthritis than in other diseases. In 4 patients with rheumatoid arthritis, autopsy revealed no evidence of atrophy or other disturbance in the intestines.

Various workers have reported frequent enlargement and tortuosity of the colon in rheumatoid and osteo-arthritis.[24,62,68] As recently as 1910, colectomy was advised to rid the body of intestinal stasis which was considered to be a causative factor in the arthritis.[50] Barrow believed it was probable that the toxins responsible for arthritis were elaborated and absorbed from the cecum.[5] Haft reported intestinal changes (such as incompetency of the ileocecal valve, lack of haustration, atony and redundancy) in patients with arthritis and also in those with chronic disability.[33] Others have seen no abnormal variation or only minor changes in the colon.[43,52]

Many patients with chronic arthritis are visceroptotic and exhibit poor posture, decreased vital capacity and an asthenic type of digestive disturbance.[31,38] Fletcher and Graham noted atony of a portion or all of the colon in 65 per cent of these patients.[25] Others have found slight dilatation in some part of the colon in 36 per cent of cases, but no instances of spastic colon.[10]

Hartsock, in a study of 100 patients of typical atrophic arthritis, noted: (1) that there was a higher incidence of gastrointestinal disturbance in rheumatoid arthritis, than in other exhausting diseases; (2) gastrointestinal findings and symptoms were as marked in early cases and in young individuals as in older individuals and in more long-standing cases; (3) treatment of the gastrointestinal tract resulted in improvement of the arthritis (in the absence of other pathologic features).

It is true that some patients with rheumatoid arthritis complain less of arthritic symptoms when their bowels are moving regularly; however, there are no constant organic abnormalities of the gastrointestinal tract which are peculiar to rheumatoid arthritis.

Autonomic Nervous System.—Patients with rheumatoid arthritis frequently exhibit vasomotor abnormalities. Cobb and his associates stressed the rôle which environmental stress, such as grief, poverty and family worry may play in the onset or exacerbations of rheumatoid arthritis;[13] psychotic individuals, however, seem to be relatively immune to arthritis.[32] A detailed discussion of psychic factors in rheumatic diseases is presented in a separate chapter.

Frequent localized outbursts of sweating are characteristic of the patient with rheumatoid arthritis. Generalized sweating is a common phenomenon in many patients with a chronic febrile illness.

The average blood pressure in patients with rheumatoid arthritis is not significantly different from control groups.

Rheumatoid arthritis tends to occur in individuals with a nervous and unstable emotional constitution.[35,61] This is manifest as an increased irritability of both the vagotonic and sympathicotonic systems, known as amphotonia; there is probably a slight tendency toward the sympathetic side. *Many patients with rheumatoid arthritis were nervous or high strung as children and came from stock with similar characteristics.*

Depressing influences have frequently been noted immediately prior to the onset of a rheumatoid arthritis. Severe and lasting autonomic changes may be produced by emotional disturbance such as fear, anger, grief or anxiety. More than 25 per cent of one series of patients with rheumatoid arthritis showed a history of worry and anxiety. Some of the typical instances of this were as follows:

Male, aged thirty-seven years, with sudden onset of typical rheumatoid arthritis two months after seeing boss cut his throat; father had gout.

Male, aged fifty-four years, with sudden onset of typical rheumatoid arthritis one month after friend attempted to murder his wife, and patient sat up all night to quiet him.

Examination of the peripheral nerves of autopsied cases of typical rheumatoid arthritis have shown small nodules in the perineurium; some individuals have considered these lesions specific for rheumatoid arthritis. The nodules were usually in the perineurium, never in the endoneurium and rarely in the epineurium. If stained with hematoxylin-eosin, they were visible to the naked eye as round or oval lesions, each varying in diameter from 0.14 to 0.2 mm.[27] Similar lesions have been found in muscles (such as the gastrocnemius) by biopsy.

Microscopically, these lesions showed three zones: (1) a peripheral ring of lymphocytes and plasma cells; (2) a middle zone of polyhedral cells with large irregular nuclei, poor in chromatin; (3) and inner pale and acellular zone with homogeneously staining masses which contained occasional small dark granules of nuclear débris. In some instances, the acellular center was not present and a central blood-vessel was seen; in others, a few epithelioid cells represented the intermediate zone.

In a control series of 100 cases of non-arthritic diseases, similar lesions were not found. The likelihood of nervous system involvement in rheumatoid arthritis has been postulated for years because of the symmetrical involvement, pain along the course of nerves, sweating, interosseous muscular atrophy, glossy skin, hyperactive reflexes, and other findings; nerve, ganglion or spinal cord involvement have been considered as possible sites of involvement.

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CHAPTER IX

PSORIASIS AND RHEUMATOID ARTHRITIS

PSORIASIS may be found in from 0.25 to 3 per cent of patients with rheumatoid arthritis.[7]

Psoriatic arthritis occurs in about 1 per cent of those with cutaneous psoriasis. Some rheumatologists believe that psoriatic arthritis is merely the occurrence of two diseases (psoriasis and rheumatoid arthritis) in the

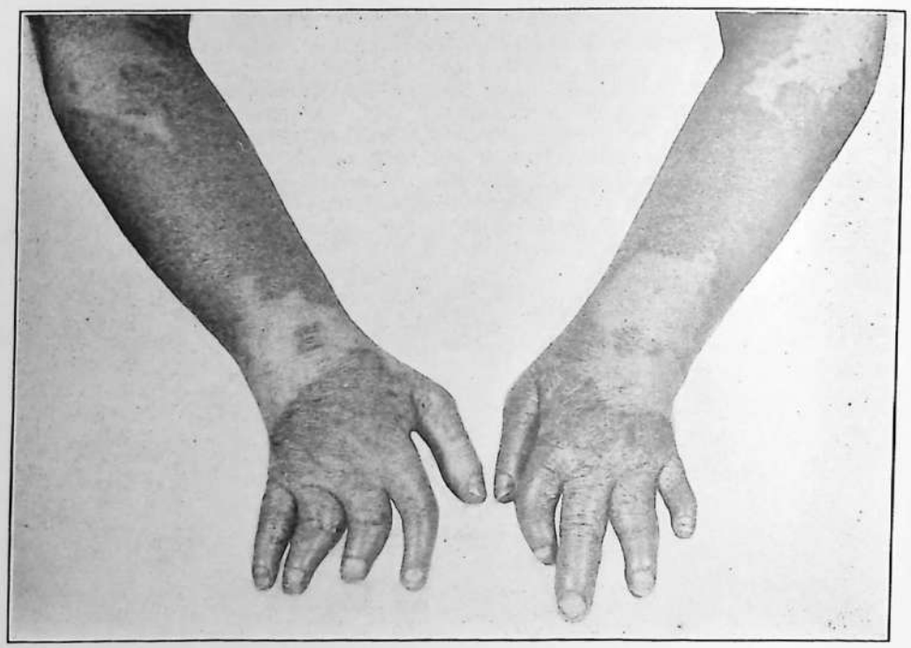

Fig. 25.—Psoriasis and rheumatoid arthritis (psoriatic arthritis).

same individual[6] while others feel that this may be a separate disease entity. Psoriasis often disappears during pregnancy, as does rheumatoid arthritis.[9] In Epstein's 22 cases, the skin lesions usually appeared on an average of 6.6 years before joint involvement was noted.[4] In 300 unselected patients with rheumatoid arthritis, Bauer and his associates found psoriasis in 2.7 per cent, as compared with an incidence of psoriasis of 0.7 per cent in a similar number of controls. *This association is not merely a coincidental occurrence of two common diseases, but is a significant one.*

Of 20 such patients observed by Dawson, the psoriasis preceded the arthritis in 15. In 9 of the patients, exacerbations and remissions of the arthritis accompanied similar changes in the psoriasis. The *arthritis found*

in association with psoriasis is often indistinguishable from the usual form of rheumatoid arthritis by clinical or roentgen examination. However, Hench has pointed out that when the hands or feet are involved, there is a tendency *toward lesions in the terminal joints of the fingers and toes. Psoriatic arthritis usually appears associated only with long-standing chronic psoriasis.* Psoriatic lesions (pitting, retraction, hyperkeratosis, etc.) may be found adjacent to the affected joints. Activity of the disease and terminal deformity

FIG. 26.—Psoriatic arthritis. (Shlionsky and Blake, courtesy of Ann. Int. Med.)

have been reported to be less in psoriatic arthritis than in simple rheumatoid arthritis, but some of these patients present marked deformities, as in severe rheumatoid arthritis.

The first comprehensive study of the clinical association of joint involvement and psoriasis was made by Bourdillon in 1888.[2] Joint pains in psoriasis were noted as early as 1822.[3] Various names have been applied to the combination of rheumatoid arthritis and psoriasis; these include psoriatic arthritis, psoriasis arthropathica, arthropathia psoriatica and

psoriasis arthritica. The occurrence of psoriasis together with degenerative joint disease is considered to be merely a matter of chance.

Dawson has found that less than 2 per cent of cases of rheumatoid arthritis develop psoriasis. He has reported 19 cases of psoriasis in patients with rheumatoid arthritis; in 7 of these the psoriasis was atypical and exudative; in 5, the psoriasis and the rheumatoid arthritis were of the usual variety, in 7 others, although the psoriatic lesions were atypical in character and distribution, the hands and feet were frequently affected,

FIG. 27.—Psoriatic arthritis. (Shlionsky and Blake, courtesy of Ann. Int. Med.)

and there was a marked tendency toward involvement of the nails in the process. In this latter group, there was an exudative eruption with waxy, translucent papules in addition to large abundant scales. Exacerbations and remissions of the two diseases coincided. The atypical feature of the rheumatoid arthritis was the **frequent involvement of the terminal interphalangeal joints.**

Although any joints were involved, the hands and feet were affected most frequently. The nails of the fingers and toes were involved in most cases.

In the group of cases with psoriatic arthritis, there is a frequent *tendency to marked destructive joint changes.* Cases have been reported[1,11–13] in which such marked atrophy and destruction occurred in the phalanges of the hands and feet that some of the phalanges were almost completely absorbed.

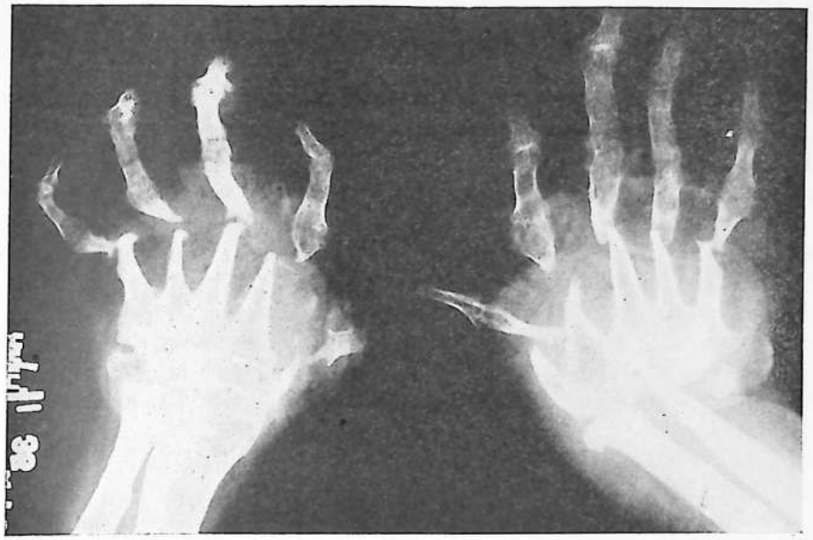

FIG. 28.—Psoriatic arthritis. (Shlionsky and Blake, courtesy of Ann. Int. Med.)

FIG. 29.—Psoriatic arthritis. (Shlionsky and Blake, courtesy of Ann. Int. Med.)

In some instances, the destructive process extended to the metacarpals and carpals.[5,14] Bony involvement in the proximal portions of the extremities is not as common. Such extensive bone and joint changes with extensive loss of bony substance and tapered narrowing of bony fragments in the hands and feet has been described in patients in whom no psoriasis was present.

Bauer and his associates observed 26 patients with chronic arthritis and psoriasis; in 23, the arthritis was of the rheumatoid type. In the 3 remaining patients, arthritis was confined to the terminal phalangeal joints (with psoriatic lesions of the nails of the involved digits).

Bauer and his associates have frequently seen, clinically and at autopsy, involvement of the terminal interphalangeal joints in patients with rheumatoid arthritis. The lesions of the terminal interphalangeal joints were similar to those occurring elsewhere in these patients. They pointed out that these lesions differed markedly from those observed in a psoriatic patient with arthritis limited to the terminal interphalangeal joints.[1]

Among the 23 patients who presented a rheumatoid type of arthritis, 17 showed psoriatic nail changes; in 13 of these there was an associated involvement of the terminal phalangeal joint. *Synovial tissues* were studied in 6 of these 23 patients and were found to be *similar to those found in rheumatoid arthritis*. These workers found chronic synovitis of the knee joints, with hyperemia, inflammatory cell infiltration with focal collections of lymphocytes (often with germinal centers), and hypertrophied villi. Ankylosis, subluxation with deformities and atrophy of bone were found in the advanced cases. In one of these patients with psoriasis of the skin and nails, there was an ankylosing polyarthritis with arthritis of some of the terminal phalangeal joints.[1]

This individual first noted arthritis in the terminal joint of the left index finger at twenty-four years of age, and subsequently developed deformities in most of the terminal joints of the other fingers and thumbs during the next forty-four years; these were associated with changes in the nails and paronychial inflammation. Psoriasis was first noted at the age of thirty-nine, fifteen years after the first joint involvement. During the last twenty-two years of his life he had also had arthritis in most of the distal joints of the toes. The shortened fingers and toes, and swollen and deformed terminal joints were readily apparent clinically. At autopsy (following a coronary artery occlusion), the joints of both feet and one hand were examined minutely.

Marked articular destruction and resorption of bone, with pronounced shortening of the middle phalanges, had occurred in the terminal phalangeal joints of the fingers and toes. In the distal phalanges, the resorptive changes were less marked but cuplike deformities were formed at the proximal ends of these bones by pronounced marginal overgrowth of bone at the sites of insertions of tendons. The diffuse atrophy usually seen in rheumatoid arthritis, was not present. Dense acellular fibrous tissue (with minimal or no inflammatory changes) replaced the joint spaces in most of the digits. There was no involvement of the proximal interphalangeal, metacarpophalangeal or metatarsophalangeal joints (which are usually affected in typical rheumatoid arthritis).

Bauer concluded that *in most instances the joint changes in rheumatoid arthritis are indistinguishable from those in rheumatoid arthritis associated with psoriasis*. However, in an occasional patient, unusual and distinctive alterations may occur in the terminal interphalangeal joints.[1]

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CHAPTER X

LABORATORY FINDINGS IN RHEUMATOID ARTHRITIS

A GREAT number of laboratory tests have been employed in rheumatoid arthritis. Aside from the complete blood count, urinalysis and blood Wassermann test which are performed routinely on all patients entering the clinic, there are only a few tests of definite clinical value to the general practitioner in this disease. The *sedimentation rate* and *roentgen examinations* of various joints are undoubtedly very helpful. Other tests may be useful in differential diagnosis—such as the blood uric acid, Gc complement fixation test, B. abortus agglutination tests, synovial fluid findings, joint culture and biopsy, B.M.R., agglutination tests for hemolytic streptococci, antistreptolysin content of serum, antifibrinolysin, precipitins, Weltmann coagulation reaction, formol-gel reaction, blood culture, serum phosphatase and various blood chemistry values (calcium, phosphorus, sugar, cholesterol, serum proteins, etc.), as well as numerous other examinations. *The general practitioner should routinely obtain a blood count, urinalysis, B.W.R., sedimentation rate* and *roentgen examinations* in his patient with rheumatoid arthritis.

Roentgen Findings.—Roentgen-rays were first demonstrated by Roentgen in 1895. Within a year following this, they were used in the diagnosis of "rheumatism." In a roentgenogram of a joint, one actually obtains:

A macroscopic positive picture of the bones.

A negative representation of cartilage obtained by the absence of a shadow.

Some knowledge concerning the soft parts.

Usually no evidence of alteration of synovial membrane.

During the progression of rheumatoid arthritis, various changes may be seen by roentgen examination. In the earliest stages, the roentgen picture may be essentially normal until one reaches the stage of slight swelling of the joint; at this time, soft tissue films will disclose the increased periarticular width and density and one may see some atrophy of the epiphyseal line. As the cartilage becomes thinned, the interosseous space becomes more narrow and further atrophy of epiphyseal bone occurs. With progression of the process, erosions and marginal bony exostoses may appear. In a more advanced stage, the interosseous space becomes obliterated (because of destruction of hyaline articular cartilage), and there is destruction of articular bone. The epiphyseal line becomes more atrophied and marginal lipping of bone may be noted. Variable degrees of articular destruction are seen on the roentgenogram. Complete bony ankylosis may occur eventually in some cases.[29]

In a roentgen study of 207 joints in 59 patients with rheumatoid arthritis, marked systemic decalcification was found in 95 per cent, occurring throughout the skeleton but being seen best in the hands and feet. *Punched out areas* (atrophic destruction of bone) were noted *in 85 per cent* of cases,

occurring as frequently as in gout (although such areas were usually larger in gout). *Effusion into the joint* was demonstrated *in 88 per cent* of cases, being dense in 80 per cent, sharp and fusiform in 73 per cent and evenly distributed in 70 per cent. *Narrowing and obliteration of the joint space was seen in 95 per cent of cases.* Swelling of the soft tissues occurred in 41 per cent of cases and atrophy in 33 per cent. **Lipping and osteophytes** (production of bone) **were seen in 77 per cent** of patients with rheumatoid arthritis. Ankylosis was noted by roentgen-ray in 26 per cent of these patients.

Roentgen Findings in Rheumatoid Arthritis

1. No changes may be apparent very early in the disease.[32]
2. The first roentgen sign is usually *periarticular soft tissue swelling*—a fusiform swelling of the soft tissues about the joint indicated by increased density of the soft tissues (due to proliferation of synovial membrane, joint effusion and thickened joint capsule).
3. Generalized *skeletal decalcification* is an early sign.[61] The decalcification is usually more marked in the articular ends of the bones. This demineralization cannot be distinguished roentgenologically from that of hyperparathyroidism, hyperthyroidism, rickets, osteomalacia or senile osteoporosis. There is rarefaction of the trabeculated ends of the bones, followed by atrophy of the ends of the bones, thinning of the articular cortices and a ground glass appearance and atrophy of disuse in the shaft.[67] Generalized decalcification of the bony skeleton is one of the most characteristic roentgen findings.[2]
4. Definite effusion within the joint presenting a dense, sharply outlined and fusiform appearance.
5. Progressive *narrowing of the joint space* (indicating thinning of the articular cartilage).
6. *Destruction of the cartilage* and the presence of cortical erosions with punched out areas of bone along the articular surfaces; these are usually smaller than those of gout (in gout, there is no alteration in bone density no matter how advanced the stage).
7. The *articular surfaces* become *irregular* and may be *destroyed;* this may be followed in some cases by *subluxations* and *dislocations.*
8. *Marked transparency of bones.*
9. *Hypertrophy of bone* may appear in the *late* stage; this may be seen relatively early in weight-bearing joints.
10. *In the very late stages* of *severe rheumatoid arthritis,* very little, if any, periarticular swelling remains, but there is marked atrophy of bone, destruction of cartilage, moderate bony lipping and osteophytes (in some cases), marked destruction of bone, disarrangement of the joint and deformity. Fibrous or bony *ankylosis* may occur, resulting in continuity of the trabeculated ends of adjacent bones.[10] Bony ankylosis is most common in joints with a narrow range of motion, such as the sacro-iliac joint or carpus. Surrounding muscles become atrophied and occasionally small calcified deposits may be seen about the joint margins. One may see deviation, luxation or subluxation, thickening of the bones and deformity of the bony ends at the joints due to mechanical forces. If the disease becomes quiescent, there may be an increase in bone density.
11. The roentgen findings of rheumatoid spondylitis are considered in a separate section; see Spondylitis.

Marked limitation of motion may occur even in the absence of obvious roentgen changes in the joints.

The earliest roentgen findings may not appear for years after the onset of joint symptoms.

The joints most often involved radiologically are the proximal interphalangeal, metacarpophalangeal (thumb usually not being involved), wrist, elbows, knees, tarsal and metatarsophalangeal; shoulders and hips are affected less often.

FIG. 30.—A female, aged thirty-eight years, with extensive involvement of all the carpal, metacarpal and many interphalangeal joints. There are marked atrophic changes with bony ankylosis involving some of the joints. **Rheumatoid arthritis.**

By a study of serial roentgenograms taken several years apart, it was found that *good function was compatible with even marked roentgen changes, and that functional recovery was possible although radiologic changes were increasing.*[51] *Changes in roentgenograms usually continued for years after the arthritis was quiescent clinically. The roentgen picture rarely showed improvement;* recalcification of bone and moderate repair was seen roentgenographically in only 2 patients.

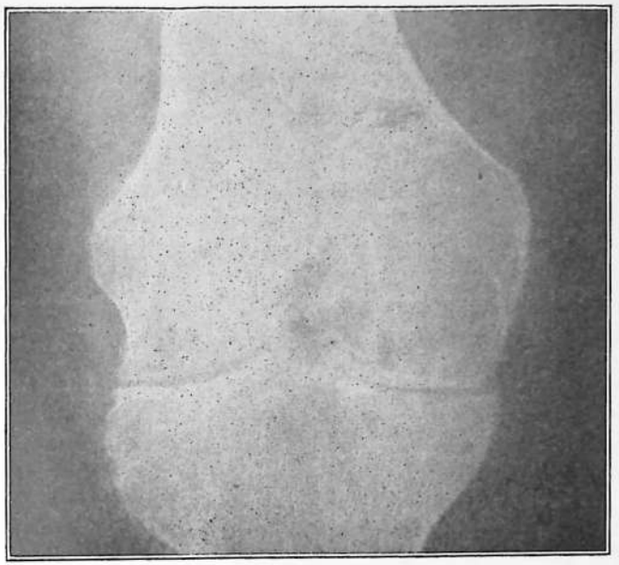

FIG. 31.—Rheumatoid arthritis. Marked narrowing of joint space at knee joint indicating destruction of cartilage; homogeneous ground glass appearance in the bones.

FIG. 32.—Advanced rheumatoid arthritis. Marked osteoporosis; deformities and luxations. Destruction of cartilage.

Roentgen Differentiation of Rheumatoid Arthritis from Gout

Punched out areas may occur in both rheumatoid arthritis and gout.
When surrounded by a sharply circumscribed swelling in the soft tissues (tophus), these punched out areas are typical of gout.
Gout rarely shows bony ankylosis.
Gout shows no systemic decalcification, less frequent thinning of cartilage, and a more asymmetrical distribution of joint swelling.
Bony cysts are usually larger in gout than in rheumatoid arthritis.
Gout is often localized in the metatarsophalangeal joints of the big toe, finger joints and wrist.

Roentgen Differentiation of Rheumatoid Arthritis and Degenerative Joint Disease (Osteo-arthritis)

	Rheumatoid Arthritis	Degenerative Joint Disease
Earliest signs	Fusiform swelling of soft tissues and rarefaction of trabeculated ends of bones	Formation of osteophytes and narrowing of the joint space
Soft tissue involvement	Fusiform swelling about joints, especially in phalanges	Usually none
Lipping and osteophytes	In about 75 per cent of cases	In every case
Heberden's nodes	None	Very frequent
Punched out areas of bone at articular margins	Very common	Usually absent
Systemic decalcification	In almost every case; may be the earliest roentgen sign	Mild and consistent with age of patient
Narrowing of joint space	In almost every case	In about 50 per cent of cases
Ankylosis (fibrous and bony)	In approximately 25 per cent of cases	Very rare; only in spine as a locking of osteophytes

Adhesions in arthritic joints have been visualized by the intra-articular injection of "arthropsin," a yellow dye (a 10 per cent solution of the disodium salt of tetra-iodo-sulfo-benzoic acid).[35]

Sedimentation Rate.—This is moderately to markedly increased in rheumatoid arthritis.[65] A normal sedimentation rate in active rheumatoid arthritis is so unusual that such a finding almost excludes this diagnosis.[25] The increase in sedimentation rate is usually roughly proportional to the severity of the process. As improvement occurs, the sedimentation rate gradually returns toward normal in most cases. In some patients the sedimentation rate remains high even though marked functional improvement has occurred. The description of this test and its relationship to various forms of arthritis is described in a previous chapter.

Blood Count.—Elevation of the non-filament count above 16 per cent occurs in over 95 per cent of cases.

W.B.C. is between 5000 and 10,000 cells per cubic millimeter in 80 per cent of cases; leukocytosis of from 12,000 to 20,000 is frequently seen in the acute stage. Leukopenia is not uncommon in chronic, long-standing cases. In children, the W.B.C. may rise as high as 50,000 during the acute stage of the disease.[2]

Polymorphonuclear neutrophils—average—64 per cent.

Slight persistent eosinophilia may occur in severe cases.

Simple hypochromic anemia is frequently present, usually of moderate degree—occasionally marked. Anemia is present more frequently in clinic than in private patients. Reduction in hemoglobin is greater than reduction in erythrocytes.

Marked anemia is often present in rheumatoid arthritis; Haden believed this was due to the toxemia of the disease, and to the frequently associated achlorhydria (resulting in impaired absorption of iron).

Haden believes that changes in the leukocytes are not characteristic in rheumatoid arthritis and that there is no significant alteration in the differential count. Although leukopenia[25] is frequent in long-standing cases, a mild polymorphonuclear leukocytosis may occur.[25] He has not observed toxic granulation in the leukocytes in rheumatoid arthritis.

Blood Volume.—About 12 per cent above normal.[66]

Blood Chemistry.—Normal values are usually found for serum calcium,[3,26,60] serum phosphorus,[33] serum magnesium,[57] serum uric acid, sugar, urea, creatinin and phosphatase.[68]

The concentration of serum phosphatase is essentially normal regardless of the severity of the rheumatoid arthritis; thus, *if the individual with rheumatoid arthritis shows a definitely increased serum phosphatase, one should suspect some coincident bone disease,* such as osteitis (Paget's disease) or malignancy.[28] Increase in the blood calcium has been noted occasionally.[31,46,73]

The serum phosphatase was normal in 30 patients with rheumatoid arthritis and in 3 patients with degenerative joint disease. In 2 patients with rheumatoid arthritis with an elevated serum phosphatase, *x*-rays of the tibiæ showed changes consistent with Paget's disease. The finding of a normal serum phosphatase in rheumatoid arthritis indicates the lack of marked osteoblastic activity in this disease, which is in contrast to those conditions in which decalcification is accompanied by marked osteoblastic activity as an attempt to produce rapid repair.

Recent investigations have shown that the *calcium and phosphorus metabolism is grossly normal in patients with degenerative joint disease and in those with rheumatoid arthritis.* Detailed analyses indicated a slight tendency toward an increased rate of calcium metabolism and increased calcium excretion in individuals with rheumatoid arthritis and a decrease in calcium excretion in those with degenerative joint disease. The magnitude of increased loss and retention was small and may not be of importance but the variations from normal cannot be explained by other factors known to influence calcium metabolism. The serum calcium and phosphorus values were essentially normal in all cases.

This work provides strong evidence against the hypothesis that rheumatoid arthritis is due to hyperparathyroidism, as was proposed by some earlier workers.[60]

The total sulfur, total nitrogen and the amino-acid content of total serum proteins from normal subjects showed no essential difference from patients with rheumatoid arthritis.[5]

Total, free and ester serum cholesterol, and total serum lipids are normal.[4,8] Serum phospholipids are slightly elevated.[4]

Total blood proteins are normal,[1,14,62] with a rise in globulin and a fall in albumin fractions, and a slight rise in fibrinogen content.

Diminution of plasma pigments.

Glucose tolerance test—normal in over 85 per cent of cases.[36]

Formol-Gel Reaction.—This is usually positive in patients with active rheumatoid arthritis or mixed arthritis.[63]

Although some workers employ the formol-gel test as an index of clinical activity of the disease, the sedimentation rate probably is a more accurate measurement of this. However, the formol-gel test gives a simple qualitative test for increase of globulin or fibrinogen in the blood; the test depends upon the change in viscosity of the body fluids after formaldehyde is added.[27]

Liver Function.—Hepburn and his associates employed the hippuric acid test to study the liver function of 28 patients with rheumatoid arthritis and degenerative joint disease. Neither group showed any greater deficiency in hepatic function than that shown by a group of individuals suffering from a systemic disease. Liver dysfunction (as determined by the Azorubin S, hippuric acid and bilirubin excretion tests) has been reported in 50 to 60 per cent of patients with severe rheumatoid arthritis.[58,59]

Blood Groups.—These are essentially what would be expected in a random sampling of the population.

Urine.—No constant abnormalities.

Synovial Fluid.—Discussed in a previous section; see synovial fluid.

Basal Metabolic Rate.—Normal in a majority of cases.

Blood Culture.—Variable results have been obtained. Most workers have been unable to culture streptococci from the blood of patients with rheumatoid arthritis.[16,24] Others[22,47] have demonstrated these organisms in the blood stream in as many as 69 per cent[11] of their patients. Dawson has concluded that *"up to the present time no convincing evidence has been brought forward that organisms are actually present in the blood or tissues of patients suffering from rheumatoid arthritis."*

Fraser made blood cultures from 61 patients with rheumatoid arthritis, employing the method described in 1929 by Cecil and his co-workers. Streptococci were not recovered from any of these. In 61 control patients, a third or more of whom had diseases due to chronic bacterial infection (chronic cholecystitis, chronic osteomyelitis), Streptococcus viridans was recovered from the blood in 3 after dental extraction for apical abscess.[21]

Fraser made a brief historical survey of blood culture studies in rheumatoid arthritis and concluded that one is not justified to state from blood culture findings that rheumatoid arthritis is a disease of infective origin. He suggested, agreeing with Okell and Elliott, that bacteremia may occur from trauma or treatment (such as massage).[55]

Spinal Fluid Findings.—These are usually normal except an increase in total protein, abnormal colloidal gold curves or a combination of the two in some patients with rheumatoid spondylitis.[44,45]

Agglutination Tests for Hemolytic Streptococci.[34,48,54]—It has been shown that the serum of most individuals with rheumatoid arthritis will agglutinate hemolytic streptococci (Group A, Lancefield) in a titer of 1:160 or more. This reaction does not become positive in most cases until some months after the disease is well established; the property is usually absent in children and adolescents. *"Agglutination in a titer of 1:160 or higher rarely occurs in any other disease except frank hemolytic streptococcus infection."*[15] Such agglutinins tend to disappear as the individual recovers. This reaction suggests an etiologic relationship between arthritis and hemolytic streptococci but certainly does not prove this. Agglutination reactions with other organisms are essentially negative; sera from individuals with other diseases do not ordinarily produce such agglutination.

Streptococcus agglutination and precipitin tests are not done routinely as they are complicated procedures and do not offer sufficient diagnostic aid to justify their expense.[2]

Antistreptolysin Content of Serum.—Antistreptolysin (antihemolysin or antistreptohemolysin) is an antibody which neutralizes streptolysin; the latter is a soluble product of hemolytic streptococci which produces hemolysis of erythrocytes. The *antistreptolysin content of the serum is increased in acute hemolytic streptococcal infections;*[9] it may be increased in the early stage of acute cases of rheumatoid arthritis but is not found in any significant titer later in the disease.[52] Antistreptolysin titers are distinctly lower in rheumatoid arthritis than in rheumatic fever.[43]

Antifibrinolysins.—Hemolytic streptococci will liquefy normal human fibrin clot; the plasma clots from the blood of patients convalescent from acute hemolytic streptococcal infections resist this fibrinolytic action. Such resistance is due to the presence of antifibrinolysins. The latter are usually present in the blood of patients with rheumatic fever but not in rheumatoid arthritis.[53,56,69,70] Antifibrinolysins are also indices of acute hemolytic streptococcal infections and as such may be present in the early acute stage of rheumatoid arthritis, but not later in the disease.

Perry found the antifibrinolysin titer to be normal in 161 out of 171 tests in 33 patients with rheumatoid arthritis; this is in marked contrast to the high titers found in rheumatic fever and does not support the thought that hemolytic streptococci cause rheumatoid arthritis. It is also evidence against a common etiology of the two diseases.

Precipitins.—Hemolytic streptococci contain a nucleoprotein (P)[38–41] (which is not specific as it gives cross-precipitation with other Gram-positive cocci), a group specific carbohydrate (c), and type specific protein and carbohydrate fractions (M and S). The nucleoprotein fractions are precipitated by sera of patients both in active rheumatic fever[12] and rheuma-

toid arthritis.[7,17,64] A close approximation has been noted between precipitin and agglutination reactions.

Skin Tests.—*Numerous investigators have reported positive skin tests to hemolytic streptococci in a large majority of patients with rheumatoid arthritis.* The significance of this is questionable. These are discussed elsewhere.

Mester Test.—As yet *there is no specific test which will differentiate rheumatic from other diseases.* Mester recently claimed that he had found such a reaction; this depended on the special sensitivity of rheumatic patients to salicylic acid.[49]

The technic of the test is as follows: A white blood count is made on the resting and fasting individual. Then 1 cc. of a 0.1 per cent aqueous solution of salicylic acid is injected intradermally, forming 5 blebs of 0.2 cc. each on the flexor aspect of a forearm. Another white count is made after thirty minutes, during which time the patient sits or lies down and does not eat or drink. In his series, nearly all rheumatic patients (acute or chronic) showed a decrease in the number of leukocytes after thirty minutes. A positive reaction was a decrease of 15 per cent or more in the white blood count, but some patients showed a drop of 50 to 70 per cent. The control group was negative while 98 per cent of the rheumatic cases gave a positive result.

Copeman's findings did not confirm this work; he found the test to be negative in frank rheumatic disease and positive in patients not having rheumatic disease and concluded that this test did not help in the diagnosis of rheumatic disease.[13]

Lenoch found the reaction to be positive frequently in rheumatic disease but also obtained positive tests in non-rheumatic patients. The test was negative in degenerative joint disease.[42]

Others used a modification of Mester's reaction and found the test to be grossly unreliable and thus of little value in differentiating certain forms of rheumatic disease.

Weltmann Coagulation Reaction and the Sedimentation Test in Arthritis.—In 1930, Weltmann described a test utilizing the observation that diluted serum required different amounts of bivalent electrolytes for heat coagulation according to the pathologic process involved. It was necessary to add higher concentrations of calcium chloride to sera from exudative processes than to normal sera to produce coagulation. Less calcium chloride was required to be added to sera from cases with fibrotic processes and hepatic damage as prominent features. This test is not dependent upon the total serum protein or the albumin-globulin ratio; it is not influenced by the concentration of calcium and chlorides in the serum. It is assumed that the reaction depends upon qualitative changes in the serum proteins caused by exudative or fibrotic local tissue changes.

Kling reported that the Weltmann coagulation reaction did not possess the general utility of the sedimentation test, which latter seemed far superior as a check on the clinical course and effect of therapy. He performed this test in a series of 250 cases of arthritis, including 98 with rheumatoid arthritis and 88 with degenerative joint disease (with 64 miscellaneous cases).

Technic of the Weltmann Reaction.—1. A stock solution of 10 per cent crystalline $CaCl_2 \cdot 6H_2O$ is used.

2. From this, 10 dilutions are prepared, ranging from 1:1000 to 0.1:1000 (the integral between each being 0.1:1000).

3. Four and nine-tenths cc. of solution is pipetted into each of 10 test tubes. These are numbered according to their concentration of calcium chloride, tube 1 containing the highest concentration—being placed on the left.

4. One-tenth ml. of clear non-hemolyzed serum is added to each tube and the tubes are shaken.

5. These are heated in a boiling water-bath for fifteen minutes.

6. After heating, the contents of the tubes may remain clear, or become turbid or flocculent.

7. The number of the tube showing flocculation is read and designates the coagulation band.

8. In normal sera, flocculation occurs in tubes 6 or 7, corresponding to 0.5 to 0.4:1000 calcium chloride. Flocculation occurring in only tubes 1 to 5 is called a short band (a shift to the left, or exudative zone). Flocculation extending to tube 8 and further is known as a long band (shift to the right or fibrotic zone).

9. Modifications of this technic have simplified this.

In Kling's cases, the coagulation band was markedly short in only 53 per cent of cases of rheumatoid arthritis while the sedimentation rate was markedly increased in 82 per cent of this same group. Thus, the Weltmann reaction was judged to be inferior to the sedimentation rate as an indicator of rheumatoid arthritis. This was also true in those cases in which both tests were used in a group of patients with degenerative joint disease (osteo-arthritis).

Milles and Salt investigated the blood of 150 cases of rheumatoid arthritis or allied diseases by means of the sedimentation rate, the formol-gel test, the Weltmann reaction (calcium chloride heat coagulation test) and the Takata-Ara reaction (mercuric chloride flocculation test). They concluded that the sedimentation rate and Weltmann reaction were very sensitive indicators of the extent of the activity of rheumatic disease, but that the two did not always parallel each other. In patients with severe rheumatoid arthritis, there was a moderate shift to the left in the serum coagulation band; acute stages of rheumatic fever produce a much greater deviation to the left. An occasional shift to the right in rheumatoid arthritis seemed to them to indicate a good prognosis.

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CHAPTER XI

RHEUMATOID ARTHRITIS: DIFFERENTIAL DIAGNOSIS

THE typical case of rheumatoid arthritis usually offers very little difficulty in diagnosis. In the early stages of the disease, or in atypical cases, one may encounter trouble. About two-thirds of all chronic arthritis is either rheumatoid arthritis or degenerative joint disease: the differentiation of these two disorders is tabulated.

Other forms of joint disease which may resemble rheumatoid arthritis include:[3]

1. Gonococcic arthritis.
2. Atypical rheumatic fever.
3. Acute gout.
4. Tuberculous arthritis.
5. Arthritis possibly associated with focal infection.
6. Periarticular fibrositis.
7. Syphilitic arthritis (uncommon).
8. Arthritis associated with lymphogranuloma venereum.

Degenerative Joint Disease.—Although it is usually relatively easy to distinguish between rheumatoid arthritis and degenerative joint disease, the two entities frequently are present in the same patient, producing a mixed type of arthritis. In the typical patient with **rheumatoid arthritis,** *distinguishing features include* the age of the patient at the onset of his disease (most often between twenty and forty), the typical fusiform swelling of the proximal interphalangeal joints of the fingers, with involvement also in many cases of the knees, metacarpophalangeal joints, wrists, elbows, ankles, etc.; symmetry of joint involvement frequently; the presence of constitutional symptoms such as loss of weight (which may be marked), fever, tachycardia, anemia and, in some cases, enlargement of lymph nodes and of the spleen; marked muscular atrophy as the disease progresses; joint effusion; and the increased sedimentation rate. In a purely **degenerative joint disease** (osteo-arthritis), the sedimentation rate is normal. The patient is well nourished unless there is some accompanying organic pathology. The typical lesions are the *Heberden's nodes* seen at the distal interphalangeal joints of the fingers; other joints commonly involved include the knees, cervical and lumbar spine and sacro-iliac joints. There are usually no constitutional symptoms, no muscular atrophy (except that which may accompany ageing), no or very little joint effusion and the process does not go on to ankylosis (which may occur in rheumatoid arthritis). The patient with degenerative joint disease is usually past forty years of age, except when the joint pathology is secondary to acute or chronic trauma, or in women who have had a premature or artificial menopause. Only a few joints are usually involved in degenerative joint disease, as contrasted with the multiple, usually symmetrical involvement

in rheumatoid arthritis. Subcutaneous nodules occur in from 10 to 20 per cent of patients with rheumatoid arthritis, but are not seen in degenerative joint disease. The roentgen findings are different in the two disorders:

	Rheumatoid Arthritis vs.	Degenerative Joint Disease
Synonyms	Atrophic; chronic infectious; proliferative	Hypertrophic; osteo-arthritis; degenerative arthritis
Age of onset	Usually between 20 to 40 years	Most often after 40 years
Prodromes	Often marked: fatigue; nervousness, chilliness, numbness and tingling in extremities, etc.	Usually none
Involvement of hands	Especially proximal interphalangeal and metacarpophalangeal joints	Especially terminal interphalangeal joints (Heberden's nodes)
Other joints commonly involved	Wrists, toes, knees, elbows, ankles, shoulders, hips, temporomandibular, sternoclavicular	Knees, lumbar spine, sacro-iliac, lower cervical spine
No. of joints involved	Usually many	Usually few
Symmetry of joint involvement	Most often symmetrical	Often asymmetrical
Type of patient	Usually thin, asthenic, viscerоptotic	Often overweight; sthenic
Type of onset	Usually insidious; may be abrupt	Usually insidious
Constitutional symptoms	Fever, loss of weight, anemia, tachycardia; often enlargement of lymph nodes and spleen	Usually none
Type of joint swelling	Usually fusiform or spindle shaped	Usually irregular or knobby
Muscular atrophy	Usually present and may be marked	Not frequent; not marked
Skin changes	Cold, clammy hands; smooth, shiny skin in extremities	None except those of age
Joint effusion	Usually present; may be marked	Usually none
Subcutaneous nodules	Present in 5 to 25 per cent	Absent
Sedimentation rate	Usually rapid	Usually normal
X-ray findings	Early: fusiform periarticular soft tissue swelling; rarefaction of trabeculated ends of bones and systemic decalcification	Early: narrowing of the joint space due to destruction of cartilage; formation of osteophytes and lipping
	Later: decrease of joint space, lipping and osteophytes, punched-out areas of bone at articular margins, and possibly ankylosis, subluxations, etc.	Later: cysts at ends of bone, deformities from pressure and traction; no ankylosis
Ankylosis	In about 25 per cent in later stages	No true ankylosis
Primary pathological change	Synovitis	Degeneration of hyaline articular cartilage; no inflammatory changes
Agglut. test with hemolytic streptococci	Strongly positive in over 50 per cent of cases after several months of illness	Usually negative
Progress of the disease	Marked tendency to progression despite treatment	Slow progress; often arrested by supportive measures
Prognosis	Very doubtful; 10 per cent progress to ankylosis and invalidism despite good therapy	Usually good as regards function; usually no crippling; no flexion deformities

in degenerative joint disease, one does not see the fusiform soft tissue swelling, nor the systemic decalcification so characteristic of rheumatoid arthritis; degenerative joint disease does not progress to bony ankylosis (as may occur in rheumatoid arthritis); one may occasionally see an interlocking of bony spurs in degenerative joint disease. Despite these clear-cut differences, one must always remember in dealing with older patients that both of these disorders may be present in the same individual.

Rheumatic Fever.—The patient with typical rheumatic fever can usually be differentiated readily from other forms of joint disease; however, in the early stage, this may be difficult and in some cases the passage of considerable time is necessary before a final diagnosis can be made. *Important diagnostic features* of **rheumatic fever** include its onset under fifteen years of age in many cases; a history of preceding upper respiratory infection in most instances; negative roentgen findings except soft tissue swellings; the absence of any permanent joint damage; the dramatic response in most instances to adequate doses of salicylates with complete relief of pain and return of temperature to normal; the occasional finding of electrocardiographic evidence of cardiac damage; occasional involvement of the pleura or lungs; negative Gc complement fixation test; absence of tenosynovitis; and the marked infrequency of an associated conjunctivitis. The antistreptolysin titer is usually increased; this titer is normal in rheumatoid arthritis (but unfortunately this test is usually not available to the average practitioner). The agglutination reaction to hemolytic streptococci is usually negative in rheumatic fever and positive in most cases of rheumatoid arthritis (after some months of the disease). *Points of similarity* between rheumatoid arthritis and rheumatic fever include the rapid sedimentation rate in both; the multiple joint involvement, often symmetrical; the occurrence of subcutaneous nodules in both diseases; the tendency to occur in the same families, chiefly in the temperate zones, often preceded by an upper respiratory tract infection; the possible etiologic rôle of hemolytic streptococci in both; and the occurrence of cardiac damage in rheumatic fever and the recent autopsy findings which show evidence of cardiac damage identical with that of rheumatic fever in a large percentage of patients with rheumatoid arthritis. The *main points of differentiation* include the age incidence (inasmuch as 90 per cent of cases of rheumatoid arthritis begin after the age of fifteen); permanent joint damage (which occurs in rheumatoid arthritis); response to salicylates (which is dramatic in rheumatic fever); the roentgen findings (which include osseous changes and evidence of cartilaginous damage or destruction in rheumatoid arthritis); the occasional electrocardiographic change (in rheumatic fever); and, if available, agglutination reaction to hemolytic streptococci and antistreptolysin titer. *Rheumatic fever is not uncommon in an army at war.*

Gonococcic Arthritis.—Gonorrheal arthritis can be diagnosed readily in most instances by a history of gonorrhea during the preceding several weeks; the findings of gonococci by smear or culture in the joint fluid (in about 25 per cent of cases) or in the urethral discharge; the positive gonococcus complement fixation test in most cases (especially if repeated); the

	Rheumatoid Arthritis	Rheumatic Fever	Gonorrheal Arthritis
Age at onset	*90 per cent of cases after age of 15*	*First attack under 15 years in 90 per cent; may occur at any age*	Most often 20 to 40 years; may occur at any age
Preceding upper respiratory infection	Frequent	In most cases	In about 10 per cent
Roentgen-rays	*Characteristic:* may be negative early; later generalized osteoporosis, etc.	*Usually negative except for soft tissue swelling*	May show rapid bony destruction
Permanent joint damage	May occur	*Usually none*	May be marked
Subcutaneous nodules	Present in about 20 per cent of cases; may persist for weeks	Present in about 20 per cent of cases	Absent
Tenosynovitis	Usually absent	Usually absent	Present *in almost 50 per cent of cases*
Response to salicylates	Temporary relief, but no dramatic response	*Usually dramatic* with complete relief of pain and return of temperature to normal *if adequate doses* are used	Not dramatic; some relief of pain and some reduction of temperature
Involvement of pleura or lungs	None	Not uncommon	None
Presence of serous conjunctivitis	Absent	Very uncommon	In about 20 per cent of cases
Response to sulfonamides	Very poor or none	Very poor or none; may be made worse in acute stage	Excellent
Complement fixation test (Gc)	Usually negative	Almost always negative	Positive in over 80 per cent of cases if repeated every 10 days for 3 examinations
Synovial fluid	No gonococci	No gonococci	Gonococci demonstrable by smear or culture (in at least 25 per cent of cases)
Electrocardiogram	Usually negative	Evidence of cardiac involvement may be seen	Usually no changes
History of gonorrhea	Usually negative; possibly coincidental	Usually negative	*Usually within several weeks preceding joint pains*
Agglutination reaction with hemolytic streptococci	*Strongly positive in most cases of some months' duration*	Usually negative	Usually negative
Antistreptolysin titer	Normal	Increased	Normal

frequent association of tenosynovitis; the absence of subcutaneous nodules; the good general health of the patient except for the fever and signs accompanying his present acute joint infection; usually, marked pain on motion of the affected joints and extreme tenderness of these joints; and the *prompt response of the infection to adequate doses of sulfonamides.* Gonorrheal arthritis is more common in males (3:1), while rheumatoid arthritis shows a marked predominance in females. One must remember that gonorrheal arthritis is usually a polyarthritis at the onset; further, the gonococcic complement fixation test may remain negative for the first four weeks of a gonococcic infection, so that a negative test certainly does not rule out this disease.

Diagnostic Pointers in Gonorrheal Arthritis

History of gonorrhea (urethritis or involvement of the cervix, conjunctiva or rectum).
Onset of joint symptoms *usually* within ten to twenty days after primary gonorrheal infection.
Predominance in males (3:1).
Usually a *polyarthritis at the onset.*
Involvement of *knees, ankles* and *wrists* in order of frequency.
Frequent occurrence of tenosynovitis about the wrists and ankles.
Occasionally associated with conjunctivitis and iridocyclitis.
Gonococcus complement fixation test of blood positive in over 80 per cent of cases (if properly performed); may remain negative for the first four weeks of the disease.
Isolation of gonococci from joint fluid (by special culture methods).

Gout.—Gout is occasionally confused with rheumatoid arthritis or gonorrheal arthritis. A few pointers will usually suffice in making the correct diagnosis.

1. *Gout occurs usually in males* (98 per cent of the cases).
2. Gout is *uncommon under* the age of *thirty-five.*
3. In typical cases, the attacks of gout appear suddenly in a previously healthy individual, persist for a few days or weeks, and then disappear, leaving the patient without any joint symptoms during the periods between attacks.
4. The **complete freedom from joint symptoms between attacks of gout is characteristic of this disease.** Attacks may occur every year or two at first, and later increase in frequency, so that they may finally come on every week, month or more often.
5. The *dramatic response* of the patient *to* adequate doses of *colchicine* is a diagnostic and therapeutic feature of gout. The pain of gout may be excruciating, but is usually relieved within twelve to twenty-four hours by adequate amounts of colchicine. If the patient does not respond to such therapy, suspect that the diagnosis is incorrect (unless the dose has been inadequate or the medication has not been retained).

6. The first attack of gout is usually *monarticular,* frequently with involvement of a great toe.
7. *Involvement of* the *great toe* occurs in 70 per cent of cases in the initial attack and sooner or later in about 90 per cent of patients.
8. *Tophi* may be seen in 50 per cent of cases.
9. The *blood uric acid* is usually elevated during attacks of the disease.

Main Symptoms and Signs of Gout

Tophi (present in only about half the cases at any given time).
Elevation of the *blood uric acid* at some time in about 75 per cent of patients.
Acute *recurrent attacks with complete remissions* (before the development of secondary changes in joints).
Predominance in males (98 per cent of cases).
Involvement of the big toe in the initial attack in 70 per cent of cases, and sooner or later in 90 per cent.
Roentgen-rays of *little diagnostic value early* in the disease; bony erosions seen in only about one-third of the cases even in the later stages.

Suspect Gout if:[2]

Acute joint symptoms follow an *operative procedure.*
There is a *family history* of gout.
There is a history of *olecranon bursitis* or Achilles tendinitis.
Acute joint symptoms follow slight trauma, or excessive holiday food or drink.
Sudden joint symptoms appear during early morning hours.
Acute arthritis occurs in men over the age of forty, in the absence of acute trauma or a history of gonorrhea.
Acute joint symptoms develop within minutes or a few hours.
Joint symptoms are *associated with a chronic nephritis or renal colic.*
Joint symptoms occur *in* patients with *polycythemia, leukemia* or after certain therapeutic procedures, such as injection of liver extract, salyrgan, insulin, ergotamine tartrate (gynergen), thiamin chloride, decholin (dehydrocholic acid) or the use of a ketogenic diet.
Joint symptoms occur after hunting and fishing expeditions.
Joint symptoms recur in the spring or fall.
Joint pain is exceptionally severe.
Acute *involvement of the great toe occurs.*
The patient appears wearing a shoe cut out over the metatarsophalangeal joint of the great toe.
Complete recovery of an acute arthritis occurs within several weeks.
Symptoms are *relieved* within twenty-four to seventy-two hours by adequate doses of *colchicine.*
The patient is absolutely *free from joint symptoms between attacks.*
Periarticular fistulas or ulcers are present.
Joint symptoms develop or are made worse following the ingestion of a high fat and low carbohydrate and protein diet for several days (P.F.C. 50–300–30).
The symptoms in a monarticular arthritis are entirely disproportionate to the findings.
The general reaction, especially the fever in any polyarthritis, is less than the findings might indicate.

	Rheumatoid Arthritis	Gout
Sex	2 or 3 females to each male	98 per cent of cases are males
Onset	Usually insidious	Attacks usually appear suddenly
Age of onset	Usually between 20 to 45 years	Usually after 35 years
Frequency of attacks	Onset usually insidious; recurrent attacks occasionally early in the disease	Varying from several months to several years
Freedom from symptoms	Symptoms usually continue for months or years without marked intermissions	Usually complete freedom from symptoms between attacks
Relief of symptoms by full dose of colchicine	None	Usually prompt
Blood uric acid	Usually normal	Usually elevated during attacks
Tophi	None	Present in 50 per cent of cases
Involvement of big toe	Not characteristic	Occurs in 70 per cent in initial attack and sooner or later in 90 per cent
Generalized osteoporosis by *x*-ray	Usually seen	Not a feature
Symmetrical joint swellings	Usually present	Not usual
Fusiform joint swelling	Usually present	Joint swelling usually asymmetrical and irregular
Sudden development of joint symptoms in minutes or hours	Does not occur this suddenly	Common
Associated olecranon bursitis	Not common	Common
Subcutaneous nodules	In 20 per cent	Not found
Prodromes	Often marked (fatigue, paresthesias, cold, clammy hands)	Usually none
Type of patient	Usually thin, asthenic	Often overweight, sthenic
Muscular atrophy	Usually present and may be marked	Usually none except terminal stages
Ankylosis	May occur	Rare
Agglutination test with hemolytic streptococci	Often positive after some months of illness	Negative
Family history of gout	Usually negative	Often positive
No. of joints involved	Usually multiple	First attack is usually monarticular
Pain	Usually mild, relieved by rest	Often excruciating; relieved by adequate doses of colchicine

10. Generalized osteoporosis, seen by *x*-rays in rheumatoid arthritis, is not a feature of gout.
11. As differentiated from rheumatoid arthritis, *joint involvement* is usually *not symmetrical* in gout.
12. Fusiform swellings of joints do not usually appear in gout, but the joint swelling is irregular and asymmetrical.
13. The subcutaneous nodules often seen with rheumatoid arthritis are not found in gout, although olecranon bursitis is commonly associated with the latter disease.
14. The patient is usually overweight and of a sthenic habitus, in contradistinction to the usual patient with rheumatoid arthritis who is weak, tired, underweight, anemic and asthenic.
15. Ankylosis is very rare in gout, while this does occur in the later stages of rheumatoid arthritis.
16. In gout, joint swelling, redness and pain may develop very suddenly, in a period of minutes or hours, while the typical joint changes in rheumatoid arthritis consume weeks and months.
17. Muscular atrophy, which is so common in rheumatoid arthritis, is not a feature of gout.
18. Most patients with gout give a family history of the disease.
19. The pain in gouty patients is often excruciating and much greater than the discomfort seen in most individuals with rheumatoid arthritis.

Tuberculous Arthritis.—Tuberculous arthritis may at times be mistaken for rheumatoid arthritis. In most instances, however, tuberculous joint involvement is characterized by the *slow, insidious development of a monarthritis*, although several joints may be involved occasionally; involvement of four or more joints is a point against the diagnosis of tuberculous arthritis. Tuberculosis of the spine is the most common form in children, occurring most often between the ages of two and ten. In adults, tuberculosis of the spine seems also to be the most common form, followed by involvement of the hip. The most commonly involved vertebræ are those between the sixth dorsal and fifth lumbar.

In about half or more of the cases, there is a *concomitant pulmonary tuberculosis*. In adults, tuberculous joint involvement is most common in the second and third decades. In those with spinal involvement, at least half of the cases develop a paravertebral abscess, which may be at times the only roentgen evidence of the underlying bone disease.

A *family history of tuberculosis*, or a previous personal history of pleurisy with effusion, suppurative cervical adenitis or fistula *in ano* may be of assistance in arriving at the correct diagnosis.

Additional aids in diagnosis of tuberculous arthritis include a biopsy of joint tissue; guinea pig inoculation of synovial fluid; microscopic examination or culture of joint fluid for tubercle bacilli; and a positive tuberculin test. The presence of considerable fluid in a joint, or the involvement of numerous (more than three) joints is evidence against the diagnosis of tuberculosis.

Symptoms of tuberculous arthritis are not characteristic, except that the disorder usually develops insidiously. Symptoms may include local pain, heat and swelling of a joint, the doughy swelling being due to enlarged synovia rather than to a great amount of fluid; muscular spasm and limitation of motion; muscular atrophy (which may appear early and may be marked); loss of weight; low grade fever and, in some cases, a normal temperature; and local tenderness, which is usually not an early symptom.

When the knee joint is involved, pain (insidious in onset) is usually the first symptom, although stiffness may appear as the initial symptom in 10 per cent. Local heat and gross limitation of motion are usually present. In these cases, bacterial examination of the pus (including guinea pig inoculation) from an abscess is a valuable laboratory aid.

If the hip joint is involved, the primary lesions are usually in the head or neck of the femur. Symptoms include its occurrence most often in children, limp, pain in the knee and thigh (from pressure upon the obturator and anterior crural nerves); muscle spasm; fullness in the groin; tenderness on pressure over the hip; slight flexion and abduction of the thigh; flattening of the buttock and disappearance of the gluteal fold on the affected side; limitation of all motions at the hip, especially of rotation and abduction; and at a later stage, adduction deformity of the thigh with the formation of cold abscess or sinuses in some cases, most often pointing on the outer side of the thigh. The synovial tissue is invaded early, followed by marked rarefaction of the bone and obliteration of landmarks; the femoral head becomes misshapen or may be entirely absorbed.

Tuberculosis of the spine is the most common form of tuberculous arthritis. The lesions occur most commonly in the bodies of the vertebræ, and in more than half of the cases the thoracic vertebræ are affected. The anterior border of the vertebral body is most commonly involved; collapse of a number of the vertebræ results in angulation of the spine with shortening of the trunk. Deformities are produced by caseous softening of the bodies of the vertebræ with the added pressure of weight bearing. The symptoms include abnormalities of gait, with awkwardness in walking; rigidity and upright position of the spine, even when bending; pain and weakness in the back, and pains and crying at night; reference of pain at times to the abdomen (along the intercostal nerves); kyphosis; tenderness and muscular spasm over the affected areas and occasionally paralysis of the legs from pressure upon the cord by granulation tissue or cold abscess. *Cold abscess formation and paralysis* are the two most common complications. A dorsal abscess may remain localized, or may travel along the anterior aspect of the spine forming a psoas abscess; in some cases, these may point in the thigh.

The roentgen findings in tuberculous arthritis are not characteristic. Active early tuberculosis may be present in bones and joints with negative roentgen findings. Soft tissue swellings and increased radiability of the bones may be noted early.

Later, one notes marginal erosion with marked irregularities of contour along the joint surfaces, evidence of subchondral invasion with apparent separation of the cartilage and narrowing of the joint space. Later, a cold abscess or bursal involvement may be noted, with marked deossification of regional bones and articular cortices and a reduced joint space which may be obliterated; subluxations are common.

Tuberculous Arthritis	vs.	Rheumatoid Arthritis
1. Usually monarticular		1. Polyarticular
2. Injection of joint fluid into guinea pig often reveals tuberculous nature		2. Injection is negative
3. Joints most often involved: spine, knee, hip, elbow, ankle		3. Any joint may be involved, but particularly the proximal interphalangeal joints
4. Associated visceral tuberculous lesion in 50 per cent of cases		4. Usually no visceral tuberculosis
5. Most common in first 14 years of life		5. Most common in middle age
6. Biopsy of synovia reveals tuberculous lesion		6. Biopsy reveals no tuberculosis
7. Roentgen findings usually progress more slowly		7. Roentgen findings often more rapidly progressive
8. Joint involvement not symmetrical		8. Often symmetrical
9. General health usually good		9. Usually thin, underweight
10. Slow insidious development		10. May be as slow, but usually porgresses more rapidly
11. History of preceding trauma in 50 per cent		11. Usually none

Fibrositis.—The patient with primary periarticular fibrositis will usually not show the marked loss of weight, anemia, fever, marked muscular atrophy, rapid sedimentation rate, roentgen joint changes and synovial exudate which are features of rheumatoid arthritis. In fibrositis, prolonged rest is usually followed by an aggravation of the symptoms with marked stiffness and discomfort, requiring a period of minutes or more for the patient to limber up before he again feels better. The patient with rheumatoid arthritis, on the other hand, is usually improved following periods of rest, although some of these individuals may show the same tendency to stiffness around joints after a period of prolonged rest in one position. There are practically no physical findings in fibrositis except the occasional appearance of tender or painless fibrous nodules of varying size in the affected areas; the finding and even the importance of such nodules has been questioned by many people in this country, and even in England, which seems to be the homeland of fibrositis. The main features of fibrositis appear to be pain, stiffness, aching and limitation of motion with little or no effect on the general health; rheumatoid arthritis, on the other hand, appears to be a systemic disease with definite constitutional symptoms. The practically normal sedimentation rate and negative roent-

gen findings in fibrositis will usually clinch the diagnosis if laboratory tests are required.

Differential Diagnosis of Fibrositis From Rheumatoid Arthritis

	Fibrositis	Rheumatoid Arthritis
Joint pain	Pain is periarticular; usually worse on awakening; improved following gentle exercise; made worse by rest	Definite joint pain; often increased following exercise; improved by rest
Joint swelling	No synovial exudate	Synovial exudate frequent
Joint tenderness	Absent or very mild	May be marked
Stiffness of joints	Rarely objective; usually subjective only	Commonly subjective and objective
Muscular atrophy	Absent	Common
Remissions	Frequent	Uncommon
Constitutional phenomena	Usually absent; usually no loss of weight	Fever, tachycardia, *loss of weight*, etc.
Sedimentation rate	Normal; occasionally elevated	*Markedly elevated* in most cases
Anemia	Rare	Common
Roentgen-rays of joints	*No bony changes*	Definite intra-articular findings
Prognosis	Good, from the standpoint of the joints; recurrences are frequent	Very uncertain; usually poor
Response to therapy	Very variable; occasionally prompt	Slow

Periarticular fibrositis is often confused with rheumatoid arthritis. It is important to attempt to separate the two because of the difference in treatment and in prognosis. The subjective symptoms of stiffness, aching and limbering up are much the same in primary fibrositis as in the secondary fibrositis which accompanies rheumatoid arthritis (as periarticular, intramuscular, tendinous, etc.). However, in true rheumatoid arthritis, the pain produced by intra-articular disease gradually increases in the damaged joints; on the other hand, real joint pain is absent in fibrositis.

Focal Infection.—One encounters a number of cases resembling rheumatoid arthritis in which the joint involvement seems to be associated with definite focal infection (as dental abscess, sinusitis, peritonsillar abscess, etc.). In this condition, only a single or a few joints are involved, as a rule, and a marked improvement in the arthritis follows proper attention to the focus of infection. This subject of so-called focal infection arthritis is still controversial but it should be remembered that when an arthritic process follows shortly after or in association with a focal infection, proper attention to the focus may result in marked lessening of the symptoms and a complete clearing of the process; unfortunately, one does not often see this picture.[3]

Diagnosis of Syphilitic Joint Disease.—Syphilitic arthritis is uncommon. The Wassermann reaction is positive in most cases and there is a definite response to antisyphilitic therapy. This diagnosis is made by a careful history (including a personal or family history of syphilis, repeated miscarriages, interstitial keratitis, iritis, lymphadenopathy, chancre, etc.), a complete physical examination, blood and spinal fluid Wassermann and

the clinical manifestations. *The Wassermann test may be negative in known syphilitic bone and joint lesions;* on the other hand, the finding of a positive Wassermann reaction does not necessarily incriminate syphilis as the cause of the joint discomfort. Too much credence must not be placed upon a negative Wassermann reaction if bone or joint syphilis is suspected; in all these cases, one should *x*-ray the tibia and fibula for additional information.

No pathognomonic roentgen signs of syphilitic arthritis have been reported; suggestive features include erosion of bone and cartilage with bony proliferation and periostitis in a relatively symptomless joint.

Clinical details favoring the diagnosis of syphilitic joint disease include absence of pain, even with marked hydrops, free range of motion, good general health despite a long story of joint pathology, precipitation of the arthritis by a blow (occurring often in syphilitics), other evidence of syphilis, lack of response to salicylates and favorable response to antisyphilitic therapy.

Suspect Syphilis:

1. The occurrence of *joint disease without* heat, *pain* or tenderness must always make one consider neuroarthropathy.[1]
2. *Rigid pupils* and *absent knee jerks* are more important than blood or spinal fluid Wassermann tests in the diagnosis of tabetic arthropathy.
3. Syphilis should always be suspected in all cases of *bilateral painless hydrops of the knees* in children.[4]
4. Suspect syphilis in any case of so-called "rheumatic fever" which does not respond promptly to adequate doses of salicylates.
5. Suspect syphilis and tuberculosis in a monarthritis or a deforming arthritis at any age.
6. Syphilis may cause arthritis; symmetrical synovitis; spondylitis; hydrarthrosis; tenosynovitis; bursitis; periostitis; juxta-articular nodes and gummata; Charcot joints and osteochondritis.

Traumatic Arthritis.—Traumatic arthritis is not often confused with rheumatoid arthritis. The *history of injury, monarticular joint involvement* (in most cases) and *absence of systemic manifestations* (*i. e.*, normal T.P.R., normal sedimentation rate and good general health) are of importance in the diagnosis of traumatic arthritis. Bilateral or polyarticular joint involvement is uncommon. In most instances, the traumatic arthritis begins within a week following the injury, although symptoms may not be noted for periods varying from several weeks to six months or more. *Years of pain and limitation of motion may follow a single strain or contusion* even in the absence of evident anatomic changes; severe single injury or repeated mild trauma may result in the picture of degenerative joint disease. One must remember, also, that injury may aggravate an existing arthritis of any type.

Joint effusion may or may not be present, and roentgen examination may be negative in the early stages. Synovial fluid usually contains large numbers of erythrocytes in the early stages and an increased amount of

bilirubin resulting from the breakdown of erythrocytes; the icterus index of synovial fluid in traumatic arthritis is usually above 5, while it is almost invariably below this figure in inflammatory effusions. The bilirubin content of synovial fluid may also be elevated occasionally in hemophilia, sarcoma, xanthoma or tabetic arthropathy.

An acute traumatic arthritis, synovitis or periarthritis is usually recognized without difficulty; however, injury may reactivate a previous form of arthritis, or an acute arthritis may develop on a basis independent of the injury.

Characteristics of Traumatic Arthritis

Joint phenomena are usually monarticular; bilateral or polyarticular involvement is uncommon.

Symptoms may result from reactions in synovial membrane, capsule or articular tissues.

Symptoms include: pain, swelling, crepitus on motion and possibly muscular spasm and limitation of motion.

Skin temperature is usually normal.

Joint effusion may or may not be present.

In most instances, a traumatic arthritis begins within a week following injury although symptoms may not be noted for periods varying from several weeks to six months or more.

Roentgen examination may be negative in the early stages and may reveal osteophytes or hypertrophic changes later. Traumatic lesions (such as tears in the cartilage, hypertrophied synovial membrane, narrowed or obliterated suprapatellar or infrapatellar spaces, etc.) may be revealed by roentgenogram following the intra-articular injection of air, carbon dioxide or contrast medium.

The icterus index of synovial fluid in traumatic arthritis is usually above 5; this is almost invariably under 5 in inflammatory effusions. The bilirubin content of synovial fluid is raised only rarely (except from trauma); uncommon causes include hemophilia, sarcoma, xanthoma and tabetic arthropathy.

Acute traumatic arthritis, synovitis or periarthritis are usually recognized without difficulty. However, injury may reactivate a previous form of arthritis, or an acute arthritis may develop on a basis independent of the injury.

Differentiation of a sprain (tearing of a ligament) from traumatic arthritis or synovitis may be difficult; in synovitis, however, considerable fluid is usually observed in the joint. Years of pain and limitation of motion may follow a single strain or contusion even in the absence of anatomic changes. Severe single injury or repeated mild trauma produces the joint picture of a degenerative joint disease. Injury may also aggravate an existing arthritis.

It has been estimated that 75 per cent of industrial workers over the age of forty suffer from some type of joint affection. Traumatic arthritis may result from severe blows or falls, from trauma incident to poor posture or from athletic injuries or occupation. In any form of joint disease, the possibility of a sudden accident or repeated minor trauma must be searched for in the history. Such slight injury as repeated pressing of a knee against

a heavy door may result in pain in that joint. Arthritis may also develop following the introduction of a foreign body (needle, thorn or bullet) into a joint. A detailed discussion of this subject is given in a later chapter.

Lymphogranuloma Venereum.—Lymphogranuloma venereum occurs more commonly in negroes. The Frei test is specific for this disease. The joint manifestations are usually milder than rheumatoid arthritis and respond more readily to rest; the process responds to sulfonamide therapy.

The arthritis with lymphogranuloma venereum is usually a chronic process, with acute flare-ups and a tendency to relapse. Polyarticular involvement is the rule. Migratory joint involvement or generalized arthralgia may occur. The joint involvement may persist for weeks or months without destruction of bone or cartilage. The swelling is usually confined to the periarticular tissues.

Other evidence of lymphogranuloma venereum which may be found includes the presence of *rectal strictures* in a large percentage and the *positive Frei test.* The sedimentation rate is usually markedly elevated. The disappearance of joint symptoms following adequate sulfonamide therapy is in contradistinction to the negative results obtained in rheumatoid arthritis with such treatment.

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CHAPTER XII

RHEUMATOID ARTHRITIS: RÉSUMÉ OF TREATMENT

UNFORTUNATELY, many patients with various forms of joint disease expect their physicians to administer some magic pill or parenteral medication which will ameliorate their symptoms or "cure" their arthritis within a few days or weeks As yet, this magic bullet has not been obtained for the arthritic patient.

FIG. 33.—Early *rheumatoid arthritis* showing very well the fusiform soft tissue swelling at the proximal interphalangeal joint of the middle finger, as well as narrowing of this joint space.

General Pointers for the Busy Practitioner

1. There are many etiologic factors in joint disease; the physician must *first obtain a correct diagnosis* of the type of arthritis his patient presents.
2. The *patient* should be well *informed as to the type of his arthritis* by means of illustrations, simple drawings and in terms which he can clearly understand. If the individual learns to know more about his arthritis, he will be better able to help himself and his physician.
3. The prognosis should be gone over in detail with the patient and his family, employing again simple non-medical terms, when possible, and not couching it in vague medical phrases. In most cases, it will be possible to give at least a fairly good prognosis and in certain types of joint disease, the prognosis is excellent.
4. The arthritic patient needs a great deal of encouragement; the patient with rheumatoid arthritis, for example, must understand that he will not be "cured" in a few weeks, but that his treatment will occupy a period of months or years, and that the ultimate prognosis is good in at least 75 per cent of the cases, with proper coöperation and care.
5. The physician should not promise miracles; these will not usually be borne out. If he is confronted with an "arthritic derelict" of long standing, with multiple flexion contractures and deformities, marked muscular atrophy and general debility, the family must understand that, although it may be possible to improve the patient by prolonged physical therapy, occupational therapy and orthopedic measures (including surgery in some cases)—plus an adequate diet and good, sensible general care—such a patient will not witness disappearance of his contractures, deformities and muscular atrophy simply by giving a few pills by mouth or a few injections. Above all, one should never make any misleading statements to the patient or the family. The patient and the family will soon lose confidence in the physician who presents a glowing account of quick "cure" of rheumatoid arthritis by various injections or medications, etc.—without a carefully prepared long-range view of therapy for the particular patient.
6. The physician must always bear in mind that although we may employ a "general routine" for the care of various forms of arthritis, "*the patient must be treated*, and not only his disease." The regimen of treatment must be varied, depending upon many factors exhibited by the individual—the state of his illness—his physical condition—his tolerance to medication and physical therapy—his mental makeup—or any new situations which may and do arise.
7. *If a given program of therapy does not produce the desired effects in a reasonable period of time*—or if untoward results are obtained—**the physician must not hesitate to alter this from time to time**; however, once a program has been worked out for the individual patient, it should be given a fair trial and not be changed from week to week without just cause.

Several forms of joint disorders are welcomed by the general practitioner because of the marked improvement shown by the patient within a relatively short time, under proper therapy. Examples of these include:

1. The prompt improvement in the patient with *gonorrheal arthritis* following the use of sulfonamides or penicillin in adequate dosage (in addition to proper supportive therapy).

2 Amelioration of symptoms in the patient with acute exacerbations of *gout* by adequate colchicine medication. It is truly dramatic to see the most favorable change in these individuals within eight to twelve hours following the use of colchicine—with relief of 90 or more per cent of the pain and discomfort.
3. The prompt relief of the discomfort in a *sprained ankle* following *procaine injection.*

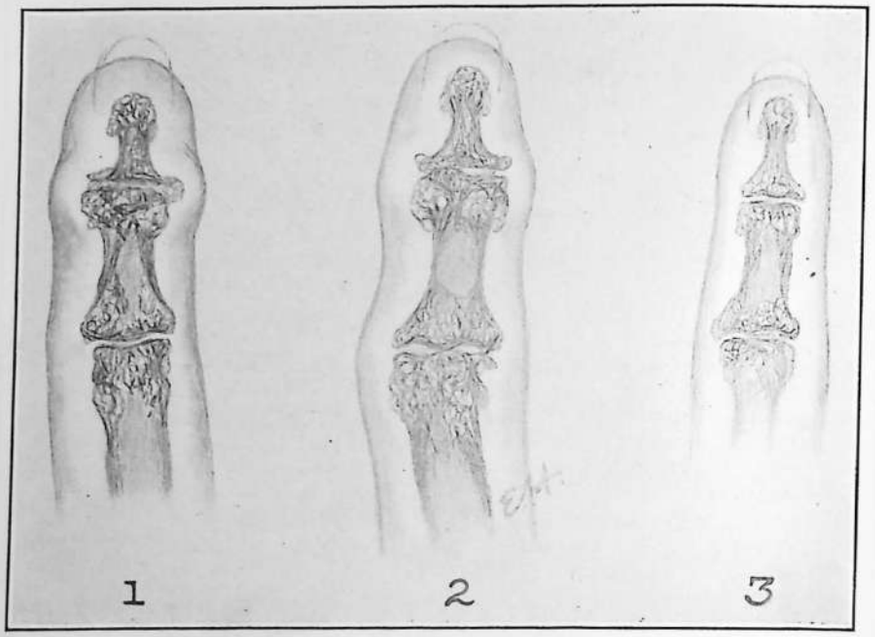

FIG. 34.—Drawing made from *x*-rays of several fingers with *degenerative joint disease.* Note the swellings at the terminal interphalangeal joints (Heberden's nodes). In the fingers, degenerative joint disease involves especially the terminal interphalangeal joints; however, the proximal interphalangeal joints are involved in the process in some cases.

4. The relief within a day or two in the majority of patients with acute episodes of *subdeltoid bursitis,* following adequate *roentgen therapy.*
5. The relief of pain in patients with *rheumatoid arthritis of the spine* (in early stages) by adequate roentgen therapy.
6. The value of proper application of heat and massage and procaine injections in *fibrositis;* this latter diagnosis should always make one suspect some underlying disorder, especially diabetes mellitus, hypothyroidism and gout.
7. The dramatic effects of salicylates upon the pain and fever associated with an acute attack of *rheumatic fever.*
8. The excellent results obtained with sulfonamides or penicillin in certain *specific forms of infectious arthritis: pneumococcal, meningococcal, gonococcal.*

9. The relief of prolonged severe low back pain and sciatica, by removal of a *protruded intervertebral disk* in patients with typical signs of this disorder.

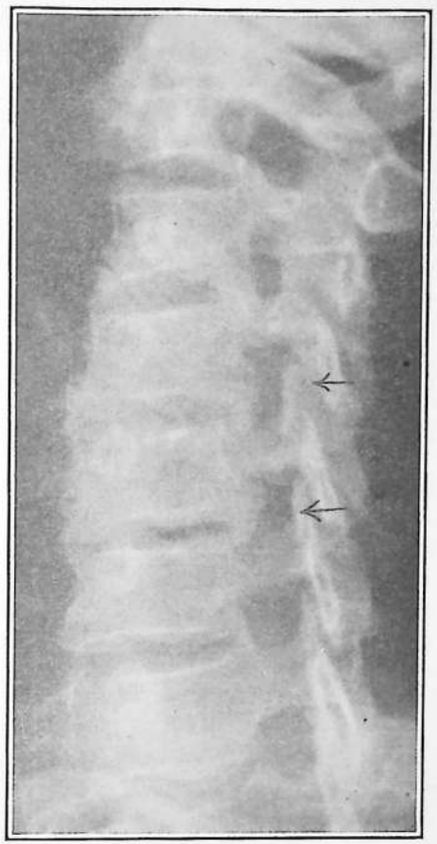

FIG. 35.—*Degenerative joint disease* in cervical spine. Man, aged fifty-nine, who has had muscle and joint pains in the neck and shoulders for fifteen years. Hypertrophic changes may be seen involving the cervical spine. These changes are particularly manifest in the region of the third, fourth and fifth cervical vertebræ. The intervertebral disk is narrow, and possibly as a result of this, the usual wear and tear on these vertebræ is enhanced. The hypertrophic changes occur anteriorly on the bodies but the more significant changes are in the region of the intervertebral foramina on either side, with distortion of these (see arrows) and probably pressure on nerves, causing pain. These changes are of long standing. The changes in the region of the intervertebral foramina may be missed unless oblique views are taken.

10. The relief of low back discomfort, sciatic pain and muscular tiredness in the legs by the use of proper shoes—which fit the foot of the individual—together with elevation of the heel when a short leg is detected, and heel wedges and other simple forms of alterations of the shoes when indicated. The use of proper shoes with *correct* wedges, heel lifts and other modifications is usually neglected by the average busy practitioner because he either feels that he does not have the

time to spend with the patient, or that this is below his professional dignity—or occasionally, he does not know how to determine the type of support the particular type of foot should have.

Unfortunately, the two most common forms of joint disease (rheumatoid arthritis and degenerative joint disease) do not fall into the category of joint disorders usually welcomed by the general practitioner—inasmuch as these two disorders require prolonged therapy with results measured in months or years—rather than in days or weeks.

The greatest arthritic problem confronting the busy general practitioner is the patient with *rheumatoid arthritis*. Unfortunately, because of the insidious development of this disease over a period of weeks, months or years in some cases, the patient may have his disease for a long period of time before it is recognized. Confronted with a patient with rheumatoid arthritis, the busy practitioner should bear in mind several important points:

1. **Do not promise the patient a cure!** It is only fair to explain to the patient that once the hyaline cartilage over the ends of the bones has been destroyed, it does not regenerate, although there may be some attempt at repair by connective tissue or fibrocartilage formation.

2. The patient should be told that there are 7 chances in 10 that he or she will obtain marked improvement, and an additional 2 chances in 10 that some improvement will follow; the family should be warned, though not pessimistically, that in about 5 to 10 per cent of cases, the arthritis continues or progresses despite all attempts to stop this. However, even in these cases, should the disease go on to bony ankylosis, one is able to splint the parts in positions of election so that the arms and legs will be in favorable positions for use when the disease process burns itself out.

3. The physician is justified in *manifesting a degree of optimism* and confidence to the patient concerning the outcome of his particular illness. However, such optimism must be genuine and not simply overenthusiasm with a promise of rapid cure.

4. The physician must stress the point that with our present knowledge, the *treatment may* in all probability *require several years*, and not weeks or months—although it is true that in exceptional cases extraordinary results may occur in a few weeks or one or two months; however, this is not the rule. If the patient realizes this at the onset of treatment, he will not experience the keen disappointment which might otherwise be noted following several months of therapy.

5. The patient with rheumatoid arthritis is a sick individual. He has a generalized disease, not only a local process of his joints.

6. Although one may outline a general or working plan of therapy for the average case of rheumatoid arthritis, this must necessarily be modified varying with the conditions which are met in that particular patient. One is justified, then, in offering the busy general practitioner, a general plan of therapy—or outline—which may serve until he has had time to digest and adequately prepare for the patient those modifications which may be necessary. If private, state or federal institutions were available

for the proper care of the patient with rheumatoid arthritis, proper treatment could be carried out in much the same manner as has been done for the tuberculous patient. The great need in this country—from the arthritic viewpoint—is large, well-managed institutions where adequate arthritic therapy could be carried out on a long range program—where the individuals could be kept for periods up to several years, or more, if neces-

FIG. 36.—*Rheumatoid arthritis* of thirteen years duration in a man of 61 years. The phalanges of the second, third, fourth and fifth fingers are dislocated at the metacarpophalangeal articulation. There is considerable damage in the wrist and at the proximal interphalangeal joints of the second, third, fourth and fifth fingers, with narrowing of joint spaces, erosion of bone and ankylosis.

sary. Until such a time has come, the busy practitioner must avail himself of every opportunity which is afforded to him for the management of these patients.

The practicing physician is often confused by the availability of a multitude of measures which may be employed for the patient with rheumatoid arthritis. *There is*, as yet, *no one remedy or specific therapeutic agent or no*

combination of remedies which is always suitable or effective for these patients; inasmuch as the cause of rheumatoid arthritis is unknown, specific therapy directed against an etiologic agent is impossible. It is usually necessary to select that treatment which applies to the particular patient in question,

FIG. 37.—Shows hand of patient at age of sixteen. At that time it was thought that the multiple joint changes and soft tissue swelling were due to rheumatoid arthritis. The proximal interphalangeal joints of the second and third fingers appeared narrowed and there was marked soft tissue swelling about the latter joint. Some soft tissue swelling was present at the wrist, but the carpal bones were unaffected. (See Fig. 38.)

employing measures which will increase his resistance and improve his general health, and in addition handle the inflamed joints properly so as to preserve function and aid healing.

When the condition is first diagnosed, the patient usually wants to know the exact cause of rheumatoid arthritis and how to get rid of it quickly.

Many of these patients are puzzled to learn that the exact cause is unknown; these individuals have learned from friends that it is only necessary to remove a tooth, or tonsils or to have a prostate massaged in an effort to provide relief. The patient will usually have a multitude of questions, which must be answered honestly, but firmly. After a few visits, he will inform the physician concerning the "cure" which he has heard of through his friends by means of dusting sulfur in the shoes, or of taking increasing amounts of lemon juice daily for twenty-four days, or of bee

FIG. 38.—Shows same patient three and a half years later. There has been a considerable progression of the lesions with numerous punched out areas in the proximal, middle and distal phalangeal bones (of both hands). Fusiform soft tissue swelling can be seen around several of the interphalangeal joints with some destruction of the joint space. These changes are typical of *gout*, although gout is not common at this age. The patient had even more marked changes in the feet. Blood uric acid was markedly elevated. His symptoms had begun with swelling and pain in the left great toe at the age of ten. At present, tophi are present in the ears and toes.

stings, or of manipulations, or of a series of 21 baths at a spa, or of a trip to Florida for a month or many other therapeutic schemes. The physician must realize that these individuals are sick, are deathly afraid of the possible crippling invalidism which may occur and are only too willing to grasp at any straw which may promise them relief. Arthritic patients

must be told that many of the measures which will be used during their treatment may appear undramatic and that their program of therapy may be long and may appear tedious, but that such a program will, in the long run, be productive of the best all around results.

Measures of Proved Value in Rheumatoid Arthritis

1. *Rest* of the body: mentally and physically.
2. Rest of the involved joints, with *splinting* of the joints to prevent deformity.
3. *Physical therapy* (heat, massage and special exercises) and *occupational therapy*.
4. A sensible *nutritious diet* containing adequate calories, vitamins and minerals and anticonstipation factors.
5. *Relief of pain* by: splinting of affected joints, rest, physical therapy, analgesics when required (especially *aspirin* and often phenacetin), and sedatives and hypnotics if needed.
6. *Avoidance of cold, dampness* and *drafts*.
7. *Psychotherapy*, and proper attention to maladjusted human relationships leading to emotional reactions, especially anxiety and resentment.
8. *Orthopedic measures* including supports, good posture, elastic bandages, braces when indicated, proper shoes, corsets, plaster casts, manipulation of joints and orthopedic surgery, if required (but only under expert supervision).
9. *Symptomatic therapy*, as indicated to combat constipation, anemia, insomnia, extreme fatigue and tiredness, undernutrition and lack of appetite, menopausal symptoms if present, etc. One must never forget that he is treating a human being, and not a disease.
10. *Periodic vacations*, when possible, possibly including a stay at a well-equipped hospital or spa.

The physician must stress to the patient that *no pill or capsule, as yet known, and no combination of oral medications will cure rheumatoid arthritis;* the patient (and also the physician) must understand that no series of injections, by itself, will cure rheumatoid arthritis. The physician and the patient must likewise know that long-standing deformities, contractures and ankylosed joints will not change as a result of oral or parenteral medication. The physician, by a thorough knowledge of the present concept and methods of therapy of this disease, should impress upon the patient that he will use that plan of therapy in each individual which is most likely to succeed; he must be honest, and truthfully optimistic, so that his patient will develop confidence in his therapy. However, he must not hesitate to change or alter his plan of therapy if necessary depending upon changes which may occur in the individual patient. The physician and patient must understand that individuals will differ in their response to treatment and that those measures which may prove useful in one individual may be of little or no value in another.

The physician must always bear in mind the fact that *rheumatoid arthritis usually proceeds through a series of exacerbations and remissions which may be mild or severe.* Exacerbations may accompany marked

changes in weather, acute respiratory infections, periods of overactivity, menses, etc., while partial or almost complete remissions may occur in pregnancy or in obstructive jaundice. The tendency to spontaneous remissions in rheumatoid arthritis must always be considered in evaluating the relief or effect produced or attributed to any special drug or regimen of therapy. However, if left alone, most patients with rheumatoid arthritis tend to become progressively worse, except for the temporary remissions usually occurring during obstructive jaundice or pregnancy. No one has yet been able to determine the effective mechanism during jaundice or pregnancy which is operative in resulting in this improvement;

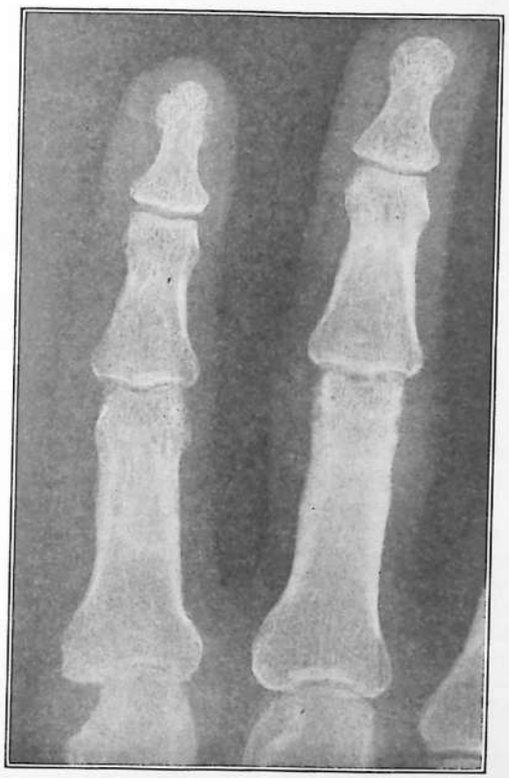

FIG. 39.—Early *rheumatoid arthritis.* Middle finger is at right. Note soft tissue swelling (fusiform) at proximal interphalangeal joint and narrowing of this joint space, with several small punched out areas of bone here, so often seen in rheumatoid arthritis.

studies of cholesterol, bilirubin, blood lipids, bile acids and sex hormones have as yet been fruitless in attempting to utilize the potent physiologic common denominator occurring in these conditions and in applying it to the service of the rheumatoid arthritic. However, *the mere fact that either* **obstructive jaundice** *or* **pregnancy** *may produce at least a marked temporary improvement in these patients* is often of great psychologic benefit to these patients in an effort to explain to them that the disease is not necessarily

relentless, uncontrollable and devastating and in buoying the hopes of physicians who treat these patients that someone may, in the near future, find the much sought for therapeutic agent in this disease. The recent work of Selye and his associates in producing joint disturbances in rats by injections of desoxycorticosterone acetate and exposure to cold offers another avenue for the experimental therapeutic approach to this problem, and again opens the door to the experimental use of various endocrine and other preparations in these animals. One must attempt to dispel the pessimistic viewpoint held by some practitioners that nothing can be done for rheumatoid arthritis.

Measures of Probable Value in Rheumatoid Arthritis

1. Blood *transfusions.*
2. *Removal* of definite *focal infection* (in an effort to lessen the burden which the patient is carrying).
3. Chrysotherapy: parenteral *gold therapy.*
4. *Procaine injections* around a joint in some cases to lessen pain temporarily.
5. *Roentgen therapy* (particularly in patients with rheumatoid spondylitis).
6. *Foreign protein therapy* (typhoid vaccine intravenously).
7. Residence in a *warm, dry climate* with avoidance of storms.
8. Mecholyl or histamine iontophoresis.

Measures of Questionable Value:

1. Vaccines.
2. Very large doses of concentrated vitamin preparations (especially vitamin D).
3. Prolonged fever therapy.
4. Endocrine products, such as estrogenic substance (which may decrease the arthralgias accompanying the menopause); desiccated thyroid gland (which will lessen the arthralgias associated with hypothyroidism); testosterone (which may diminish the fatigue and lassitude of the middle-aged or older man); thus far, no endocrine preparations have resulted in marked improvement of the rheumatoid arthritis, *per se.*
5. Other preparations given parenterally such as sulfur, bee venom, chaulmoogra oil and others which are probably not of specific value but may exert a foreign protein reaction in addition to the possible psychic value of the injection of any harmless substances, even physiologic saline.
6. The value of iodides and arsenicals is still open to much question.

Measures Employed by Some in Rheumatoid Arthritis but Probably Not of Value:

1. Operations such as splenectomy, parathyroidectomy and thyroidectomy.
2. Dietary fads.
3. Cobra venom.
4. High colonic irrigations and the use of intestinal antiseptics.

Inasmuch as rheumatoid arthritis is subject to spontaneous remissions and relapses and occasional patients recover fairly promptly, one should not be too dogmatic in stressing the value of any one single measure to the exclusion of others. The late Prof. Alfred Stengel once said that the most effective medication for the patient with rheumatoid arthritis is that medicine which he took just before he began to improve. We are treating a systemic disease so that the entire patient must be treated and not only his joints.

Rheumatoid arthritis is a systemic generalized disease for which as yet we have no specific, rapid cure; the joint manifestations are only one part of the trouble. In order to obtain the best results in the treatment of rheumatoid arthritis, it seems reasonable that we must treat the whole individual including both his body and his mind.

Treatment will vary with the stage of the disease process. In the very early stage of the disease, it is often possible to obtain marked improvement or arrest of the disease process. In those individuals in whom the disease, although of several years duration, is now relatively quiescent, a regimen of management can usually be adapted to the patient's occupation and general manner of living; however, if an exacerbation of the disease occurs, strict attention must be paid to rest and all of the details as outlined for treatment during the active stage of the disease.

The patient must not expect constant improvement without interruption; the response to therapy will be variable from week to week and minor setbacks will occur in most patients.

GENERAL OUTLINE OF THERAPY OF RHEUMATOID ARTHRITIS FOR THE BUSY PRACTITIONER

1. **Adequate Mental and Physical Rest.**—(*a*) Change in environment may be required: hospitalization; spa; vacation, etc.

(*b*) A period of several weeks or months of rest in bed should be suggested in all cases of active generalized rheumatoid arthritis.

(*c*) If the patient must continue his occupation, daily rest periods should be instituted.

Almost all of these patients complain of fatigue. **Fatigue is One of the Greatest Contributing or Precipitating Factors of All Disease.** The elimination of fatigue will do much to restore the mental and physical endurance of these individuals; this is very difficult to accomplish in some cases.

Unfortunately, the patient with rheumatoid arthritis often thinks he must exercise his affected joints as much as possible to prevent stiffness and deformity. Nothing can be further from the truth. *A period of several weeks or months of rest in bed should be suggested in all cases of active generalized rheumatoid arthritis.* This does not mean, however, that the patient is to remain motionless for this period, or that he may rest in bed in any position which he deems best.

The Patient With Rheumatoid Arthritis is a Sick Individual. He Has a Generalized Disease, Not Only a Local Process of His Joints. As in pulmo-

nary tuberculosis, where few physicians doubt the value of rest in bed during the active stages, a proper amount of rest in bed during the active stages of rheumatoid arthritis will usually pay large dividends. During this period, ankylosis, contractures and muscular atrophy are prevented by physical therapy and proper orthopedic care. As a general rule (except in acutely inflamed joints), each joint should be moved through as full a range of painless motion as possible at least one or two times a day to prevent the development of adhesions. Properly administered massage (of the muscles between the joints) and cautious use of active and passive movements will maintain muscular tone and result in larger and more powerful muscles.

The affected joints should be properly splinted in many instances for periods during the day and night, in an effort to prevent trauma to joints and to promote muscular relaxation. Methods of splinting and positions to be used are discussed in a separate section. Many of these patients can spare themselves years of discomfort by carrying out a program of adequate rest at the onset of their illness. The physician is often at fault for not insisting upon rest in bed; in the case of a wage earner, although such rest may deprive that individual of several months' earnings at the onset, it may save him years of idleness at a later date.

This period of rest in bed varies with the individual. In some, a stay of one or two weeks may be markedly beneficial, while in others from four to six months may be required to produce any marked benefit. When rest in bed is absolutely impossible, periods of rest upon a couch for an hour or two at noon are worth insisting upon. These patients should go to bed immediately after the evening meal. If the arthritis involves weight-bearing structures such as the knees, the patient must remain off of the affected limbs until the acute stage of the process has passed. Even in the subacute stages, freedom from weight bearing will usually produce added improvement. *The climbing of stairs is to be especially avoided by those individuals with arthritic involvement of the knees, feet or hips.*

In the average mild or early cases of rheumatoid arthritis, most workers agree upon a program of from ten to twenty hours of complete rest each day. If the patient is ambulatory and his arthritis is not severe or disabling, he should be encouraged to work if this is a non-traumatizing form of activity. If the individual is worn out and tired, he must rest for a period varying from ten to twenty hours a day, but special exercises must be prescribed along with the rest so that the patient will not become stiff. **The rest must be prescribed with the joints in favorable positions and good body mechanics must be preserved.** Exercises should not be severe enough to produce pain which persists for periods of more than one hour, and should never tire the patient.

If possible, patients with rheumatoid arthritis should be hospitalized at the onset both for diagnostic purposes but more especially so that the patient may be taught various methods and principles of therapy, including rest, exercises, massage and physical therapy, and obtain any necessary plaster splints or orthopedic apparatus that may be needed.

Rest in Bed Should be Insisted Upon in:

1. The acute, febrile stage of the disease with severely inflamed joints.
2. Patients whose arthritis is progressing rapidly, even though there may be no fever and no acute inflammation of the joints.
3. Individuals with marked deformity or inflammation of weight-bearing joints.
4. Those with marked constitutional manifestations.

Duration of Rest in Bed.—(1) This is variable, as is the case in pulmonary tuberculosis, and depends upon the response of the patient. (2) If possible this should be prolonged even after definite improvement has begun, with the individual then getting up very gradually. (3) With a subsidence of fever, tachycardia, signs of inflammation in the affected joints, lowering of the sedimentation rate and improvement in the patient's general health, he may then be allowed to sit on the edge of the bed or be carried into a chair; after a period of ten to fifteen minutes, he should be put back to bed again. (4) If there is no deleterious effect noted from this, the periods in which he is allowed in a chair are increased slowly, and as the patient responds, he is permitted to take a few steps, usually with support (such as a walker) at first.

If Prolonged Rest in Bed is Necessary:

1. Be sure that the bedclothes are not pulled tight enough to produce footdrop; a pillow placed under the covers at the foot of the bed will keep the covers lifted off of the feet.
2. A cradle may be placed over the legs if these are involved.
3. Daily good nursing care must be provided to keep the skin in the best condition, and to assist in bathing, application of heat and massage. A member of the family can often be trained to carry out these items satisfactorily.
4. **The patient must not lie in bed like a lump of putty,** *but should have a firm mattress and a non-sagging spring.* He should be taught the proper positions of rest for his extremities to avoid contracture deformities, muscle setting exercises, and when tolerated, should be taught graded mild exercises.

Weight Bearing is Inadvisable and not Permitted if:

1. Weight must be borne on acutely inflamed or hot joints.
2. There is marked muscular atrophy of the legs.
3. The knees are fixed in flexion or the legs are not in good alignment.

In patients with mild rheumatoid arthritis in whom prolonged rest in bed may not be necessary, a period of ten to twelve hours of rest in bed each night should be insisted upon, as well as from one to two hours each afternoon. If the patient is fatigued despite this program, he must spend more time in bed, up to twenty or more hours out of the twenty-four. *The benefit to be derived from rest in bed cannot be overemphasized.* Even as

the patient improves, he should continue to obtain, if possible, his afternoon rest, rest after dinner in the evening, and a long period in bed each night. This is one safeguard in avoiding fatigue. Hospital beds, with adjustable springs can usually be rented or purchased; this will greatly facilitate the handling of many of these patients.

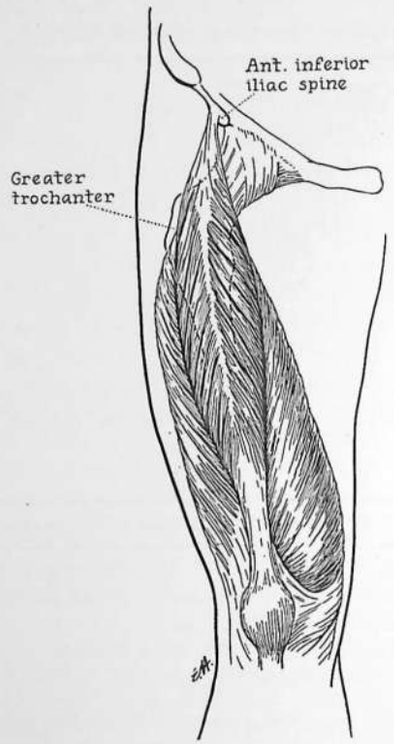

FIG. 40.—Sketch of *quadriceps muscle.* Muscle "setting" exercises are of extreme importance in the treatment of rheumatoid arthritis. In individuals with marked damage to the knee joint or in acute arthritic episodes when movement of the knee joint is impossible either because of pain or because of the use of a plaster cast, the quadriceps muscle must be exercised by the patient or this will undergo marked atrophy. The patient can be taught to contract or "set" this muscle if he understands its position and function. One of the best methods of teaching this is to explain to the patient that he must attempt to voluntarily contract this muscle by trying to squeeze it (by pushing the knee backward against the mattress—without using the hands). When done properly, the muscle mass will rise slightly and the quadriceps tendon will be felt to move below the patella. The exercise is performed most easily if lying down with the foot held at right angles.

2. **Splinting of Affected Joints.**—(*a*) Marked improvement may be noted if acutely inflamed, tender joints are placed in positions of physiologic rest at the onset of an arthritis.

(*b*) Acutely painful joints should be immobilized (whenever possible) by splints—with the joint in a position of physiologic rest (and one of election if ankylosis is inevitable). These positions include 10 to 15° of flexion for the knee; dropping the foot 10 to 15° beyond the right ankle; about 30° of dorsiflexion (*i. e.*, extension) of the wrist; semiflexion of the fingers and metacarpophalangeal joints, with adduction of the thumb. These are described in detail in the chapter dealing with splinting of affected joints.

FIG. 41.—Long-standing *rheumatoid arthritis* in a woman aged fifty-one years, who has had involvement of the hands and feet since fourteen years of age, with marked residual deformity. At present, she has leukopenia and splenomegaly. The small joints of the hands (especially the metacarpophalangeal joints and wrist) show advanced destructive changes. The cartilage has been completely destroyed and the articular ends of the bones are moth-eaten. Dislocations have occurred at some of the metacarpophalangeal joints.

(*c*) The splints should be removed each day and the joints moved once or twice through a full range of painless motion (varying with the tolerance of the patient); following this, the splints are reapplied. The aim is to permit sufficient motion to prevent ankylosis and sufficient rest to permit healing.

(*d*) Splints may be of molded plaster of Paris, Castex, leucite, etc. Elastic bandages are an economical method of applying light additional support for involved joints, especially the knee joints.

When Weight Bearing is Begun.—If the lower extremities have been involved in the arthritic process: (1) Support affected weight-bearing joints by splints, braces or bandages (elastic). (2) Begin weight bearing

with assistance of a walker, if obtainable. (3) Gradually increase weight bearing until patient can manage on one or two crutches.

Prevention of Deformity in Arthritis

1. It is usually much easier to prevent a deformity in arthritis than to correct one. *It is the duty of the general practitioner to prevent the occurrence of deformities*, as most deformities in arthritic patients *can* be prevented.
2. In the early stages of arthritis, deformities result from muscular spasm with muscular strain and joint irritation from faulty body mechanics.
3. Flexion and adduction deformities are most common, as these positions afford the patient the greatest relief from pain and because of the more powerful action of most flexor and adductor groups of muscles over the corresponding extensor and abductor groups.
4. A *non-sagging mattress* must be used (often with boards 1 foot wide and 1 inch thick placed lengthwise under this); pressure from bedclothes must be avoided (by means of a *pillow or cradle at the bottom of the bed* with the sheets passing over this). *Avoid a soft mattress and sagging spring.*
5. Place only one pillow under the head—in some cases—none; several pillows may force the cervical and dorsal spine into forward flexion.
6. For many arthritic patients *resting in a chair for long periods is not advisable* as the position thus assumed leads to flexion contractures of the spine, hips, knees, elbows and wrists, with internal rotation and adduction of the shoulders and pronation of the forearms.
7. **Muscular spasm must be inhibited promptly or actual shortening may occur in the joint capsule, and fibrous or bony adhesions may result.**
8. **Never place pillows under the knees** if arthritis involves the knee joints; this error is the commonest cause of a flexion deformity at the knee.
9. If the knee is involved, do not permit weight bearing until the acute inflammation has entirely subsided, and a period of exercises, without weight bearing, has produced no harmful effect.
10. When the feet are involved in an arthritic process, no weight bearing is permitted until pain and swelling of the feet have entirely subsided, and until the foot muscles have been strengthened by non-weight bearing exercises. A cradle should be placed over the feet, so that the weight of the bedclothes will not eventually produce a position of plantar flexion (equinus).
11. If there is a tendency to footdrop, the foot may be supported at right angles to the leg, by sand bags or molded plaster splints, or by a foot board at the bottom of the bed (with the head of the bed elevated 6 to 12 inches).
12. All arthritic deformities are not avoidable. A great number of these should not occur. However, in some cases, despite the best care and attention, sudden exacerbations of the arthritic process may result in contractures within a very short period.
13. For prevention of deformities at the hip, spine, wrist, fingers, elbows, etc., see the detailed discussion in the chapter devoted to Splinting of Affected Joints.

3. Physical Therapy.—(*a*) Physical therapy is an important adjunct to other treatment in the arthritic. Its injudicious use may cause harm.

Physical therapy alone does not cure arthritis and should not be employed to the exclusion of other anti-arthritic treatment.

If Plaster Splints or Bivalved Casts Are Needed for Prevention or Correction of Deformities of the Legs:

1. Place the extremities in as nearly normal position as possible before application of plaster.
2. If the patient is not in the acute stage of the disease, better position and further muscular relaxation can usually be obtained if the patient is anesthetized and the plaster applied during anesthesia.
3. All bony prominences should be well padded before application of the plaster to prevent pressure sores.
4. The foot is kept at a right angle to the leg and the plaster should usually extend from the gluteal fold downward to beyond the toes (if the knees are involved); the lower end of the cast is left open so that the toes may be inspected frequently (especially during the first twenty-four hours) for evidence of circulatory embarrassment.
5. If both legs are held in plaster casts, prevent outward rotation of the legs by incorporating a wooden crossbar into the casts posteriorly at the lower legs or heels.
6. Bivalve the cast after several days so that heat, massage and exercises may be given, as indicated. The posterior portion should be retained as a splint, which may later be required only at nights to maintain proper position of the legs.
7. As soon as possible, each affected joint should be moved through a full range of painless motion several times each day to prevent the occurrence of adhesions.

(*b*) This special therapy should be dispensed with care, under the direction of a physician. The coöperation of a well-trained physical therapist is always advisable, so that members of the patient's family can be instructed in the proper use of massage, exercises, hot applications, etc.

(*c*) Patients with most forms of arthritis are usually grateful for the local application of heat. In those individuals whose symptoms are aggravated by heat, this should be discontinued or modified. The greatest value of heat is in chronic arthritis.

(*d*) In the acute stage of an arthritis, **do not employ** diathermy, ultrashort wave therapy or hot baths.

(*e*) In the subacute or chronic stage, heat is a useful weapon and when possible should be applied daily to the affected joints, followed usually by carefully applied massage of *the muscles between these joints, with lighter stroking of the joints themselves.* If the arthritic process is more or less generalized, the hot water tub bath may be very effective.

(*f*) Heat increases blood and lymph flow in the affected area, thus aiding in the removal of waste products and hastening the absorption of inflammatory exudates. It also increases local nutrition and relaxes muscles, which may be in spasm. Mild local heat often relieves pain.

(*g*) Physical therapy includes the use of such physical agents as heat, cold, rest, muscular exercise, massage, hydrotherapy, light and electricity.

(*h*) **Under-water therapeutic exercise is very effective** in stimulating flabby muscles and in restoring their normal use. The buoyancy of the water, and its efficiency in overcoming the force of gravity, is the great advantage of under-water treatment. The weight of the body is almost completely carried by the sustaining power of the water, permitting freedom of movements and hastening the process of rebuilding atrophic muscles. A patient who weighs 132 pounds will weigh only 6 pounds when lying in water—thus permitting the weakened muscles and joints to perform actions under the water which would be impossible out of the water. Aided by the buoyancy of the water, extension of affected joints may be performed in marked comfort under water. *Buoyancy is present in any water, whether it is at the most expensive spa or in the cheapest home-made pool or bath tub.* Hydrotherapy is contraindicated in acute rheumatoid arthritis because of the probability of fatigue and increased activity of the disease.

(*i*) Hot paraffin applications are often useful in the treatment of chronic arthritis of the hands and wrists. For detailed technic, refer to the chapter on physical therapy.

(*j*) *Heat,* followed by properly administered *massage* and regulated *exercises* are extremely important factors in the treatment of chronic arthritis. Heat may be applied by bakers, hot water bottles, electric pads, hot fomentations, hot bricks or sand bags, heat lamps, hot foot or arm baths, hot whirlpool baths, diathermy, ultra-short waves, infra-red irradiation, hot paraffin applications, hot air douche, electric cabinet baths, etc.

(*k*) Any object which has been heated to a higher temperature than its surroundings emits infra-red rays. Infra-red rays produce an instantaneous sensation of heat when the intensity is great. Mild infra-red radiation often relieves the pain in acute arthritis. This may be given for fifteen to thirty minutes several times a day, and the maximum heat tolerated is focused upon the painful joint. The infra-red generator is placed from 2 to 3 feet from the patient depending upon the type of reflector and the sensitivity of the parts to be treated. Avoid blistering of the skin.

(*l*) The effects produced by *diathermy* or short-wave therapy can be attributed solely to the generation of heat, and not to any specific biologic or bactericidal action. Diathermy has had wide vogue in recent years in the treatment of many subacute and chronic painful, inflammatory conditions, especially in deep-seated portions of the body. Its greatest usefulness in rheumatoid arthritis applies to the chronic stage of this disease; in these patients diathermy or short-wave diathermy may be applied to the affected joints daily, or two or three times a week, followed by massage. Medical diathermy in chronic arthritis should always be supplemented by other forms of heat two or three times a day. The first several doses should be of low intensity and for a short period. In some cases of arthritis diathermy seems to aggravate the local symptoms; if this continues, such treatment must be abandoned. Local diathermy is not used in acute infectious arthritis. In fact, diathermy is not essential in the treatment of rheumatoid arthritis. In chronic arthritis, subjective and objective improvement following the judicious use of bakers or infra-red therapy may

equal that obtained by expensive diathermy machines. However, short-wave diathermy will heat the deeper tissues and will often have a good psychic effect upon the individual. It is especially valuable in low back strain and in subdeltoid bursitis.

(*m*) In chronic arthritis, general heliotherapy may be of tonic value to improve the appetite and sense of well-being. Do not employ heliotherapy or ultra-violet irradiation in patients with acute rheumatoid arthritis because of the possibility of producing increased activity of the joint process. **No one has ever demonstrated that sunlight has any specific beneficial effect upon the course of rheumatoid arthritis.**

(*n*) Occupational therapy may be of value in keeping the patient's mind occupied so that he does not constantly think about his illness; the work involved often results in rehabilitation of the individual.

Massage in Rheumatoid Arthritis

1. Unless contraindicated, every patient with non-acute rheumatoid arthritis deserves proper massage. **Contraindications to massage include** an acutely inflamed joint, a temperature above 100° F., a suspicion of malignancy or phlebitis or increased pain, swelling or stiffness in a joint persisting (more than two hours) following the massage.
2. *Beneficial massage should never cause pain while being given. Always give too little, rather than too much massage. Do not massage painful or markedly swollen bones or joints.*
3. Begin the massage with gentle stroking and later use deeper stroking motions, kneading and friction of the surrounding muscles for ten minutes twice a day. This is best performed after at least fifteen minutes of heat to the tissues. See chapter on Massage for details.

Exercise in Rheumatoid Arthritis

1. Proper exercises are extremely important in treatment of the patient with rheumatoid arthritis. *Too much exercise may cause irreparable harm to the arthritic.*
2. *At least one or two painless gentle movements of the affected joints must be performed daily even in the acute stage of the disease so that dense adhesions are not allowed to form.* Definite exercises should not be begun until a joint is without pain while at rest. As the patient improves, the amount and usually the type of exercise is modified and increased.
3. **Never exercise a patient to the point of persisting pain or fatigue.**
4. Any motion which is attended, or followed within twenty-four hours, by an increase of pain, swelling or stiffness in the joints has been excessive; this must be reduced or modified. Proper exercises should cause no severe pain during their performance.
5. Even in the more chronic stages of an arthritis, not more than an hour of continuous exercises should be performed.
6. The occurrence of muscular spasm during an exercise is a signal for cessation of the movement.
7. Fibrillation of muscles indicates that the active exercise is too severe.
8. All movements must be slow, deliberate and not jerky or tiresome. All periods of strenuous exercise must be followed by an equal period of rest. See chapter on Exercises for details.

The patient with rheumatoid arthritis must spend from ten or twelve up to twenty-four hours a day in bed, varying with the stage of his disease. In the acute stage of the inflammatory process, the affected joints must be rested and very little motion or exercise must be undertaken. When the arthritis has become subacute, carefully performed motions and exercises should be performed, increasing these as the disease becomes less active and a chronic stage is reached. If exacerbations of the disease occur with further inflammatory changes in the joints, periods of complete rest in bed may be necessary, especially if weight-bearing joints are involved.

In the acute stage of rheumatoid arthritis, massage and exercise should not be undertaken, but physical therapy should be confined to the application of heat. Although many individuals feel that any form of heat is as useful as another, we have found that the patient with an acute exacerbation obtains more relief when *hot fomentations* are used *over the affected joints* for twenty or thirty minutes, three or four times per day. This may be done by using pieces of old blankets or turkish towels, which have been soaked in hot water (with or without epsom salts) and wrung out fairly well, and wrapped around the joints involved. The wet blankets or towels may be covered by dry towels or blankets to preserve the warmth as long as possible; the blankets are soaked in the hot water every several minutes and reapplied as often as necessary (to prevent cooling of the fomentation). This application and reapplication of the hot wet blanket or towel, wrapped around the joint, is continued for twenty or thirty minutes three or four times a day. When hot fomentations are not available (which is rare), one may employ a heating pad, hot water bottle, home-made baker, infra-red lamp or other source of heat. That form of heat which is most analgesic should be continued.

As the disease becomes subacute and the patient attempts to make a few movements. one may begin effleurage (very light massage) and (cautiously) exercises in bed (without weight bearing). Too much motion may cause further joint damage and lead to crippling; too little motion in this and later stages may permit dense adhesions to form. It must be stressed to the patient that as his disease passes from the acute to the subacute stage, he must not overdo either rest of the joints or exercise. Unfortunately, the patient or occasionally his physician will feel that he must move his joints continuously in an effort to prevent adhesions; such motions will usually lead to further joint disability. Other individuals may wrongly perform no motions of the joints because of possible damage which may result from such motions; this view is also usually wrong because prolonged inactivity without even a few motions may lead to crippling, or marked limitation of function.

In resting the involved joints, one must include instructions as to reduction or possibly complete *avoidance,* if possible, *of postural, static, recreational or occupational trauma.* The static trauma from marked obesity or poor posture may be lessened by decrease in weight, supports (elastic bandages, corrective shoes with properly placed pads or metatarsal bars, corsets, braces, etc.) and postural exercises. One must be very careful in advising arthritic patients to give up their jobs when such individuals are responsible for the family income; in some cases, this procedure may be absolutely essential. If the patient must continue at some kind of work for the support of his family, it may be possible to modify his job or his hours of work, or to provide additional rest periods during his work, or frequent vacations from his job.

Heat, massage and exercises improve the circulation of the affected parts and assist in decreasing or preventing muscle spasm and deformity and improving muscular tone; they are often of benefit in decreasing the inflammatory process.

It should be emphasized to the patient by his physician that **physical therapy should be carried out daily** at the patient's home by means of simple inexpensive apparatus; members of his family may be trained in the essentials of this undertaking. In addition to this, a trained physical therapist may treat the patient at his home or office, if possible, two or three times a week so that careful supervision may be maintained and the therapy may be regulated or modified according to the particular needs of the patient at that time. In patients with chronic rheumatoid arthritis, it is often advisable, in addition, to spend several weeks once or twice a year in a well-equipped hospital or spa for more intensive therapy, general check-up and possibly, the benefits of a vacation away from home. The patient must definitely realize that 21 baths at some particular spa will not cure his rheumatoid arthritis. The individual must be told that despite the claims of any particular spa concerning its mineral waters and their supposed virtue, *it has not been shown that appreciable amounts of any element are absorbed from such waters or muds,* but that the value of such spa or hospital therapy includes the rest, vacation away from the cares of the household, and the value obtained from heat, massage and hydrotherapy.

It cannot be stressed too often that home physical therapy should not take the place of professional physical therapy, but that it should supplement this. One can seldom train a member of the family to perform skilled massage, and it is dangerous for the family to attempt any special exercises or other measures except in the presence of the physical therapist and with the full knowledge of the physician in charge.

Most patients with rheumatoid arthritis obtain a soothing effect from the application of moist heat in the form of hot fomentations. After the acute stage of the disease has passed, it is often difficult to continue the time-consuming job of continuing these applications several times a day. At this stage, one may change to any form of heat which seems preferable to the patient, whether this be dry heat, heating pad, baker, hot water bottle, infra-red lamp, diathermy, etc. The psychic effect of the latter is sometimes enormous; this psychic effect cannot be ignored.

During the subacute or chronic stage of the process, pool baths are especially valuable. **Hydrotherapy is an extremely important tool which has not been used to advantage by many physicians.** *Hydrotherapy is contra-indicated in acute rheumatoid arthritis* because of the probability of fatigue and increased activity of the disease. Underwater therapeutic exercise is

very effective in stimulating flabby muscles and in restoring their normal use. The buoyancy of the water and its efficiency in overcoming the force of gravity are great advantages of under-water therapy. Inasmuch as the specific gravity of water is about the same as that of the body, the weight of the latter is almost completely carried by the sustaining power of the water, permitting much greater freedom of movements and hastening the process of rebuilding atrophied muscles. There is also a great stimulating effect upon the patient's morale when he finds that he can readily move his limbs when immersed in water (although this may have been almost impossible when in bed). Aided by the buoyancy of the water, extension of affected joints may be performed in marked comfort under water (as compared to the discomfort of weights and pulleys attached to a bed).

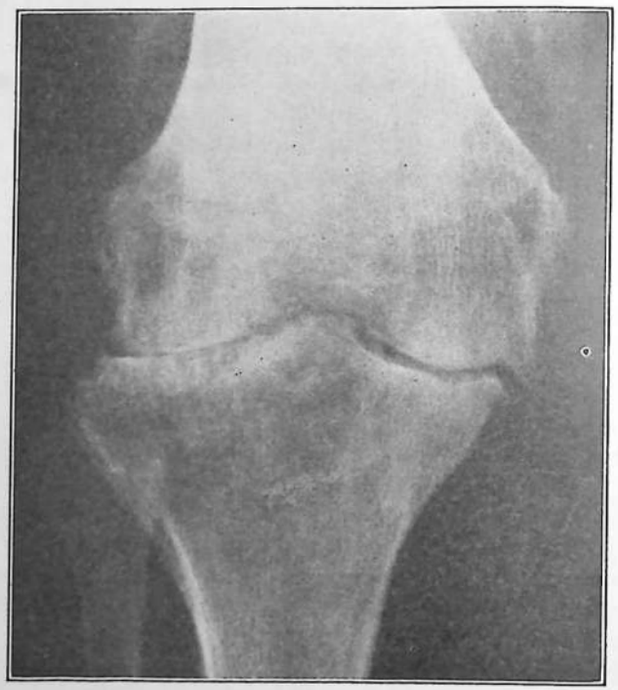

FIG. 42.—Long-standing *rheumatoid arthritis* in a woman aged sixty years. There are extensive changes involving the articular surfaces and adjacent portions of the bones of the knee joint. There are also changes in the soft tissues. On the mesial side of the knee joint, there are some hypertrophic changes. Note the marked narrowing of the joint space, indicating destruction of hyaline cartilage.

Occasionally, it may be impossible to exercise joints because of marked pain; in such instances, if muscle setting exercises cannot be performed, faradic or sinusoidal currents may be used to produce relatively painless contractions of muscles, thus reproducing the effects of active exercise and preventing atrophy of adjacent muscles.

Most physicians do not employ **occupational therapy** to the full extent

which may be utilized. It is often possible to accomplish results by occupational therapy which would have been considered impossible by other methods. By proper occupational therapy, the patient is taught special exercises in an effort to improve the function of his affected joints. The patient learns to regard the work as a valuable medical exercise and not merely a pastime or drudgery. Such occupational therapy may include washing dishes, using a screw driver, shelling peas, weaving rugs, typewriting, making beds, shooting marbles, etc. *The prescription for occupational therapy should be definite and the patient must be given definite instructions together with the reasons behind these particular actions.*

Properly Performed Graded Exercises

1. Must not fatigue the patient.
2. Must not cause continued pain after their completion.
3. Must not be begun too soon in the acute stage of rheumatoid arthritis.
4. Probably aid in the absorption of joint exudates.
5. Greatly encourage the patient.
6. Are carried out first with the patient flat in bed.

Heat, in the Patient With Rheumatoid Arthritis

1. Will *usually* lessen joint pain.
2. Will usually be followed by greater movement.
3. May occasionally aggravate the symptoms during the acute stage of the process; discontinue it temporarily in these few patients.
4. May be varied; acceptable forms are: hot wet packs to the affected joints, for periods from twenty to thirty minutes three or four times a day; bakers; electric pads; diathermy, etc.
5. Should not tire or fatigue the patient.
6. Should not cause discomfort.
7. Should be applied several times a day.
8. May cause excessive sweating and tend to weaken the patient; these individuals must be given additional fluids and sodium chloride.
9. May be ordered as warm tub baths or bubble baths when multiple joints are involved unless the patient is debilitated or if the arthritis is in the acute stage.

Warm Tub Baths

1. Are often valuable in the subacute or chronic stage of rheumatoid arthritis.
2. Do not use in debilitated patients or in those with acutely inflamed joints.
3. First treatments should be not more than five or ten minutes in the tub, with the temperature gradually raised to not more than 100° F.
4. Increase the length of stay in the tub up to twenty, or a maximum of thirty minutes, and the temperature up to 103° F. or more, if tolerated.
5. If excessive sweating and weakness occur, give the patient added fluids and sodium chloride.
6. Contrast baths are often of value in treatment of rheumatoid arthritis of the feet and ankles.
7. Mild exercises may be begun underwater in the tub or Hubbard tank or pool; mild massage may be given in the tub or pool.

Diet in Rheumatoid Arthritis

1. In most cases, a high caloric, high vitamin, anticonstipation diet is indicated (unless the patient is overweight).
2. *There is no specific diet applicable to all patients with rheumatoid arthritis* except a sensible intake for that individual: high in vitamins and calories and adequate in calcium, phosphorus and iron.
3. There is no definite evidence that any of the following diets are of specific value in rheumatoid arthritis: low caloric; low carbohydrate; changed acid-base balance of the diet; omission of so-called acid fruits and vegetables; allowing only one type of food substance at a meal; omitting foods to which the patient is hypersensitive; or a low protein diet. Walter Bauer has carefully evaluated these special diets and found no substantial evidence to support their specific use. Most rheumatologists agree with his findings in this regard.
4. The daily diet should contain two or three glasses of milk, several glasses of tomato juice or fruit juice; meat, seafood or fowl; cheese; fresh green vegetables; butter, cream and eggs as desired, and any additional foods chosen by the patient provided that these contain a sufficient number of calories to maintain weight, or to increase this if the patient is grossly underweight (or that the patient does not gain weight if he is already overweight).
5. Meat is needed by the arthritic because of its vital body building contents. Citrous fruits are also required because of their large content of vitamin C.
6. In those undernourished and weakened individuals with rheumatoid arthritis, it seems ill-advised to prescribe a low caloric diet with marked limitation of carbohydrates. High protein and high carbohydrate diets with large amounts of citrous fruits are definitely beneficial (rather than harmful) to the individual with rheumatoid arthritis. *There is no good reason why any food or class of foods should be restricted from the diets of these individuals* unless such articles cause gastrointestinal upsets or the patient is allergic to such foods.

4. **Diet in Rheumatoid Arthritis.**—One of the first questions which the arthritic patient asks his physician is "What kind of diet do you advise?" These patients have heard of the supposed virtues (?) of various types of diets (low carbohydrate, low sodium, high potassium, low caloric, low protein, etc.) and are at times amazed when the intelligent physician explains to the patient that his diet should be sensible, well balanced and nutritious, containing an adequate amount of calories, vitamins and minerals and usually an anticonstipation regimen. There is no proof that lack of any specific food is a causative factor in rheumatoid arthritis.

We usually prescribe a high caloric diet, inasmuch as most patients with rheumatoid arthritis have lost weight and are under par as regards their energy. An abundance of calcium, phosphorus and vitamin D (to insure absorption of these minerals from the gastrointestinal tract) is advisable in the diet in an effort to combat the local and diffuse atrophy of bone which is the usual occurrence in these individuals. Meat (particularly liver) is helpful in the prophylaxis and treatment of the hypochromic anemia which so often accompanies the disease; additional iron should often be prescribed (such as ferrous sulfate). If the patient is overweight, carbohydrate may then be restricted; in some instances, one must restrict the carbohydrate somewhat so that the patient will consume an adequate amount of fresh green vegetables, fruits, milk and meat, which seem more essential.

In addition to a well-balanced diet with an adequate caloric intake, one should add vitamin concentrates as capsules or other forms; thus far, there seems to be no specific effect upon the arthritis which is achieved by any one particular vitamin. Nausea, vomiting and the dangers incident to a persistently elevated blood calcium may follow the prolonged use of massive doses of vitamin D.

In patients who are greatly underweight, we often advise 1 or 2 ounces of cod-liver oil morning and night, if tolerated, in an effort to supply vitamins A and D, as well as added calories; some patients cannot tolerate this even though it is kept cold in the refrigerator. In these individuals, Maltine with cod-liver oil may be tried.

If the patient drinks a full glass of orange juice daily, it does not seem necessary to add extra ascorbic acid (vitamin C) to his diet. In some of these individuals with poor appetites, vitamin B complex (capsules) often aid in improving the appetite and the general sense of well-being. If these are not tolerated, one may try thiamin chloride alone (5 mg. b.i.d.), or in combination with riboflavin (1 to 2 mg. per day) and niacinamide (100 mg. per day). *Great care must be taken not to overload the rheumatoid arthritic with medications which may upset his gastrointestinal tract and thus decrease his dietary intake,* or cause nausea and vomiting; this must be adjusted to the individual patient.

5. **Relief of Joint Pain.**—The physician must attempt to relieve pain in the patient with rheumatoid arthritis. *Pain prevents proper rest, appetite, sleep and exercise, which are important factors in the recovery process.* Peripheral circulation and muscle function may be decreased by persisting pain, possibly through nervous impulses.

If drugs are required to relieve pain, *aspirin is usually the most effective.* Because of the marked sweating from very large doses of aspirin, we prefer to give equal amounts of aspirin and phenacetin as needed, ranging from 0.3 to 0.5 gm. (5 to 7½ grains) on awakening, at noon, after supper and at bedtime; this may be increased if necessary. If these medications cause gastric irritation, sodium bicarbonate may be prescribed, 0.3 to 0.5 gm. (5 to 7½ grains), with each dose of the analgesics.

There is little justification for the intravenous use of salicylates in the patient with rheumatoid arthritis; most of these patients tolerate oral medication if prescribed intelligently.

In some of these patients, small doses of phenobarbital, 15 mg. (¼ grain), may be given two or three times a day, and 30 mg. (½ grain) or more at bedtime, if required because of nervousness or sleeplessness. Other hypnotics may be needed and should be prescribed unless the insomnia is due to pain.

Some of these patients obtain relief from pain *by infiltration of 1 per*

cent procaine hydrochloride around the joint; at times, this relief of discomfort is more than a temporary affair.

Psychotherapy is often of definite value in lessening the patient's discomfort. In some of these patients anxiety, worry or grief may be a major contributing factor in the progress of the disease.

Relief of Joint Pain

1. *Proper splinting of acutely inflamed joints* in positions of physiologic rest will often produce marked relief of discomfort.
2. Considerable amounts of anodynes are usually not required in the arthritic patient, except during the acute stages of the process (in some patients).
3. If pain persists despite splinting in a position of physiologic rest, give aspirin and phenacetin, 0.5 gm. (gr. vii $\overline{ss}$) of each, every three or four hours, as may be necessary during the acute stage of the disease. Salicylates are analgesics and vasodilators. The analgesic action of aspirin seems superior to that of sodium salicylate in rheumatoid arthritis. Aspirin must be used with caution in asthmatic patients and especially when nasal polyps are present (because of the increased sensitivity to this drug in some of these patients). Plenty of water should be taken with the aspirin to lessen gastrointestinal discomfort.
4. Although it should be avoided in the average case of rheumatoid arthritis, it may occasionally be necessary to add small amounts of codeine phosphate, 10 to 15 mg. ($\frac{1}{6}$ to $\frac{1}{4}$ gr.), every four hours if the pain is severe, in the early stages, or if absolutely necessary—dilaudid, 3 mg. ($\frac{1}{20}$ gr.), three or four times a day, may be given.

If narcotics are required over a lengthy period the diagnosis should be checked; a malignancy may be present.

5. Alternating hot and cold douches to inflamed joints often relieve pain; use warm water for one and a half minutes, then cool water for one-half minute and continue to alternate these in this manner for twenty or thirty minutes.
6. Although aminopyrine relieves joint pain, it has been incriminated as an important causative factor in agranulocytic angina and is not recommended because of this.
7. Many physicians employ cinchophen for the relief of pain in various forms of arthritis and gout. This drug is not recommended because liver damage (including acute yellow atrophy and cirrhosis) has followed its use; intoxication has also been noted following the use of neocinchophen.
8. Cobra venom has been used for the relief of intractable pain. Two or three days of successive injections are required for the production of analgesia by cobra venom. An ampoule (5 mouse units) is given intramuscularly daily until an analgesic effect is obtained and then two or three times a week in order to keep the patient comfortable. Such injections have been continued for months. Cobra venom is usually ineffective in relieving pain in rheumatoid arthritis.

6. **Psychotherapy.**—Individuals with rheumatoid arthritis are usually introspective, fretful, lack confidence in themselves (and occasionally in their physician) and are often seemingly unreasonable; *psychotherapy*, whether

used consciously or unconsciously, is *absolutely essential to the proper handling of these patients*, who are especially amenable to suggestion.

Psychotherapy is an extremely potent weapon in the armamentarium of the physician treating rheumatic patients. This includes the improvement which follows telling the patient that his swelling of the knee has decreased $\frac{1}{16}$ inch (by actual measurement) from the previous measurement, the effect of the hypodermic or intramuscular needle, confidence of the physician, the patient's knowledge that his physician has other lines of attack which he may fall back upon if the first or second lines fail, gradually increasing mobility of stiff joints, even slight decreases in the sedimentation rate or increases in the hemoglobin content, etc. The physician should never withhold any good news from his arthritic patient.

The arthritic patient must be questioned carefully about his intimate relationships with members of his family, his friends and business associates; these individuals are usually eager to unburden themselves. One must try to expose to the patient his resentments, conflicts or actual hatreds and his fears. One may then explore the individual's personal beliefs or his own standards of living. People do not often like to face unpleasant truths about themselves and one must guide them to get their own convictions of where they are wrong and causing them to want to change them. Swaim believes after long personal experience with large numbers of arthritic patients, that if people would cultivate a mature, developed faith and belief in a super-force as part of their lives, they would not react with fear and anger the way they do under emotional strain and their health would be better and they would be less susceptible to disease; he feels that this spiritual factor may turn the tide of success or failure in some cases.

7. **Orthopedic Care in Rheumatoid Arthritis.**—Whenever possible, one should obtain the coöperation of an orthopedist early in the course of the disease. Many of the deformities which are seen in arthritic patients are the result of prolonged muscular spasm; in the early stages of the disease, this spasm can usually be overcome by periodic rest, decrease in trauma and the use of supports such as corsets, braces, proper shoes (with pads and metatarsal bars if needed) or plaster splints. If the muscular spasm is allowed to persist, contractions of the joint capsule and tendons occur. Most of these deformities with their antecedent muscular spasm result from the attempt of the patients, or often of oversympathetic relatives, to keep the joints in those positions which are least painful. This includes the placing of pillows under inflamed knees, prolonged flexion of various joints, etc. In our experience, the quickest and surest method of producing flexion contractures and deformities of the knees is to permit the prolonged use of pillows or folded towels under the knees. Every physician must warn the patient and family concerning this and must examine the patient on each subsequent visit to be sure that either the patient or family, or both, have not weakened and replaced the harmful pillow under the knees.

Orthopedic Care in Rheumatoid Arthritis

1. Orthopedic care aims to preserve the function of joints or to restore such function if this has been impaired.
2. *There is less danger of ankylosis, adhesions and loss of motion if inflamed joints are rested by splinting them, than if they are constantly moved in an effort to prevent stiffness and loss of motion.* Ankylosis does not develop follow ng temporary complete immobilization of joints.
3. Molded plaster splints should be used to immobilize acutely involved joints where possible; these should be removed once daily to permit one or two full painless motions of all joints included in the splinted area. This fact cannot be overemphasized.
4. Manipulations of deformed joints should be avoided if possible; they should never be employed during the active stage of an arthritis because of the possibility of a resulting flare-up or ankylosis. Manipulations should be performed only by those well trained and skilled in such procedures.
5. Orthopedic surgery in chronic arthritis may be of great value if associated with intensive training in muscular exercise for at least several weeks prior to the operation.
6. Do not perform joint operations during the acute stage of an arthritis; in most instances joint surgery should be considered only after a period of at least six months of quiescence from joint activity.
7. Surgery should aim to obtain a functionally useful joint. Begin postoperative joint motion as soon as possible (when soreness has disappeared). *Do not maintain the joint in one position for long periods postoperatively, unless absolutely essential.*
8. Arthrodes's (removal of articular surfaces in order to produce bony fusion) is ill-advised in most patients with rheumatoid arthritis except in some instances when the midtarsal and subastragalar joints of the feet are involved.
9. *Orthopedic surgery in rheumatoid arthritis includes* synovectomy (in selected cases), posterior capsuloplasty of the knees, osteotomy, arthroplasty (construction of a new joint), reconstructive operations, excision of the patella, drilling of the femoral head, etc. See chapter on Orthopedic Surgery for details. Sympathectomy is seldom employed in chronic arthritis at present.
10. Arthroscopy (direct visualization of the joint cavity) may be practiced by means of an arthroscope without fear of infection or trauma to the joint, but should be used only by one trained in this procedure.

Manipulation of Joints in Rheumatoid Arthritis

1. Flexed knees may be straightened safely, in most cases, by application of a plaster cast from the upper thigh to the toes, with the knee in a position of maximal correction; this may be bivalved in a few days to permit heat, massage and mild exercises. New casts may be applied every week or ten days as the flexion deformity lessens, using the posterior portions of these as posterior molded splints (after the cast is bivalved).
2. In some cases with marked deformity, gentle manipulation of the joint may be performed (by one trained in this work) under anesthesia, followed by the application of a plaster cast or splint. *Manipulation should not be undertaken in acutely inflamed joints.*

Manipulation of Joints in Rheumatoid Arthritis—(*Continued*)

3. Wedging casts may be used to correct a flexion deformity of the knee (except in those cases when the synovial membrane is acutely inflamed or if there is a posterior subluxation of the tibia).
4. In some intractable flexion deformities of the knee, moleskin adhesive traction may be required (avoid irritation of the skin by applying Tr. of Benzoin to the skin generously before application of the adhesive); surgical procedures such as posterior capsuloplasty may be needed.

Surgery of Arthritic Joints

1. *Do not operate on a joint during the acute stage of rheumatoid arthritis.*
2. When possible, attempt to obtain a functionally useful joint.
3. Begin postoperative motion as soon as possible—when soreness has disappeared.
4. Do not maintain the joint in one position for long periods postoperatively unless absolutely necessary.
5. Develop the surrounding muscles by exercises for days or weeks prior to operation.
6. *Synovectomy* at the knee joint may be advisable when prolonged conservative treatment has failed to render the process quiescent, or in those chronic cases with persisting swelling and chronic hydrops from hyperplastic synovial membrane, thickened and thrown into folds, filling the anterior portion of the knee under the patella.
7. *Arthroplasty* (*i. e.*, the reconstruction of a new joint) is the only surgical treatment of ankylosis. This operation attempts to model an artificial joint. *Arthroplasty should never be attempted unless the joints are in a quiescent phase;* it is especially beneficial in the elbows, knees and hips. The usual technic is to divide the bones at the line of the former joint, smooth and trim the ends to simulate normal articular configuration and to stitch a sheet of fascia lata (removed from the thigh)—or in some clinics, a sheet of sterile cellophane—between the joint surfaces. The fascia or cellophane prevent the recurrence of ankylosis.
8. *Arthrodesis* is the removal of articular surfaces to produce fusion of bony surfaces (ankylosis)—or in some instances, the use of a graft to bridge a joint. This operation may be performed while the arthritis is still active. One hesitates to suggest this operation in patients with rheumatoid arthritis (except in some instances when the midtarsal and subastragalar joints of the feet are involved). Occasionally, arthrodesis may be advisable in a severely damaged joint with loss of all useful motion in which it is advisable to produce a painless, stiff joint, especially in weight-bearing joints where stability is more essential.
9. *Posterior capsuloplasty* may be advisable in some cases of contracted knees when there is no severe damage of the articular surfaces and when capsular contraction posteriorly prevents full extension and results in flexion contractures.
10. *Osteotomy* (cutting of the bone close to the joint) may be indicated occasionally in flexion contractures of the knees if the articular cartilage is so badly damaged that operation upon the joint capsule is unlikely to result in joint motion, or in some instances, to correct bony ankylosis in a deformed position.

Additional Symptomatic Care

This includes attention to insomnia, fatigue, constipation, anemia, undernutrition, lack of appetite, associated menopausal symptoms, varicose veins, etc.

1. **For insomnia: If due to pain,** aspirin, 0.6 to 1 gm. (gr. x to xv), triple bromides, 1 gm. (gr. xv), and chloral hydrate, 0.6 gm. (gr. x), given thirty minutes before bedtime. A palatable mixture is:

℞—KBr	30	ʒi
Chloral hydrate	20	ʒv
Water	15	ʒss
Flex. glycyrrhiza	15	ʒss
Syr. glycyrrhiza	q.s. ad 120	ʒiv

S.—Teaspoonful thirty minutes before bedtime. Aspirin may be taken separately with a full glass of water.

   If bromides and chloral prove unsuitable, phenobarbital may be used, beginning with 30 mg. (½ gr.) one hour before bedime and increasing if necessary to 0.1 to 0.2 gm. (1½ to 3 gr.). If sleep is not obtained, seconal or nembutal may be tried, 45 to 90 mg. (gr. ¾ to 1½).
2. **Extreme fatigue and tiredness: Amphetamine (benzedrine) sulfate,** 5 to 10 mg. on arising and again at noon. This dose may be doubled if necessary. **Glycine** is sometimes helpful (3 to 5 gm., three times each day). Ephedrine sulfate, 25 mg. (⅜ gr.), three times a day, may be tried.
3. **Constipation:** Abdominal massage, additional fluids prunes, abdominal exercises, regular bowel habits, mineral oil or granular agar agar, and if necessary, cascara. Treatment of constipation is considered in detail later.
4. **Undernutrition and lack of appetite:** Tr. nux vomica, 0.3 cc. (m. v), three times a day, before meals may serve as a mild gastrointestinal tonic. In selected cases, try small doses of **insulin** before meals for a period of several weeks (beginning with 5 units before breakfast and supper increasing to 10 or 15 units several times each day); **cod-liver oil** in generous doses—1 ounce two or three times a day (if tolerated); Brewer's yeast tablets, 9 per day and vitamin C, 100 mg. twice a day.
5. **Association with menopause:** If indicated give adequate amounts of estrogenic substance (such as stilbestrol, orally, 0.5 mg. per day) to relieve menopausal symptoms and some arthralgias associated with the menopause. In men, if arthritis is associated with hypogonadism and neurasthenia, cautiously try doses of testosterone propionate, 5 to 25 mg., three times per week (intramuscularly) in addition to small daily oral doses (10 mg.); increase dose cautiously if advisable.
6. **Anemia:** Ferrous sulfate, 0.2 gm. (gr. iii) or ferrous gluconate, 0.3 gm. (gr. v), from four to six tablets per day; or iron and ammonium citrate, 6 gm. per day (usually given as a 10 or 20 per cent solution). If achlorhydria is present, give dilute HCl (1 to 4 cc.) in water (preferably with added orange juice and sugar), three times a day, with meals. This should be taken through a straw to prevent damage to the teeth. *Blood transfusions* are often of value in initiating the recovery process even when only a slight to moderate amount of anemia is present. One should always search for and eliminate, if possible, any possible cause of loss of blood. In many patients as the rheumatoid arthritis improves the hemoglobin level returns toward normal without iron indication.

Additional Symptomatic Care—(*Continued*)

7. **Prostigmin bromide:** 10 to 15 mg., and atropine sulfate, 0.2 mg.—each t.i.d. orally—may be of benefit in relieving or *lessening muscular spasm.* Prostigmin methyl sulfate, 1 cc., 1:2000 solution may be given hypodermically together with 0.6 mg. of atropine sulfate every two, three or four days in an attempt to lessen muscular spasm. Recent investigations have shown that prostigmin is of very little value in patients with rheumatoid arthritis.

Foci of Infection

1. Remove or treat any definitely infected focus.
2. If removal of infected tonsils or teeth is required to clear up an infected focus, give the adult patient from 0.5 to 1 gm. of sulfamerazine every eight hours for twenty-four hours prior to operation and again every eight hours for twenty-four or forty-eight hours immediately following operation; this is done in an effort to prevent postoperative flare-ups in affected joints.
3. Foci of most importance include teeth, gingiva, tonsils, hyperplastic lymphoid tissue in the pharynx, paranasal sinuses and prostate; foci of questionable importance include the cervix of the uterus, phlebitis, appendix, gall bladder and the gastrointestinal tract.
4. *In patients whose arthritic symptoms come on acutely following the appearance of an infected focus,* dramatic results may be obtained at times following the proper removal of the focus; in general, it may be said that *the more rapid the onset of an arthritic process, the more favorable the prognosis.* Unfortunately, the number of patients with rheumatoid arthritis benefited (arthritically) by removal of infected foci is very small.
5. *Explain to the patient and his family that mere removal of focal infection will not cure his rheumatoid arthritis;* in a small percentage of cases, it may be followed by marked improvement. *The focus is being removed as a general health measure*—as would be done if the patient had no arthritis—in an effort to lessen the increased load which he is carrying.
6. Most medical men know too little about teeth and their disorders; a competent dentist should be urged as a consultant in all patients with rheumatoid arthritis. All adults suffering with rheumatoid arthritis should have a full mouth *x*-ray and the dentist must correlate his clinical and roentgen findings.
7. In greatly weakened, emaciated patients, attempt to improve the general health, if possible, before attacking foci.
8. If infected teeth must be removed, do this slowly, removing only one or a few at a time, permitting several days between extractions.
9. Except in the early stage of a rheumatoid arthritis, there is some question as to whether expensive combing of the patient for focal infection by consultants is warranted; in any case, the physician in charge must interpret these with a broad viewpoint of the patient's general condition, as a whole.
10. We advise a conservative attitude toward focal infection; we do not think that they should be neglected.

Blood Transfusions.—In some individuals, two or three small transfusions may initiate the recovery process, even when only a slight or moderate degree of anemia is present. In addition to its red cell and hemoglobin content, the blood may exert a beneficial non-specific effect and possibly aid in stimulating antibody formation—or may supply antibodies (as yet unknown). Transfusions also assist in raising the serum protein level.

Gold Therapy in Rheumatoid Arthritis

1. The status of gold therapy in rheumatoid arthritis is still under investigation.
2. At present, many large arthritic clinics in teaching hospitals (including our own) employ gold therapy in patients with rheumatoid arthritis.
3. Although chrysotherapy is not without definite dangers (such as numerous complications and an occasional death from purpura hemorrhagica, agranulocytic angina, liver damage, etc.), the current view of most rheumatologists is that it is an extremely useful adjunct in the treatment of patients with rheumatoid arthritis.
4. **Gold therapy should not be used by the general practitioner unless he familiarizes himself with all of the details of this therapy, the dangers involved, the precautions to be observed, and the DO's and DON'T's of gold therapy** See chapter on Gold Therapy for details.
5. Most clinics have reported optimistically on the use of gold by injection so that the Committee of the American Rheumatism Association which prepared the 1942 Primer on Arthritis wrote that a continuance of trial of this form of treatment under controlled conditions is justifiable and desirable in view of the large number of individuals who have been greatly relieved.
   In their last review, the editorial committee of the American Rheumatism Association agreed that it was not yet possible to give a final evaluation of *chrysotherapy*.
6. The effectiveness of gold preparations appears to depend on the gold they contain.
7. Gold remains in the body for months after treatment is stopped.
8. Colloidal gold salts have been reported to be of little or no value because the colloidal gold particles are quickly phagocytized.
9. It is not yet known how, where or why gold salts are of benefit in some cases of rheumatoid arthritis: it seems to lessen or arrest the inflammatory changes.
10. Gold salts do not straighten deformed joints and do not repair damaged cartilage.

Foci of Infection.—The average patient with rheumatoid arthritis tells his doctor that the latter must find the offending focus of infection in his body so that he can get rid of his arthritis. Many or most of these patients are firmly convinced from their conversations and reading that some focus of infection must be found, which, when eliminated, will result in the clearing of their disease. Unfortunately, this is seldom the case—*i. e.*, although numerous areas of infection may be found in the teeth, tonsils, sinuses, prostate—it is seldom that the mere removal of such offending foci alone will result in any great ameliorating effect upon the arthritis *per se*. The hypothesis for focal infection in rheumatic disease dates back to the

improvement noted by Benjamin Rush in 1801 in his female patient whose rheumatism disappeared from her hip after an aching tooth was removed. Very few individuals carried the work of Rush further until Billings in 1912 brought forth the thought of focal infection which spread very rapidly over the country. The pendulum is now swinging away from the focal infection theory for rheumatoid arthritis but we still do not really know the exact rôle which focal infection may play in this disease.

Foreign Protein Therapy.—(*a*) Foreign protein therapy benefits permanently only a small percentage of patients with rheumatoid arthritis.

(*b*) It is of greatest value in those patients whose symptoms have reached a standstill during the recovery stage, and stiffness remains as one of the outstanding symptoms.

(*c*) Substances used for foreign protein therapy include typhoid vaccine intravenously, Coley's fluid intramuscularly, Aolan or boiled milk intramuscularly, and intramuscular injections of whole blood from the patient himself.

(*d*) Although temporary symptomatic improvement often results from foreign protein therapy, this improvement is permanent in only about 10 to 20 per cent of patients.

Roentgen Therapy in Rheumatoid Arthritis

1. Roentgen therapy produces marked subjective and objective improvement in a large percentage of patients with *rheumatoid arthritis of the spine* (rheumatoid spondylitis). Care must be taken in treating over the female pelvis to avoid sterilization.
2. Although it may temporarily lessen pain and swelling, and increase joint motion in peripheral joints involved by rheumatoid arthritis, our present concept is that roentgen therapy does not produce a permanent improvement in rheumatoid arthritis of peripheral joints (except in a very small percentage of cases).

Effect of Climate on Rheumatoid Arthritis

1. Recent work by Selye and his associates suggests that exposure to cold may play an important rôle in the etiology of experimental lesions resembling rheumatic fever; the rôle of cold in rheumatoid arthritis has not yet been determined.
2. Exposure to cold, dampness and drafts usually results in greater pain and stiffness in the average patient with rheumatoid arthritis.
3. It has not yet been proven that a warm, dry climate has, by itself, any particular beneficial effect upon rheumatoid arthritis; however, if one had his choice, he would probably be more comfortable in such an environment.
4. It is usually stated that rheumatoid arthritis is rare in the tropics, but this has not been satisfactorily established as yet.
5. Even though the individual moves to a warm, dry climate, his arthritis should be carefully supervised by an individual trained in this work.
6. At present, we do not know enough concerning the effect of climate upon the average case of rheumatoid arthritis to advise that he make a great financial sacrifice or give up his means of livelihood in order to move to a warm, dry climate.

Triple typhoid vaccine given intravenously is still the favorite form of foreign protein therapy; moderate febrile reactions are usually strived for in rheumatoid arthritis—*i. e.*, a sufficient amount of the vaccine intravenously so that the patient develops a fever of from 102° to 104° F. for at least several hours. The amount of vaccine necessary to produce this varies with the age and general resistance of the patient, previous injections of typhoid vaccine, and the brand of typhoid vaccine used. The physician would do well to use only a single manufacturer's brand of typhoid vaccine, so that he will get to learn its potency and will be better able to predict the dose to be used to produce the desired effect. It is wise, also, to further dilute this vaccine so that one has, in addition to his stock bottle, a second bottle containing approximately 100 million organisms per cc. This will permit more careful control of dosage. If the desired fever is not attained by the first injection, and the temperature begins to fall within several hours, it is permissible, and often necessary, to give a second dose of typhoid vaccine from two to four hours after the first injection to boost the temperature to the desired level. This must be done cautiously, and only by one experienced with this form of therapy. Typhoid vaccine must never be given promiscuously intravenously to induce fever, as deaths have been reported as often as 1 per 1000 patients following such therapy. Such therapy may be repeated every four to five days for a series of from 4 to 6 treatments if beneficial results are obtained. Such therapy should not be given in general to patients with cardiovascular renal disease, hypertension, those who are markedly debilitated, or to individuals over fifty years of age except in very particular instances. The details of such therapy are outlined elsewhere in this volume.

Iontophoresis with mecholyl or histamine has no important advantages over other methods of physical therapy and the disadvantages attending its application preclude its widespread use.

Vaccine Therapy

1. No one stock or autogenous vaccine produces distinctly better results than another in patients with rheumatoid arthritis.
2. *Vaccines will not straighten out chronically deformed, ankylosed joints.*
3. There is as yet no proof that any one organism causes rheumatoid arthritis.
4. It has been suggested that subjective improvement following vaccine injections in arthritis results from its psychic effect.
5. Vaccines are no longer used in rheumatoid arthritis in many of the better arthritis clinics throughout the country. The question of desensitization to specific organisms found in foci in the patient's body, with attempts at immunization later, is still unsettled.
6. Although some optimistic reports are still forthcoming on the use of non-specific vaccines and of foreign protein therapy (such as typhoid vaccine, etc.) in rheumatoid arthritis, the value of these preparations is still to be determined and the reported results vary widely.

Massive Doses of Vitamin D.—*The value of massive doses of vitamin D in rheumatoid arthritis* is still open to question. Although encouraging results have been reported in some quarters, other clinics have not obtained any striking improvement with similar doses, or have reported discouraging results. The possibility of harm from calcification (renal, subcutaneous) from continued use of very large doses of vitamin D must be borne in mind. This is discussed in detail in a later chapter.

Fever Therapy.—(*a*) Fever therapy produces lasting improvement in only from 10 to 20 per cent of patients with rheumatoid arthritis.

(*b*) Short periods of fever therapy (thirty minutes per day) with temperatures up to 101° F. may be of value in rheumatoid arthritis but should not be used in very weak or elderly patients. This amount of fever may be induced by a fever cabinet, hot tub bath or blanket pack.

Endocrine Products

1. *Estrogenic substances* and *stilbestrol* definitely lessen the pains and muscular aches often associated with the menopause. However, there does not seem to be any definite value of these drugs in shortening the course of rheumatoid arthritis.
2. *Desiccated thyroid* substance should be given to those patients with marked lowering of the metabolic rates (hypothyroidism). Although this will ameliorate the symptoms of myxedema, it will not usually improve the arthritic manifestations to any degree except when the diagnosis has been in error. In these patients the joint pains are not due to rheumatoid arthritis, but solely to the arthralgia so often associated with hypothyroidism.
3. The *ameliorating effect of pregnancy upon rheumatoid arthritis* is well known (similar to the relief by obstructive jaundice); however, most of these patients have a return of their joint phenomena shortly after delivery (or after disappearance of the jaundice in the icteric individuals).
4. The relationship of the pituitary gland to bone and joint changes is under investigation at present.
5. Parathyroidectomy is no longer recommended in this country in the treatment of rheumatoid arthritis.
6. In an occasional diabetic patient with rheumatoid arthritis, the use of an adequate amount of insulin with proper dietary regimen is followed by a dramatic improvement in the arthritis; this must be studied further.
7. Adrenal cortical extracts have not proved of definite value in lessening the articular manifestations of rheumatoid arthritis. *Desoxycorticosterone* has been employed in the experimental production of arthritis.
8. *Testosterone* may be of value in decreasing the tiredness and lassitude in male patients (of middle age) with rheumatoid arthritis. The effect of the testosterone upon the rheumatoid arthritis is under investigation at present. In some patients this greatly increases the stiffness and discomfort.

Sulfur, Bee Venom and Chaulmoogra Oil.—(*a*) These are no longer used by most large arthritis clinics in teaching hospitals.

(*b*) There is no good evidence of sulfur deficiency or abnormal sulfur metabolism in rheumatoid arthritis.

(*c*) Any of the above may produce a slight foreign protein reaction followed by temporary improvement: there is no good indication for their parenteral use at present.

Outline of Therapy in Rheumatoid Arthritis

1. Adequate mental and physical rest.
2. Whenever possible, splint acutely painful joints in positions of physiologic rest; remove the splints daily and move the joints several times through a full range of painless motion. Prevent deformities—see text.
3. Physical therapy—especially the judicious use of heat, massage and exercises; see text. Occupational therapy may be very valuable. Properly given physical therapy should not cause pain. Never exercise a patient to the point of pain or fatigue.
4. A high caloric, high vitamin, anticonstipation diet in most cases (unless the patient is overweight).
5. Relieve pain by proper splinting of acutely inflamed joints. In the acute stages of the process, give (if necessary) aspirin and phenacetin, 0.5 gm. of each every three or four hours. Occasionally, it may be necessary to give codeine phosphate, 10 to 15 mg. ($\frac{1}{6}$ to $\frac{1}{4}$ gr.), every four hours in adults in the acute stages; *check the diagnosis if narcotics are required.* Aminopyrine and cinchophen are not recommended because of possible toxic reactions. Cobra venom has not been of value for relief of pain in rheumatoid arthritis.
6. Avoid cold, dampness and drafts.
7. Psychotherapy.
8. Orthopedic care—to preserve the function of joints or to restore such function if this has been impaired. Avoid manipulation of deformed joints if possible.
9. *Symptomatic treatment* is necessary: give attention to insomnia, fatigue, constipation, anemia, undernutrition, anorexia, associated menopausal symptoms, varicose veins, etc. See text.
10. Periodic vacations if possible, at well-equipped hospitals or spas.
11. Blood transfusions may be of value.
12. Remove or treat any definitely infected focus; see text for use of sulfonamides pre- and postoperatively. Mere removal of focal infection will not usually improve the arthritic picture greatly.
13. Gold therapy is still under investigation in rheumatoid arthritis. At present, most rheumatologists consider it a useful (but dangerous) adjunct in the treatment of these patients. The general practitioner should not use gold therapy unless he familiarizes himself with all of the details of this therapy, the dangers involved and the do's and don'ts of chrysotherapy. See text for details.
14. Periarticular injections of procaine may be of value in decreasing pain in some cases.
15. Roentgen therapy is of value in rheumatoid arthritis of the spine, but is not extremely beneficial in rheumatoid arthritis of peripheral joints.
16. Foreign protein therapy and fever therapy are occasionally of benefit in a small percentage of cases.
17. Residence in a warm, dry climate, with avoidance of storms, may be helpful in some cases.
18. Vaccines are no longer used routinely in rheumatoid arthritis in most arthritic clinics.

Outline of Therapy in Rheumatoid Arthritis—(*Continued*)

19. The value of large doses of vitamin D in rheumatoid arthritis is still open to question. It is not without danger.
20. Estrogenic substances, stilbestrol and desiccated thyroid should be used when needed for an accompanying menopausal syndrome or hypothyroidism respectively; the use of testosterone is being investigated.
21. Most large clinics no longer routinely use parenteral injections of sulfur, bee venom or chaulmoogra oil in rheumatoid arthritis.
22. The use of iodides and arsenicals is empiric. Their value is still questioned.
23. Mecholyl or histamine iontophoresis do not appear to be of sufficient value in rheumatoid arthritis to justify their use.
24. Dietary fads, high colonic irrigations, splenectomy, parathyroidectomy and thyroidectomy should be discouraged.

Outline of Treatment of Rheumatoid Arthritis of the Spine (Rheumatoid Spondylitis)

1. *Properly administered roentgen therapy is probably the most valuable single therapeutic procedure available to these patients;* in most early cases, it will relieve or abolish pain, improve motion and prevent the occurrence of deformities. Gold is of value in some cases,
2. Every effort must be made to maintain the patient's spine in an optimal position.
3. In most cases, *rest in bed on a firm hair mattress, with fracture boards under the mattress, is advisable for several weeks or months.*
4. Hot fomentations may be used on the entire spine twenty minutes twice a day.
5. Plaster body shells, lined with felt, may be needed to prevent or correct flexion deformity.
6. Flexion deformities may be lessened by placing a small firm pillow or blanket roll under the most prominent part of the spinal deformity for thirty minutes several times a day.
7. In some cases, it may be necessary to attempt (gentle) hyperextension of the spine under anesthesia, followed by the application of a plaster body jacke ; this may be repeated every five to seven days, with lessening of the deformity being achieved with each subsequent jacket.
8. Deep breathing exercises should be encouraged to increase the thoracic capacity.
9. When maximum correction has been attained, a waxed leather or a celluloid jacket may be worn for a year or more to maintain this.
10. Every effort must be made to prevent muscular atrophy during the period of inactivity by heat and massage, and by exercises for the gluteal, intrascapular and abdominal muscles.
11. If the hip or knee joints are involved in an acute process, it may be necessary to extend the cast to cover these areas and the whole leg, or in some instances to apply moleskin adhesive traction. Involvement of the hip joints is often a serious problem in these patients and demands additional attention.
12. If there is acute involvement of the cervical spine, traction by means of a head sling may be needed, followed by use of a felt collar or a Thomas collar.
13. When the active disease becomes quiescent, begin exercises with the patient sitting on the edge of the bed, and later in a chair; avoid muscular spasm.

Iodides and arsenicals have been employed empirically for years in rheumatoid arthritis. They are discussed in later chapters.

Sulfonamides have not proved of value in rheumatoid arthritis.

Penicillin has not been of value in rheumatoid arthritis. Several independent investigations have confirmed this.

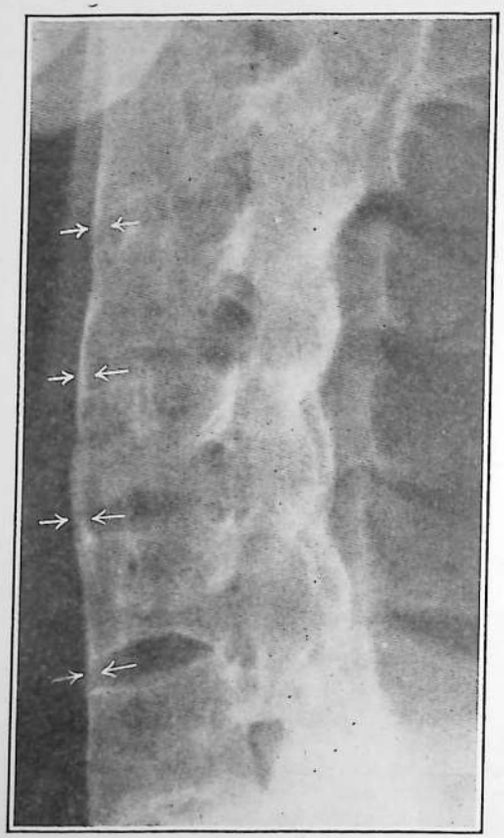

FIG. 43.—*Rheumatoid arthritis of the spine* with calcification of the anterior ligament in the cervical region.

Unfortunately, physicians occasionally become so enthusiastic with a special therapeutic agent that they neglect: (1) the patient's general health; (2) the patient's comfort; and (3) the prevention of deformities.

Improvement in rheumatoid arthritis is usually gradual; **do not expect dramatic changes in a short period** in these patients. **Do not allow the patient to become discouraged.** The physician must always remember that **there is at present no one drug or therapeutic procedure which is specific for rheumatoid arthritis**; and, that although he may wish to remove focal infection, to administer gold preparations, etc., during the entire period, the patient's program of rest, physical therapy, exercises and adequate caloric and vitamin intake must be maintained. One must not be content with merely

administering gold preparations once a week, but attention must be given constantly to the patient as a whole and to his joints.

One of the most constant worries in these patients is fear of progressive deformity and despair of cure. Inasmuch as more than 70 per cent can be greatly benefited (or have natural remissions of their disease), the *physician is justified in manifesting a degree of optimism and confidence to the patient concerning the outcome of his particular illness.* However, *such optimism must be genuine and not simply overenthusiasm with a promise of rapid cure.*

Patients, in general, appreciate being told that in about 20 per cent or more of cases of true rheumatoid arthritis, the condition appears to be amenable to therapy and that in an additional 50 per cent, moderate or marked improvement is the rule. This accounts for 70 per cent of these persons. An additional 15 to 20 per cent are definitely improved to some extent. *One should mention that in from 5 to 10 per cent of patients with moderately advanced rheumatoid arthritis, no form of therapy has produced noticeable improvement. The patient then realizes the entire truth of the situation; he knows that his physician is not a quack who is promising him a 100 per cent chance of cure of his disease.*

Most patients with arthritis have tried various home remedies, fads and patent medicines before a physician is consulted. They have come in contact with Mrs. X whose arthritis was cured (?) by a special diet consisting of the juice of 1 lemon daily, increasing each day by 1 lemon until 20 have been taken and then decreasing to 1. Many have heard of and have visited individuals whom the patients felt might cure arthritis by various manipulations. Some have worn special ivory bracelets while others have placed powdered sulfur in the shoes for twenty-one days. Almost all of these patients are constantly seeking new information concerning the treatment of their disease; medical and pseudo-medical articles in newspapers and magazines are eagerly digested and well remembered.

The physician handling patients with arthritis must be well versed in the literature of this disease. At the same time, it is necessary that he persuade the patient that, in the long run, his form of therapy is the one which is most likely to produce the greatest amount of improvement in the largest number of cases. **The Word Cure Should Not Be Used in Discussing Results in Rheumatoid Arthritis.** Very few of these patients experience what may be termed a complete cure of the disease; however, an arrest of the process is often obtained.

After the *physician* has convinced the patient that he has four chances in five of being improved, he *must also stress the point that the treatment will in all probability require several years, and not weeks or months.* It is true that in exceptional cases, extraordinary results may occur in a few weeks or one or two months; however, this is not the rule. If the patient realizes this at the onset of treatment, he will not experience the keen disappointment which might otherwise be noted following several months of treatment. *Results are usually measured by months rather than by weeks.* This cannot be emphasized too strongly. Although some patients may

disappear from one's office after one or two months of treatment (to take up some new "cure" [?] for this disease), most of these will return more strongly convinced than ever that the original physician knew whereof he spoke, and was doing all that was possible to produce improvement in his patient.

However, this does not imply that the physician is to be content to rest upon his laurels month after month if no improvement occurs. The diagnosis should be checked; it may be possible that the disease is not a true rheumatoid arthritis but may, in reality, be gout. Few practitioners realize that only 50 per cent of patients with gout manifest tophi in the early stages of the disease. The history, physical examination and laboratory findings should be checked frequently. If these are unchanged, the physician must persist with this treatment. Individuals who do not obtain marked improvement during the first several months are fortunate that the disease has not progressed and that contractures and deformities have not developed.

In general, it *remains for the physician to constantly buoy up the spirits and hopes of these patients to a key pitch*, not with false hopes of a rapid cure, but *with true enthusiasm of the results of careful, deliberately planned treatment.* In some cases it may be necessary and financially possible to send the patient on a vacation where he or she may be away from the cares of children, the constant ringing of the phone, the worries of the office, etc. The good results sometimes seen following well-managed spa treatment are due in large part to a change of environment with absence of worry and the beneficial effects of well-administered physical therapy.

Patients with rheumatoid arthritis are unable to obtain adequate hospital care; free patients with this disease are seldom welcome in the wards for more than four or five weeks. The major needs for the proper treatment of arthritic patients in this country include: (1) Better institutional facilities; (2) funds for research; (3) physicians well trained in this work; and (4) adequate instruction to medical students. Too little space is devoted to arthritis in the medical curriculum and in current textbooks of medicine. In one recent edition, although more than thirty pages are devoted to typhoid fever, arthritis is covered (?) in seven and a half.

CHAPTER XIII

SPLINTING OF AFFECTED JOINTS

All acutely painful joints should be immobilized (whenever possible) by molded splints; the latter are removed daily and the joints are moved once or twice through a full range of painless motion (varying with the tolerance of the patient), following which the splints are reapplied. If acutely inflamed joints are placed in positions of physiologic rest at the onset of an arthritis, marked improvement may be noted. *Motion aggravates acute arthritis. Absolute rest of the part permits the formation of adhesions.* There must be sufficient motion to prevent ankylosis and sufficient rest to permit healing. If this principle were adhered to, physicians and patients would be gratified by the good results obtained.

Molded Plaster of Paris Splints.—Molded plaster of Paris splints are usually advisable. These should be light, but thick enough to prevent breaking of the splint. The plaster may be applied over stockinet and felt padding which has been placed on the affected limb. Where facilities are limited or for use in the home, strips of impregnated plaster will prove useful. These are sold already cut in various lengths, which need merely be immersed in water, quickly withdrawn and applied to the part involved. These are available in several different sizes—3 x 15, 4 x 15 and 5 x 30 inches, in boxes of 50 sheets each. They are relatively inexpensive, a box of 50 of the largest size costing approximately $2.50. From 7 to 15 layers (or sheets) may be needed for each splint depending upon the joint involved. It requires not more than a second or two for the splints to become saturated in water; the plaster sets within eight to ten minutes.

When Applying Plaster:

1. Pad all sharp, bony prominences which may be covered: malleoli, anterior-superior spines, crest of the ilium, knee, etc. If the patient is thin and the spinous processes are prominent, use padding over these.
2. If all prominences are padded, additional padding elsewhere is unnecessary.
3. If the ribs flare, these must be padded.

The "Major" plaster bandage may be used for strong, light-weight casts and has the advantage that it may be bivalved very easily by the general practitioner by applying hot water from a syringe along a scored line. The plaster is disintegrated by the hot water and the crinoline may be cut with bandage shears, leaving a neat edge; no plaster is lost in the removal. These plaster bandages come in various sizes including 2 inches x 3 yards, 3 inches x 3 yards, 4 inches x 5 yards, 5 inches x 5 yards and 6 inches x 5 yards, ranging in prices from $1.45 to $3.05 per dozen rolls.

Care must be taken that bony prominences are well padded to prevent pressure. Inasmuch as the plaster is not being used as a cast, but merely as a splint permitting daily inspection of the affected part, no pressure points should appear.

The plaster of Paris bandage was invented by A. Matthysen, a medical officer in the Dutch Army in 1852. Plaster of Paris is still the most universally acceptable cast material available.

Plaster of Paris, or anhydrous calcium sulfate, exists in underground deposits as gypsum, or hydrated calcium sulfate, which is a solid crystalline material. Gypsum is heated to 120° C. to remove most of the water of crystallization; impurities such as silicates, iron and carbonates are then removed and a proper accelerator is added to reduce the length of the setting time.

When soaked in water, plaster of Paris takes up water and reverts to the solid crystalline state with a rapid formation and interlocking of crystals. Much of the ultimate strength of the plaster is derived from this tight interlocking of the crystals. *The plaster must remain undisturbed during most of the setting process so that this interlocking of crystals occurs properly.*

After the plaster sets, the excess water disappears by evaporation. This requires a few hours in thin casts or days in thick ones. The rate of evaporation varies with the temperature and humidity of the air and its circulation around the cast. Some casts may appear dry on the surface and yet contain a considerable amount of water in their deeper layers. *Heavy casts may remain wet and weak for days in wet, humid climates* so that in these areas, a cast drier with free circulation of air around the cast is advisable.[11]

Defective plaster casts may have been weakened by having been bent or compressed following the critical point in the setting process. If the application of the cast is too slow or if the setting time of the plaster is too rapid, movements may occur in the cast long after the critical point. *If the position of the cast is changed after it has been applied, the cast may be weakened* by disrupting the setting process and pressure points and wrinkles may be set up inside the cast. In general, the *critical point* is that time when the plaster is about the consistency of thick cream and begins to lose its glistening wet character; the critical point has been passed when the plaster has become dull and the cast has become firm. *In an analysis of defective casts,* Luck found that the *most common single cause of impairment was manipulation after the critical point.* This is particularly important when large casts are made, such as body casts and shoulder and hip spicas. In these cases, the patient should not be moved too soon after the cast has been applied. A break or bend in the cast frequently occurs when the patient is moved onto a litter or turned in bed. If a break or bend occurs in the cast after this has become solid, it may be so weakened that it is useless.[11]

Thick padding beneath a cast is another frequent cause of a weakened cast. Such padding usually necessitates extensive molding and heavy rubbing,

which, if prolonged, can greatly weaken the cast. In most instances, only a thin layer of padding is indicated. If thick padding is used, there is a great tendency to apply the plaster bandage under tension, which is dangerous; **plaster bandages should not be drawn on, but should be laid on.** Thick padding also increases the opportunity for friction to occur between the cast and skin so that painful points may occur more often by friction in a thickly padded cast than by pressure from casts with little or no padding.

The layers will fuse to form a solid cast if plaster bandages are applied in rapid succession. When one layer of plaster-crinoline dries before another is applied, lamination of the cast occurs with non-fusion. **If it is necessary to wrap fresh plaster over a dry plaster surface,** one should first roughen the dry surface by scratching this with a sharp knife.

It is unwise to make wedges or large windows in green casts if this can be avoided. Wedging a green cast results in compression at that site and possible bending or breaking of the cast.

Good plaster technic includes use of a minimum of plaster, rapid application of the cast and protection and comfort to the patient.

In most instances, one should avoid the use of salt or borax to accelerate or of sugar to retard the setting time. *The setting time can be retarded effectively,* if needed, *by cold water,* while warm or hot water will accelerate this. One can usually regulate the setting time by varying the length of the soaking time and the temperature of the water. One usually employs a soaking time of about a minute in water at a temperature of from 70° to 95° F.

The ultimate strength of the cast will be greatest in those which are completed early in the setting process and kept quiet until this has set.

The plaster bandage should not be squeezed immediately on removal following soaking as this presses much of the plaster out of the bandage; proper technic includes the gentle removal of the wet bandage from the bucket and allowing this to drain for ten seconds or more into another bucket before gently compressing out the excess of water which remains. The rolls are drained outside the soaking bucket as any plaster of Paris which accumlates in the soaking water tends to accelerate the setting time. It is good plaster technic to change the soaking water after each six bandages.

It has been shown that when plaster splints or slabs are used alone (*i. e.,* without incorporating these into a cast), thin and broad splints should be used and should be placed on one or both *sides* of joints such as the knee and elbow (joints moving predominantly in one plane). This is just the opposite of the use of plaster splints when they are used to reinforce casts encasing such joints (as the knee, elbow, wrist or ankle).

It has been shown by calculation that it is possible for lateral splints to be sixteen times as resistant to bending stresses as similar splints which might be placed anteriorly and posteriorly.

If one desires to reinforce casts that encase the aforementioned joints, the reinforcing splints will contribute more to the strength of the cast if they are narrow and thick; these splints should be placed on the front and

back (and not on the sides) when being incorporated into plaster casts. Reinforcement for hip spicas should be placed with the strongest reinforcement laterally but with reinforcement also anteriorly and posteriorly; reinforcement in shoulder spicas should be over the top of the shoulder and in the axilla.

Application of Plaster Splints.

A. If one desires to **splint the knee joint,** the simplest method for the general practitioner is as follows:

1. The patient lies on his abdomen.
2. A thin coating of petroleum jelly is applied to the thigh and leg.
3. Plaster strips are wet and are then placed over the back of the thigh, leg and plantar surface of the foot. This may be built up until from 7 to 15 layers of plaster gauze are applied, as needed. If rolls of plaster are used, these may be cut into strips of the desired length, or rolled onto the leg wet.
4. The plaster is allowed to dry.
5. The splint can then be lifted away easily because of the petroleum jelly coating.
6. The splint may be placed on a radiator to dry more quickly. Splints may be made much lighter than usual if these are allowed to harden completely before using.
7. The splint may then be coated with cellulose acetate, shellac or varnish; these are useful in waterproofing the cast.

B. **Splinting of the lower leg and ankle.**

1. The patient should lie prone (on his abdomen).
2. The ankle may be kept in the ideal attitude by having the toes hang over the edge of a table or litter.
3. Petroleum jelly is applied to the lower leg and foot and the strips of wet plaster are then applied. Each layer is placed so that it partly overlaps that one next to it.
4. Plaster strips may be purchased in varying sizes, or the physician may cut these from the rolls of plaster. If he desires, he may form his plaster splint by wetting the roll of plaster and rolling this back and forth on the back of the leg and sole of the foot.
5. The plaster splint is removed when firm, and dried over a radiator; this may then be waterproofed by coating with cellulose acetate, shellac or varnish.

C. The technic, as described for splinting the knee joint may also be used to make a **molded posterior body shell.** When a large shell is to be made, numbers of criss-cross plaster strips should be placed, especially about the hips and buttocks, where strain is to be expected.

D. For the **hands and fingers,** three to seven thicknesses of plaster gauze can be held against the hand and forearm which have been coated previously with petroleum jelly; these are held in the desired position until hardened. They are then lifted away and dried on a radiator and waterproofed. In making plaster splints for the hands and fingers, the plaster is applied to the flexor aspect of the forearm and palm, usually leaving the thumb out of the splint.

It is sometimes difficult to remove snugly fitting casts. Wilson has suggested a simple method of applying plaster casts in order to provide for easy removal. The part is enclosed in stockinet or wrapped with sheet cotton as usual. A strip of piano felt (about 2 inches wide and long enough to extend through the entire cast) is placed on the anterior surface of the part, for example from the toes to above the knee, if a cast is being made for the lower leg. On top of this is laid a thick rubber strip, such as a fan belt (from an automobile), which has been cut to produce a long strip; the widest surface of the fan belt makes contact with the felt. The strip of fan belt is rubbed with paraffin so that it does not stick to the cast, when applied. Two strips of plaster bandage are placed, one on either side of the rubber strip, in order to cover the felt and to restore a rounded contour. The circular cast is then applied in the usual manner. While the plaster is still wet, and as soon as it has set, the rubber strip is pulled out, one hand being placed over the instep to eliminate the danger of breaking the cast.[28]

There remains a finished circular cast with a grooved inner surface extending throughout it. Inasmuch as the margins of the groove are padded, no discomfort results to the patient. The groove readily admits the lower jaw of the usual cast cutter so that the cast can be removed easily. The rubber strip is removed each time so that it may be used again and again in further casts.

Waterproofing the splint results in a cleaner, firmer and harder splint and prevents moisture and perspiration leading to deterioration of the splint. When one employs cellulose acetate or a solution of celluloid in acetone to coat the splint, one can make lighter splints because these substances give the splints greater strength.

Cellulose Acetate for Covering Plaster Casts.—This solution was devised at the Children's Hospital School in Baltimore; it has proved very satisfactory for waterproofing plaster shells.[12] To obtain strength in light shells they are made next to the skin, using 6 to 8 layers of plaster. After they have taken the shape of the extremity they are removed and placed on a radiator for twenty-four hours. At this time they are painted with the Cellulose Acetate Solution.

Material	*Company and address*
Cellulose Acetate (Lacquer Viscosity 5)	Celanese Corp. of America, 180 Madison Ave., New York, N. Y.
Dimethyl Phthalate A	Kay-Fries, 180 Madison Ave., New York, N. Y.
Acetone: U.S.P.	

Formula

⅔ jar cellulose actate (2 qt. jar)
3 oz. sizing (Dimethyl Phthalate A)
Acetone to fill jar

Stir occasionally to assist in dissolving the cellulose acetate. Before applying cellulose acetate, add enough acetone to make solution slightly thinner than when first dissolved. Apply the solution as paint, using ordinary paint brushes. Three coats are usually used and each must be allowed to dry completely before applying the next coat. The cast must be dry before the cellulose acetate is used. If the atmospheric humidity is high, the cellulose acetate will absorb water and turn white. It will later dry out, but may not be completely satisfactory. For this reason it is better to apply the solution on clear, dry days. It is best to prepare the solution a day before using it.

Celluloid or Cellulose Acetate Splints.—If such light splints are desired, they can be made as follows: The part to be splinted is coated with petroleum jelly and a piano or other wire is laid longitudinally. Layers of plaster are applied over this forming a light plaster casing. When this has hardened, the wire is pulled taut and the plaster is cut along this wire, enabling one to remove the plaster without danger of cutting the skin. The plaster mold can be removed from the part and closed. This hollow mold is then filled with a mixture of plaster and water, or plaster, sawdust and water so that a cast of the leg is made within the mold. When this has hardened, the outer mold is removed and one is left with a cast of the part. Surgical stockinet is then pulled over the plaster cast of the part and this is painted with celluloid or cellulose acetate mixture. When this coat has hardened, a second is applied. Another stockinet is then applied over this and several more layers of celluloid or celluloid acetate are painted on. This is repeated until the splint is sufficiently thick (usually 2 or 3 layers for the extremities and 4 to 8 for body jackets). When this has dried, one may cut it longitudinally and remove the newly made celluloid jacket or leg cast. Because of the resiliency of the material, the cut edges can be spread and the cast may be placed on the part at will. A shoemaker or surgical supply store can sew leather strips on to each side of the longitudinal cut edges. If eyelets are placed in the leather strips, the celluloid jacket or leg cast may be laced as desired.[14]

The Plaster Bed.—Tippett has used the plaster bed or plaster shell for patients with generalized joint involvement.[25] With the patient lying on his face (and the mattress supported by fracture boards), the limbs and back are covered with olive oil and plaster bandages are applied (about twenty 8-inch bandages), starting at the sole of the foot and continuing layers of plaster up the leg, back, shoulders and neck and sometimes over the scalp and forehead (after protecting the hair by a rubber bathing cap) until a layer approximately ½ inch thick is obtained. This includes some criss-cross plaster layers. After the plaster has set, the shell is lifted off carefully, dried near a radiator or fire for several days, and is then used by the patient. This large plaster shell is unnecessary if only one or two limbs are involved.

If the knees and hips are involved, such a shell may be made extending to the mid-back. It may be stabilized by a wooden cross-bar incorporated into the shell, joining the lower portions of the shell at about the ankles.

Light Plaster Body Cast.—If a light body plaster cast is desired, it should be split while damp (but after it had thoroughly hardened); it is then allowed to dry (over a radiator or under a baker) and may be coated with celluloid or cellulose acetate.

Whenever a body cast is desired, reinforcements of additional plaster strips must be placed longitudinally down both sides and along the back, which are then incorporated in the circular plaster rolls around the body.

Castex Splints.—Castex splints have been used by some physicians. This material utilizes a synthetic resin and is an aid in the treatment of arthritis and various orthopedic conditions, including fractures. The

Castex is, on the average, *only one-third as heavy as a plaster* splint of equal strength. *The former is waterproof and is pervious to roentgen-ray.* With plaster splints, roentgenograms cannot be made without removal of the splint.

This type of waterproof cast was introduced in 1939 by Thorndike and Garrey[24] of Boston. These bandages (moistened with a quick drying solvent) are supplied in various widths in 10 yard lengths; the bandages are rolled, tinted unbleached cotton sheeting cut on the bias. The cellulose compound which is used to impregnate the cotton sheeting is pyroxylin ("airplane dope") in acetone.

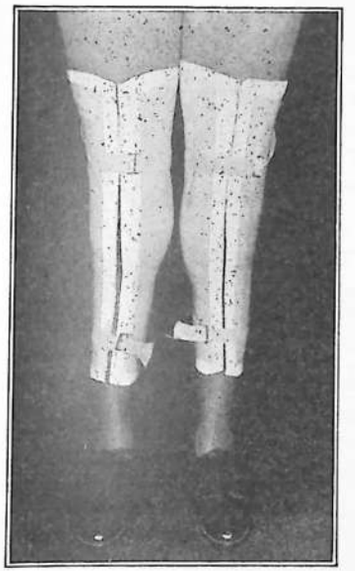

FIG. 44

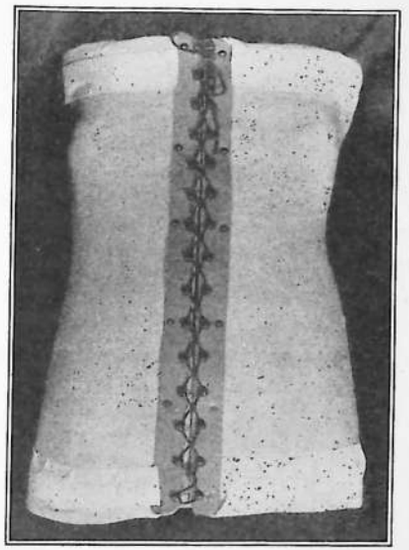

FIG. 45

FIG. 44.—Anterior view of a pair of cellulose-compound (Castex) walking cylinders. Note the neat appearance which is made possible by the elasticity of the material which permits it to conform to the parts to be protected. (Joplin, courtesy of Jour. Bone and Joint Surg.)

FIG. 45.—Cellulose compound jacket with a strip of hooks braided on either side to facilitate lacing. (Joplin, courtesy of Jour. Bone and Joint Surg.)

Disadvantages of Castex.—(1) The disagreeable odor of acetone (until the bandage is dry); (2) a longer setting time than plaster (usually requiring from fifteen to thirty minutes); (3) shrinkage of the cloth on drying (applying the bandages without stretching minimizes this); (4) inflammability—*the bandage is inflammable when wet* but much less so than collodion or ether; when dry, it is no more inflammable than wood; (5) high cost.

Advantages of Castex.—(1) Ease of application; (2) lightness; (3) permeability to roentgen-rays; (4) waterproof; (5) easily repaired if cracked; (6) small quantity of material needed (so that strong splints are extremely thin).

In addition to the Castex bandage, a lacquer is available for use on the surface of the cast after the latter has dried; this is useful in adding a finish to the cast, sticking down any rough edges and producing a smooth appearance. The use of this lacquer is not essential to the waterproof, durable nature of the cast.

Application of Castex Splints.—Cover the part to be splinted with orthopedic stockinet (which can be purchased in various widths, depending upon the size of the part to be splinted; this costs approximately 25 cents per yard length).

Wear rubber gloves or cover the hands with a coating of petroleum jelly or boric acid ointment; otherwise, a collodion-like film will be deposited upon the hands.

Do not open a can of Castex until ready to use; open only one can at a time as the material begins to harden on exposure to air. When the can has been opened, the bandage should be applied immediately.

Castex should not be brought near an open flame as it is inflammable until thoroughly dry.

The Castex bandage is ready to use without the addition of any further substance. **Use practically no tension in its application because shrinkage of the cast occurs**; the Castex must never be stretched. Apply the bandages smoothly.

In most instances, 3 spirally wound overlappings form an adequate cast; the edges must overlap so they will fuse together. These 3 layers will form a cast from $\frac{1}{8}$ to $\frac{1}{4}$ inch thick which has greater strength than a plaster cast almost three times as thick.

If it is desired to build reinforcements into the cast, horizontal splints of this material may be placed wherever desired between the first and subsequent spiral windings.

The Castex becomes rigid in from fifteen to thirty-five minutes depending upon weather conditions and the amount of padding or stockinet used beneath it. If it is desired to produce rapid drying, warm air can be circulated around the cast with a heat lamp or electric fan. Expose the cast to the air until completely dry.

In most cases of arthritis, the operator desires to remove the cast or splints daily. Ordinary surgical scissors cut this material if only 3 layers have been applied. Because of the resilient nature of the Castex, the cast is easily spread and removed when it has been cut lengthwise; when reapplied it assumes its original shape. Because of shrinkage which may occur, it is advisable to leave the Castex cast in place for at least several hours (and preferably a day) before cutting this. This prevents turning under of the cut edges and other untoward contractions in the material which might occur otherwise.

When the cast is reapplied, it may be held firmly in place by webbing straps and buckles or by small strips of adhesive tape. This gives Castex an additional advantage over plaster of Paris, the bivalving of which requires two cuts; plaster lacks the resiliency of Castex.

If desired, metal braces, reinforcements, etc., may be incorporated into the cast.

The Castex lacquer may be applied (with a brush or with the gloved hand) to the outer surface of the cast when this has dried, in order to give the cast a more finished appearance and a smoother surface. The lacquer is also useful in making minor repairs in the casts or as a cement in securing the straps and fittings. For those individuals who are mechanically inclined, a zipper may be incorporated into the cut edges of the cast so that it may be fastened readily.

Light Aluminum Splints.—Koch and Mason[9] have detailed the making of light aluminum splints for the hand. They employ sheet aluminum, 0.064 inch thick, designated as No. 2S $\frac{1}{2}$ H. This is rigid enough to maintain any desired shape and light enough not to be cumbersome. By means of a few tools (snips, hammer, jigsaw, bending irons, file, sandpaper, small drill, rivets and a vise) splints can be prepared promptly for many purposes.

Leucite Splints.—The Du Pont preparation, *leucite*, may be used as a splinting agent. Thin strips of this colorless smooth substance may be softened in warm water and quickly molded to the affected part; the substance hardens readily on cooling. The material is very light, clean and possesses some obvious advantages.

Tenite Splints.—Tenite (produced by Eastman Kodak) is a material which softens at the temperature of boiling water and molds well. This may also be used for splinting.

Elastic Bandages.—When an individual is ambulatory, elastic bandages are a convenient form of additional support to involved joints. The familiar "Ace" and similar elastic bandages are an economical method of applying light additional support for involved joints. These are especially useful in involvement of the knee and ankle joints. The pressure may be adjusted by varying the tightness of application of the bandage, but edema of the extremity should not result. These bandages have the advantage of being washable without impairing the elasticity of the cotton material. They are available in widths of 2, $2\frac{1}{2}$, 3, $3\frac{1}{2}$, 4, 5, 6, 8 and 10 inches; all are $5\frac{1}{2}$ yards long. When Ace bandages are used for women, a special silk and cotton flesh tinted, rather inconspicuous bandage with woven edges (known as Ace No. 7) may be used. This is made in widths of $2\frac{1}{2}$, 3 and 4 inches. The Ace ankle roller ($2\frac{1}{2}$ inches wide and 3 yards long) in white or neutral flesh color may be used where the longer bandages are inconvenient. One may also employ elastic knee caps or anklets or laced elastic knee supports with or without lateral hinge joints incorporated into the support.

In patients with varicose veins or mild phlebitis of the legs, *elastic adhesive bandages* (such as Elastoplast or Ace No. 10 adhesive bandage) may be applied from the foot to the upper portion of the thigh. Bony prominences and the under surface of the knee must be protected with adequate padding. Such bandages may be left in place two or three weeks unless irritation of the skin occurs. If the leg is painted with compound tincture of benzoin prior to the application of Elastoplast, much less itch-

ing or irritation of the skin will occur. In many overweight women with large legs, pain in the knees is greatly reduced by this procedure. If marked improvement is noted, a heavy elastic two-piece stocking may be applied to each leg (from foot to upper thigh) when the elastic adhesive bandage has been removed. Such stockings may be washed; their cost ranges from $15 to $20.

Gelatin Boots.—The gelatin or Unna boot may be applied in the conventional fashion with the heated Unna paste and gauze. The conventional or already prepared gelatin gauze bandages are often useful in phlebitis and low grade inflammatory processes in the legs.

One may purchase gauze bandages already impregnated with a gelatin paste containing zinc oxide, kaolin and calcium chloride (such as Cruricast, produced by the E. K. Demmel Company, Brooklyn); this is ready for use coming in a width of 4 inches and a length of 10 yards. One package is usually sufficient to prepare a gelatin boot for a lower leg. Gelatin boots are useful in some cases, especially where one hesitates to employ adhesive or Elastoplast to the thin delicate skin with varicose veins in patients who may be sensitive to adhesive. After the ready prepared gauze bandage impregnated with the gelatin paste has been applied to the leg, overlapping each layer about half way over the preceding one, the whole bandage is covered with 1 or 2 layers of plain gauze until this hardens. A formalin solution is also supplied which may be sprayed on to the gelatin boot to make this harder.

RELIEF OF MUSCLE SPASM

It has recently been suggested that the muscle spasm in rheumatoid arthritis may be relaxed by the use of prostigmin.[22] Kabat and Knapp previously reported encouraging results with prostigmin in the subacute and chronic stages of poliomyelitis.[8] These investigators believed that prostigmin had an action on the spinal cord.

Trommer and Cohen observed relaxation of muscular spasm in 13 of 19 patients with rheumatoid arthritis following administration of prostigmin; although prostigmin did not relieve the pain produced by the joint pathology, muscular spasm was relieved even though this had persisted for some years, so that motion could be carried out more easily and with less pain in the affected joints.

As a general routine, if relaxation of muscular spasm is desired, prostigmin methylsulfate is given subcutaneously in doses of 1 cc. (of the 1:2000 dilution), together with atropine sulfate (0.6 mg.). Atropine is given together with the prostigmin in an effort to prevent any undesirable effects which the prostigmin may produce by stimulation of the myoneural junctions of the parasympathetic nervous system. The injections may be given three or four times a week and should be supplemented by prostigmin given orally in doses of from 15 to 30 mg. of prostigmin bromide three times a day. When the prostigmin is given orally, it is advised that it be accompanied by tincture of belladonna, in doses of from 0.2 to 0.4 cc. three times per day.

In some cases of rheumatoid arthritis, one may notice relaxation of muscular spasm in from five to fifteen minutes after subcutaneous administration of prostigmin. It has been reported that such relaxation may persist for several days.

Although prostigmin is effective in relieving muscular spasm in some cases of rheumatoid arthritis, no one knows exactly how it exerts its beneficial action. Trommer has suggested that it may act directly upon the central nervous system.

It is of some interest that in the experiments reported in the literature with prostigmin, atropine is given concomitantly (in an effort to prevent untoward effects from the prostigmin). Some patients have noted definite lessening of stiffness and muscular spasm following the administration of tincture of belladonna alone (which may have been given because of gastrointestinal disorders). Following the injection of atropine, patients also have described a definite relief of muscular spasm. Work is in progress at the present time to determine what rôle the atropine may play in decreasing muscular spasm and whether some of the benefit attributed to prostigmin may have, in reality, been due to atropine. In this connection, one is reminded of the lessening of the stiffness in Parkinsonism by drugs closely allied to belladonna (hyoscine, stramonium and others). The action of these latter drugs is also being studied at present to determine if they will relax the spasm found in rheumatoid arthritis.

PREVENTION OF DEFORMITY

It is usually much easier to prevent a deformity in arthritis than to correct one. Correction of deformities belongs to the realm of the orthopedic surgeons; fortunately, these men have been able to perform "miracles" with some of the "wrecks" which have been thrust upon them at times. However, *it is the duty of the general practitioner to prevent the occurrence of these deformities* where this is possible. The average deformities in most arthritic patients can be prevented. If it is evident that deformities will occur in any given case, it is essential that the part be held in a position in which optimum function of the part is permitted.

During the early stages of an arthritis, *deformities result usually from muscular spasm* with muscular strain and traumatic joint irritation due to faulty body mechanics. *Such deformity is ordinarily in a position of flexion* and often adduction because this affords the patient the greatest relief from pain and because of the more powerful action of most flexor and adductor groups of muscles over the corresponding extensor and abductor groups. Other factors which favor muscular contraction and aid in the development of deformities include pressure of the bed clothes, a sagging bed, muscular strain, static influences and gravity. If the patient with rheumatoid arthritis lies in bed for long periods without proper care, extensive muscular and bony atrophy and deformities will occur. Several pillows under the head may force the cervical and dorsal spine into forward flexion. A soft mattress and sagging spring may produce increased flexion

of the thoracic spine with strain on the posterior spinal ligaments and compression of the anterior parts of the vertebral bodies and intervertebral structures.[16] Other untoward effects include straightening or even kyphosis of the lumbar spine resulting in additional strain on the posterior spinal ligaments and muscles and fascia of the back, flexion of the hips with contraction and shortening of the powerful ilio-psoas muscles and Y ligaments of the hip joints, hyperextension of the knees, straining of the posterior muscles of the thighs and calves and the posterior part of the joint capsule.

Resting in a chair for long periods is not advisable for many arthritic patients as the position assumed leads to flexion contractures of the spine, hips, knees, elbows and wrists with internal rotation and adduction of the shoulders and pronation of the forearms.[2]

One of the main principles in preventing such deformity is to *inhibit muscular spasm.* If this is not accomplished promptly at the onset of the disease, actual shortening occurs in the joint capsule and in other structures about the joint on the side of maximum contracture, and fibrous or bony adhesions may occur.

In cases in which there is a possibility of ankylosis, the parts should be maintained as much as possible in positions which would give the patient maximum function of the ankylosed part.

Positions of Election if Ankylosis is Inevitable[3,15,17,18,19]

Knee: A few degrees of flexion (10° to 15°).
Ankle: The foot should be dropped 10° to 15° beyond the right angle; very slight pronation is also sometimes advisable.
Hip: Slight flexion (20°) of the hip with the leg practically straight; in neutral position or even slight (5° to 10°) adduction and slight external rotation.
Shoulder: Not more than 45° between the humerus and the posterior border of the scapula; forward flexion (30° to 45°) and rotation of the arm in slight supination (10° to 15°), so that the hand will reach the mouth.
Elbow: Depends upon the patient's occupation and wishes; in general, approximately 70° to 80° of flexion is usually best with forearm in slight supination (10° to 15°).
Wrist: About 30° of dorsiflexion (*i. e.*, extension).
Hand: Semiflexion of the fingers and metacarpophalangeal joints; adduction of the thumb.
Jaws: Open $\frac{1}{4}$ inch.[4]

Most deformities can be prevented if the physician has a thorough understanding of the problem at hand and if the patient is coöperative. It has been stated that "*Correction of deformity should be labeled correction of mistakes made by the doctor and patient.*"

These four principles will, in general, be the physician's best safeguard against the occurrence of deformities. The use of plaster or Castex splints has already been commented upon in detail. These splints should be applied to the joint in a position such that if ankylosis does occur, maximum function will result (*i. e.*, in the position of election should ankylosis

be inevitable). The application of these principles to individual joints is as follows:

Prevention of Deformities in Arthritis[5,20,21]

Physiologic rest of the involved joints by:
Rest in a non-sag bed (in acute arthritis) using a firm mattress, under which fracture boards have been placed; periods of special postures.
Splinting of the affected joints (by the use of casts, splints or other apparatus) to prevent trauma, weight bearing or strenuous use.
Movement of all joints through as wide a range of painless motion as possible one or two times a day.
Special graded muscular exercises for each joint involved, with periods of rest following such exercise.
Maintenance of good posture (see section on orthopedics).

The Knee.—The knee is the largest joint in the body and is frequently attacked because it is a weight-bearing structure. Atrophy of ligaments and muscles about the joint may appear rapidly. Common findings include narrowing of the joint space, fixation of the patella against the femoral condyles (often with fusion), subluxation of the tibia on the femur, and contracture of the joint capsule, hamstring muscles and periarticular structures.[7]

In acute arthritis involving the knee, the patient should be put to bed at once and a posterior molded splint made for the involved limb extending from the upper portion of the thigh to the ankle. In most cases it is advisable to include the foot so that foot drop will not occur (from pressure of bed clothes, etc.). The knee is splinted in a position approximately 10° to 15° less than full extension, with the foot held in 90° of flexion. This splint is worn day and night in acute cases, but is removed at least once every twenty-four hours to permit several motions of the joint (to prevent adhesions) and to permit the use of hot fomentations which aid in lessening pain and muscular spasm.

One of the fears of the patient with acute joint involvement is excruciating pain which may occur on slight motion of the joint. With the joint held firmly in a splint or bivalved cast, movement of the joint is impossible. He is thus relieved of pain which otherwise might occur with every movement of the knee.

It is essential that the splint or cast be removed daily and the affected joint be inspected. Any points of pressure from the cast are searched for and if found, additional padding is applied or the proper adjustments made in the splints. The knee is moved through as great a range of painless motion as possible. This is done by the patient and the motion is slow, easy and deliberate. One or two full motions of the affected joint usually suffice to break up any adhesions which have formed; not more than 1 or 2 full motions daily are advised during the acute stage. *Any joint adhesions forming within twenty-four or forty-eight hours can usually be readily destroyed by several movements of the joint.* If active movements (by the patient him-

self) cannot be performed, the physician must slowly move the joint through as great a range of motion as possible one or two times without causing the patient undue pain. Following this, the splints are reapplied. As long as joint pain, tenderness and swelling persist, it is advisable that splints be used for the affected joint during at least part of the day or night so that muscular spasm will not cause contracture deformities.

Prevention of Flexion Deformity of the Knees

Never place pillows under the knees for relief of pain in these patients; this is the commonest cause of such a flexion deformity.

Use a posterior molded splint or cast extending from the upper part of the thigh to the toes with the knee in about 10° of flexion. Make the splint with the patient face down; if desired, the plaster bandage can be placed directly on the non-hairy leg (after this has been coated with petroleum jelly) as plaster may be washed off easily. The plaster splint may be sandpapered inside if required.

Incorporate a tongue depressor into the leg splint posteriorly at the ankle to control rotation of the hip joint.

Remove the splint once daily and allow one or two active (or if necessary, passive) movements (short of severe pain); this prevents the formation of dense adhesions.

Apply hot fomentations to the knee twenty to thirty minutes several times a day.

Employ muscle setting and non-weight-bearing exercises when the acute inflammation has subsided.

With improvement, lessen the period of splinting gradually so that within a short time, splinting may be required only at night and several hours during the day.

Even in the later stages of the process, permit the splints to remain in place all night as flexion of the knees is common during sleep (unless prevented by splints).

Do not permit weight bearing until the acute inflammation has subsided entirely and a period of exercises without weight bearing has produced no harmful effects; the leg muscles must be in a condition to support this weight. **The patient must not walk on a flexed knee joint,** *or he will produce distortion in both lower extremities and in the trunk.* He should not be allowed to walk until he is able to hold the extended leg horizontally while sitting. During the first few weeks or months of walking, it may be advisable to use a stiff-legged brace, an elastic (Ace) bandage or a cane. A metal or wooden walker is of great assistance to these patients when they attempt weight bearing.

Begin **massage** of the muscles (excluding the affected joint) within twenty-four hours of the period of original splinting except in acute arthritis. This should consist of light upward stroking movements directed at the calf and thigh muscles in an effort to improve the local circulation. This must be done daily and should be increased gradually, beginning with 10 or 15 upward movements to the calf and to the thigh daily; the area of the affected joint must not be included in the massage in acute cases.

The commonest deformity of the knee is marked flexion, subluxation and outward rotation. The cause of this flexion deformity is the desire of the patient to relieve the pain in the acute stage of the disease. Flexion of the knee is the most comfortable position when swelling is present as it lessens the pull of the hamstring muscles on the posterior portion of the capsule, thus relaxing the latter, and permitting additional room for the swollen tissues of the joint. Most of these patients initiate the flexion deformity by placing pillows under the knees to relieve discomfort. Other individuals bend the knees while lying on the side. As the disease progresses further flexion is required for relief of pain and the deformity grows progressively worse. Because of this continued flexion of the knee, actual shortening occurs in the hamstring muscles so that the weight of the leg rests upon only the heel and the hip. With loss of support for the tibia, this latter drops down or back upon the condyles of the femur and subluxation occurs. This is due, at least in part, to contracture of the hamstrings. Outward rotation of the tibia on the femur usually follows within a short space of time. The prevention of such flexion deformity of the knee rests in general with the practitioner.

When correction of deformity of the knee has been attained in a bedridden patient by Buck's extension or serial plaster splints or casts (with or without wedging or the use of a turnbuckle) and one is ready to begin weight bearing, support and protection must be given because of the weakened quadriceps muscles and other periarticular structures. During the period of rest in bed, exercises and massage must be employed to strengthen the quadriceps muscles. In severe cases, it may be necessary to use metal leg caliper braces, with leather casing at the thigh and leather support over the patella, the metal uprights fitting into appropriate receptacles in orthopedic shoes. The braces may be locked at the knee when the patient is up and about keeping the leg straight. The lock may be opened permitting bending of the knee when the patient sits down for short periods.

In other cases, plaster or Castex cylinders (with a hole for the patella) may be employed to support the knee joint until the quadriceps muscles have become stronger. If the quadriceps muscles are strong, one may employ a laced elastic knee support with or without metal hinge joints, or simply elastic bandages with a strip of felt incorporated on either side of the patella for additional support. Occupational therapy and proper exercises, heat and massage help to strengthen the atrophic muscles.

Ankle and Foot.—The commonest deformities in arthritis involving the feet are the stiff, pronated or flat feet and deformities of the toes.[26] Most, if not all, of the disability of the feet can be prevented by proper early treatment.[10] With the occurrence of an arthritis involving the feet, there results a limitation of motion in the tarsal and phalangeal joints, accompanied by muscular weakness. The main factor in the progression of deformity in the feet is this muscular weakness and the added burden of weight bearing.

Deformities at the hip or knee may also result in foot strain and deformity. Continued flexion of the knee may cause strain and acute pain in the heads of the metatarsals. External rotation of the leg results in pronation and strain of the longitudinal arch. Long periods of confinement in bed

result in relaxation of ligaments and muscles so that foot strain appears when weight bearing is resumed.

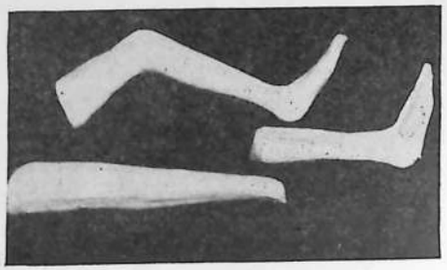

FIG. 46.—Plaster leg supports. Upper: a half leg shell for rest with the top a protective splint. Below and to the right: a night boat for toe drop and painful ankle and foot. Below: a slab splint for painful contraction of the knee. (Swaim, courtesy of Am. Jour. Nursing.)

FIG. 47.—Rheumatoid arthritis. This young girl, aged twenty-six years, had rheumatoid arthritis of multiple joints for two years. The right hip joint (seen at the left side of the roentgen-ray) shows a rather well-advanced rheumatoid arthritis; there is loss of the normal joint space with cystic changes in the bones around the joint. The right sacro-iliac joint also showed a low grade arthritic process.

Deformities seen in the arthritic foot include: (1) rigid valgus, (2) equinus and (3) flat anterior arch with cocked up toes. The position of equinus or plantar flexion results from the pull of the strong gastrocnemius muscle, from the weight of the bed clothing, and in part, from gravity. When such patients are allowed to walk, this deformity is aggravated by the fact that walking is frequently done mainly on the anterior part of the foot (because this results in least jarring and consequently in less pain in

the foot). Contractures of the toes are not infrequent with hyperextension at the metatarsophalangeal joints and flexion at the interphalangeal joints. Hallux valgus is also common.

Pain in the foot in arthritis may be due to: (1) localized pressure; (2) muscular or ligamentous strain; or (3) acute or chronic inflammatory changes.[23] *This foot pain may be relieved by* restoration and maintenance of normal function of the foot.

Pain due to excessive pressure may result from irregularities of the foot pressing against a smooth shoe or from an undue amount of weight being placed on certain bones of the foot. *The commonest cause of pain in the feet and legs is foot strain, a result of long hours of standing, excessive weight and faulty body mechanics.* The foot pain may be the result of an actual inflammatory process occurring in the joints of the feet or along the ligaments or tendon sheaths.

Prevention of Deformity of the Foot in Arthritis

Place a cradle over the feet or a pillow under the covers at the foot of the bed so that the weight of the bed clothes will not eventually produce a position of equinus (or plantar flexion).

If any tendency to foot drop is seen, support the foot in a position at right angles to the leg by sand bags or by molded splints (to be removed daily for motion of the foot).

In arthritis involving the foot, **permit no weight bearing until pain and swelling have completely disappeared,** and until the muscular tone of the foot muscles has been re-educated.

Prevent muscular atrophy in the foot by proper exercises while the patient is confined to bed.

Weak feet may be strapped in dorsi-flexion and inversion for seven to ten days; later one should emphasize proper walking posture and corrective exercises and should employ proper shoes with pads, wedges or metatarsal bars if necessary.

Figure-of-8 canvas ankle straps may provide additional support to the longitudinal arch and prevent widening of the ankle mortise.[7]

Remember that it is easier to prevent deformities of the foot than to correct them.

Serial plaster casts may be required to correct deformities. These should be bivalved in two days to permit joint motion and physical therapy.

Employ proper physical therapeutic procedures, including contrast baths after the acute stage of the arthritic process has subsided. Contrast douches may be given in a tub with a hand shower allowing about one and a half minutes of warm shower to the feet, followed by one-half minute of cool, repeating the entire procedure five or ten times as tolerated. One should always begin and end with warm water. This frequently results in marked relief of pain in the feet in these individuals and in more freedom of motion. The same effect can be accomplished by immersing the feet alternately into two buckets, one containing warm, and the other cool water, for similar periods of time.

One of the most frequent causes of deformity of the foot in the arthritic and in other individuals is rest in bed over long periods of time with the bed clothes tightly drawn so that the feet are pulled into a position of

equinus (plantar flexion). In any patient where rest in bed is required, it should be a rule that the bed clothes be placed loosely over the feet, or if this cannot be done, that a cradle be placed over the feet so that the bed clothes rest upon this or upon a pillow placed under the covers at the foot

To Prevent Foot Drop (Equinus) in Bed Patients:

1. Elevate the head of the bed 6 to 12 inches.
2. Place a board upright at the foot of the bed and allow the foot to push against this with the weight of the body tending to cause the latter to slide down in bed.
3. If required, a posterior molded plaster splint may be used to hold the foot in the proper position.

of the bed, rather than the feet; otherwise, lying in bed leads to external rotation of the hips, slight flexion of the knees and mild equinus of the feet. A simple cradle covering to relieve the feet of weight of the bed clothes can be made by removing one side from an ordinary small orange crate,

Metatarsal Support

1. Cut a piece of felt or sponge rubber about 1 inch in diameter (varying in size and shape with the foot).
2. Cover with cotton so that the support is not over $\frac{1}{4}$ to $\frac{5}{16}$ inch thick.
3. This is pressed against the sole of the foot and is placed just proximal to the heads of the metatarsals with the greatest thickness just behind the second and third metatarsals.
4. When properly placed, this may be strapped on to the foot with adhesive, or glued into the shoe.
5. Most support is given behind the second and third metatarsal heads with less behind the fourth and none behind the first or fifth metatarsal heads.
6. Another method of metatarsal support is the Jones bar.

Jones (Metatarsal) Bar

1. This is a curved or straight piece of leather approximately $\frac{1}{4}$ inch high and $\frac{3}{4}$ inch wide, skived toward the front. This is placed on the outside of the sole of the shoe and follows a line of the ball of the foot (from the head of the first metatarsal to the tuberosity of the fifth).
2. Another method of support is to begin at the anterior part of the heel (at its center) with a piece of leather or heavy rubber and continue this to a point located behind the heads of the second and third metatarsals. This support is from $\frac{1}{2}$ to $\frac{3}{4}$ inch wide and runs along the sole, being the same height as the heel posteriorly, and following the conformity of the sole, being from $\frac{1}{4}$ to $\frac{5}{16}$ inch in height anteriorly.

and placing the crate over the feet with the bed clothes carried over the top of crate. If there is any tendency to foot drop, the foot should be held in a position 10° to 15° beyond the right angle by means of sand bags or a posterior molded splint (removed once or twice a day to permit free

motion of the foot), or the head of the bed may be elevated 6 to 12 inches allowing the body to slide down somewhat in bed so that the feet may press against an upright foot board.

With the onset of rheumatoid arthritis in the ankle, muscular spasm tends to produce a valgus position of the foot, while the pull of the gastrocnemius muscle, the weight of the bed clothes and gravity lead to an equinus position of the foot. The **equinovalgus** which occurs leads to depression of the metatarsal arch, cocking up of the toes and in some instances to hammer toes.

When the feet are involved in an arthritic process, **no weight bearing should be permitted until pain and swelling of the feet have entirely subsided,** *and until the foot muscles have been strengthened by non-weight-bearing exercises.* The muscles of the feet will become weak and atrophied with prolonged rest in bed from any cause, unless active movements and massage of the feet have been carried on throughout the illness. Such a condition must be overcome before weight bearing is permitted. The patient must have proper fitting shoes (with supports if necessary) before attempting to walk again.

Felt or sponge rubber metatarsal support or a Jones (metatarsal) bar may be of value in lessening the discomfort commonly experienced over the second, third and fourth metatarsal heads in these patients.

Hip.—The type of deformity most often seen in arthritis involving the hip is a flexion contracture with adduction and at times internal rotation. This may precipitate flexion of the knee, knock knee, weak foot, equinus of the foot, tilting of the pelvis and increased lumbar lordosis.[16] If the deformity is of long standing dislocation of the hip may occur.

In some cases, the first symptom of arthritis of the hip is pain referred to the medial aspect of the knee and the anterior thigh. In these cases, examination may disclose some muscular spasm after exercise and a slight permanent flexion deformity.

Deformities of the hips and spine go hand in hand. Flexion of the hips follows distortion of the trunk, due to the changed line of gravity. Flexion deformities of the hip result in forward tilting of the pelvis which precipitates extreme lumbar lordosis; the latter is practically always an accompaniment of flexion of the hips.

In some cases, one must use heavy posterior plaster shells, to be worn in bed in an effort to prevent flexion contractures. If there is a beginning flexure contracture at the hips, it may be necessary to place small pillows under the buttocks, the patient being on a firm mattress under which fracture boards have been placed.

Shoulder.—*Pain in the shoulder region is due most often not to arthritis, but to the condition commonly known as subdeltoid or subacromial bursitis or involvement of the shoulder cuff area* (which includes the muscular attachments to the tuberosities). The bursitis is often secondary to tears, hemorrhage or calcification in the supraspinatus tendon. This subject has been discussed in another section.

The main deformity occurring in arthritis involving the shoulder joint is

adduction and internal rotation of the arm; true ankylosis of the shoulder joint rarely occurs in arthritis.

Prevention of Deformity of the Hip Joint in Arthritis

Rest in bed.

Early tendency to flexion of the hip joint secondary to distortion of the trunk may be controlled by placing a small pillow or folded towel under the sacrum and under any point of maximum kyphosis of the spine; use a very thin support at first, and gradually increase its thickness.[16]

If permanent hip flexion is to be prevented or avoided, *the patient should lie on his abdomen* (prone) *for thirty minutes several times a day, beginning with one or two pillows placed under the abdomen.* The pillows are gradually removed as tolerated permitting the stretching of the anterior structures which will allow the hip to assume a neutral position. If the flexion deformity is slight, it may be corrected by traction in the line of the deformity, together with hot fomentations for thirty minutes two or three times a day.

If there is a tendency to rotation and adduction deformity, hold the lower extremities mid-way between external and internal rotation, and, at frequent intervals, separate the thighs to prevent shortening and contraction of the strong adductor muscles. Such positions may be maintained by sand bags or splints, if required.

In the acute stage of arthritis of the hip joint, fix the hip in a position of full extension, 20° abduction and in mid-rotation by the use of traction (Buck's extension) or a plaster splint with a cross-bar under the ankle to prevent rotation; *prolonged use of a plaster cast must be avoided or ankylosis may result.* If Buck's extension is used, attach the traction area well above the knee to avoid stretching the knee joint.

In acute painful hip joints, it may be necessary to apply a unilateral or bilateral plaster spica in order to secure relief or to correct deformities; this must be altered or changed frequently until the desired good position is obtained. When weight bearing is begun, one may employ a light-weight celluloid hip spica which encloses the abdomen and the affected thigh.

Frequently change the patient's position. The bed must have a firm non-sagging mattress (with fracture boards under the mattress) and a prone position is used at intervals throughout the day to stretch any early flexion deformity which may have developed.

If ankylosis is inevitable, the extremity is held in the position of optimum function.

Proper exercises should follow the subsidence of the acute process.

Permit no weight bearing until pain, tenderness and muscle spasm have disappeared.

Prevention of Deformity of the Shoulder in Arthritis.—In an effort to prevent deformities of the shoulders, one should avoid the use of excessive pillows beneath the head, neck or back in semi-reclining and reclining positions. A firm mattress should be used and only a small pillow (if any) should be permitted under the head. In acute arthritis rest the shoulder with the arm in 90° abduction and full external rotation; this may be maintained in bed by pillows, sand bags or plaster splints. This will prevent the tendency to forward drooping of the shoulder with adduction and

internal rotation of the arm which commonly accompanies arthritis of the shoulder. Adhesions are apt to form less readily in this position.

Hot fomentations should be applied to the shoulder for thirty minutes several times a day during the acute stage of an arthritis to relax muscular spasm and relieve discomfort. Several motions must be attempted each day following the hot fomentations to prevent the formation of dense adhesions.

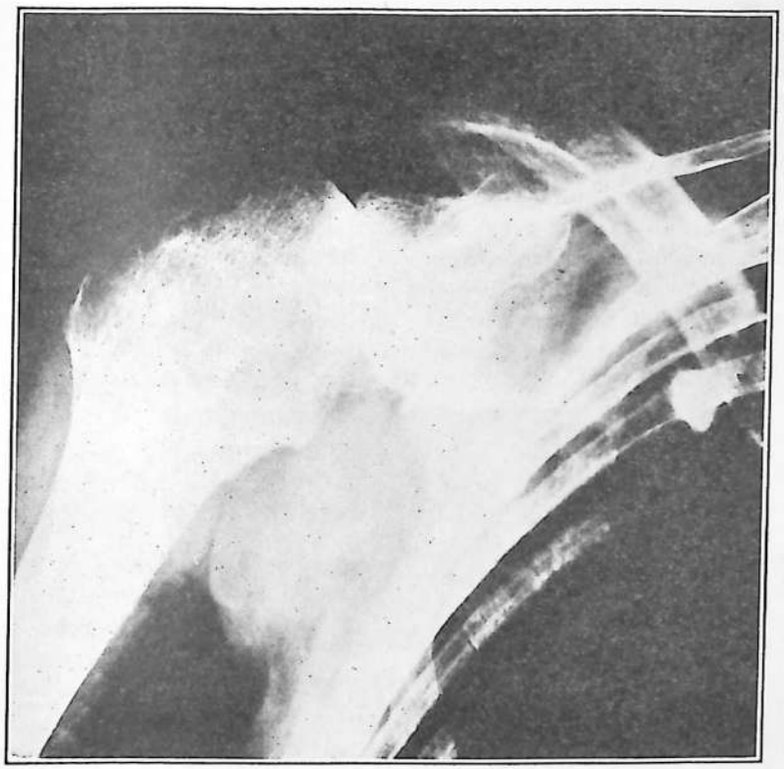

FIG. 48.—Rheumatoid arthritis in a woman, aged sixty-nine years. Patient had arthritis for twelve years with greatest involvement of hands, hips and right shoulder. Motion at right shoulder joint was about 30 per cent of normal. Roentgen-ray reveals almost complete obliteration of the normal joint space at the right shoulder with atrophic and cystic changes in the bones around the joint. There is considerable soft tissue atrophy in this area. The patient's hands also showed a rather well-developed rheumatoid arthritic process. (Note: A calcified Ghon tubercle can be seen in the apex of the right lung.)

When the patient has improved and is ambulatory, an airplane splint may be used to maintain this position. In either case, splints must be removed daily and motion of the joint must be allowed (several times) to break up any adhesions which may have formed.

In painful adducted shoulders, abduct the arms gradually by inserting small pillows between the arm and the chest wall. As soon as tolerated,

perform exercises to maintain abduction and external rotation. Employ proper physical therapy following subsidence of the acute process.

Active exercises may be encouraged by the use of pulleys or screw eyes attached to the head of the bed with a rope passing through these. The ends of the rope are held in the patient's hands and as he pulls down on one end of the rope, the other arm is pulled up and *vice versa*. In chronic arthritis of the shoulders, the patient should attempt to clasp the hands behind the head and flatten out the flexed elbows on the bed for periods of thirty minutes three times a day. The reader should refer to the chapter on "The Painful Shoulder" for a more detailed discussion of this subject.

Elbow.—Unfortunately, the elbow joint is often misused by arthritic patients. It is designed to be a non-weight-bearing joint, but the arthritic individual soon supports himself by holding on to chairs or tables, or by using a cane, thus employing the elbow as a partial weight-bearing structure. This additional trauma to the elbow joint is of importance in leading to limitation of motion and deformity. The customary attitude assumed by the arthritic patient in bed with the elbow at an angle of 90° or less and the hand in pronation on the chest assures the patient of comfort during sleep, but encourages a flexion contracture deformity.

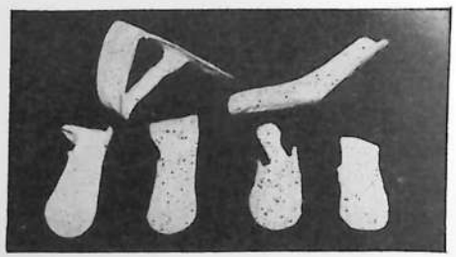

FIG. 49.—Upper left: shoulder splint for abduction of painful shoulder. Upper right elbow slab splint to rest the elbow. Below: set of hand, wrist and finger supports. (Swaim, courtesy of Am. Jour. Nursing.)

Hot fomentations may be used to lessen muscular spasm and pain at any stage of the arthritis. When the process has passed from the acute stage, hot fomentations may be followed by gentle stretching of the elbow by the operator, or by permitting the patient to carry buckets containing increasing amounts of water. Traction is an additional method which may be used in some cases to relieve flexion deformities.

The commonest deformity at this joint is flexion. **Prevent such deformity by:**

In acute arthritis of the elbow, a splint should be worn with the elbow in full extension; this is removed at least once daily to permit several active movements of this joint.

If ankylosis seems inevitable, the elbow is flexed to 70° to 80° and the forearm is splinted in slight supination (10° to 15°).

Special exercises and physical therapy are instituted as soon as possible.

The Wrist and Hand.—Palmar flexion is the deformity of the wrists which must be avoided. In the fingers, if neglected, there may occur ulnar deviation, hyperextension at the metacarpophalangeal joints and acute flexion of the interphalangeal joints.

Deformity of the wrist is due to the pull of the strong flexor muscles and to gravity. The palmar flexion of this joint which results, decreases the normal grasping power of the hand.

Too much weight bearing on the outstretched hand may lead to a flat hand. This is usually brought about by marked weight bearing on the palms as in getting up from a chair or bed, or by the improper use of crutches.

Bony ankylosis is uncommon in the fingers. **Prevent these deformities by:**

1. A cock-up splint from the upper forearm to the finger tips to hold the wrist in 30° of dorsiflexion and the fingers in semi-flexion; ulnar deviation must be prevented by a flange on the ulnar side of the splint.
2. Special exercises.
3. Physical therapy and occupational therapy.

If ankylosis is inevitable, the wrist should be allowed to fuse in a useful position (of dorsiflexion with semi-flexion of the fingers).

Paraffin wax dips are especially useful as a method of applying heat to the wrist and hand; this should be followed by voluntary exercises.

Manual traction may correct subluxation of the interphalangeal joints with the reduction maintained by small *wooden splints* cut from tongue depressors; these should be padded before using. They may be held in place on the palmar aspects of the fingers by adhesive tape. *Banjo splints* with wire mesh finger stalls or with adhesive finger traction may be beneficial in reducing subluxating interphalangeal joints. Swaim's clam shell plaster cuff (over a piece of felt) is effective in preventing interphalangeal and metacarpophalangeal subluxations. The plaster cuff is molded (about felt) over the subluxed joints in a corrected position and can be slipped on and off of the hand easily, being held by an elastic band or webbing strap.

Jaw.—Ankylosis of the temporomandibular joints rarely occurs in rheumatoid arthritis. If these joints are involved, attempts to prevent ankylosis include the application of external heat for periods of fifteen to twenty minutes three times a day, a soft or liquid diet, and the chewing of gum several times each day. The temporomandibular joint may be splinted by an elastic strap under the chin, fastened to a cloth skull cap.[2] Wooden wedges (such as several tongue depressors taped together) or corks may be inserted at first between the incisor teeth and later moved back between the premolars. These should be inserted after the periods of chewing. Increased pain and stiffness within twenty-four hours following the chewing or wedging are indications that these have been carried too far.

Acute involvement of the temporomandibular joint occurs in 20 per cent of patients with rheumatoid arthritis. During the acute state, the teeth should be in centric occlusion, barely separated, and held in this position even while eating or talking. The patient must not stretch his jaw to see how he is progressing. Mild exercises may often be instituted in a few days when muscular spasm and pain have lessened considerably. Heat

should be applied locally over the joint fifteen to twenty minutes several times a day. Mild gum-chewing exercises may then be begun, chewing at first a soft and later a harder gum for fifteen to thirty minutes several times a day. At a later stage one may insert a soft sponge rubber horseshoe ¼ inch thick (cut to fit the shape of the occlusal line). Firmer rubber may be used later, and the device may be kept in the mouth several hours each day. Jaw function increases gradually for a month or two after the acute stage.

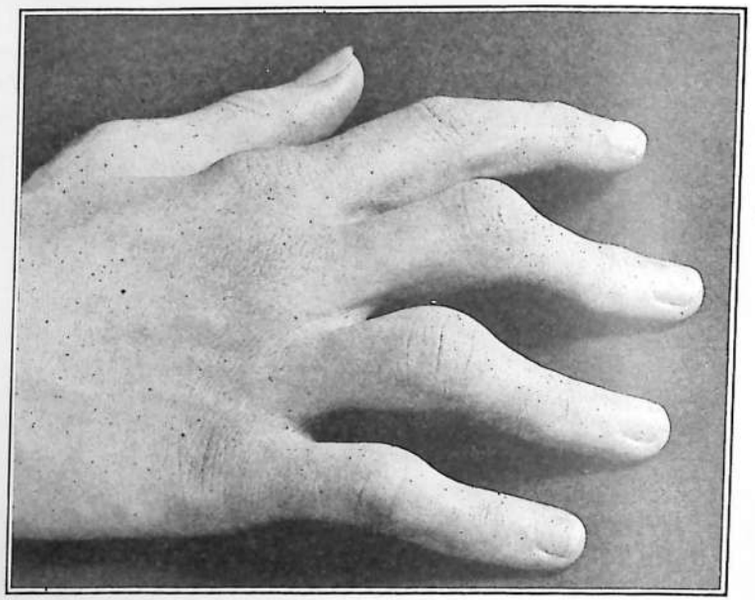

FIG. 50.—Rheumatoid arthritis showing swelling and deformities at proximal interphalangeal joints.

Spine.—If the cervical spine is involved, the tendency toward a flexion deformity (with at times rotation of the head) may be overcome by using only one small pillow under the head, and by lying flat in bed without a pillow several hours each night. The patient must not continually look in one direction, as this leads to a rotation deformity of the neck.

In arthritis of the cervical spine, lateral tilting or rotation of the head may prevent normal vision and become unsightly. This may often be prevented by a *Thomas collar*, or by a piece of *felt* wrapped around the neck each night. The latter should be about ⅜ inch in thickness, 1 yard long and be about 2½ inches in width at one end, gradually increased to a width of approximately 3½ inches at the other. This is wrapped around the neck each night (weather permitting) beginning with the narrow end; it may be held in place by a safety pin or strip of adhesive. A medium sized turkish towel, folded twice lengthwise, may be used in a similar manner. If the patient is confined to bed, it may be necessary to employ

sand bags, hot fomentations, traction or *plaster collars* for support and muscular relaxation.

To avoid deformities of the thoracic and lumbar spine:

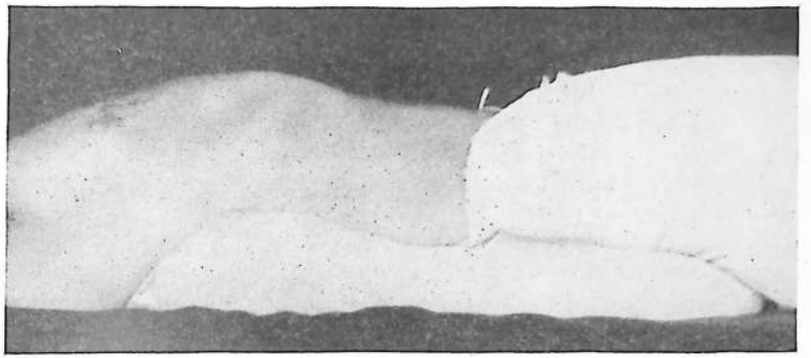

FIG. 51.—Plaster shell used to increase expansion of the chest in hyperextension; used for longer periods of rest in bed and even at night. (Swaim, courtesy of Am. Jour. Nursing.)

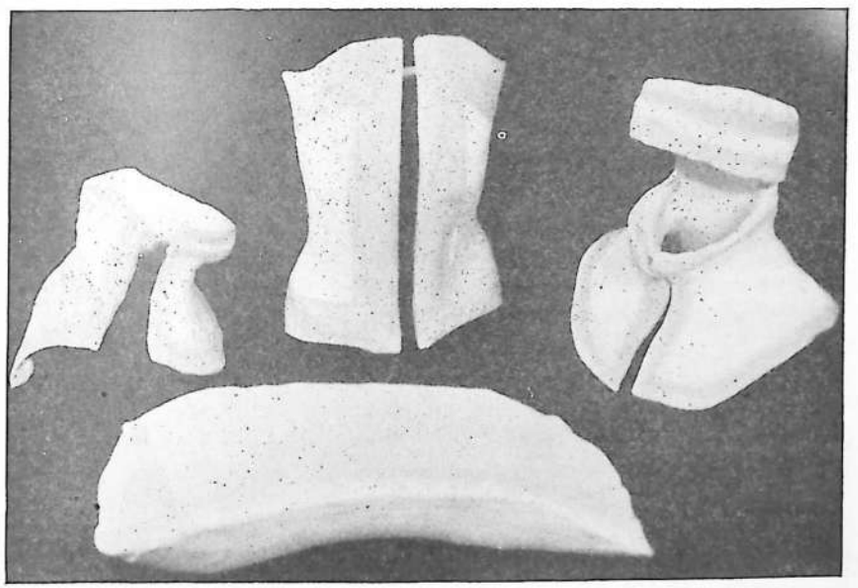

FIG. 52.—Plaster supports: Jackets for the body (above); shell to be used in bed (below); collar for the neck (left); neck support with a halo which effectively immobilizes the lame arthritic neck (right). (Swaim, courtesy of Am. Jour. Nursing.)

1. Discourage the patient from lying on his side (shoulder and hip) as this may lead to severe lateral deviation of the spine. Rest and relaxation may be obtained by the lateral oblique position (on the scapulæ and buttocks with a pillow behind the back); this avoids distortion and soft tissue strain.[16]

2. The patient must lie on the back for thirty minutes after each meal, with the hands beneath the head and the elbows flattened on the bed; deep breathing exercises to elevate the ribs are performed in this position. During these exercises, a small hard pillow may be placed under the thoracic spine (between the scapulæ) but no pillow should be used under the head.

3. A non-sag, firm mattress or large boards placed longitudinally under the mattress will prevent sagging; only one pillow should be used under the head.

4. A small thin pillow may be placed in the small of the back while the patient is in bed to maintain the normal lumbar lordosis.

5. In any tendency toward increasing dorsal kyphosis or flexion of the hip, place a small pillow or folded towel under the sacrum and under the point of maximum kyphosis.[22]

6. A Taylor brace with shoulder straps may be used when the patient is ambulatory.

7. Plaster and, later, celluloid or waxed leather body casts may be extremely beneficial in patients with rheumatoid spondylitis.

Hench and his associates do not believe that all arthritic deformities are avoidable. A great number of these are needless, but in some cases, despite the best medical and orthopedic attention, sudden exacerbations of the arthritic process may result in moderate to marked contractures. It has been shown that such *joints may "freeze up" within three or four days.*[4]

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CHAPTER XIV

PHYSICAL THERAPY IN RHEUMATIC DISEASES

Physical therapy is merely an adjunct to other treatment in the arthritic. Its injudicious use may cause harm. *Physical therapy alone does not cure arthritis and the use of this measure to the exclusion of other antiarthritic treatment will not produce the best results. In individuals with chronic arthritis physical therapy is probably the most important single factor in treatment: its main value is to increase blood flow in the periphery.*[11]

Physical therapy should not be given haphazardly, but should be dispensed with care under the direction of a physician. When limitations of budget so require, members of the patient's family can be instructed in the proper use of massage, hot applications, etc. The coöperation of a well-trained physical therapist is always advisable.[53]

Physical therapy is the use of physical agents, such as heat, cold, rest, muscular exercise, massage, light and electricity in the treatment of disability and disease. Physical therapeutic measures which have been used in the treatment of rheumatic diseases include *heat*[1] (as diathermy, short wave, ultra short wave, baking, electric pad, hot water bottle, infra-red lamp, hot baths, hot packs, steam bath, mud bath, fever therapy, light cabinet baths, hot air douches, paraffin dips, etc.); *massage; hydrotherapy,* including whirlpool baths and under water exercise; special active and passive movements (*exercise* and rest); occupational therapy; heliotherapy; iontophoresis (histamine or mecholyl); faradism and galvanism (to restore wasted muscles); roentgen therapy; colonic irrigation and the use of electric currents.[26] It is impossible to describe these various measures in detail as volumes have been written upon each. An attempt will be made to give the general practitioner a working knowledge of the usefulness of these measures, their indications, contraindications and methods of application.[32]

Piersol has stated that physical therapy is less well understood and less frequently employed by the internist than any other form of therapy. He has found that very little time is devoted to physical therapy in the average medical school and that the courses which are available are inadequate and superficial. Thus, one of the oldest and most useful branches of therapeutics is practically unknown to the average physician.[45]

Unfortunately, conservative physicians occasionally look upon physical therapy with distrust because of its wide exploitation by charlatans and quacks, and because of the unwarranted, exaggerated statements made as to its value by overenthusiastic individuals. Physical therapy was reborn in World War I because of the urgent need of returning the wounded to the front lines rapidly or of rehabilitating them sufficiently for non-combatant duty. Copeman has discussed physical therapy in the field in war zones.[10]

One must be very careful not to employ physical therapy for malingerers and professional accident seekers.

Physical Therapy in Rheumatoid Arthritis

Because of the various methods available to the physician, he is somewhat at a loss to determine which measures to apply in a given case. In general the practising physician may employ the following plan:

1. Application of moist heat **(hot fomentations)** to acutely inflamed or subacutely inflamed joints.
2. No massage or exercise during the acute stage of an arthritis, except that the involved joint should be moved through a full range of painless (or nearly painless) motion two or three times each day to minimize the formation of dense adhesions.
3. **In the subacute and chronic stages, the triad of heat, massage and graded exercises should be used in practically every patient every day.** The form of heat at this stage should be that which is most comfortable to the patient and at the same time, productive of best results. The form of heat may be varied several times a week and one may then continue that which seems to suit best that particular patient, whether it be moist heat, infra-red lamp, baking, warm paraffin applications, warm tub baths, diathermy, warm cabinet baths, etc.
4. In the subacute and chronic stages, physical therapy must be carried out at least once (and preferably more) each day, regardless of whether the patient is at a spa, hospital or in his own home. If he is at home, daily home physical therapeutic measures can be supplemented, in most cases, by treatments several times a week by trained masseurs, in addition to warm baths, pool treatments, diathermy or other measures which may be indicated, if the patient is ambulatory. **Physical therapy must not be confined to two or three diathermy or other treatments per week at the physician's office!**
5. It is practically always possible to train some member of the patient's family or some friend to assist in the proper home physical therapeutic measures which are desired.
6. **The physician** or his assistant **must take the time to explain in detail the rôle of physical therapy in the home to the patient and his family.** Unfortunately, the reason for poor results in many cases is the indifference or ignorance of the physician to the great importance of heat, massage and graded exercises in the care of the chronic arthritic.
7. Occupational therapy should be instituted as soon as possible in most patients with rheumatoid arthritis.

Physical therapy must be conservative in older people as *aged individuals do not tolerate the application of physical measures as well as do young persons.*

Carey has emphasized that *physical therapy must not be neglected in the medical curriculum for medical students.*[5]

It has often been pointed out that in order to be effective in a therapeutic program in the arthritic patient, *physical therapy must be carried out daily in the home* for a long period of time. The physician must prescribe for each patient individually and should give several demonstrations of treatments. If sufficiently detailed instructions are given, a large number of these patients continue the treatments at home and are enthusiastic about the benefits they derive from these.[55]

Physical therapy technicians should be well trained and should be under the supervision of a physician.

At present there are 28 approved schools for Physical Therapy Technicians. There were about 720 graduates in 1944.

Most of these schools require two years of college credit as the minimum entrance requirements. The average tuition is about $200 per year.

Individuals desiring registration may write to the American Registry of Physical Therapy Technicians, 30 North Michigan Ave., Chicago 2, Ill.

The regular physical therapy course averages from nine to twelve months. Emergency courses set up during the war may usually be completed in six months.

The most universally applicable modality in physical therapy is **moist heat.** Some patients with chronic rheumatoid arthritis spend a large amount of time in the warm bath. This is in reality a method of low grade fever therapy. If a patient is submerged in a bath at 110° F. for twenty minutes, he develops an oral temperature of 102° F. This form of generalized application of heat by the hot bath is most acceptable and of distinct therapeutic value to many patients with multiple joint pains. In addition, there are available various forms of moist heat for local application, such as hot fomentations, soaks, paraffin baths for the hands or feet, whirlpool baths, steam jets, etc.[51]

Although, in general, **moist heat appears to produce more relief** (both subjectively and objectively) to most patients with various forms of arthritis, dry heat is definitely acceptable and is frequently used either in general form such as cabinet baths or locally as diathermy, infra-red radiation, radiant heat lamps, hot pads, etc.

One of the least expensive and most valuable forms of physical therapy is **exercise.** Proper exercises require perseverence, patience and time on the part of both physician and patient; improperly performed exercises may cause definite harm.

The importance and the technic of **massage** are not fully appreciated by most physicians. Massage is extremely useful in most patients with chronic forms of arthritis. **Properly performed massage should not cause pain and must not tire the patient.** The technic of massage must be learned from one trained in this art. The physician treating patients with arthritis would be well repaid for his time and effort if he learned the indications, technic and use of massage from one well experienced in this field; in addition, he should allow the expert masseur to practice on the physician himself so that he would experience first hand the "feel" of the amount of pressure exerted, the method of avoiding bony prominences, the direction of the stroking and other pointers which he should master. Because of the extreme importance of this subject, a special chapter has been devoted to instructions for the physician concerning massage and an attempt has been made to portray as well as can be done by photographs and sketches, the various methods which may be used, the direction of the stroking, the use of frictions about various joints, the positions of the part being massaged, etc.

Other physical therapeutic measures for the arthritic include the use of ultraviolet irradiation or sunshine, iontophoresis, the use of static electricity (to reduce swelling in some inflammatory lesions of joints), etc.

Physical therapy is not used for its psychologic effect upon the arthritic, but if properly used, it will aid in reducing joint swelling, relieving pain, improving the general condition of the individual, preserving and improving joint function and lessening deformity. Although a general plan of attack must be available, the **treatment must be individualized for each case.**

In the patient with **rheumatoid arthritis** whose joints are temporarily immobilized by splints (which are removed daily for several movements), gentle heat will often hasten reduction of the swelling and alleviate pain; *intense heat must not be used* at this stage as this may aggravate both pain and swelling. If possible, the involved extremities should be elevated from time to time to lessen the degree of swelling. When the patient passes into a subacute phase with decrease in the swelling and lessening of the muscular spasm, mild stroking massage may be begun, with the stroking being performed in the direction of the venous flow. It is extremely important to *massage the muscles above and below the affected joints;* when the hand reaches the affected joint, it is raised so that it passes over the joint surface very lightly.[51] Graduated exercises may also be used as the evidence of acute inflammation subsides.

The Rheumatoid Arthritic Patient in Bed

When the patient with rheumatoid arthritis is confined to bed, he must be encouraged to observe several general principles:

1. *Deep breathing exercises,* with 8 to 10 deep breaths each hour while awake. The breathing should be thoracic, with the abdomen "pulled in."
2. The patient or nurse must be sure that the covers are not pulled tightly over the patient's feet to prevent foot drop; a pillow or cradle should be placed under the supporting sheet so that the blankets do not rest upon the patient's feet.
3. *Muscle setting exercises* can be begun almost at once in any arthritic patient who is confined to bed. These include the voluntary contractions of the *quadriceps muscles, gluteal muscles,* etc., eight to ten times per hour, or more as tolerated without causing pain or fatigue.
4. In acutely inflamed joints, much of the pain, swelling and muscular spasm around the affected joints can be relieved by frequent *hot fomentations,* much in the manner of the Kenny packs of the affected parts for poliomyelitis. The use of such hot packs should be encouraged by the practising physician; the marked improvement resulting will more than compensate the physician and the patient for the time and apparent inconvenience.
5. As the patient improves, moderate *exercises* can often be performed with the assistance of weight and pulley arrangements, care being taken to prevent friction of the bed clothes on the buttocks, heels and elbows.
6. *Exercises* should be *increased gradually* in range and time, but these *must never cause pain or fatigue.*
7. *Proper massage can usually be begun as soon as the acute stage of the process has disappeared.*

When the shoulders and hips are involved, it is a great advantage to have available a large therapeutic tank for under-water exercise. After the patient has been placed in the tank or pool, the temperature may be gradually increased until the water reaches 100° or 102° F. (as in the Hubbard-Currence tank). In some of these tanks, whirlpool arrangements are provided which may be directed at the painful joint, relieving discomfort and encouraging active movement.

In some cases of persistent edema at the ankles or wrists, the use of *static wave current* may hasten the removal of this fluid; if this is not available, mild graded exercises will be very helpful and if required, one may prevent pain by an *ethyl chloride spray* to the part prior to the exercise. Some individuals have used iontophoresis with procaine to lessen or prevent the pain prior to such exercise.[51]

In the patient with **degenerative joint disease,** no one form of heat is always preferable to another. One may employ hot fomentations, infrared lamps, heating pads, mud packs, contrast baths, hot soaks, hot paraffin applications, diathermy, cabinet baths, warm tub or pool baths, etc. In most of these cases, heat will improve the circulation within the joint capsule. If muscular spasm is present, mild, graded exercises will lessen this spasm if performed without causing pain.

Patients with degenerative joint disease may develop pain in some cases from prolonged lack of movement and pressure on opposing joint surfaces where the cartilaginous insulation is gradually disappearing. Non-weight-bearing activity may lessen such discomfort.

In **degenerative joint disease affecting the cervical spine,** the pain is usually mainly mechanical with narrowing of the intervertebral spaces (through which the spinal nerves pass) and limitation of the range of motion of the neck. In the early stages of the disease, pain occurs only with certain movements of the head and neck and is often more pronounced on one side than the other; the pain later becomes radicular with a segmental distribution along the distribution of the cervical nerves. This picture is often confused with neuritis, bursitis, myositis, etc.

The **Sayre head sling** is especially applicable for patients with degenerative cervical joint disease. One should employ radiant heat for thirty minutes, followed by effleurage of the neck and shoulder. The Sayre head sling may be attached to an ordinary ice scale and from 70 to 80 pounds of traction may then be applied slowly. One may make a head sling (as suggested by McFarland and Krusen) of double thickness cable felt 2 inches wide with the center loop of the sling fitting under the chin and occiput and the two free ends slipped over the hooks.[42] When one has reached the desired number of pounds of pull, the head may be rotated gently to the right and to the left, following which the traction is slowly released. It is preferable to carry out this procedure twice a day for a week or more, following which the patient should be instructed in the use of the apparatus at home. Firm traction on the neck is produced by the patient flexing his knees slightly to permit pull on the overhead apparatus.

Such a procedure is a rational method of treating these cases because the intervertebral foramina have been narrowed and the spinal nerve roots emerging from these have been compressed by the degenerative bony

changes. This pressure results in inflammatory changes in the nerves, which will subside if the neck is extended by stretching; the latter procedure widens the foramina and removes pressure upon the nerves. *Narrowing of the intervertebral foramina may be missed by the roentgenologist unless oblique views are taken.*

In **degenerative joint disease of the lumbar spine**, exercise may aggravate the symptoms in structurally weak backs. In these individuals, it is often advisable to limit motions by curtailing such activities as golf which results in torsion strain to the lower dorsal spine and lumbar muscles. If there is marked thinning of a disk or marked hypertrophic changes, passive manipulations of the lumbar spine and straight leg raising exercises must be forbidden.

In those patients with degenerative joint disease of the lumbar spine and obesity, one should prescribe high back *corsets* or *braces* together with supports for a sagging abdomen; these relieve the overburdened, acutely angulated lumbosacral joint of much of the weight and distribute it over a wider longitudinal area of the back. These individuals are also often benefited by a *firm mattress*, with fracture boards under this, *heat* to the back, *massage*, *muscle setting exercises* and later proper active exercises.

In patients with **involvement of the knees and ankles**, every step must be taken to prevent the possibility of invalidism. If these individuals are greatly overweight, this should be reduced to lessen the load upon the lower limbs. The early use of *crutches* will assist in weight bearing in these individuals and should be used unless there is an associated arthritis of the hands, wrists and elbows. *It is often difficult to persuade patients with mild synovial irritation to use crutches, but they are extremely valuable if used early.*

In general, the use of a cane is discouraged because of the increased strain which is thrown upon the elbow joints and wrists. When canes are used, the individuals usually walk with flexion of the hip and lumbar spine, which is inadvisable.

When the **hip joints** are involved in a **degenerative process**, *diathermy* often affords marked relief if it is applied directly through the hip joint. In the *early stages* of *degenerative hip joint disease*, the patient will usually be benefited symptomatically by a regimen of limited activity, and judicious use of diathermy and massage. All of these patients should have daily *massage*, especially of the adductors of the thigh. Proper *exercises* must be performed several times a day and one should attempt to maintain as much motion as is possible, without pain by passive stretching of the hip joint and fascia of the thigh.

The patient must realize that physical therapy alone does not cure rheumatoid arthritis or degenerative joint disease, but periodic, properly directed physical therapy will help greatly to maintain their activities.

Some teachers do not realize that **medical motion pictures** are available on a *loan basis* from the American Medical Association. These may be borrowed by medical societies, medical schools, hospitals and other scientific groups. Requests for these should be instituted as far in advance as is possible so that proper reservations may be made. The only expense incurred is that of transportation both ways. Requests should be sent to the Director, Scientific Exhibit, Am. Med. Assn., 535 North Dearborn St., Chicago 10, Ill. Among the films which would be of special interest to rheumatologists are those on "Aids in Muscle Training;" "Massage;" "Occupational Therapy" and "Underwater Therapy." All of these mentioned are 16 mm. silent films.

Fibrositis is considered to be a subacute or chronic inflammation of the white fibrous tissues of the body; the latter is found in ligaments, tendons, fasciæ, aponeuroses, muscle and nerve sheaths, periosteum and subcutaneous tissue so that fibrositis may affect practically any region of the body. The *main symptoms of fibrositis* are aching, pain, stiffness and fatigue. Fibrositis is usually divided etiologically into the primary type and the secondary fibrositis (associated with gout, influenza, rheumatoid arthritis or degenerative joint disease, etc.).

In any patient with symptoms of fibrositis, one must think of an associated diabetes mellitus or hypothyroidism; these two diseases are frequently associated with symptoms of fibrositis.

Primary fibrositis can be divided anatomically into the intramuscular form and the periarticular form. Intramuscular fibrositis includes such conditions as *torticollis*, *pleurodynia*, *cephalalgia*, *lumbago* and affections of other muscular groups without particular designation.

Periarticular fibrositis is found in the tissues immediately around the joint and may be misdiagnosed arthritis. *Rheumatoid arthritis* can usually be *differentiated from periarticular fibrositis* by the findings in the former of an increased sedimentation rate during the active stage of the disease; the positive roentgen findings; swelling of the joint with the appearance of fluid in the joint; atrophy of the adjacent muscles; and the systemic manifestations accompanying the process. In primary periarticular fibrositis, the cardinal symptom is marked stiffness, especially on awakening in the morning which requires considerable movement before this symptom subsides.

Primary fibrositis is not accompanied by roentgen changes, muscular atrophy, rapid sedimentation rate, marked systemic manifestations, marked loss of weight or the other findings so common in rheumatoid arthritis.

Fibrositis may produce nodules or indurations in the muscles at the back of the neck, especially along the nuchal line at the back of the head; these indurations, which can be palpated, are tender and may give rise to headache in the back of the neck or in the frontal region, or extending over the top of the head between these regions. Krusen has found that the fibrositic nodules in this region can be palpated most readily by allowing the patient to rest his forehead against the examiner's chest in order to relax the muscles of the neck; in this position, the fingers of the examiner can locate the tender indurations.

Fibrositic headaches are usually worse on awakening in the morning. Prolonged application of heat and heavy local massage are credited with affording marked relief to these individuals.

Krusen has reported that the indurations or nodules of intramuscular fibrositis are found, in order of frequency: (*a*) in the lower lumbar muscles and along the iliac crest; (*b*) along the nuchal line at the back of the neck; (*c*) in the upper border of the trapezius muscle; (*d*) the scapular or interscapular regions; (*e*) the intercostal muscles; and (*f*) the gastrocnemius, pectoral and rectus abdominis muscles.[35] The indurations are felt very infrequently in the muscles of the forearm or thigh.

Most European writers agree that the application of heat followed by deep, local massage is almost specific for this syndrome. Although heat and firm massage are credited with controlling the fibrositic flare-ups, *they do not prevent recurrences.* In our experience, *recurrences are most common following exposure to cold and drafts.*

The massage must be firm, beginning gradually at the first session and slowly increasing in intensity; it is directed toward any painful region or palpable thickening. Severe massage is not employed in the acute stage of the process. The massage is increased in intensity so that in about two weeks more pressure can be exerted with very little pain and the nodules can then be manipulated by the finger tips or knuckles, in conjunction with deep effleurage of the whole part.

Each affected region should be treated for ten to fifteen minutes daily. Treatment of advanced cases may require four to six weeks, while soft, recent thickenings may be dispersed in seven to fourteen days. In some instances, hard, fibrous indurations may persist despite any prolonged, vigorous massage.

Periarticular fibrositis is treated in a similar manner with initial applications of heat, followed by very firm local massage.

In patients with fibrositis, one often employs exercises which will stretch most efficiently the involved tendons, periarticular tissues or muscles.

In *massaging the fibrositic nodules,* **friction massage** is usually employed, *i. e.,* the finger tips remaining in firm contact with the skin, with the skin gliding over the subcutaneous structures; no lubricant is used on the skin in this procedure. One may use the hyperextended finger tips or the ball of the thumb in circular movements over the affected areas.

Some pain is always caused by this heavy massage of the indurated areas, but this may be minimized by slow, gradual and careful manipulation. Stretching exercises are an important therapeutic procedure to be used following the massage. *One must avoid injury to tendon sheaths or nerves which may result from too forceful manipulation.*

One should warn the patient that he may feel somewhat worse for several days following the massage and stretching exercises; although the soreness may increase temporary, many of these patients will feel less stiff and less fatigued. *One is usually gratified by a continued program of heat, deep friction massage and stretching in these patients with fibrositic indurations.*

The indurations in muscles and subcutaneous tissues are felt only with difficulty by the beginner in physical therapy. As one improves his technic, he will feel these indurations in the subcutaneous tissues and muscles much more frequently and in some cases actual fibrous bands may be detected in the muscles.

Although one can usually promise the patient with primary fibrositis a good immediate result (within a few weeks), he must be warned that recurrences of his fibrositis will probably be very common.

The local application of *heat and massage* are often beneficial in the patient with **senile osteoporosis,** together with postural exercises and the use of a back brace or corset with stays for support of the back. The use of vitamin D, calcium and estrogenic substances to promote calcification and lessen discomfort are discussed elsewhere.

In some cases of **spondylolisthesis** in which surgical treatment may not be indicated or in which it is refused, *palliative measures* include the use of radiant heat, massage and a well-fitted back support. About 10 per cent of cases of anterior displacement of one vertebra on another are asymptomatic.

A **painful coccyx** (coccygodynia) is usually not removed unless this is due to trauma. *Conservative treatment* for coccygodynia includes internal massage as advocated by Thiele (to reduce pain by relieving spasm of the pyriformis muscle), hot sitz baths and the use of a rubber ring.

Hibben has summarized the **medico-legal aspects of physical medicine.** According to the Council on Physical Therapy of the American Medical Association, physical therapy is the treatment of disease by various non-medical and non-surgical means, comprising the use of heat, light, water, electricity, massage, exercise and climate.

By law, the physician is bound to use such reasonable ordinary care, skill and diligence as physicians and surgeons in the same neighborhood in the same general line of practice ordinarily exercise in like cases. **If the physician departs from recognized remedies or methods of treatment or diagnosis, he does so at his own peril and risk,** and *if such departure results in injury to the person treated, the* **physician is liable for damages** *no matter how good his intentions may have been.*

The physician is, of course, responsible for any negligence on the part of his nurse, physical therapy technician or assistant whom the physician has assigned a duty, even though she is employed and paid by the patient. *The majority of medico-legal cases involving physical medicine are brought against physicians who dabble in physical therapy.*

Hibben suggests that only equipment which bears the approval of the Council on Physical Therapy of the American Medical Association be used, and that this should be inspected every six months or oftener by a capable supervisor.

The *Council on Physical Therapy of the American Medical Association* has *accepted various devices* such as a *hot water bottle with an electric metal heater* protruding down into the water so that it may be controlled at a constant temperature (usually not over 125° F.); an *automatic electric blanket* (thermostatically controlled to maintain the temperature at a constant level despite environmental changes); *Cooley compresses*—to keep a constant warmth in wet dressing by a waterproof electric pad; etc.

Except for roentgen and orthopedic practice, physical therapy heads the list for the number of damage suits and number of judgments rendered the plaintiff. Every physician using physical therapy should be well covered with a standard insurance policy in a good company; the local county medical society will assist him in determining which insurance carrier is the most reliable and offers the best coverage.

Great care must be exercised even in the prescribing of such apparently simple measures as a hot water bottle. In general, the temperature of the water put into the bottle is 120° to 125° F.; unfortunately, some nurses are not familiar with this. The physician must specify the part of the body to which the hot water bottle is to be applied; whether there should be an intervening material between the skin and the bottle; the length of application; and *the bottle should be removed if the patient says that it is too hot.* Hot water bottles should not be applied to sleeping or unconscious patients without adequate supervision. These same general principles apply to the use of other physical agents such as infra-red lamps, bakers, heating pads, ultra-violet radiation, short wave diathermy, paraffin baths, etc.

It is essential that patients have privacy and that they be properly draped with clean linens. *The patient should be instructed to report even the slightest discomfort during any treatment.* A *record should be kept of each treatment* given, including the kind and length of treatment and any other significant data. The *physical therapist must avoid any hasty unguarded statements as to prognosis.* In general, the patient should understand in advance the approximate number of treatments which may be required, together with their approximate cost.

HEAT IN RHEUMATOID ARTHRITIS

The beneficial effects of heat in arthritis were well known to the ancient Romans and Greeks, who constructed elaborate baths and utilized natural hot springs in the treatment of these conditions. Patients with most forms of arthritis are usually grateful for the local application of heat. Heat relaxes muscles, increases local nutrition, increases the flow of blood and frequently relieves pain. Heat may cause harm if applied injudiciously. In some patients with acute arthritis, the application of heat in any form seems to aggravate the local condition; **in individuals who are made worse by heat, it should be discontinued or modified.** An acute flare-up of a local arthritic process may follow mild exposure to heat. Some individuals feel that heat should not be applied to an acutely inflamed joint when the overlying skin is hot or red.[21] With the above exceptions in an occasional patient, heat is not contraindicated in the acute stage of arthritis; however, *the greatest value of heat is in chronic arthritis.*

Forms of heat to which the body may be exposed include: (1) **ultra-violet rays** (short wave lengths—1850 Angstroms—artificial light; 2900 to 3600 Angstroms—sunlight); (2) **visible light**—3600 to 7500 Angstroms; (3) **infra-red** of various wave lengths: (*a*) 7500 to 15,000 Angstroms (near infra-red), (*b*) 15,000 to 30,000 Angstroms (intermediate infra-red), (*c*) over 30,000 Angstroms (far infra-red—such as heat from hot water bottles, hot baths).

Body tissues may be heated in one of three ways: (*a*) by conduction, (*b*) by radiation, or (*c*) by the electric current.

In heating tissues by **conduction**, a substance whose temperature is higher than that of the body is placed in contact with the skin so that the heat is conducted from the warmer object to the body tissues. This includes the use of electric pads, hot water bottles, hot water or hot air baths, paraffin baths, etc. The skin is a poor conductor of heat; *very little rise in temperature can be expected in the deep tissues following the local application of heat, because this is rapidly carried away by the blood supply beneath the skin.*

Application of Heat in Rheumatoid Arthritis

In the acute stage, with red, hot, swollen, tender joints, if the patient can be made comfortable by the use of splints, rest and medication, do not apply heat to the joints. If, in spite of such care, the joints remain painful and there is no tendency toward quiescence of the disease, one may apply hot wet dressings of a saturated solution of magnesium sulfate to the affected joints for periods of from five to thirty minutes several times each day. If hot fomentations result in further pain, they should be discontinued at once. If the patient does not permit the weight of the dressing upon the affected joints (because of pain) employ radiant heat as infra-red rays for from five to twenty minutes several times a day, unless this produces an aggravation of symptoms.

In the acute stage, *do not employ* diathermy, ultra-short wave therapy or hot baths. Poor results have been obtained to date with fever therapy in rheumatoid arthritis.

As the patient improves and the disease passes *into the subacute or chronic stage* (as judged by sedimentation rate, symptoms, joint swelling and tenderness on motion), *heat is one of the most useful weapons available. When possible, in such individuals, apply heat to the affected joints several times per day, followed usually by massage of the muscular tissues between the joints,* with only the lightest stroking of the joints themselves. If the disease is more or less generalized, use the hot water bath (gradually bringing the temperature of the water from 95° to 98°, 99°, 100°, 101° or 102° F.) with the patient remaining in the bath up to twenty or thirty minutes, depending upon his tolerance. If the bath is followed within twenty-four hours by an exacerbation of symptoms, this should be modified or discontinued.[12]

For involvement of individual joints in chronic or subacute rheumatoid arthritis, some of the common heating methods which may be employed include bakers (which can be made inexpensively), ordinary small hand heating lamps, infra-red lamps, hot paraffin applications, contrast baths, conventional diathermy and ultra-short wave therapy. For home therapy, use a heat lamp for thirty minutes two or three times a day at a distance of comfortable toleration over the affected joints.

In heating by **radiation**, the shorter the wave length falling on the skin, the greater is the penetration, and thus more energy can be tolerated

without damage to the surface tissues.[52] In treatment with infra-red radiation, when penetration is desired, a source with as high a temperature as tolerated is used. The *main effect of infra-red radiation is hyperemia in the part treated;* one of its uses is as a counterirritant for the relief of pain in lumbago and fibrositis.

Heating by the **electric current** is rather easily understood. *Heat is produced when an electric current passes through a resistance. An electric current of sufficient strength sent through the body tissues would heat these.*

Should heat be applied to the joints of patients with rheumatoid arthritis?—The answer to this question in general should be **yes.**

Should heat be applied to joints in the acute stage of rheumatoid arthritis?—Answers to this may vary. If properly applied, it is often beneficial. Some individuals feel that heat is distinctly contraindicated in the acute stage of an arthritis. If heat aggravates the symptoms it should be discontinued. Hot fomentations are often beneficial during the acute stage of rheumatoid arthritis.

Value of Heat in the Treatment of Arthritis.—A proper amount of heat *increases blood and lymph flow* in the affected area, thus aiding in the removal of waste products and hastening the absorption of inflammatory exudates; it also *increases local nutrition.* Heat *relaxes* tissues, especially *muscles,* which may be in spasm. Mild local heat often relieves pain. Available forms of local heat include heat lamps, infra-red (non-luminous) heat generators (the most efficient means of applying heat in the office or home), hot wet compresses or poultices, hot water bottles, heating pads, heated bricks, bags of salt or sand, etc. Luminous heat penetrates somewhat more than infra-red.[28] The greatest amounts of heat are obtained from carbon or tungsten filament lamps as heat rays from luminous sources penetrate more deeply than those from non-luminous origin. One may obtain penetration of heat into human tissue up to 1.5 cm. from luminous bulbs.[33]

One must be extremely careful in applying heat to a diabetic or to any patient with loss of sensation, scars or impaired circulation.

Very little careful work has been done on the question of how much heat should be introduced in various conditions and we have no good information, for example, as to whether it is better to treat a sprained ankle by a temperature of 102° or 103° or 106° F.

HYDROTHERAPY

The *advantages of water* as a medium *for* the application of *thermal stimuli* include:[8] it is a good conductor of heat; it eliminates the force of gravity to a large extent; more intense and less variable thermal stimuli are derived; immersion in water relieves muscular spasm, stimulates cutaneous and venous circulation and produces a good psychologic effect upon the patient. Some of the most commonly used methods of hydrologic treatment include reclining baths of plain and mineral water, or carbon dioxide or salt; a deep pool at 110° F.; mud packs; manipulation douches

(Aix and Vichy type) at 102° F.; cabinet vapor baths; foam or bubble baths.[54]

Hydrotherapy is contraindicated in acute rheumatoid arthritis because of the probability of fatigue and increased activity of the disease.

Under-water Treatment in Arthritis.—Currence has described an improved method of under-water treatment of arthritis.[15] He has designed a tank which permits a complete range of motion of all joints. If necessary, patients may be swung on to a stretcher (supported by an overhead carrier) and lowered slowly into the tank. By means of an overhead trolley, the patient may swim supported by a belt.

FIG. 53.—Modified Hubbard tank. Note overhead pulley and head rest. (Currence, courtesy of Arch. Phys. Ther.)

In a hydrotherapeutic pool, the temperature of the water is kept between 90° and 96° F.; this is comfortable to the patient and encourages muscular relaxation.[19] Pool baths also improve the morale of the patient; the appetite is often stimulated following the under-water exercise.

The temperature of the water may be changed rapidly. In subacute or chronic stages of rheumatoid arthritis, the patient may be placed in the tank with the temperature of the water approximately 100° F. After four or five minutes, this is gradually increased to 104° to 106° F. so that about 1° F. rise in body temperature is produced; the temperature of the water is then rapidly reduced. The patient is given several gentle painless

passive movements of affected joints, and is returned to bed. Treatments may be given every two or three days if no reaction occurs within twenty-four hours. Inexpensive types of Hubbard tanks are available for home use.[57]

FIG. 54.—Modified Hubbard tank. (Currence, courtesy of Arch. Phys. Ther.)

Nylin's Technic of Under-water Exercises in Arthritis[44]

Temperature of water when patient enters tub is 96° to 98° F.
Gradually raise temperature to 101° to 102° F. during first three or four minutes, placing cold wet compress as a turban to patient's head to prevent unpleasant symptoms; change compress frequently.
Turn on shower at bottom of the tub, if available (see bubble baths).
If desired, apply gentle stroking and kneading in the neighborhood of joints, rather than to the joints themselves.
Active under-water exercises should not be attempted (especially if the patient is frail or if the joints are very painful) until several baths have been given.
Bath may last from ten to twenty minutes.
Treatments are given from two to three times per week, with local heat and massage on the days on which under-water exercises are not used.
Such exercises relieve pain and strengthen atrophied muscles.

Under-water therapeutic exercise is very effective in stimulating flabby muscles and in restoring their normal use.[30,31,38,43] The *buoyancy of the water* and its *efficiency in overcoming the force of gravity* are the greatest advantages of under-water treatment.[39] Inasmuch as the specific gravity of the water is about the same as that of the body, the weight of the latter is almost completely carried by the sustaining power of the water.[50] This permits much greater freedom of movements and hastens the process of rebuilding atrophied muscles. There is also a great stimulating effect upon

the patient's morale, when he finds that he can readily move his limbs when immersed in water (although this may have been almost impossible when in bed).

When a body is submerged in water, it will lose as much weight as the displaced water. A patient who weighs 132 pounds will weigh only 6 pounds when lying in water.[18] Because of this, weakened *muscles and joints can perform actions under water which would be impossible out of the water. A dramatic psychologic improvement often results* in helpless bed-ridden patients when they are able to move their affected parts in a pool. Aided by the buoyancy of the water, extension of affected joints may be performed in marked comfort under water (as compared with weights and pulleys attached to the usual ward bed). When traction is exerted on a table under water, the body will often float free of the traction table.

Buoyancy is present in any water, whether it is at the most expensive spa or in the cheapest home-made pool or bath tub.

The therapeutic pool is not used in patients with pulmonary tuberculosis or in those with evidence of arteriosclerotic heart disease.

In **patients with multiple joint involvement**, we frequently employ the **following program** of physical therapy:

1. General *hot bubble baths*, 100° to 102° F., ten to twelve minutes, with the patient moving her affected joints while submerged in the bath. The bubble bath is given *three times a week.*

2. *On the alternate days*, when bubble baths are not employed, *baking, massage* and *passive* and *guarded active movements* are prescribed for the upper and lower extremities and the neck.

The Hot Bath.—This is a very useful procedure in individuals with subacute or chronic generalized rheumatoid arthritis.

Warm baths exert a sedative effect and a mildly stimulative effect upon the circulation.

Many arthritic patients do not tolerate overexposure to heat and are easily exhausted from prolonged hot baths.[37] Thermal baths (97° gradually raised to 102° F.) are those most frequently used in the treatment of arthritis. They may be used for five to thirty minutes two or three times a week. They are also useful (especially in cold weather) in older patients with degenerative joint disease. They are given as follows:

The *patient is placed in a tub* of warm water (*temperature about* 98° F., determined by a bath thermometer and not by the elbow). One or 2 pounds of sodium chloride may be added to each 20 gallons of water. Some patients prefer baths containing 1 ounce of pine extract to each 20 gallons of water.

An attendant is present during the entire duration of the bath.

The temperature of the water is raised gradually to between 98° and 102° F. or in some cases, to 105° F. by running hot water into the tub.

The first bath lasts only five or six minutes and is given at least two and a half hours after a meal.

The duration of subsequent baths is gradually increased up to thirty or

forty minutes if well tolerated (*i. e.*, if there is no exacerbation of joint symptoms and no undue weakness).

A cool wet towel placed around the head lessens discomfort while in the tub.

Weakened or emaciated patients may respond poorly to such measures.

The use of several cupfuls of magnesium sulfate in the water may result in greater comfort to the patient and adds an important psychologic factor; most patients will readily submit to magnesium sulfate baths but may not appreciate the value of warm water alone.

The patient may be sponged with alcohol after the bath, dried well, and returned to bed wrapped in a light blanket, without exposure to drafts.

Fluids are given freely after the bath (or during the bath if undue sweating occurs); *if sweating is excessive, enteric coated tablets of sodium chloride* (1 gram each) *are supplied.* Use between 2 and 6 of these depending upon the amount of perspiration.

The patient should rest in bed for at least an hour following such a bath.

Foam Bath.—This is prepared by placing about 10 cc. of a saponin solution in about 3 or 4 inches of water in a tub. Foam is then formed by forcing air or oxygen into this mixture through a long frame with perforations immersed in the water. After the tub has been filled with foam (in ten to fifteen minutes) the patient enters the tub. Radiation of heat from his body is restricted by the foam which covers the patient. Perspiration is produced. In nervous patients, such baths have a sedative effect.

Bubble Bath.—A metal plate with numerous perforations covers the bottom of the tub or tank.

Pipes are placed under the plate so that water (or air) under high pressure may be forced upward through the holes, as a shower.

This produces a smooth and even mechanical stimulation directed uniformly over the whole body surface.

The tub is filled with warm water (96° to 98° F.) and the patient enters this before the shower at the bottom of the bath (bubble bath) is turned on. Most arthritic patients experience marked amelioration of their symptoms following such a bubble bath.

Hot Foot or Arm Baths.—These may be given by using a large bucket or specially constructed container with water at a temperature of 100° F. After the arm or foot has been immersed in this container, the temperature is raised rapidly to 115° F. and is maintained there for twenty to forty minutes. Many patients will not take advantage of warm foot soaks at home, unless some medication is added, such as an ounce of magnesium sulfate per gallon. Such therapy is of most benefit in the chronic stage of the arthritis.

The Whirlpool Bath.—This is a bath of whirling aërated water developed by French army surgeons during the war of 1914–1918. The water in this bath is kept at a temperature of approximately 110° F. The whirling of the water adds an element of soft and gentle massage to the heating effect. These baths are useful in treating traumatic, circulatory and

chronic rheumatoid conditions of the upper or lower extremity. The aërated water results in peripheral vasodilatation, soothes peripheral nerve endings and induces muscular relaxation. Although they are somewhat expensive, a useful form of whirlpool bath can be constructed by a local plumber at a moderate expense by using a copper wash-boiler and ordinary pipe fittings. Details of such a bath have been given.[13]

The apparatus suggested by Boynton consists of a copper tank 28½ x12 x 27½ inches with 2 inlets connected through a T-joint with a common pipe from a mixing valve.[3] Each of the inlets is equipped with a ½ inch McDaniel suction tee, with an air vent pipe from the tee to the top of the bath to provide adequate air mixing. The inlets are placed 4 and 18 inches above the bottom and near the outlet. Standard 1-inch galvanized pipe is used. A 2½-inch overflow outlet is placed at one end near the top to keep the water at a safe level.

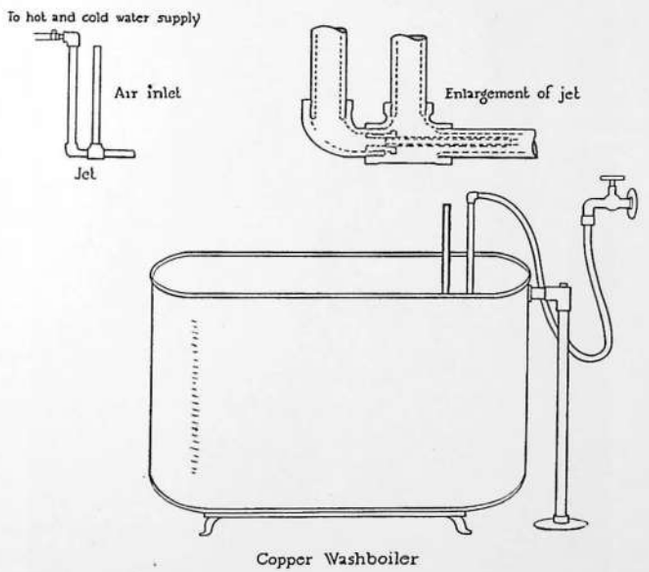

FIG. 55.—Home-made whirlpool bath. (Council on Physical Therapy of the American Medical Association.)

The Full Wet Pack.—The hot full wet pack is beneficial especially in individuals with subacute or chronic rheumatoid arthritis, who should not or cannot be moved to a tub. Muscular relaxation and relief of discomfort usually follow this procedure. This pack serves also as a useful preliminary measure for general or local massage. It requires no expensive equipment and no special training of the operator. The whole procedure can be performed by one individual. The method of application is as follows:

The room must be warm (temperature about 72° F.).

Place a rubber sheet or a large piece of oilcloth over the entire bed, and a large blanket on top of this; use an old blanket as this will become wet.

Fold a large sheet lengthwise in plait-like folds about 8 inches wide so that it may be spread conveniently and quickly under the patient.

Dip the sheet into hot water (about 110° F.) and wring this out.

Quickly spread the sheet under the patient (who is nude).

With the patient's arms elevated, fold one-half of the sheet about the patient from the neck downward.

Wrap the other half around the individual after the arms have been lowered, the upper edge of the sheet surrounding the neck.

FIG. 56.—Contrast baths to lower extremities. (Krusen, Physical Therapy in Arthritis, courtesy of P. B. Hoeber, Inc.)

Place a layer of sheeting between the legs and wrap the lower end of the sheet around the feet.

Wrap the sheet snugly around the patient.

Wrap the blanket (under the sheet) closely around the sheet, tucking this in under the feet and around the neck (the head of the patient being the only exposed portion).

Wrap one or two additional blankets around the patient, depending upon the degree of warmth developed in the particular patient.

Apply an electric pad or hot water bottle if the patient's feet become cold.

Continue the hot pack—from thirty minutes to an hour, as tolerated.

Contrast Baths.—Contrast baths (or contrast sprays or douches) are of assistance in relieving pain and stiffness in joints especially in degenerative joint disease. *They often have a more analgesic local action than heat alone, and may be used in the treatment of any stiff, painful joints in rheumatoid arthritis.* When other conservative measures (such as splinting, rest and mild analgesics) do not relieve discomfort in an acute arthritic flare-up, contrast showers may be given to practically any joint in the body. They are often of value in those individuals with cold, clammy extremities.

Contrast Showers

If the patient can be moved into a tub, a small bath spray is attached to the common outlet for hot and cold water. If, for example, the knees are involved, a spray of warm water (as tolerated) is directed against these for one and a half minutes, followed by a spray of cool water for one-half minute. This alteration of heat and cold is continued for twenty to thirty minutes, always beginning and ending with warm water. The spray may be directed in a similar manner to any involved joint.

If the patient cannot be moved to a tub, a large rubber sheet is placed under the patient and the head of the bed is elevated 4 to 6 inches (on wooden blocks or a small chair). A rubber hose connection extends from the common outlet for hot and cold water to the patient's bed. The spray is now applied in a similar manner with an assistant regulating the change of hot and cold water in the bathroom, changing these as enumerated above. The longer the rubber tubing, the more time is required for the water to reach the patient. The water is allowed to drip from the rubber sheeting into a bucket placed on the floor at the foot of the bed.

In the case of *hands or feet*, if sprays are not available, the same effect can be obtained by the *use of two large buckets or containers, one* partially filled with *cold water* (temperature about 60° F.) and the *second* with *warm water* (about 105° to 110° F.). The hands or feet are immersed in the warm water for one and a half minutes, followed by the cool for one-half minute; this is repeated for a total of from twenty to thirty minutes.

The general procedure of the contrast douches is immersion or spraying of the affected part for approximately one and a half minutes with warm water, followed by a shorter period (one-half minute) of cool water. This alternation of immersion or spraying with warm and cool water is continued for twenty to thirty minutes, as tolerated, always beginning and ending with warm water.

This simple procedure is either not well known or is used but little in general practice. It is one of the more useful and simple methods of affording symptomatic improvement to these patients.

HOT PARAFFIN APPLICATIONS

An effective method of applying heat to individual joints is to coat them with several layers of warm paraffin, and to allow this to remain in

FIG. 57.—Warm paraffin bath. (Handbook of Physical Therapy, Am. Med. Assn.)

FIG. 58.—Method of applying hot paraffin with a paint brush. (Krusen, Physical Therapy in Arthritis, courtesy of P. B. Hoeber, Inc.)

place for thirty to sixty minutes. The paraffin is easily peeled off at the end of this period. It results in a moderate hyperemia with reduction of pain and stiffness. It is also (as in the case of contrast showers) an excellent psychologic form of therapy. Many patients enjoy more relief from paraffin applications than from electric pads. This is useful especially in the treatment of joints in the chronic stage of rheumatoid arthritis in homes where electricity is not available and where one cannot utilize bakers, infra-red radiation, etc. It may be used two or three times a day.

Technic of Paraffin Application

About 4 pounds of ordinary paraffin (such as is used to cover home-made jelly) is melted in the upper portion of a double boiler.

Paraffin wax melts at various temperatures, the most frequent melting point of commercial preparations being 120° F.

The temperature of the melted paraffin should range from 120° to 130° F. In order to obtain this temperature, one may use 4 parts of paraffin and 1 part of liquid petrolatum. A bath thermometer may be used to determine the temperature of the mixture.[20]

This is allowed to cool until a thin scum forms on the top.

It is now ready for use. In the case of the fingers, the hands are dipped into the paraffin and removed after several seconds. The fingers should be held apart while immersing the hand. The fingers must be held still; otherwise, the paraffin will crack on hardening. The patient must not touch the very hot bottom or sides of the container. As soon as this layer of paraffin has hardened (within five to ten seconds) the hands are dipped into the wax again. This procedure is repeated until 8 or 10 coats have been applied.

The wax coated hands are wrapped in a warm blanket for thirty to sixty minutes.

Because of the sweating which occurs beneath the paraffin, this latter is easily peeled off when desired.

The mixture of oil and paraffin may be used repeatedly as this sterilizes itself.

In hairy parts, the hair must be shaved before the application of paraffin; otherwise dermatitis may result.

In involvement of other joints (knees, shoulders, elbows, etc.), 8 or 10 coats of wax may be painted on the involved part by a brush or a large soup ladle wrapped in gauze.

Be extremely careful in patients with thin, tender skins; these persons may experience a burning sensation from the wax. In these individuals, add about 1 ounce of mineral oil to each pound of wax to lower the melting point of the mixture.

Do not spill wax on floors because of the resulting danger of slipping on the wax.

BAKING APPARATUS

In chronic stages of rheumatoid arthritis, properly and systematically performed baking coupled with massage may work wonders in the treatment of painful stiff shoulders, knees and other joints. Unfortunately, many individuals look upon this as an office or a hospital procedure. An effective baker may be built cheaply for the home.

A simple inexpensive model has been prepared for such use.[31] This consists of a frame of strap iron ($\frac{1}{16}$ x $\frac{5}{8}$ inch) to which is riveted a reflector made of highly polished tin sheeting. The sheeting is placed on top of the frame in a slight arc. Just below the tin sheeting are set two double receptacles in which are placed a total of four 60-watt bulbs. These receptacles are connected in multiple with heavy lamp cord about 6 feet long. The dimensions of the baker (for use over arms or legs) are 17 inches in length, 14 inches in width, and 14½ inches in height. If the baker is to be used over the body, the upright supports should be about 2 or 3 inches longer. By means of such a baker, any variation in heat from 1 to the full 4 bulbs may be used.

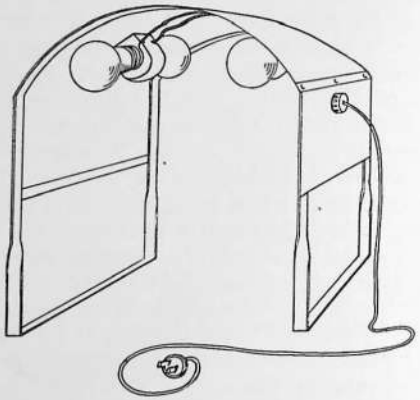

FIG. 59.—Home-made baker. (Council on Physical Therapy.)
Specifications: (*a*) Frame—1/16 by 5/8 inch strap iron; (*b*) reflector—highly polished tin sheeting; (*c*) 2 double receptacles, General Electric Company, Catalogue 66722; 250 volts, 650 watts; (*d*) 4 60-watt mazda lamps.
The tin is riveted to the strap iron. Receptacles connected in multiple with heavy lamp cord 6 feet long. Hubble plug at the end of cord. The above baker is designed for applying heat to the legs or arms. If baker is to be used for the body, supports should be 2 or 3 inches longer.

Although this apparatus is simple to construct, some individuals may not be able to complete the equipment or may not be in proximity to a shop where the framework may be constructed. A simpler (but less effective) apparatus may be made in the following manner. About 4 feet of heavy garden wire fencing 2 feet in width is bent into an arc, the top of the arc being approximately 1½ to 2 feet high. A large extension cord is obtained and the end containing the bulb sockets is fastened to the top of the wire. By means of several double or triple sockets, 4 to 6 bulbs may be fastened on the under surface of the wire. This should be prepared so that the hot bulbs cannot come in contact with the skin. The apparatus is adjusted over the part to be treated, and several heavy blankets cover the outside of the wire fencing. The bulbs are lighted in sufficient number to produce the desired amount of heat; when this has been obtained, most of the bulbs may be turned off and a small amount of heat will maintain the desired temperature. If desired, the apparatus may be made large enough to include the entire body, except the head. The body baker is preferable to the hot wet pack as the former is simpler to use, and there is not as much initial heat with gradual lowering of the temperature as occurs with the wet pack.

Baking produces muscular relaxation, increased blood flow to the part (which furnishes increased nourishment for these tissues and hastens the removal of waste products), and relief of pain and stiffness in joints which are the seat of a chronic arthritic process. *Heat*, followed by properly administered *massage* and regulated *exercise* are extremely important factors in the treatment of chronic arthritis.

Additional Pointers in the Use of Bakers.—Care must be taken that the electric bulbs or hot metal do not come in contact with the skin.

The point of heat tolerance of the individual must not be exceeded.

Baking of an arm or leg should consume from thirty to forty-five minutes, unless this is not well tolerated. Heat must never result in blistering or burning of the skin.

Thirty to forty-five minutes of baking followed by ten to twenty minutes of properly administered massage daily may prove very beneficial.

The part must not be cooled or chilled immediately following baking.

A period of rest following and equal to that of the baking and massage is usually prescribed if generalized treatment is being given. Otherwise, the individual is encouraged to move the joints immediately after the baking and massage.

OTHER METHODS OF APPLYING HEAT

These include hot water bottles, electric pads, hot fomentations, hot bricks or sand bags, heat lamps, hot foot or arm baths, hot whirlpool baths, diathermy, ultra-short waves, infra-red radiation and heliotherapy. In the use of *hot water bottles*, care must be taken to prevent burns. It is especially important that the bottle be wrapped so that *no metal may come into contact with the skin* of the patient. Temperatures at which the ordinary electric pad is set are approximately 108° F. (low), 180° F. (medium) and 245° F. (high).

Hot Fomentations.—These may be applied by dipping heavy Turkish towels or doubled woolen blankets or woolen cloths into a bucket of hot water (about 115° F.), wringing these out, and applying them to the part to be treated. To prevent the rapid escape of heat, they are covered by a hot water bottle and a small blanket or by a blanket alone. These hot applications should be changed from every one to five minutes, as cooling occurs. They are renewed for a period of fifteen to thirty minutes at each treatment. Hot fomentations are of great value in the treatment of acutely painful, swollen joints; the addition of a cupful of magnesium sulfate to the bucket of water may produce better symptomatic results. If joint pain and swelling increase following such applications, the treatment should be discontinued. Hot fomentations are valuable also in the treatment of painful stiff joints in the chronic stage of rheumatoid arthritis. Such moist heat may produce more symptomatic relief than dry heat.[49]

Hot Air Douche.—One may use an ordinary hot air blower as is employed to dry hair. This is fixed on a stand and hot air may be blown against painful joints (such as fingers and hands) for periods of from ten to thirty minutes.[28]

Heat Lamp.—A simple, inexpensive heat lamp has been devised by Krusen for local application of dry heat. This consists of a cup-shaped polished reflector similar to those used by photographers with photoflash bulbs and a clamp by which it may be attached to a chair, bed or other object. Within the polished reflector is placed a 250-watt Mazda CX bulb. Such an outfit costs not more than four or five dollars and serves as an effective means of applying radiant heat. It has been advised that this lamp be held about 18 inches from the body and that local treatment should not exceed thirty or forty-five minutes one or two times a day. Similar lamps attached to an adjustable stand with a flexible elbow can be purchased usually for less than ten dollars.

FIG. 60.—Large luminous heat generator on stand. (Courtesy of The Burdick Corporation.)

In some communities it may be necessary to revert to older methods of applying dry heat, such as the *heated brick, bag of sand or salt, or iron.* In any of these great care must be taken that the hot object does not come into direct contact with the skin of the patient. Special caution must be used when tissues are heated in the region of scars or when sensory nerve function is impaired. One of the favorite methods of applying heat to the low back in former days was to place a dry towel over the low back and to iron the towel back and forth with as hot an iron as the patient could tolerate.

Heating Jacket.—It is very difficult to heat all surfaces of the shoulder by ordinary means because of its irregular contours. When an ordinary heat lamp is employed, the rays converge on only one surface so that little heating of the periarticular structures in the axillary space is obtained. The usual baker does not produce uniform heating of the periarticular tissues of the shoulder. Krusen has devised an inexpensive device to provide controllable uniform heating of all surfaces of the shoulder girdle and upper arm.[34] This is a modification of the electrically heated sleeves or cuffs devised by Brown and Allen, constructed in the manner as the electrically heated diving suits used by naval deep-sea divers, in which the temperature was controlled by an accurate thermoregulating device;[4] by means of this type of device, regulated conductive heat can be applied to all surfaces of almost any region of the body, such as the hip, foot or shoulder, without resorting to an elaborate and expensive diathermy apparatus which is not essential for warming the superficial structures.

Electric Cabinet Baths.—These are used in the hospital or office only in robust patients. Treatment lasts from five to fifteen minutes, and is followed by a warm shower and gentle massage. The patient should then lie down and rest and gradually cool off for forty-five minutes or an hour after the bath. Such baths may be employed once or twice a week, and prove of benefit in robust individuals with generalized chronic arthritis and especially in patients with fibrositis.

ULTRA-VIOLET AND INFRA-RED RADIATION

During the past ten or fifteen years, advertising campaigns stressing the value of special treatment with these forms of radiation have reached great heights. As Coblentz pointed out, *it is difficult to think of any warm object which does not emit infra-red rays;* this is in contradistinction to the thought of many laymen and a few physicians who believe that infra-red radiation is new and somewhat mysterious.[7]

Any object which has been heated to a higher temperature than its surroundings emits infra-red rays; thus, coal fires, hot stoves, steam pipes, light bulbs, etc., are all examples of sources of infra-red rays. These rays are emitted also from hot water bottles and electric pads, but are of low intensity. The spectral quality and total intensity of infra-red rays emitted by radiant heaters for the purpose of warming rooms is the same as that produced by expensive infra-red lamps, but the heat rays are not focused sufficiently for use in arthritis. Over 60 per cent of natural sunlight is infra-red radiation. Near infra-red rays may penetrate through the skin, subcutaneous tissues and superficial muscular layers as well as accessible tendons and bones.

One of the main differences between infra-red and ultra-violet rays is that the former produces an instantaneous sensation of heat when the intensity is great, while the ultra-violet rays act slowly and insidiously so that their effect is not perceived in most instances until four to eight hours after their application. Severe skin burns and conjunctivitis may result from undue exposure to ultra-violet radiation; because of this, dark glasses are worn during all ultra-violet radiation treatments.

Glass in most windows does not usually permit the passage of ultra-violet rays, due chiefly to their content of iron oxide. Practically all of the ultra-violet rays of sunlight are transmitted by glass made of clear silica (quartz); such special glass (Uviol or Vita glass[2]) has been installed recently in many homes, offices and hospitals.

Ultra-violet rays may penetrate through skin for a depth of 0.1 to 1 mm. and infra-red rays many times this distance. The amount of ultra-violet radiation required to be of use in therapy may be obtained from electric arcs between electrodes of metal, carbon or mercury vapor in a closed tube of quartz glass. The only natural source of such radiation is the sun, the temperature of whose surface is probably about 10,000° F.

Ultra-violet Therapy

In chronic arthritis, general heliotherapy is often of tonic value; small areas of the skin (one-fourth of the body) should be exposed at a time. If ultra-violet radiation is used, treatments should be given not oftener than every second day (because of the time required—about forty-eight hours—for the skin erythema to reach its maximum). Do not employ heliotherapy or ultra-violet irradiation in patients with acute rheumatoid arthritis because of the possibility of producing increased activity of the joint process.

If an ultra-violet lamp is used, the initial treatment should not exceed one-half minute and should not cover more than one-fourth of the body surface; gradually increase the portion of the body exposed and the length of exposure. The ultra-violet generator is usually kept about 30 inches from the body. Care must be taken not to burn the patient. Occasionally, one may note untoward symptoms such as dizziness, nausea, vomiting, diarrhea, insomnia, etc.; in these cases treatment should be discontinued temporarily; when this is again begun, the dosage must be smaller.

If direct sunlight is used, the initial treatment should consist of about five minutes anteriorly and posteriorly; treatments may be given daily if no untoward symptoms result, increasing by five minutes anteriorly and posteriorly to a total of one hour's exposure per day.

Ultra-violet light is not used to produce local heat in the joint; its chief effect is a general tonic one upon the organ as a whole. It has been suggested that such an "insult" to the skin may directly improve immunity.[23]

The room should be warm and well ventilated.

Goggles must be worn.

If the skin is sensitive, the face should be covered by a thin towel and the nipples and genitalia with heavy paper or toweling.

The advantage of the carbon arc lamp is that it is always ready for use as soon as it is lighted.

The hot mercury vapor lamp requires an interval of several minutes until the full ultra-violet radiation is produced.

General irradiation should be divided equally between the anterior and posterior surfaces.

A time clock with a bell alarm attachment should be used.

Dangers include hypersensitivity to ultra-violet rays and overdosage.

Contraindications to Ultra-violet Therapy

Active pulmonary tuberculosis, advanced cachexia, cardiac decompensation, renal or hepatic insufficiency, marked arteriosclerosis, lupus erythematosus, generalized dermatitis or patients receiving sulfanilamide. Nervous individuals or those with fever may be made worse.

The use of ultra-violet light or sunlight is often of distinct value to the patient with chronic rheumatoid arthritis. A decrease in pain and muscle spasm and a general tonic effect may follow exposure to direct sunlight, or to other ultra-violet radiation. *Some rheumatologists have cautioned against the use of heliotherapy in the acute stage of the disease or in the presence of fever. Others have extolled the value of heliotherapy early in rheumatic joint disease* stating that the ultra-violet irradiation of the skin may accelerate

the absorption of joint effusion, decrease joint swelling and deformity and increase joint movement.[17] A maximum erythema dose of ultra-violet radiation has been advised in this connection. The erythema produced by the ultra-violet radiation is a useful method of producing counter-irritation.

In our experience, direct sunlight or ultra-violet irradiation seems to act as a tonic to patients with rheumatoid arthritis after the acute stage of the disease has passed. Following such exposure in gradually increasing amounts, the patient's appetite seems to improve and a general sense of well-being often results. However, it must be remembered that sunlight or ultra-violet irradiation does not cure rheumatoid arthritis.

Ultra-violet radiation is of no value as a method of applying heat locally to joints, because of the time required to produce an erythema. If ultra-violet therapy is used, the first several treatments should be of very short duration (one-half minute each) and must be directed at only one-quarter of the body at a time.

Ultra-violet lamps vary in erythemal potency so that they should be standardized. Adequate ventilation must be provided in the treatment room and a shield or goggles must be used over the eyes of the patient. The genitalia and breasts (in women) should be covered. One must remember that the senile or keratotic skin may predispose to the development of cancer. *Patients with diabetes frequently react unfavorably to irradiation* resulting in local and general petechial hemorrhages. A safeguard which should always be provided is the sounding of a clock to measure the length of exposure.

The available ultra-violet lamps include the carbon arc lamps, the hot quartz lamps, the low pressure mercury arcs, cold quartz lamps and electrodeless induction lamps. The closest approach to sunlight is the carbon arc, which is the hottest artificial source of radiation. In the use of artificial heliotherapy in rheumatoid arthritis, the carbon arc irradiation is probably the preferable source of ultra-violet light as it also produces heat radiation, which may be beneficial. The *advantages of the carbon arc lamps* are: (1) the full power develops instantly; (2) the lamps are portable and are not easily damaged by moving. Their disadvantages are the large amount of current which they consume and the adjustment and replacement of carbons which is necessary from time to time.

The *quartz mercury vapor arcs* are based upon the principle that mercury, when vaporized by intense heat in a quartz container, emits a spectrum which is rich in ultra-violet.[25] Because of the ease of handling these lamps and the slight upkeep, they are one of the most widely used sources of ultra-violet light; they require only about one-fourth as much electrical energy as other sources.

Infra-red Rays.—These are also useful in relieving painful, swollen joints. A rapid (but often only temporary) analgesic effect frequently occurs following their application. Mild infra-red radiation often relieves pain in acute arthritis. The *advantages of infra-red radiation* are the penetration of its rays, the absence of pressure on the treated parts, and the

fact that the tissues may be observed constantly while being treated. The short infra-red rays are the most valuable therapeutically, as these possess the power of greatest penetration. One of the best bulbs for this purpose is a tungsten filament lamp consuming from 100 to 1500 watts, depending upon the size of the lamp. The small lamp produces as much deep heat as the larger one, but the latter affects a greater area. The concave mirror reflector is suitable for the heating of affected joints.

If thermal radiation is used one obtains superficial heating with penetration of not over 3 mm. in the region of the far infra-red (as produced by hot water bottles or electric pads, hot water or paraffin baths, an electric heater, etc.). The average skin temperature is about 89.6° F.; this cannot be raised more than 18° F. without producing a bleb or blister. In all cases, the patient's comfortable sensation to heat should be the final criterion.

Infra-red radiation is more economical, easier to manipulate, easier to control and less dangerous than short wave diathermy. The part being treated is more easily inspected if infra-red radiation is used and the effectiveness of the treatment may be varied by the application of hot wet compresses or counterirritants; wet compresses may be kept on continuously during the treatment if moist heat is desired as the compress is kept hot by the lamp and more water may be poured on the cloth periodically to replace that which evaporates.

This form of treatment may be given several times a day, if necessary, and the maximum heat tolerated is focused upon the painful joint. The distance of the infra-red generator from the patient will vary from 2 to 3 feet depending upon the type of reflector and the sensitivity of the parts to be treated. If heat radiation is the main object of the treatment, this is continued from thirty to sixty minutes; if the part is being warmed merely as a preliminary to massage, twenty to thirty minutes is sufficient. The occurrence of burning sensations during treatment indicates that the lamp should be moved farther from the patient.

Infra-red Radiation

Infra-red radiation is used chiefly because of its local heating effect. It may be useful in relieving 1 or 2 painful joints in the acute stage of an arthritis or in decreasing pain, stiffness and muscular spasm in joints which are the seat of a chronic rheumatoid process.

It may be given several times a day; the maximum heat which can be endured by the patient is focused upon the painful joint for a period of from fifteen to thirty minutes. Blistering of the skin must be avoided. Usually allow three to five minutes to heat the generator before applying.

Infra-red radiation is often a valuable method of heating tissues before the administration of massage.

DIATHERMY AND SHORT WAVE DIATHERMY MACHINES

Heat is produced when an electric current is passed through tissues. This action is utilized in diathermy machines. The only type of current

which can be passed safely through the body at a high enough intensity to appreciably raise the temperature is the high frequency current. *One of the most important features of the diathermy machine is that it delivers a high frequency current.* Medical diathermy has been defined as the therapeutic use of heat generated in the tissues of the body by high frequency currents, insufficient in amount to result in temperatures which are high enough to destroy tissues or to impair their vitality.[14] **This is the most efficient form of heating deeper tissues and is especially valuable in treating the spine and large joints.**

Diathermy may be indicated in contusions, muscle strains, myositis ossificans, sprains, bursitis, tenosynovitis, chronic arthritis, myositis, myofascitis and fibrositis. In traumatic arthritis, one often obtains marked relief by diathermy followed by static electricity or massage.

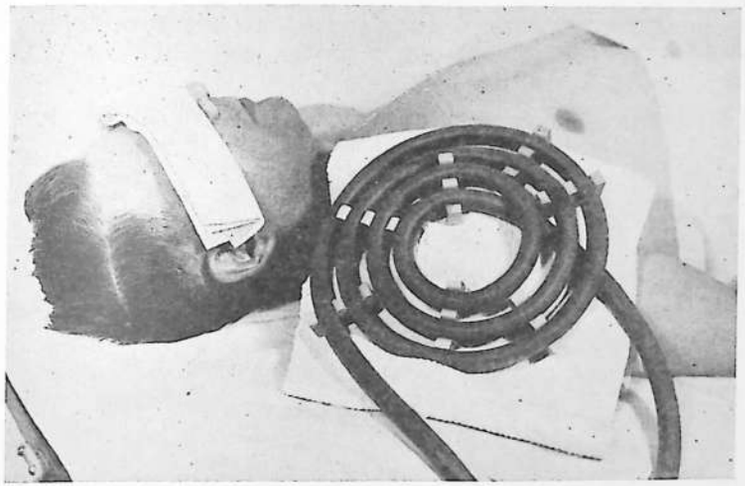

FIG. 61.—Short wave diathermy to the shoulder. Pancake coil technic—electromagnetic induction. (Coulter, courtesy of Med. Clin. North America.)

In contusions or muscle strains, diathermy may be used to increase the local circulation after a period of twenty-four hours during which the swelling, tenderness and ecchymosis have been reduced by cold compresses or an ice bag. The first treatment of a muscle strain should be brief and at a low intensity.

In some cases of acute **bursitis**, pain is relieved by the use of short wave diathermy for twenty minutes each day (in addition to infra-red radiation for thirty minutes twice a day); with a diminution in the pain, proper massage and relaxed motion is then used, and later, active exercise. If several applications of diathermy aggravate the bursitis, this should be discontinued, and one should consider the use of procaine injections, roentgen therapy or other measures. In *chronic subdeltoid bursitis*, short wave diathermy, infra-red radiation, massage and exercise are often of great value. Diathermy may aid in the absorption of calcified deposits about the shoulder joint.

In **degenerative joint disease** of middle-aged individuals with bilateral involvement of the knees or hips, rest and proper administration of diathermy often result in relief of symptoms.[28] Although diathermy may be of great value in chronic arthritis, some cases of degenerative joint disease may be aggravated by heat; for this reason, the first few treatments with diathermy in chronic arthritis should be of low intensity and for only a short period. *The first short wave treatment is often followed by an aggravation of symptoms in many forms of arthritis.*[40] If such distress recurs regularly following these treatments, diathermy must be abandoned.

Conventional diathermy machines produce wave lengths above 30 meters; *short wave diathermy* denotes wave lengths from 3 to 30 meters, and *ultra-short wave diathermy*, wave lengths less than 12 meters.[16] The Council on Physical Therapy of the American Medical Association has suggested that the term *short wave diathermy* be applied to the therapeutic heating of body tissues by an oscillating electric current of high frequency, *i. e.*, from 10,000,000 cycles per second and a 30 meter wave length to 100,000,000 cycles per second and a 3 meter wave length.[14] *Synonyms* which have been used previously *for* such *short wave diathermy* include: short wave therapy, ultra-short wave therapy, radiothermy, parathermy, neodiathermy, short diathermy, etc.

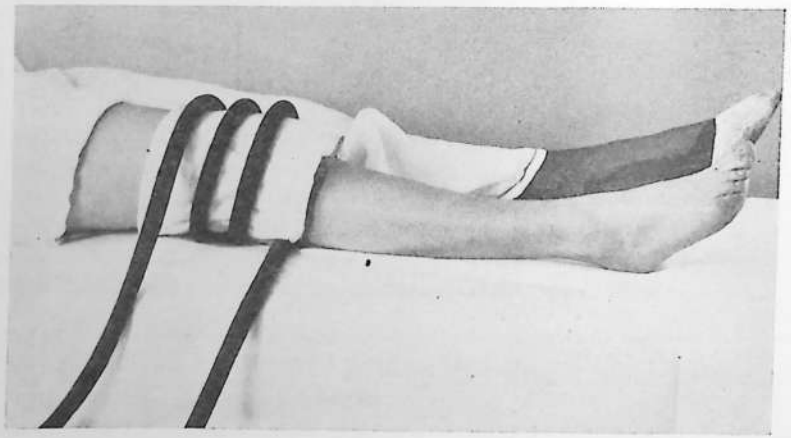

FIG. 62.—Short wave diathermy to the knee. (Coulter, courtesy of Med. Clin. North America.)

Short wave diathermy was introduced therapeutically only within the past fifteen or twenty years.[48,56] The two types of apparatus commonly used are the vacuum tube or valve machine and the spark gap device. Many physical therapists seem to prefer the tube apparatus although a properly constructed spark gap machine may be effective. In the selection of such equipment, the physician should purchase those machines which have been approved by the Council of Physical Therapy of the American Medical Association. The free booklet of this Council listing the accepted machines may be procured by any physician.

Three methods have been used to apply these currents locally: (1) with a contact metal electrode; (2) with a high frequency alternating electric field electrode; or (3) with a high frequency electromagnetic field coil.

The *conventional diathermy* utilizes the contact metal electrode, the frequency of oscillations being from 500,000 to 2,000,000 cycles per second. A frequency of from 10,000,000 to 100,000,000 oscillations per second is used by the second method; in this form (short wave diathermy) the metal electrodes are insulated with rubber or felt, and when applied are usually separated from the skin by a $\frac{1}{2}$ inch layer of towel or felt. This layer of toweling between the electrodes and the skin prevents burns (as surface moisture cannot accumulate into small conducting areas). The double cuff electrodes and the air spaced electrodes are superior to the pad electrodes in heating deep tissues.

The other method of heating tissues by high frequency currents is by *electromagnetic induction*. This is believed to be the most efficient and convenient method for using short wave diathermy. A flexible, heavily insulated cable carries the current to the patient. This cable is wound around the part to be treated in coils or loops. The part being treated is separated from the coil of cable by a dry towel.

The Council of Physical Therapy of the American Medical Association has found that "*burns may occur with any method of applying short wave medical diathermy as well as with the contact metal electrodes of conventional diathermy.*"[14] *This Council was unable to accept the claims of manufacturers of these machines that burns were impossible as evidence was available that such burns did occur. These burns are slow to heal* and resemble those caused by electrocoagulation.

In many instances, such burns are due to collections of moisture between the skin and the electrodes resulting in a concentration of energy at these points. Patients who have been burned have experienced a distinct sensation of the burning. *The operator should always be informed at once by the patient if any burning sensation is noted.*

When diathermy is used, all metallic substances (knives, keys, money, watches, etc.) must be kept out of the field of operation and chairs and tables must have no metal parts. Beds with inner spring or wire mattresses are a fire hazard. When diathermy is employed, the degree of heat must be limited so that no unpleasant sensation or pain is produced. Inflammatory tissues tolerate less heat than normal tissues.[20]

It is usually conceded that the **effects produced by diathermy or short wave therapy are to be attributed solely to the generation of heat, and not to any specific biologic or bactericidal action.** With the introduction of relatively safe and efficient apparatus, diathermy has had wide vogue in recent years in the treatment of many subacute and chronic, painful, inflammatory conditions, especially in deep-seated portions of the body.

It has been claimed that short wave diathermy is simpler, safer and gives more effective heat penetration than ordinary diathermy, and that ultra-short wave therapy results in selective heating and specific effects upon acute inflammatory processes in the skin and body cavities. Kovács

questioned these claims and suggested that beginners in this field employ a standard type of diathermy machine rather than a flimsy make of short wave apparatus.[24]

Superiority of Short Wave Diathermy Over Conventional Diathermy.—Coulter believes short wave diathermy is the most efficient method of applying deep heat to human tissues. He no longer employs long wave diathermy machines for medical diathermy because these are more apt to produce burns, more difficult to apply and are less efficient in producing deep heat than short wave diathermy machines. Using efficient machines and proper technic, he has found a rise in temperature in the quadriceps muscle 2 inches below the surface of the skin of 0.25° F. with a hot water bottle, 2.25° F. with conventional (long wave) diathermy, and 7° F. with short wave diathermy.

Others have not seen marked differences between the two as heat production seems to be the essential feature of both; however, there seems to be a deeper penetration of heat with the short wave apparatus, and the heating may be more uniform. The short wave apparatus may be applied more readily than the conventional diathermy. However, with conventional diathermy, the dosage is easier to control than with the short wave machine; *this lack of any convenient method of determining dosage is a disadvantage of the short wave machine.* The dose must be regulated by the patient's skin tolerance to temperature. The effects of the various wave lengths between 3 and 30 meters have been found to be approximately the same.

Short wave diathermy does not produce any specific physiologic effects other than those attributable to the heating of the tissues.

Suggestions in the Application of Short Wave Diathermy[12]

No unpleasant or painful heat sensation should be produced.
Remove all metallic objects from the area of treatment, including knives, keys, money, watch, etc.
The patient must not be allowed to regulate the current: a trained technician should be nearby at all times.
Exercise great care in patients with cardiac disease; if tachycardia occurs, decrease the current.
Use chairs and tables without metal parts for treatment; an inner spring mattress must not be used because enough heat may be generated in the springs to ignite the mattress.
Pain occurring while treating a region with a bony projection may be due to overheating of the periosteum; in such cases treatment should be stopped at once.
Turkish toweling should be used to separate the electrodes from the skin; the toweling must not become too moist (as from perspiration).
Metal must *not* come in contact with the patient or the machine.
Do not treat through clothing.
In treating the head, therapy must be stopped if untoward symptoms occur

Diathermy and Short Wave Diathermy

The term "short wave diathermy" is used to denote treatment with wave lengths from 3 to 30 meters; it includes both short and ultra-short wave therapy.[22]
Advantages of short wave diathermy: deeper, more uniform heating, greater ease of application and less likelihood of burns. *Disadvantages:* no accurate method of controlling dosage except by the patient's sensations.
Other than noted above, there is no great difference between the effects produced by conventional diathermy and short wave diathermy.[27]
The preferred type of apparatus is one using the vacuum tube with high wattage output; a good spark gap apparatus is also effective.
The effects of the various wave lengths from 3 to 30 meters are approximately the same.
There results from diathermy or short wave diathermy no specific physiologic or bactericidal effects not attributable to heat; there is no demonstrable selective thermal action of either.[29]
At present, the indications for the use of diathermy or short wave diathermy are the same.
Diathermy and ultra-short wave diathermy are contraindicated in patients with acutely inflamed joints.
The average effective duration of a diathermy or short wave treatment is from twenty to thirty minutes.
Never apply the electrodes to the broken skin; great caution must be taken in applying these over scar tissue.
Diathermy and short wave machines should be operated only by individuals trained in this work; it is advisable that one obtain special training in this field rather than rely upon the instructions given by the salesmen. An up-to-date text on the subject should be available for points of detail in its application.[6,25,36]
The greatest usefulness of these forms of therapy in rheumatoid arthritis lies in the chronic stage of this disease; in these patients, diathermy or short wave diathermy may be applied to the affected joints daily or two or three times a week, followed by massage.
Medical diathermy in chronic arthritis should always be supplemented by other forms of heat two or three times a day. The first several doses should be of low intensity and for a short period. In some cases of arthritis, diathermy seems to aggravate the local symptoms; if this continues, such treatment must be abandoned.

Disadvantages of Short Wave Diathermy.—These include burns (if improperly used), and the difficulty of properly measuring the dosage.[47] However, the danger of burns is less likely with the short wave apparatus than with conventional diathermy.

Is Diathermy Essential for the Proper Treatment of Rheumatoid Arthritis?—No, diathermy is contraindicated in the acute stages. In the chronic stages, subjective and objective improvement following the judi-

cious use of bakers or infra-red therapy may equal that obtained by the expensive short wave machines. It is true, however, that short wave diathermy will heat deeper tissues and will often have a good psychic effect upon the individual; it is especially valuable in low back strain, subdeltoid bursitis and degenerative knee or hip joint disease. In degenerative joint disease of the spine, diathermy, massage and support are palliative measures which often prove useful. Head traction is often beneficial when the cervical spine is involved. However, the physician who cannot afford to install a diathermy machine may obtain good results in his patients with chronic arthritis by other less expensive forms of apparatus, as described previously.

Contraindications to Local Diathermy[12]

Acute infectious arthritis (and certain other acute inflammatory processes, such as an acute non-draining cellulitis).
Through the pelvis, lower back or abdomen during menstruation or pregnancy.
Over a suspected malignancy.
Any condition with a tendency to hemorrhage (as gastric ulcer).
Over areas of anesthesia or diminished sensation (as in peripheral nerve injuries).
Where simpler methods of applying external heat give good results.

Diathermy in Subdeltoid Bursitis.—Satisfactory results have been obtained in a majority of cases of **subdeltoid bursitis** by diathermy, massage and active and passive exercises, emphasizing abduction. One must always be on the lookout for a possible rupture of the supraspinatus tendon, in which disorder the patient cannot begin abduction with the arm at the side of the body or has marked difficulty in performing the first 20° of abduction.

In patients with a **"frozen shoulder"** which is of short duration, the orthopedist must always use extreme caution during the manipulation to avoid fractures or dislocations. It is advisable to obtain a roentgenogram in all cases following manipulation. Following the manipulation, the arm should be placed in a splint in abduction and external rotation or should be tied (in this position) to the head of the bed. Hot packs will help alleviate pain and muscle spasm for the first few days following manipulation. Passive and active motion may be begun in most cases about twenty-four hours following manipulation but the arm should be kept in abduction and external rotation when not in use. *The use of diathermy and massage will hasten the period of convalescence.* The treatment must be persisted in and *exercises must be prescribed for months* following the manipulation if a permanently good result is to be anticipated.

Faradism and Galvanism.—A galvanic (or continuous) current may be produced by a dry-cell battery with sufficient voltage to overcome the resistance of the skin (a bad conductor) and to stimulate the muscles and nerves beneath; a faradic (or interrupted or induced) current differs from the galvanic in that it flows intermittently and for very short periods of time.[46]

Faradism may be used to promote muscular contraction in cases where active muscular movement may be impossible. It is a valuable adjunct to active movements and exercise and aids in the development of a particular muscle group. It may be used in most forms of muscular wasting. It must not be substituted for active use of the muscles. The Bristow-Smart coil is less painful than the ordinary faradic coil and a surging current is usually employed.[9] It may be used for twenty to thirty minutes with rest periods during the treatment.

Faradism stimulates muscles through their motor nerves rather than by direct action on the muscle fibers. In order to stimulate any muscle most efficiently by this means, the faradic current is applied to the motor point (the point of entrance of the nerve into the muscle); this results in a maximal contraction of that muscle. Whole groups of muscles may be stimulated by applying the electrode over a nerve trunk.

Galvanic currents stimulate motor nerve fibers only at the time of opening and closure of the current (if the strength of the current remains the same). Galvanism produces contractions in muscle fibers even when the nerve may be degenerated.

It is of use in the re-education of groups of small muscles as the interossei or lumbricals of the hand. If the positive pole is applied to the painful joint, one produces vasoconstriction and reduction of nerve irritability. If the negative pole is applied to the joint, counterirritation results from vasodilatation and increase of nerve irritability.[41]

With galvanic currents, treatment is continued for thirty to forty-five minutes using as much current as the patient can comfortably bear; this produces a prolonged cutaneous hyperemia and reflex dilatation of the deeper vessels. It is often used as a form of counterirritation in rheumatoid arthritis.

Sinusoidal Current.—This is a slow alternating current used to produce muscular contraction. It may be used in slow, rapid or surging intensity. It is usually given by means of the Schnee 4 cell bath, and although uncomfortable, may be efficacious in patients with pronounced muscular wasting and sluggish peripheral circulation.[9] In practice, 2 leg and 2 arm baths are half filled with water and placed in an electric circuit. One limb is in each bath, and galvanic, sinusoidal or faradic current is passed through the body. The sinusoidal current is useful in this form from arm to arm in treating marked muscular atrophy in rheumatoid arthritis. The duration of the treatment averages from twenty to thirty minutes several times a week.

High Frequency (Oudin) Treatment.—This is obtained from the single high voltage (oudin) terminal of a spark gap diathermy apparatus executed through a condenser (vacuum glass) tube. Moving the electrode back and forth in slight contact with the skin produces mild or heavy sparks varying with the strength of the current and the distance of the electrode from the skin.[28] This acts in a manner of counterirritation and may be

useful in diffuse neuritic pains or subacute or chronic arthritis involving several joints. It is usually given after the application of heat.

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CHAPTER XV

OCCUPATIONAL THERAPY

Occupational therapy includes any prescribed mental or physical activity which may aid in recovery from a disease or injury and render the convalescence more pleasant. This may be of value at times in keeping the mind of the patient occupied so that he does not constantly think about his illness; in other cases, the work involved results in rehabilitation of the individual. It is important that individuals with chronic forms of arthritis obtain mental and physical diversion by means of occupational therapy; the latter, in addition, will be of great assistance in restoring muscular power and increasing the range of motion in the involved joints.[1,3,5]

Purposeful activity which achieves something is usually preferable. *Crafts* which have been *used frequently in occupational therapy* include jig-saw puzzles, weaving, rug making, bookbinding, metal work, needle work, crocheting, knitting, celluloid etching, block printing, chair caning, simple wood work, gardening, pottery, leather work, simple jewelry work, basketry, typewriting, etc. Other simple measures which Hench and Krusen have recommended include ironing, sweeping, raking leaves, using a foot power sewing machine, washing dishes, polishing silver or shoes, shelling peas, using a screw driver, making beds, shooting marbles, piano playing, playing parchesi or crokinole, riding a stationary bicycle, clay modeling, etc.[6]

Occupational therapy should be prescribed as carefully as physical therapy, using those forms of work or play which will result in the desired exercise for the involved parts. Many patients who will not follow specific directions concerning ordinary exercises to be prescribed at home, will perform the desired occupational therapy (which often produces the same desired end-result[7,8,9]). One must *be careful* that the *patient does not overwork.*

Occupational therapy is an objective type of treatment prescribed by the physician in an effort to hasten the patient's recovery from injury or disease or to contribute to his adjustment to hospitalization. Occupational therapists are trained to carry out the prescription of the physician by selecting and adapting activities which meet the patient's psychologic and physical needs. These activities must be interesting enough to the patient to induce him to participate actively in these efforts. While the patient is under observation, the occupational therapist should provide the physician with information as to the patient's reactions and progress.

In the handling of the arthritic patient, there must be close coöperation between the internist, the orthopedist, the physical therapist and the occupational therapy department. These individuals should frequently exchange information in an effort to provide the most intelligent and

effective therapy in an effort to return the patient to normal activity in the least possible time. The physician, in general, should furnish both the physical and occupational therapy departments with a prescription regarding treatment, after consultation with these departments. In the ideal set-up, rounds should be made as often as possible by the four interested groups: internist, orthopedist, physical therapist and occupational therapist to permit an interchange of ideas.

Occupational therapy is a well recognized form of treatment for the arthritic patient and should be included as a definite part of a hospital regimen in treating these individuals. Long before active exercise is indicated, this form of therapy, which holds as its basic principle, the psychology of organized activity, mental or physical, may contribute greatly in preserving or restoring morale, in the correction of bad posture and above all, *in reducing the fear element*, which is a potent factor to be dealt with.

When the lower extremities only are affected and the patient must remain in bed with complete rest for the affected parts, some *diversional occupation* should be given *which will not only interest the patient but will serve to inspire confidence and a degree of hope.* Such a procedure is definitely helpful and prepares the way to the next step which is active exercise as prescribed treatment.

The field of occupational therapy covers a wide range, including the playing of games which require little or no exercise, on through to strenuous work on large rug looms, block printing equipment and the bicycle saw. The therapist should not be at a loss to find the right course to follow. This therapy should be prescribed as early as possible, *because anxiety and the dread of being hurt by any form of exercise is much more difficult to cope with if diversional therapy does not enter into the program before the more strenuous type of activity is introduced. Once a patient's confidence has been won, he becomes far more coöperative and is usually eager to follow suggestions.* He will endure discomfort better if he has an intelligent understanding of the wisdom of graded activity.

In the active treatment work as ordered for this type of patient, *the psychology of progressive exercise by means of interesting occupation* holds a place of utmost importance. The work prescribed because of its adaptability to the limitation of the individual case becomes more and more interesting and means far more to him than mere exercise. *It becomes a means of self expression and develops his personality, in his ability to create.*

To the bed patient, for whom occupational therapy has been prescribed, a mild type of craft work is given, always chosen to meet the definite order, but at the same time, with consideration for the desire and will of the person to be treated. *Various pieces of equipment are available which are adjustable to the bed and position of the patient and such occupations as colonial mat weaving and cord knotting may be undertaken.* These adjustable frames support the work for the patient and he may be concerned wholly with the development of his craft and is in no way hampered by the anxiety of holding equipment as he works.

It must be clearly understood by the therapist that the work should be supervised so that correct posture may be maintained and that the activity may not go beyond the fatigue point. *Short periods of activity with intervening rest periods* is a definite principle of occupational therapy.

Having established a contact with the patient in his room or ward, he is later referred to the work shop. There is practically no limitation which may not be met and dealt with to some advantage for the arthritic. He finds himself in a *cheerful atmosphere of activity* and almost immediately, the patient referred for specific exercise, feels assured and his morale is noticeably stepped up.

Large looms, graded for the effort required in their operation, offer exercise for the upper extremities. Loom weaving holds a definite interest for the patient and at the same time may produce graded exercise for prescribed joint and muscle action for arms and shoulders. *For shoulder motion, wheel cushion weaving* is first given on a frame adjuster to the particular limitation and is an easy step to the upright loom which entails more strenuous activity for shoulder flexion and abduction. Any of these crafts are also adaptable as exercise for the flexors or extensors of the fingers. Other equipment available for active treatment work for the upper extremities is that used in *block printing*, particularly recommended for action of wrist, forearm and fingers, *cord knotting* and all of the tools to be found at a work bench: hand saws, brace and bit, and planes for shoulder and elbow action, as well as hand drills, and the ordinary screw driver which offers pronation or supination of the forearm, depending on the drive of the screw.

For the lower extremities, the most valuable piece of equipment is the *bicycle saw*. This is adjustable in the length of the pedals and the position of the seat and may be graded for resistance.

In treating the arthritis patient by occupational therapy, the work must be prescribed and the order should carry with it the exact motion required and a suggestion as to the approximate length of the exercise period.

Occupational therapy technicians must be properly trained in an accredited school so that they receive training in the fundamental sciences, learn to understand the individual and the physical and mental diseases and the injuries which they may be called upon to assist in treating. The occupational therapist must also have a broad training in the activities or occupations which may be applied to motivate the patient and must have a knowledge as to which should be selected and changed to control the desired mental processes and reactions of the patient or his specific needs for exercise. She must coördinate all available resources in an effort to hasten the mental, physical and social rehabilitation of the patient; to this end, her imagination and ingenuity will be taxed in adapting processes to carry out the physician's prescription and to aid in the patient's recovery.

Occupational therapy includes work which is planned in an effort to prepare the individual for his return to his previous occupation; intelligently planned activities which will aid in restoring muscular and articular function, building up the patient's physical endurance, improving his general condition and assisting in his mental rehabilitation if this is necessary; as well as rest and controlled general exercises and diversions.

The effect of occupational therapy may be physical, economic, social or mental. Physically, it is of value in improving muscular power and restoring function to disabled joints. *Economically*, occupational therapy will detect skills and capacities for vocational guidance, permitting those individuals who may be hospitalized permanently to participate in the activities of that institution. *Socially*, the morale of the patient is improved greatly, as well as that of the other patients in the ward or institution. *Mentally*, occupational therapy offers encouragement to the individual, occupying his mind and easing any tendency for emotional stress and restlessness.[4]

Occupational therapy in the arthritic patient must include adequate rest during the acute stage of the arthritis by centering the attention of the patient objectively in constructive channels in an effort to reduce anxiety, tension and nervousness. When the arthritis has become subacute, carefully graded exercises will preserve function and aid in restoring muscular power and joint motion. During the long period of convalescence, occupational therapy will tend to prevent the development of a neurosis and convince the patient that he is not helpless and that usually he can be taught an occupation which will assist him in becoming independent.[2]

Most arthritic patients are afraid to use painful joints because of the resulting pain on motion. Because of this, the average patient, if left alone, would develop flexion attitudes with flexion of the wrists and fingers, ulnar deviation of the hands, adduction and internal rotation of the humerus, flexion of the head, hips and knees, a flat chest and very little, if any, use of the intercostal muscles in breathing. Such positions further aggravate the joint phenomena, reduce the circulation and tend to develop flexion contractures and deformities.

Unfortunately, pillows are frequently placed under the knees leading to further flexion contractures, or under the thighs with subsequent subluxation of the knee. Foot drop occurs very often in the average arthritic patient at home because of the pressure from tightly drawn bed clothes; rotation of the hips is another consequence of this.

During the acute stage of an arthritic process, the joints should be held with proper support (often plaster shells or casts) in optimal positions, with daily motion (2 or 3 guided movements) so that dense adhesions do not occur.

The arthritic patient must be stimulated to exercise during the subacute stages of his disease as he is afraid to move his joints to any great extent because of fear of pain. Carefully selected activities should provide the necessary stimulus and incentive for such motion, permitting the patient to relax, lose his fear of pain and perform such exercise as will produce the maximal useful function. Occupational therapy will thus aid in muscular and joint coördination and hasten the return of function.

Occupational therapy is also of value in the arthritic to combat dis-

couragement and the development of neurosis by employing interesting constructive activities to satisfy any emotional outlets, while at the same time providing the desired physical exercise.

Flexion deformities are frequent at the hips and knees, but can be prevented if the patient is not permitted to place a pillow beneath his knees; it is often necessary to employ plaster splints or a plaster shell from the low back to the tips of the toes to splint the affected joints, permitting several painless motions each day in an effort to prevent the development of dense adhesions. Following this, when the acute stage of the disease has passed, non-weight-bearing exercises may be employed, and various forms of apparatus may be devised to strengthen the involved muscles. A knee push may be constructed which can be operated in bed by the patient. The *bicycle jigsaw* may be used to increase the strength of the flexors or extensors of the knee and hip. This may be built with special pedals with adjustable short and long shafts for the pedals and the seat; extension is increased by pushing the pedals while pulling the pedals around increases flexion. This latter may be done with the feet strapped on the pedals so that they may be pulled in reverse action.[4]

Deformities of the knees and hips produce pain and deformity in the feet and ankles because of the additional strain on these parts. Foot drop may be produced by lack of proper support for the feet or by pressure of the bed clothes. In these patients, it is necessary to regain flexion of the toes and ankles. This may be done by non-weight-bearing exercises. The feet may be exercised by having the patient pick up marbles with his toes, while flexion may be aided by use of the bicycle jigsaw in reverse (with the feet strapped to the pedals).

Involvement of the *shoulder* in the arthritic process often leads to forward drooping of the shoulder, with internal rotation of the humerus and limitation of flexion and abduction of the upper arm. The posture is usually poor and the chest is often flat. The ingenious occupational therapist may employ weaving, carpentry and the tying of sailors' knots to improve function in the upper extremity.[4]

If the *elbows* are involved, a flexion deformity frequently occurs as the elbow is strained when the individual pulls himself up or holds on to tables or chairs. These patients are given exercises to increase extension of the elbow and supination of the forearm.

Ulnar deviation and subluxation of the *fingers* should be prevented by proper splints extending from the upper part of the forearm to the tips of the fingers, with the wrist in a cock-up position and the fingers in slight flexion. If flexion deformities of the fingers are present, one should avoid fine handiwork. One should avoid muscle fatigue which may follow prolonged muscular contraction; it is preferable to have activity which employs muscular contraction followed by relaxation. If the wrist is involved, craft work may be done while the wrist is in the cock-up splint as such work would exercise the dorsiflexors. The splint may be discarded when muscular power returns and the individual can maintain dorsiflexion without the splint.

When the patient is referred to the occupational therapist, one should specify the type of the arthritis, the stage of the disease, the amount and type of activity which may be indicated for any specific joints, the length and frequency of the exercise period and general or special cautions which should be observed, including cardiac involvement and the avoidance of function in certain joints. The occupational therapist should also be aware of the patient's emotional status.

There are at present 13 *approved schools for occupational therapy* technicians in the United States and Canada. Approved schools of occupational therapy have been established in Boston, Philadelphia, Toronto, St. Louis, Kalamazoo, Milwaukee, etc.

These individuals are trained adequately in the crafts, arts, medical subjects and biologic sciences. Entrance requirements in these schools usually demand at least a High School education and often one year of college. Tuition averages $250 per year. The duration of the course in most of these schools is three years; the school at the University of Toronto completes its course in two years.

Most of these schools are affiliated with colleges, including the University of Pennsylvania; Ohio State University; New York University; University of Toronto; University of Kansas; Washington University (of St. Louis); Columbia University; University of Michigan; Western Michigan College of Education; William and Mary College; Mount Mary College and the Milwaukee-Downer College. The Boston School of Occupational Therapy, 7 Harcourt St., Boston, is not yet affiliated with a college. In 1944, there were about 250 graduates from these schools.

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CHAPTER XVI

MASSAGE IN RHEUMATOID ARTHRITIS

THIS is one of the most useful, easily obtained and frequently neglected methods of physical therapy in arthritis. *Proper massage requires the assistance of one trained in this field.*[1,11,13] Unfortunately some physicians

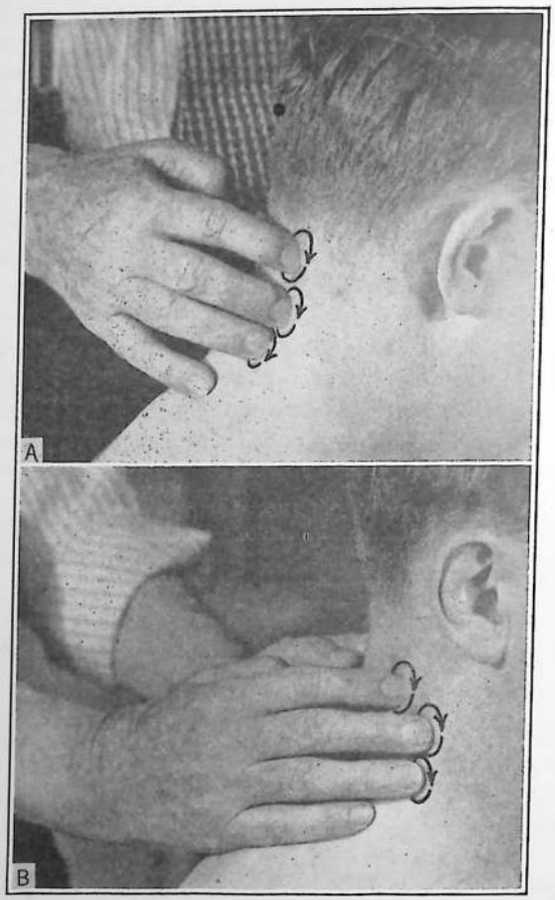

FIG. 63.—*A*, Friction; the fingers are placed alongside of the cervical spine, slightly lateral to the spinous processes. The head is supported by the opposite hand to enable the muscles of the neck to relax. *B*, Friction of the neck. The fingers move in small circles. The underlying soft tissues move with the movements of the operator's fingers.

order patients with chronic rheumatoid arthritis to "apply heat to the joint and massage it once in a while." Massage is a double-edged weapon which may result in marked improvement if properly performed, but in further damage when given indiscriminately.[14]

Massage is a medical art which was founded on physiologic and anatomic knowledge; it is not a tool of charlatans or a black art. It is not mere rubbing but is a series of manipulations performed best manually with an exact technic, on the surface of the body. Physiologic effects are produced by mechanical stimulation and by reflex action.

A physician would not consider sending a patient to the drug store with an order to "get a sedative"; however, this is equivalent to the order frequently given, "massage the joint every several days." In fact, the latter is worse, for the druggist may come to the patient's aid in the case of a sedative, whereas the patient has usually no more idea of how to obtain massage than the young bride has of making a mustard plaster.

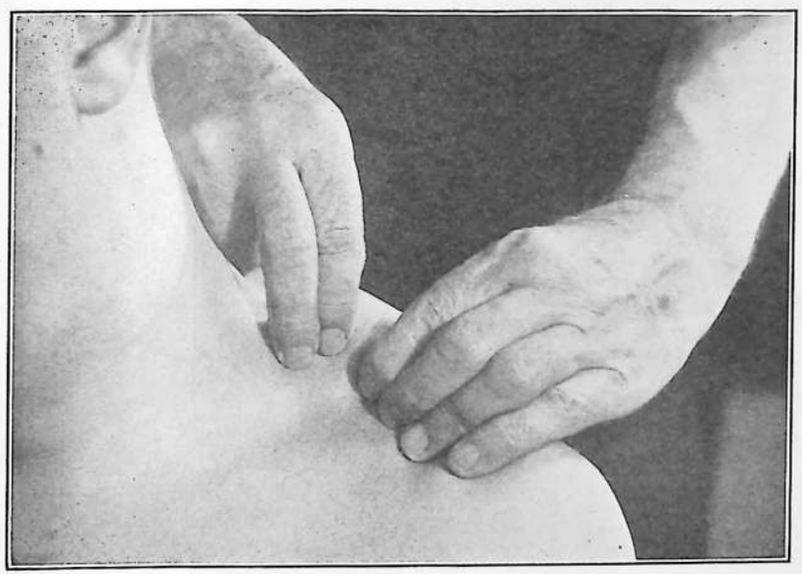

FIG. 64.—Pinching pétrissage: the soft tissues are being picked up by one hand of the operator while the fingers of the other hand perform circular movements of the raised soft tissues. This is especially valuable in fibrositis.

Another reason for the lack of utilization of massage by the general practitioner is that he had not been trained in its indications, contraindications, and in the proper methods of performing massage.[16] Some physicians consider it below their dignity to massage an involved limb or even to instruct a member of the family in this procedure. Massage given by trained physical therapists may be expensive, but is desirable, if possible; failing this, our next aim is to train the general practitioner not only in the mechanism of massage, but in its usefulness in various conditions.

Mechanically, massage assists the circulation in the venous system and aids in the movement of lymph; massage may also produce tension on a structure to free or stretch it.

The vasodilatation resulting from massage assists in the absorption of

inflammatory or waste products. Massage is very useful in preventing muscular atrophy.[10]

Some writings on arthritis are vague in their description of the subject of physical therapy. It is difficult to portray technics of massage; however, as many physicians have not had special training on this subject and do

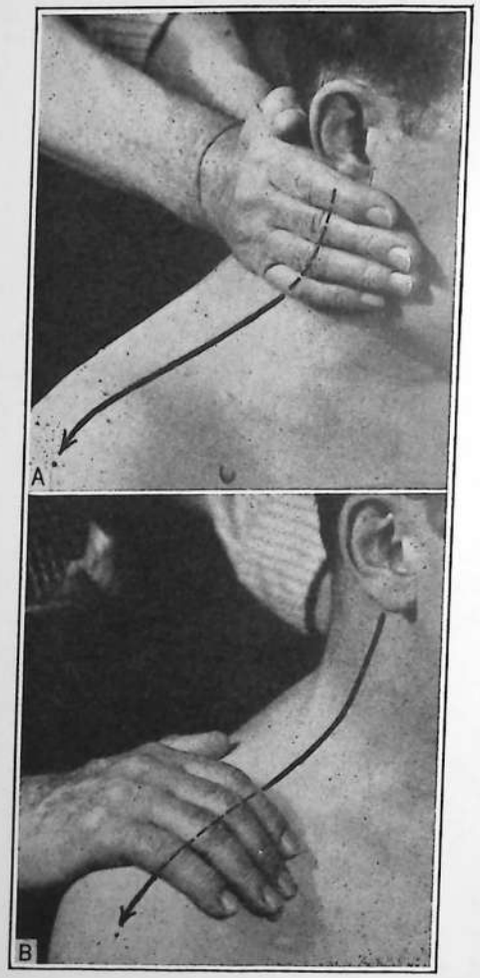

FIG. 65.—*A*, Effleurage of the neck. This figure illustrates beginning of movement, which is seen almost completed in next figure. *B*, End of movement of effleurage of the neck, begun in *A*.

not have time available to obtain experience in this field, one must endeavor to describe these insofar as such is possible. Those who are interested in the theory and finer details of massage may consult the sections on this subject prepared for the Council on Physical Therapy of the American Medical Association.[7]

There is evidence to suggest that there is no lymphatic drainage except that which accompanies the massage resulting from movements of the part. If a joint is inflamed so as to prevent or greatly limit motion, there is inadequate lymphatic drainage of this area.

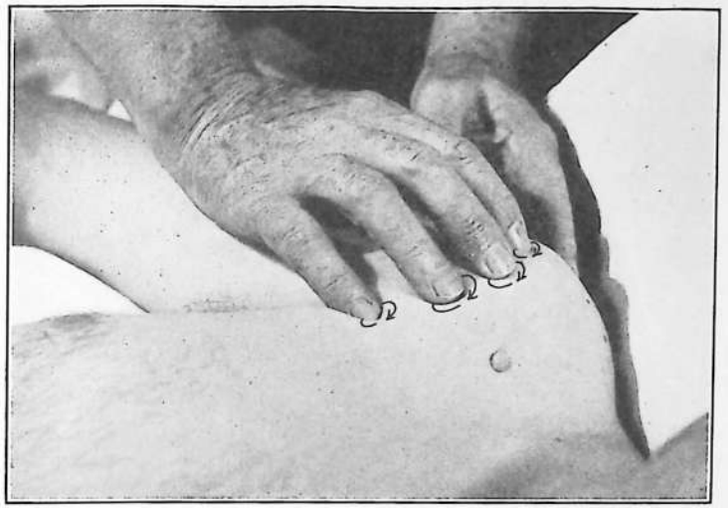

FIG. 66.—Frictions at the shoulder, performed with both hands simultaneously.

Indications for Massage in Rheumatoid Arthritis

Unless contraindicated, every patient with non-acute rheumatoid arthritis deserves proper massage.

Contraindications to Massage in Rheumatoid Arthritis

1. An acutely inflamed joint.
2. Temperature above 100° F.
3. Suspicion of local malignancy.
4. Phlebitis (because of the danger of embolism) or lymphangitis.
5. *Increased pain, swelling or stiffness in a joint persisting (more than two hours) following massage.*
6. Acute burns or acute dermatitis.
7. Persistent hypertension or advanced atheroma of blood-vessels.
8. Suspicion of osteomyelitis or tuberculous lesion of bone.
9. Abdominal massage is contraindicated if intra-abdominal organic disease is present.

Hyperesthesia or pain associated with inflammation may result from the liberation of a chemical substance by the nerve endings;[3] if this takes place over a considerable area, the part becomes hyperesthetic. Massage removes lymph and this hyperesthetic material, whatever it may be, for only in the presence of massage does anything get into the lymphatics.

There is much disagreement among rheumatologists as to the best form

of massage to employ under various circumstances. *The following directions apply only in a general manner and each arthritic individual will require variations from this depending upon his general and local conditions.*

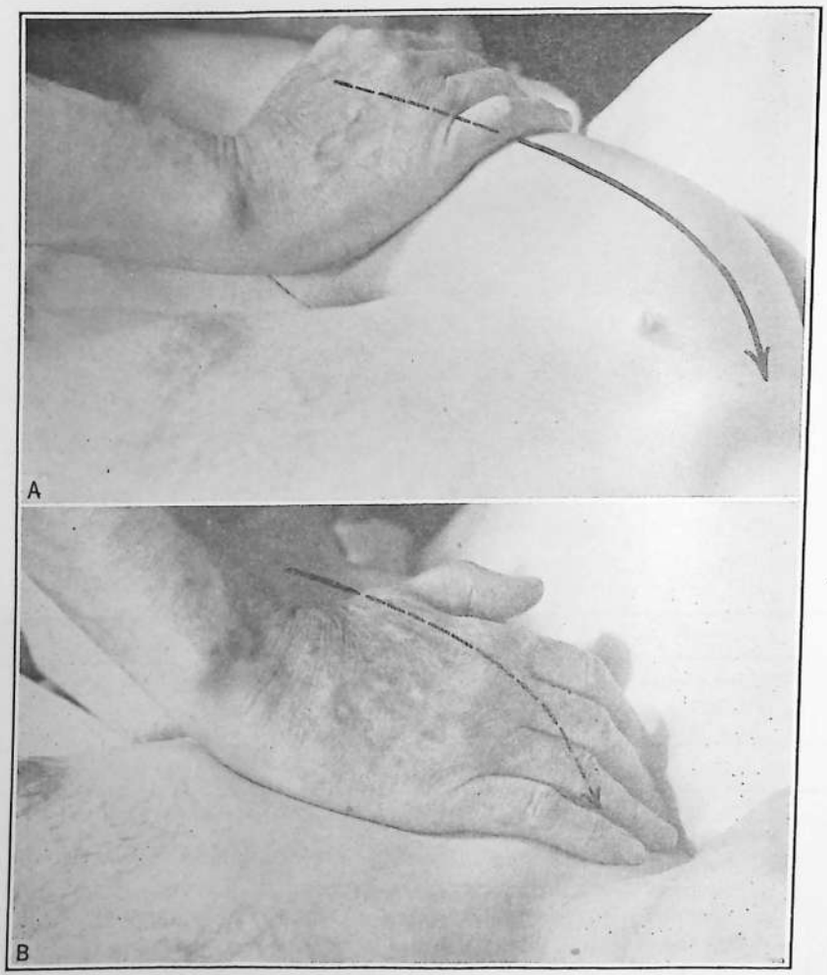

FIG. 67.—*A*, Effleurage of the shoulder and upper arm, illustrating the necessity of even pressure. The hand is molded so that it always conforms to the contour of the part over which it is gliding. This picture illustrates beginning of this motion. *B*, Illustrating end of motion begun in *A* (effleurage of the shoulder and upper arm).

In patients with chronic rheumatoid arthritis or with degenerative joint disease, massage should consist of light to deep stroking movements (effleurage) of the muscles with only light stroking carried out over the joint itself. Kneading, light or deep (pétrissage) may be used proximal and distal to the involved joint and frictions with the finger tips are often of value when performed in the immediate vicinity of the joint.

In the average patient with chronic rheumatoid arthritis, the involved

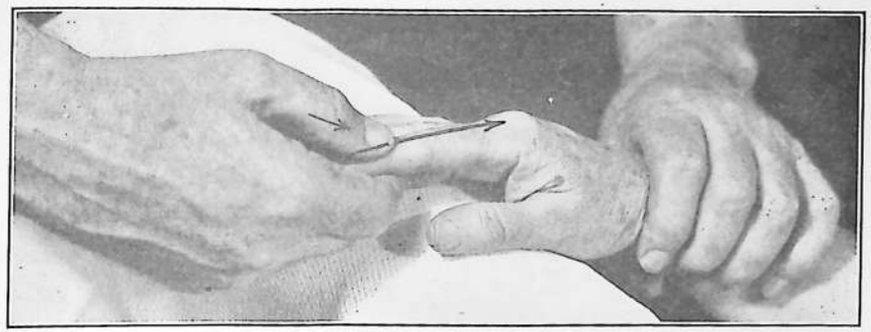

FIG. 68.—Effleurage of a finger. Stroking is performed by the operator's forefinger on one side of the digit and the thumb on the other. The operator's other hand supports the arm and hand.

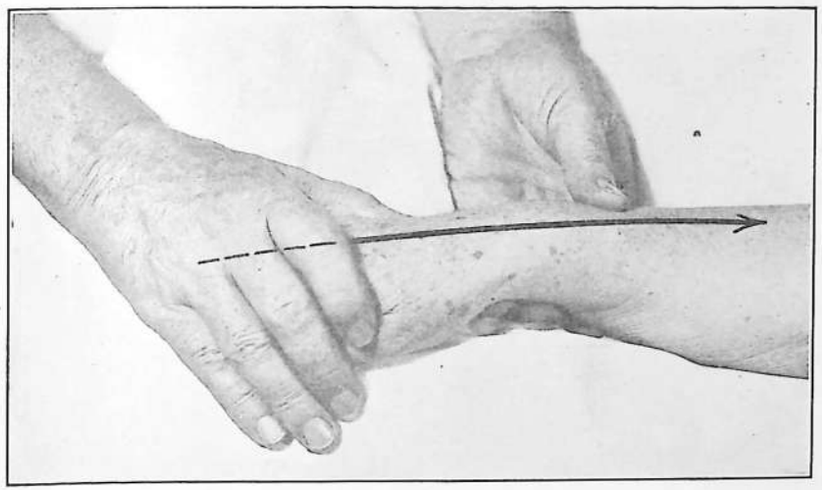

FIG. 69.—Effleurage of the hand. The operator molds the hand to the part, and supports the extremity, with his other hand.

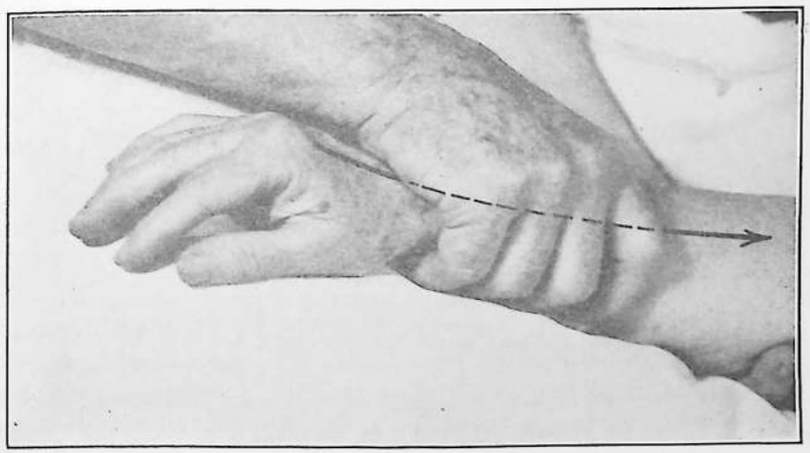

FIG. 70.—Effleurage of the hand and forearm.

limb may be massaged for ten to fifteen minutes daily, using mainly stroking movements (about 15 to 20 strokes per minute). Massage must

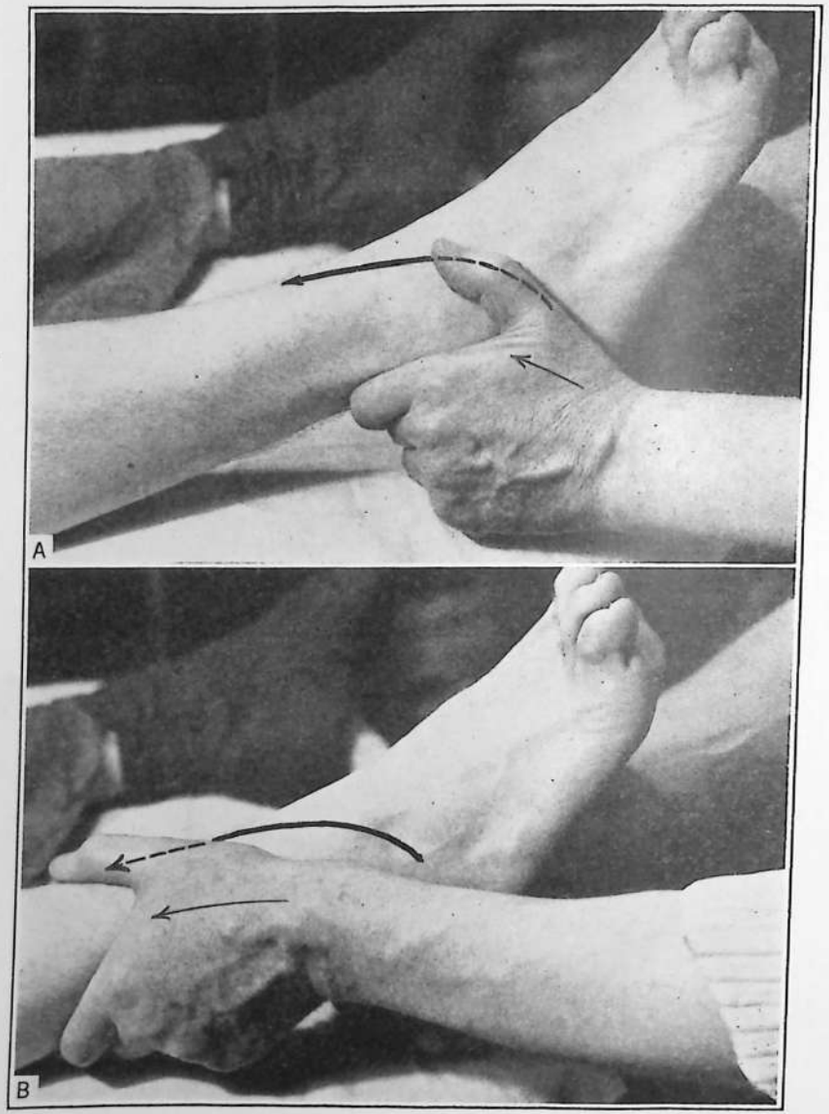

FIG. 71.—*A*, Massage of the ankle joint; it is very important that the movements be performed around (instead of over) the malleolus. Pressure is exerted by the thumb or thenar eminence. This figure illustrates beginning of the movement. *B* indicates end of movement begun in *A:* massage of the ankle. The thumb goes around, not over the malleolus; pressure is not exerted upon the malleolus.

not increase the inflammation present in the joint and should thus be practised in the neighborhood, but not immediately over, the affected

joint. **Movements are made in the direction of the venous and lymphatic flow,** *and only very light massage is permitted over the joint itself.*[15]

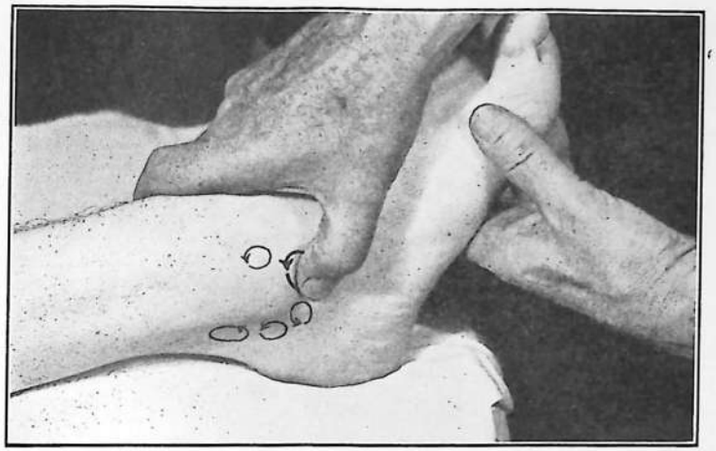

FIG. 72.—Friction (circular movements) of the thumb around the external malleolus.

General Regimen of Massage for the Patient With Rheumatoid Arthritis

1. *Acute stage:* No massage.
2. *Subacute stage:* Begin with gentle stroking and later use deeper stroking motions, kneading and friction of the surrounding muscles for ten minutes twice a day; superficial very light stroking may be begun over the joint.[5] This is best performed after at least fifteen minutes of heat to the tissues. **Never cause pain.** The time required for such massage is approximately from five to ten minutes for an arm, ten minutes for a leg, and from ten to fifteen minutes for the back. If multiple joints are involved, all of the areas may be included in the massage. **Always give too little, rather than too much massage.** In involvement of the arm, massage should be begun from the elbow and extend toward the shoulder, then from the wrist to the elbow, and last, from the finger tips to the wrist. If the leg is involved, massage begins from the knee toward the hip to open the vascular channels, then from the ankle to the knee, and finally from the toes to the ankle. In lesions of the back, begin the massage on the sides of the spinal column from the buttocks toward the neck, and then from the buttocks toward the axilla.
3. *Chronic stage:* As in subacute arthritis.

Precautions must be observed in the ordering and performance of massage **to avoid legal action.** The patient must not be exposed to a chill after heat has been applied. If an extremely hairy part is to be massaged, irritation must be avoided so that a folliculitis or pustular infection does

not result; in some of these individuals, with the patient's consent it is advisable to shave the part. In addition, the physician must remember

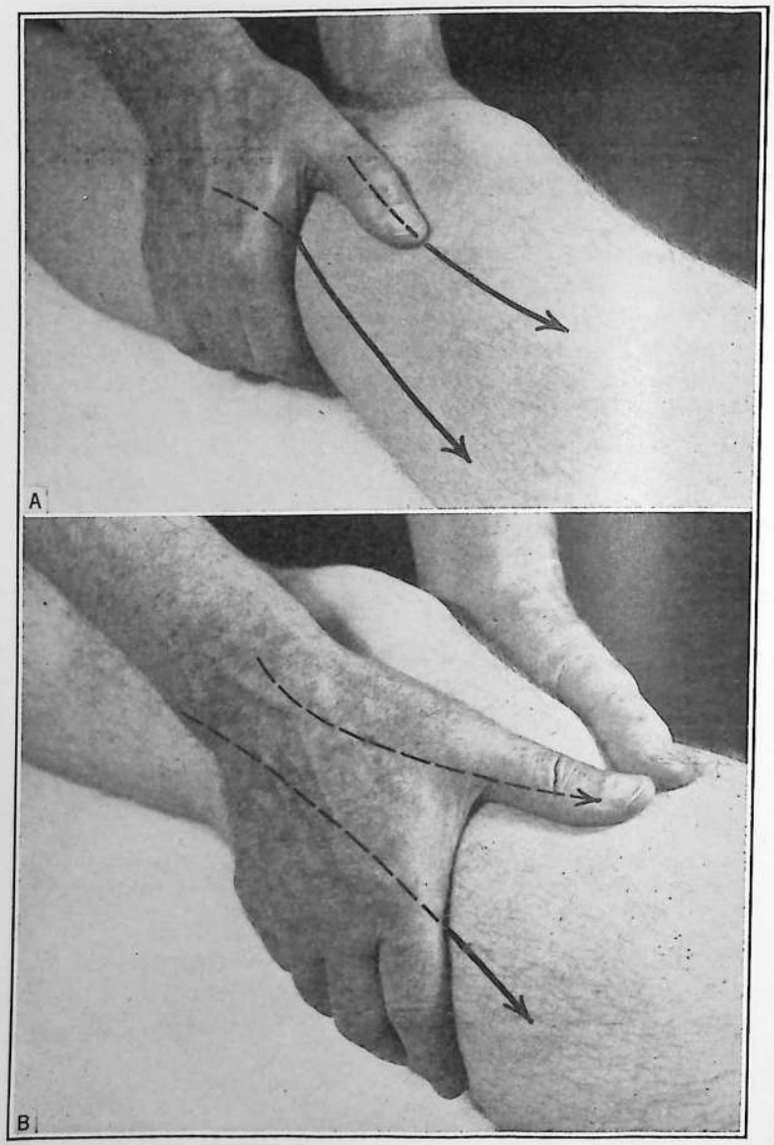

FIG. 73.—Effleurage of the knee: the operator's hand goes around, instead of over the patella. This also illustrates the even pressure of the hand which must conform to the contour of the part. *B*, End of effleurage of the knee, begun in *A*.

the important contraindications to massage such as swellings which may possibly be malignant, dermatoses, acute arthritis and systemic diseases with marked febrile reactions.[8]

VARIETIES OF MASSAGE[5]

1. Superficial or deep stroking movements (effleurage).
2. Compression movements, including kneading (pétrissage) and friction.
3. Percussion movements, such as hacking, clapping, slapping, tapping and beating (tapotement).
4. Vibratory and shaking movements.

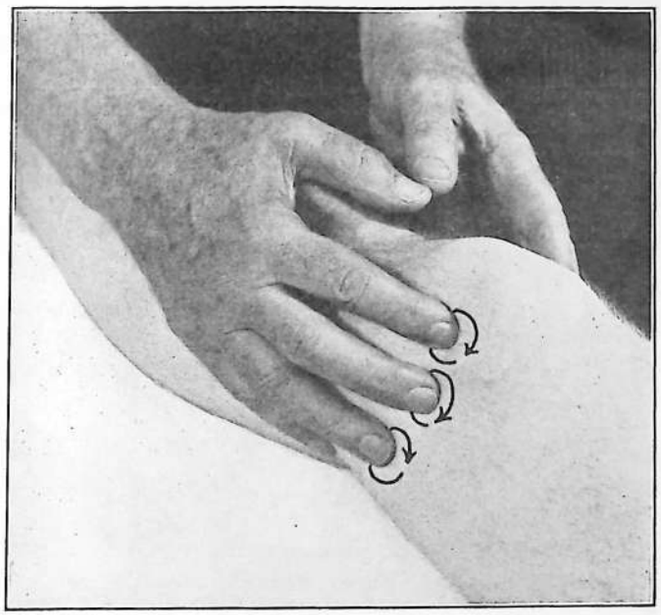

FIG. 74.—Frictions around the knee joint. Arrows illustrate small circular movements with the finger-tips. After frictions are performed in this position, the fingers are moved slightly and the motions repeated, etc., around the patella.

THERAPEUTIC VALUE OF MASSAGE

1. Increases local blood supply to joints.
2. Hastens drainage from region of involved joints thus decreasing the periarticular swelling.
3. Produces muscular relaxation.
4. Increases lymphatic and venous return.
5. Massage and relaxed movements prevent fibrosis and adhesions in muscles and decrease the tendency to muscular atrophy.

Should Massage be Given to Acutely Inflamed Joints? *No!*

Stage at Which Massage Should be Begun in Rheumatoid Arthritis.—Massage should be begun as soon as the acute inflammatory process in the joints has subsided. This should be given at the onset only to the tissues surrounding the joints and then to the periarticular tissues, rather than to the joints themselves. Massage may be given within several weeks after the onset of an acute arthritis requiring rest in bed; if the knees are involved,

the muscles of the thighs and lower legs are massaged to aid the return of venous blood and to prevent muscular atrophy from disuse. When the acute inflammatory process has subsided, as judged by subsidence of redness, swelling and tenderness, the periarticular tissues may be massaged lightly to promote absorption of the periarticular edema. In chronic stages of arthritis, the surrounding muscles and the areas over the joints themselves may be included in the massage, if this latter is given very lightly.

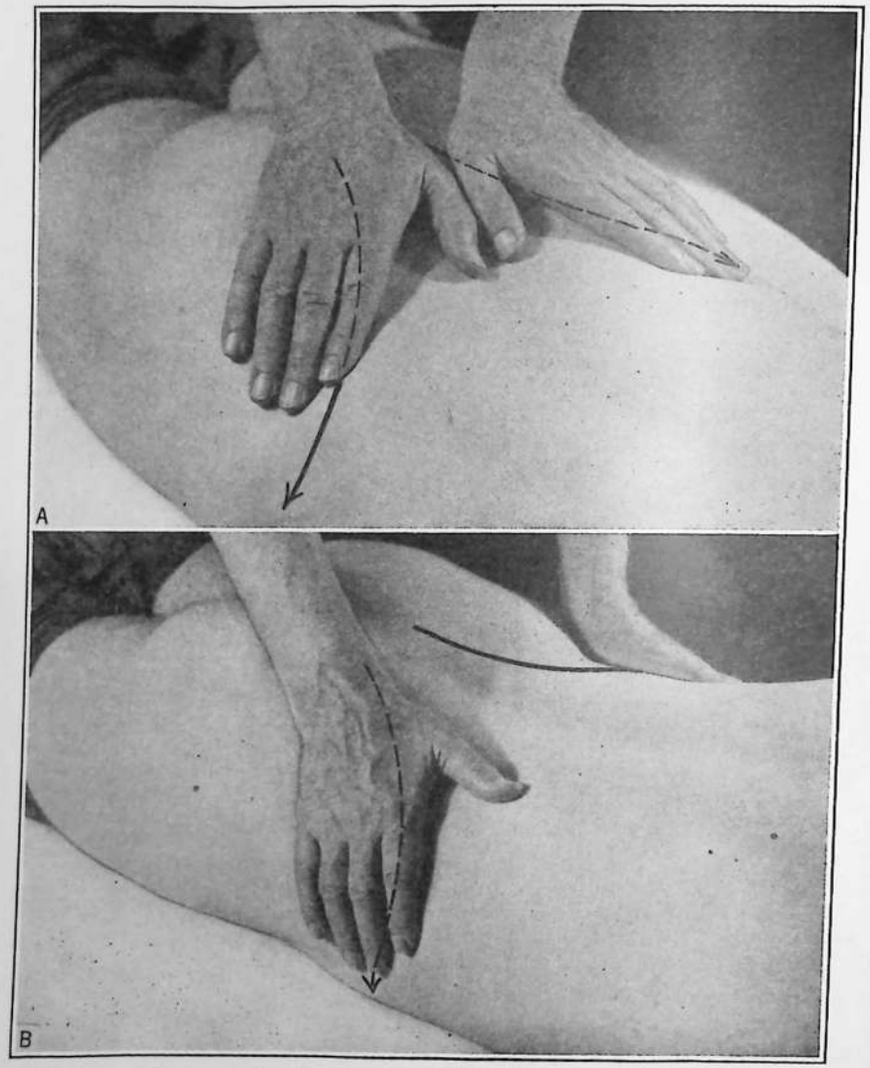

FIG. 75.—*A*, Effleurage of the low back; beginning of motion. Note that the operator's hands are molded to the part being massaged. *B*, End of motion of effleurage of low back begun in *A*.

Should the Parts be Heated Before Massage is Given?—In general it may be stated that the application of local heat for a period of from thirty to forty-five minutes (directly preceding the massage) is beneficial in that it induces a mild hyperemia and muscular relaxation.[6]

FIG. 76.—Frictions at the lumbosacral joint for arthritis or fibrositis. This is one of the important movements in massage of a joint. Friction should always be followed by effleurage.

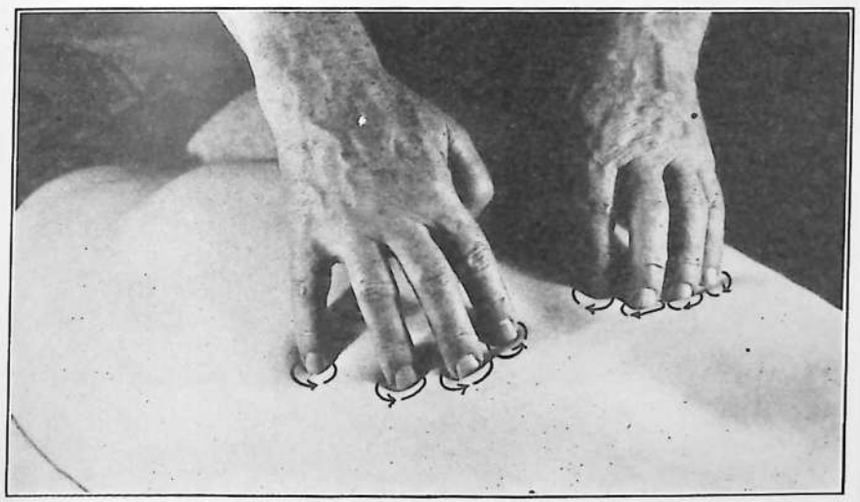

FIG. 77.—Bilateral frictions to the low back.

Massage Should Not Produce Pain.—Massage should never cause pain while being given. Slight discomfort during or for a short period following massage is not avoidable in some cases; however, if discomfort persists (for more than two hours) following massage, it is probable that the massage was given incorrectly or that it was contraindicated in that particular case.

Should Powder or Cream be Applied to the Skin Preceding Massage?—If the skin is dry, a small amount of cold cream should be used during the massage. If the skin is moist, some individuals prefer drying this and using fine powder.

The Varieties of Massage.—*Stroking movements* are long smooth motions toward the heart over the involved part. After the upward movement, the hand is raised from the part and reapplied to the lower portion. Stroking movements are carried out only in the direction of the venous flow and must be gentle, slow and rhythmic.[7] Light stroking produces a reflex effect. Deep stroking empties the lymphatics and the veins. Because of the low venous pressure, heavy pressure is not required. *One of the main purposes of massage is to return stagnant venous blood to the general circulation.* Upward stroking helps to empty the veins whose blood may have been stagnant, because the pumping action of the surrounding muscles is lost if the patient is not active. During stroking movements even pressure is applied in upward movements only and the hand is molded in such a manner that it conforms to the configuration of the part massaged. When large muscle groups are massaged the entire hand is used, while only the ball of the thumb or the thumb and one or two adjoining fingers are used on small parts.

Pointers on the Technic of Massage.[2,12,17]

The room temperature should be approximately 70° F.
Only the portion of the body being treated should be exposed; the remainder of the body must be covered with a blanket or sheet.
Both patient and operator should be in comfortable positions; the patient should always be recumbent to take advantage of gravity (in influencing the lymphatic and venous circulation).
The joint must be relaxed and comfortable; remove any constricting bands or clothing.[9]
One of the best ways to acquire the proper technic for massage is to have several treatments from a good masseur.
The hands of the masseur must be dry, warm, soft and must possess some strength.
If hairy parts must be massaged frequently, the skin should be shaved to avoid irritation.
Massage in arthritis should never produce pain which persists for several hours.
Movements should be slow and rhythmical.[4]
It is almost impossible to massage one's-self well.
Heat should be applied before the massage.
Do not massage painful or swollen bones or joints.
When stroking motions are used, these are made in only one direction, *i. e.*, toward the heart.

Kneading consists of circular movements with both hands. One hand is placed on one side and the other on the opposite side of the extremity, pressure being exerted toward each other. Kneading is always alternated with stroking (effleurage) to assist return of venous circulation.

Friction consists of circular movements of the fingers on the tissues using deep pressure so that the surface structures are moved over the deeper tissues.

Percussion movements include: (*a*) Hacking (striking with the ulnar side of the hand); (*b*) clapping (slapping movements using alternate palms held partly open); (*c*) slapping (with the palms); (*d*) tapping (with the finger tips as in playing the piano); (*e*) beating (percussion with the partially closed fist). Percussion or vibratory movements are bath house procedures and have no place in the treatment of arthritis.

Vibratory movements consist of trembling movements of the fingers, or hands in contact with the body; similar motions performed more coarsely are known as shaking motions.

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CHAPTER XVII

EXERCISE FOR THE ARTHRITIC PATIENT

Unfortunately many patients with rheumatoid arthritis labor under the delusion that the joints must be exercised as much as possible to prevent stiffness; by this procedure, irreparable harm may be done to the involved parts. On the other hand, there are those physicians who advise the patient with arthritis to go to bed and to remain there until his joint swellings and pain have disappeared. Such a practice may cause muscular atrophy and weakness and in part defeats the purpose of the treatment.

Proper exercise will decrease the tendency toward muscular atrophy and aid in preventing the formation of dense adhesions in the joint. Such adhesions can usually be prevented by one or two daily painless movements of the joint. *One should never perform movements which cause marked pain.* It is far better to carry one movement through a full range of motion than to perform a series of shorter movements. The exercise should be considered to be excessive if the patient experiences an increase in pain, swelling or stiffness within twenty-four hours following this.

Legal action frequently follows manipulation of deformed joints. Those individuals employing mechanical apparatus for manipulations on patients with arthritis or deformed joints must realize that this is a very dangerous procedure because of the possibility of producing fractures of the weakened bones and the ankylosis which sometimes results.[3]

Several simple general rules may be noted concerning the use of exercise in the arthritic patient. These are described in detail in the Handbook of Physical Therapy published by the American Medical Association.

During the acute stage of an arthritic process, only slight daily painless motion of these parts is performed by the physician or assistant. One or two daily painless movements of the joint usually prevent the formation of dense adhesions; in some instances, such motion may cause slight discomfort. No movements should be performed which cause marked pain; any pain (in the joints) which persists for more than several hours following motion indicates that the motion has been excessive. When the acute phase of the disease has passed, one may note slight attempts at motion of the affected parts by the patient himself, especially if enough encouragement is given by the physician. It is at this time, in the subacute stage, that active exercises are instituted and are increased gradually, as tolerated.

When the arthritic patient is confined to bed, he should be placed on a firm, non-sagging mattress and should practise *postural exercises* three times a day after meals. The pillow is removed from under his head and the patient lies in a horizontal position of hyperextension with the hands placed behind the neck or head, if possible, to help raise the ribs and flatten the dorsal spine. A small pillow may be placed under the chest

in the beginning. Following thirty minutes in this position, the patient lies on his abdomen in the face prone position for fifteen to thirty minutes, with a pillow placed lengthwise under the chest and abdomen. This helps to counteract the gravitational effects which may increase the strain on the body and the sag of the various organs. These two positions have been emphasized as the ones which give the nearest approach to complete physiologic relaxation, resulting in elevation of the diaphragm, more active circulation through the vascular channels in the abdomen, increased range of respiratory movements and a greater vital capacity.

Exercise for the Arthritic Patient

At least one or two gentle painless movements of the affected joints must be performed daily even in the acute stage of the disease so that dense adhesions are not allowed to form. In acute cases, relaxed motion is advisable.

Definite exercises should not be begun until a joint is without pain while at rest.

As the patient improves, the amount, and usually the type of exercise is modified and increased. Attention should be given to underwater exercise and sling suspension exercises. The Hubbard tank may be used for all exercises in the early arthritic cases; a simple tank may be made for home use.

Active movements (by the patient himself) are always to be preferred to passive movements (by an assistant).

Never exercise a patient to the point of pain or fatigue.

Any motion which is attended or followed within twenty-four hours by an increase of pain, swelling or stiffness in the joints has been excessive; this must be reduced or modified. Proper exercises should cause no pain during their performance.

When the acute stage of the disease passes, and the patient moves the affected joints slightly, active therapeutic exercise is begun.

Never force a joint; this may result in a flare-up of a smoldering arthritic process.

Muscle training exercises are of extreme importance and *must be performed whether the patient is confined to bed or not.* A **simple overhead frame** may be used for bed patients with elastic loops for the legs and a bar for the hands; these will enable exercises which would not be possible otherwise.[5,6]

Muscle setting consists of contracting a muscle or group of muscles without moving the joints. Muscle setting exercises should be begun as soon as any acute inflammation subsides. This should be increased as tolerated, exercising chiefly those muscles which combat deformity: extensors, abductors and external rotators. Muscle setting may be begun with a few contractions each hour and may be increased to five minutes or more per hour as long as they do not produce pain or fatigue.

Muscle setting exercises should be followed in time by *assisted active exercises, exercises without weight bearing* and last, *weight-bearing exercises.* Proper exercises must never result in an increase in the activity of the arthritic process.

Many physicians do not have available lists of exercises which may be consulted for use in such patients. Each general practitioner should have on hand a copy of the Handbook of Physical Therapy, published by the American Medical Association, and authorized for publication by the Council on Physical Therapy. The following is presented for practitioners who desire a list of some of the exercises available for these patients. It must be understood in each instance that these exercises are to be instituted only after the stage of acute inflammation has passed, and that only one or two exercises are to be used at the onset. These are performed not more than three or four times a day. As the patient improves, they may be increased in number and duration but any **increase in pain, swelling or stiffness occurring within twenty-four hours following the exercises indicates that these have been excessive and that they must be decreased or modified.** Any pain incident to the exercises which disappears within an hour, is usually considered not to be detrimental. Some individuals feel that pain persisting for two or three hours only is likewise not harmful. It is preferable that exercises cause no pain whatsoever.

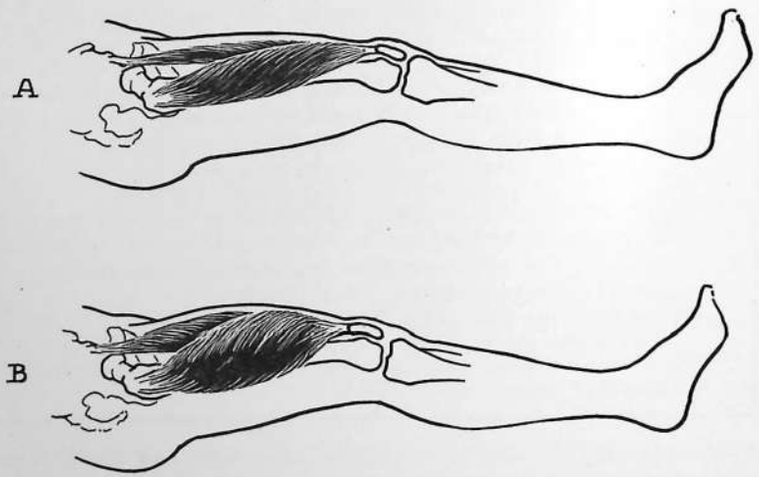

FIG. 78.—*Muscle setting. A* shows the quadriceps muscle at rest. *B* shows this muscle contracted (or set). This is performed with the leg extended. The position (origin and insertion) of the muscle is explained to the patient. He usually learns to "set" the quadriceps by attempting to "squeeze" the muscles of the mid-thigh. In some patients this is learned best by pushing the knee backward against the bed.

The following additional notes may prove useful to the physician in a better understanding of such exercises:

Passive motion is movement performed by an operator without the assistance or resistance of the patient.

Active motion is that done by the patient.

Even in the more chronic stages of an arthritis, **not more than an hour of continuous exercises should be performed.**

The occurrence of muscular spasm during an exercise is a signal for cessation of the movement.

Exercises for the Bed Patient

1. Exercises should be individualized for the patient, varying with his general health, joints involved, type of arthritis, etc. However, in the average patient with widespread involvement of multiple joints by rheumatoid arthritis, the following program may form a basis for such exercises.
2. Exercises must never be carried to the point of fatigue.
3. Exercises must never cause pain which persists.
4. The patient should attempt to "*set*" (contract) the *quadriceps muscles* while in bed, beginning with four or five times each hour while awake, and increasing this as tolerated to fifty or one hundred times an hour if this does not cause fatigue or pain. This simple exercise will be extremely valuable when the patient is ready to walk again.
5. *Squeeze the gluteal muscles together* four or five times each hour while awake, increasing this to eight or ten times per hour, if tolerated.
6. Take *ten deep breaths each hour*, elevating the chest with each inspiration and pulling in the lower abdomen with each expiration. Note that Nos. 4, 5 and 6 should be done each hour unless these tire the patient.
7. The following exercises should be performed *two or three times a day:* morning, afternoon and evening, if tolerated:
   (*a*) *Hands*
   (1) Touch the tip of each finger to the tip of the thumb. Repeat eight times. As improvement occurs, touch the tips of the second, third, fourth and fifth fingers to the thenar eminence instead of the tip of the thumb.
   (2) With the fingers together, attempt to stretch the fingers long and to raise the tips of the fingers dorsally. Relax. Repeat eight times.
   (3) Separate the fingers widely; relax. Repeat eight times.
   (4) Make a tight fist (or squeeze a small rubber or woolen ball); relax. Repeat eight times.
   (*b*) *Wrists*
   (1) Extend (dorsiflex) the hand at the wrist; relax. Repeat eight times.
   (*c*) *Forearm*
   (1) Flex the forearm and then attempt to extend this against the patient's own resistance. Repeat eight times. This must be taught to the patient by having the physician apply gentle resistance against the forearm when the patient attempts to straighten this. The patient will soon learn to produce his own resistance voluntarily.
   (2) Touch the shoulder with the fingers of the same side; repeat eight times. Open arm to right angle at the elbow before each attempt at flexion.
   (*d*) *Shoulder*
   (1) Raise the arm horizontally (laterally) over the head. Lower. Repeat eight times.
   (2) Raise the arm vertically over the head and lower by moving the arm laterally and downward. Repeat eight times.
   (3) Place both hands under the back of the head and attempt to flatten out the elbows on to the bed. Hold this position a few seconds or more, as tolerated. Repeat eight times.

Exercises for the Bed Patient—(*Continued*)

(*e*) *Foot*
- (1) Dorsiflex the foot. Relax. Repeat eight times.
- (2) Move the foot in large circles by having the great toe move in large circles. Repeat eight times.

(*f*) *Knee*
- (1) Flex knee and extend fully. *Repeat this exercise only once or twice each day if attempts are being made to correct flexion deformity of the knee.*
- (2) As the patient improves, with the patient on his back, gently flex both knees with the feet resting on the bed. Then flex the right leg to its maximum on the abdomen, straighten out the lower leg so that the whole leg is straight and makes an angle of 45° with the horizontal, then return to the position of flexion of the knee on the abdomen, and finally relax the leg. Repeat with left leg. This may be repeated as tolerated. This exercise cannot be done until the patient has gained considerable strength.

(*g*) *Hip*
- (1) With the leg extended, abduct the leg at the hip; return to resting position. Repeat eight times.

(*h*) *Low back*
- (1) Flex the knees, contract (squeeze) the low back and gluteal muscles and attempt to gently elevate the buttocks from the bed (while lying on the back). Repeat eight times.

(*i*) *Neck*
- (1) Turn the head to the right, then to the mid-line, then to the left, then return to the mid-line. Repeat eight times.
- (2) Hyperextend the head, then completely flex this (with the chin on the chest). Relax. Repeat eight times.

8. *Caution.* Although the instructions as given above will apply to the average case of rheumatoid arthritis, no 2 patients are exactly alike. *The tolerance of patients varies greatly.* Although many individuals will be able to tolerate the exercises planned, repeating these eight times, three times a day, some may be able to tolerate these only once a day, and perhaps only one or two movements at each joint. *The amount and type of exercise must be individualized.* **Never tire the patient; never cause persisting pain!**
9. The exercises will accomplish several features.
   (*a*) They will improve the muscular tone and prevent muscular atrophy.
   (*b*) They will aid the movement of venous blood and stagnant lymph.
   (*c*) They will usually greatly improve the patient's morale and will keep his mind occupied a large part of the day. He will feel that he is really accomplishing something while in bed and not merely lying as a lump of putty.
10. When the patient is able to be up, exercises should be continued and modified as desired. If a therapeutic pool is available, all exercises may be performed much more readily.

Fibrillation of muscles indicates that the active exercise is too severe and that it should be reduced, or assistance should be given by the operator.

One movement through a full range of motion is preferable to a series of shorter movements.

All movements must be slow, deliberate and not jerky or tiresome.

All periods of strenuous exercise or exercises following other strenuous measures as hot baths, must be followed by an equal period of rest.

The arthritic patient should not spend many hours sitting in a chair as this permits the affected joints to remain for long periods in positions of the common flexion adduction deformities.

In the past several centuries, the three principal *systems of exercises* have been the Turnverein system (introduced in Germany by Ludwig Jahn); the French system (introduced by Delsarte) and the Swedish (by Pehr Ling and Adolph Spiess). A system of gymnastics was developed by American physicians and educators about one hundred years ago. At present there is a tendency for the development of *corrective exercises* for therapeutic purposes.

Elkins has pointed out that although motion is probably the most important function of life, and the skeletomuscular system thus constitutes a very important part of the body, very little time is spent in teaching the function or malfunction of this system in comparison with that spent on systems of much lesser consequence.[2]

Muscles are of use because they *are able to produce motion by tension between their attachments.* Muscular tension is made useful by its action on rigid skeletal levers. Muscular action regulates motion.

Muscular action aids in the stabilization of joints. The attachment of muscles is such that they produce a pull almost parallel to the long axis of the bones; the strong component of force which is produced in this direction tightens the joint capsule and pulls the articular surfaces together. If this stabilizing action were not present, the joints would show many more strains and stresses and subluxations would occur.

Elasticity and extensibility are two important physical properties of muscles. When a muscle is stretched, it is capable of being extended. These properties permit smoothness of motion and aid in relieving a sudden impact which might follow the inertia of load, preventing rupture of muscle fibers.

Muscular exercises should be performed slowly because rapid motion fatigues muscle quickly and sudden, rapid, jerky movements may result in further damage to arthritic joints, leading to more muscular spasm with resultant loss of muscular power, elasticity and contractility.[2]

Exercises may be used to increase the tone of muscles. Complete muscular relaxation occurs during life only under deep anesthesia. Simple muscular tone is the state of muscular tension between contraction and complete relaxation. Lack of muscular tone may add to the instability of joints. *Muscle setting exercises,* in which the muscle is tightened without produeing motion, *will increase the muscular tone.*

Contractures in rheumatoid arthritis may lead to a disturbance of proper posture, especially if there is flexion of the knee and hip. Minor flexion deformities of the knee throw the center of gravity backward requiring the spinal column and trunk to compensate by assuming a forward position,

EXERCISES FOR THE KNEE JOINT

This is the largest and most complex joint in the body. Its strength is due largely to its ligaments and only secondarily to its muscles. Flexion is performed chiefly by the hamstrings, in addition to the sartorius, gracilis and gastrocnemius. Extension is mainly a result of the action of the quadriceps extensor, and in addition, the tensor fascia lata.

Passive Movements (to be used first).—The patient lies on his side with his knee resting on a large powdered piece of cardboard. With the operator holding the patient's ankle and thigh (while standing behind the patient) slow, steady flexion of the knee joint is performed. The joint is maintained in this position of flexion for three to five seconds and is then extended in a similar manner. If the patient is in the sitting position, place the thigh of the patient over the operator's thigh to fix this, and then move the leg as desired.

Active Movements.—The same as the above except that the patient performs this himself while in bed.

Contraction of the extensor muscles above the knee (quadriceps) to move the patella without moving the knee itself. This is performed more easily if an assistant exerts constant slight pressure against the plantar surface of the forefoot. The patient learns this exercise more quickly if he attempts to squeeze the mid-thigh, or if he pushes the knee backward onto the bed.

Lying on the back with the leg extended, attempt to flex the knee as much as possible.

Lying on the back with the knee flexed, attempt to straighten the leg as far as possible.

Lying on the abdomen, with the knee flexed, attempt to straighten the leg.

Lying on the abdomen, with the leg extended, flex the knee.

Sitting on the edge of the bed with the leg hanging, attempt to straighten the leg.

Sit on the edge of the bed, swing the leg back and forth in pendulum fashion.

Sitting, with the leg extended, raise the leg from the bed.

Bicycling of the legs while lying on the back; later stationary bicycling.

Standing on the toes, bend the knees and return to standing position.

Performance of the above against resistance (usually offered by the operator or a member of the family).

EXERCISES FOR THE FOOT AND ANKLE

Passive dorsal and plantar flexion of the foot while lying on the affected side. These movements are carried out by the physician who grasps and supports the ankle with one hand, so that the heel is above the bed, and moves the foot with the other. The foot is held for several seconds in a position of dorsiflexion and then in plantar flexion.

Dorsal and plantar flexion of the foot by the patient while lying on the affected side.

Circular motion of the foot while in a sitting position.

Rolling a tennis (or rubber) ball between the soles of the feet.

Sitting, with the feet placed on a board so that the toes project over the edge of the board; bend the toes downward over the edge of the board.

Sitting, pick up marbles from the floor with the toes.

Same as the above, but bringing marbles to the opposite band from the foot.

Sitting, with the feet parallel and 6 inches apart, raise the inner margins of feet, keeping the toes flexed.

Standing with the feet placed on a board so that the toes project over the edge of board; bend the toes downward over the edge of the board.

Standing, rise up on the toes and bear weight on the outer portion of the feet, lifting the inner margins of the feet.

Walk on the outer borders of the feet with the toes flexed.

Cross the ankles when sitting and allow the feet to rest on the outer borders.

Sitting, heels apart and big toes together, raise the front part of the foot from the floor as far as possible.

EXERCISES FOR THE LOW BACK

(Repeat each exercise five to ten times varying with the physical conditions.)

1. Squeeze the gluteal muscles together and maintain this for seven seconds.
2. Contract the gluteal and abdominal muscles together while lying on the back.
3. Contract the abdominal muscles and raise the head with the hands behind the head; maintain this for seven seconds.
4. Lie on the back; do straight leg raising to 90° with one leg straight and the other flexed at the knee. Bring the leg up fast and allow it to drop slowly. Repeat five or ten times and then perform the exercise with the opposite leg.
5. Lie on the back; forcibly dorsiflex and invert the foot with straight leg raising and stretch until a pull is felt in the hamstring muscles.
6. To stretch the ilio-tibial band: stand 2 feet from a table. Place one hand on the table and lean toward the table, keeping the feet flat on the ground.
7. Lie on the face; raise one leg slightly and lower. Follow with opposite leg.
8. Lie on the back; knees flexed. Raise each knee on the abdomen alternately.
9. Lie on the back. With the hands in front, sit up slowly without using the hands.

Despite the mass of literature concerning the effects of **poor posture,** very

few physicians employ a course of training in postural exercises which proves clinically useful in correcting the poor posture, especially in school age children.

Scoliosis seldom causes radicular pain. *Kyphosis* and *lordosis* are seen most often in asthenic individuals with hypotonic muscles and an unstable spinal column. Postural exercises are included in the chapter on Orthopedic care. A few simple *principles for improving posture* should be given to the patient and he must practise these several times a day.[5] These pointers include:

1. Raising the thorax with deep breathing exercises through the chest with the abdomen pulled in.
2. Attempting to stretch the top of the head and shoulders toward the ceiling. The patient should sit, stand and walk "as tall as possible" at all times.
3. Walking with the feet pointed straight ahead.

In functional scoliosis, in which roentgenograms show a normal structure of the spine, corrective exercises are always indicated. One should not expect severe structural scoliosis to respond to exercises. If the curve remains stationary in cases of functional scoliosis with therapeutic exercise, one does not urge surgical treatment; if the curve increases, it should be corrected on a hyperextension frame or turnbuckle cast, following which spinal fusion is advisable.[1]

EXERCISES FOR THE HIP JOINT

Passive motion of the knee and hip with the patient lying on the back, the leg being held in a position of flexion for from three to five seconds.

Same as above but with active motion.

Lying on the back, raise the leg slowly with the knee straight.

Lying on the back, legs straight, separate legs widely and return to normal position.

Lying on the face, lift the leg upward as far as possible.

Lying on the back, bend forward to touch the toes.

Lying on the back with the knee straight, force the leg down to the bed after the operator has raised it.

Lying on the affected side, raise the knee toward the chest as far as possible and return.

Touch the toes from a sitting position.

Standing between 2 chairs, swing the affected leg to and fro.

Walk up and down stairs.

Bicycle in bed and on a stationary bicycle.

EXERCISES FOR THE SHOULDER JOINT

The abductors (deltoid and supraspinatus) are comparatively weak. The adductor (pectoralis major) is strong. The internal rotators (latissimus dorsi and subscapularis) are much stronger than the external rotators (infraspinatus and teres minor).

Exercises.—Passive motion of the shoulder joint in all directions.

Lying flat in bed, gently abduct the arm.

Lying flat in bed, externally rotate the arm.

Lying on the back with the elbow flexed to 90°, raise the arm upward and return.

Shrug the shoulders.

Lying on the abdomen, clasp the hands behind the head, raise the head and elbows from the bed.

Standing, gradually reach as high on the wall as possible with the fingers.

Sitting or standing, swing the arms to and fro.

Standing, arms outstretched shoulder high at the side of the body, rotate the hands in circles getting progressively larger.

Raise the arms upward and sideward from the body until the palms meet above the head.

Same as the above, bringing the backs of the hands together.

Arm hanging by the side, hold a bucket containing increasing amounts of water; rotate the hand to and fro.

Play pool.

Standing, body bent forward, swing the arms antero-posteriorly and then laterally.

Attach a cord over pulleys at the head of the bed, about 2 feet above the level of the mattress. Hold one end in each hand. Pulling the cord with one arm draws the opposite arm upward, and *vice versa*.

EXERCISES FOR THE ELBOW JOINT

Flexion is performed through the biceps, brachialis anticus, muscles arising from the internal condyle of the humerus and the brachioradialis. Extension is accomplished by the triceps and anconeus, aided by the extensors of the wrist and fingers. The arm is supinated by the biceps and supinator brevis. Pronation is performed by the pronator radii teres and the pronator quadratus.

Exercises.—Passive motion (flexion and extension).

Lying on the abdomen with the affected arm held in extension (by the operator) at the side of the patient's body and over the side of the bed, the patient attempts to bend the elbow farther and farther (assisted by gravity).

Lying on the back, attempt to straighten the elbow (which has been flexed by the operator).

Touch the shoulder with the hand. Later do the same with increasing weights in the hand.

Lift increasingly heavy weights.

Saw wood; pitch quoits; use a screw driver; weave baskets.

EXERCISES FOR THE HAND AND WRIST

Passive exercise in the following combinations: Flexion of the fingers with extension of the wrist.

Flexion of the wrist with extension of the fingers.

Pronation of the forearm with extension of the wrist.

Active Exercises.—With the hand resting on the little finger (on a table) bend the wrist forward with the fingers straight.

Same as before bending the wrist backward.

Palm on the table, move the hand toward the little fingers.

Palm on the table, move the hand toward the thumb.

Make a tight fist and then fully extend the fingers.

Turn door handles.

Same as the above, with an assistant holding the other side of the door handle.

With the fingers of each hand bent, pull the fingers of the other hand.

Wring out wet cloths of various sizes.

Spread the fingers apart widely; bring the fingers together.

Squeeze a rubber ball and let go.

With the palm on the table, raise and lower the fingers, one at a time.

Touch the tip of the thumb to the tip of other fingers, one at a time.

With one hand, crumple a page of newspaper into small ball.

With a rubber ball resting on the dorsum of hand, flip this into the air and catch it in the palm.

Wind twine into a ball.

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CHAPTER XVIII

ORTHOPEDIC MANAGEMENT OF ARTHRITIS.

Unfortunately, *many physicians have used orthopedic measures* (including splints) *only as a last resort in the arthritic patient* rather than as an aid at the onset of treatment. Early orthopedic management is an important phase of therapy in arthritis. **The main aim of the orthopedic surgeon, in arthritis, is to preserve the function of joints or to restore such function if this has been impaired.** *He must constantly realize, however, that he is treating a patient and not merely several involved joints;* in rheumatoid arthritis, particularly, it is true that the whole is greater than any of its parts.[66]

The duties of the orthopedist include:

1. Recognition of the early signals of abnormalities in body framework and evaluation of these.[64]

2. Use of proper *splints* early in the course of the disease *to rest the affected joints*, decrease inflammation, *prevent deformities* and *maintain joint function.*

3. *Aspiration of large joint effusions* in order to prevent stretching of the joint capsule and ligaments, and to relieve pain; strict asepsis must be employed and a pressure bandage should be applied following the aspiration. The fluid obtained may be examined, if desired, for diagnostic purposes.

4. Maintenance of affected joints in positions best suited for ankylosis should this be inevitable.

5. Attempts to *develop* and *maintain good body posture,* and thus overcome muscular strain and traumatic joint irritation. Minor aids in weight bearing which may be prescribed by the orthopedist include elastic supports, corsets, back braces, corrective shoes, etc.

6. *Correction of deformity* if such has developed (by means of splints, plaster casts or surgical procedures: manipulation, arthrotomy, osteotomy, synovectomy, capsuloplasty, arthroplasty or arthrodesis). Correction of deformities has been termed correction of mistakes of the doctor and patient.[42]

7. Suggestions as to physical therapy; however, the orthopedist must not wear out the patient with excessive physical therapy.

The sheet anchor in the orthopedic management of arthritic joints is rest. The arthritic patient with painful joints is not put to bed and left there without proper care. If adequate management is not prescribed, extensive muscular atrophy will occur, together with atrophy in the bones and joint cartilages because the limbs are held rigid by muscular spasm, leading to well-recognized deformities. *The affected part must be supported comfortably in the position of optimal use should limitation of motion or ankylosis occur.* Several active or guided movements daily will prevent the formation

of dense adhesions within the joint and prevent contractures of the periarticular structures.

One of the most important principles in the treatment of arthritis is to rest the affected joint. There is less danger of ankylosis, adhesions and loss of motion if inflamed joints are rested by splinting them than if they are continually moved in an effort to prevent stiffness and loss of motion. Ankylosis does not develop following temporary complete immobilization of joints; it is usually the result of an inflammatory process leading to destruction of the articular cartilage.[86] The concept that joints must be moved vigorously to prevent stiffness has been shown not only to be erroneous but actually dangerous; modern rheumatologists would no more consider strenuously exercising acutely inflamed joints than would surgeons attempt to obtain motion at the site of a fracture immediately after the injury. The orthopedist must constantly watch for any sign of muscular contracture which might lead to deformity.

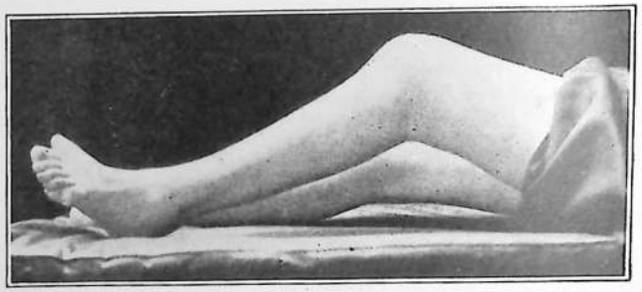

FIG. 79.—Early flexion deformity of the knee in arthritis. (Swaim and Kuhns, courtesy of Jour. Am. Med. Assn.)

One of the fears of the arthritic patient is pain resulting from motion of the inflamed joint. A sore joint produces fear as motion or slight jarring of an inflamed joint may cause excruciating pain;[89] this is the basic cause of many of the flexion deformities in this disease. General relaxation of the patient may be obtained by proper use of splints in order to break down this mental complex and to assure him that he can have no motion (or pain) of the splinted parts. If he could be *assured* that no pain could result from motion, his mental attitude would be greatly improved and the spasm of muscles surrounding the joints would be abolished. *By the use of molded plaster splints, painful motions can be abolished in acute arthritis, and flexion contractures can be avoided;* this applies especially to those joints commonly affected in rheumatoid arthritis (knees, wrists, elbows, fingers, spine and toes).

Some of the **common deformities** seen in patients whose **arthritis** has been neglected are flexion contractures of the knees, flexion of the wrists (thus almost destroying the use of the hand inasmuch as the fingers are placed in a poor position for grasping), flexion and limited motion of the elbows, extension and subluxation of the toes, flexion deformity of the

thoracic spine with bony ankylosis of the ribs and spine and contractures of the fingers.[95] The optimum position for splinting these joints is one of muscular relaxation and one in which, if ankylosis should occur, the most useful position will be attained. Such positions include a straight (but not hyperextended) knee with several degrees of flexion; slight extension of the wrist; a straight flat spine, etc. These optimum positions have been enumerated elsewhere.

Proper Splinting of Arthritic Joints Will Not Lead to Ankylosis.—Such splints should provide firm support for the affected joint but should not be so tight that painful pressure results. Protective splints should be made for inflamed joints as soon as pain occurs. The method of making and applying these is discussed in detail elsewhere (see Chapter XIII). Plaster casts or splints eliminate muscular spasm, contractures and flexion deformities.[94]

Unless properly splinted, deformities of joints may occur early in the course of a rheumatoid arthritis. Swaim believes that one-half of the inflammatory reaction within the joints is continued because of use of the joint during the acute stage of the disease, and that lessening of trauma to the involved joints would greatly reduce the amount of capsular thickening and extension of pannus on the joint surfaces.[91] *Supports must be used for long periods not only during the active stage of the disease, but also in the after-care of the patient.* If used early enough, protective splints will prevent ulnar deviation of the fingers and subluxation and flexion at the metacarpophalangeal joints.[88]

Almost all deformities in arthritis are those of flexion. Because of pain, the patient voluntarily attempts to splint acutely inflamed joints; this produces muscular contraction and spasm, followed later by contractures. The *joints are invariably flexed during sleep by these individuals to relieve pain;* this emphasizes the importance of proper splinting at night. Molded plaster splints should be used to immobilize the affected joints where possible; these should be removed once daily to permit one or two full painless motions of all joints included in the splinted area. Fisher has devised a "Universal" splint for the joints of the upper extremity and one for those of the lower extremity.[20] An elastic bandage is very useful in adding support to strained joints.

Medical men should learn and be willing to use plaster in patients with arthritis. Splinting can usually be accomplished by plaster splints, or by circular plaster casts which may be bivalved later. If involvement of the hip joint requires immobilization, splints upon the lower extremities may be connected by cross boards.[5] **Plaster shells can be made quickly at home by** cutting a piece of felt $\frac{1}{4}$ inch thick, and about 5 inches wide (when the forearm and hand are to be splinted). The felt should extend from the upper forearm to just beyond the finger tips. An indentation is cut out for the thumb along one side. Wet plaster (from rolls) is then turned back and forth over the felt for 6 to 8 thicknesses and the wet splint is applied to the forearm and hand with the felt side next to the skin. A gauze bandage is then wrapped around this to hold it in place. The hand, wrist and fingers

are placed in a proper position with the wrist in extension and the fingers in midflexion. The splint may be trimmed with adhesive tape after it has dried. Such a splint will aid greatly in immobilizing acutely inflamed, tender joints and prevent subluxation of the carpal joints and ulnar deviation of the fingers.

Such splints must be removed each day for several motions of each of the affected joints so that dense adhesions are not allowed to form, and may be removed for the use of hot fomentations, and later for massage and exercise.[75]

Plaster shells may be made in a similar manner for the lower extremities in order to prevent outward rotation of the tibia and foot and to support the joints in favorable positions. The shells may be changed every week or ten days in the legs if needed to correct early contractures.

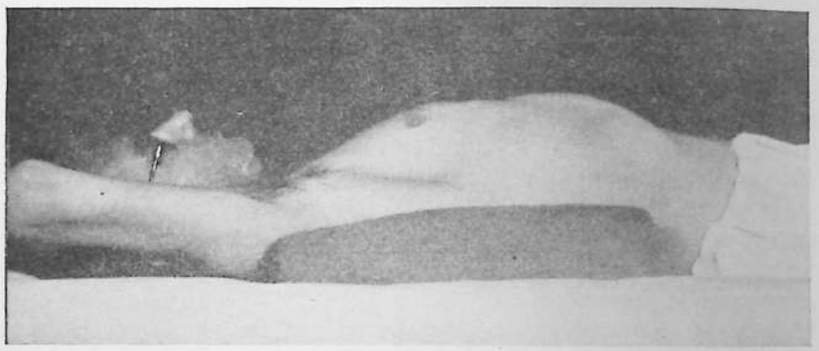

FIG. 80.—*Hyperextension* to expand the chest, raise the diaphragm, increase abdominal breathing and circulation and correct round shoulders. Use this position for one-half hour after meals; pillow under the shoulders and a pillow under the knees (to relax the back); pillows must not be kept under the knee for long periods or contracture deformities may result. (Swaim, courtesy of Am. Jour. Nursing.)

In individuals who have been confined to bed for long periods, it is often advisable to make *posterior light splints* or *walking slabs* which extend from the midthigh to just below the calf. These may be made in a manner similar to that previously described and may be bound to the leg with flannel bandages so that the knee joints will be supported in weight bearing and to avoid additional strain of motion.

CORRECT POSTURE

Andry,[3] Goldthwait[26] and Osgood[68] have stressed the importance of perfect mechanical alignment in these individuals. All unnecessary mechanical strains must be eliminated. The *importance of correct posture* cannot be overestimated. **Poor posture is one of the most important causes of strain of the back, foot, knee and hip.** Faulty posture may be caused by disease, carelessness, fatigue, style, ignorance, hypothyroidism, illness or psychogenic states.[31,90] "A man stands as he feels."

Although patients with poor posture may manifest no strain of the joints for years, a period of overwork or debility from illness may result

in symptoms of joint trouble, as may any sudden strain, such as suddenly stepping down from a curb, jerking open a window or carrying a heavy suitcase. **Signs of poor posture** include abdominal ptosis, excessive lumbar lordosis and anterior position of the head.

The *commonest postural defects* are round shoulders (kyphosis), prominent abdomen, pronated feet (pes planus), knock knee (genu valgum) and increased lumbar curve (lordosis). It has been claimed that poor posture resulting in ptosis, mechanical interference, drag and pressure may be indirectly responsible for gastrointestinal stasis (due to imperfect assimilation and excretion) and for poor digestion.[96]

LaPlace and Nicholson have described the *physiologic effects of the correction of faulty posture.*[54] They concluded that correct posture was of appreciable advantage to the respiratory and circulatory functions in a majority of individuals but that in some a postural defect may be a compensating mechanism which it is not advisable to disturb.

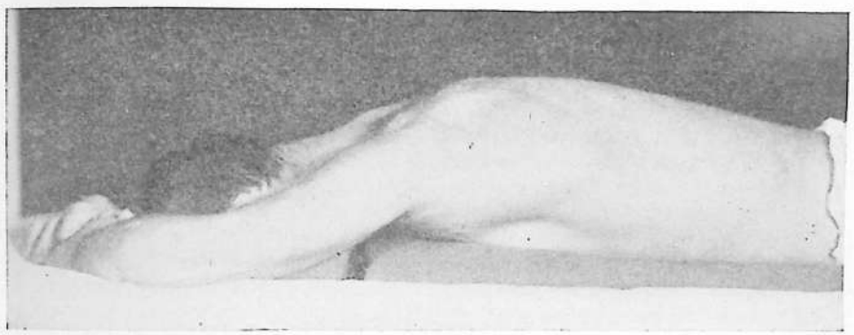

FIG. 81.—*Face prone position;* support under the abdomen to flatten the back. (Swaim, courtesy of Am. Jour. Nursing.)

The correction of poor posture in the arthritic individual is begun while he is in bed even during the acute stage of his arthritis. **Most patients with active rheumatoid arthritis and multiple joint involvement do best if maintained flat on the back with only one pillow under the head during the day and night, being allowed up only for meals.** After fatigue has lessened and the patient has improved, short periods of sitting upright are permitted with some form of support (brace or plaster shell) to keep the chest high and prevent drooping.[93] In patients who are continually propped up in bed with pillows, a prominent kyphosis may develop.

In addition to rest and the use of postural exercises, *correction of deformity by the orthopedic consultant* may be accomplished by Buck's extension; back braces: Thomas collars and corsets; molded plaster splints; turnbuckle casts; special corrective splints such as the Thomas caliper splint for the knees, airplane splints for the shoulders, cockup splints for the wrists and banjo splints for the hands; breaking up of adhesions by manipulation under anesthesia; and operative procedures.

Postural Exercises for the Standing Position.—When the patient has improved sufficiently so that *standing exercises* may be used, the following postural aids as recommended by Swaim will be found useful:

Correct Standing Position.—Feet straight ahead, body weight forward and borne on outer side of feet, abdomen in, back flat, chin in and attempt to stretch body upward. If one can hold a half dollar in the fold of the buttock, the posture is good.

Hands on the hips, abdomen in; breathe deeply, elevating chest; exhale by drawing in the abdomen.

Standing with the back to the wall and the heels 4 inches from wall; head and shoulders touching the wall; draw the abdomen in and up to flatten the back against the wall.

Standing; place one hand behind the neck and one on the opposite thigh; bend the body toward the side of the lower hand; keep the abdomen in. Repeat toward the other side.

Standing, raise the arms above the head, up on the toes, inhale; lower the arms and heels; exhale.

Positions and Exercises for Improvement of Posture of the Bed Patient

Exercises to improve circulation, tone and nutrition of muscles must be begun as soon as the acute inflammation has subsided. These are instituted while the patient is still in bed in an effort to improve body posture and physiology. Swaim has advised the following exercises for bed patients in an effort *to improve body posture:*

Position of Hyperextension of the Trunk (lying on the back, no pillow under the head; pillow under the shoulders and ribs; pillow under the knees; hands under the back of the head, maintain this position for thirty minutes after each meal): Lessens drag upon abdominal viscera, elevates ribs and diaphragm, flattens lumbar spine and tilts pelvis backward. The position of hyperextension will promote better circulation in the abdomen and extremities.

Prone Position.—Lying on the abdomen, legs straight arms placed on the bed above the head, pillow under the abdomen; assumed for fifteen minutes three times a day following the hyperextension position.

Breathing Exercises.—While in the hyperextended position, breathe deeply, raising the chest and exhale by drawing in the lower abdomen (*maintaining the elevated chest*). This tends to raise the chest from its drooped position. This should be performed three times a day during the period of hyperextension, beginning with five breaths each time. A complete set of postural aids has been presented by Goldthwait and his associates in their excellent monograph on "Body Mechanics."[27]

Lying on the back, knees flexed, hands on the head, back flat and chin in. Breathe deeply, elevating the chest, and maintain this elevated position of the chest; exhale by drawing in the lower abdomen.

Lying on the back, knees flexed, hands on the head: pull in the lower abdomen and relax; repeat five times.

Similar position: pull in the lower abdomen, squeeze the buttocks together and attempt to lift the buttocks slightly; relax. Repeat five times.

Similar position: attempt to stretch one side of the body, pulling the ribs apart; relax. Stretch the opposite side. Repeat five times.

Similar position but with the hands on the lower rib margins anteriorly; breathe deeply and pull the lower margin of the ribs up and out; maintain this pull while exhaling (by drawing in upper abdomen). Repeat five times.

Hall feels that the key exercise for improvement of body mechanics is what he terms the *pelvic roll*. This is done with the patient on his back in bed or on the floor, with the knees flexed, chest raised and the hands under the neck. The patient contracts his abdominal muscles until the lumbar spine touches the bed and the tip of the spine rises from the bed. When the patient has mastered this, it is made more difficult by only partly flexing the knees and performing the same exercise; the exercise is finally performed with the knees completely extended. When the patient is able to assume the upright position, he should attempt to stand for portions of the time with the gluteal and abdominal muscles contracted.

Manipulation of Deformed Joints

Should be avoided if possible.

Should not be done during the active stage of an arthritis because of the possibility of a resulting flare-up or ankylosis.

Should be performed only by one well trained and skilled in such procedures.[110]

Occasionally is indicated in selected cases of deformity due mainly to adhesions of the knee, wrist, shoulder, hip or foot.

Is not often successful in the fingers.

In degenerative joint disease rarely produces permanent improvement in range of motion or relief of pain; may cause fracture of an osteophyte and loose bodies in a joint.[4]

Has been advised under anesthesia for extensive fibrositic adhesions of the shoulder and lower spine. Manipulation under anesthesia is usually necessary to secure motion in the "frozen shoulder" seen with chronic (adhesive) subdeltoid bursitis. Platt has warned that if extensive capsular adhesions are present, short gas anesthesia should not be used as the patient may awaken with severe pain.[76] In the lower spine, advanced degenerative joint disease is a definite contraindication to manipulation.

Great care should be taken during the manipulation because of the possibility of producing fractures of the atrophied bones.

Holbrook and Hill have pointed out that although most individuals advise against manipulative procedures to joints during the active phase of the disease, this phase may persist for months or years. They have felt that the hazard of waiting more than a few weeks or months is much greater than that of attempting to straighten a joint in any stage of activity. Manipulation under anesthesia may be helpful in correcting minor deformities of position such as stiff, adducted shoulders; slightly contracted knees; rigid feet; and permanently flexed wrists.

ORTHOPEDIC SURGERY IN ARTHRITIS

The *surgery of chronic arthritis has been called patchwork surgery.*[67] Operative procedures have been most successful in the elbows and knees and much less so in the shoulders, fingers and hips. *If the arthritic process has been quiescent for six months and if the patient is gaining weight and strength,*

operations on arthritic patients result in no greater likelihood of infection, ankylosis or lack of union than in other diseases.

Osgood has stressed certain features which must be obtained before surgery is even considered in arthritic patients:

Quiescence of the arthritic process for months.
Optimal general condition of the patient.
Good morale of the patient.
Adequate facilities for hospital and follow-up care.

The *importance of intensive training in muscular exercise* and control for a period of *at least several weeks prior to the operation* has been stressed. The patient is taught preoperatively to perform those exercises which will be required following the surgical procedure.[85]

Before the orthopedic surgeon operates upon any affected joint, Wilson has suggested that he **determine the following:**[111]

1. Is the arthritis still active?
2. Is the patient in good enough physical condition to permit extensive surgical procedures?
3. Considering the age of the patient, his physical state and the number of operations required, is the expected gain in function from operation worth the effort?
4. Should the operation be delayed for any reason for institution of other therapy?
5. Will the patient be able to coöperate in after care with the proper morale and willingness to endure some pain?
6. Are the facilities necessary for successful treatment present in the hospital?
7. Will the patient be able to receive proper after care on leaving the hospital?
8. Is the patient's financial status such that he can go through the entire treatment?

The flexors of a joint are usually stronger than the extensors and the pronators are nearly always stronger than the supinators. When a joint is inflamed, the muscles surrounding the joint may show considerable spasm, with the stronger muscles outpulling the weaker ones, and causing deformities.

We *disapprove of manipulation of the joint in the acute stage of an arthritis,* but muscle setting and massage of muscles above and below the joints may be carried out as soon as the subacute stage has appeared. If surgery is done too early in the arthritic, it can do more harm than good. Surgery should always be looked upon as an adjunct in the treatment of arthritis. In quiescent or arrested cases, surgery may be required to increase the mobility of the body with a minimum expenditure of the patient's energy. Surgery deals with the mechanical factor, mainly, in the arthritic; with very few exceptions, it should be resorted to only in arrested cases.

Ankylosis, per se, does not require surgery. If the ankylosis is in poor position resulting in increased muscular fatigue, one should then employ the simplest type of osteotomy to correct the deformity. No surgery will

be required if proper preventive orthopedics has been used early in the disease as the ankylosis, if it occurs, will be in an optimum position.

Unfortunately, there is no hard and fast rule which must be followed as regards surgery in the arthritic. One should discover whether the deformity is due to contraction of tendons or capsules or whether there is an actual bony deformity. One will then decide in that given case whether tendon lengthening, capsulorrhaphy, osteotomy or another procedure is preferable.

Joint disability is often one of painful mechanics with a slight amount of bony deformity within the joint, such as bony spurs. Such bony spurs are found most often around the hip joint. In some cases, removal of these heavy spurs on the edge by acetabuloplasty is very beneficial.

Willard believes that the thickened, swollen synovia may be a source of infection in some cases, leading to involvement of other joints. Some surgeons perform synovectomies rather early in rheumatoid arthritis. Although the consenus of opinion is that the disease should be quiescent before performing this operation, some patients may never have a lowering of the sedimentation rate to normal and one must use his clinical judgment in suggesting that synovectomy be performed in certain patients when the maximum amount of clinical improvement has been obtained.[109]

Flexion contractures of the knees are common in the experience of the orthopedic surgeon. A flexion of over 15 or 20 degrees results in an unsteady gait in these individuals so that standing or walking may become very difficult. These flexion deformities are almost always due to contracture of the soft tissues—ligaments, muscles, fasciæ, tendons, etc.; such flexion contractures are very seldom the result of bony changes.

In **many minor contractures,** these **can be overcome by** the judicious use of serial plaster splints or casts, Buck's extension, turnbuckle casts and the like. In some, it is advisable to administer a general anesthetic and to then apply a cast when the maximum correction has been obtained by very gentle manipulation of the knee joint.

Major contractures may require surgical intervention. Posterior capsuloplasty of the knee joint, as devised by Wilson, is a very useful procedure in many of these individuals.

In those patients whose knee joints are filled with large, thickened synovial villi, often appearing at operation as numerous rice bodies, a **synovectomy may be performed** after the active disease has disappeared, or after activity has greatly lessened. These individuals have motion of the knee but complain of pain on motion with resulting increase in swelling and pain, in some cases with complete disability. In some instances one may notice herniations posteriorly into the popliteal space (formerly called **Baker's cysts**). At operation in these cases, one evacuates the contents of the joint and dissects the synovial membrane from above downward. It is sometimes difficult to completely clear the lateral recesses of the inflamed membrane. The pannus is scraped away from the cartilaginous surfaces. It may be necessary to remove the entire patella or to remove the posterior longitudinal half if its surface is greatly eroded, in order to avoid subsequent

traumatic arthritis. The infrapatellar fat pad is usually removed at this time. If an extension is present in the popliteal space, a posterior incision will be required for its removal.

Post-operative care is extremely important in patients following **synovectomy** of the knee. The posterior molded splint is usually kept in place for a week (extending from the fold of the buttock to the Achilles tendon). Early active motion is then encouraged to avoid adhesions. If the patient does not coöperate, after two to three weeks one must give pentothal or gas anesthesia and flex the knees forcibly to break up the early adhesions. Baking and massage are carried out during this period to improve the muscle tone. Weight bearing may be permitted in from two to six weeks.[73]

Principles in Joint Surgery[111]

Do not operate during the active stage of an arthritis; joint surgery should be attempted only after a period of at least six months of quiescence from joint activity.

Patients who have been inactive for years are poor operative risks.

Attempt to obtain a functionally useful joint. Orthopedic surgical procedures upon the upper extremity are concerned with the use of the arms, particularly for eating, dressing and personal care, while the leg problem is concerned with the ability of the patient to be able at least to arise from a chair and sit down again.

When both arms and legs are disabled, an attempt should be made to solve the arm problem completely before the leg condition.

Begin postoperative joint motion as soon as possible (when soreness has disappeared).

Do not maintain the joint in one position for long periods postoperatively unless absolutely essential.

Develop surrounding muscles by exercises for several weeks prior to operation.

Orthopedic surgical procedures available to the arthritic patient include: arthrotomy, osteotomy, synovectomy, capsuloplasty, arthrodesis, arthroplasty, forage, etc.

SYNOVECTOMY

Volkmann is credited with having performed the first synovectomy; this was done in a patient with tuberculous arthritis of the knee (in which condition the operation is now contraindicated).[82] Others have subsequently discussed this procedure.[25,62]

Synovectomy of the Knee.—Hyperplastic synovial membrane thickened and thrown into folds may cause disability even after the arthritis is inactive. Such hyperplasia in the knee often causes chronic effusion, pain and limitation of motion on attempts to fully extend the knee, tenderness around the infrapatellar fat pad, and roentgen evidence of soft tissue changes with only slight damage to the joint cartilage.

Swett performed his first synovectomy in 1911 and worked on this subject until 1922 before reporting his results. Since his report in 1922, a voluminous literature has been published on the value of *synovectomy* in rheumatoid arthritis so that the operation *is now regarded as a safe procedure in proper hands and in selected cases.* The results of synovectomy have been gratifying in some instances and disappointing in others so that the important problem is the selection of appropriate cases for this operation. Swett suggested in his first article that the operation was most likely to succeed when performed where the damage was entirely synovial with an extensive effusion and without ulceration of cartilage. In his cases, there was palpable synovial thickening, coarse crepitation, flexor spasm, generalized tenderness and painful movements of the affected joint. Roentgen examination revealed evidence of synovial thickening with very little if any diminution of the joint space and with slight, if any atrophic changes in the ends of the bones. He called attention to the distinction between the brittle, grating feeling caused by the rubbing of the bare ends of the bones as contrasted with the soft, squashy feeling due to tabs and fringes. Swett thought that maximum benefit was to be expected from synovectomy if this were performed before extensive atrophic changes had occurred in the ends of the bones and before the cartilages were ulcerated. He considered as unfavorable for synovectomy those cases with inflammation involving predominantly the articular cartilages, the ends of the bones, the joint capsule and the muscles.[103]

Selection of Cases for Synovectomy[44,45,100,101]

This is used most often in cases of rheumatoid arthritis with little or no involvement of the cartilage or bone (with most of the process localized to the synovial membrane).

Synovectomy at the knee is indicated chiefly in some chronic cases where synovial tissue and pannus have filled the anterior portion of the knee under the patella.

Patients with persistent swelling and chronic hydrops resisting other forms of therapy are often candidates for synovectomy.

There should be a preceding trial of conservative therapy, including splinting, corrective and muscle setting exercises before surgery is performed.[72]

Do not operate until acute inflammation has subsided.

Patients will require prolonged postoperative physical therapy.

There should be considerable effusion into the joint in suitable cases.

Synovectomy is most adaptable to the knee joint. It has been done for chronic synovitis, rheumatoid arthritis, traumatic arthritis, synovial osteochondromatosis, xanthoma of the synovial membrane, osteo-arthritis and intermittent hydrops.[21] In a series of 90 patients, following synovectomy, 35 obtained excellent results, 33 per cent were improved and 32 per cent were in poor condition. Cure or improvement followed the operation in all of the cases of traumatic arthritis, degenerative joint disease, osteochondromatosis and xanthoma. In chronic synovitis, 77 per cent were cured or improved; in chronic infectious arthritis, 56 per cent were improved or cured. Synovectomy is the choice of surgical treatment in intermittent hydrarthrosis.[49] It resulted in improvement in about 50 per cent of the selected cases of rheumatoid arthritis which were operated upon.[102]

Following synovectomy in selected cases, large compression dressings are used and active motion of the joint is begun several days postoperatively, with weight bearing after three to seven weeks. Some surgeons apply a cast for ten days and then begin passive and later, active movement; at a later date, manipulation may be necessary.[21]

New synovial tissue forms from the surrounding connective tissue after synovial membrane has been excised.

Inge has shown that *tuberculosis of the synovial membrane is a distinct contraindication to synovectomy.* He has reported a series of 86 cases of chronic non-tuberculous synovitis of the knee which were treated by synovectomy with good results.

Synovectomy of the knee is the removal of the synovial tissue of the knee as completely as possible; *i. e.*, dissecting out the anterior synovial membrane, the infrapatellar fat pad and the suprapatellar pouch. If there is an extension into the popliteal space (Baker's cyst), remove this through a posterior incision. In patients with marked erosion of the patellar surface, the entire patella or its posterior half should be removed. Wilson has advised that it is unwise to interfere seriously with pannus developing over the surfaces of the joint. He believed that this operation was indicated when there was a persistence of chronic thickening and swelling of the knees, with pain on motion and in some cases, chronic effusion, after other joint symptoms have disappeared. He advised that the semilunar cartilages be allowed to remain in place in arthritic conditions. Others believe that many failures in synovectomy are due to incomplete operation.[18] They state that successful synovectomies in the knee usually include removal of all hypertrophic osteophytes, semilunar cartilage and all cartilage destroyed or partly destroyed by pannus, leaving behind only that cartilage which appears satisfactory and undamaged.

In recent years, some workers have performed synovectomy relatively early in selected patients with rheumatoid arthritis and believed that their results were very encouraging. Previous workers had advised against early operative intervention in these patients because of the possibility: (1) of ankylosis, and (2) spread of the disease process. Work now in progress may help to decide this question. When one has seen the marked synovial hyperplasia with rice body formation in some of these knee joints, it is difficult to imagine how any procedure short of operative intervention could greatly aid such joints.

POSTERIOR CAPSULOPLASTY

Wilson has advised posterior capsuloplasty for some cases of contracted knees, when no severe damage of articular surfaces is present. Capsular contraction posteriorly in the knees prevents full extension and results in flexion contractures, although motion in flexion is preserved. Capsuloplasty is especially successful in those cases of flexion deformity of the knee when the patella is free and only a slight subluxation exists. Posterior capsuloplasty frees the capsule and is helpful in many of these cases.

ARTHROTOMY

This is rarely indicated in arthritis except in degenerative joint disease for removal of joint mice or hypertrophied villi.

OSTEOTOMY

Cutting of the bone close to the joint is occasionally indicated in flexion contractures of the knee if the articular cartilage is so badly damaged that operation upon the joint capsule is unlikely to result in joint motion, and to correct bony ankylosis in a deformed position. Various forms of osteotomy have also been advised in the hip region in the case of some of the flexed and adducted deformities in degenerative joint disease. Osteotomy of the lower end of the radius has been used in cases of fixed palmar flexion of the wrist.

ARTHROPLASTY[14–17,23,55,77]

The only surgical treatment of ankylosis is *arthroplasty* (*i. e.*, the construction of a new joint).[83] This is an operation to model an artificial joint. Satisfactory results have been obtained, especially in ankylosis of the *hips, elbows* and *knees;* results have been less good in the shoulders, fingers and jaw. Cases of ankylosis due to trauma usually respond well to arthroplasty.

Operation in an ankylosed temporomandibular joint is a necessity. Arthroplasty of metatarsophalangeal joints often produces satisfactory results; arthroplasty of the ankle is never justified.

In brief, the *technique of such an operation* is to divide the bones at the line of the former joint, smooth and trim the ends to simulate normal articular configuration and to stitch a sheet of fascia lata (removed from the thigh) or cellophane between the joint surfaces. The fascia prevents recurrence of the ankylosis. In fibrous ankylosis, fascia lata or cellophane is placed between the bony ends after the joint surfaces have been removed. Kirschner introduced the transplantation of fascia in 1909. Since then, it has been used in bridging hernial gaps, reconstructing muscle and tendon defects, and in arthroplasties to prevent ankylosis. Fascia may serve as living suture material. It is very resistant to infection and absorption.

The most favorable age group is from twenty to forty-five: operation under age of eighteen endangers epiphyseal growth. Arthritis should be inactive for at least six months before attempting operation. Operation is contraindicated in tuberculous joints because of the danger of stirring up a latent infection.

In ankylosis of both knees, arthroplasty of only one of these may be sufficient. When both hips are ankylosed, bilateral arthroplasty may be necessary. If both hips and both knees are ankylosed, arthroplasty is advised in one hip and the opposite knee. Following arthroplasty of the knee or hip, no weight bearing should be permitted even with braces for at least three months.[32]

The Smith-Petersen arthroplasty for the hips includes the ingenious procedure of placing vitallium molds over reconstructed femoral heads.[39] *Vitallium*, *leucite* and *cellophane* have been used successfully as interposition substances in arthroplasty in patients with rheumatoid arthritis.[78] *Vitallium* (65 per cent cobalt, 30 per cent chromium and 5 per cent molybdenum) is one of the best metals to employ in bone surgery. It results in no irritation of the surrounding soft tissue or bone and no discoloration or staining of the structures.[13]

Arthroplasty of the hip has become much more frequent since the introduction of the leucite and the vitallium cups. It is very important that proper exercise be performed beginning about ten to fourteen days after this operation. Emphasis should be placed on abduction exercises (because of the tendency for development of an adduction deformity) and on exercises for extension, flexion and internal rotation. *Short wave diathermy is not desirable after a vitallium cup arthroplasty* as the heat may be concentrated in the vicinity of the metal cup. One must also be careful in the use of heat if the cup used is of plastic material with a low melting point.

An *arthroplasty splint* has been devised for use following this operation. This splint extends the entire length of the leg, being broken at the knee with a movable hinge; it holds the foot at right angles to the leg. Universal rollers, which can move in any direction, hold the foot about 2 inches off of the surface of the arthroplasty board. A rope is fastened to the knee portion of the splint; this runs through an overhead pulley, attaching at the other end to a handle within reach of the patient.[106] Motion following arthroplasty of the knee should proceed more slowly than at the hip so that a lax and unstable knee does not result.

Tantalum, a little known metal (the seventy-third element in the periodic table) is superior to all other surgical metals except vitallium.[9] Tantalum is as strong as steel, resists corrosion and can be stamped, machined and drawn into wire. It appears to be absolutely inert in the human body. It has been used in orthopedic and dental work (machined to make arthroplasty cups, nails for femoral neck and intertrochanteric fractures, and screws to be placed in the jaw and capped by teeth). Unfortunately, it is expensive ($60 per pound); a $\frac{3}{4}$ inch screw costs $1.50; and a $3\frac{1}{2}$ inch bone plate, $5.00. At present, these must be made to order by hand.

CHEILECTOMY OR RECONSTRUCTIVE OPERATIONS

These include excision of any obstructive or interfering parts of the joint surfaces, such as excision of bony spurs (osteophytes) or the more complicated remodeling operations advised for the deformity of the hip joint in degenerative joint disease (morbus coxæ senilis), residual suppurative arthritis of the hip, etc.[33] Cheilectomy (removal of osteophytes) may be indicated in some cases of degenerative joint disease when the osteophytes definitely block movement.

ARTHRODESIS

Arthrodesis is the removal of articular surfaces in order to produce fusion of bony surfaces (ankylosis) or if performed as an extra-articular operation, the use of a graft to bridge a joint. It is useful in monarticular arthritis in which it is advisable to produce a painless, stiff joint, especially in weight-bearing joints where stability is more essential.[7] It may be done in the knee, hip, sacro-iliac joint, spine, shoulder, wrist, subastragalar or ankle joints. At the time of operation, any deformity may be corrected by the removal of an adequate bony wedge, allowing the joint to fuse in a position of optimum function. *Arthrodesis is ill advised in rheumatoid arthritis except in some instances when the midtarsal and subastragalar joints of the feet are involved.*[112] In rheumatoid arthritis of the spine, spontaneous ankylosis will usually obviate the necessity of surgical interference.

DRILLING OF THE FEMORAL HEAD (FORAGE)

Some investigators feel that the nutrition of a damaged hip joint may be improved by opening channels in cancellous tissue of the head of the femur. This operation, which is easy to perform, consists in drilling one large or several fine tracks in the long axis of the neck of the femur through a small incision over the great trochanter. Its exponents claim relief of pain within a few days and considerable improvement in joint function.[28]

The idea of bone drilling in arthritis resulted from improvement in a case of arthritis of the hip after fracture of the neck of the femur. MacKenzie claimed dramatic results following this therapy in many cases of all types of arthritis.[56,58] Improvement was noticed also in joints other than those treated. Kersley noted some improvement from this therapy in patients with gout and rheumatoid arthritis; the most striking improvement occurred in the group with degenerative joint disease.[48]

EXCISION OF THE PATELLA

Satisfactory results have been obtained in a small series of patients with chronic rheumatoid or degenerative arthritis of the knee by excision of the patella; these were patients who did not respond to conservative treatment.[6] This is an outgrowth of the work of Brooke, who removed the patella in treating fractures of this bone.[8]

In these patients, the greatest pain is present in the anterior part of the knee, and is aggravated by attempts to extend the knee (usually accompanied with subpatellar crepitation, and at times, irregular, cog-wheel like movements of the patella). The patella normally lies behind the quadriceps tendon, and is useful especially in that it enhances the power of the quadriceps and helps to form the capsule of the knee joint; it is not necessary to good knee function, as is seen in those patients with congenital absence of the patella.

Surgical excision of the patella is not a specific treatment for arthritis of the knee joint, but is used to eliminate a mechanical impediment to

function due to arthritic changes; following the operation, there results a decrease or absence of pain on extension as well as increased motion of the knee. The operation is undertaken only if the arthritis is quiescent and in the presence of definite roentgen-ray evidence of chronic arthritis of several years' duration, which has not responded to conservative therapy. It has been called a radical operation, but in some cases, seems to be justified. It appears that this operation may be of value in chronic burnt out arthritis where the patellar movements are limited and are the chief source of pain. It may be especially useful in some cases of traumatic arthritis.

MANIPULATION OF DEFORMED ARTHRITIC JOINTS

Hill and Holbrook have had marked success with manipulations of the knees, with follow-ups of good results many years later. Flexion deformities of the knees have been corrected by manipulation. Many knees which were stiff in extension were mobilized with good results, but some of these individuals develop postpatellar adhesions that make the situation more difficult, especially from the standpoint of pulling loose the patellar tendon. Following the manipulation, the legs are kept in a straight cast but are moved frequently to prevent recurrence of adhesions. If this is adhered to, contracted knees will not develop. Attempts to restore some motion in stiff knees (following arthritis) leaves one with all to gain and very little to lose if this is performed when the arthritis is quiescent. Manipulation of knees during the active stage of an arthritic process may lead to flare-ups of the disease in other joints.[43]

SULFONAMIDES AND PENICILLIN IN ORTHOPEDIC SURGERY

Implantation of sterile sulfanilamide or sulfathiazole powder in clean operative wounds decreases the incidence of postoperative infections and is a safe procedure. In contaminated wounds, the local use of sulfonamides lessens the incidence and severity of infection whether the wounds are sutured or left open. Sulfonamides are used locally and by mouth in full doses in acute pyogenic infections; chronic pyogenic foci in soft tissues and bone can be excised and the wound closed if sulfonamides are given in full dose by mouth and implanted in the wound before suturing this. Key has summarized the use of sulfonamides in orthopedic surgery.[50]

The use of penicillin in joint surgery is in its infancy. When susceptible organisms are present, 10,000 units of penicillin may be placed in the joint cavity each day in sterile saline.

JOINT TRANSPLANTATION

Transplantation of an epiphysis or one-half of a joint, especially if done autogenously, is a well-established procedure.[60] Transplantation of entire joints is impractical. Tietze was probably the first to perform a genuine joint transplantation, replacing the resected lower portion of the radius with a phalanx of a great toe.[104] Judet, in 1906, found that cartilage

was preserved in autogenous transplantations of joint surfaces in rabbits.[47] It is believed that the most favorable conditions in joint transplantation are the replacing of only half of a joint with preservation of the capsule of the host joint. Gill autotransplanted the second long metatarsal bone in dogs from one to the opposite paw and found the articular surfaces normal and the joints in perfect function, with no evidence of necrosis of bone eight months later.[22] Others (Haas, Phemister) concluded that the articular cartilage offers the greatest possibilities for successful transplantation of various parts of a bone, and that although the bone itself degenerates, it later is regenerated from periosteum and endosteum.[29,75]

Joint transplantation has been performed in human beings (Lexer), but this was soon given up and replaced by simpler procedures such as arthroplasty with interposition of fascia or fat tissue.

MOBILIZATION OF STIFF JOINTS BY OXYGEN INFLATION

Henson has used oxygen inflation under known pressure in an attempt to mobilize stiff joints.[40,41] He has preferred this procedure to manipulation under anesthesia and has not observed exacerbations of old infection or untoward symptoms in six years' experience with this form of therapy. Results include the mechanical breaking up of adhesions and stretching of inelastic capsules.

The method as employed is as follows: Procain anesthesia of the skin.

Introduction of a fine gage needle into the joint space.

Entrance of oxygen into the joint space through the needle with the gages set at 1 pound pressure.

Increase in pressure to tolerance (no narcotic is used except in very nervous patients). Most individuals will permit 2 pounds of pressure.

Withdrawal of the needle and compression of the needle opening.

Slight massage of the joint with attempts at motion.

Repetition of the procedure in five to ten days, beginning at the pressure previously tolerated (usually 2 pounds) and increasing to the point of discomfort (probably 3 pounds); this may later be raised to 4 pounds.

Roentgen films immediately after inflation to determine the amount of oxygen in the joint space (1 to 10 cc.).

Best results have been obtained in dry joints. Cases treated included stiff joints from prolonged immobilization, traumatic joint hemorrhage, specific or non-specific infections, etc. Henson has also used oxygen inflation to prevent the occurrence of adhesions in acute infections and in acute traumatic lesions of joints. More work will be necessary before this procedure can be properly evaluated.

AMNIOTIC FLUID CONCENTRATE

Amniotic fluid may be injected intraperitoneally without harm and may cause a stimulation of the defense mechanism of the peritoneum. Shimberg believed that if human and bovine amniotic fluid was effective in hastening the process of repair within the peritoneal cavity, it might

be of value in other serous cavities such as those of the joints.[79] He concentrated, fractionated and purified the amniotic fluid of cows two to four months pregnant. Such fluid contains small amounts of allantoin, which is of value in promoting repair.

When injected into joint cavities, amniotic fluid may: (1) separate the injured surfaces by distending the cavity; (2) stimulate a defense reaction in the intra-articular tissues with the formation of much fibrin; (3) reduce oozing; and (4) influence the lubricant factor in joint function.

The amount of fluid injected varied with the joint involved, being as much as 100 cc. in the case of a knee joint. Sufficient concentrate was introduced to distend the joint cavity and to produce at least a minimal separation of the articular surfaces.

Among 68 cases treated by this method there were 8 patients with rheumatoid arthritis, 1 with gonorrheal effusion and 4 with joint effusion of unknown etiology. In over one-half of the cases, the knee was the joint treated; other treated joints included the shoulder, elbow, hip, ankle and wrist. The therapy was also used in arthrotomies and in fractures involving the joints. The number of arthritic patients so treated was too small to permit adequate deductions but the results suggested that further work along this line might lead to encouraging results, especially in the prevention of intra-articular adhesions.

CONSERVATIVE TREATMENT OF DEGENERATIVE HIP JOINT DISEASE

If well developed roentgen changes of degenerative joint disease are manifest in the hip, conservative measures will usually produce only temporary palliation. Relief may be obtained by rest with avoidance of weight bearing, use of heat and analgesics. Temporary relief may be gained at times by local infiltration with procaine or procaine and eucupine.

Conservative Treatment of Degenerative Hip Joint Disease:

1. *Rest in bed* in *recumbency* on a *firm mattress.*
2. *Buck's extension* applied to the lower limb to relieve pain and correct the flexion and adduction deformity.
3. Application of heat several times per day: *hot fomentations* or radiant heat—thirty minutes three times a day and diathermy, once or twice a day, if available.
4. *Analgesics,* as needed: aspirin and phenacetin, āā 0.3 gram (gr. v) t.i.d.; it may be necessary to add small amounts of codeine phosphate, 10 mg. ($\frac{1}{6}$ gr.) q. four hours for a short period at the outset to control pain.
5. *Local injections* of *procaine* or procaine and eucupine may afford temporary relief.
6. If the patient must be ambulatory, *crutches* should be used and periods of rest in bed must be insisted upon daily.
7. *Immobilization* in a *plaster cast* or the use of a light ambulatory plaster hip spica will usually produce relief of pain, but are unpleasant; the short leather and steel spica to reduce hip motion to a minimum is usually not very effective in these patients.
8. The relief of pain is usually in proportion to the protection from weight bearing.
9. Surgical methods will usually secure relief from discomfort and require only two to three months in bed.

When pain is a prominent feature, the patient should be put to bed on a *firm mattress and extension applied to the lower limb;* in the presence of a movable hip, this is the most effective single medical procedure to relieve pain and to correct the flexion and adduction deformity. If the patient must remain ambulatory, crutches should be prescribed, together with periods of rest in bed each day. Moist or radiant heat or diathermy will also afford some relief of discomfort. *In most instances, the degree of relief from pain will be proportional to the protection from weight bearing,* and will be greatest in those with unilateral involvement.

SURGERY IN DEGENERATIVE HIP JOINT DISEASE

Although significant relief from pain and disability may be obtained for varying periods by conservative treatment of degenerative hip joint disease, *surgery offers the only lasting relief from pain and disability when conservative therapy fails.* In individuals who are fair or good risks for major surgery, arthrodesis and arthroplasty are the procedures of choice. Individuals with bilateral degenerative hip joint disease requiring surgery should have an arthroplasty performed. When one desires a movable hip, cup arthroplasty is the preferable procedure.[35,36]

Vitallium cups were introduced by Smith-Petersen for hip joint arthroplasty; since then, a synthetic plastic resin, leucite, has been used similarly. Leucite is also non-irritating and inert in tissues and is, in addition, radiolucent so that one can readily visualize changes taking place in the bone within the cup (which cannot be done if vitallium is used).

When *cup arthroplasty* is performed, relief from pain occurs within several days postoperatively and is permanent unless mechanical complications develop in the joint. Motion is restored at the joint with an average of about 30 degrees of flexion. Some degree of abduction and rotation are also regained. *In proper hands, the operation should produce an excellent result in about 75 per cent of the cases.*

In some cases, fixation of the hip by operative means (*arthrodesis*) is indicated. This is the *most certain* and *most conservative* procedure and should be done if the patient demands a relatively certain outcome. It is *especially of value* in individuals with unilateral degenerative hip joint disease whose occupation requires strenuous and prolonged use of the limb (clerks, laborers) and in the elderly who might not withstand the long period of operation needed for arthroplasty. Solid bony union may be obtained by dislocating the hip and removing the cartilage from the acetabulum and the femoral head and employing autogenous bone grafts from the tibia.[35]

Following arthrodesis, the patient is free of pain and his gait is improved. The disadvantages claimed include the increased likelihood of low back pain and the unsatisfactory sitting attitude; these are more theoretical than real.

Other operative procedures have been performed in degenerative hip joint disease. Watson-Jones secured ankylosis in these hips by a relatively minor surgical method of driving a *three flanged pin* (on a guide wire) *through the femoral neck and head and into the pelvis* (under roentgen control).[107] This procedure does not produce shock and can be completed in from fifteen to thirty minutes.

In patients with unilateral degenerative hip joint disease who are poor operative risks and who will not submit to arthrodesis, McMurray has performed *subtrochanteric osteotomy* with displacement of the lower fragment mesially to establish contact between the upper surface of the osteotomy and the pelvis. He reported relief of symptoms when an adequate shift in position was obtained.[61]

Surgeons have not reported as enthusiastic results as Mackenzie's own findings following *drilling of the femoral neck* in hip joint disease; Mackenzie reported that 80 per cent of his patients had good results. It has been suggested that the benefit may be due to relief of tension in the cyst-like cavities seen commonly in the femoral head.[57]

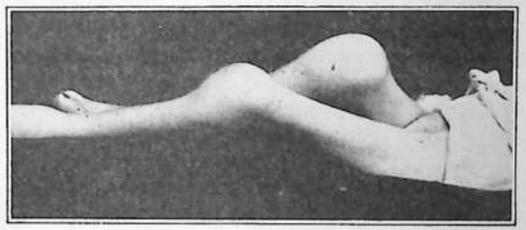

FIG. 82.—Flexion and adduction of the hips and flexed, swollen knees; extensive atrophy of the thigh and calf muscles. (Swaim and Kuhns, courtesy of Jour. Am. Med. Assn.)

Cheilotomy or *exostosectomy* (advocated by Jones and Smith-Petersen) of the femoral head or of the anterior and inferior acetabular rim with removal of osteophytic new bone will probably be replaced by cup arthroplasty when the patient desires a movable hip.[80,81]

ORTHOPEDIC SURGERY IN DEGENERATIVE JOINT DISEASE

This is limited to the removal of joint mice and to relief of a painful stiff hip (malum coxæ senile). *In degenerative joint disease one must strive for good alignment of the weight-bearing mechanism;* this may include treatment of weak feet to relieve strain at the knee, support for a pendulous abdomen to relieve strain at the hip and low back, and proper braces for relief of spinal strain.[65] *Foot drop is caused too often in arthritic patients by tightly drawn bed clothes.* Attempts should always be made to correct flat feet and flexed knees and hips; strain from these result in further strain in the back, neck and shoulder.

Arthrodesis is rarely indicated in degenerative joint disease except in some cases affecting the hip joint.[2] Whitman has devised a reconstruction operation for these cases.[108]

Arthritis Involving the Knees[46]

1. The knee joint, the largest joint in the body, is often attacked because it is a weight-bearing joint.
2. *Pain, swelling* and *permanent flexion* are the first signs of impending deformity, followed soon by atrophy of muscles and ligaments about the joint, narrowing of the joint space, subluxation of the tibia on the femur, fixation of the patella against the femoral condyles, contracture of the hamstring muscles, joint capsule and periarticular structures, and occasionally ankylosis.
3. In any acute arthritis involving the knee, lessen pain and prevent flexion deformity by the application of a *posterior molded plaster splint, extending from high in the thigh to the tips of the toes;* this must be removed at least once each twenty-four hours to permit several motions through as full a range as possible to prevent the development of adhesions. *Hot fomentations* may be applied for thirty minutes before moving the knee each day.
4. If *flexion deformities* are present, these may be *corrected by* the application of *serial casts* or *posterior molded splints,* or by *traction in the line of the deformity,* by wedging the casts or by the use of turnbuckle casts or in some cases if the arthritis is not too active, by gentle manipulation under anesthesia followed by the application of a plaster cast to maintain the correction. If plaster casts are used, these should be bivalved within forty-eight hours in most instances, using the posterior half as a resting shell. The knee joint should be moved through as full a range of painless motion as possible each day to prevent the formation of dense adhesions.
5. If *serial casts* or *plaster shells* are used to reduce flexion contractures, these are renewed every seven to ten days as improvement occurs. Hot fomentations may be applied to the knee joint several times a day to relieve discomfort and lessen muscular spasm.
6. When maximum correction has been obtained, this may be maintained by a *light metal leg brace or caliper splint, or by a light plaster shell* or a celluloid posterior splint. The patient should continue to use the plaster or celluloid shell during at least the entire night for months following the correction so that flexion contractures do not recur.
7. When a patient has been confined to bed for long periods with involvement of the knee joint, the quadriceps muscle should be strengthened as much as possible by *muscle setting exercises* performed every hour while the patient is awake, up to the limit of tolerance (without producing pain or fatigue). This will lessen the tendency to weakness of the quadriceps muscles and periarticular structures.
8. *Weight bearing should not be begun until* the patient can raise the lower leg and hold the leg extended horizontally over the side of the bed. If the quadriceps muscles are well developed, the patient may then attempt weight bearing with support of the knee joint by an elastic bandage with felt strips incorporated into this on both sides of the patella.
9. In patients confined to bed for long periods with arthritis of the knee, the knee must be supported more firmly when weight bearing is begun. One may employ caliper splints or a plaster leg cylinder with a hole cut out over the patella.
10. The atrophied muscles should receive *daily physical* and *occupational therapy* such as heat, massage and exercises employing the *overhead sling suspension cuff.*

Degenerative joint disease of the spine may require a spinal jacket or brace, Thomas collar, or the Goldthwait low short back brace with abdominal supporting pad; this latter is useful in cases of low back pain and sciatica.

Excision of the patella has been advised in early osteo-arthritis of the knee joint to relieve pain, especially when there is localized osteo-arthritis following fracture of the patella. Haggart has discussed the surgical treatment of degenerative knee joint disease.[30]

SYMPATHECTOMY IN CHRONIC ARTHRITIS

The sympathetic nervous system may exert an inhibiting influence upon the dilatation of blood-vessels. Although patients with rheumatoid arthritis have a high vascular-tone,[63] sympathectomy has not been particularly beneficial in these patients. This operation was suggested in an attempt to improve the circulation in peripheal joints in those with marked peripheral vasospasm.[1] Although it did result in some relief of pain, the course of the disease was not changed significantly. Such an operative procedure may be preceded by spinal anesthesia or foreign protein therapy; the amount of relief obtained by these procedures may give some gage of the improvement to be expected from sympathectomy. The operation is seldom performed at present for relief of arthritis.

ARTHROSCOPY

Direct visualization of the joint cavity may be obtained (if desired) by means of an arthroscope. Burman has found that injection of eosin into joints may cause diffuse staining of normal synovia and cartilage although such injection will usually permit better visualization of eroded cartilaginous areas.[11,12] Eosin is not irritating when injected into the joint cavity and thus may be a distinct aid in arthroscopy.

Arthroscopy may be done aseptically without fear of infection or trauma to the joint. Such examination provides a diagnostic arthrotomy without operation.[10,19] It is especially useful in patients who refuse arthrotomy or in medico-legal cases when operation is refused. This instrument should not be used until one has obtained considerable experience on cadavers.

DEFORMITIES OF THE KNEE FROM ARTHRITIS

The *natural tendency* for individuals with arthritis of the knee *is to flex the knee* to decrease pain and to relieve tension and the pull of the hamstring muscles upon the posterior portion of the joint capsule. This *results in shortening of the hamstring muscles and contraction of the capsule, and leads to further flexion of the knee.* **This deformity is usually begun by allowing a pillow to be placed beneath the knee.** Further contractions lead to subluxation of the knee (downward dropping of the tibia on the femoral condyles) and outward rotation of the tibia on the femur.[97]

A flexed knee causes shortening in the extremity, resulting in equinus and an unstable weight-bearing surface. The latter produces abduction and

external rotation of the hip to obtain a broader base on which to stand. Later, there follows a tilt of the pelvis and shoulders to the shorter side with lateral deviation of the spine (scoliosis).[87] Thus, flexion deformities of the knee may precipitate a traumatic flat foot, strain of the internal

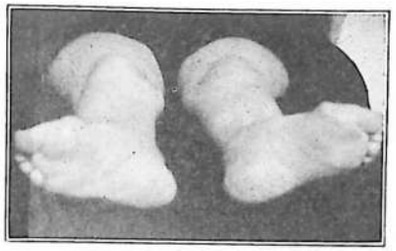

FIG. 83.—Outward rotated lower leg; note position of patella and of malleoli. (Swaim and Kuhns, courtesy of Jour. Am. Med. Assn.)

Prevention of Deformities of the Knee in Arthritis[98]

Flexion contracture and deformity of the knees in arthritis are signs of mismanagement on the part of the physician if he has been in charge of the patient since the onset of the arthritic process.

Permit **no pillow or other substance under the knees** (except as specified by the physician).

Insist upon rest in bed with absolutely no walking while the knees are swollen and painful.

Use a posterior molded plaster splint extending from the hip to the toes with the leg extended, and the ankle flexed to 90°, day and night, until all signs of acute inflammation have subsided. Keep the knee in about 10° of flexion (not hyperextended) in a splint; the nurse must not hold the leg only by the heel during the application of the splint.

Remove the splint daily to permit use of *hot fomentations* to the knees for fifteen minutes, and two or three full painless movements of the affected parts (to prevent the formation of dense adhesions).

Use splints for at least the entire night for several months to prevent the tendency to flexion, which may occur during sleep.

No manipulation should be performed during the active stage of an arthritis.

Following subsidence of acute inflammation, institute exercises for the knee; the *canvas cuff sling suspension* outfit or Russell traction may be of use.[38,59] See Figures 84 and 85.

Use extreme caution in exercises, never being so strenuous as to produce discomfort in the joint lasting more than several hours; ill-advised exercises may result in acute flare-up of the arthritis.

Do not allow the patient to get out of bed until he can fully extend the leg and hold this horizontally while sitting.

lateral ligament of the knee, knock-knee and traumatic synovitis, later followed by changes in the hip and scoliosis.

Canvas Cuff Sling Suspension.—The use of the *canvas cuff sling suspension* may prevent subluxation at the knee.

Apparatus for this consists of:[92]

Canvas—6 to 8 inches wide and 18 inches in length.

Heavy elastic cord (as in home exerciser sets) 2 to 3 feet long.

Felt padding, the same width as the canvas, 8 inches long and ¼ inch thick.

The 8-inch ends of canvas are sewn together forming a canvas cylinder (or cuff); the elastic cord is attached to this as shown in Figure 85. The cord is suspended from a horizontal bar (approximately 4 feet above the mattress) running lengthwise over the bed as in a Balkan frame, with the

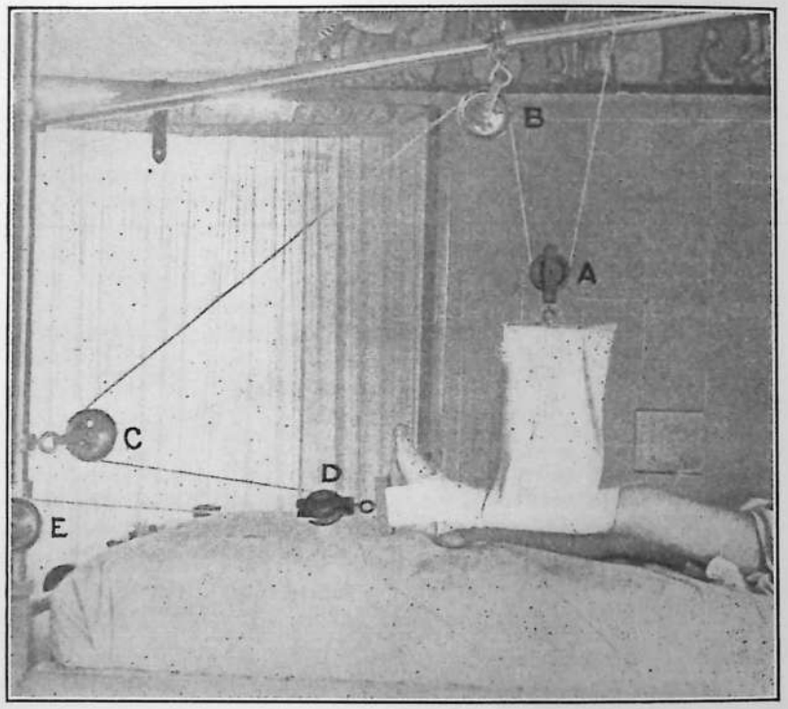

FIG. 84.—Modified form of Russell traction. *A*, *B*, *C*, *D* and *E* are pulleys. (Marians, courtesy of Jour. Bone and Joint Surg.)

canvas loop hanging from its lower end. The felt padding is sewn into the inside of the lower portion of the canvas cylinder; the sling is now pulled down upon its elastic cord, and the leg is slipped through this, with the upper portion of the tibia resting upon the felt padding inside the canvas cuff. This apparatus can be made easily in the home and costs very little. It also serves as a useful method of exercising the leg when this is permitted.

Correction of Flexion Deformities of the Knees.—If flexion deformities have occurred (usually from poor treatment or from failure of the patient to adhere to treatment), conservative orthopedic management may be sufficient to restore these to a practically normal position.

Use **posterior molded plaster splints** from the hip to the toes, removing

these once daily for hot fomentations, joint motion (if possible), and massage to the surrounding muscles if the acute stage of the process has

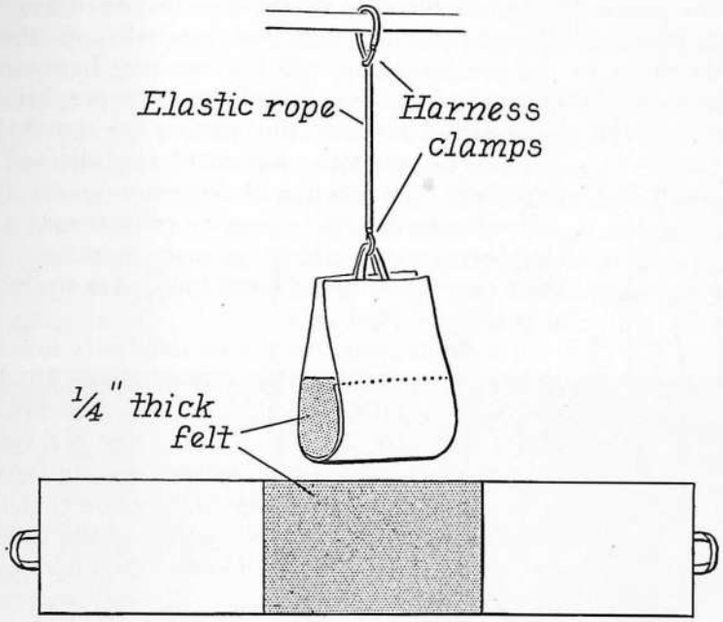

FIG. 85.—*Canvas cuff sling suspension* for support of the leg and for assistance in exercises in patients with arthritis of the knees.

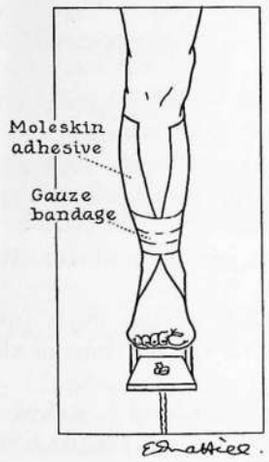

FIG. 86

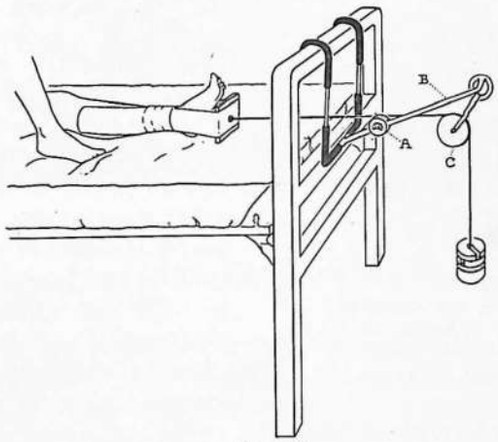

FIG. 87

FIG. 86.—*Buck's extension.* The moleskin adhesive is applied after the leg has been shaved and painted with compound tincture of benzoin.

FIG. 87.—*Buck's extension.* Traction to the lower leg. *A* and *B* are adjustable; *C* is a metal pulley wheel. Traction is exerted in the line of the deformity.

passed. Splints may permit muscular relaxation with progressive decrease in the deformity. Make new splints every two or three weeks as the condition improves until maximal improvement has been maintained.

As an alternative procedure, Buck's extension may be used applying traction in the line of the deformity. The leg should be shaved and *tincture of benzoin* should be applied liberally to the skin before applying the moleskin adhesive. The weights are increased gradually, as tolerated. When the deformity has been overcome, this position may be maintained by a light plaster splint until walking cylinders or proper braces are obtained. During the period of traction, the weights are removed once daily for two or three motions of the affected joints to prevent the formation of dense adhesions.

FIG. 88. — Thomas caliper splint: The lower ends of the splint fit into the heel of the shoe and part of the body weight is transmitted to the ground by the direct thrust of the rings against the tuberosity of the ischium. The opposite shoe should be slightly elevated. (Whitman.)

If a flexion deformity persists, *calipers* may be used for short periods (thirty to sixty minutes) several times each day with the knees buckled down as firmly as possible without pain.

In some patients, the flexion deformity is not completely overcome by serial plaster splints, Buck's extension or turnbuckle casts. In these, the remaining flexion contracture can usually be corrected by slight traction under anesthesia, followed by application of a light plaster cast or splint to maintain this.

Caliper Splints.—These are useful in the individual with some flexion of the knees and in whom one wishes to avoid further flexion when the patient is up and about. The main features of these splints are as follows:

Lateral steel uprights, one extending upward from the heel to the upper thigh on each side of the leg.

A light combination steel and leather thigh band attached to the upper end of the uprights.

Leather straps anteriorly at the knee joint and posteriorly below the knee joint to prevent further flexion of the knee.

Leather strap at the ankle joint to maintain the lateral uprights in place.

Sockets in the lateral aspects of the heels (anteriorly) into which the lower portions of the calipers fit.

Corrective turnbuckle casts may be useful in a flexion contracture deformity of the knee.[53] A simple form may be made as follows:[37]

Apply stockinet to the entire leg and use additional felt padding over the heel, Achilles tendon, malleoli, patella, quadriceps tendon and proximal third of the tibia.

Apply a long leg cast from the groin to the toes, incorporating in this a piece of copper screen 2½ inches wide and 6 inches long (over the patella), and 2 pieces of mild steel, 1 inch wide, bent at right angles (one proximal and one distal to the knee).

When the plaster has hardened sufficiently, cut the cast under the knee leaving a hinge of uncut plaster just proximal to the patella (and about the width of the patella).

Add plaster ropes from the ends of the projecting steel bars to the cast to prevent bending of the bars.

Attach a piece of clothes line between the projecting part of the bars.

Increase the tension in the rope for periods during the day; this tends to straighten the knees. See Figures 89 and 90.

One may omit the steel uprights and rope, if desired, *and simply insert a wooden wedge between the cut edges of the cast* and retain this in position by cotton sheet wadding with a thin layer of plaster covering; the size of the wooden wedge may be increased every four or five days.

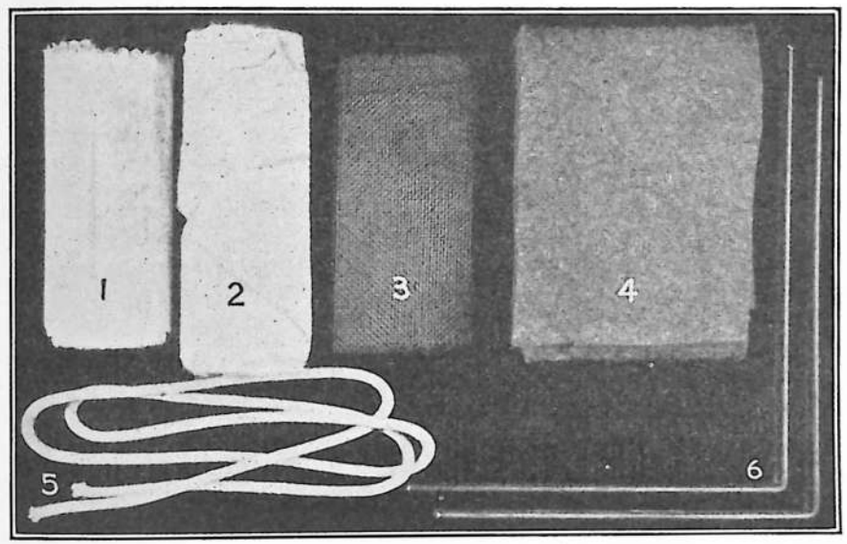

FIG. 89.—Materials used for wedging casts to correct flexion contracture deformities of knees: *1*, Plaster; *2*, sheet wadding; *3*, copper screen; *4*, felt; *5*, heavy clothes line; *6*, mild steel bars. One also employs a segment from a broom handle to twist the rope. (Hart, courtesy of Jour. Bone and Joint Surg.)

If deformities do not respond to splints, traction or wedge casts in a reasonable period of time (three to six weeks) one may resort to **surgical procedures** including capsuloplasty, tendon lengthening, and in some cases, osteotomies.

Posterior capsuloplasty produces good results when the posterior capsule is tight from adhesions, and flexed knees have motion with the patella free. *Synovectomy* is successful in some selected cases where pannus has formed in excess between the tibia and femur and synovial tissue has filled the suprapatellar pouch around the patella. *Arthroplasty* may be required after the process has become quiescent. *Arthrodesis* is seldom recommended as motion is then impossible; most orthopedists are opposed

to fusion operations on the knees; *the orthopedic surgeon should aim to preserve or increase joint motion when possible in arthritis rather than to eliminate it.*

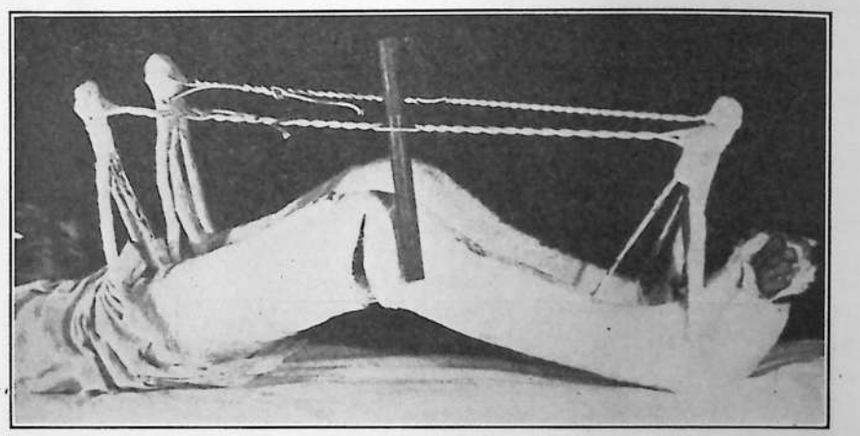

FIG. 90.—*Wedging casts* for correcting flexion contracture deformities of the knees. (Hart, courtesy of Jour. Bone and Joint Surg.)

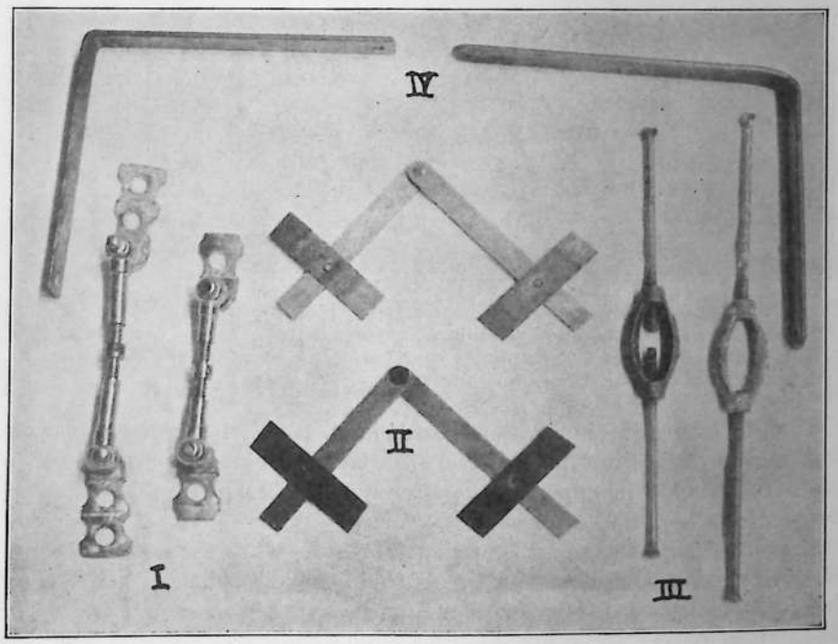

FIG. 91.—*I*, Hackenbruck type of *turnbuckle; II*, steel hinge with band on each end; *III*, ordinary buck saw type of turnbuckle; *IV*, piece of cold rolled steel, $\frac{5}{16}$ x $\frac{7}{8}$ inch bent at right angles. See Figure 92 for application of these. (Kulowski, courtesy of Jour. Bone and Joint Surg.)

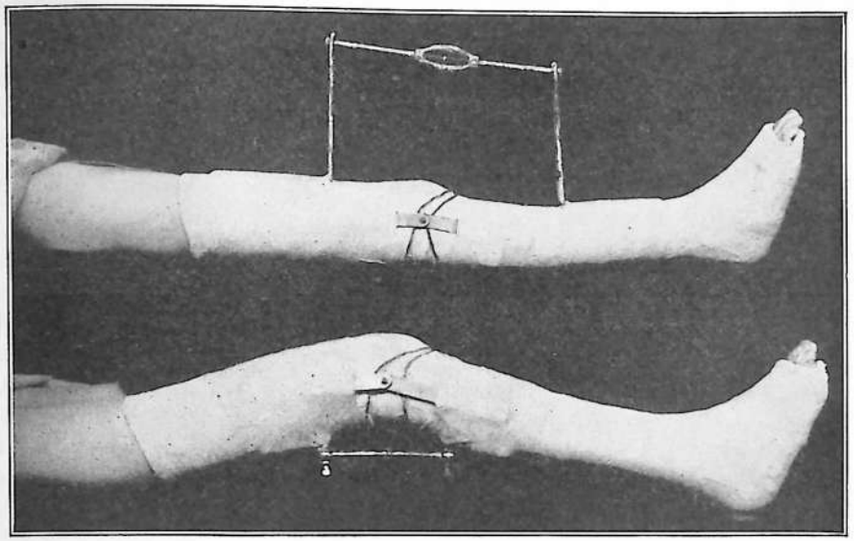

FIG. 92.—Upper figure: *regular type of turnbuckle cast* to obtain initial correction. In the majority of cases one obtains full correction with this. One may need to reapply turnbuckles after their full excursion range has been reached. Lower figure: Quengel method—used in cases offering considerable resistance to terminal stages of correction. (Kulowski, courtesy of Jour. Bone and Joint Surg.)

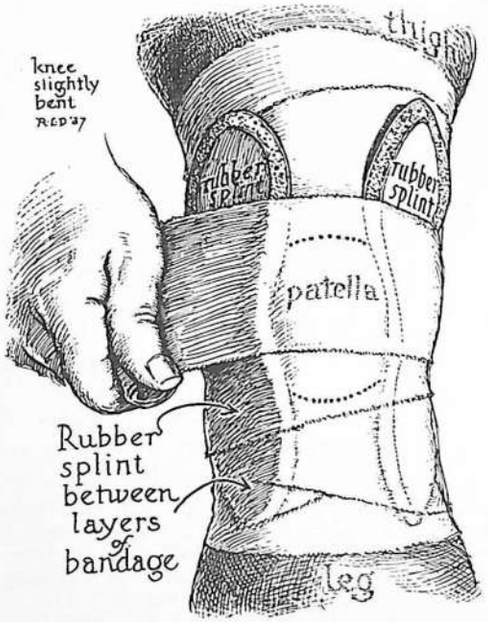

FIG. 93.—Combination *sponge rubber and elastic bandage*, partly applied to the left knee, featuring the shape and position of the rubber sponge splints. To complete the support, the bandage is continued to the top edge of its inner layer. (Truslow, courtesy of Jour. Am. Med. Assn.)

Supports for the Knee.—Elastic bandages: strapping should be begun below the knee joint and continued upward, covering an area of about 5 inches on each side of the joint. An inconspicuous bandage, known as Ace No. 7, has been designed for women. This can be obtained in widths of 2, 2½, 3, or 4 inches (with prices ranging from $1.00 to $1.50). The bandage is almost imperceptible under stockings as it has woven edges and a mercerized finish.

Truslow has described a simple method of support for injured knees which may prove useful in sprains, internal derangement, etc.[105] He winds a layer of 3 inch elastic bandage loosely around the knee (for 4 or 5 inches above and below the patella) and then applies lengthwise two long ovoid pieces of sponge rubber, one on each side of the patella; this is followed by another layer of elastic bandage with firm traction applied at each turn. The rubber sponges are approximately 8 inches long, 2½ inches wide and ¼ inch thick. A small crescent is cut out of each inner aspect to fit around the patella and the entire periphery of each sponge is bevelled (see Fig. 93). The bandage is applied with the knee flexed 10 to 15 degrees. (Heavy piano felt pads may be used if rubber sponges are not available.)

Elastic knee supports, with or without lacing, are often of great value in weak knees; if lacing is used, the felt pads as described above may be used on either side of the patella.

Elastic knee supports with hinged side joints: In place of the lateral felt pads, thin covered steel strips may be incorporated into the outside of the laced elastic knee support. Such steel strips may be approximately 10 to 12 inches long for adults, ¾ inch in width and made of 14 to 16 gage steel; these are hinged so that the joint comes at the level of the knee joint. The supports may be laced up the front. These cost approximately $12.

Adhesive strapping of the knee:

Ochsner's method of strapping is described elsewhere (see traumatic arthritis). The author employs a modification of the method of *strapping with adhesive* as described by Pemberton and Osgood.[71] Details of this are as follows:

The hair around the knee joint is shaved (with the skin dry).

Tr. benzoin comp. is applied liberally over the area to be strapped (to lessen irritation from the adhesive).

LEGEND FOR FIG. 94

FIG. 94.—*Metal leg brace* with hinge lock joint at the knee. Brace can be made without joint at knee if desired. Lower end of brace fits into metal sockets in shoes, from which they may be removed easily. Note laced leather thigh and leg supports and protective pad over patella. This type of brace costs approximately $100 per pair; straight leg braces, without the hinge joint and lock (seen best in *C*) cost approximately $75 per pair. Such braces prevent recurrence of flexion deformity but permit bending of the knee when the patient sits down (by opening the lock at the knee).

Note the laced leather cuffs above and below the knee and adjustable leather support over the knee. The metal hinge and lock is shown best in *C*. The lower ends of the leg caliper fit into metal sockets in the shoes.

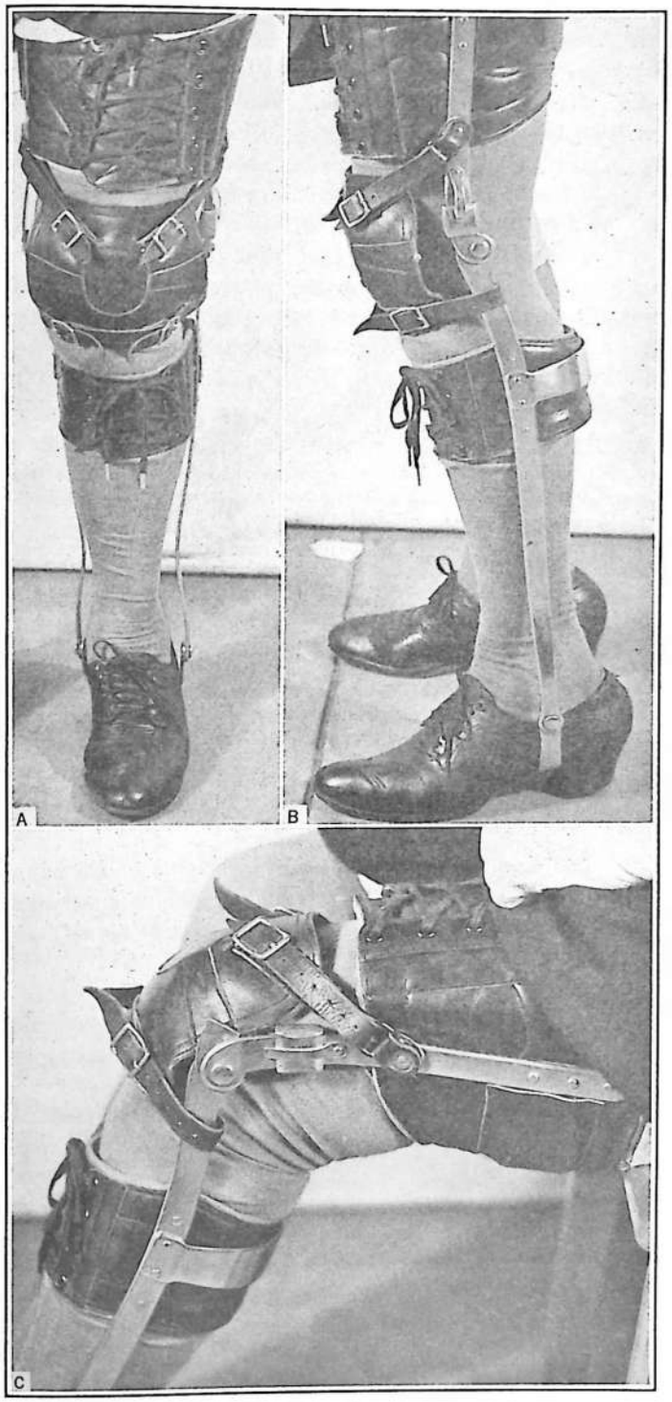

FIG. 94.

Straps of waterproof adhesive approximately ½ to 1 inch wide are used.

The *first strap* extends from 2 inches below the patella on the inner aspect of the knee upward and outward at an angle of about 30 degrees with the horizontal to the outer aspect of the knee. Strapping is applied while the knee is held in very slight flexion (just less than full extension). Straps are not extended posteriorly.

The *second strap* extends from 2 inches below the patella on the outer aspect of the knee, upward and inward at a 30 degree angle with the horizontal to the inner aspect of the knee.

Additional straps are applied alternately beginning internally and externally, each being somewhat higher than the preceding strap and overlapping this until the strapping reaches approximately 2 inches above the knee joint.

Straps are then applied transversely (horizontally) across the knee joint on top of the previous strapping to further strengthen the support. These begin with a strip directly over the patella and strips are placed above and below this in order until the diagonal strapping is covered.

A strip of adhesive is then applied longitudinally on each side of the knee joint to cover the cut ends of the adhesive.

Gauze bandage should be applied over the adhesive for several hours until the latter is held firmly in place. Liberal use of talcum powder over this support prevents sticking to the clothing.

PREVENTION OF DEFORMITY AT THE HIP

In arthritis of the hip, pain may be referred to the knee or to the thigh anteriorly.[98] *The first sign of deformity in arthritis of the hip is inability to completely extend the leg at the hip due to a flexion contracture.* Reflex muscular spasm results in flexion, adduction and in some cases, internal rotation and dislocation of the hip.

A fixed flexion and adduction deformity of the hip may cause shortening of the leg, equinus of the foot, flexion of the knee, knock-knee, flat foot, strain on the internal lateral ligament of the knee, lumbar lordosis and marked lateral deviation with rotation of the spine.[87]

Measures of value in arthritis involving the hip joint include:

1. Elimination of weight bearing by *rest in bed;* this relieves muscular spasm and pain.
2. The patient should lie on his back on a firm, non-sag mattress.
3. Maintain extension of the hip by sand bags or traction.
4. In hip joint disease, use traction with Buck's extension early, or in the presence of severe pain, apply a plaster cast with the leg extended as much as possible without discomfort (without abduction, adduction or rotation), changing the cast each three days until complete extension has been obtained.
5. Apply hot fomentations for thirty minutes two or three times a day.

6. When complete extension of the hip has been obtained, use a posterior molded splint and remove this daily for exercises and physical therapy.
7. The patient should not get out of bed until he can keep the thigh fully extended (*i. e.*, flex one knee with the other leg over the side of the bed; fully extend the latter leg without tilting the back).. Weight bearing is not permitted until all muscular spasm has disappeared.
8. If necessary, use surgical procedures such as manipulation under anesthesia (followed by use of a plaster cast for three or four days, enclosing the entire affected leg and pelvis); long continued traction or operative procedures.

Arthrodesis of the hip may be required in the later stages of a unilateral degenerative hip joint disease causing marked symptoms in young or

Arthritis Involving the Hip[46]

1. The first symptom may be pain referred to the anterior portion of the thigh and mesial aspect of the knee.
2. Examination usually reveals some muscle spasm after exercise and in some cases, a flexion deformity. If improperly treated, the process will usually progress to permanent flexion deformity and marked muscular atrophy.
3. *Hip flexion* may be *prevented or corrected* in many cases *by* having the patient *lie prone* (on his abdomen) in bed at least forty-five minutes per day with a pillow or two beneath the abdomen. The pillows are removed gradually as tolerated, allowing the hip to assume a neutral position when the anterior structures have been stretched.
4. If the flexion deformity is slight, it can usually be corrected by *hot fomentations* (twenty to thirty minutes three times a day) *and traction* in the line of deformity.
5. Muscle tone may be preserved by supporting the lower leg in a padded canvas cuff which may be suspended by an elastic cord from an overhead frame. This permits the patient to exercise that leg almost as easily as though he were in a Hubbard tank.
6. In acutely painful hip joints, it may be necessary to apply a unilateral or bilateral *plaster spica* to secure relief. It may be changed every week to ten days as an improved position is secured. In the presence of rheumatoid arthritis, the spica should not be used for long periods because of the possibility of formation of dense adhesions and diminished motion.
7. *If a deformity is present* without bony ankylosis, much of this can often be corrected by very *gentle manipulation under anesthesia* with maintenance of the correction by plaster spica casts.
8. *When* the patient becomes *ambulatory,* a *light-weight spica* may be worn; this may be removed when desired for physical therapy.
9. If ankylosis appears inevitable, the hip should be held in slight flexion, about 10° of abduction and without rotation.
10. *If conservative measures have failed* to relieve pain in degenerative hip joint disease, or if ankylosis has occurred in a poor position in the hip in rheumatoid arthritis, an *arthroplasty* with the aid of a vitallium or leucite cup should be considered for the production of a painless, movable joint.

middle aged patients in good general physical health; the reconstructive operation of Whitman is more applicable in elderly patients. A form of reconstructive joint surgery known as acetabuloplasty has been devised by Smith-Petersen in which there is a free excision of the upper and anterior portions of the hypertrophied margin of the acetabulum, in addition to a large triangular piece of the anterior portion of the capsule.[80] Arthroplasty with the aid of a vitallium or leucite cup will usually be preferred if conservative measures have failed to relieve the pain of degenerative hip joint disease.

Support for the Hip.—This may be obtained by a figure-of-8 application of *wide elastic bandage* to the pelvis and thigh as a spica, with strips of ¼ inch felting incorporated in the bandage.

A *Thomas leg splint* is often used; this consists of lateral steel uprights ending above in a leather covered and well padded ring which is so placed that the ischial tuberosity fits into the ring. The weight is transmitted through the tuberosity down the steel uprights. The lateral steel rods may end below as in the caliper splints (in the heel) or in a rubber covered base which acts as the lower end of the apparatus, and upon which weight is borne.

PREVENTION OF DEFORMITY OF THE FOOT

Strain or deformity may result from deformity of the knee or hip. Flattening of the so-called anterior arch frequently results from flexion of the knee.

The feet are pulled into plantar flexion (equinus) in patients confined to bed because of the weight of tight bed clothes, gravity and the pull of the strong gastrocnemius muscle. *Prevent toe drop while the patient is in bed with any illness.*

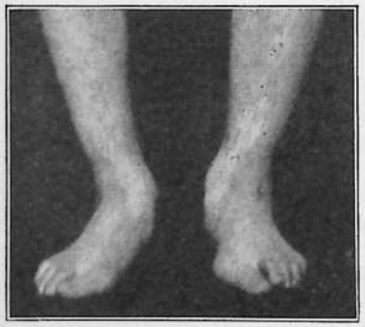

FIG. 95.—Early stage of deformity in arthritis, showing valgus and swelling about the ankles. (Swaim and Kuhns, courtesy of Jour. Am. Med. Assn.)

Foot drop may be prevented by:

1. Use of a cradle (such as an orange crate with one side removed) over the feet so that the weight of the bedclothes is removed from the feet.
2. Intermittent use of sand bags or pillows against the soles of the feet.
3. If a tendency to foot drop is noted, apply posterior molded splints to the feet for several hours each day, and during the entire night.
4. Slight elevation of the head of the bed and use of a board placed vertically at the foot of the bed against which the feet may rest.

In cases of sprain, one may use adhesive support and later an ankle corset or a canvas anklet reinforced with whalebone. Most cases of simple sprain respond well to immediate procaine injection with support to the ankle by strapping; ordinary motion should be encouraged immediately following the procaine injection.

A large soft foot covering (called the Mollo-Pedic Shoe) is available to cover swollen, tender feet or those in bandages or plaster. It consists of a sponge rubber sole and a fabric upper.

Detailed instructions on the care of painful feet are presented elsewhere (see chapter on Painful Feet).

Arthritis Involving the Feet[46]

1. Deformities at the knee or hip may result in foot strain by undue strain on the metatarsal heads (as may occur with a fixed flexed knee) or to strain of the longitudinal arch or pronation of the foot (as may follow external rotation of the leg).
2. Foot strain may appear on weight bearing in individuals who are confined to bed for long periods with resulting relaxation of the muscles and ligaments.
3. A valgus position of the foot may follow the muscle spasm associated with arthritis in the ankle or tarsal joints. An equinus position may result from the pull of the gastrocnemius muscle, gravity and the weight of the bed clothes. This equinovalgus deformity results in depression of the front arch on weight bearing and cocking-up of the toes with resultant hammer toe deformities.
4. **Weight bearing is contraindicated when the feet are painful and swollen** (in the acute stage of any arthritis). A plaster molded posterior splint may be used in these patients extending from below the knee to beyond the tips of the toes; this is removed *daily* for the application of *hot fomentations and motion* of the affected joints to avoid the formation of adhesions.
5. A *cradle* should be placed *over the feet*, or a pillow placed under the sheet at the bottom of the bed to keep the weight of the bedclothes off of the feet.
6. *When acute pain has subsided, foot exercises* may be begun.
7. *If deformities have occurred*, these may be *corrected by* the application of *serial casts or splints*, which are removed every day to permit slight motion and hot applications. If it is necessary to perform *gentle manipulation under anesthesia* to gain a better position, the plaster cast should be maintained for four to five days before bivalving. If necessary, fusion operations may be performed or hammer toes may be corrected radically.
8. Weight bearing is permitted when pain and swelling have disappeared; *good shoes* and adequate support must be applied to the feet.
9. *Figure-of-8 canvas walking straps* may be used to give additional support to the longitudinal arch and to prevent widening of the ankle mortise. A soft leather *laced anterior arch cuff* may be worn just proximal to the metatarsal heads, to give additional support, if indicated.
10. The question of proper shoes and supports for the feet is discussed later. (See chapter on Painful Feet.)

SUPPORT FOR THE LOW BACK

(Lumbo-sacral and Sacro-iliac Joints)

Adhesive strapping is the simplest form of support for the low back.

Proper *corsets* may be required (such as a modified corset brace with 2 para-spinal steel uprights, or if necessary, a metal pelvic band or a Camp

or Gossard low back corset). A polo belt or a motorcycle leather back support may also be employed as support for the low back.

The Goldthwait or Knight short back brace may be used.

Low back and abdominal supports and corsets are valuable only when they are fitted properly to the individual patient in an anatomic and physiologic manner. Because it believed that corsets and abdominal supports do not receive the attention of specialists, the Council on Physical Therapy will no longer consider or approve any special make of these products; firms having accepted products were asked to delete any seal or mention of Council acceptance.

Most orthopedic supply houses will fit a good low back support (for the male) which includes several para-spinal metal reinforcements for approximately $12.00. Polo belts cost only $2.00 or $3.00. The Taylor type back brace with shoulder straps and low back corsets costs approximately $30.00. Camp and Gossard corsets usually range from $8.00 upward.

Adhesive Strapping for the Low Back

1. Shave the hair on the low back and lateral portions of the lower abdomen.
2. Paint the low back with compound tincture of benzoin extending laterally to well beyond the anterior-superior spines.
3. If possible, the patient should stand erect.
4. Use strips of waterproof adhesive 2 inches wide, beginning just behind and above one trochanter and passing to a similar position on the opposite side.
5. The second strip is placed higher and in the opposite direction, overlapping the first by about an inch.
6. The third is still higher, extending to the iliac crests and is applied in the direction of the first strip.
7. The fourth strip is higher and runs in the same direction as the second.
8. A piece of cardboard, about 6 inches long, 3 inches wide at the top and 5 inches wide at the bottom, is covered by cotton wadding and gauze bandage. This is then placed over the adhesive, and is held in position by several diagonal strips of adhesive (running from below upward and outward).
9. The support is completed by 4 or more strips of adhesive placed horizontally as the initial strips.
10. Several short strips of adhesive are placed vertically anteriorly to cover the cut ends of the adhesive.
11. The adhesive is then well powdered.

SUPPORT FOR THE THORACIC SPINE

Adhesive strapping is the simplest form of support. If necessary, one may use a back brace with shoulder straps (such as a Taylor back brace with shoulder straps, incorporated with a low back corset).

By the use of plaster jackets, spasm of the erector spinæ muscles is relieved and flexion deformities of the spine may be prevented. If a body cast is used, this should be high in front and should come to the point of

Arthritis Affecting the Spine[46]

1. In *degenerative joint disease* in middle-aged or older patients, *most patients respond well to* heat, rest, support (with adhesive strapping, corset supports, steel back braces if required with elastic shoulder straps if the thoracic spine is involved) and correction of poor posture (if the latter is present).
2. In *rheumatoid arthritis of the spine*, the sacro-iliac joints are usually affected early and may fuse before marked involvement of the spinal column is noted. Chest motion may be limited and vital capacity be decreased by involvement of the rib articulations; the patient may be unable to turn to either side without moving the whole body.
3. **Roentgen therapy is one of the most useful measures in relieving pain and muscle spasm and in stopping the progress of a rheumatoid spondylitis.**
4. In rheumatoid spondylitis, the patient should rest in a *firm, non-sagging bed* and may obtain relief by the use of *hot fomentations* to the back. Boards may be placed between a thin hair mattress and the springs. In bed-ridden patients, a simple *posterior plaster body shell* may suffice temporarily.
5. The patient with rheumatoid spondylitis should have the spine immobilized in a plaster body jacket, and later in a leather or celluloid laced body jacket. The jacket may be removed for the use of heat and massage.
6. In *arthritis of the cervical spine*, rotation and lateral tilting of the head may be prevented by a *Thomas collar*, or by a felt collar or by the use of a medium-sized Turkish towel folded lengthwise twice and wrapped around the neck several times, and secured with safety pins. If the patient with cervical arthritis is confined to bed, sand bags may be used to hold the head in place and *head traction* may be required if deformity has occurred or if severe pain is present. *Hot fomentations* are useful in relieving pain and muscular spasm.

Strapping for the Thoracic Spine

1. The part to be strapped is shaved if necessary and is then painted with compound tincture of benzoin.
2. Adhesive tape 2 inches wide is used.
3. The strapping runs obliquely from below upward.
4. The *first strap* begins at the crest of the left ilium at its margin nearest the sacrum and runs upward and outward across the spine to the axillary border of the right scapula.
5. The *second strap* begins on the right side and runs from the right iliac crest to the axillary margin of the left scapula.
6. Three or four further strips are used on each side, alternately applying the right and the left, each one overlapping the previous strap on that side. Each succeeding strap begins further laterally below and ends further internally above the preceding strap on that side.
7. This will remain in position better if finally two or three strips of adhesive are placed longitudinally over this on each side of the spinal column, from above downward.
8. Powder is dusted over the adhesive strapping.

the kyphos posteriorly. A snug fit is desirable around the abdomen; this is loose above to encourage the patient to move the chest in breathing. It may be desirable to use a head-chin traction of 5 pounds while the cast is in place.

SUPPORT FOR THE CERVICAL SPINE

Thomas Collar.—This is easily made and consists of many layers of cotton sheet wadding, thin felt or cardboard (covered with cotton wadding or gauze and pulled through a stockinet) wrapped around the neck and fastened in position by a thin strap about the mid-portion. A satisfactory support may be obtained by wrapping around the neck a piece of thick felt about 3 feet long, and 4 inches wide at one end, tapering to 2½ inches at the other.[52]

A Thomas collar may prove very useful in the patient with arthritis involving the cervical spine. If felt is not available, a piece of cardboard may be used at home. This should be approximately 4 inches wide, being curved high to fit under the ears and cut out below to fit over the trapezius muscles. It should dip down in front to fill the distance between the mandible and clavicles. The collar may be fastened in the back of the neck by a gauze tie or buckle. This will relieve some of the strain on the cervical spine by allowing the weight of the head to be borne in part directly upon the shoulders.[75]

In some patients with degenerative joint disease of the cervical spine with narrowing of the intervertebral foramina, it may be necessary to employ traction by means of the Sayre suspension apparatus or utilizing a modified football helmet or heavy canvas straps, which are attached by a cord, passing over a pulley at the head of the bed. Weights may be used to produce traction, as tolerated. When symptoms have been relieved by traction, a light plaster or celluloid collar may be utilized.

SUPPORT FOR THE ENTIRE SPINE

One may utilize plaster casts or shells; Castex or waxed leather jackets (see Spondylitis) or a Bradford frame. A firm non-sag mattress and fracture boards should be used.

THE SHOULDER

Inflammations around the shoulder joint produce adduction and internal rotation of the arm. Stiffness of the shoulder may be caused by adhesions around the bursæ and joint and contractures of surrounding tissue (capsule, muscles, bursæ). *True ankylosis of the shoulder is rare in arthritis.*[99]

Limitation of motion at the shoulder usually results from calcification in the supraspinatus tendon with an associated subdeltoid bursitis. This is discussed elsewhere (see chapter on The Painful Shoulder). If proper positions and exercises are not performed following subsidence of an acute subdeltoid bursitis, marked limitation of motion may occur at the shoulder, resulting at times in a "frozen shoulder." The latter usually requires

gentle manipulation under anesthesia. This must always be followed by proper exercises of the shoulder for months to prevent a recurrence.

Keep the shoulders abducted and externally rotated to lessen pain and prevent deformity. To avoid deformity in arthritic conditions involving the shoulder, the patient should lie on the back for several short periods each day (thirty minutes, three times a day) with the hands under the head and the elbows flattened out and on the bed.

In the early stage of arthritis involving the shoulder, a cushion or muff in the axilla will provide an adequate amount of abduction. Plaster splints, casts or aeroplane splints may be used with the arm in abduction.

Avoid Deformities at the Shoulder by:[46]

1. Use of a flat, firm bed, with only one small pillow, if any, beneath the head.
2. The patient should lie with his *hands clasped beneath his head* several times a day for short periods; such a position permits abduction and external rotation of the shoulder.
3. In arthritis involving the shoulder, *support the arms* by sand bags, pillows or plaster splints *in moderate abduction* and *external rotation* to prevent forward drooping of the shoulder with consequent adduction and internal rotation of the arm. If the patient is ambulatory and other joints are not involved, an airplane splint may be used.
4. Pain and muscular spasm are relieved in the active stage of an arthritis of the shoulder by applications of *hot fomentations* for thirty minutes three or four times a day, following which the joint is moved actively or passively through as full a range of *painless motion* as possible for several movements to prevent adhesions. This motion may be assisted by the use of *shoulder pulleys* (consisting of awning pulleys or screw eyes and rope with the free ends of the rope held in the hands and the pulleys placed at the head of the bed).
5. Following subsidence of the acute stage of an arthritis, heat and massage of the shoulder may be followed by gradual stretching using the shoulder pulleys or *occupational therapy*.
6. *Gentle manipulation under anesthesia* is a safe and effective method (in skilled hands) to obtain increased range of shoulder motion if conservative treatment fails. Shoulder motions must be continued at least several times a day following manipulation to prevent recurrence of adhesions.
7. Ankylosis is rare at the shoulder joint; surgical intervention is necessary in such cases for restoration of motion.

Abduction of the shoulder in ambulatory patients can be maintained by a shoulder abduction cap with a wrist strap.[70] This consists of an ordinary canvas or leather cap which fits snugly over the head and buckles under the chin, with a reinforced loop on the top of the cap. A leather wristlet is applied to the wrist of the involved arm; the latter is raised and the wrist is placed on top of the head. It is held in this position by buckling a small leather strap from the wristlet to the head cap. This may be of use in patients with subdeltoid bursitis.

When an arthritis becomes quiescent, deformities may be overcome by exercise, traction or manipulation under anesthesia (see section on the Painful Shoulder).

ELBOW

Contractures may be prevented by the application of a molded plaster splint to the flexor surface with the elbow in full extension; this may be removed several times each day for physical therapy and exercise. One may employ elastic bandages, elastic elbow cuffs or elbow braces.

If ankylosis is inevitable, the elbow should be kept at slightly less than

Management of Arthritis of the Elbow[46]

1. The elbow joint is often misused; deformity and limitation of motion may result from making the elbow a weight-bearing joint when the arthritic patient uses a cane or supports himself by holding on to tables and chairs.
2. In the acute stage, rest the arm and hand in a *molded plaster splint* in extension (as tolerated). Active or guided *motion* must be performed several times a day to prevent formation of dense adhesions.
3. If there is any suggestion that ankylosis may occur, the joint should be placed in a useful position: usually slightly less than a right angle with supination of the forearm (or a position midway between supination and pronation).
4. Following the acute stage, apply *hot fomentations; gentle manual stretching* may be attempted following the application of heat. Stretching may also be accomplished by having the patient carry increasingly heavy weights, such as sand or water in a bucket. Traction may be required.
5. *Manipulation of the elbow is not advised* as it usually leads to further stiffness.
6. If the disease is quiescent, an *arthroplasty* may be performed if ankylosis has occurred in bad position.

Arthritis Involving the Wrist[46]

1. Gravity and the pull of the flexor muscles produce a deformity of palmar flexion, thus decreasing the normal grasping power of the hand.
2. Apply a molded *plaster* or Castex *splint* with the joint in about 30° of dorsiflexion, extending from the upper forearm to the palmar crease; the splint may include the fingers if these are painful and swollen.
3. *Remove the splint at least once a day* to permit several active or assisted *motions* to prevent the formation of dense adhesions.
4. If marked pain and muscle spasm are present, *hot fomentations* may be applied several times per day.
5. If deformity is present, a series of cock-up splints may gain increased motion.
6. If ankylosis is inevitable, allow the wrist to fuse in a useful position and, if necessary, manipulate the wrist under anesthesia to obtain sufficient correction before firm union of the bones occurs.
7. If the bones have ankylosed in a bad position, this may be corrected surgically producing a fusion with 30° of dorsiflexion.

90 degrees of flexion. If ankylosis has occurred, surgical procedures (such as arthroplasty) will often produce a useful joint.

Manipulation of the elbow usually leads to further stiffness and is inadvisable.

WRIST AND HAND

The use of a *plaster or metal cock-up splint* will usually prevent flexion deformity. The wrist is held in about 30 degrees of dorsiflexion. Such a

Arthritis Involving the Hands[46]

1. Prevent ulnar deviation of the hand and poor position of the fingers by the use of a *molded plaster splint* extending from the forearm to the finger-tips, with dorsiflexion of the wrist and moderate flexion of the fingers; a flange on the ulnar edge at the hand prevents ulnar deviation of the fingers.
2. *Warm paraffin dips* may be used in the acute or chronic stage to relieve muscle spasm and pain. Five to eight thicknesses of wax are applied by dipping the hand into the melted paraffin wax in a double boiler and allowing the wax to cool on the hand, forming a glove. This may then be covered by a warm towel or blanket and the heat dissipated gradually in from thirty to sixty minutes, following which the wax glove may be peeled off and placed back in the pot for use the next application. (See instructions in chapter on Physical Therapy for details.)
3. *Finger exercises* daily will aid in maintaining motion. These include squeezing a rubber ball; attempting to extend the fingers; attempts to separate the fingers from each other; and touching the tip of each finger to the tip of the thumb several times per day.
4. Wood carving, needle work and other forms of *occupational therapy* are very helpful in these cases.
5. Subluxation of interphalangeal joints may be corrected by *manual traction*, followed by application of small wooden splints cut from wooden tongue depressors and held in place on the palmar aspect of the fingers with adhesive tape. Banjo splints with finger traction are often of value.
6. The *"clam-shell" cuff* devised by Swaim to prevent interphalangeal and metacarpophalangeal subluxations consists of a piece of felt and a plaster slab which is molded and permitted to harden around the subluxed joints in corrected positions. When dry, it may be held on the hand with an elastic band or webbing strap. It is easily slipped off and on the hand.
7. Avoid excessive weight bearing on the outstretched hand as this may produce a "flat" hand with loss of the palmar arch.
8. If stiffness of the hand is inevitable, it should be placed in a position of dorsiflexion at the wrist, flexion at the metacarpophalangeal joints, partial flexion of the fingers and apposition of the thumb.

splint extends from the upper third of the forearm to a point just distal to the metacarpophalangeal joints, or if the phalanges are involved, to the tips of the fingers. The fingers and metacarpophalangeal joints are held in slight flexion (see Fig. 96). A flange is placed on the ulnar side to prevent ulnar deviation of the fingers. Such a splint may be worn during

the night only, or may be used during the entire twenty-four hours if comfortable, being removed at intervals for physical therapy and exercises. A plaster splint can be made easily if the hard coated plaster bandage is used; this will harden within five to eight minutes. This splint will prevent the common tendency toward the ulnar deviation of the fingers.

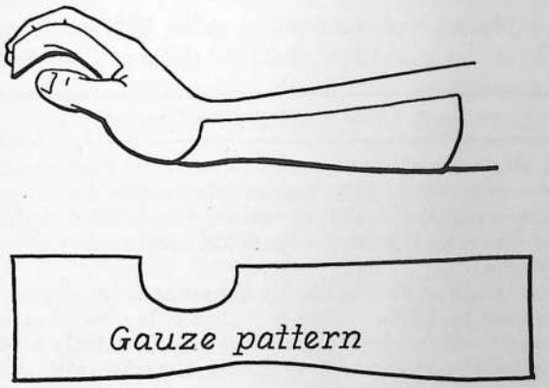

FIG. 96.—Cock-up plaster splint for rest of arthritic wrist or fingers

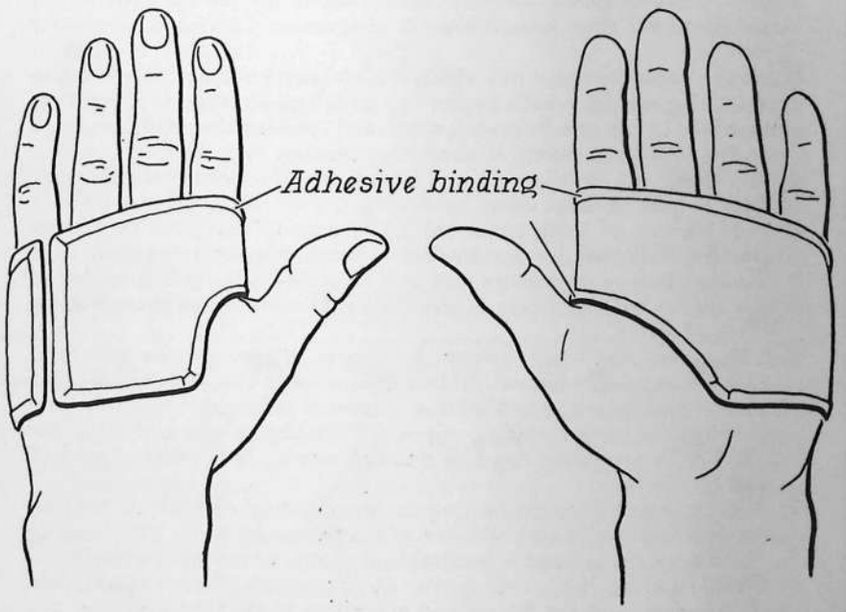

FIG. 97.—*Plaster cuff* for arthritic hand (to prevent ulnar deviation of fingers; suggested to the author by L. T. Swaim.)

A small *cuff of plaster* may be made to extend from the base of the thumb to just beyond the metacarpophalangeal joints.[92] This splint leaves the thumb free. It is from 3 to 4 inches long and is cut on the ulnar side so that it may be easily applied and removed. It can be held in position by

1 inch tape fitted with a buckle. This simple splint will prevent ulnar deviation of the fingers (see Fig. 97).

The patient must not bear weight upon the palms while moving in bed. The deformities which occur most commonly in the fingers include ulnar deviation of the fingers producing the flipper hand; hyperextension or flexion contracture of the metacarpophalangeal joints; interphalangeal dislocations and interphalangeal flexion contractures.[84]

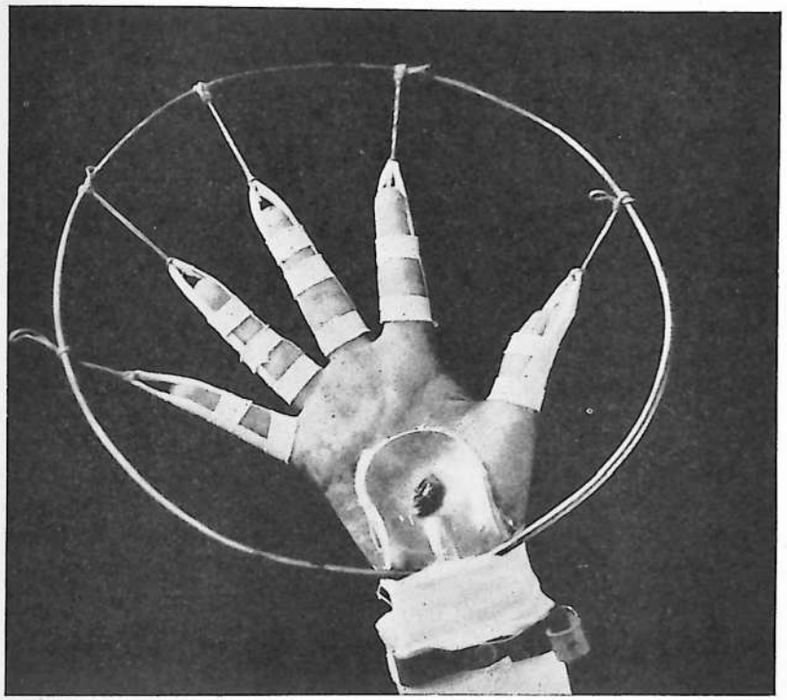

FIG. 98.—Banjo splint used to obtain extension of fingers and wrist. (Meyerding, courtesy of Proc. Mayo Clinic.)

Deformities may be overcome in some cases by *traction*, as by the *banjo splint* with adhesive strips on the dorsal and volar aspects of the fingers; or by plaster splints, changing the splints as the deformities lessen. A *banjo splint* is merely a loop of 3/16″ wire, fastened to a splint at the wrist and continuing in a circular fashion about 3 or 4 inches beyond the finger tips. In deformities of the fingers, small canvas loops are attached to the wire frame by elastic bands so that traction is exerted upon the deformed finger (usually being applied at the onset in the line of the deformity) (see Fig. 98).

If necessary, manipulation may be used to correct the deformity, followed by immobilization of the hand in splints for several days; physical therapy and exercises are then begun. Kuhns has advised that no manipulation be undertaken in the hand unless the arthritis has been quiescent

for at least twelve months.[51] A number of operative procedures have been described for deformities of the wrist and hand.[24,34]

Arthritis of the Temporomandibular Joint[46]

1. Limited motion occurs frequently, but *ankylosis is rare.*
2. *Hot fomentations* are useful in the acute stage.
3. Limitation of motion may be prevented by the *chewing of gum* after the subsidence of acute symptoms.
4. The range of motion may be increased by *cork or wooden wedges* inserted between the teeth.
5. *Arthroplasty* of the temporomandibular joint is difficult and should be performed only by one trained in this field; it may be resorted to in severe cases as it is attended by a high degree of success.

Miscellaneous Aids.—For the arthritic invalid who is unable to ascend or descend stairs, a *small elevator or ascending stairs* can be built into the home. Such small elevators can be made large enough to accommodate a wheel chair. The escalator can be made so that the patient may ascend while seated in a chair. These services are available in practically all large towns.

Counterbalanced Stairway Chair.—One of the misfortunes in arthritic patients with involvement of the knees or hips is that they should not climb steps; thus, the patient is often confined to the second floor unless sleeping and lavatory facilities are available on the ground floor. This can be overcome rather easily in the average home by building (at about $15.00 expense) an **apparatus** by means of which the patient can be safely **transported up and down a stairway.** About half the width of the average stairway is usually required for this purpose.

The apparatus is made as follows: Two long two by fours are fastened parallel on to the steps, beginning at the top, and extending to the bottom of the stairway; they are placed about 10 inches apart. A wooden chair is made with two short legs posteriorly and 2 long legs in front. Roller skate wheels are fitted into the bottom of each leg so that the chair will slide up or down on the two by fours on its roller skate wheels. A wooden guide is placed along the outside of the two by fours so that the chair cannot slip off of the track.

At the top of the stairs is placed a grooved wooden pulley wheel. Passing over this is a $\frac{1}{4}$ inch marine tiller line (non-stretching cable) which is fastened at the upper end to the chair and at the bottom of the stairs to a small wooden box whose width is equal to the distance between the two by fours (about 10 inches). The box has attached at its sides (below) roller skate wheels. The box is filled with steel plates sufficient in amount to counterbalance the weight of the patient who will be placed in the chair at the top. The rollers on both sides of the bottom of the box slide along the inner side of the track made by the two by fours.

An important feature of the apparatus is a hook placed at the bottom to hold the chair at the bottom when it is not in use; this should be constructed so that it cannot be easily unhooked by children or others, but so that it can be easily unhooked by the patient after she sits upon the chair. This is extremely important as the main hazard of the apparatus is that someone may unhook the empty chair at the bottom (allowing the weight box to crash rapidly to the bottom of the two by fours).

Inasmuch as the patient is counterbalanced by the weights in the wooden box, the individual is slowly taken downstairs in the chair as the counterbalanced box rises. When the individual is ready to come upstairs again, very slight pushing is required to have the chair rise on the two by fours as the weighted box descends to the bottom.

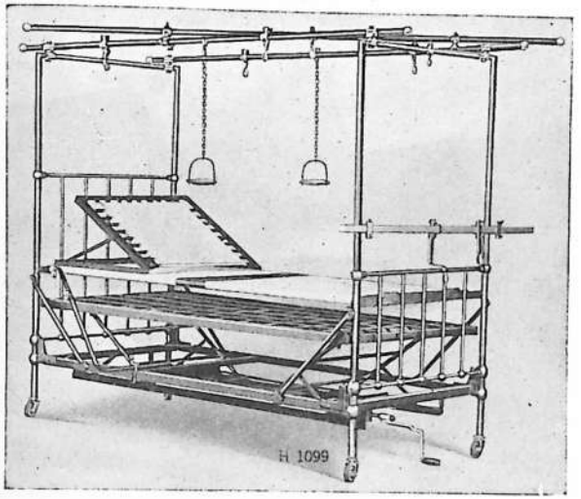

FIG. 99.—The Hanflig Orthopedic Bed. (Frank A. Hall & Sons.) Note the adjustable over-head Balkan frame; the Bradford frame (which is adjustable, supported at both ends, and permits shifting of the canvas to vary the position of the bed pan opening); the spring and mattress (may be raised or lowered); movable canvas back rest and arm supports.

A **wooden folding chair** with arm rests and a canvas seat and back, with roller wheels is a great aid to the arthritic patient. Such a chair is manufactured by the Gold Medal Folding Furniture Company of Racine, Wisconsin. It usually sells for approximately $15.00. This type of chair has been available at Sears, Roebuck and Company and by Abercrombie, Fitch and Company, of New York City.

Hanflig has described an **improved orthopedic bed** which may be useful in treating arthritic patients who are confined to bed.[34] The essential features are: an apparatus simulating a Bradford frame consisting of 2 long bars, which slide across 2 shorter cross bars at the ends of the bed by means of adjustable stops.

Two pieces of canvas are attached to the long bars which can be narrowed or widened by turning the long bar with a rotating ratchet device on its end so that the canvas can be made tight at any width.

A gear arrangement with a crank at the foot of the bed permits the spring with the mattress on it to be lowered and raised to the Bradford frame at will. The spring is really a flat metal sheet with many circular openings to give firmness and some flexibility.

Wheels made with long shackles slide up and down in the bed posts so that the head or foot of the bed can be raised.

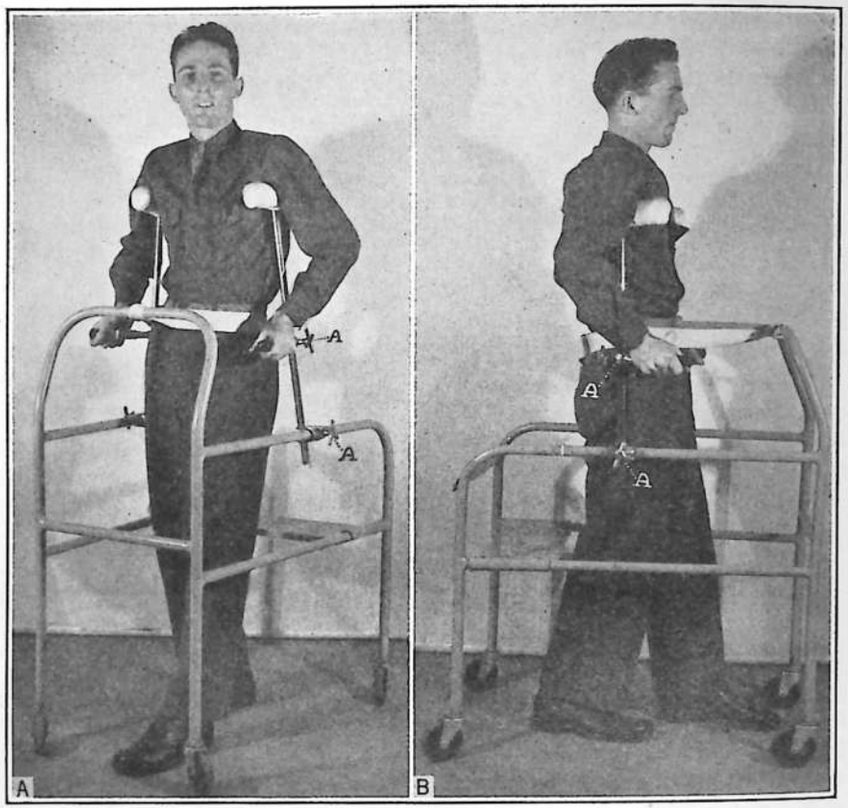

FIG. 100.—A metal walker which is very useful in reëducating the arthritic patient to walk after he has spent some time in bed. Arm and hand rests are adjustable by screws at *A*. Canvas strap holds patient and gives him added confidence. Metal seat behind patient may be used if patient tires. Walker costs approximately $75; this can usually be rented at low rates.

A light aluminum pillow rest, hanging on two pins at the head of the bed, raises the patient's pillow.

Other apparatus which may be useful in the office or hospital where large numbers of arthritic patients are seen include:[92]

The High Seat Chair.—In such a chair, the legs are especially long (the seat averaging 25 inches from the floor). The primary purpose of this chair is comfort for individuals with ankylosis or deformity of the knees or hips. These patients can sit on high chairs with comfort without bending the affected joints.

The Metal Roller Chair.—Bathing the arthritic patient is often difficult. This is simplified by lifting the patient on to a metal chair with rubber rollers inserted into its legs so that the chair can be wheeled to the bathroom and into a stall shower. Following his shower, the patient can be dried or wheeled back to his bed where final drying may be completed. This is especially useful in large institutions dealing with many arthritic patients.

The High Toilet with Arm Rests.—The toilet is raised (built up) so that the seat is about 10 to 12 inches above the usual position. The arthritic patient with involvement of the hips or knees can sit comfortably upon this if horizontal bars (metal) are placed as rails (just below shoulder level) which the patient may grasp in sitting or rising.

The Individual Pool.—A small pool lined with stainless steel can be built inexpensively by any large institution. With the aid of a plumber and engineer (usually available in hospitals) such a small pool may be built for less than $75. The water is maintained at any desired temperature (usually about 100 degrees for home exercises) and the patient is lifted into and out of the pool by a Bradford frame so that no discomfort is caused by handling. If chlorine is added to the water, it may be used all day in the clinic.

A portable, waterproof canvas tank, 24 inches deep, 6 feet long and 4 feet wide, suspended from a frame 43 inches high, has been devised.[69] Standard faucets are incorporated into 2 opposite corners to permit rapid filling and emptying. The frame consists of interlocking galvanized pipe which can be taken apart easily. When the tank is not in use, it can be folded. The bottom of the tank is kept rigid by folding boards held under it by a folding steel framework. An angulated Bradford frame is suspended from one edge of the frame and rests on the bottom of the tank. The patient's head rests upon canvas straps which are slung across the head of the tank. Water may be run in by a garden hose. The tank may be sterilized by chlorinated lime, and washed with soap and water.

Combination Reading Lamp and Book Holder.—Arthritic patients who are confined to bed and deprived of free use of the hands will appreciate such a device which is built on a floor stand. Such an apparatus can be purchased from the Milwaukee Book Bracket Co., 622 Colby Abbot Building, Milwaukee. The cost is approximately $16.25. The patient can read a magazine or heavy book, while in any position, without holding the book.

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CHAPTER XIX

DIET IN RHEUMATOID ARTHRITIS

UNFORTUNATELY, some arthritics have deleted meat from their diet because, possibly, of their confusion with the restriction of purines by some in the diet of gouty individuals. Inasmuch as the patient with rheumatoid arthritis needs these important body-building substances, meat must be consumed in ample quantities.

Other arthritics have shied away from oranges and other citrous fruits because of the old thought that these substances produced "acids" in their bodies and aggravated their rheumatoid arthritis. Because of their content of vitamin C, citrous fruits should be taken liberally by these individuals.

It has been shown that the calcium and phosphorus metabolism is grossly normal in patients with rheumatoid arthritis although there is a slight tendency toward an increased rate of calcium metabolism and increased calcium excretion in patients with rheumatoid arthritis; the magnitude of this loss is small.[24]

Numerous investigations have been made of the diet of patients with various forms of arthritis.[2,7,9,11–13,18–20,23,27–30]

There is no proof that lack of any specific food is a causative factor in rheumatoid arthritis.[3] Bayles and his associates studied the dietary history of 31 patients with rheumatoid arthritis for the year preceding the definite onset of the disease process. The food intake was apparently the same as that of a cross section of the families in that section. However, *more than 60 per cent* of the patients *fell short of* the National Research Council's recommendation for *calcium, thiamine* and *riboflavin; 50 per cent had an inadequate intake of vitamin C* and a small number were deficient in iron, protein or vitamin A or its precursors. These workers felt that if a food deficiency contributes to the onset of rheumatoid arthritis, it must be caused by an increased total requirement of the patients rather than in a deficiency in his average diet.

A large number of patients with rheumatoid arthritis exhibit a red smooth tongue and skin changes seen in vitamin B deficiency. The frequency of evidence of deficiency states in patients with rheumatoid arthritis suggests that this condition may increase the food requirements, especially of the vitamin B complex.[3]

There is no good experimental evidence to support most of the diets that have been advocated in the past in patients with rheumatoid arthritis. No one has shown any valid reason for restricting any class of foods in the diet of these patients.[28]

In an excellent article entitled "What Should a Patient With Arthritis Eat?" Bauer[2] has shown that there is no one specific diet applicable to

patients with arthritis (except in cases of gout); he employs a diet high in vitamins and calories (unless the patient is overweight), and adequate in calcium, phosphorus and iron.

He has evaluated the following diets in rheumatoid arthritis: (1) low carbohydrate, (2) reduced caloric intake, (3) altering the acid-base balance of the diet, (4) omission of so-called acid fruits and vegetables, (5) allowing only one type of food substance at each meal, (6) omitting foods to which the patient is hypersensitive, and (7) a low protein diet. He has clearly shown that *there is no good evidence to support the use of any of these in patients with rheumatoid arthritis.* At present, most rheumatologists agree with his findings in this matter.

Most rheumatologists employ what appears to be a sensible diet for the patient with rheumatoid arthritis, *i. e.*, one which seems to fit best his bodily needs. Inasmuch as most of these patients are underweight, have a low grade fever and appear chronically ill, we use a **high caloric diet** (except in those few who are obviously overweight), which is **high in vitamin** content, **abundant in calcium and phosphorus** *and* in addition, acts as *an anti-constipation regimen.* Such a diet would contain the following each day:[28]

1. *Milk*—2 or 3 glasses or more per day.
2. *Fruit juice* or tomato juice—2 glasses or more per day.
3. At least several *fresh green vegetables.*
4. *Meat, fish, fowl or eggs* as desired—at least once per day, and preferably twice.
5. Butter, cream and cheese as desired.
6. Bread and cereal as desired, provided that this does not interfere with the consumption of fruits and green vegetables.
7. Starchy (20 per cent) vegetables as desired again provided that this does not interfere with the consumption of fresh fruit and fresh green vegetables.
8. In the markedly underweight patient, *cod liver oil*—an ounce two or three times a day is useful in putting weight on the patient (and in supplying vitamins A and D). If this is not tolerated, concentrated vitamin preparations are available in capsule form.
9. *Vitamin B complex* is often useful in stimulating an appetite. If used in capsule form, a preparation should be ordered which supplies each day at least 3 mg. of thiamin chloride and the same amount of riboflavin, together with small amounts of niacin amide and other available fractions of the B complex.
10. It is often desirable to give *additional nourishing feedings* as fruit juices or milk or milk shakes between meals and at bedtime in the markedly underweight patient.

It is usually advisable to include in the diet an adequate amount of meat (particularly liver) and iron because of the hypochromic anemia which often accompanies severe cases.

If the patient shows evidence of a *spastic or irritable colon*, one must curtail the amount of roughage in his diet; in these instances, small amounts

of Tr. Belladonna (0.3 to 0.6 cc.) may be given three times a day following meals.

SAMPLE DIET:

Breakfast:

One orange or the juice of 1 or 2 oranges (caution: orange juice loses 50 per cent of its content of vitamin C if allowed to stand at room temperature for several hours).

Oatmeal (or other hot or cold cereal) with sugar, milk and cream.

A slice of whole wheat or rye bread (or toast or rolls) with a generous helping of butter.

Eggs and bacon (if possible).

Glass of milk.

Lunch: Similar to dinner.

Dinner:

Broth or tomato juice or fruit cocktail.

Serving of any desired meat, fish or fowl.

Large serving of a 5 or 10 per cent vegetable (see below).

Average serving of a starchy (20 per cent carbohydrate) vegetable.

Additional vegetables as desired.

One slice of whole wheat or rye bread (or toast or rolls) with plenty of butter.

Large glass of milk.

Salad as desired, including mayonnaise.

Dessert: preferably fresh fruit.

Mid-morning, mid-afternoon and bedtime: added fruit juices, milk, or milk shakes and crackers as desired.

Additional:

a. An ounce of *Cod Liver Oil Two or Three Times Per Day* if tolerated. If this is kept in the refrigerator, its unpleasant taste is decreased. This is also lessened in the mint flavored cod liver oil and in Maltine with cod liver oil. If not tolerated, one capsule of halibut liver oil or 5 drops of percomorph oil are given per day.

b. Vitamin B as Brewer's Yeast, whole wheat germ, "Bemax," etc., 1 or 2 tablespoonfuls in the morning cereal; or two or three Brewer's yeast tablets three times a day; vitamin B_1 may be given as thiamin chloride, 5 mg. one or two times a day. It is preferable to supply the whole vitamin B complex: many preparations of this are now available.

c. Cevitamic acid 100 mg. twice a day for the first several weeks of treatment.

d. Insulin may be of value in stimulating the appetite and aiding in the metabolism of carbohydrates in these patients. It may be begun in doses of 5 units of old (regular) insulin given hypodermically from twenty to thirty minutes before breakfast and supper. If required, this may be increased gradually until a total of 30 or 35 units is given daily. In most cases, it will be possible to withdraw the insulin after four to six weeks without ill effects.

e. If the patient drinks an insufficient quantity of milk, administer calcium gluconate, 1 gram (gr. xv) three times a day.

If the individual is of average weight, preference should be given to those foods which supply vitamins and minerals, with the high carbohydrate foods being eaten after the patient has assured himself that he can finish the *meat, milk, fresh green vegetables* and *fresh fruits.* Unfortunately, in the past, many of these individuals "filled up" on sugar, syrups, starchy desserts, pastry, ice cream, noodles, white bread, etc., and then did not have a desire to eat their meats and green vegetables and fresh fruits.

If the diet contains at least a pint of milk per day, green leafy vegetables, fruits, meat or eggs, this will tend to correct any deficiency of vitamins A, riboflavin and C as well as calcium deficiency.

The physician must remember that *deficiency states may result even when the diet is adequate in vitamin content;* in such cases, one must consider defective absorption, impaired utilization or possibly, increased intestinal destruction of these food substances.[21]

Unfortunately, many patients with rheumatoid arthritis avoid those vegetables and fruits which have a sour taste because of their mistaken view that their body is swamped with acids. Ironically, they usually substitute for these foods high carbohydrate foods which are really acid-ash foods. **Fresh vegetables and fruits should be taken in abundance** by the patient with rheumatoid arthritis as a source of minerals and vitamins and also for their high base and high cellulose content.

Protein is obtained mainly from animal foods, including meat, fish, eggs, milk and cheese and vegetable foods such as nuts, whole grain cereals and leguminous seeds.

The preparation *Dietene,* which has been accepted by the A.M.A. Council on Foods, contains a low fiber content (0.2 per cent), low fat (0.18 per cent) and a *high protein content* (32.5 per cent). It may be of value in hypoproteinemia by presenting to the patient a readily assimilated form of protein, with added vitamin and mineral content. It is also useful when tube feedings are indicated. Three ounces of Dietene (the suggested daily dosage) contains 27.6 grams of protein, 1 gram of calcium, 0.8 gram of phosphorus, 12.75 mg. of iron and the following vitamins: A—5000 U.S.P. units; thiamin chloride—1.9 mg.; riboflavin—2.8 mg.; C—63.7 mg.; D—500 U.S.P. units; nicotinic acid—19.5 mg. The preparation contains skim milk solids, with added sucrose, malt, saccharated iron oxide and added vitamin preparations. It may be of value in undernourished, markedly underweight individuals in supplying a high vitamin, high protein, high mineral complement to the diet.

One should make every attempt to increase the vitality and resistance of the patient by improvement in his nutritional state. If the patient is markedly undernourished, a high carbohydrate diet is often essential. In some of these patients, small doses of insulin may be employed several times a day to stimulate the appetite.[6,16]

The average diet is usually deficient in *calcium*. For this reason, the arthritic patient should drink at least a pint of milk per day as the calcium of milk is readily utilized. Other sources of calcium include cheese, eggs and fresh vegetables and fruits.

Most patients and a few physicians have no knowledge of the caloric value of foods. Some believe that one of the most nourishing foods is spinach and do not realize that it requires approximately 6 servings of spinach to equal, in caloric value, 1 medium sized potato. Because of popular misconceptions as to diet, it is advisable to furnish each patient with a sheet containing the approximate food values of many common foods. Such tables can be printed or mimeographed at very low cost and will obviate the possibility of misinterpretations in diet. On such a diet the following information may be placed:

Vegetables are classified according to their carbohydrate content. This ranges approximately from 5 per cent to 20 per cent. Five per cent carbohydrate foods contain many less calories than an equivalent amount of 20 per cent ones. In attempting to gain weight, large portions of the 20 per cent vegetables should be consumed. However, the diet should include an adequate amount of 5 per cent vegetables which contain large amounts of certain protective vitamins.

5 Per Cent Vegetables:

Fresh asparagus, celery, cucumbers, lettuce, mushrooms, cooked rhubarb, sauerkraut, spinach, Brussels sprouts, cabbage, cauliflower, egg plant, green peppers, radishes, tomatoes, watercress. An average serving of any of these furnishes approximately *16 calories.*

10 Per Cent Vegetables:

Beets, canned green peas, carrots, onions, squash, pumpkin, fresh string beans, turnips. An average serving of any of these contains approximately *28 calories.*

High Carbohydrate Vegetables (averaging 20 per cent):

Potatoes (white or sweet), corn, fresh green peas, lima beans, baked beans, artichokes, parsnips (and most cereals, including macaroni, spaghetti, noodles and rice). An average serving of any of these is approximately 100 calories. For example, a medium sized ear of corn furnishes as much caloric value as any of the following: 6 tomatoes, 3 cupfuls of spinach, 6 cups of cabbage, 12 cups of watercress, 60 large leaves of lettuce, 25 long stalks of asparagus or 30 radishes.

Fruits Containing 10 Per Cent Carbohydrate or Less:

Orange, grapefruit, cantaloupe, peach, pineapple, strawberry, cranberry, watermelon, olive. An average serving of any of these contains approximately 45 calories (such as a small orange, one-half of a small grapefruit 4 inches in diameter, 1 medium sized peach, 1 slice of pineapple three-quarters of an inch thick, three-fourths of a cup of strawberries, etc.).

High Carbohydrate Fruits (15 to 20 per cent):

Apples, apricots, bananas, cherries, grapes, grape juice, pears, plums. The caloric value of these foods is as follows: an average sized apple, 80 calories; medium sized apricot, 20; average sized banana, 95; medium sized cherry, 2 calories; medium sized grape, 3–4 calories; small glass of grape juice, 120; average sized pear, 100; plum, 30 calories. In attempting to gain weight, generous portions of the higher carbohydrate fruits should be consumed, in addition to sufficient citrous fruits to maintain a normal vitamin C intake. In regard to dried fruits, the caloric values are: 1 date, 30 calories; fig, 60; prune, 35; large raisin, 8 calories. Most individuals do not realize that prunes should be included in a high caloric diet. The average serving of prunes (6) contains the caloric equivalent of approximately 5 small oranges.

Other values which may be included in the list given to the patient are:

Cooked oatmeal	½ cup	79 calories
Cooked rice	½ cup	70
Cornflakes	1 cup	57
Shredded wheat	1 biscuit	110
One saltine—2″ square		17
Graham cracker		40
One egg		75
One large glass of milk (8 ounces)		160
One large glass buttermilk (8 ounces)		85

It Should be Emphasized That a Glass of Buttermilk Contains Approximately Only Half the Caloric Value of a Glass of Whole Milk.—It is a popular misconception that buttermilk is milk with added butter and that it is more nourishing than plain milk.

One large glass (8 ounces) of skimmed milk (cream removed)	85 calories
American cheese—1″ cube	90
Cottage cheese—1 rounded tablespoonful	30
(Cottage cheese is relatively much lower in caloric content than is American or Swiss cheese)	
Swiss cheese slice—4″ x 4″ x ⅛″	90
Cooked lean beef, lamb or mutton (1 slice 4½″ x 2½″ x ¼″)	120
Smoked ham—similar slice	175
(The high caloric value of ham, pork and bacon is due to its large content of fat)	
Liver or veal—slice 2″ x 3″ x ½″	130
Lean fish (cod, flounder)—slice 4″ x 2½″ x ¾″	90
Fatty fish (salmon, mackerel, shad), similar slice	170
Oyster—medium size	16
Lean chicken—slice 2″ x 2″ x ¼″	50

Among the foods which are *high in fat content are:*

Butter—square 1″ x 1″ x ⅜″	75 calories
40 per cent cream, 1 tablespoonful	75
20 per cent cream, 1 tablespoonful	40
(The cream usually served to the home is 20 per cent cream)	
Green olives, each	12
Ripe olives, each	8
Peanut butter, level tablespoonful	90
Salad oil, tablespoonful	135
Large almond	10
One peanut	4 or 5
Pecan—1 large	20
Cocoa (tablespoonful) (powder)	50
Avocado (alligator pear)	210
Cod liver oil—1 tablespoonful	99

Desserts:

Apple pie—average portion ($\frac{1}{6}$ medium sized pie)	400 calories
Cocoanut custard pie—average portion	450
Custard pie—average portion	260
Mince pie—average portion	460
Pumpkin pie—average portion	350
Strawberry short cake—average serving with 2 tablespoonfuls whipped cream	350
Medium sized chocolate cup cake with chocolate icing	265
Pound cake—slice $2\frac{3}{4}''$ x $3''$ x $\frac{5}{8}''$	125
Cruller—$2\frac{1}{2}''$ diameter	150
Large macaroon	110
Vanilla ice cream—$\frac{1}{8}$ quart	250
Strawberry or peach ice cream—$\frac{1}{8}$ quart	210
Chocolate ice cream—$\frac{1}{8}$ quart	285
Orange ice—$\frac{1}{8}$ quart	150
Hershey chocolate bar (5c plain)	260

Beverages:[2]

Cup of cocoa (with 5 ounces of milk)	175 calories
Coca Cola—6 ounce bottle	85
Ginger ale—6 ounce bottle	65
Carbonated beverages—6 ounce bottle	65
Sweet cider—8 ounces	120
American ale—8 ounce glass	100
Average beer—8 ounce glass	115
Champagne—3 ounces	85
Port wine—1 ounce	50
Sherry wine—1 ounce	40
Gin, rum or whiskey—1 ounce	75

Breads:

White bread—1 slice, average	65 calories
White bread—1 slice, thin	52
Whole wheat bread—1 slice, average	74
Graham bread—1 slice, average	65
Rye bread, light—1 slice, average	76
Rye bread, dark—1 slice, average	95
Cracked wheat bread—1 slice, average	66
French (or Vienna) bread—1 slice $3\frac{3}{4}''$ x $2\frac{1}{4}''$ x $1''$	53
Bran bread—1 slice, average	76
Hollywood Health Bread—1 slice	45
Melba toast—1 slice $3\frac{1}{2}''$ x $1\frac{1}{2}''$ x $\frac{1}{4}''$	20
Zwieback—1 piece	34
Rye Krisp—1 piece $1\frac{7}{8}''$ x $3\frac{5}{8}''$	20

Sandwiches (with 2 pieces of white bread):

Ham	250 calories
Chicken salad and lettuce	230
Cream cheese and jelly	370
Bacon, lettuce and tomato	340

Sandwiches will vary with amount of butter and filling; values are only approximate.

For anyone desiring an excellent short résumé of the caloric, vitamin and mineral content of commonly used portions of foods, I can highly recommend the small manual of Bowes and Church.[5]

Jacobson and his associates were able to *decrease the joint fluid* in a case of rheumatoid arthritis *by the use of a low sodium diet* and the *ingestion of ammonium chloride*, and to increase the joint fluid by feeding sodium bicarbonate.

Pelner has reported on the effectiveness of a low sodium, neutral or acid ash diet, together with ingestion of 6 grams of ammonium chloride per day in the removal of effusion in traumatic hydrarthrosis; these patients received no sodium chloride at the table, and no salt was used in cooking their foods.[22]

Rheumatologists vary widely in their choice of diets for patients with rheumatoid arthritis. Although certain individuals have advised the use of a low starch diet in the treatment of this disease most rheumatologists have not found that patients improve more on such a diet than on a high starch diet.[14]

Neuwirth believed that decreasing the carbohydrates in the diet was desirable in patients with rheumatoid arthritis because an abundance of carbohydrate increases the requirements of the body for thiamine chloride and limits the sources of proteins, vitamins and minerals;[21] some workers have claimed that a high carbohydrate diet produces water retention in the body.[1,26]

In some patients with rheumatoid arthritis, joint pain and swelling may be definitely lessened by a diminished intake of starchy vegetables and other high carbohydrate foods, with the patient receiving an adequate amount of meat, eggs, dairy products and 5 or 10 per cent vegetables and fruits.[25]

Holbrook has summarized the literature on the **raw food diet** as a therapeutic agent.[15] Bircher-Benner used the so-called Rohkost Diet in 1895.[4] This diet was composed predominantly or exclusively of uncooked edible plant life such as fruits, eggs, cheese, white flour, alcohol, etc.; in 1897, he opened his Zurich clinic for the treatment of the sick with food and reported a far-reaching, non-specific therapeutic influence in various human disorders. This diet was based on the thought that the heat of cooking may spoil the biologic powers of vegetables; Bircher-Benner questioned the nutritional value of meat. The current interest in raw foods is a result largely of his enterprise; this is manifest today by health cocktails made of the juices of various vegetables; vitamin bars; and numerous vegetables and fruit-juice presses, shredders and extractors.

Of the 89 articles on raw foods in the Index Medicus over a period of thirteen years recently, only four were in the English language. Holbrook could find no pertinent discussions of the raw food diet in recent textbooks of nutrition or medicine.

Hans Eppinger, Professor of Medicine at the University of Vienna, believed that the raw food diet would be of therapeutic value in some disorders because it was *poor in sodium* and *rich in potassium*.[8] He believed that such a diet *would increase the excretion of water* and sodium chloride in such conditions as cardiac decompensation, nephrosis, rheumatoid arthritis and other disorders.

The raw food diet should not be continued more than five or six weeks as it is poor in protein, calcium and sodium, although it is rich in potassium, carbohydrate and some vitamins.

Other diets which have been advocated in the past include the "salt

free diet" which probably originated with Van Noordan in 1902 as used in renal disease; the Gerson diet; and the "*vegetarian diet.*" The latter varies with the individual; the eating of meat is forbidden in this diet (because meat must be obtained by killing). The vegetarian diet differs from the raw food diet in that the former permits considerable cooking.

The "*salt free diet*" was originally used to produce diuresis. Volhard insisted that this diet contain no more than 1 gram of sodium chloride per day.

The *Gerson diet,* or the *S.H.G.* (Sauerbruch-Herrmannsdorfer-Gerson) diet originated when Sauerbruch, a surgeon interested mainly in tuberculosis, persuaded Herrmannsdorfer to study the effects of diets on wound healing and wound infection. The latter visited Gerson and brought him to Munich so they could continue their studies.[10] This diet emphasizes the use of raw fruits and vegetables, eliminates salt and salty foods, but permits cooking and the use of up to 1500 cc. of milk per day and 500 grams of meats per week. It is claimed that this diet supplies generous quantities of all minerals except sodium.

The "*raw food diet*" is *primarily a high potassium, low sodium diet.* It has been estimated that the average American consumes and excretes about 10 grams of sodium chloride daily. Healthy individuals can ingest up to 40 grams of sodium chloride daily without the appearance of edema. About 2 or 3 grams of potassium is consumed daily by the average adult. The potassium ion has been considered to have a diuretic action.

Holbrook has outlined his *concept of a raw food diet.* This includes:

Breakfast: raw 10 per cent fruit; cooked egg; a thin slice of toasted, salt free bread; a square of butter; clear tea with lemon; sugar; marmalade or honey.

Lunch: 1½ cups of 5 or 10 per cent vegetables; an average serving of raw fruit (from 5 to 15 per cent); salt free butter; teaspoonful of sugar; clear tea with lemon and salt free mayonnaise.

4 P. M.: serving of 10 per cent raw fruit; thin slice of salt free bread with a square of salt free butter; clear tea with lemon and sugar and marmalade or honey.

Supper: cooked egg or cheese; serving of 10 per cent raw fruit; nuts; clear tea with lemon and sugar; salt free butter and 2 teaspoonfuls of jelly or fruit preserves.

It must be emphasized again that *such a diet is deficient in calories and protein for most adults,* and *is lacking in vitamin D;* although the values for vitamins A and C are high, the amount of thiamin obtained is only about half normal. The diet is high in potassium and low in sodium. *Such a raw food diet must not be used for long periods,* even in healthy individuals, because of the food deficiencies mentioned. It has been suggested that this type of diet may be of some value in at least temporarily decreasing the swelling seen in some individuals with rheumatoid arthritis, if continued for several weeks only.

Without discussing the type of arthritic involvement, Turnbull has recently reported relief in most arthritic patients of all or the greater part of their symptoms by a diet which excluded foods to which patient gave positive reactions by cutaneous sensitization tests.[31–33] Other workers have not been able to confirm these results. Vaughan found that 20 per cent of 1000 consecutive adults with allergic disorders (asthma, hay fever, urticaria, angioneurotic edema, etc.) complained or had complained of rheumatic pains. Of these 200 patients, 90 showed some evidence of articular pathologic changes. Only 10 per cent of these 90 patients had rheumatoid arthritis. Sixty-nine per cent of the 200 patients with rheumatic pains showed evidence of food allergy.[34]

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CHAPTER XX

FOCI OF INFECTION IN RHEUMATOID ARTHRITIS

THE hypothesis of focal infection in rheumatic disease was born in 1801 when Benjamin Rush removed a painful tooth from a woman with a rheumatic hip, resulting in a disappearance of the pain and complete clinical recovery from the disorder.[56] The idea lay practically dormant for over one hundred years until Billings (1912) again brought it to the fore. Despite numerous reports since that time, *we do not yet know the rôle of focal infection in most cases of rheumatoid arthritis.*

In a small percentage of patients, rheumatoid arthritis begins atypically with a rather sudden onset and is often monarticular in these cases. In these particular individuals, the removal of a focus of infection may be followed by a disappearance of the arthritis. These patients have been classified by some as having "focal infection arthritis" or "infective arthritis, etiology unknown."[11] Such "focal infection arthritis" cases are very uncommon. It is in these patients that the cleaning up of purulent sinuses, or a tonsillectomy (following a peritonsillar abscess) or removing a tooth which is the seat of a periapical abscess—may be followed by dramatic improvement in the arthritis.

The presence of a focal infection alone does not necessarily lead to arthritis. Among 239 psychiatric patients, Brown found infected foci in the nose and throat in 44 per cent, in the teeth in 37 per cent and in the urinary tract in 17 per cent—yet none of these individuals had arthritis.[13]

At present, a conservative attitude should be adopted concerning focal infection in rheumatoid arthritis. The importance of focal infection in the production of rheumatoid arthritis is still unknown.

Removal of focal infection in the patient with *advanced rheumatoid arthritis rarely results in a beneficial effect upon the arthritis per se,* although it may improve the patient's general health and lessen the load of infection which he is carrying. The practitioner must realize that *a tonsillectomy or removal of an infected tooth will not straighten out deformed joints,* which have resulted from contractures, adhesions and ankylosis. However, in our present state of knowledge, *it seems unwise to allow any definite focal infection to remain in the patient's body if this can be eradicated without detriment to the patient.* Removal of focal infection must be undertaken with care in elderly patients; likewise, important teeth must not be sacrificed in debilitated patients at the risk of interfering with their nutrition unless these teeth are definitely infected and the infection can be eliminated in no other way.

In those individuals whose arthritis begins shortly after a tonsillitis, peritonsillar abscess, sinusitis or discovery of a periapical abscess, it is sound clinical practice to **treat or remove the infected area as soon as is possible.**

Present evidence suggests that the appendix, gall bladder, female genito-urinary tract and constipated bowel are not important as foci of infection

in patients with rheumatoid arthritis; if these areas are definitely diseased, they should be treated according to the principles which one would use had the patient no rheumatoid arthritis.

Short and Bauer have stated recently that expensive combing of the patient for sources of infection is seldom warranted; they have stressed the importance of having the physician in charge of the patient carefully interpret and correlate such reports before acting on isolated areas.[99]

Although Hippocrates has been credited as having relieved two patients with rheumatism by dental extractions, Hench has not been able to verify this by careful search in the available translations.[55]

Focal Infection in Rheumatoid Arthritis

1. *Removal of infected foci seldom produces marked improvement in rheumatoid arthritis, except when done at a very early stage.*
2. One should not expect that removal of focal infection will heal extensive and irreparable articular damage.
3. *Removal of focal infection* is not unwise in most cases of rheumatoid arthritis, but it *will not accomplish the impossible;* it is justified in most instances in lessening the burden of infection which the patient is carrying.
4. It is possible that infected joints themselves may act as foci.
5. A conservative attitude should be adopted at present concerning the removal of focal infection in rheumatoid arthritis:
   - (*a*) Remove (or treat) obviously infected foci (especially teeth, tonsils, sinuses, gingivæ and prostate), in accordance with good clinical judgment of the patient's general condition, his reaction to anesthesia, etc.
   - (*b*) Do not expect dramatic improvement of the arthritis following such removal of focal infection.
   - (*c*) *Never promise the patient or his family that removal of focal infection will greatly improve his arthritis.*
   - (*d*) Remove definitely infected foci as early in the course of rheumatoid arthritis as possible.
   - (*e*) Best results from removal of focal infection are noted in those (few) patients whose rheumatoid arthritis has begun rather acutely.

One must never rely upon removal of focal infection as the sole therapy in rheumatoid arthritis. Certain individuals have recently expressed grave doubts concerning the importance of focal infection.[21,86] Some of these persons have gone so far as to say that focal infection should not be removed and that its whole concept is false. Unfortunately, there is no practical way to evaluate the damage that a suspected focus may cause.

Level headed general practitioners realize that collections of pus in the maxillary sinuses must be drained, that proved abscesses about the roots of teeth as well as periodontitis (pyorrhea alveolaris) must be attended to, that a chronically infected cervix with marked leukorrhea should receive proper care, that chronic prostatic infection needs medical attention, and that definitely infected tonsils which are the seat of repeated attacks of sore throats, tonsillitis or peritonsillar abscess should be removed.

Definite focal infection in the body may be harmful to the patient regardless of his disease.[5] By the time some of the recent writings decrying focal infection reach some practitioners, these statements are so distorted or are presented in such a manner that he is led to believe that: no tonsils or teeth should ever be removed, no prostate gland requires massage, purulent infections in paranasal sinuses will clear up without treatment and many other falsifications. *The present wave of extreme antagonism to the concept of focal infection (presented by some internists) may cause immeasurable harm.*

From clinical experience, I believe that some cases of arthritis, nephritis, iritis, endocarditis and other diseases are definitely begun by infection originating in sites which we at present term foci, probably better labeled *portals of entry of infection* (tonsils, paranasal sinuses, infections of the gums and periapical regions, prostate and other organs).[9,10,60] Clinicians with wide hospital ward experience have seen instances of bouts of acute nephritis, arthritis or iritis follow shortly after the onset of an acute tonsillitis or an acute paranasal sinusitis or other infection.[63,88–94] The frequency of such occurrences is greater than would be accounted for by a chance coincidence of the two diseases.

How the infection in tonsils, paranasal sinuses and other areas leads to infection in joints, eyes, kidneys or other organs is a matter of speculation. This is possible in several ways: (1) the bacteria themselves could be transported by way of the blood stream to other organs; (2) bacterial toxins could be carried from foci to areas of lowered resistance; or (3) the protein fraction of the bacteria could act as an antigen and sensitize tissues such as synovial membrane, so that further additional bacteria or protein coming into contact with the synovial tissue could produce an allergic reaction. There is laboratory evidence being accumulated at the present time to suggest that this last mechanism, a sensitization phenomenon, may be much more important than has been formerly thought. Several carefully controlled experiments may illustrate this.

When rabbits were injected intravenously with heat killed streptococci, and bacteria were injected intravenously into these rabbits at a later date, it required a smaller number of organisms to produce arthritis in the previously injected rabbits than in normal animals. In another set of experiments, small amounts of heat killed streptococci or nucleoprotein fraction were injected into the right knee joint of a number of rabbits. When these animals were later injected intravenously with live cultures of the same bacteria, the previously injected joints were much more susceptible to infection than other joints injected with heat killed staphylococci or horse serum.[20]

Organisms can and do pass from so-called foci of infection into the blood stream. It has been definitely shown that the mere act of chewing on hard candy or massage of the gingivæ in individuals with periodontitis (pyorrhea alveolaris) is followed by the finding of the mouth organisms in the blood stream.

To sum up the preceding paragraphs, infected tonsils, paranasal sinuses, gums, etc., contain virulent pathogenic bacteria; these bacteria or their products do at times gain access to the blood stream. By some mechanism, possibly associated with sensitization of distant tissues, foci of infection may at times be responsible for the onset of some cases of infectious arthritis, nephritis, iritis and other diseases.

General Considerations

1. Remove or treat all *definitely infected foci* as early in the course of the arthritis as possible (unless the patient is markedly debilitated, or runs an acute course with high fever).[26,83]
2. In extremely emaciated patients, attempt to build up the general health before performing any operative procedures; if improvement in nutrition does not occur within a reasonable amount of time (two to four weeks), proceed cautiously to attend to foci of infection.
3. During acute exacerbations of the disease, with high fever and marked joint symptoms, treat the patient conservatively for at least several (two to four) weeks; if the patient fails to improve or becomes definitely worse, cautiously attempt to eradicate definitely infected areas.
4. If operative procedures are required in an effort to eliminate focal infection, give sulfamerazine, 1 gm. (in adults), with an equal amount of sodium bicarbonate or potassium citrate every eight hours for twenty-four hours preceding operation and a similar amount every eight hours for twenty-four hours following the operative procedure; since instituting this program, I have not seen any instances of postoperative joint flare-ups. One must be certain that the patient has never previously been known to be sensitive to sulfonamides.
5. **Tell the patient that the removal of the infected focus probably will not cure the arthritis**; however, this may improve his general health and lessen the load of infection which he is carrying.[34,59]
6. In *atypical rheumatoid arthritis*, where the joint phenomena have developed within a short period of time, results following removal of infected foci may be very gratifying; if the onset of joint symptoms follows shortly after an acute flare-up in an infected focus, removal of this focus may produce dramatic results. Unfortunately, this type of case is the exception and not the rule.
7. In order of probable importance, the foci in arthritis include the teeth and gums, tonsils and infected lymphoid pharyngeal tissue, the paranasal sinuses the prostate and of much less significance, infection in the gall bladder, pelvis of the kidney, cervix and tubes, etc.
8. In many instances, infected foci often pass undiagnosed or are incompletely eradicated.[5]

Focal infections probably play a rôle in keeping an arthritis active but they must not be considered to be the sole etiologic factor; the treatment of rheumatoid arthritis should include the eradication of any definitely infected focus but should not end here.[6] *The removal of focal infection, per se, will not result in cure or even improvement of the joint pathology in many cases of infectious arthritis.* Once the arthritic process has been begun, inflamed synovial tissue itself may possibly act as a secondary focus from which infection may be further disseminated. One must not expect miracles; long-standing contractures and deformities will not vanish overnight

after the removal of infected tonsils or the extraction of teeth with extensive periapical abscesses.[82] Such patients require adequate orthopedic and physical therapy, in addition to general care in an effort to even slowly improve their status. Furthermore, only a small percentage of individuals with definite infection in teeth, sinuses, tonsils or other areas develop arthritis.

Indiscriminate removal of tonsils, teeth, etc., may cause further damage to already infected joints. At least a temporary blood stream infection frequently follows the removal of infected teeth and other organs. It is possible that if many infected teeth were removed at one time in a debilitated person, the accompanying blood stream infection might produce considerably more damage in the already inflamed synovia.

What attitude should then be taken concerning definitely infected foci? Our working hypothesis is as follows: definitely infected tonsils, gums, sinuses, etc., are no more in keeping with good health than a furuncle or carbuncle. Even in otherwise normal subjects (without arthritis), adequate attention should be given to these infections. We are all willing to have a competent dentist treat areas of periodontitis (pyorrhea alveolaris) in our mouths or an otolaryngologist perform radical antral surgery if conservative measures fail to improve a chronic, purulent maxillary sinus infection. **We advise the removal of, or proper attention to every area of definite infection which can be found in the arthritic body in the same general manner as this would be attended to even if the patient did not have an arthritis.** These must be *definitely infected areas* and not questionable sites of infection. *Do not promise the arthritic patient that his arthritis will be improved following such procedures*, but one should say that these areas will be eradicated as a general health measure in order to lighten the load of infection which his body is bearing, in the same manner as would be done in our own bodies even without the presence of an arthritis.

In order that the removal of such infection may be attended by a minimal amount of further damage to joints, we have used the general principles enumerated on page 359:

Marked improvement does not often follow mere removal of focal infection for various reasons:

1. There may be additional foci present in the body.
2. The disease may be of such long standing that the foci are no longer actively concerned in the further course of the arthritis.
3. The foci may be unrelated to the arthritis[67] and merely an accompanying disease—another manifestation of the lowered general resistance of the patient permitting infections to develop in joints, teeth, sinuses, etc.
4. Removal of foci may result in a transient bacteremia and further joint damage.

Unfortunately, some physicians feel that removal of focal infection is the only treatment required in rheumatoid arthritis. These persons find some justification for the procedure regardless of the result: (1) if the arthritis is improved following the removal, this "proves" that the foci were at fault; (2) if the arthritis is made worse following their removal, this likewise "shows" a definite connection between the two; while (3) if no change occurs following removal, this is said to be fine, *i .e.*, the patient has no reaction and joint improvement will follow slowly. **It cannot be overemphasized that the patient must be told that mere removal of an infected focus will not cure his arthritis.** The patient's morale will not then be depleted if joint phenomena do not improve following the operation. One may tell the patient that he has definitely infected foci (if such are present) which *may* be related in a causal manner to his arthritis. The presence of definitely infected foci even in individuals without arthritis requires treatment as a general health measure.

One should obtain the assistance (whenever possible) of experienced consultants in special fields (otolaryngology, dentistry, gynecology and urology). It is advisable to speak in person with the consultant after he has examined the patient rather than to accept merely a typewritten report.

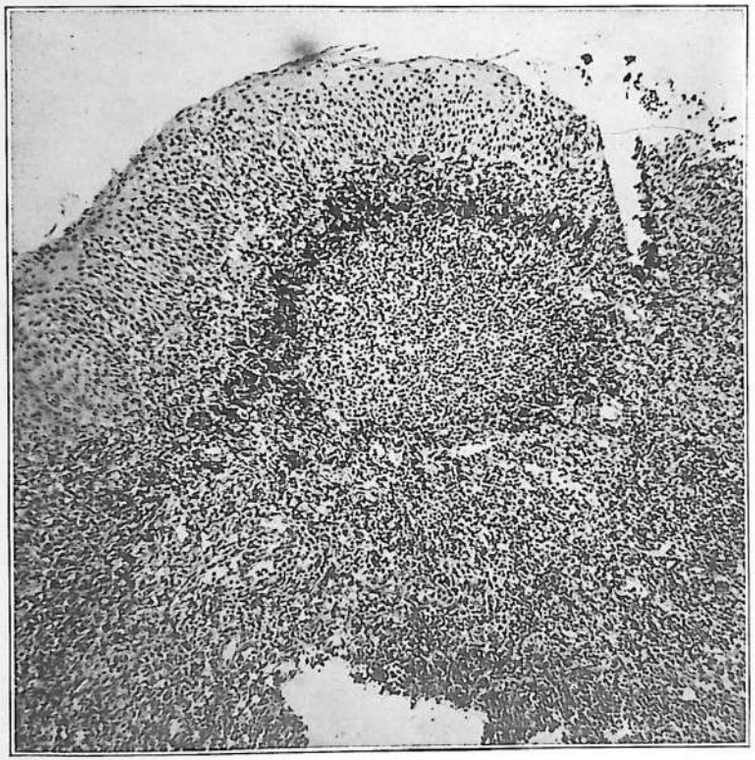

FIG. 101.—Pharyngeal tissue from a child with choreiform movements. × 72. (Schenck, courtesy of Arch. Otolaryngol.)

INFECTED TONSILS

Some authorities consider infected tonsils to be the commonest cause of rheumatoid arthritis.[96] Definitely infected tonsils should be removed early in patients with this disease not because they are the cause of the

disease, but as a general health measure.[11] *However, routine tonsillectomy is not indicated in any form of arthritis* because it does not cause improvement in any convincing number of cases.[11] Infections of the nose and throat in rheumatoid arthritis must not be ignored and the physician must not neglect such infection.

It should be emphasized that *there is a group of patients with a chronic form of rheumatic fever which resembles rheumatoid arthritis.* In these individuals, the joints may have the typical appearance of rheumatoid arthritis, with symmetrical fusiform swellings, etc.; the blood sera of these cases do not agglutinate hemolytic streptococci but do uniformly exhibit a high antistreptolysin titer. Almost all of these individuals give a history of frequent sore throats or sinus infections. It is extremely important that diseased tonsils be removed and sinusitis be carefully treated in this particular group.[11]

When an arthritis follows closely after a tonsillitis or peritonsillar abscess, it is extremely important to eradicate the tonsillar infection as rapidly and completely as possible. If the joint phenomena begin abruptly, one can usually predict a much better prognosis for the arthritis.

It has been found that after the first year of life there are no tonsils in which pathologic changes cannot be found.[109] Infected tonsils have been seen in the majority of cases of rheumatoid arthritis.[12] Tonsillar infection should be suspected in the presence of any of the following:

Suspect Tonsillar Sepsis if:

There is enlargement of the tonsillar lymph nodes under the angles of the jaw.

There is a history of repeated attacks of sore throats or tonsillitis; however, some of the best clinical results have followed removal of tonsils which were not obviously diseased and which had never been acutely infected.

An attack of arthritis follows shortly after a sore throat.

There is congestion of the mucous membrane of the anterior pillars and soft palate or ecchymosis at the base of the uvula.

A paste-like or milky secretion exudes from the tonsillar crypts on pressure.

Marked difference exists in the appearance of the two tonsils.

Embedded tonsillar stumps are found.

Tonsillar stumps have been considered to be one of the most vicious of all types of tonsillar infection in producing distant symptoms. Otolaryngologists agree that *tonsils cleanly removed in patients over the age of six seldom recur.* If tonsillar stumps appear infected, they should be removed.

Clean surgical removal of the infected tonsils is the most certain means of eradication of this focus. Antiseptics locally applied to the tonsils do not cure chronic tonsillar sepsis. Although roentgen therapy may shrink the tonsil, it does not eliminate its contained infection; positive bacterial cultures can be obtained immediately following such treatment. Electrical coagulation is not considered an efficient method of removing every vestige

of tonsillar tissue as small pieces may be buried under the mucosa which has grown over them; abscesses may later form in such bits of tissue.

Except in cases of extreme infirmity, *age alone is not a contraindication to tonsillectomy and patients over fifty years of age are often less upset by the procedure than are younger people.* In one series of 1800 tonsillectomies performed at the Hospital for Ruptured and Crippled in New York City, not a single death occurred from any cause although the patients ranged from one to seventy-eight years.[12]

FIG. 102.—Normal pharyngeal tissue from a child. × 72. (Schenck, courtesy of Arch. Otolaryngol.)

Schenck has demonstrated the presence of infection in patches of lymphoid tissue in the pharynx.[97] He has advocated their removal in so far as this is possible by electrocoagulation. There are numerous lymphoid patches in the gastrointestinal tract (ileum, appendix, cecum?) which may become infected and which of course are not amenable to therapy. Roentgen therapy is sometimes used in the treatment of pharyngeal lymphoid hyperplasia.

Some workers feel that sinus infection may act as a focus only because of absorption of toxic drainage material by the rich vascular supply in the pharynx, and that very little toxin is absorbed through the relatively poorly vascular maxillary sinuses themselves.

It has been suggested that at least a transient blood stream infection

follows tonsillectomy in about 25 per cent of the cases. In one series of 22 patients in whom tonsillectomy was performed, 4 showed growth in the postoperative blood culture and 3 in the preoperative cultures.[106] In no

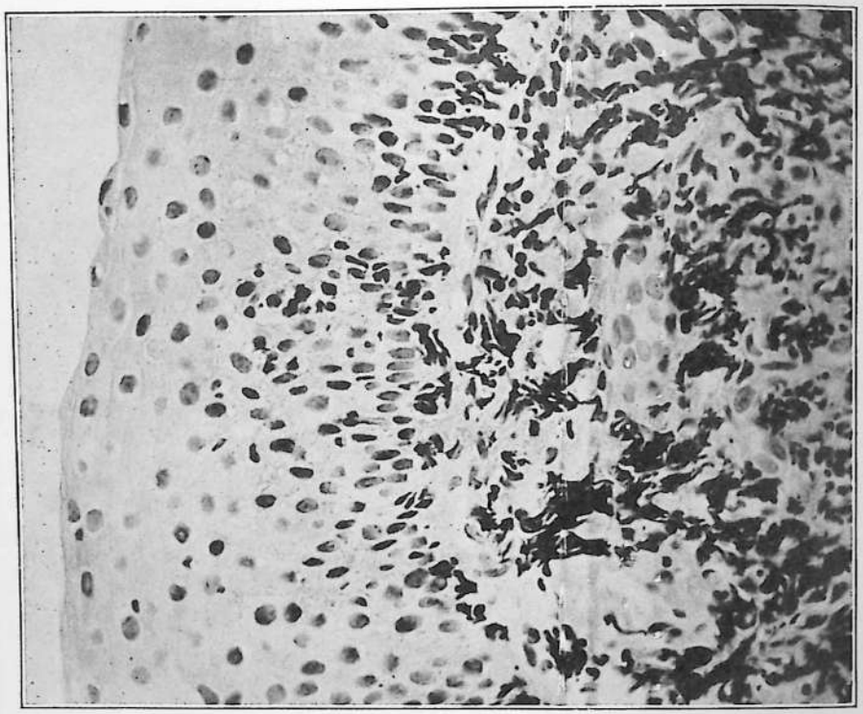

FIG. 103.—Pharyngeal tissue from a child with articular pains. × 306. (Schenck, courtesy of Arch. Otolaryngol.)

case was there growth both preoperatively and postoperatively in the same patient. No constant relation could be shown between the clinical course and the presence or absence of a positive blood culture.

PARANASAL SINUSES

Boots and McCollom found that *rheumatoid arthritis was ushered in by an upper respiratory infection in 20 per cent of their 180 cases.* Seventeen per cent of these cases showed purulent sinus infections; in the latter group, improvement usually occurred following adequate treatment of the sinus infection. It was also observed that any intervening upper respiratory infection greatly increased the activity of the rheumatoid arthritis.

The sinuses of every arthritic patient should be examined, both clinically and roentgenologically, as sinus involvement may give few or no symptoms. Infection in the paranasal sinuses may initiate or aggravate some cases of rheumatoid arthritis.[72,104,115] Attacks of arthritis may begin shortly following an acute sinusitis; in a small percentage of individuals, marked

improvement in the arthritic process may follow eradication of sinus infection. If the latter can be cleared by conservative methods, this should be done; if, in spite of a reasonable period of conservative treatment, usually from one to two months, pus persists in considerable amounts in the maxillary sinuses, one should resort to puncture and drainage or radical sinus surgery. Some investigators believe that sinus infection has systemic significance only because absorption of sinus drainage occurs through the mucosa of the pharynx or alimentary tract.

In many patients the diagnosis of sinusitis is readily made from the history of a purulent nasal discharge, especially when this is unilateral, and from the finding of a localized stream of pus in the nares; some latent cases with scanty discharge are difficult to recognize. Transillumination of the frontal and maxillary sinuses is of some aid; a marked relative opacity of one antrum is especially significant, but this may be the result of an old and cured inflammatory process, mucocele or neoplasm.[7] On the other hand, *pus may be encountered in an antrum which appears clear on transillumination.* The frontal sinuses are often asymmetrical and transillumination is then of less assistance; cloudiness of a frontal sinus may be due to disease, or to small cavities with thick walls as a result of infection in infancy or childhood. Roentgen examinations are helpful, particularly if a fluid level is seen. Thickening of the mucosal lining of the sinuses may be the result of acute infection, cystic degeneration, neoplasm, allergy or previous inflammation. Roentgenograms of the sinuses should be taken by experienced technicians and read by experts in this field.

Snyder and his co-workers have been convinced of a close relationship between chronic arthritis and sinus disease; they have reported that rhinologic treatment of the diseased sinuses has often materially aided in the clinical improvement of chronic arthritis.[103] They did not observe any case in which competent and careful rhinologic treatment caused any ill effect upon the arthritic picture. By means of roentgen examinations, they were able to disclose many cases of sinus disease which otherwise might have escaped detection. In practically 25 per cent of 386 cases of chronic arthritis seen in office practice they demonstrated roentgenologically and clinically proven sinusitis, most often of an asymptomatic or "silent" type. In these individuals with sinus infection, the arthritis "showed with few exceptions a most favorable response to the rhinologic treatment of the diseased sinuses."

Hurd found that the ethmoids were always involved in these cases of sinus infection, most often in association with the maxillary antra, but occasionally with involvement of the frontal or sphenoid sinuses.[61] In some of these individuals, all of the paranasal sinuses were involved simultaneously. The ethmoid infection in these cases was usually of the hyperplastic type with thickening of the mucosa and sclerosis of the bony walls; only a small amount of mucopus was present. In his series, more than two-thirds of the individuals had previously had tonsillectomies without benefiting the arthritis, while the sinus infection was probably overlooked. *The majority of these patients had no subjective symptoms of sinus disease*

and were not aware of their sinusitis. In only about one-third of the cases was there a purulent nasal discharge, often called postnasal drip. Sinus headaches were uncommon.

Hurd emphasized the importance of careful and complete examination, including the condition of the mucosa, the presence of secretion, transillumination of the antra and frontal sinuses after shrinkage of the nasal mucosa, and the character of the mucosa and secretion about their orifices as seen by a nasopharyngoscope. The appearance of the middle turbinates and the mucosa of the middle meatus gave considerable information concerning infection in the ethmoid, frontal or maxillary sinuses. In involvement of the ethmoid or frontal sinuses, he did not believe it possible to find a clear, smooth, glistening or normal colored mucous membrane of the middle turbinate; inasmuch as this turbinate is a part of the ethmoidal labyrinth, it assumes the same mucosal change as the ethmoidal cells. One of the first changes observed in involvement of the ethmoid sinuses was a change in the character of the mucosa of the middle turbinates, particularly along their lower border and at the anterior and posterior ends.

Careful repeated intranasal observations form the only reliable method for diagnosis of ethmoid activity.[77] An ethmoid region which is repeatedly swollen and congested over a period of time with a tendency to stuffiness on that side of the nose (of which the patient is usually not aware) is usually infected; free pus is found here only rarely. Transillumination is of no value for the ethmoid and sphenoid sinuses because of their inaccessibility.

In the **treatment of sinus infection,** conservative measures are used if possible; these include nasal shrinkage to promote adequate ventilation and douching.[113] Sulfonamides are usually given in adequate doses by mouth in severe sinus infections. In those who do not respond, solutions of penicillin may be placed directly in the pus containing antra.

For shrinkage of the nasal mucosa, one may employ a solution of physiologic saline containing 0.25 per cent of neosynephrin (or from 0.5 to 3 per cent ephedrin sulfate or paredrine hydrobromide or tuamine sulfate) and 0.5 per cent antipyrine, permitting 4 or 5 drops to run into each nostril every two or three hours, as necessary. In addition, unless contraindicated (hyperthyroidism, extreme nervousness, sleeplessness, etc.) a benzedrine inhaler may be used; two long sniffs are taken through each nostril every hour or two if required. Care must be taken not to overdose with benzedrine as nervousness and sleeplessness may result. Most rhinologists employ constricting solutions which contain sulfonamides for local use in the nose in patients with sinusitis and purulent nasopharyngitis. These include paredrine sulfathiazole, tuamine sulfate with sulfathiazole, etc. If the nasal mucosa becomes raw or irritated, a few drops of liquid albolene may be instilled into the nose two or three times a day. If desired, the shrinkage solution may be used as a nasal spray in an all glass atomizer; because of the possibility of "lipoid pneumonia," sprays of oily solutions are not advisable.

Bacteriologists and rhinologists consider silver salts of little or no value, except for their psychic effect. Daily douching in infections of the maxillary

sinus may obviate the necessity of surgery in some cases. The *Proetz suction* is generally accepted as the best method of diagnosis and treatment in these patients.[98] In about 15 per cent of cases, douching is impossible through natural orifices and needle puncture is necessary; about 85 per cent can be reached with a Pierce cannula.

If conservative measures do not relieve the condition, surgery must be resorted to for removal of the accessible infected tissue. *Surgery is required* in the following cases:[61] (1) polypoid degeneration of the ethmoidal mucosa with an associated osteitis of the walls of the cells (which can be determined by inspection and stereoscopic roentgen-rays); (2) greatly thickened frontal or antral mucosa (in which case it is better to remove the infected mucosa; in any case, drainage, at least, should be established); (3) badly infected ethmoids which have failed to improve following conservative therapy.

If operation is decided upon, this must be thorough; whenever indicated, a submucous resection is performed. This provides a better view of the ethmoidal labyrinth.[87] If the ethmoids are involved, complete exenteration must be done; if some of the cells remain, no improvement may be noted. The removal of all infected, degenerated membrane in the sinuses has been advised.

The majority of sinus infections can be cured if these are handled intelligently and skilfully; the saying, "Once a sinus case, always a sinus case" is not true. Hurd warns that although sinus infections may be lessened or improved by a stay in a dry mild climate (such as that of Arizona), lasting climatic benefit will not be achieved unless by permanent residence.

TEETH

Dental infection in arthritis usually implies periodontitis (pyorrhea alveolaris) or periapical infection.[62,79,95] Unfortunately, **most medical men know very little concerning the teeth and their disorders;** *however, they do not hesitate to inform the dentist that certain teeth must be extracted.*

The dentist must be considered as a consultant rather than as a mechanic. Dentists are frequently able to save important teeth and to eradicate infection by means other than extraction. Both clinical and roentgenologic[22] examinations of the teeth should be made as the roentgen-ray may reveal the bony appearance to be fairly good when the condition is much worse clinically; on the other hand, roentgen examination may disclose lesions in a clinically "clean" mouth.

A full mouth x-ray should be taken in all patients with rheumatoid arthritis because of the frequent presence of abnormalities which may be overlooked clinically. *Thirty per cent or more of edentulous patients harbor retained roots, many of which are infected.*[25] The patient seldom realizes that these are present. The *dentist must look for* pulpless teeth, impacted teeth with their attending infections, infected cysts, residual infection of the alveolar process, gingivitis, periodontitis (pyorrhea alveolaris), ill constructed (poor) dentistry (such as overhanging fillings, poorly fitting crowns or bridges), dental caries and Vincent's infection.[30] A well-known

saying is: "Infected is the tooth that wears the crown." Sorrin has commented upon the frequency of *pulp stones* and *hypercementosis* in arthritis.[105]

The dentist must never give advice concerning extractions merely by looking at dental x-rays. He must check the mouth clinically, and must check the *x*-ray films with the patient's mouth (fillings, missing teeth, etc.) in as much as **it is very easy to mix up dental films** in a large *x*-ray laboratory. Unfortunately, individual dental films are not marked by most laboratories; this is an error as patients are often given someone else's dental films or a set containing his maxillary teeth and another patient's mandibulars. *A careful dentist will, of course, always check the films with the mouth.*

The internist must not offer advice concerning dental x-rays and their interpretation, unless he is especially trained in this work, and then only after carefully checking the films with the teeth clinically. One should always seek the advice of a competent dentist.

Advice Concerning Dental and Gingival Infections in Rheumatoid Arthritis

Abscessed teeth must be extracted. Do not remove anterior pulpless teeth unless there is a definite history of dental infection related to the arthritis. Pulpless teeth may be removed if there is a recent dental history related to arthritic symptoms.

All *periodontal disease* (pyorrhea alveolaris) *must be eliminated.*

The teeth must be checked periodically by clinical and roentgen examination.

The *dentist must check the dental x-rays with the patient's mouth;* the patient is frequently given another patient's films.

Treatment and care of the gums and teeth should be undertaken as would be done had the patient no arthritis (with the following exceptions):

That in the case of undernourished extremely weakened individuals, attempt should be made to build up the general resistance and strength of the patient before extractions are performed.

Do not remove more than one infected tooth at the first sitting, allowing a rest period of at least three or four days; if a flare-up of arthritic phenomena occurs after an extraction, a period of at least a week must be allowed before further extractions are performed. After the first extraction, 2 or 3 infected teeth may be removed at one sitting but under no circumstances should more than 4 infected teeth be removed at any one time.

Administration of sulfamerazine and sodium bicarbonate for twenty-four hours prior to and following extractions as noted before. (See General Considerations in the Treatment of Focal Infection, page 359.)

Pack from .15 to .3 gm. (2½ to 5 grains) of sulfanilamide into the socket following dental extraction, thereby decreasing the incidence of local postoperative infection, hemorrhage and dry sockets.

BACTEREMIA FOLLOWING DENTAL EXTRACTION

A transient bacteremia has been reported after dental extraction in more than 60 per cent of cases in one series; the incidence of positive cultures was correlated directly with the severity of the dental infection.[80] These extractions were performed under nitrous oxide anesthesia. No

serious systemic manifestations were noted. Cases of subacute bacterial endocarditis have also been reported following dental extractions.[33]

Burket found *bacteremias in 16 per cent of patients after removal of teeth under procain anesthesia* and in a much higher percentage following nitrous oxide.[14,16] The frequency of these bacteremias could not be associated definitely with the degree of oral sepsis or the severity of the operative procedure. Repeated blood cultures showed that *such bacteremias were transient,* and no specific serologic changes in the blood stream were observed following them. It was demonstrated that **organisms in the gingival sulcus could be forced into the blood stream during dental extractions**; this emphasizes the necessity for improved methods of sterilization of the gingival sulcus prior to extraction, and for caution in performing intra-oral operative procedures on chronically ill patients.

Attempts to sterilize the gingival sulcus showed the inadequacy of the method and antiseptic used. The bacterial flora was quantitatively reduced after treatment but in many cases pure cultures of Streptococcus viridans were grown from the gingival sulcus; this fact is of significance in the interpretation of periapical cultures on extracted teeth.

IMPORTANCE OF PERIAPICAL INFECTION

Hunter was concerned with sepsis from accumulated filth around ill-fitting crowns and bridge work and artificial dentures—so-called *"gold traps of sepsis."*[31] Frankel found no correlation between systemic disturbance and the extent of dental restorations (crowns, bridges and pulpless teeth) in a series of 19,000 persons.[37] *Appleton observed no increase in the amount of systemic disease in those with the greatest periapical infection as judged by clinical and roentgenologic examination.*[2] *Burket concluded* (from laboratory studies) *that evidence of periapical infection as a cause of systemic disease is even less convincing.*[15] He has shown further that organisms present in the blood stream may localize in the periapical region of

Pointers Concerning Dental Focal Infection in Rheumatoid Arthritis

There is *no pathognomonic test to determine infection of a tooth in the mouth.*

Chronic periapical lesions often exist without clinical signs or symptoms.[32] Haden considered chronic periapical infection to be by far the most common dental cause of systemic disease.[47]

Pathologic lesions harboring bacteria can be demonstrated about the apices of teeth without roentgenologic changes. *Roentgen evidence of periapical disease is dependable in proportion to the skill of the examination and to the judgment used in interpretation of the films.*[100]

In many instances, areas of suspected dental infection can be eliminated by surgical procedures other than extraction.

Periodontal disease (pyorrhea alveolaris) may be as important as periapical infection; there is no decisive evidence as to which is more important.

Unless there is a recent dental history related to arthritic symptoms, *pulpless teeth may be no more dangerous in the patient with rheumatoid arthritis than in the individual without arthritis.*

Are All Pulpless Teeth Infected?

NO! **The pulpless tooth is not a dead tooth** but has vital relationships with the surrounding tissue through the alveolodental periosteum.[43]

Histologic examination has shown that the pulps and periapical tissues of healthy teeth are free from evidence of irritation or bacteria.[35] **It is practically impossible to remove a tooth aseptically without actual cauterization of the gingival sulcus** because bacteria are forced back into the pulp and periapical tissues during the extraction.

If vital pulps are exposed and cultured by a method which eliminates contamination from the gingival sulcus, no growth is obtained.[45] Therefore, *cultures from extracted teeth do not reflect the true bacteriologic status of vital or pulpless teeth unless the gingival crevice is first actually cauterized.* Grossman has recently estimated that growth will be obtained from only about one-third of pulpless teeth if the proper technic is employed.[43] He minimizes the importance of some studies concerning periapical infection in which extracted teeth were used as the source of material. Some workers feel that pulpless teeth cannot be sterilized;[71] others have expressed a contradictory viewpoint.[3,22]

A negative roentgenogram of a pulpless tooth does not eliminate the tooth as a possible focus of infection.

Experimental evidence indicates that the loss of the dental pulp may render the periapical area more susceptible to infection.

Multirooted pulpless teeth (molars in general) should receive serious consideration because of the difficulties in technic and greater number of possibilities (root canals) for infection.

Extraction of Pulpless Teeth in Rheumatoid Arthritis[42,74,111]

At present, no dentist or physician can say with assurance whether bacteria are present or not in the periapical region of a given tooth regardless of the presence or absence of periapical roentgen changes.

One must always consider the dentist's opinion concerning the ease with which the lost teeth can be replaced, the roentgen appearance over a period of time and the intrinsic value of the tooth to the patient.

There is no more evidence to warrant extraction of pulpless teeth in a patient with rheumatoid arthritis than in a patient without arthritis.[4] An average of 3.9 pulpless teeth per mouth has been found in adults of all ages seen in a large dental clinic. The fact that a patient has rheumatoid arthritis does not alter the question concerning infectivity of pulpless teeth.

Pulpless teeth should be removed if there is: (*a*) Roentgen evidence of extensive rarefying osteitis involving more than half of the root; or (*b*) a long history of soreness of the tooth ("the lame tooth"), with tenderness to percussion.[45]

In a given case, *if the patient has had conservative therapy* (including removal of all definitely infected foci) *without improvement in his arthritis, one may consider removal of pulpless posterior teeth.* However, this must not be insisted upon and the patient should be told that such extractions may not improve his arthritis.

If pulpless anterior teeth are important foci, the disease area may be eliminated by root amputation (surgical removal of the diseased area).

In general, bridgework should not be attached to a pulpless tooth.[85]

teeth with apical disease and that *periodontal infection may be as important as periapical infection* in the causation of disease at a distance.

Cultures from the periapical region following dental extractions have shown streptococci in most instances. In some series Streptococcus viridans was reported in every culture.[24] In others, positive cultures were noted in about 67 per cent of the cases.[65] Burket reviewed the literature on this subject and showed that results of postmortem bacteriologic studies were similar to clinical findings.

Cook found that as many as 89 per cent of pulpless teeth may produce bacterial growth in glucose brain broth as compared with 4 per cent of control teeth.[27–29]

Dental extractions must not be thought of as mere mechanical procedures. The advisability of and the time for the removal of infected teeth must be considered carefully; improper removal may result in further harm to the patient. Indiscriminate removal of teeth may produce needless

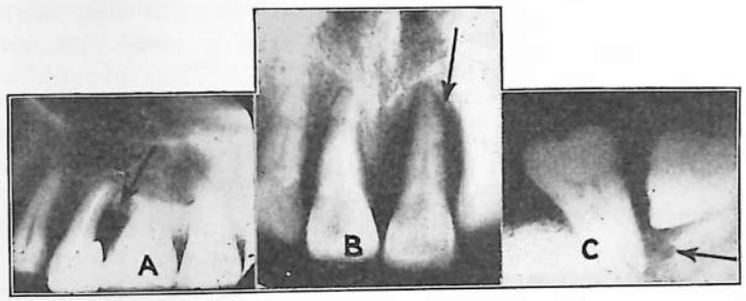

FIG. 104.—*A*, Deep pocket under overhanging filling; *B*, pyorrheal pockets around upper incisor; *C*, area of active osteomyelitis around impacted third molar. (Haden.)

edentia. *In any case of doubt concerning the care of certain teeth, the physician or dentist should consider himself as in the patient's position and act accordingly.*

IMPORTANCE OF PERIODONTAL DISEASE (PYORRHEA ALVEOLARIS)

The importance of periodontal disease in rheumatoid arthritis has not been evaluated.

It may be of great significance because:

a. A large total area is usually involved.

b. Trauma during mastication may force bacteria into the circulation.

c. Slightly loosened teeth may act as pistons forcing bacteria into the blood stream.

d. Severe periodontal disease may interfere with proper dietary regimen.

In view of the types and frequency of the bacteria in the periodontal regions, disease here should receive treatment because of the general improvement in health and resistance which may follow, and possibly because of its (unproven) exciting or aggravating rôle in some cases of arthritis.

REMOVAL OF IMPACTED TEETH

This should be done when the crowns have erupted through the alveolar process, or when they are causing symptoms or pressure.

DIAGNOSIS OF PERIAPICAL INFECTION

Acute Stage: This is relatively easy because of the subjective symptoms of pain, elongation, mobility of the tooth, etc., and the objective signs of swelling and (usually) fever.

Chronic Stage: This is extremely difficult.

There is no means of telling whether a given tooth is or is not infected except by making bacteriologic studies of the periapical region with the tooth in situ.

There is *at present* **no available means of determining whether or not a given tooth is acting as a focus of infection.**

Numerous clinical and laboratory studies give information in this respect:

Physical Examination:

Color: usually more opaque or dull gray.

Pressure over the apical region may produce pain not present on pressure over normal teeth.

Subjective (of importance when present):

Periodic episodes of pain associated with the tooth. This may also be present with uninvolved teeth; when associated with a pulpless tooth, it is important.

Pain on pressure may also be present in periodontal infection.

Roentgen Studies:

The absence of a roentgen-ray lesion does not eliminate the possibility of a tooth harboring infection or having an actual lesion.

The presence of a roentgen lesion does not indicate active infection. This may be evidence of a past lesion of bacterial origin, or of a lesion resulting from trauma, etc.

The characteristics of a "roentgen-ray lesion" do not indicate its quiescent, progressive or dormant nature.

Roentgen periapical changes indicate only destruction of the bony tissues in this region. *Those areas from which negative cultures are obtained have no characteristic roentgenologic appearance which distinguishes them from lesions with positive bacteriologic cultures.*

Other Suggested Aids in Diagnosis:

Short wave irradiation of the affected area followed by several sedimentation rates.[44]

Transillumination.

Root amputation and culture.

Main Types of Periapical Infection:

Acute: Dental abscess, probably of little significance in chronic rheumatoid arthritis, but may play a rôle in acute forms of infectious arthritis.

Chronic: So-called "dental granuloma," "blind dental abscess." May be an exciting or aggravating factor in rheumatoid arthritis. May occur about a tooth whose pulp has been removed, or whose pulp has undergone necrotic changes.

Sources of Periapical Infection:[1]

From the dental pulp (due to caries or deep cavities, accidental exposure during dental operations, or improper technic during removal of pulp); this is the commonest source.

From the gingival sulcus and periodontitis (pyorrhea alveolaris) by direct extension or through the lymphatics.

From the blood stream; this is uncommon.[18]

Methods of Treatment of Periapical Foci:[41]

Removal of the tooth and eradication of the granulating focus.

Apical root resection (apicoectomy) in some cases (only in anterior teeth).

Adequate root canal treatment in selected cases.

Is There Any Reliable Laboratory Evidence to Show That Dental Focal Infection May Cause or Aggravate Rheumatoid Arthritis?—No! Although dramatic improvement (or an exacerbation of symptoms) may occasionally occur following the removal of dental infections, this is the exception rather than the rule.

GASTROINTESTINAL TRACT

There is little evidence that absorption of toxic products from the intestinal tract plays an etiologic rôle in many cases of arthritis.

We have not been convinced that infection in the intestinal tract plays any important rôle in the etiology of the great majority of cases of rheumatoid arthritis; Hench has pointed out that arthritis has never been reported as a sequel to Hirschsprung's disease and that it is uncommon as a complication of typhoid fever or bacillary or amebic dysentery.

Some arthritic patients probably suffer because of malfunction of the digestive tract, but as yet there is no method of determining what sort of disturbance of the gut (if any) may lead to arthritis or make an existing arthritis worse. It is true that some individuals with rheumatoid arthritis note increased pain and swelling in the affected joints when they are constipated; one should make every effort to combat constipation by an adequate diet, fluid intake, abdominal exercises, proper bowel habits and drugs if required. It is important to carefully check all of the factors in the patient's diet and to see that his colon functions well as a general health measure.

The rôle of the gastrointestinal tract in arthritis has been stressed by various authors.[8] Substances may pass from the gastrointestinal tract to joint tissues.[17,67,68] Some have suggested that absorption of toxic products from this site is an important factor in the causation and continuance of cases of arthritis.[101,112] In one series of 60 arthritic patients, 53 were found to have streptococci on the rectal mucosa while only 20 per cent of controls showed similar findings; the serum of the patients contained strong agglutinins to these organisms.

Difficulty in the utilization of starch has been reported in the arthritic individual.[75] Seventy-nine per cent of the stools of a series of arthritic patients showed an excess of starch as compared with slightly over 25 per cent in a control group. In this same group, iodine staining organisms were found in the stools in 90 per cent of patients as compared with 22 per cent of controls. The fermentation test was positive in over 80 per cent of the cases as compared to 24 per cent in the controls. In all of these examinations, no definite pathologic condition was found except the presence of starch in excess in the arthritic patients.

Suspicion has been raised that some of the benefits following changes in diet in arthritic patients may be due in part to *changes in the bacterial flora in the gastrointestinal tract.*[108] Greater numbers of bacteria have been found in the stools of arthritic than of normal persons. This has been explained in part by the frequency of anatomic and functional abnormalities of the gastrointestinal tract in such patients (especially changes in the colon).

Much of the work on the digestive tract has not been properly controlled. *The finding of starch in the stools means* either abnormal starch intake, rapid small gut motility, deficient amylase secretion or an inadequate gut pH for starch splitting. Several widely different types of digestive disturbances will all give excessive starch in the stool. One is left in doubt as to whether it is the presence of the starch, or the disturbance which causes it to be there that is of any importance in arthritic patients.

The *bacterial count* depends largely on the solidity *of the stool,* multiplication being slow in a stool that is desiccated early in its transit of the colon, while a stool kept liquid by cathartics may remain a good culture medium all the way around to the rectum. One individual may show a variation in the bacterial content of the stool from day to day depending on the use of cathartics and on the relation between the taking of the cathartic and food. A saline, for example, that traverses the small gut rapidly, may carry a large amount of good culture material into the cecum which ordinarily would have been absorbed in the small gut. The importance of achlorhydria is probably due to its bearing on the emptying time of the stomach for the same reason.

The finding of abnormalities such as *elongation, dilatation, tortuosity and reduplication of the colon,* as well as gastrointestinal stasis have been stressed.[84,107] The distorted gastrointestinal tracts of these individuals may return to normal under proper dietetic therapy.[36] A deficiency of vitamin B may produce similar gastrointestinal findings, leading to atrophy of gastrointestinal mucosa.

Because he considered intestinal stasis to be the cause of arthritis, Lane advised colectomy in selected cases some years ago; this is not advocated at present.[70] Goldthwait and Osgood recognized the importance of proper elimination and function of the gastrointestinal tract.[39] Hartsock, in a study of 100 patients with rheumatoid arthritis, concluded that the percentage of cases with gastrointestinal disturbance is greater than in other exhausting diseases, and that the gastrointestinal symptoms and

findings are as marked in early cases and in young individuals as in old, long standing cases.[48] He found that in the absence of other pathologic features, the arthritis responded to treatment of the gastrointestinal tract alone. Freund suggested that abnormal acids may be produced in the intestinal canal by processes of abnormal fermentation and that these

Combat Constipation and Gastrointestinal Stasis

In the habitually constipated patient, administer 2 glasses of *warm water* containing ½ teaspoonful of *sodium bicarbonate* and the *juice of half a lemon* on awakening; lie on the right side for five minutes. Follow by *light exercises* (lie flat in bed; bring each knee toward the chest as far as possible three times; repeat three times with both knees together; sit up in bed three times without bending the knees).

Add to the diet a dish of *prunes* each morning and *applesauce* or an *apple* each night.

Insist upon a *regular time for bowel movement,* usually just before or just after breakfast.

If necessary, take *granular agar-agar,* 2 tablespoonfuls mixed with the morning cereal, or *metamucil* (1 tablespoonful stirred well in a glass of water—once or twice a day); *or mineral oil,* 1 tablespoonful morning and night. *Mineral oil is practically tasteless if kept in the ice-box.*

If required, administer small doses of *cascara* (such as a teaspoonful of the aromatic fluid extract of cascara sagrada each night) until the bowels have become regulated. This dose of cascara coupled with mineral oil as outlined above produces effective, soft movements in most instances.

Magnesium sulfate (2 teaspoonfuls) or pluto water (1 glass) has been advised on three consecutive days of each month if there is marked joint swelling.[110]

Abdominal massage may be useful in such patients. This is performed in a circular manner without the use of too great pressure. Massage should commence in the left lower quadrant and work downward; when this portion of the bowel has relaxed under the hand, the massage may be done in the left upper quadrant, again working downward. Following this, one should begin in the right upper quadrant and slowly work down the bowel, with the final massage beginning in the right lower quadrant then working along the course of the colon.

Colonic irrigations are no longer employed routinely in most clinics.[102]

In those individuals whose *constipation* is on a somewhat *spastic* basis coupled *with nervous tension,* the following prescription may be used to relieve spasticity and nervousness:

℞—Tr. hyoscyamus,			
Tr. belladonna	āā	16	℥iv
Sod. bromide		30	℥j
Flex. casc. sagr. arom		45	℥jss
Elixir digestive comp. . .	q.s. ad	180	℥vj

Sig.—One teaspoonful, three times a day, after meals.

In patients with large, atonic colons, small doses of *tincture of nux vomica* (0.3 cc. or 5 minims several times a day) are often useful in relieving constipation.

In some cases of marked constipation, good results may follow modification of the intestinal flora by milk soured with B. acidophilus, together with the administration of lactose (several ounces per day).

acids may exert a harmful action on joint membranes; he thought that intestinal disinfection and simultaneous alkalization may be a therapeutic method of treating rheumatism.[38]

Disturbances in the pattern of the colon, anacidity and abnormality of carbohydrate tolerance are probably not the cause of the arthritic picture, but are merely the result of a prolonged illness.[78] Some have found no constant deviation from normal gastrointestinal function in patients with arthritis.[66]

Achlorhydria.—The importance of achlorhydria in patients with rheumatoid arthritis is debatable. This is noted in from 20 to 50 or more per cent of these patients.[19,23,50,76] Gastritis is not found at necropsy in these individuals and it is doubtful if achlorhydria is of primary etiologic significance.[52] If free acid is absent from the gastric contents, one may employ a teaspoonful of dilute hydrochloric acid in water, to which is added the juice and pulp of an orange and a small amount of sugar to render this more palatable. This may be taken through a straw if desired (because of damage to the teeth) three times a day with meals.

PROSTATIC AND PELVIC INFECTION

Some arthritic patients with chronically infected prostates improve following a series of prostatic massages. These individuals are usually examples of *atypical rheumatoid arthritis* and in most instances have had a rather abrupt onset of their joint phenomena. Occasionally, even light stroking of the prostate has caused an acute flare-up in their joints.

At least two microscopic examinations of the fresh unstained prostatic secretion (forty-eight hours apart) must be made before eliminating the prostate as a focus of infection.

A focus of infection in the genito-urinary tract has been found in only 5 per cent of patients giving a negative past urologic history.[67] In the study of 1000 consecutive cases, 40 per cent of arthritic patients admitted a past urologic history; of these, 65 per cent harbored infection. In some, genito-urinary tract infection was thought to be the sole cause of the arthritis.[116]

The majority of cases of chronic prostatitis are due to non-specific infection and not to gonorrhea. Prostatic massage may (at times) *be associated with a sharp exacerbation of joint pain and swelling.*

In the treatment of a chronic prostatic infection in an individual with rheumatoid arthritis, the massage must be very gentle during the first several treatments. Such massage must be continued one, two, or three times a week until the prostatic secretion shows not more than 5 to 10 leukocytes per high power field. The intervals between such massages are then lengthened to one, two, or three weeks and massage is finally discontinued if the prostatic secretion contains no pus. The oral administration of sulfonamides may be indicated to clear up a prostatic infection. An infection in the prostate or seminal vesicles may be secondary to infection elsewhere in the body (teeth, tonsils) and may not respond to treatment as long as foci persist elsewhere in the body.

Pelvic Sepsis.—Most internists agree that *non-specific pelvic infections* (in the female) *are seldom directly related etiologically to an arthritis.* A few

authors believe that cervicitis may be a frequent cause of rheumatoid arthritis. This has not been true in our experience.

The cervix has been called the pelvic tonsil.[40] Robinson stated that *"chronic infective arthritis in women is due, in the vast majority of cases, to an infected cervix."*[53] (This view is not held by most rheumatologists.) He was impressed by the frequency with which patients dated the onset of an arthritis to marriage, childbirth or abortion, and found that treatment of the cervix resulted in improvement of the arthritis. In his series, cervicitis was 15 to 20 per cent more common in arthritic than in non-arthritic women. He stressed the fact that chronic cervicitis may be present with an apparently normal cervix, and that even though the cervix is considered to be a focus in arthritis, it is rarely treated with success. He feels that cauterization, swabbing or amputation of the cervix do not completely cure the cervicitis. In his experience, the best method of treating cervicitis is intrapelvic and intracervical diathermy, heating the whole pelvic contents including the cervical glands throughout their entire length, and also the parametrium. It is his contention that most cervicitis is puerperal in origin and that obstetricians bear the burden of preventing many cases of chronic infective arthritis in women.

Although salpingo-oöphoritis is mentioned by some as a possible cause of arthritis, this must be very rare except in cases of gonorrheal involvement.

The question of menopausal arthralgia and arthritis (climacteric or chronic villous synovitis) is discussed elsewhere.

GALL BLADDER

Although some individuals consider an infected gall bladder to be a significant focus in arthritis, very few patients with cholecystitis have developed rheumatoid arthritis and most individuals have seen no permanent joint benefit following removal of infected gall bladders in patients with rheumatoid arthritis.

Hench has found **little or no evidence that cholecystitis causes arthritis.** Cholecystectomy is warranted only because of the findings in the gall bladder *per se*, and not because of any relationship to the arthritis.

If chronic cholecystitis complicates rheumatoid arthritis, treat the gall bladder disease in the same manner as if arthritis were non-existent; in the majority of these patients, a medical regimen is used and surgery is resorted to only if symptoms are not relieved or if frequent attacks of gall stone colic occur.[114]

In one study of several hundred patients with rheumatic disorders, the incidence of gall bladder infection was no higher than in the usual group of hospital admissions; in no instance (in this series) was the cholecystitis found to be a definite factor in the etiology of the arthritis.[49] Less than 5 per cent of these patients gave evidence of gall bladder infection.

The indications for cholecystectomy in chronic arthritis should be determined by the pathologic process in the gall bladder and not by the arthritis.[58] Recent reports emphasize that cholecystitis is rarely a focus (less than 5 per cent

of cases) in rheumatoid arthritis and that the effects of cholecystectomy in this disease are rather unimpressive, even though markedly infected gall bladders are removed. Relief of rheumatoid arthritis after any operation may be due solely to a non-specific effect.[64,81]

OTHER FOCI OF INFECTION

Appendix, Bronchi, etc.—Boots and McCollom reported a young girl who exhibited recurring *hydrarthrosis* of the knee for a long period. With each flare-up of the knee, there was definite tenderness over the appendix. The knee became entirely well after removal of the appendix.[11] Although spectacular results have been reported very occasionally in rheumatoid arthritis following *appendectomy*,[52] these are likely to be a non-specific effect due to the hospitalization, rest in bed and anesthesia.

Rheumatoid arthritis has not been reported as a complication of acute or chronic appendicitis, despite the frequency of appendicitis.

One is not impressed by any definite relationship between appendiceal infection and rheumatoid arthritis, although it seems logical that this may be found occasionally.

It is conceivable that in certain cases of *bronchitis*[51] and *bronchiectasis*, the infected areas may serve as a focus of infection; their treatment, however, is much the same as though no arthritis were present. It is probable that the use of penicillin may result in marked improvement in many patients with bronchiectasis, and will be especially useful pre- and postoperatively; lobectomies may be performed with much more safety following a course of penicillin.

Phlebitis.—Infection in the veins of the legs and neck has been mentioned as a frequent cause of joint disease.[73] Meyer believes that latent chronic phlebitis of the veins of the legs or jugular veins or both is responsible for more rheumatic disease than any other known foci. He had noted sensitivity to pressure over inflamed jugular veins as well as over veins in the legs. His treatment of jugular phlebitis is by application of several leeches over each jugular vein so involved, varying with the age and weight of the patient. He believes that the jugular phlebitis results from inflammation of the tonsils. It is true that phlebitis often causes pains in the lower extremities and may simulate joint involvement; phlebitis should always be searched for, especially in overweight women with pains in the legs and knees. The use of Elastoplast or similar bandages to the affected extremity will often result in marked improvement of the phlebitis (with the patient allowed to walk around as usual). The concept and treatment advocated by Meyer has not been accepted in rheumatic diseases.

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CHAPTER XXI

MISCELLANEOUS ITEMS OF THERAPY

RHEUMATOID arthritis produces a greater amount of crippling than any other form of rheumatic disease. These patients should have early institutional treatment. The inflammation of articular and periarticular tissues will lead, if not arrested, to muscular atrophy, deformity and ankylosis. In the great majority of these cases, no definite infecting organism has yet been isolated.

The practicing physician must appreciate the natural history of rheumatoid arthritis. A small percentage of these patients will go on to ankylosis and progression of the disease despite every available medical attention. *The great majority of patients with rheumatoid arthritis will eventually have an arrest of their disease with little crippling whether adequate treatment is employed or not.* Most of these patients can be greatly benefited by proper regimen of therapy which is individualized in each instance in an effort to prevent muscular atrophy, weakness, loss of weight and deformity.[2]

It may be difficult in some instances to make the diagnosis of rheumatoid arthritis in the early stage of the disease. In some cases, the onset may present a confusing picture resembling peripheral vascular disease, rheumatic fever, fibrositis, gonorrheal arthritis and other disorders.

Although the disease may begin with an acute febrile polyarthritis, the onset is usually insidious with the disease running a course of many years with remissions and exacerbations. Many of these individuals are of slender build, with poor posture, cold clammy hands and feet, tired, worried, tense, underweight and overworked. In a carefully taken history, one will often obtain a history of emotional strain, overwork, worry, sudden shock, malnutrition and in many instances, a family history of rheumatic disease.

The patient is often a young woman with vague aches and pains, anorexia, fatigue, loss of weight, tachycardia, backache and who sooner or later notices some puffiness or swelling of one or more joints such as the proximal interphalangeal joints of the hand, knee, wrist or ankle. *This stiffness and swelling may be transient at first* and the diagnosis is frequently overlooked at this stage. Although *the pain and swelling may disappear following symptomatic care* (such as rest or aspirin), several months later the patient again develops pain, swelling and stiffness in one or more proximal interphalangeal joints, wrists, ankles or knees together with a definite limitation of joint motion. These individuals *frequently complain of stiffness on awakening in the morning.* There is definite tenderness on pressure laterally on the puffy interphalangeal joints with pain on motion of these joints. At a later stage, the physician observes atrophy of muscles and skin.

It is usually easy to differentiate the disease from degenerative joint disease which usually occurs later in life and is characterized by the tell-tale Heberden's nodes—swellings of bone and cartilage at the base of the terminal phalanges; *the individual with degenerative joint disease usually has* a normal sedimentation rate, very little or no excess of fluid in the joints, no constitutional phenomena and does not go on to ankylosis.

It may be difficult to make a definite roentgen diagnosis of rheumatoid arthritis at an early stage as the enlarged soft tissue shadow may be the only evidence of the disorder. At a later stage, there may be a decrease or loss of the joint space, osteoporosis of the epiphyses and still later a generalized decalcification of the shaft of the bones.

Although the *sedimentation rate* is non-specific, it *is probably the most valuable laboratory evidence* which may be used in support of the diagnosis of rheumatoid arthritis. One must remember, however, that many chronic infections and other disorders which produce tissue breakdown (such as coronary artery occlusion, metastatic malignancy, etc.) may produce a marked acceleration of the sedimentation rate.

One must also recall that a patient with degenerative joint disease may have a rapid sedimentation rate if there is a concurrent sinusitis, bronchitis or other subacute or chronic infection present; *i. e., a rapid sedimentation rate in the presence of joint phenomena does not always mean rheumatoid arthritis or specific arthritis.* On the other hand, *it is unusual to see a patient with active rheumatoid arthritis with a normal sedimentation rate.* **If the sedimentation rate is normal, the diagnosis should be checked.** There are definite instances, however, in which an active rheumatoid arthritis is encountered with a repeatedly normal sedimentation rate for some months; these cases are in the great minority.

As the rheumatoid arthritis progresses, definite *periarticular swelling* and *effusion into the joints* may occur; this is especially notable in the knee joints. If untreated, the individual may then progress to severe *muscular atrophy, deformity* and *crippling* with *stiffness* or actual *ankylosis* in multiple joints. He is then left in a helpless condition with *flexion deformities* of the knees, *subluxation* of the tibia on the femur, stiffness of the shoulders and marked ulnar deviation and flexion deformities of the fingers and hands.

The point which is uppermost in these individuals' minds is the *fear of becoming crippled.*[2] The physician must point out to the patient in a manner which she understands, with diagrams when necessary, that if she follows a carefully prepared regimen, she will not become crippled and that she will have 9 out of 10 chances of being benefited, with about 7 chances out of 10 of being markedly improved. *The patient must not be given the picture of a "quick cure" of this disease* because such is not possible in the great majority of these individuals with our present knowledge. The patient must understand that the treatment will require months and probably several years, and that she should be under careful supervision for the remainder of her life.

The physician must gain the confidence of the patient, not by false promises

or a false hope of quick cure, but by an honest and sincere discussion of the problem as it applies to that particular individual. *Most patients with rheumatoid arthritis will be greatly benefited by a stay of at least a month or two in a hospital* under careful supervision; this should be suggested in practically all cases, when at all feasible. This will enable the physician to observe the patient at least daily and to perform any necessary laboratory work (complete blood count, urinalysis, blood Wassermann reaction, sedimentation rate, blood uric acid, *x*-ray of at least one of the involved joints—preferably a hand, and any other tests which may be indicated if there is a question as to diagnosis: chest *x*-ray, basal metabolism, tests for Brucellosis or gonococcal infection, dental *x*-rays, examination of prostatic secretion, examination of the nose, throat and sinuses, gastric analysis or biliary drainage if indicated, synovial fluid examination in some cases, orthopedic consultation, etc.).

When the patient is hospitalized, definite advantages should follow, including:

1. *Rest* of the individual—both physically and mentally.

2. Careful *rest of the joints* by proper splinting where indicated in positions of physiologic rest; orthopedic consultation is often very valuable in this respect.

3. *Adequate diet,* with interval feedings, insisting upon the daily intake of at least a pint of milk, 200 cc. or more of citrous fruit juice, at least 2 fresh green vegetables, at least one serving of meat, fish, eggs or cheese, in addition to other items which may be necessary to bring the total calories to the desired level so that the undernourished individual gains in weight (whole wheat or rye bread, butter, nuts, cereals, starchy vegetables, etc.) The patient must understand that the milk, fresh fruit, fresh green vegetables, meat or fish portion of the diet is a necessity (unless not tolerated) and that the bread, cereal, starchy vegetables, etc., should not be eaten first and thus "fill" the patient so that she can not eat the former.

4. The physician by repeated questioning can usually gain the confidence of the patient and thus dispel any fears or worries which she may have. The *social service department of the hospital may be of assistance* in working out details at home so that the patient's cares and worries will be lessened considerably. In addition, this department will usually be able to arrange for a prolonged stay, if desirable, in a *convalescent home* when the patient is ready to leave the hospital.

5. *Adequate physical therapy* should be available in the hospital so that when the patient is able to tolerate this, it can be given most efficiently and with great comfort to the patient. It is amazing to see the amount of mental and physical comfort which the patient derives from a properly given warm bubble bath, with mild massage. *Underwater therapy* permits the individual to perform many movements which were impossible without marked pain out of the pool. This not only assists the patient physically, but greatly improves her mental attitude when she sees that she can actually bend the involved joints without discomfort, and that this improvement continues daily. In addition, *the department of physical therapy*

instructs the patient concerning proper exercises and massage so that this is not given in a haphazard manner when the patient returns to her home.

6. The department of *occupational therapy* should be of equal assistance in patients with rheumatoid arthritis in both keeping the patient's mind occupied and in prescribing those bits of handicraft which will exercise the desired muscles, even without the patient's realization at times that she is performing such exercises.

7. The kind, *considerate care of well trained nurses who are genuinely optimistic* in their dealings with these patients is of inestimable value.

The *most important principle in treatment of the patient with rheumatoid arthritis* is that she must be rested. *Rest* in bed will lessen both mental and physical fatigue, assist the undernourished patient to gain weight and permit one to remodel the body by correcting any functional deformities which are usually due to poor posture; proper body mechanics can be restored.

A good *masseuse* can assist in obtaining relaxation of the patient. *Graduated exercises* are begun as soon as the acute stage of the disease has passed. *These exercises are performed first in recumbency,* and *later,* while *sitting* or finally, *standing.* During the period of rest in bed, exercises must be planned carefully to encourage deep breathing and to improve the tone of the quadriceps, gluteal, hamstring and leg, foot, arm and hand muscles.

If flexion deformities are present, these should be corrected by *serial plaster shells* or *Buck's extension,* followed by *plaster shells or casts.* In some instances, further relaxation may be secured by *periarticular injection of procaine* or the use of an *ethyl chloride spray* just prior to the application of the splints or casts to permit the greatest possible correction. In some individuals, the use of prostigmin (with atropine) may aid in decreasing muscle spasm and in lessening deformities.

Some rheumatologists feel that exposure to the *sunshine* or to *ultra-violet irradiation* in gradually increasing amounts is of definite tonic value to these patients. Others prescribe ionization to the affected joints, with reciprocating faradism to the extensor and flexor muscles of the wrists, and the vastus internus and hamstrings of the knees.

If a large amount of *fluid* is present in the knee joint, this may be *aspirated* in an effort to prevent stretching of the joint capsule and stretching of the ligaments before application of the plaster splints or casts.

When the patient becomes ambulatory, proper *orthopedic support* must be prescribed as needed. In those with marked involvement of the knees, light metal leg *braces or walking calipers* or light plaster or Castex *walking cylinders* (cut out anteriorly for the patella) may be necessary. In some, it may be necessary to give *support to the back* by a low or high back *corset,* with or without additional metal support in the corsets. *Proper shoes* must be worn which fit the foot of the patient, with adequate width anteriorly, a heavy, long counter, a snug heel fit, and adequate support for the longitudinal and anterior arch if necessary. If there is marked tenderness over the heads of the second, third and fourth metatarsals, a *metatarsal bar* may be needed on the sole of the shoe. In some of these patients, an elastoplast strapping may be useful placed just proximal to the heads of the metatarsals.

Although aminopyrine is definitely of value in diminishing joint pain, it is considered too dangerous to use because of the possible sensitivity with resulting agranulocytic angina, which may prove fatal. The combination of *aspirin and phenacetin,* 0.3 gm. (gr. v) of each, together with *phenobarbital* 15 mg. (¼ gr.) has proved most useful in relieving pain and stiffness in these individuals. Occasionally, at the onset of treatment it may be necessary to add small amounts of codeine phosphate 10 to 15 mg. (⅙ to ¼ gr.) every four to six hours, but this is not advisable as a routine. *Any patient who requires narcotics for relief of joint pain may have a malignancy;* the diagnosis should be checked in such cases. Codeine may become habit forming and may cause marked constipation, nausea, vomiting and indigestion; if required, this drug must not be continued for more than several weeks. *Sedatives* may be required at bedtime for a short period in these individuals. These may include *phenobarbital* 30 to 90 mg. (½ to 1½ gr.), *seconal* 45 to 90 mg. (¾ to 1½ gr.), *bromides* 1 gm. (gr. xv) or many of the proprietary preparations which are available. The individual seems to fall asleep somewhat more quickly with seconal than with nembutal, but the effect of the latter usually persists longer. There appears to be no or very little hangover in the morning following the administration of seconal; slight morning drowsiness or dizziness occasionally follows the use of nembutal. None of these preparations are required for long periods in these individuals; as the joint pain, swelling and stiffness decrease, the individual sleeps well without sedation.

Various *liniments* have been applied to the painful joints. These include chloroform liniment, soap liniment, methyl salicylate (oil of wintergreen), etc. These produce a local counterirritant effect and assist in relieving the discomfort in some individuals. Some of the salicylate is absorbed from methyl salicylate ointment applied locally so that some analgesic effect may also result from this source. Local application of liniments often aid psychologically to a large extent in some patients.

Symptomatic therapy may be necessary to correct anemia, achlorhydria, anorexia, vitamin deficiency, or endocrine dysfunction. Most physicians prescribe adequate supplementary vitamins daily for these individuals, in an effort to supply at least the daily minimal requirements, when known (as vitamin A—4000 U.S.P. units; thiamin hydrochloride—1 mg.; riboflavin—2 mg.; niacin amide—minimal daily requirement not yet established—possibly 10 to 20 mg.; vitamin C—75 mg.; vitamin D—400 U.S.P. units). Most manufacturers market concentrated vitamin preparations which meet these requirements by one small capsule per day (such as multicebrin—Lilly; special vitamin formula—Squibb, etc.).

The physician must remember that these individuals must eat an adequate diet and that there may be additional vitamins and other substances in our food which have not yet been isolated in pure form. The patient must never think that "because he is taking his vitamin capsules *each day" he can neglect* his proper diet.

If *menopausal symptoms* are present, one may employ stilbestrol in doses of 0.5 mg. (to 1 mg. if necessary) to control the flushes and arthralgias which may accompany this syndrome. In an occasional patient, stilbestrol is not tolerated, even when the dose is reduced to 0.1 mg. per day because of subsequent nausea and vomiting. In these individuals, estrogenic substances or synthetic preparations may be given parenterally.

As the patient improves, there is a gain in weight, decrease in stiffness and joint swelling, lessening of pain and an improved sense of well-being. *The sedimentation rate usually returns to normal gradually* as the disease becomes inactive. Inasmuch as recurrences of the disease are common, the patient should be seen at regular intervals for *follow-up examination* in an effort to detect a recurrence at the earliest possible time and to begin additional therapy, if required.

A satisfactory therapeutic regimen must combat all impinging destructive agencies including fatigue, faulty nutrition, anemia, endocrine or nervous imbalance, focal infection and postural defects. An effective therapeutic program involves total support of the patient, *i. e.*, a correlation of all therapeutic procedures which might be of some assistance in that particular individual.[76]

Adequate therapy must include removal of worry, fatigue, focal infection and faulty body mechanics and one must supply rest, sedatives, physical and occupational therapy, optimal nutrition, iron to combat the anemia, replacement therapy in the endocrine or gastrointestinal system and tonics.

Short reported *improvement in 53 per cent of 300 patients with rheumatoid arthritis on general measures* (rest, analgesics, physical therapy, good diets, etc.); these patients were followed for periods up to ten years. In 16 per cent, the disease had become arrested.[90] Scores of drugs have been used in the treatment of various forms of arthritis. Many of them have little or no rationale.[91] Some of them have been designed to relieve pain; others (vitamins, sulfur) have been prescribed to correct some supposed (but often unproved) deficiency; still others (as gold) are used to lessen a supposed (but unidentified) type of infection.[81]

Sulfonamides have as yet been found to have *no beneficial effect* in cases of active rheumatic fever or rheumatoid arthritis and are of no value in degenerative joint disease.[4] *Sulfonamides should be used only to treat specific arthritides of known bacterial origin* (gonococcal, staphylococcic, hemolytic streptococcic, pneumococcic, meningococcic, etc.) which are known to be susceptible to the action of these drugs. *Penicillin is likewise of value in the same group of specific arthritides*, but *has not been shown to be of value in rheumatoid arthritis.*

The use of massive doses of vitamins, particularly of vitamin D (in doses of from 200,000 to 300,000 units per day) is still open to question. Unfortunately, undue advertising ballyhoo has made the medical profession somewhat skeptical of their value so that it has been very difficult to arrive at a logical conclusion as yet. Several groups of workers have reported definitely good results in patients with rheumatoid arthritis following the use of large doses of vitamin D; other equally reliable workers

have not seen such marked improvement follow this drug in their patients (although they used exactly the same drug and dosage, in many instances). *Some patients with rheumatoid arthritis have definitely improved following massive doses of vitamin D; others have had toxic reactions*, including nausea, vomiting, nocturia and occasionally the formation of renal calculi. If the physician employs large doses of vitamin D, it is suggested that he read the pros and cons on this question very carefully, as enumerated in the Chapter on Vitamins in Rheumatoid Arthritis. Large doses of vitamin D may be toxic, and certain preparations of this drug are definitely more toxic than others. *In the light of our present knowledge, it is permissible to employ large doses of vitamin D in patients with rheumatoid arthritis, provided that* the patient be watched carefully for signs of toxicity, and that the blood calcium be checked every month; if this rises markedly the drug should be at least temporarily discontinued.

Aspirin (acetylsalicylic acid) *remains the safest and generally the most effective analgesic drug and may be prescribed fairly generously*, provided that those who receive it do not rely upon it alone and thus omit other more important remedies. We frequently employ a combination of aspirin and phenacetin (0.3 gm.—gr. v. of each) together with small amounts of phenobarbital (15 mg.—$\frac{1}{4}$ gr.) three or four times a day in patients who are having marked joint discomfort. It is often advisable to have the patient place this medication on a table at the bedside, together with a glass of water, on going to bed. *On awakening early in the morning* (whether it be 5, 6 or 7 A.M.), *the patient may take this medication immediately*, and then roll over for an additional nap if so desired. By the time he is ready to arise, much of his pain and stiffness will have disappeared. This procedure may be of great symptomatic value to many individuals with rheumatoid arthritis and fibrositis. *The physician must not depend upon analgesics to relieve the discomfort in rheumatoid arthritis permanently.* These are used as a temporary expedient until the patient is taught to rest properly with his joints in positions of physiologic rest, with the addition often of molded plaster splints; *heat, massage and proper exercise* will help greatly in improving the individual and thus reduce his discomfort.

Opinions continue to differ widely on the use of gold in rheumatoid arthritis. Authorities are agreed that gold salts are of no value in degenerative joint disease or fibrositis and that if gold is employed in arthritis its use should be restricted to rheumatoid arthritis. Some physicians of wide experience in rheumatic disorders consider chrysotherapy the treatment of choice in rheumatoid arthritis while others equally well trained employ it only after conservative measures have failed. Some have thought that its toxic effects outweigh its beneficial action. *Most recent articles on the subject favor the use of gold therapy*, but in doses much smaller than were originally employed. *We have used gold salts parenterally in rheumatoid arthritis in small doses and have been very favorably impressed with the marked beneficial effects* which have followed in most cases, with very little evidence of severe toxic reactions (WHEN SMALL DOSES WERE USED). In our hands, gold salts have been of greater value in rheumatoid arthritis than any other single

Relief of Joint Pain

Splinting of involved joints in positions of physiologic rest, together with carefully supervised rest in bed, and the use of hot fomentations to the affected joints for twenty to thirty minutes two or three times per day—usually relieves joint pain.

With proper care, most individuals need only small amounts of analgesics (aspirin and phenacetin) after several weeks of therapy. One does occasionally encounter patients who require codeine despite proper treatment.

Aspirin 0.3 gm. (gr. v) and phenacetin 0.3 gm. (gr. v) may be used every three or four hours as necessary during the acute stage of the disease; splinting in positions of physiologic rest usually make this unnecessary.

One of the mistakes in giving aspirin is that only 0.3 gm. is used at a dose; at least 0.6 gm. should be given each time, or a combination of 0.3 gm. of aspirin and 0.3 gm. of phenacetin; the smaller dose will usually not relieve pain and will thus leave the patient pessimistic concerning his joint symptoms.

If absolutely necessary, small amounts of codeine phosphate—10 to 15 mg. ($\frac{1}{6}$ to $\frac{1}{4}$ gr.) may be given several times per day and at night (for very short periods); however, this is inadvisable. If narcotics are required, the diagnosis should be checked; a malignancy may be present.

The patient should remain in bed if pain persists in weight-bearing joints. If this principle were adhered to, fewer deformities would be seen in these patients.

Alternating warm and cold douches to inflamed joints often relieve pain; use warm water for one and a half minutes, then cool water for one-half minute, and continue to alternate warm and cool water for a total of twenty or thirty minutes, always beginning and ending with the warm water.

Procaine injections, used cautiously, may be a useful measure in relieving pain, at least temporarily, especially that due to fibrositis. Such injections should not be substituted for extensive endeavor to discover the cause of the pain.[1,100]

Some physicians prefer *aminopyrine* (formerly amidopyrine; trade name pyramidon) in 0.3 gm. (5 gr.) doses four times a day; the analgesic actions of this drug are excellent. Aminopyrine is probably the most important causative factor in agranulocytic angina and has been responsible for numerous deaths.[16,19] It is not recommended because of this.

Cinchophen is the standby of some physicians in rheumatoid arthritis. The average dose is 0.5 gm. ($7\frac{1}{2}$ gr.) three or four times a day.[43,92] It should be used with sufficient alkali to keep the urine alkaline and should be given on only three consecutive days, to be followed by a period of abstinence from this drug for four days. Its dangerous potentialities are recognized by well-informed physicians.[16] *This drug is not advised* because liver damage such as acute yellow atrophy and cirrhosis (with more than 200 deaths) has followed its use, and because as yet no completely safe method of administration has been presented. Intoxication has also been noted following the use of neocinchophen.[18] Proprietary preparations belonging to the quinine group of drugs include:[21] atophan, neocinchophen, tolysin, Cinsa-Vess, atoquinol, gorun, hexophan, novatophan and others. The toxic properties of neocinchophen are in general similar to those of cinchophen.[16]

drug. However, *injections of gold are not the final answer to rheumatoid arthritis* inasmuch as the gold may be toxic, relapses do occur when gold therapy is stopped (in a fair number of individuals) and not all patients with rheumatoid arthritis respond to gold therapy. Because of the possible toxic effects, *we do not feel that gold should be employed as a rule by the general practitioner in the treatment of rheumatoid arthritis unless* he has

Notes Concerning Aspirin and Salicylates

Salicylates are the safest analgesics; they are also vasodilators.[74]

Aspirin is insoluble in water *and should be prescribed in dry form. It is soluble in a solution of potassium citrate.*

A soluble preparation, calcium acetylsalicylate, is available.[72] Calcium aspirin may be given in doses of 0.6 to 1.0 gm. (10 or 15 gr.) three times a day.

The analgesic action of aspirin seems superior to that of sodium salicylate in rheumatoid arthritis.

Proprietary preparations containing aspirin or salicylate include[21,80] empirin, alka-seltzer, anacin, novaspirin, salsil, kalmopyrin, aspro, genaspirin, aspriodine, aspergum, calsapirin, acetidine, Dr. Miles' aspirmint, M. Matte's German asthma powder, etro, etc.

The *main disadvantages of aspirin* are the tendency to produce sweating and gastrointestinal irritation; if sweating is excessive, aspirin may be combined with equal amounts of phenacetin, the total dose of aspirin and phenacetin being 0.6 to 1 gm. (10 or 15 gr.) several times a day. Much less gastrointestinal discomfort will be noted if a glass of water is taken with the aspirin. Some individuals prefer to dissolve the aspirin in water and potassium citrate rather than swallow the aspirin tablets; less gastrointestinal reaction has been noted. *Salysal* (salicylic acid) does not cause gastrointestinal irritation.

Sensitivity to aspirin is probably the commonest form of drug allergy.[11,106] Reactions may include asthma, urticaria, angioneurotic edema, vasomotor rhinitis, abdominal cramps, purpura and pain simulating angina pectoris. *Deaths may occur following the administration of aspirin due to sensitivity resulting in severe asthmatic attacks.* This common sensitivity to aspirin is not fully appreciated. The time interval between the administration of aspirin and the onset of symptoms of hypersensitivity varies from ten minutes to two hours. **Aspirin must always be used with caution in the treatment of asthmatic patients and should not be employed when nasal polyps are present** because of the increased sensitivity to this drug of patients having these conditions.

Poisoning from overdosage of acetylsalicylic acid has been reported.[31,73]

Acute intoxication may occur following large doses of sodium salicylate. Symptoms include oliguria, albuminuria, delirium, somnolence, vomiting, coma, deafness and dyspnea.

Methyl salicylate should be restricted to external applications as relatively small amounts have caused death.[33]

Large doses of sodium salicylate may be given by rectum if desired; this does not often cause gastric irritation. Unfortunately, with this method the exact amount of salicylate absorbed is unknown. If the patient is in the hospital, one may give (an hour after an enema) as much as 10 gm. (150 gr.) of sodium salicylate rectally by Murphy drip in 120 cc. (4 ounces) of water, morning and night.[51] This may be increased if necessary.

studied the subject thoroughly and will examine and question the patient carefully each week, as well as perform a complete blood count and urinalysis at each visit. The physician should preferably have had some actual experience with gold therapy at a recognized arthritic clinic. This subject of gold therapy is discussed in detail in a separate chapter.

Aspirin has been used in enormous quantities throughout the world for almost fifty years. The majority of individuals take it with apparent impunity while many seem to have a mild idiosyncrasy to it or other salicylates. In relation to the huge quantities consumed, severe reactions to aspirin are rare. *Deaths from aspirin* have been more frequent in England than in the United States.[34]

Honigsberger reported the development of *nosebleed* in 3 cases *after* taking large doses of *aspirin* and the frequent occurrence of bleeding in patients with rheumatic fever while they are receiving large doses of salicylates.[46] Experimental evidence suggested that if salicylic acid is given over a long period to rats or to human beings, hemorrhages resulted (with a fall in prothrombin levels).[60,69,88] This fall in prothrombin level could be prevented if vitamin K was administered simultaneously with the salicylic acid; however, after the production of hemorrhage by salicylic acid, administration of vitamin K was of little use. Dicumarol, given by mouth, induces a shortage of prothrombin in the blood. Dicumarol can be qualitatively degraded to salicylic acid.[47]

In view of this evidence, *it may be advisable to give the patient prophylactic doses of vitamin K if salicylates are to be administered over a long period in large doses.*

Most evidence indicates that aspirin and salicylates are among the least toxic of active pharmacopœial preparations.[35]

Following the *local application of methyl salicylate ointment,* a counter-irritant action is noted, together with a systemic action which is analgesic and antipyretic. Drasche showed in 1876 that salicylates appeared in the urine almost immediately after application of an alcoholic solution of salicylic acid to the skin.[30] Other workers have confirmed the rapid percutaneous absorption of salicylic acid.[10] Inasmuch as salicylic acid itself is too irritant for use in inunctions, several of its esters were used and were found to be more rapidly absorbed from the skin; methyl salicylate is the drug used in this manner at present.[48,86]

Workers have found from *0.5 to 9 per cent of applied methyl salicylate* to be *absorbed from the skin* while others found the absorption to be considerably delayed.[41]

Beutner and his associates made a total of 103 inunctions on 86 patients. They determined the salicylate excretion after inunction of methyl salicylate and compared it to that occurring after the oral administration of 0.3 gm. of aspirin. The methyl salicylate was prepared in various vehicles with a 20 per cent salicylate content. The type of ointment used was found to have little influence upon the amount of methyl salicylate absorbed. Ten grams of inunction were used each time.[7]

It was found that the excretion of salicylates in the urine was complete in almost all cases in twelve hours following either oral administration of aspirin or the use of the inunction. Urine was analyzed quantitatively for salicylic acid. An average of 66 mg. of salicylic acid appeared in the urine during twelve hours following administration of 0.3 gram of aspirin; *from 48 to 90 mg. of salicylic acid appeared in the urine during the twelve hours following the application of 2 grams of methyl salicylate* (contained in 10 grams of an ointment) which was rubbed into the skin, and allowed to remain for at least thirty minutes.

Cohen summarized the literature on **phenacetin** (acetophenetidin) and concluded that there is little or no evidence to justify its inclusion in the category of dangerous drugs. *Phenacetin is a valuable analgesic.* A careful analysis of reported cases of alleged acetophenetidin intoxication showed that erroneous inclusion of cases actually due to allergy, overdosage, etc. have created a false impression of the toxicity of this substance. Phenacetin had been stigmatized as a dangerous drug largely on the basis of its relation to and derivation from aniline.

In small doses, phenacetin is an efficient analgesic for mild rheumatic pains, headache, etc.; the average dose for this purpose is 0.3 gm. (gr. v) three to five times a day. Aspirin and other drugs have replaced it as an antipyretic as it requires large doses of phenacetin for its antipyretic action. Following the paper of Morgan and his associates in 1909 on the untoward effects of antipyrin, acetanilid and phenacetin, many of the potential dangers of these drugs were widely publicized.[71] Cohen's re-analysis of the toxic reactions from phenacetin in the reported cases shows that *in ordinary doses phenacetin is a valuable analgesic and should not be included in the category of dangerous drugs.*

IRON AND TRANSFUSIONS

Most patients with rheumatoid arthritis are anemic. This is usually a hypochromic (secondary) anemia, which may result from the arthritis *per se* (as an infection), from a deficient diet or from some other totally unrelated cause. In a given patient, it should not be assumed that the arthritis is the sole cause of the anemia; every effort should be made to locate a source of bleeding, a history of inadequate diet or other cause. A hemoglobin estimation should be done routinely on all patients with arthritis; *anemia practically never exists if the hemoglobin percentage is normal.*

Hartung has found that **blood transfusions** repeated each week for ten to twelve weeks were definitely beneficial in rheumatoid arthritis, resulting in remissions in some cases. Copeman has also stressed the value of blood transfusions in rheumatoid arthritis.[20,42] Repeated blood transfusions are not completely without danger. In one large series of 3500 transfusions, 7 deaths were found to be directly attributable to the transfusions.[27] Five of these patients died of renal insufficiency and 2 of pulmonary edema.

Pointers of value in attempting to prevent such reactions include:

Use typing sera of high titer.

Discontinue the transfusion at once if any unusual symptom occurs.

Alkalinize the urine prior to transfusion; this prevents obstruction of the renal tubules by hemoglobin (as shown in animal experiments).

The use of universal donors may be dangerous unless the agglutinins of the donor have been titrated and have been found to be weak.

Patients with cardiac damage and those with elevation of the blood urea nitrogen apparently do not tolerate blood transfusions well.

Test for the Rh factor in all patients when repeated transfusions are planned.

Some patients with rheumatoid arthritis have been subjected to venesections, followed by a *transfusion of this blood after it has been exposed to ultra-violet irradiation*. There have appeared in the literature no carefully controlled experiments on this subject. The method may have been instituted as a mild foreign protein therapy or its use may have arisen following the reports of massive vitamin D therapy in rheumatoid arthritis.

Treatment of Anemia in Arthritis

Search for and correct any possible cause (bleeding hemorrhoids, deficiency of iron or vitamin C in the diet, peptic ulcer, etc.).

In many patients, as the rheumatoid arthritis improves, the hemoglobin level returns towards normal even without iron medication.

Adequate intake of iron, such as *ferrous sulfate*, 0.2 gm. (gr. iii), or ferrous gluconate, 0.3 gm. (gr. v), four times per day. Other preparations which may be employed include *iron and ammonium citrate*, 6 gm. daily (usually given as a 20 per cent solution); reduced iron, 3 gm. (45 gr.) a day; freshly prepared Blaud's pills (4 pills, three times a day). Ferrous sulfate or gluconate do not often cause indigestion; they are two of the most effective of all iron preparations. Only small doses are required for adequate results. However, *any preparation of iron may cause indigestion and severe constipation.* Iron occasionally gives rise to diarrhea. A useful method of prescribing iron in solution is the following.[28]

℞—Ferri et Ammon. Cit. . . .	45	℥iss
Syr. Tolu	45	℥iss
Aqua q.s. ad	360	℥xij

S.—One tablespoonful three times a day, after meals, with water (taken through a straw).

Inasmuch as many of these patients have low or absent free HCl in the stomach, it is advisable to supply this. Hydrochloric acid may assist in the absorption of iron from the upper part of the small intestine. A suggested method of prescribing such acid is as follows:[29]

Add 4 cc. (one teaspoonful) of dilute HCl to a glass of water, into which has been placed the juice and pulp of an orange and a teaspoonful of sugar.

Three such glasses should be drunk daily with meals.

Rinse the mouth with water containing a small amount (½ teaspoonful) of sodium bicarbonate as soon as possible following the acid medication to prevent harmful effects upon the teeth.

Treatment of Anemia in Arthritis—(*Continued*)

In some patients, iron cannot be tolerated by mouth. In these individuals, a change may be made to an arsenical preparation such as:

Sodium cacodylate, 15 mg. (¼ gr.) three times a day by mouth.

Fowler's solution—from 0.3 to 0.6 cc. (5 to 10 minims) several times a day as tolerated (by the skin and gastrointestinal tract).

Hypodermic injections of preparations containing iron or iron and arsenic. An injection may be given two or three times each week if iron or arsenic cannot be tolerated by mouth; this is necessary in only a few individuals. The amount of iron which can be injected is very small and such therapy is not advised in anemias unless the oral administration is not possible; *iron should not be given intravenously as this procedure is dangerous.*

When it is possible, *blood transfusions are often used in rheumatoid arthritis.* In addition to its red cell and hemoglobin content the blood may exert a beneficial non-specific effect and possibly aid in stimulating antibody formation.[21] It is possible that the blood of normal individuals may contain some (as yet) unknown antibodies which may be beneficial in rheumatoid arthritis. It also assists in raising the serum protein level to normal in those persons whose diet may have been deficient in protein. Small transfusions (250 to 300 cc.) may be given every two or three days until the hemoglobin has been brought up to 85 per cent or more. Some clinicians prescribe a small transfusion (250 cc.) each week for ten to twelve weeks in patients with severe rheumatoid arthritis. If the patient is markedly anemic (45 per cent or less) transfusions are often advised before attempting to remove any evident foci of infection. In some individuals, *two or three small transfusions may initiate the recovery process*, even when only a slight to moderate degree of anemia is present.

LIVER EXTRACT

Crude liver extract does not cure rheumatoid arthritis. Although this may be a valuable method of supplementing the vitamin B and other fractions in the undernourished patient with rheumatoid arthritis, the results of its use in this disease have been disappointing, except in a few cases in which dramatic improvement has been reported.

Davis reported on the use of a crude liver extract (Campolon, Winthrop) in a series of 25 patients with gout.[25] He described his results as favorable. He used crude liver extract in these patients because it was thought that there might be some deficiency in a particular fraction of the liver, which substance might be removed by concentrating the liver fraction. Previous workers had shown that *preparations of concentrated liver were ineffective both in gout and rheumatoid arthritis* and that acute attacks of gout were precipitated in some individuals by the administration of concentrated liver preparations. Davis began his therapy with an average of 1 or 2 cc. of crude liver extract intramuscularly, giving injections daily and increasing the dose up to 10 cc. The injections were then reduced to one or two a week, and were continued for a protracted period. At present, most workers have not been able to confirm Davis' results in gouty patients

using crude liver extract. One must be extremely cautious in the use of crude liver extract as untoward reactions are by no means rare, and one may encounter generalized urticaria, severe local reactions and bronchial asthma in some individuals.

FOREIGN PROTEIN THERAPY

Foreign protein therapy produces remissions only occasionally in patients with rheumatoid arthritis, and is rarely curative.[43] It is most useful after the recovery phase has come to a standstill and marked stiffness remains as an outstanding symptom. The contraindications are definite and are defined later.

Foreign Protein Therapy in Rheumatoid Arthritis

Substances used: typhoid vaccine, Coley's fluid, boiled milk (Aolan).

Contraindications:[18] Pregnancy; chronic alcoholism; renal disease or marked hypertension; serious valvular, coronary artery or myocardial heart disease; diabetes; obvious circulatory weakness; weakened, markedly undernourished or seriously ill patients. *No patient should be subjected to protein shock unless it is evident that he can bear the additional strain.*

Doses must be minimal in: Patients with a history of asthma or other allergic disease, or in the feeble or aged.

Indications: Patients with rheumatoid arthritis whose symptoms during the recovery stage have reached a standstill and stiffness remains as one of the outstanding symptoms, especially in the atypical (focal infection) type of rheumatoid arthritis;[89] failure of all other forms of conservative therapy.

Prior to fever therapy with typhoid vaccine, the patient should be given at least 8 to 10 gm. of sodium chloride per day in enteric coated tablets, together with an abundance of fluids. On the day of the treatment, the patient should be placed between blankets and should have a total of four hot water bottles or several electric pads on or near his extremities for several hours prior to the injection of typhoid antigen or vaccine. These should be kept warm during the entire treatment. No aspirin or other salicylates should be given on the day of the treatment or within twelve hours prior to this. If the temperature rises too high, the heating pads are removed, an ice cap is applied to the head, large amounts of aspirin may be given and tepid sponges may be used if needed, to reduce the fever. This is seldom necessary.

Dose for adults: 10 to 25 million (dead) typhoid bacilli given *intravenously;* second dose—25 to 50 million given after twenty-four hours of normal temperature (and not oftener than every four or five days); third dose—40 to 100 million; fourth dose—60 to 150 million; fifth dose—80 to 200 million. If the patient has a severe reaction to any dose of typhoid vaccine, it is often advisable to repeat the same dose (without increase) at subsequent injections (unless febrile paroxysms do not occur). *If no improvement has occurred following 4 injections, discontinue.* If improvement continues, a series of 5 or 6 such injections may be given unless the patient is unduly worn out by the febrile responses. Always use typhoid vaccine made by the same drug firm so that you know the result to be expected.

Foreign Protein Therapy in Rheumatoid Arthritis—(*Continued*)

In some patients, a dose as low as 10 million organisms will produce the desired febrile response. Occasionally, it will not be necessary to markedly (if at all) increase subsequent doses for a therapeutic effect. *The dose varies greatly with each patient.*

Some workers prefer to use the *typhoid H antigen.* The dose of the H antigen required to produce a febrile response is often double that of the whole typhoid organism.

If an adequate rise in temperature is not obtained, or if the temperature is not rising within several hours, the dose of typhoid antigen or *vaccine may be repeated* once or even twice.

When individuals have been immunized previously with typhoid vaccine, it is extremely difficult (and often impossible) to obtain a marked rise in temperature by intravenous injections of typhoid antigen or vaccine.

Some physicians prefer *Coley's fluid* as a foreign protein and begin with 0.01 cc. given intramuscularly; others use boiled milk (or *Aolan,* a somewhat similar preparation), giving from 1 to 10 cc. intramuscularly; some recommend *intramuscular* injections of from 5 to 10 cc. of the *patient's own blood,* withdrawn just before being injected. If one wishes to use milk as foreign protein therapy, ordinary skimmed milk may be boiled for ten minutes. In adults, the first dose usually consists of 5 cc. injected into the gluteal muscles (when the milk has been cooled to body temperature). This may be increased by several cc. at each injection to a maximum of 12 to 15 cc.

The thought underlying foreign protein therapy is the stimulation of antibody production, leukocytosis and the production of proteolytic ferments in the blood.

The chill following *typhoid vaccine* is the most constant and may be fairly severe. *This is the most commonly used foreign protein* as it is readily available and accurate dosage is easily obtained. Reactions from milk are less severe but may be more prolonged; it may be used if a chill is to be avoided.

Reactions from milk are less constant as they may depend to some degree upon the bacterial content of the milk.

Reactions from Coley's fluid are inconstant, being severe at times, and absent at others.

Reactions are usually diphasic, resulting in an intensification of the disease both generally and locally, and later, a constructive phase in which there occurs general euphoria and diminution of the disease symptoms; in general, the more severe the first phase, the greater the clinical benefit.

Therapeutic results should not be expected when the organism is no longer capable of response to stimulation; repeated injections at very short intervals may fail to produce a reaction and may actually do harm.[79]

The use of typhoid vaccine as non-specific therapy followed its intravenous use[54,101] in the treatment of typhoid fever; equally good results were obtained in typhoid fever with colon bacillus vaccine[55] and later with albumose[63] given intravenously, or boiled milk[87] injected intramuscularly. Typhoid vaccine was given intravenously in this country for fever therapy by Miller and Lusk in 1916; these workers employed it in the treatment of arthritis.

Opinions are divided concerning the usefulness of this procedure. Good

results have been reported occasionally in rheumatoid arthritis following intravenous injections of colon bacillus vaccine,[57] casein[62] and typhoid vaccine.[15] Others obtained "cures" in only 6 of 70 patients, all being less than two years duration.[26] Foreign protein therapy is not advised in an undernourished patient with rheumatoid arthritis; it has been compared to "the technique of opening an abscess by means of a kick with a football shoe."[21]

Some of the *substances* employed *for non-specific protein therapy* include bacterial vaccines (especially typhoid); Coley's fluid and tuberculin (bacterial extracts); milk, casein and egg albumin; normal human serum or diphtheria antitoxin; peptones, proteose, albumose and histamine; and enzymes and tissue extracts. Malarial infection, bee venom and possibly some preparations of colloidal sulfur may be examples of such therapy.

Although symptoms following typhoid vaccine vary markedly, the usual reaction to the first injection is a chill or chilliness from thirty to sixty minutes following the injection, general aches and pains, followed by a fever of from 101° to 105° F., beginning from two to four hours after injection. The febrile period may last from three to fourteen hours. One may see a secondary spike of fever after the temperature has returned to normal, five to eight hours after the injection of typhoid vaccine. This secondary rise of temperature persists for two to four hours. A transient polymorphonuclear leukopenia occurs immediately after the injection, followed by a polymorphonuclear leukocytosis (beginning with the chill); this may persist for six to twelve hours.

Results to be Expected From Foreign Protein Therapy.—A small number of patients with rheumatoid arthritis will obtain symptomatic improvement following such therapy. In about 10 per cent the improvement may be permanent; in the remainder, the benefit persists for only several weeks or months. The administration of intravenous typhoid vaccine to a markedly underweight, very anemic, weakened patient is inadvisable.

We no longer use foreign protein therapy in rheumatoid arthritis except in the fairly well nourished individual who has improved moderately with other therapy, but has then remained at a standstill, with stiffness as an important symptom. In such individuals, mild fever therapy with typhoid antigen may hasten the recovery process.

Mortality Following Typhoid Vaccine Given Intravenously.—In a series of 2500 patients given intravenous typhoid vaccine at the Mayo Clinic, death occurred in 3 cases.[44] There were 14 instances of untoward reaction such as appendicitis, cholecystitis, enteritis, pleurisy, iritis, glaucoma, pericarditis, adenitis, renal insufficiency and vascular thromboses.

PROCAINE

Procaine has been employed by various workers in the field of arthritis. In traumatic arthritis, procaine has been suggested actively and prophylactically.[75] Procaine is in wide use in the treatment of sprains and contusions. Others have reported encouraging results by infiltrating the

ligaments and muscles about joints which are affected by degenerative joint disease.[36,99]

Procaine eliminates the pain impulses and overcomes the local hyperemia due to vasodilatation. Leriche believed that procaine was of value in these conditions because it suppressed by its local anesthetic action the nerve irritation which was inaugurated by trauma. There is a rich nerve supply particularly in ligaments in the region of an articulation. Following trauma, these nerves are violently excited resulting in a disturbed vasomotor functional state clinically appearing as muscular spasm, pain, tenderness and loss of motion. Procaine eliminates the pain impulses that emanate in automatic waves from the area of trauma, resulting in a return to the normal vasomotor tone at least temporarily.

Gorrell injected *procaine, nupercaine* or *eucupin* into 295 patients *to counteract pain* (due mainly to sprained ankle, degenerative joint disease or "rheumatic conditions").[38] A trigger point was sought before making the injection. If the patient did not wince when pressure was made on the point, this was probably not the one sought. When pressure with the finger tip caused the patient to say, "That is my pain," relief was predicted confidently. Each area with wincing tenderness was injected. Procaine relieved the great majority of muscle, fascia and ligamentous pains. Although the relief was only temporary in many instances, it was often gratifying. Gorrell emphasized that although the injection afforded temporary relief, the organic disorder causing the pain must be treated to obtain lasting relief.

Fenz has summarized the use of local anesthesia in the treatment of rheumatoid arthritis, bursitis, neuritis, neuralgia and spondylitis. The patients were given an average of 3 or 4 procaine infiltrations resulting in freedom or almost complete freedom from pain in 70 per cent of cases; only 7 per cent were said to be totally unimproved.[37]

PROSTIGMIN

The *Kenny concept and treatment of poliomyelitis* aroused considerable interest in the *muscle spasm* which is said to be characteristic of the disease, and against which the treatment is particularly directed.[24] The muscles which are in spasm are painful on pressure or stretch; this condition may lead to dysfunction and deformity. Although it is claimed that *spasm* is present in every case, there *seems to be no direct relation between it and motor paralysis*.

Present investigators agree that the *spasm* is not due primarily to a process within the muscle but that it *is a reflex phenomenon*. Bouman and Schwartz concluded that in poliomyelitis, impulses from higher centers which are inhibitory of muscular contraction, are blocked in very close proximity to the motor neurons.[9] Kabat and Knapp reported that in *acute and subacute poliomyelitis spinal anesthesia* (with procaine) *markedly relaxed the muscular spasm*.[50] In one individual, intravenous injection of beta-erythroidine hydrochloride (which blocks the myoneural junction)

was followed by relaxation of muscle spasm and of pain on passive motion. In 68 cases of poliomyelitis, an internuncial cord lesion was found almost constantly; in 26 of these cases, the anterior horn cells were relatively normal. Chronaxia measurements gave no evidence of relation between muscle spasm and damage to the neurons in the anterior horns.

Such investigations tend to show that *in poliomyelitis, the spasm is apparently neurogenic,* due to the blocking of reflexes by lesions in the gray matter of the cord.

Kabat and Knapp reported that *subcutaneous injections of prostigmin significantly increased the range of passive motion,* decreased or eliminated deformities in some instances by relaxation of hypertonus and in some cases improved active motion in 20 patients in the subacute stage of *poliomyelitis.* They noted that *muscle spasm lessened* more rapidly when prostigmin was added to the Kenny routine. Prostigmin appeared in these cases to decrease skeletal muscle hypertonus and muscle spasm (proprioceptive reflex hyperirritability). *The effects were evident an hour after the subcutaneous administration of the drug.* Kabat and Knapp believed that these actions of prostigmin depended on alteration of function of synapses in the spinal cord and were not antagonized by atropine.[49]

Prostigmin is believed to depress the function of the enzyme cholinesterase, which latter is responsible for the rapid splitting of acetylcholine, rendering it inactive. By such inhibition of cholinesterase prostigmin permits acetylcholine to accumulate to a greater extent than normal at the synapses, parasympathetic nerve endings, myoneural junctions and in the blood.[93] *Prostigmin relieves fatigue of skeletal muscles in myasthenia gravis, but aggravates the muscular rigidity and tremor in postencephalitic Parkinson's disease.*[70] These actions of prostigmin have been attributed to the inhibition of cholinesterase at the myoneural junction of skeletal muscle.

The *simultaneous administration of atropine* apparently does not inhibit any important effect of prostigmin on muscle function but *does eliminate those unpleasant parasympathetic effects* permitting the giving of larger doses of prostigmin resulting in more pronounced muscular effects (in poliomyelitis) without unpleasant symptoms.

Relaxation of muscle spasm was the most significant effect of prostigmin in patients with poliomyelitis. These changes in muscle function apparently do not result from the action of prostigmin on the myoneural junctions in skeletal muscle as the accumulation of acetylcholine due to inhibition of cholinesterase at the myoneural junction would increase hypertonus of skeletal muscle.

If prostigmin is injected intraspinally into patients with poliomyelitis, a greater degree of muscular relaxation results than that which follows subcutaneous injection of the drug. The effects on muscle function preceded any significant absorption of the drug into the general circulation, pointing to the spinal cord as the locus of action of the drug. Kremer recently demonstrated a depression of spasticity and deep reflexes following intrathecal injection of prostigmin in man.[56]

In poliomyelitis, Kabat and Knapp gave subcutaneous injections of 1.5 to 2 mg. of prostigmin methylsulfate together with 0.6 mg. (gr. 1/100) of atropine sulfate in adults. Subcutaneous administration was found to be more effective than oral medication. In most cases, the drug was given orally, in addition, in doses which were gradually built up to 45 mg. of prostigmin bromide and atropine sulfate 0.6 mg. (gr. 1/100) three times a day (for adults, with smaller doses for children). Unpleasant symptoms were avoided by carefully adjusting the balance of prostigmin and atropine in the individual patient. *Some of the patients took these doses for two months without harmful or unpleasant effects* and almost all of these patients showed some improvement in muscle function from the drug.

The muscular spasm and atrophy in rheumatoid arthritis bear some resemblance to these symptoms in poliomyelitis. In rheumatoid arthritis, the muscular spasm, if untreated, leads to deformities, fatigue and pain.

Prostigmin is a synthetic compound possessing the desirable properties of physostigmine (eserine) with supposedly very few of its toxic by-effects. It has been used to restore normal tone and peristalsis in the smooth muscle of the intestine by its action on the parasympathetic innervation; this action has found usefulness in preventing and terminating abdominal distention and postoperative intestinal atony. In some cases, it may cause an excessive response with abdominal cramps. It has also been used postoperatively to overcome urinary retention and atony of the bladder. The action of prostigmin is so efficient and selective in *myasthenia gravis* that its action has been used as a diagnostic test in this disease. The weakened and flaccid muscles of the individual with myasthenia gravis respond within a few minutes following the subcutaneous administration of the drug.

Prostigmin is marketed in tablet and ampoule form. The tablets contain 15 mg. of prostigmin bromide (in tubes of 20 or bottles of 100). Ampoules of prostigmin methylsulfate are sold in two strengths, 1 to 2000 and 1 to 4000; both are 1 cc. ampoules, sold in boxes of 12 or more. There is also available an ampoule which contains prostigmin methylsulfate 1.5 mg. together with 0.6 mg. (gr. 1/100) of atropine sulfate.

Experimentally, prostigmin has been used as a test for early pregnancy; in the treatment of delayed menstruation; in atrophic rhinitis; in paroxysmal tachycardia; in the elimination of gas shadows in fluoroscopy and roentgenography; in expulsion of ureteral calculi and in some cases of tinnitus and deafness, as well as in certain peripheral vascular disorders (especially where vascular spasm has caused blanching and sensory disturbances of the fingers, toes or limb).

Trommer and Cohen observed *a relaxation of muscle spasm* in 13 of 19 cases of *rheumatoid arthritis* following the administration of prostigmin. No demonstrable effect was noted on the pain produced by the joint lesion but active and passive motion could be carried out more easily and with less pain.

Prostigmin acted rapidly (within three to fifteen minutes) when given subcutaneously; in some cases, the action persisted for several days. A more

prolonged effect was obtained in the interim between injections when prostigmin bromide was given orally along with the subcutaneous administration.[103]

In the patients treated by Trommer and Cohen by prostigmin, patients were selected who had a maximum of deformity with loss of function, but with a minimum of active joint involvement. These patients were usually given subcutaneously 1 cc. of prostigmin methylsulfate (neostigmine methylsulfate) 1 to 2000 (0.5 mg.) and 0.6 mg. (gr. 1/100) of atropine sulfate every other day. In addition, the patients were given orally from 7.5 to 45 mg. of prostigmin bromide, together with from 0.6 to 1.2 cc. of tincture of belladonna daily.

These workers *believed that prostigmin relieved the muscular spasm in rheumatoid arthritis, and thereby relieved pain.* It was suggested that if muscular spasm exists, these patients should receive from 1 to 2 cc. of prostigmin methylsulfate 1 to 2000 together with suitable amounts of atropine subcutaneously three to four times per week, together with from 15 to 30 mg. of prostigmin bromide orally three times a day. The dose of atropine or tincture of belladonna must be so regulated as to prevent any untoward side effects of the prostigmin.

We have used prostigmin and atropine in a number of patients with rheumatoid arthritis. *Patients vary greatly in their reaction to prostigmin.* Some experience marked *dizziness* and the drug must be discontinued. In some, even when atropine is administered, *severe abdominal cramps* occur with *precipitous bowel movements* which do not give the patient much warning before evacuation is essential. In some individuals, there is a definite sense of improvement in strength and general well being following the prostigmin. In one patient, an acute flare-up of rheumatoid arthritis occurred following three days of prostigmin medication; it is of course impossible to say whether the prostigmin contributed to this. Other patients have noticed no definite change following the administration of the prostigmin. We have not been favorably impressed with the results of prostigmin therapy in patients with rheumatoid arthritis. There is no definite improvement in the rheumatoid arthritis and there is very little evidence in our work or in other recent work to suggest that this drug will be of any permanent value in these patients.

SPLENECTOMY

Because of the finding of an enlarged spleen in some patients with rheumatoid arthritis, it has been suggested that splenectomy may be of value in relieving the arthritic phenomena. There is no rationale for splenectomy in rheumatoid arthritis. Steinberg concluded that splenectomy did not directly alter the course of rheumatoid arthritis.[94]

Chevallier, in 1930, suggested that splenectomy might bring about a regression in some of the arthropathies.[17] Hanrahan and Miller in 1932 noted marked improvement in the joints of their patient (female—aged fifty years); this patient had the chronic arthritis, large liver and spleen, slight secondary anemia and leukopenia (1000 WBC) described by Felty.[40]

Following splenectomy, the affected joints became less painful and stiff and the WBC rose to 10,000 per c.mm. The improvement was maintained five months after the operation. No specific changes were noted microscopically in the spleen. Craven's case of rheumatoid arthritis in a woman of forty years (with the Felty syndrome) showed striking subjective improvement with increased joint mobility following splenectomy; however, little permanent improvement was noted eight months later.[23] Both Hanrahan's and Craven's patients died within eighteen months following the splenectomy. The case of Loeper and his associates, of rheumatoid arthritis in a woman of twenty-eight years, also showed immediate marked subjective improvement after splenectomy, but died thirty-five days after the operation because of bronchopneumonia.[61] The woman aged forty-two years with advanced rheumatoid arthritis of seven years duration reported by Villaret and others improved markedly following splenectomy.[105] Bach and Savage described 3 cases in which splenectomy was performed in rheumatoid arthritis; amyloid change was found in 2 of these.[3] The patients have been followed for at least nine months. Marked clinical improvement followed the operation in 2 instances and the disease became quiescent in the third.

Surgical intervention such as cholecystectomy, tonsillectomy or removal of focal infection *may be followed by transient clinical improvement in the patient with rheumatoid arthritis.* Some have claimed that this was due to a non-specific protein shock mechanism with stimulation of the reticuloendothelial system, or to the period of rest following the surgery.

AMPHETAMINE (BENZEDRINE)

Patients with degenerative joint disease who are markedly overweight frequently inquire concerning the use of *amphetamine sulfate* (*benzedrine sulfate*) to aid in the control of their obesity. This drug has been accepted in New and Non-official Remedies with well defined uses including the treatment of narcolepsy and certain depressions accompanying psychopathic conditions. The Council concluded that *in view of the dangerous effects which might come from exploitation of this drug for use in obesity, it disapproved of general recognition of claims for use of amphetamine in obesity.* It was thought that whatever effectiveness the drug might have might possibly be due to undesirable properties.[22]

NEO-ARSPHENAMINE

In some cases of rheumatoid arthritis, improvement may follow the intravenous injection of neo-arsphenamine.[39] With the introduction of gold salts, neo-arsphenamine is now seldom used in rheumatoid arthritis. It is worth a trial in individuals who can not continue gold therapy. Arsenic has also been used with benefit in psoriatic arthritis. The reported effects of arsenic include stimulation of hematopoiesis and general metabolism, vasodilatation and in some cases an amelioration of discomfort.[77,78] The exact manner in which the beneficial effects of arsenic are obtained is unknown.

Neo-arsphenamine alone will not cure rheumatoid arthritis. Although slight improvement may follow the first or second injection of this drug, subsequent injections may seem to be of no value in some of these patients. It is merely an empirical adjunct to other forms of therapy. One may give 0.2 to 0.3 gm. intravenously once a week for 10 injections (if there are no contraindications to such therapy).

Precautions in the Administration of Neo-arsphenamine[96]

Each dose must be freshly prepared and must not remain in solution more than twenty minutes before being used.

The ampoule of neo-arsphenamine should be immersed in alcohol before being opened to be sure that it is air tight.

The drug should be dissolved carefully in sterile water or special solvent so that no residue remains; it should go into solution within two minutes.

The *solution must not be aërated* as this increases its toxicity; shaking or allowing the solution to stand does likewise.

The *injection should be made slowly*, taking at least a full minute.

Caution must be taken that *no leakage occurs into* the subcutaneous *tissues;* otherwise, sloughing may occur.

If a temperature above 99° F. is present, the dose is reduced to 0.15 gm.; if the patient's temperature is above 100° F. no injections are given.

Contraindications. Hepatitis, cirrhosis of the liver, coronary artery heart disease, aneurysm, acute pulmonary infection (including tuberculosis), nephritis or nephrosis, allergic history, history of previous dermatitis or eczema, or inflammatory lesions of the brain or cord.

Reactions which may occur include: nausea, vomiting, headache, diarrhea, fever, chills, abdominal pain, and less frequently, flushing, backache, pains in the legs and dizziness. Later reactions include stomatitis, urticaria, Herxheimer reaction, purpura hemorrhagica, exfoliative dermatitis and acute yellow atrophy of the liver.

Adrenalin should always be on hand for hypodermic use should a nitritoid crisis occur (flushing, choking, coughing, dyspnea, wheezing, unconsciousness, rapid, thready pulse, etc.).

Always look for jaundice, inquire concerning itching or skin eruption, and record the temperature before giving neo-arsphenamine injections. No injections are given if there is icterus, a skin eruption or a temperature above 100° F.

VENOM

Cobra Venom.—Talkov and Bauer found cobra venom of no value in relieving pain in rheumatoid arthritis.[98] Cobra venom has been used in arthralgia and for the relief of intractable pain.[85,95] Scientific experiments with cobra venom at the Pasteur Institute showed that it was useful in relieving severe pains accompanying malignancy. No demonstrable damage results to the circulatory, renal or hepatic tissues following moderate doses (5 to 10 mouse units). There seems to be a wide margin of safety.

Cobra venom is said to relieve pain by a sedative action on the higher centers of the brain (as does morphine), but induces analgesia more slowly

than morphine, requiring two or three days of successive injections. The analgesia, when induced, persists for longer periods than that due to morphine.

An ampoule (5 mouse units—manufactured by Hynson, Westcott and Dunning of Baltimore) is given *intramuscularly* daily until an analgesic effect is obtained, and then two or three times a week in order to keep the patient comfortable. Such injections may be continued for months.[102]

Other Snake Venoms.—Venom from several species of snakes including the cobra, pit viper, water moccasin and copperhead, have been employed by various workers in the treatment of arthritis and allied conditions. Physicians have not been impressed by their virtues although laymen have long favored ointments of snake oil and the stings of bees in the treatment of rheumatic disorders. **Most qualified workers at present find a lack of scientific evidence for any of these venoms in the treatment of rheumatic conditions.** *None of the snake venoms have an established place in the treatment of arthritis.*[83]

Steinbrocker and his associates reported that about 60 per cent of their patients with "arthralgias and related conditions" obtained "more or less symptomatic relief" when treated with neurotoxic venom.[95]

Burkhardt employed viper extract intracutaneously in 64 patients.[12] Almost all of the 17 patients with various types of neuralgia experienced complete alleviation following a short course of therapy. Seventy-five per cent of those with rheumatoid arthritis showed constitutional improvement and improvement in the joints locally, with decreased pain and swelling and a reduction in the sedimentation rate.

Butler noted marked improvement in 90 per cent and symptomatic cures in at least 85 per cent of patients with "arthritis, arthralgia, neuritis and allied affections" by the intradermal injection of moccasin venom (Moccasin "Venomin," obtained from the Venomin Company, Lorain, Ohio).[13] The venom was obtained from freshly captured snakes; this was immediately dried with vacuum under carbon dioxide snow freezing. The author began his treatments with 0.05 cc. of the weakest dilution (Vial "A") always given intradermally.

Snakes have long been a symbol of the art of healing, exemplified mainly by the pictures of Aesculapius and Hygeia and by the Roman caduceus. Various snake venoms have been commented upon in recent literature. It has been stated that the venom of the European sand adder is superior to cobra venom in analgesic effect.[12] Venom of the pit vipers such as the copperhead, water moccasin and rattlesnake have also been discussed.

We have not been able to find in the literature carefully controlled experimental work showing any particular value from snake venoms in rheumatoid arthritis. Their use in this disease is certainly not advocated in the light of our present knowledge.

CALCIUM

Copeman has used intravenous injections of 5 to 10 cc. of 10 per cent *calcium gluconate* two or three times a week, especially in intervals between

gold therapy.[21] He thinks it has decreased the liability to the development of late evidence of intolerance to gold.

Some workers believe that muscular spasm may be lessened in rheumatoid arthritis by intramuscular injections of calcium gluconate.

XANTHIUM

Krantz and his associates have discussed the use of an extract of xanthium burs in various forms of arthralgias.[53] Their attention was called to the use of a tea of xanthium by an Indian guide in the Maine woods; the tea was used in the treatment of arthralgias.

Xanthium (or cockle bur or spiny clotbur) is a common weed which was introduced into this country from Europe. The burs used in this investigation were Xanthium spinosum. The extract of xanthium burs was found to be innocuous when administered to Macacus rhesus monkeys. The depressor response found following xanthine extracts was attributed to choline which was identified (or to some precursor of this amine).

Xanthium did not produce analgesia in rats with experiments conducted according to the technic of Macht.[67]

Mecholyl (beta-methylacetylcholine chloride) has been used in the treatment of some arthralgias and the benefit obtained was due in part to the mechanism of peripheral dilatation. "Favorable results" have been noted in 60 per cent of patients with "arthritis" (200 patients) in a preliminary study.[52] We have seen only 2 patients who received tablets of Xanthium. In one young girl there was some alleviation of discomfort; in a woman aged thirty-five years, the drug had no effect.

ANTI-RETICULAR CYTOTOXIC SERUM (ACS)

Various investigators in Russia have claimed that small doses of an "anti-reticular cytotoxic serum" greatly stimulate mesenchymal cells, while large doses block these cells. The anti-reticular cytotoxic serum (ACS) is usually prepared for use in humans by immunizing an animal (horse, donkey, sheep, goat) with antigen taken from the spleen and rib marrow of a human cadaver (in cases who have had no infectious diseases) not later than six to ten hours after death.[8]

These individuals stress the "functional condition of the connective tissue" of the body; the local and general reactivity of the body in the course of disease is said to depend to a considerable extent upon the connective tissue. This connective tissue is viewed as forming a peculiar physiologic system with complex functional manifestations.

The functions of this system include:

1. A *trophic function*, which regulates cellular metabolism, nutrition and acts as a depot; disorders in this function may contribute to precocious senescence.
2. A *plastic function*, aiding in the regeneration of tissues and in the healing of wounds, including fractures.

3. *Protective* functions by phagocytic and other activity against infection and development of new growths.
4. An *autoregulative function*, through various internal secretions.
5. A *mechanical function*, assisting the osseous and elastic skeleton.

It is thought that the health and age of the body are determined largely by the physiologic conditions and age changes in its connective tissue.

Mechnikov, in 1900, suggested that it might be possible, with small doses of a cytotoxic serum, to strengthen the functions of the most valuable elements in the body and to weaken the aggressive tendencies of the phagocytes.

Bogomolets proposed an anti-reticular cytotoxic serum to affect the condition of the physiologic system of the connective tissue. *Injections of this serum into a rabbit markedly increased the concentrations of agglutinins and hemolysins in the blood. In mice, "ACS" saved the lives of 70 per cent when infected with recurrent typhus* (which caused up to 100 per cent mortality in control mice).

"ACS" has been used in Russia with apparent success in highly diversified diseases. It is thought that it is capable of stimulating the functions of the connective tissue only if used in small doses (averaging 0.05 to 0.1 cc.) as large doses usually inhibit the system. It is hypothesized that "ACS" acts by stimulating the trophic, protective and plastic functions of the connective tissue system. The earliest manifestations, usually seen after the second injection of serum, consist of a relative lymphocytosis, dilatation of the capillaries and increased permeability of the "hemato-parenchymal barrier" (which latter is the minute or crude dispersion of connective tissue fibrils which fills the space between the capillaries and the parenchymal cells).

"ACS" may increase the amount of complement in the blood as well as the opsonic index and the phagocytic activity of the leukocytes. Following adequate doses of "ACS," the sedimentation rate is said to fall to normal.

In the *preparation of "ACS"* for use in humans, one should refer to Marchuk's article.[68] Briefly, this consists in preparing an antigen by mincing 5 to 15 gm. of tissue in the proportion of 8 to 9 parts of the spleen of the human cadaver (not later than six to ten hours after death) with one to two parts of marrow. These are ground in a porcelain mortar and a volume of physiologic saline is added equal to five times the weight of the tissues. Half of the resulting suspension is centrifuged at 1000 r.p.m. for three to four minutes. The layer of fat is removed from the supernatant fluid; the remainder then serves as the antigen for intravenous injection while the uncentrifuged antigen is given orally.

The best method of immunizing animals to obtain "ACS" is by intravenous injection. The antigen is injected five to six times in increasing doses at intervals of four to five days and the titer of cytotoxins in the serum is determined by complement fixation eight or nine days after the last injection.

It was reported that: "*In the acute rheumatic process*, a single injection may stop the disease from progressing."[97]

The Conference called by the Institute of Clinical Physiology of the Academy of Sciences of the Ukrainian S.S.R. and by the Ukrainian Institute of Experimental Biology and Pathology of the People's Commissariat of Public Health of the U.S.S.R., held in Ufa July 12–14, 1942, together with delegates from the Moscow, Kiev, Kharkov and Bashkirian medical institutes, as well as evacuation hospitals of different regions and towns, heard over *40 reports* and communications *based on 2500 clinical observations* presented there and passed the resolution that:[32]

"On the basis of over 2500 clinical observations presented at the conference, *the therapeutic effect of anti-reticular cytotoxic serum was clearly established* in war traumatisms and the following diseases: (1) Frostbite and wounds; (2) Infectious diseases (including spotted typhus, puerperal and gynecological sepsis, *rheumatism*, unresolved pneumonia and lung abscesses, tonsillitis); (3) Diseases of the nervous system, including neuritis, meningo-encephalitis, disseminated sclerosis as well as schizophrenia and various psychoses; (4) Eczema, peptic ulcers and ozena."

The Conference regarded it as an "absolute necessity" to use "ACS" extensively in the therapeutic practice of military hospitals and civilian medical institutions.

Bogomolets' "ACS" was first tried on human patients in 1936.[68] Only preliminary observations have been reported on the use of "ACS" in the treatment of "sepsis and *acute rheumatism*," but the results are hopeful and suggest that it may act favorably on other chronic diseases in the second stage of their course. *A warning is issued against the use of the serum*, especially intravenously, *in patients with endocarditis and myocarditis*.

It is stated that the serum is of value in so many diverse diseases because it does not act upon bacteria or their toxins directly, but upon the reactivity of the physiologic system of the connective tissue. The serum is given in infectious diseases to intensify the creation of an active immunity. Bogomolets believes that the basis of the stimulating action of "ACS" is autocatalysis.

The most suitable cadavers for preparation of "ACS" are persons who died a sudden death and possessed no infectious diseases.

Animals may be immunized with the antigen of human marrow and spleen. It is suitable for use when the titer of cytotoxins is 1 to 100 or more (as determined by complement fixation) and the hemolytic titer is not more than 1 to 16.

Adults have been given as much as 0.1 to 0.25 cc. as a stimulating dose, and in some instances, 0.3 cc. It is injected only in a 1 to 10 solution of physiologic saline. If given intravenously, from 1 to 2.5 cc. of the diluted (1 to 10) serum solution may be given; this may be repeated and tripled after an interval of two or three days but the second injection should not be given more than ten days after the first. The same dose is advised for subcutaneous use.

Two days prior to injection, the serum is diluted in sterile physiologic saline (1 to 10); this solution is then poured into ampoules and sealed and

stored in the refrigerator and is warmed to 37° C. in warm water before injection.

The titer of cytotoxins is maintained in the serum for four to six months; in some sera, the titer did not change after one or two years. Total darkness and a temperature of 4° C. are the best conditions for preserving the serum, under which conditions the serum may be preserved for an average of six months. The titer is lowered after two months by light, freezing, room temperature and the addition of preservatives. The titer of the cytotoxins may be preserved for fifty days in serum diluted 1 to 10, but this lowering of the titer limits the use of the diluted serum to ten days.

Our recent studies on this substance have shown it to be of very little value in rheumatoid arthritis.

IODIDES

These have been used empirically in the treatment of rheumatoid arthritis, especially in the chronic stages of the disease. Some physicians think that iodides exert a synergistic effect with aspirin and advise 0.3 cc. (5 minims) of a saturated watery solution of potassium iodide two or three times a day. In some patients, iodides are not tolerated because of gastrointestinal irritation or eruption on the skin.

Intramuscular or intravenous injections of 10 per cent sodium iodide (such as Naiodine) have been used for the relief of arthritic and sciatic pains. Such injections are not advocated in most major arthritic clinics.

Iodides have enjoyed a reputation in the past as alteratives. The term alterative is essentially a word screen for ignorance. With increases in our information and clearer concepts, such vaguely defined terms fall into disrepute and the word alterative is now rarely used by modern writers.[82]

Unless new data are forthcoming, the rôle of iodides in most forms of joint disease is a thing of the past.

GLYCEROPHOSPHATE

Various physicians in Italy have given daily intravenous injections of 10 cc. of a solution containing 2.5 gm. of *glycerophosphate* in distilled water in various rheumatic diseases.[14,59,64–66,104] The treatment appeared to be harmless and temporary improvement consisted of a rapid decrease in pain and swelling, an improvement in the general sense of well-being and an increase in appetite. This was based upon the treatment of Rinaldi, a physician in a small village near Tuscany—who kept his method a secret until this was published after his death following an inquiry by the Department of Health of the Italian Home Office. According to available information, Rinaldi's injections consisted essentially of strong doses of sodium glycerophosphate together with small doses of sodium cacodylate and strychnine.[45]

OTHER DRUGS

Because of the slow improvement which may occur in rheumatoid arthritis, patients may be led to try self medication with proprietary

measures. Such a practice is not limited solely to sufferers from this disease. Physicians, disheartened by the results of conservative therapy, also turn to these products. It is fair to say that some of them may temporarily relieve pain in arthritis because of their content of salicylates, cinchophen or other substances.

It should be remembered that arthritic symptoms may occasionally follow the administration of some drugs (arsenic, bismuth, mercury, gold, barbiturates, sulfonamides, etc.). Furthermore, spontaneous remissions may occur at any time in true rheumatoid arthritis, although these are not common.

Before prescribing any proprietary preparation, the physician should be aware of its contents. Advertising may be misleading at times to both patient and physician. In cases of doubt concerning proprietary medication in arthritis, the general practitioner will not err if he employs preparations which have been approved by the Council on Pharmacy and Chemistry of the American Medical Association.

There are no specific drug remedies for the treatment of rheumatoid arthritis. The Council on Pharmacy and Chemistry of the American Medical Association recognizes the proper use of drugs which have analgesic properties for the relief of pain in arthritis. Some of the anti-arthritic preparations which have been examined and found to be *not acceptable* for inclusion in the NNR[58] (New and Non-official Remedies) are Lyxanthine-Astier; Oxo-ate and Oxo-ate "B;" Causalin (Causyth); Farastan; Condol; Ertron; Aciform II; and others. To date, the Council has not accepted for inclusion in NNR any preparation of collodial sulfur or gold for parenteral use in the treatment of arthritis.

In the 1940 edition of New and Non-official Remedies, numerous analgesic drugs are included; these include *phenetidin* derivatives such as acetophenetidin (phenacetin), phenetsal (phenetsalum or salophen); *pyrazolon* derivatives such as antipyrine, melubrin, aminopyrine (one trade name of which is pyramidon); *cinchophen* and its derivatives (chloroxyl, neocinchophen); and derivates of *salicylic acid* (including acetylsalicylic acid [aspirin], novaspirin [salicitrin], salysal, sal-ethyl carbonate and others).

The occasional occurrence of agranulocytic angina following administration of aminopyrine and of liver damage following cinchophen derivatives is well known.[14]

Bernhard reported the effects of *causalin* in 286 cases of "arthritis."[6] He recommended that the drug be discontinued if gastric symptoms appeared early in the treatment. He gave up to 4 gm. (60 grains) daily with no signs of toxicity. Bernhard stated that a causalin tablet contained about 0.12 gm. (1.82 grains) of aminopyrine. Berghausen reported a *fatal leukopenia following* internal administration of *causalin* in a dose of 3 capsules per day for a month.[5] Rawls reported marked symptomatic relief in several patients with rheumatoid arthritis following cinchophen toxicity and urticaria, without jaundice.[84]

A list of patented medications sold in American drug stores for the treatment of arthritis, gout and neuritis sent to the American Medical Association in 1931 by Dr. P. S. Hench of the Mayo Clinic included about 550 drugs. It was noted that such "rheumatic cures" often contain salicylates or cinchophen, an alkali and a mild cathartic. Although such drugs may be of value in temporarily relieving pain, they will not, *per se*, cure arthritis.

New drugs are proposed annually in the treatment of arthritis. According to the 1938 Federal Food, Drug and Cosmetic Act, a new drug cannot be manufactured until a permit has been obtained from the Secretary of Agriculture. In application for this permit, the manufacturer must state its composition, a full list of articles used as components of the drug, reports of all investigation to show whether or not the drug is safe to use, a description of the method of manufacture, processing and packing the drug, five copies of each label and a complete package of the drug. This law should decrease the number of ineffective proprietary preparations available.

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CHAPTER XXII

GOLD THERAPY IN ARTHRITIS

RHEUMATOLOGISTS agree upon the necessity of individualizing the program for the care of the patient with rheumatoid arthritis. This program should consist of mental and physical rest (with avoidance of emotional upsets if possible); local rest of the involved joints by splinting where indicated; a nourishing, well balanced, high vitamin, anti-constipation diet; adequate physical and occupational therapy; orthopedic care; treatment or removal of any (focal) infection as a general health measure to lessen the load which the individual is carrying; and symptomatic care as required (including analgesics when needed, blood transfusions and iron medication, sedatives if indicated, etc.). Very few internists now believe that rheumatoid arthritis is greatly benefited by such measures as injections of sulfur, bee venom, chaulmoogra oil, snake venom, endocrine preparations (except when specifically indicated), special diets (except a well-balanced, sensible high vitamin diet, selected to meet the caloric requirements of the individual patient), splenectomy, etc.

Marked difference of opinion still exists among students of rheumatic diseases as to the virtues of such measures as injections of gold, oral or parenteral administration of massive doses of vitamin D or the value of roentgen therapy in rheumatoid arthritis (although most observers agree that roentgen therapy is of definite value in rheumatoid arthritis of the spine—rheumatoid spondylitis). Discussions on massive doses of vitamin D and of roentgen therapy are presented in separate chapters later in this book.

The *following conclusions* are drawn from a careful analysis of published reports (of competent observers), opinions of leading rheumatologists obtained personally and experience gained from a study of our own private and clinic patients in whom chrysotherapy was employed.

1. *Injections of some gold salts in proper dosage are followed by definite subjective and objective evidence of improvement in the majority of patients with rheumatoid arthritis.*

2. Gold salts are not the ideal drug we are looking for in rheumatoid arthritis because:

   (*a*) They are *toxic.*

   (*b*) *Not all patients* with rheumatoid arthritis *respond* to injections of gold salts.

   (*c*) In a moderate percentage of patients, the *arthritis recurs following cessation of chrysotherapy.*

3. As yet, *gold salts are not advised routinely at the onset* of rheumatoid arthritis. Conservative therapy results in improvement in a majority of patients with rheumatoid arthritis (when followed carefully over a period of from five to ten years). *If the rheumatoid arthritic obtains no benefit from*

conservative measures over a period of three or four months, or if he goes downhill rapidly even before this amount of time, *gold therapy is advised if it can be given by one thoroughly acquainted with this subject.*

4. *Gold therapy in rheumatoid arthritis is still too dangerous for common use by the general practitioner.*

5. *Individual doses of gold salts should be much smaller than those used previously.* This will greatly decrease the incidence of toxic reactions while therapeutic results will be lessened only slightly (and not at all in many instances).

6. With smaller individual doses, the number of injections per series may be almost doubled; *i. e.,* each series may be continued over a period of five months or more. This may result in fewer relapses, when gold therapy is finally stopped.

7. The optimum dose of gold salt per injection, the interval between injections and the exact length of each series of injections are still unknown. In view of our present knowledge, it is suggested that, when preparations containing 50 per cent of gold are used (Myochrysine or Solganal B Oleosum), the individual dose should not exceed 50 mg. of the gold salt. *Present evidence suggests that results are almost as good when individual doses do not exceed 25 mg. of the gold salt.* We have observed good results in some of our own patients with rheumatoid arthritis when the individual doses of gold salts did not exceed 10 mg. (per week); unfortunately, this latter group of patients has been observed for not more than two years in most instances. Despite the insistence of some authors that smaller doses of gold salts do not produce brilliant results in rheumatoid arthritis, it is suggested that the dose of gold salts be gradually increased (when Myochrysine or Solganal-B-oleosum are used) until 25 mg. is being given each week; if signs of improvement (both clinical and laboratory—including a lowering of the sedimentation rate) do not occur after eight weeks of this dosage (25 mg. of the gold salt per week), the dose of gold salts should be increased to 50 mg. per week, but should not exceed this figure (unless the physician is fully prepared to assume all risks for severe toxic reactions). (See dosage schedule presented later in this chapter.)

8. *Toxic reactions may occur following even small doses of gold salts;* however, these are usually much less severe and of much shorter duration than those reactions which may follow large doses of gold salts.

9. *Gold salts are of value in no rheumatic disease except rheumatoid arthritis.*

10. TRY TO AVOID SERIOUS TOXIC REACTIONS BY: (1) questioning the patient at each visit concerning pruritus, sore mouth or gastrointestinal disturbances; (2) examining the skin, gingiva and scleræ at each visit for dermatitis, stomatitis, purpuric spots and icterus; (3) performing a WBC, differential count and Hb and a urinalysis (for albumin, red blood cells and casts) *prior to each injection.*

11. *Gold salts should not be given to any patient with a history of agranulocytic angina, purpura, exfoliative dermatitis, renal or hepatic disease. Gold salts should not be given during pregnancy.* It is inadvisable to give gold salts to a diabetic, unless this is very mild and the patient has a severe

rheumatoid arthritis which is going downhill despite all attempts at conservative care; gold salts may aggravate the diabetic state.

12. Conservative care should be continued during chrysotherapy: mental and physical rest; splinting of the affected joints where indicated; an adequate, sensible well balanced diet; physical and occupational therapy; orthopedic attention; psychotherapy; and symptomatic care when indicated (including analgesics, sedatives, hematinics, anti-constipation regimen, etc.).

13. There is no method of preventing toxic reactions, although these are usually mild and of relatively short duration if small doses of gold are used per injection. Treatment of these toxic reactions is non-specific.

14. *Gold should be stopped if an untoward reaction occurs.* If this is agranulocytic angina, purpura hemorrhagica, exfoliative dermatitis, jaundice (hepatitis) or severe renal damage, the gold should never be resumed. Following the subsidence of *mild* reactions (pruritus, dermatitis, gastrointestinal upsets), gold may be resumed in smaller doses. Some workers merely decrease the dose of gold in individuals with these milder reactions; such a procedure is taking an unnecessary risk, not in accord with good clinical judgment.

15. If the rheumatoid arthritis is not improved following injections of 25 mg. of Myochrysine or Solganal-B-oleosum for eight weeks, together with injections of 50 mg. of the drug each week for an additional twelve weeks, it is unlikely that improvement will come as a result of further gold salt therapy in that particular individual.

16. If improvement occurs during the course of gold therapy, it is advisable to repeat the entire series of gold salts at least once after an interval of six to eight weeks during which no gold is given (or during which not more than 10 mg. of the gold salt is given per week).

Introduction of Gold Salts in the Treatment of Arthritis.—Gold has been employed as a therapeutic agent for many centuries. *In the eighth century it was recommended as a panacea, and in 1500* (in a compound of mercury) *it was advocated by Paracelsus as an elixir of life.*[83] Koch demonstrated (1890) that gold cyanide inhibited the growth of tubercle bacilli *in vitro.* Inorganic preparations were in use until 1916, since which time many organic compounds have been developed.

Feldt showed (in 1913) that certain preparations of gold which contained a sulfhydryl group possesses anti-bacterial properties.[19] Fourteen years later, he found that Solganal was effective against experimentally produced streptococcal and spirochetal infections in mice.[20] Lande and Pick reported on the use of gold preparations (Solganal and Triphal) in human chronic polyarthritis in the same year.[49,60] The work of Forestier, who treated large numbers of arthritic patients with gold preparations, popularized this form of therapy.

The widespread use of gold therapy has been somewhat limited by its untoward reactions, the crudeness of the available preparations and the uncertainty of dosage. Among the preparations, which have been developed are "Solganal," "Neo-Solganal," "Solganal B," "Myochrysine," gold potassium cyanide, "Krysalgon," "Triphal," "Sanocrysin," "Aurocein," "Aurol-Sulfide," calcium aurothiomalate, etc.

Mollgaard reported favorable action of gold salts in experimental tuberculosis in calves.[55] Other clinicians felt that gold compounds could also check the progress of human tuberculosis. Seligson has recently reported gratifying results in a few cases of even far advanced pulmonary tuberculosis with gold therapy; the predominantly exudative cases responded most favorably.[80] Cavities and infiltrations disappeared and the sputum became free of tubercle bacilli. Injection of gold salts into experimentally infected animals was reported to control the infection by means of stimulation of a defense reaction.[18] *Gold salts were employed experimentally in chronic rheumatism by Feldt in 1928,* following their previous use in pulmonary tuberculosis. *On the hypothesis that if gold salts were active in a chronic disease such as human tuberculosis, Forestier believed this metal should be of value in rheumatoid arthritis and in infectious forms of arthritis.*[24,25] Accordingly, he began the use of gold salts in the treatment of arthritis in France in 1928. On the basis of hundreds of cases of arthritis, he has written that *injections of gold salts when properly administered have given better results in the treatment of rheumatoid arthritis and all forms of infective arthritis than any previous method employed in France.*

Although there seems to have been no sound reason for the use of gold salts in rheumatoid arthritis, recent experimental animal and human clinical work suggests that gold is an important addition to our therapeutic armamentarium. The work of Sabin, Freyberg and others showed gold to be definitely beneficial in arthritis produced experimentally in mice by pleuropneumonia-like organisms. Rheumatoid arthritis in human beings has been reported to be definitely benefited by injections of gold by such authorities as *Freyberg, Dawson, Boots, Hartung, Cecil, Snyder* and others.

On the other hand, it must be remembered that other eminent rheumatologists are not impressed by the results of gold therapy. *Walter Bauer,* several years ago, observed that only 4 of 30 patients treated with gold showed marked subjective and objective improvement.[2] The sedimentation rates returned to near the normal range in 5 of the 30. *In his experience, these results were no better than the usual treatment with rest, high caloric high vitamin diet, analgesics and simple physiotherapy. He believed that ten years from now, it would go the way of many previous rheumatic remedies and would not be considered the meritorious anti-rheumatic remedy it is hailed today.* Furthermore, neither *Holbrook* nor *Hench* (who together with Bauer have directed the care of rheumatic diseases in the Army) have yet published their experience with gold salts in rheumatoid arthritis. Some of these individuals continue to use chrysotherapy with hopes, but not high hopes.

Gold Therapy in Experimentally Produced Arthritis in Animals.—Many stocks of mice carry a number of biologically and immunologically different types of *filtrable microörganisms of the pleuropneumonia group.*[71] In normal mice these microörganisms are found chiefly in the mucosa of

the sinuses and nose and on the conjunctiva; occasionally, they may be found in small numbers in the brain and lungs. Despite the presence of these microörganisms normally, examination of thousands of mice showed no spontaneous arthritis in these animals.[74] When, however, some of these filtrable organisms are cultured *in vitro* and later are *given intravenously to mice of the same stock, they produce a proliferative, progressive chronic arthritis*. After one intravenous injection of ½ ml. of a growing twenty-four- to forty-eight-hour culture of a type B strain (having its chief affinity for joints), from 90 to 100 per cent of the mice develop a polyarthritis; this is at first migratory, and finally, chronic and progressive, often resulting in ankylosis from two to four months later. In some series, this ankylosis may occur in as many as 70 per cent of the animals.

Clinically, the mice show fusiform swelling of the digits with anterior and posterior extremities being affected equally and often symmetrically; the knee joints may also be involved. Pathologic changes are found in the synovia, cartilage, capsule and subchondral bone marrow.[73] There is a marked proliferation in the synovia which later affects the capsule and perichondrium so as to at times obliterate the free joint space because of overgrowth of proliferative tissue. In the fourth week after inoculation of the animals, there occurs actual destruction of cartilage and at seven weeks, one may find replacement of the normal articular cartilage by undifferentiated cells. After several months, complete ankylosis may result with distorted articulating surfaces, joined by fibrous tissue.

Despite the resemblance between this form of experimentally produced proliferative, chronic arthritis in mice and rheumatoid arthritis in man, investigators have not been able to obtain similar microörganisms from exudates and tissues of human beings with rheumatoid arthritis.

The arthritis disappeared completely in 96 per cent of 603 mice with established experimental arthritis (from pleuropneumonia-like organisms) when treated with adequate doses of suitable gold compounds.[75] On the other hand, the arthritis disappeared in only 5 per cent of 100 mice when treated similarly with various other preparations such as *colloidal* gold, bismuth subsalicylate, neoarsphenamine, sulfathiazole and sulfapyridine, silver arsphenamine, fuadin (antimony) and sodium salicylate. In 184 control mice with experimentally produced arthritis, the arthritis disappeared in only 5 per cent.[70,72] Freyberg repeated some of these experiments with similar results.[61]

It was noteworthy that the *curative effect of the gold was elicited in these mice only when therapy was begun during the first three weeks after the onset of the arthritis*. Following this, *when considerable destruction of cartilage and obliteration of the affected joints occurred in mice, the effect of the gold was slight or absent.*

A complete curative effect can be obtained in practically 100 per cent of mice when treated early with adequate doses of gold; when the dose is reduced, a smaller number of animals are cured and the incidence of spontaneous recurrences after cessation of therapy becomes greater.[69] The gold compounds which are therapeutically effective against this arthritis

in mice do not exhibit a microbial action *in vitro* in the microörganism causing this arthritis.

Sabin found that in mice, the toxicity of gold depended upon the solubility of the compound and the nature of the radical to which the gold was attached; however, with soluble gold compounds, therapeutic effectiveness in mice depended chiefly on the concentration of gold. Calcium aurothiomalate, an insoluble compound, was found to be therapeutically effective against experimental mouse arthritis in the same or even smaller dose than the soluble sodium aurothiomalate (Myochrysine).

Following the use of 1 mg. or more of calcium aurothiomalate, the experimentally produced arthritis disappeared completely in 90 per cent of 70 mice; if the dose was reduced to 0.5 or 0.25 mg. of the gold salt, less than 25 per cent of those treated recovered. In 30 control mice which were untreated, no recovery occurred. The curative effect was found whether the entire dose was given at once or whether the dose (1 mg.) of calcium aurothiomalate was divided into 10 equal doses and given at forty-eight hour intervals. Although sodium aurothiomalate was an effective prophylactic and therapeutic agent against this experimental arthritis in mice, sodium thiomalate itself was without value even when given in doses ten times greater than the effective amount of the gold salt. The disulfide form of sodium thiomalate was also ineffective.[61] That the improvement was not due to the sulfur which is often present in these preparations of gold salts was shown by the effectiveness of sodium succinimido-aurate, a gold salt which does not contain sulfur. Gold chloride was also effective therapeutically in the experimental mouse arthritis, although this preparation is very toxic. Studies such as these show that the *value of gold salts in these animal studies depended on the presence of gold and was not due to sulfur or other non-metallic substances.*

Sabin and Warren showed that colloidal gold or gold sulfide were therapeutically inert against experimental chronic arthritis in mice.[74,76]

One must remember that experimentally produced arthritis in animals differs in certain respects from rheumatoid arthritis in man. In addition to the work of Sabin and his associates, other reliable reports have given similar findings. Findlay and others have shown that gold compounds are effective against infectious arthritis produced in rodents with certain pleuropneumonia-like organisms.[11,21,22] Heilman reported that a single injection of gold (Myochrysine) protected mice against rapidly fatal doses of Streptobacillus moniliformis (while sulfapyridine and neosarphenamine were ineffective against this organism).[44] The Streptobacillus moniliformis is constantly associated with a pleuropneumonia-like organism which it is believed may be found to represent a phase in the life history of the Streptobacillus moniliformis.[15,47] The intra-abdominal injection of minute amounts of virulent cultures of this organism into Swiss mice often results in an arthritis of one or more joints; larger amounts result in septicemia and death. Heilman found that a single injection of Myochrysine given intramuscularly (0.25 gm. per kilogram of mouse) at the time of injection of the microbic culture (or twelve hours before or after) protected the

mice against 100 to 1000 times the smallest amount of the culture required to cause death in 80 per cent of the mice in three days. Angevine showed that gold salts prevented hemolytic streptococcus arthritis in rats.[66]

In the chronic proliferative arthritis of mice produced by injections of pleuropneumonia-like organisms, it was found that COLLOIDAL GOLD COMPOUNDS WERE INEFFECTIVE. Therapeutic results were obtained, however, by *soluble organic gold compounds*, such as Myochrysine, Solganal and Triphal; by *insoluble organic compounds suspended in oil*, as calcium aurothiomalate, by *inorganic gold* as Sanocrysin and by injections of gold chloride (although the latter is by far too toxic to be used in human beings).

It had been pointed out that practically all of the gold compounds used in the treatment of rheumatoid arthritis contained sulfur; some individuals had suggested that the effectiveness of the compounds was due largely to the latter, rather than the gold. However, it was shown that injections of gold chloride (without sulfur) also exhibited a curative effect on experimental mouse arthritis.[74]

Sabin and Warren found in mice that the greater the dose of an effective gold compound, the more complete and more rapid was the disappearance of the arthritis. A given total dose which was curative when given within a short period in a small number of divided doses was at times ineffective when given over a much longer period and divided into a larger number of small doses. There was a high incidence of spontaneous recurrence after cessation of treatment with doses which were on the borderline of effectiveness.

The therapeutic response was more rapid and more complete the earlier in the disease the treatment was started. In experimentally produced mouse arthritis, when the treatment was delayed for four weeks after the onset of the arthritis, there was such a progression of involvement of some joints in a majority of the mice that gold was no longer effective.

Neither the blood of mice treated with large doses of gold nor the gold compound itself were effective in preventing the growth of the etiologic agent *in vitro;* even though the microörganisms were grown in the presence of an active gold compound for three generations, they did not lose their ability to produce arthritis.

Most rheumatologists believe that rheumatoid arthritis is a chronic inflammatory disease of unknown etiology and that treatment must be individualized for each patient aiming: (1) to promote optimal general health and comfort; (2) to prevent deformities and crippling; and (3) if possible, to arrest the inflammatory process.[27]

No one therapeutic measure should be used to the exclusion of others but the well balanced program will include rest, physical therapy, nutritious diet, relief of discomfort, adequate orthopedic care and symptomatic care where indicated.[93] *Gold therapy seems to be the greatest single drug showing promise in checking the activity of the disease in many cases*, but this is still imperfect and must be used with great care.

Gold seems to lessen or arrest the inflammatory process in some manner in some cases of rheumatoid arthritis. The general practitioner must remember

that gold will not reconstruct joints nor will it repair damaged cartilage or bone.[31] It will not rehabilitate the arthritic derelict with crippling deformities whose arthritis has been burned out following years of activity of the process. The patient who will have most chance of benefit from proper chrysotherapy is the active rheumatoid arthritic, preferably early in the course of his disease.

Good results have been reported by numerous authors using gold salts in rheumatoid arthritis.[9]

Almost all physicians who have had considerable experience with gold therapy in rheumatoid arthritis report better results than can be obtained with other types of therapy.[26]

Fredrik *Sundelin* and Ruiz-Moreno collected reports of 3800 cases of arthritis treated with gold (up to 1941) and found that an average of 81 per cent were improved.[68,88]

Reports in foreign countries (England, Germany, France, Scandinavia, Argentina and others) vary from mild approval to marked enthusiasm concerning the results.

Although gold compounds have been used on the continent for the treatment of arthritis since 1929, American physicians hesitated at first to "go on the gold standard" in the treatment of arthritis because of the frequency of severe complications and also because of the mortality of at least 1 per cent which has accompanied the treatment.

Phillips, who presented one of the earliest American reports on chrysotherapy in rheumatoid arthritis, was not favorably impressed.[59] Oren reported a good response in 60 of 66 cases of rheumatoid arthritis following administration of gold salts.[56] Key and his associates reported in 1939 that gold salts definitely ameliorated the course of the disease in the majority of 70 cases of rheumatoid arthritis, regardless of the duration of the disease. Sashin and his associates found moderate to marked improvement in 85 per cent of 80 patients with rheumatoid arthritis following gold salts while Snyder in the same year noted improvement in only 48 per cent of 50 cases.

Freyberg believed that gold salt therapy is the best treatment now available to arrest or lessen the inflammatory process in rheumatoid arthritis.[26]

Dawson, Boots and *Tyson* concluded that chrysotherapy offered greater promise than other forms of therapy in the treatment of rheumatoid arthritis, and thought that despite its dangers, which should be emphasized, this therapy should be continued by careful clinicians.[14]

Ellman and his associates[17] divided 90 typical cases of rheumatoid arthritis into 3 groups; one of the groups served as a control and the other two were treated with large and small doses of gold salts (Solganol B) respectively. These workers obtained evidence that injections of gold compounds favorably altered the course of the disease.

Of the 30 patients used as a control group, only 1 was rendered inactive; 22 were improved; 6 observed no change and 1 became worse. These patients did not receive gold therapy. Of the group receiving large doses

of gold, 14 of the 30 were rendered inactive and 14 more were improved while 2 showed no change. With small doses of gold salts, 8 were rendered inactive, 20 were improved, 1 experienced no change and 1 became worse. Inactivity of the arthritis included complete freedom from subjective joint pain, a normal sedimentation rate and if limitation of motion remained, this was painless.

In those treated with large doses, 0.2 gm. of Solganol B was given intramuscularly per week (after first testing for sensitivity by 3 initial weekly doses of 0.01, 0.05 and 0.1 gm. of the material); occasionally, as much as 0.3 gm. of Solganol B was given to resistant patients. In the group treated with smaller doses of gold, after two initial injections of 0.01 and 0.05 gm., 0.1 gm. was given into the gluteal muscles each week. In the control group, similar amounts of sterile almond oil were injected; this is innocuous and was often used for suspending the gold compounds. Each treatment consisted of two courses, with an interval of six weeks between courses of therapy.

In the control group, at the end of nine months after treatment, the sedimentation rate was increased in 17, lowered in 13 and under 10 mm. in 4. *In those treated with large doses of gold, the rate was increased in only 2, was reduced in 28 and was less than 10 mm. in 23.* In those receiving smaller doses of gold, the sedimentation rate was increased in only 1, was lowered in 29 and was under 10 mm. in 11. In addition, using the larger doses of gold, there was a decrease of swelling of the joints in 66 per cent of the patients and in 59 per cent with the smaller doses of gold, as compared to only 32 per cent in controls. These results are very significant and must not be underestimated.

Price and Leichentritt observed 101 cases of rheumatoid arthritis treated with gold therapy over a six year period. They concluded that gold was an effective remedy for rheumatoid arthritis, assisting in the alleviation of all joint symptoms and in rehabilitating these individuals. *They also found that aurotherapy was most effective in the early stages of the disease but that it frequently relieved pain and stiffness even in advanced cases and is worthy of trial in such patients.* They again stressed the importance of careful and repeated follow-ups on such patients before drawing any final conclusions from gold therapy because of the high incidence of relapse and remission during the natural course of rheumatoid arthritis.

Hartung observed a remission of the rheumatoid arthritis in 54 per cent of 264 patients treated with gold salts; all of these patients had their arthritis more than six months and most of them, for more than a year. In those individuals who responded favorably to gold therapy, clinical improvement was evident about the eighth week of treatment; a decrease in the sedimentation rate was evident about the same time.

Hartung has reported that blood transfusions repeated each week for ten to twelve weeks were definitely beneficial in rheumatoid arthritis, resulting in remissions in some cases. In patients who were intolerant to gold salts, he recommended the use of multiple transfusions.

Cecil reported that 31 per cent of 235 patients with rheumatoid arthritis who received adequate therapy had a complete remission (with respect to pain and swelling in the joints). Improvement was definitely greater in those whose chrysotherapy was begun early in the course of the rheumatoid arthritis; 39 per cent of early cases showed remission with gold therapy as compared with 29 per cent of late cases. The severity of the disease was of no significance in determining the improvement which was to follow as the *incidence of improvement was similar in the mild or severe cases.*[8] In the group who obtained a remission of their disease, 77 per cent had received a total of 1 gm. or more of gold salt while only 41 per cent of those who showed no improvement had this much total therapy.

Hartung believes that therapy with gold salts is indicated in patients with active rheumatoid arthritis if the disease has been present for more than three months.[41] He stresses that other important factors in treatment include rest of the patient (with rest in bed if fever is present) and rest of severely involved joints by the use of splints, braces or casts; avoidance of emotional fatigue; a well balanced high vitamin diet; treatment of any diseased condition which is found (including infected sinuses, tonsils and teeth); securing regular bowel elimination and symptomatic therapy when indicated (such as the use of hydrochloric acid if achlorhydria is present; iron medication for hypochromic anemia, etc.). It is important to stress over and over again that the proper treatment of rheumatoid arthritis is a general regimen adapted to the particular patient, and not merely the injection of a single drug once a week.

Hartung emphasized that gold salts should not be used in any other form of arthritis than rheumatoid arthritis and that especially, they should not be employed in arthritis associated with lupus erythematosus, acute rheumatic fever—with or without cardiac involvement, renal disease or blood dyscrasia. Tegner pointed out that a drug (gold) of unknown action (but widely known toxic properties) was being used to treat a disease of unknown etiology.

In our present state of knowledge, one can not recommend that the general practitioner use gold salts in the treatment of rheumatoid arthritis unless he has studied the problem carefully and has (if possible) observed their administration in an arthritis clinic at a recognized institution.[28]

The unpredictable nature and time of the toxic reactions following gold therapy is the chief hazard in their use.[52] It is dangerous and unwise to use gold salts indiscriminately because of their potential toxicity. Gold should not be used if the patient cannot be observed carefully for early indications of toxic effects or if blood counts, urinalyses and sedimentation rates cannot be performed.[62]

Sundalin was of the opinion that large doses of gold were no more toxic than small doses; most observers do not agree with this.

The results from chrysotherapy do not approach the striking improvement observed in patients with rheumatoid arthritis who have acquired intercurrent jaundice or in most women patients who become pregnant.[55]

Although Forestier and Certonciny have stated that only 1 or 2 per cent of patients with rheumatoid arthritis make a spontaneous permanent

recovery, Short has reported complete inactivation of the disease in 16 per cent of cases without gold therapy, when followed over a period of ten years.[9] Short studied 300 patients with rheumatoid arthritis who were followed for ten years; he noted that "satisfactory improvement" occurred in about half of the cases without gold therapy and that complete inactivation of the disease occurred in 16 per cent.[81] A reactivation of the arthritic process may occur even after years of quiescence in the patient with rheumatoid arthritis.

Snyder and his associates advocated gold therapy only when the arthritis was refractory to other forms of therapy; Cecil, on the other hand, was quite convinced that gold salts were most effective in the treatment of early cases.

Forty-eight per cent of Snyder's patients with rheumatoid arthritis (whose arthritis had previously proved refractory to most of the commonly accepted forms of treatment) showed definite clinical improvement when treated by gold sodium thiosulfate.[85]

Cecil and his associates found remission or marked improvement in 62 per cent of 245 cases of rheumatoid arthritis treated with gold salts; however, only 1 patient of 10 with ankylosing spondylitis was benefited.[9]

Dawson, Boots and Tyson reported that 51 out of 100 cases of rheumatoid arthritis showed a *striking* improvement following gold therapy, with an additional 25 showing moderate improvement; 15 of the group showed no improvement.[14] *In 22 of the 51 cases, the disease was apparently rendered inactive with persistently normal sedimentation rates following treatment. These cases are the most convincing argument in favor of chrysotherapy as similar results have not been obtained with any other form of therapy to date.*

Sixty per cent of the 101 patients studied by Price and Leichentritt showed a definite improvement with 13 of 14 patients with mild rheumatoid arthritis being improved, 24 of 30 patients with moderate rheumatoid arthritis being improved, and 23 of 57 patients with severe rheumatoid arthritis noting improvement. Twenty-nine per cent of the cases showed no improvement at any time and 2 patients became worse.

Hartfall and his colleagues in England reported their results with chrysotherapy in the treatment of 900 cases of arthritis, 750 of which were of the rheumatoid type.[37–40] *These workers found that preparations of gold constituted the best single form of treatment in rheumatoid arthritis.* In their opinion, rheumatoid arthritis, if seen in its early stages, was curable by gold salts; *they further stated that there were few cases which could not be improved to some extent.* Other British physicians have found it of value in the treatment of arthritis.[32,57,82,91]

Until recently gold salts were not used widely in this country; this may have been due in part to the frequency of complications and the mortality of approximately 1 per cent which occurs following gold therapy.

In an early report on over 200 patients with rheumatoid arthritis treated with gold therapy, Cecil noted striking improvement in many and a few remarkable recoveries.[7] He believed that gold therapy was an important advance in the treatment of rheumatoid arthritis. Sashin and his associates

reported marked improvement in 44 per cent and slight or moderate improvement in an additional 39 per cent of 80 patients with advanced rheumatoid arthritis who were treated with gold; these patients had been refractory to other therapeutic measures.[78] These authors concluded that gold therapy was superior to any other form of treatment known at present for patients with rheumatoid arthritis.

Snyder and his colleagues treated 50 cases of rheumatoid arthritis with parenteral gold.[86] There was only 1 excellent result, while good results were obtained in only 5 additional patients. *In over one-half of the cases the results were poor.*

Graham and Fletcher reported good results in the treatment of 100 patients with rheumatoid arthritis of more than average severity;[34] in some instances, the results were dramatic. They concluded that the incidence of toxic reactions and their occasionally severe character were serious considerations but were scarcely comparable to the grim prognosis of polyarticular rheumatoid arthritis which is taking a downward course. They believed that gold materially shortened the duration and moderated the severity of the active and crippling stage of patients with classical, chronic, progressive rheumatoid arthritis (with involvement of multiple joints). In milder cases, they advocated conservative therapy first, without gold. They again warned that there was no justification for the use of gold in any rheumatic or joint affection other than rheumatoid arthritis. It has been of no value in various forms of fibrositis, myositis, neuritis, rheumatic fever, gout, degenerative joint disease or menopausal arthralgia. In their series, patients with rheumatoid arthritis of the spine (Marie-Strümpell spondylitis) did not respond favorably to gold therapy (as many others have noted; inasmuch as roentgen therapy has been shown to be valuable in this latter disorder, it should always be employed—together with rest and orthopedic and physical therapeutic measures in the treatment of rheumatoid spondylitis).

Traut and Barton reported on the failure of gold therapy to improve a series of 16 patients with rheumatoid arthritis.[94] Gold sodium thiosulfate was used in 5 patients; exfoliative dermatitis appeared in two of these. This is an extremely unusual incidence (40 per cent) of exfoliative dermatitis and suggests that other unknown factors may have played a rôle. Their 11 clinic patients were treated with gold sodium succinamide, a drug which has not yet been proven of value in rheumatoid arthritis. Four of the nine clinic patients developed toxic reactions which were thought to be severe enough to stop therapy; only one of these patients improved temporarily. The toxic reactions reported by these authors are so out of line with those reported in large series by other workers that one hesitates to accept the lack of improvement in their patients following their therapy.

Use of Gold as the Sole Therapeutic Agent in the Treatment of a Group of Patients With Rheumatoid Arthritis.—Hartfall and his associates employed chrysotherapy without the addition of any other measures in the treatment of their large series (900) of arthritic patients. Neither

physical therapy, removal of focal infection, vaccines, diets, vitamins, nor any other special procedures were used. The good results obtained, therefore, must be attributed either to the gold employed, to the psychic effects of injections, or to a natural remission of the disease. This latter is improbable as spontaneous remissions in true rheumatoid arthritis of more than a year's duration do not occur in a large percentage of cases within one or two years.

Mode of Action of Gold Salts in the Treatment of Arthritis.—The mode of action of gold salts in arthritis is not known.

Various writers have attributed the action of chrysotherapy in rheumatoid arthritis to (1) *stimulation of the reticulo-endothelial system;* (2) *bacteriostatic;* (3) *shock-producing;* or (4) *immunologic to joint structures.*[48,53,86,92]

Dawson and Hobby showed that *gold salts had marked chemotherapeutic properties against hemolytic streptococcal infection in mice,* comparable to that obtained with the sulfonamide derivatives. Gold sodium thiomalate showed a marked bacteriostatic effect *in vitro* against the Streptococcus hemolyticus in a 1 to 10,000 dilution. Rothbard, Angevine and Cecil found that *sodium aurothiomalate was an effective chemotherapeutic agent in the prevention of arthritis in rats produced by the hemolytic streptococcus,* but that it did not cure the disease once it was established. *Hartung reported that subcutaneous injections of gold salts were followed by a marked increase in the basteriostatic power of the patient's serum against the hemolytic streptococcus,* the effects being in direct proportion to the amount of gold given.

1. The *bacteriostatic theory* was advanced by Hartung and Cotter, who reported that serum from patients with rheumatoid arthritis became bactericidal against a strain of hemolytic streptococcus when treated with gold.[42] Gold does not prevent the development of new rheumatic tissue as 6 per cent of patients with rheumatoid arthritis in one series developed rheumatic nodules while under gold therapy.

2. Gold salts are deposited in the reticulo-endothelial system.[48] Kling, Saskin and Spanbock thought that the gold acted by *stimulating the reticulo-endothelial system,* but evidence for this is not complete.

3. Leichentritt and his associates believe that gold in suitable combinations may produce a *diuretic effect* with loss of tissue fluid and improvement in joint symptoms.[45] Such an improvement in joint symptomatology with loss of water from periarticular tissues following diuresis by ammonium chloride and mercupurin had been reported previously.

Some have suggested that gold therapy is a *mild shock therapy of prolonged action.*

Fate of Gold in the Body.—*Gold is excreted chiefly through the kidneys* and to a lesser degree through the intestine and bronchial mucosa. If large amounts are given to rabbits, an experimental nephritis results. The glomeruli show no damage except slight congestion in a few instances; excretion occurs mainly through the convoluted tubules. A quantitative or rapid elimination of gold does not occur, so that some of the gold remains in the tissues for a considerable time.

Much experimental work has been done in animals in an effort to determine the localization and excretion of gold salts. It has been shown that gold is capable of promoting cicatrization of foci and healing thereby, and that when given to rabbits, it is stored in the connective tissue of the spleen, in the Kupfer cells of the liver and to some extent in the lungs.[79] Gold has also been found in the blood serum and in the lymph after its administration.

In patients with rheumatoid arthritis and in animals with experimental arthritis treated with gold compounds, gold may be found in the synovial membrane in much larger quantity than in the same membrane of normal animals receiving gold injections.[54] Gold has been detected spectrographically in aspirated synovial effusions from knee joints of patients with rheumatoid arthritis treated with gold salts.

Metallic gold, such as is used for pill coating, is said to be harmless as none is absorbed.

Gold enters almost every cell of the body shortly after injection with *greatest concentrations* being found *in the liver, spleen, kidneys and skin.*[65]

The blood level of gold increases as the amount of gold injected increases. Daily plasma values for gold fluctuate somewhat from day to day, but generally are increased after each injection and remain at this higher level or decrease slightly until the next injection is given. The excretion of gold in the urine increases similarly as larger amounts of gold are injected, but this increased excretion is not directly proportional to the increased administration of gold.[5,30,43]

Gold salts are absorbed into the blood stream and are transported in the plasma following intramuscular administration.[29] When sodium aurothiomalate (myochrysine) or sodium aurothiosulfate are injected intramuscularly in amounts supplying equivalent amounts of *gold,* the plasma gold concentrations and urinary excretion of gold are similar. Gold values of plasma and urine vary with the size of the weekly dose but are not directly proportional to this.

Following administration of *colloidal gold sulfide* intramuscularly or by mouth, the urine and plasma gold values vary greatly in different patients. In some, no gold is found in the plasma or urine after large amounts of colloidal gold sulfide are given. Others absorb the gold and have urinary and plasma values comparable to those seen with soluble gold salts. *Most patients, when given colloidal gold sulfide, have very low plasma gold concentrations and excrete very little gold in the urine.*

When 100 mg. of sodium aurothiomalate is being given weekly, the plasma gold concentration remains between 0.4 and 0.8 mg. per 100 cc.[26]

The large retention of gold during its administration is an important point which must be remembered. The average daily excretion of gold between injections of 50 mg. of gold (100 mg. of the salt) is only about 1 mg. or 7 to 10 mg. per week—about 20 per cent of that injected. *During a course of gold therapy, about 80 per cent of the injected gold apparently remains in the body.* This increasing retention of gold is important clinically as *gold may be found in the plasma or urine for many months in diminishing amounts after the last injection of gold.* When 50 mg. of gold is injected each week,

the urine may contain gold for as long as eight to ten months after cessation of injections; *when the weekly dose of gold is reduced to 25 mg., the post-treatment excretion is much shorter.*

Freyberg found that *calcium aurothiomalate* is not absorbed in any appreciable amount during the first twenty-four hours following intramuscular injection.[26] In general, plasma gold values were found to be considerably smaller with this preparation than with comparable amounts of sodium aurothiomalate. The urinary excretion of gold was also markedly smaller with calcium aurothiomalate and on many days the urine contained no gold. However, gold was found in the plasma and urine for many months following cessation of treatment. Freyberg noted considerable variation in the behavior of this preparation from patient to patient.

Block and Buchanan devised a highly accurate and specific photoelectric colorimetric *microchemical method for the determination of gold* in biological fluids and tissues.[4,6,29]

No one has yet determined the form in which the gold exists in the blood.[28] Freyberg and his associates found that the *absorption of gold compounds after intramuscular injection* in rats appeared to be *related to the solubility of the injected compounds.* Colloidal preparations of gold were absorbed poorly, compared to the crystalline salts in aqueous solution.

Following intramuscular injections of various gold compounds in rats, gold was found in the kidneys and liver in larger amounts than in other organs; much less was found in the spleen and other organs contained insignificant quantities. Following administration of crystalline gold salts, the amount of gold found in the kidneys was greater than in the liver; *gold was found in larger amounts in the liver after injection of colloidal preparations.* Following injection of crystalline preparations of gold, the urine contained much more gold than did the feces; the *feces were the main route of excretion of gold following injections of colloidal gold preparations.* The urinary excretion of gold is much greater on the day of injection of gold salts than on other days, due probably to the quick rise in plasma gold concentration after the injection.

Some individuals absorb gold from the intestinal tract when colloidal gold sulfide is given orally, while in others, there is considerable variation in this absorption.

Kling, Sashin and Spanbock reported that in experimental animals, gold was deposited in high concentration in the synovial membrane. In some individuals, plasma and synovial fluid gold values are almost identical while in others, much less gold is found in the synovial fluid than in plasma. In some instances, no gold is found in synovial fluid. In some cases, gold diffuses quickly into the joint space. *Gold is not concentrated in synovial fluid following injection of gold salts.* It is found in joint fluid in an amount equivalent to or less than that present in plasma.[29] *The concentration of gold in synovial fluid is not significantly higher than the plasma gold concentration.* Gold did not remain in synovial fluid after it had disappeared from the blood.

When the weekly doses of gold are kept at a lower level, gold disappears from the urine more rapidly. Although gold may be found in the urine for ten months or longer following the cessation of weekly injections of 100 mg. of myochrysine (*i. e.*, 50 mg. of gold), if the weekly injections are limited to 50 mg. of myochrysine (25 mg. of gold), the urine may show no gold

Gold Salts in Rheumatoid Arthritis

1. Following administration of gold salts, only insignificant traces of gold are found in the red blood cells; *the gold is found almost exclusively in the protein fraction of the plasma.*[31]
2. When increasing amounts of gold are injected each week, there results a corresponding steplike increase in the plasma gold content; when weekly doses are kept constant, the plasma gold concentration does not continue to increase significantly. (These statements do not apply to colloidal gold preparations which disappear from the blood stream very rapidly because of phagocytosis of gold by the reticulo-endothelial cells).
3. *Gold is excreted chiefly in the urine.*
4. *Gold may be retained in the body for long periods and has been found in the blood and urine as long as a year after treatment has been stopped.*
5. Blood and urine become free of gold in shorter periods if smaller doses are injected.
6. Soluble crystalline gold salts are absorbed quickly after intramuscular injection.
7. Gold therapy has been of value in some, but not all cases of rheumatoid arthritis; in some cases gold therapy seems to be followed by dramatic improvement.
8. Toxic reactions are numerous following gold therapy and *not everyone can tolerate gold salts;* in general, about one-third of patients show some toxic reaction from chrysotherapy.
9. *Toxic reactions,* in order of frequency, include *dermatitis* (63 per cent) —often preceded by generalized *pruritus; stomatitis* (32 per cent); *renal toxicity* (albuminuria, hematuria and casts in 29 per cent)— and less frequently gastrointestinal symptoms, "gold bronchitis" and purpura.
10. The therapeutic effectiveness of gold probably depends upon its absorption and its remaining in the circulation until it arrives at the site of its action: *i. e.*, at present, it would seem that the gold preparations most likely to be of value in rheumatoid arthritis are sodium aurothiomalate (Myochrysine); sodium aurothiosulfate; and aurothioglucose (Solganal-B-oleosum).
11. *Calcium aurothiomalate* is not yet available commercially. Experimentally, it proved to be the least toxic of all gold salts and was effective prophylactically and therapeutically against experimentally produced arthritis in mice. Judgment must be withheld as to its ultimate value in man until further reports are available. This is an insoluble salt, which is absorbed slowly and its behavior as to its rate of excretion and plasma gold values is irregular.
12. *Colloidal gold preparations* (such as colloidal gold sulfide) *are not expected to be effective therapeutically* because of the rapid phagocytosis of the particles of gold by the reticulo-endothelial system with insignificant plasma gold concentrations.
13. Toxic reactions occur most often after a considerable amount of gold has been injected.

Gold Salts in Rheumatoid Arthritis—(*Continued*)

14. *Therapeutic effects are not usually observed until after 6 or 8 injections.*
15. There is no reliable test yet devised which would predict whether a patient would tolerate gold salts or not; patch tests are unreliable.
16. Recent reports suggest that the incidence of toxic reactions may be decreased by including large amounts of fruit juices and liver (¼ pound three times a week) in the diet.[10]
17. Once begun, there is no obvious dependable method to inhibit the effect of gold.
18. *Toxicity is definitely reduced when smaller doses of gold are given each week.*
19. *Gold salts are usually given intramuscularly;* there appears to be no advantage in their intravenous administration.
20. Although colloidal gold preparations produce only few and mild toxic reactions, the gold particles are generally removed from circulation so rapidly that toxic reaction is prevented therapeutic effect is also prevented. Some clinics have discontinued the use of colloidal gold preparations because of the poor therapeutic results.
21. Toxic reactions may occur when the plasma gold content may be below the average values seen with similar amounts of gold and without toxicity. It is doubtful if a check of the plasma gold value will prevent many toxic reactions to gold.
22. Relapses in the disease may occur several months after gold therapy has been discontinued.
23. *No one yet knows how, where or why gold salts result in improvement in rheumatoid arthritis.*
24. Early cases of rheumatoid arthritis appear to obtain more benefit from chrysotherapy than later cases.

within three months after the last injection. *When the dose of myochrysine is decreased still further to 25 mg. (i. e., 12.5 mg. of gold), the urine may show no gold four weeks after the last injection.*

Gold Preparations Available for Therapy in Arthritis.—The following have been used in the treatment of rheumatoid arthritis:

1. "Myochrysine" (sodium aurothiomalate –for intramuscular use).
2. "Solganal B oleosum" (for intramuscular use).
3. "Sanocrysin" (double thiosulfate of gold and sodium).
4. Aurol-sulfide (orally as a 1 per cent solution and intravenously or intramuscularly as a 1 or 2 per cent solution) (colloidal gold sulfide).
5. "Myoral" (calcium aurothioglycolate).
6. "Triphal" (sodium-auro-thio-benzimid-azole carboxylic acid).
7. "Parmanil" (intramuscular) (methyl glucamide of auro-thio-diglycollic acid).
8. "Lopion" (for intravenous use) (auro-allyl-thio-urea benzoate of sodium).
9. "Allochrysine" (aurothiopropanol sulfonate of sodium).
10. "Crisalbine" (gold sodium thiosulfate—for intravenous use).
11. "Aurocein" (intramuscular) (sulfhydryl gold naphthyl tri-sulfo-carbonium derivative).

Myochrysine.—Myochrysine (gold sodium thiomalate) is marketed by Merck & Co., Inc., Rahway, N. J. It is a pale yellow powder, freely soluble in water. It contains 50 per cent of gold. It is supplied in 1.1 cc. ampoules in four strengths: 10 mg., 25 mg., 50 mg. and 100 mg. of the gold salt. Each strength is marketed in boxes of 6 ampoules.

Solganal B Oleosum.—This is aurothioglucose; it contains 50 per cent of gold. Solganal-B Oleosum is marketed by the Schering Corp., Bloomfield, N. J., in ampoules of 1.5 cc. (in boxes of 1 or 10) in strengths of 10, 25 and 50 mg. of the gold salt per cc.; in ampoules of 2 cc. in boxes of 1 or 10 containing 100 mg. per cc.; and in 5 cc. vials in two strengths: 20 or 200 mg. per cc. It is a monovalent gold compound, soluble in water, but suspended in oil.

Sanocrysin.—This is a double thiosulfate of gold and sodium. It contains approximately 37 per cent of gold. It is a crystalline substance which is soluble in water, decomposes slowly, does not readily precipitate serum and is not supposed to form gold ions within the body.[67] The minimal lethal dose in experimental animals has been 30 to 50 mg. per kilo. Mollgaard has found that it retarded the growth of the tubercle bacillus in a 1 to 1,000,000 dilution and that it prevented its growth in 1 to 1000 dilution. In animal experiments, it appeared to be more toxic than Myochrysine.[74]

Aurol-sulfide.—This is said to contain 86 per cent of metallic gold and 14 per cent of sulfur in colloidal form. It is prepared as a 1 and a 2 per cent solution for intravenous, intramuscular or oral use. Injections of this preparation in mice produced severe tremors and prostration and death in about one-third of the animals. Sabin found that it had no therapeutic effect against experimentally produced chronic proliferative arthritis in mice.[74]

Myoral.—This is the aurothioglycolate of calcium and may be obtained in this country through Fougera & Co. It is an insoluble organic gold compound containing 64 per cent of gold. It has been injected intramuscularly as a 10 per cent suspension in oil of sweet almonds and is slowly absorbed. Recent experiments have shown that this may be a valuable form of gold for use in arthritis. It deserves further study.

Parmanil.—Hartfall and his associates used this gold preparation in 50 cases of rheumatoid arthritis. It has a gold content of 50 per cent and is put up as a 2 and 5 per cent oily suspension. Beginning with 0.5 cc. of the 5 per cent suspension (0.025 gm. of gold) the dose was increased at weekly intervals to 2 cc. (0.1 gm. of gold). A course consisted of 12 injections (or a total of approximately 0.6 gm. of gold).

Crisalbine and Lopion.—These are preparations for intravenous use. Crisalbine is gold sodium thiosulfate; Lopion is gold sodium allylthio-urea benzoate. One should not encourage intravenous gold therapy inasmuch as intramuscular injections are equally effective and theoretically less dangerous. The intramuscular route is often easier in the more crippled cases, and the more severe reactions are less frequent than following intravenous therapy.

Aurocein.—This is a sterile 5 per cent solution of a sulfhydryl-gold-naphthyl-trisulfo-carbonium derivative; it is supplied in 2 cc. ampoules, and is distributed in this country by Vincent Christina & Co., Inc., New York City.

Calcium Aurothiomalate.—This has been prepared as a suspension in oil of sweet almonds. Sabin and Warren have shown that in mice, the margin of complete safety is more than 100 times greater for calcium aurothiomalate than for sodium aurothiomalate. This preparation is not yet available commercially.

Approximate Expense to the Physician of Gold Salts.—This is variably but averages $.60 for an ampoule containing 0.05 gm. and $.90 for 0.1 gm.

To date no gold preparation for use in arthritis has been accepted be the Council on Pharmacy and Chemistry of the American Medical Association.

Dose of Gold Salts in Rheumatoid Arthritis

The following schedule results in the least number of toxic reactions; although doses are small, therapeutic results with this dosage schedule are in the long run approximately as good as with the larger individual doses; relapses of the disease occur less often. It must be remembered that any schedule of dosage for gold salts is subject to change inasmuch as the optimal dose is not yet known, and patients vary markedly in their tolerance to gold salts.

1. If either gold sodium thiomalate (*Myochrysine*) *or* gold thioglucose (*Solganal-B-oleosum*) are used:
   - (*a*) *All injections* are given *intramuscularly.*
   - (*b*) All doses as specified hereafter are in *mg.* of the *gold salt, i. e.,* mg. of Myochrysine or Solganal-B-oleosum.
   - (*c*) The first 4 injections are 5 mg. each, injections being given twice a week.
   - (*d*) *All subsequent injections are given at weekly intervals..*
   - (*e*) Fifth and sixth dose: each 10 mg.
   - (*f*) Seventh to fourteenth injections, inclusive: each 25 mg.
   - (*g*) *If the patient is improving,* and the sedimentation rate is decreasing, continue weekly injections of 25 mg. each for an additional twenty-four weeks, *unless signs of toxicity appear.*
   - (*h*) *If the patient has not improved following the first 14 injections,* increase the weekly dose to 50 mg. and continue this dose for a total of twelve weeks. *If no improvement is noted at the end of this period, it is doubtful whether further courses of gold therapy will result in improvement.* In our experience, it is usually futile to repeat the course of therapy in those who have not been improved at all by such a first course.
   - (*i*) *If improvement has occurred following either* (*g*) *or* (*h*) *above,* give a rest period of six to eight weeks during which no gold is given. If a relapse in the arthritis occurs, gold therapy may be begun in six weeks; in other cases, permit eight weeks between courses. Then *repeat the entire course of therapy from* (*c*) *to* (*g*) *inclusive;* as before, if improvement does not occur following 14 injections, the dose may be increased to 50 mg., as in (*h*).
   - (*j*) The dosage per series (as above) totals 840 mg. of gold salt, or 420 mg. of metallic gold.

Dose of Gold Salts in Rheumatoid Arthritis—(*Continued*)

   - (*k*) *In some cases,* if gold is tolerated, if improvement is occurring, but if the sedimentation rate has not yet become normal, *a third course of gold may be given.*
   - (*l*) Some workers believe that one should give approximately 50 per cent more Solganal-B-oleosum than Myochrysine to obtain biologically equivalent effects inasmuch as plasma gold concentrations following Solganal-B-oleosum average about 65 per cent of those following a similar dose of Myochrysine. We have been able to obtain results from the smaller doses of Solganal-B-oleosum which seemed comparable in the long run to similar doses of Myochrysine.
2. If *gold sodium thiosulfate* is used, the *above doses should each be increased by approximately one-third* inasmuch as this preparation contains only 37 per cent metallic gold (as compared to 50 per cent in Myochrysine and Solganal-B-oleosum). Although this salt may be given intravenously, intramuscular injections are advised as there seems to be no particular advantage to the intravenous route.
3. *Other gold preparations:*
   - (*a*) Most workers agree that *colloidal gold salts* usually produce results far inferior to other gold preparations, noted previously. Colloidal gold salts are not recommended, at present, in rheumatoid arthritis.
   - (*b*) *Calcium aurothiomalate* (gold calcium thiomalate) varies widely in its behavior from patient to patient. Insufficient data have been reported as yet on this preparation to come to a final conclusion concerning it; it is not yet available commercially. It was hoped that toxic reactions would be fewer with this substance.
   - (*c*) Although many other preparations of gold are available, insufficient work has been published concerning these to permit a final evaluation. *The three preparations recommended at present* are *Myochrysine, Solganal-B-oleosum* and *gold sodium thiosulfate.*

Precautions During Gold Therapy

1. *The patient or his family must appreciate the possibilities of toxic reactions.*
2. The physician must recognize the early signs of intolerance.
3. The patient should be questioned and examined before each injection —for signs of toxicity—especially pruritus, rash, sore mouth or metallic taste, anorexia, jaundice or persisting indigestion.
4. It is preferable to *examine the urine* and to *estimate the white blood cells, hemoglobin and differential pattern before each injection.*
5. It is advisable to *stop treatment* (at least temporarily) *if any evidence of toxicity appears;* some of these individuals can then tolerate subsequent treatment.
6. *Absolute contraindications to gold therapy* include a history of purpura or agranulocytic angina; definite renal or hepatic disease; severe diabetes mellitus; pregnancy; hemophilia; severe anemia or hemorrhagic tendency; colitis; severe eczema or chronic dermatitis or urticaria; severe bronchial asthma; and the presence of any serious systemic illness other than the rheumatoid arthritis. Psoriasis is not a contraindication to therapy.

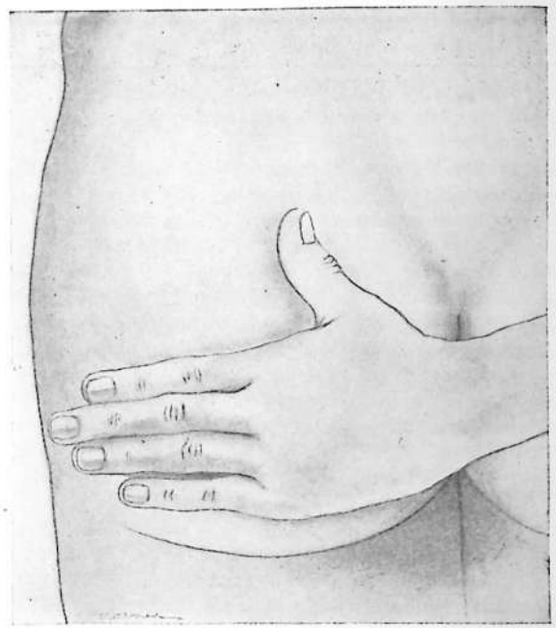

FIG. 105.—Technic of intramuscular injection. Left hand draws down buttock (Stokes, Modern Clinical Syphilology, courtesy of W. B. Saunders Company.)

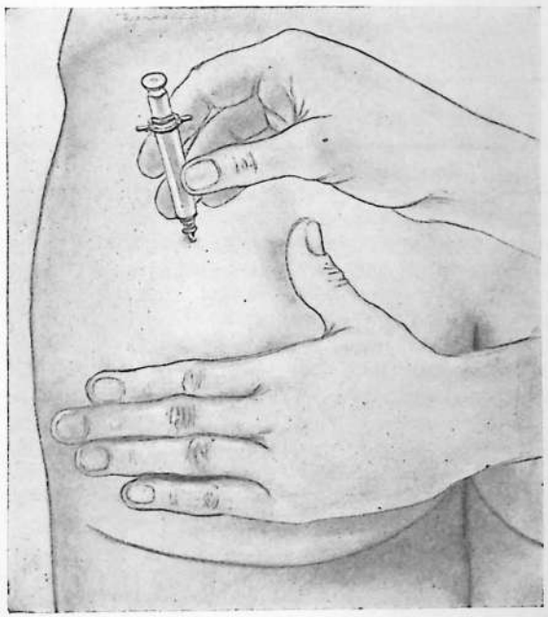

FIG. 106.—Introduction of syringe. (Stokes, Modern Clinical Syphilology, courtesy of W. B. Saunders Company.)

FIG. 107.—Aspiration to test for the presence of the needle in a vein. (Stokes, Modern Clinical Syphilology, courtesy of W. B. Saunders Company.)

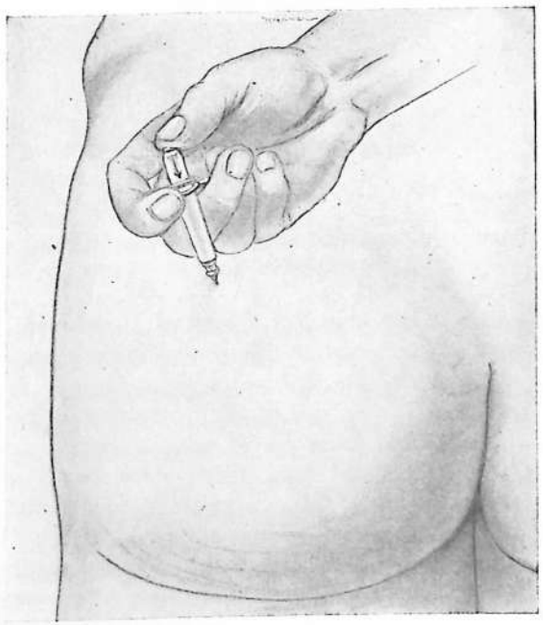

FIG. 108.—Injection; the solution should flow in freely and should not have to be forced. (Stokes, Modern Clinical Syphilology, courtesy of W. B. Saunders Company.)

GOLD SALTS USED MOST OFTEN IN THE UNITED STATES IN THE TREATMENT OF RHEUMATOID ARTHRITIS

Chemical name	*Trade name*	*Soluble in water*	*Sold commercially as*	*Per cent of metallic gold*
1. Sodium aurothiomalate	Myochrysine	Yes	Aqueous solution	50
2. Aurothioglucose	Solganal-B oleosum	Yes	Suspension in oil	50
3. Sodium aurothiosulfate	....	Yes	Aqueous solution	37
4. Calcium aurothiomalate	Used only experimentally thus far	No	Suspension in oil	50
5. Colloidal gold sulfide	Aurol sulfide	No	Colloidal suspension	87

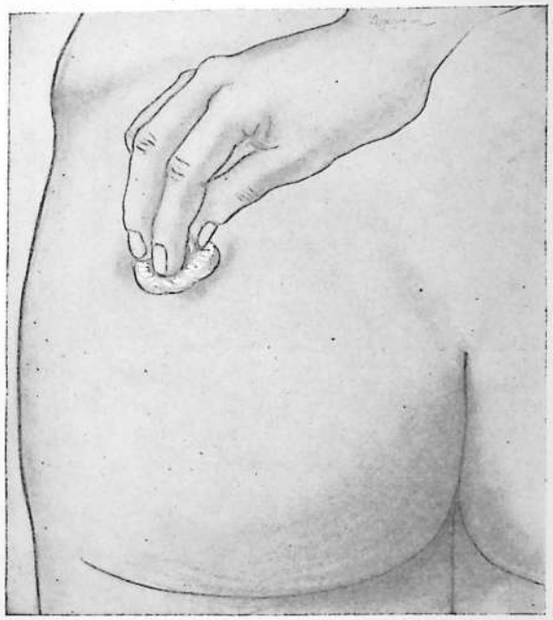

FIG. 109.—On release of the left hand, the tissues of the buttock return to their normal position, producing a valve action. The fluid is spread out in the epifascial tissue and leakage is prevented by massage with a cotton pledget. (Stokes, Modern Clinical Syphilology, courtesy of W. B. Saunders Company.)

Some investigators have found that *50 mg. of Myochrysine* (sodium aurothiomalate) (*i. e.*, 25 mg. of gold) *weekly gives clinical results equally as good as with the 100 mg. doses of the gold salt with a reduction in toxic reactions from 41 to 17 per cent, and with a marked reduction in the severity of the reactions. With the smaller doses of gold salts, severe reactions such as prolonged exfoliative dermatitis and nephritis are very uncommon.* When one attempts to reduce the dose of Myochrysine still further, *i. e.*, 25 mg. per week (equivalent to 12.5 mg. of gold), good results may be obtained in some cases, but Freyberg has reported that clinical results were usually inferior to the 50 mg. dose.[26] When 100 mg. of gold salt was given weekly, 65 per cent of patients were benefited; when 50 mg. was given weekly, 63 per cent were benefited. When 25 mg. was given weekly 50 per cent were benefited.[84] Price and Leichentritt agreed with Smyth and Freyberg that smaller doses of gold (50 mg. of gold salts per week) gave results just as satisfactory as larger doses. We were able to confirm these observations and, in addition, we observed definite improvement following doses as small as 10 mg. of the gold salt.

Lansbury used small doses of gold salts (10 to 25 mg. per week of the gold salt) and reported that improvement occurred more slowly than with larger doses and relapses occurred more quickly but that *the ultimate results compared favorably with those using larger doses.*[50] It was his opinion that the far greater degree of safety (following his smaller doses) compensated for the slower response. He concluded that gold therapy was justifiable and that it was a valuable supplement in the treatment of rheumatoid arthritis. He continued these smaller doses until a total of from 500 to 600 mg. of gold salt was given as a course.

In giving any gold preparation, one should begin with very small doses and gradually increase these until the desired therapeutic dose is attained, if no untoward reactions have occurred.[3] Although it is true that preparations of gold may be toxic, practically all therapeutically effective drugs are potentially toxic.[26]

Gold may be given to older patients. In one series, 55 per cent of the patients were past the age of fifty when gold therapy was begun. Cecil treated two children with rheumatoid arthritis with gold salts; individual doses did not exceed 25 mg. of gold salt and the total dose of gold salt did not exceed 0.5 gm. In one child, a complete remission occurred following gold therapy, persisting to date over five years. The other child improved under treatment (and was still under treatment at the time of publication of the report).

Cecil reported that if the patient received no benefit from the first series of gold, he was not likely to obtain a striking beneficial effect from further courses. On the other hand, Forestier noted that he never saw a permanent cure from only one series of injections.

In Cecil's group, each patient received a total dose of gold between 1 and 1.5 gm. per series; most patients required at least two courses and many had four, five or more courses. Gold was discontinued immediately if toxic reactions were observed.

No one has yet been able to determine the factors which are essential in obtaining a remission with gold salts. Cecil found that the majority of those who developed remissions were (1) treated early in the course of their disease, (2) received the larger doses of gold, and (3) showed a rather rapid drop in their sedimentation rate.

Using gold sodium thiosulfate, Snyder gave 14 injections per series, beginning with 5 mg. of the gold salt, then increasing this to 10 mg., 15 mg., 35 mg., 50 mg., 75 mg. and then giving 8 weekly injections of 100 mg. of gold salt each.[85] His patients were given at least two courses of treatment, with an interval of at least two months between treatments. Treatment was discontinued at the end of three courses if improvement

had not occurred. Snyder gave the gold salt intravenously; we see no advantage to the intravenous over the intramuscular route.

Freyberg reported that 55 per cent of patients who completed one course of gold therapy were moderately or markedly improved while 68 per cent of those who completed two courses of therapy were similarly benefited.

Of 12 patients treated with colloidal gold sulfide, only 25 per.cent showed a moderate degree or more of benefit while from 64 to 66 per cent were moderately improved (or more so) following either sodium aurothiomalate (Myochrysine) or sodium aurothiosulfate.[84]

Tarsy reported that 19 of 36 patients with rheumatoid arthritis showed definite improvement following three series of injections of *colloidal gold;* 1 gm. of metallic gold was the total injected per series; an interval of six weeks was allowed between each series.[89] There was an almost complete lack of toxicity in the series. Tarsy believed that the deposition of finely divided particles of gold in the histiocytes of the synovial membrane should result in the removal of inflammatory products, stimulate the general reticulo-endothelial system, and increase the defense mechanism in the synovial membrane. Freyberg and his associates, however, *discontinued the use of colloidal gold because results with the colloidal gold preparations were so poor;* they agreed that toxic reactions did not occur but attributed this to the fact that the colloidal gold was removed from the circulation so quickly.[31] Freyberg's group believed that one cannot expect therapeutic value from gold preparations if they are absorbed from the circulation so rapidly that significant plasma gold concentrations are not maintained.

Hartung reported his observations on the treatment of 264 cases of rheumatoid arthritis with gold salts.[41] He used gold sodium thiomalate (Myochrysine) intramuscularly, beginning with 10 mg. as the first dose, 25 mg. as the second, and 50 mg. as the third and subsequent doses in the series. The 50 mg. (of Myochrysine) was repeated each week until a total of 1000 to 2000 mg. had been given. Intervals between injections varied from three to seven days. (In our patients, although the first 4 injections of small amounts of gold were given twice a week, all subsequent injections were given weekly. This probably is of benefit in decreasing the toxic reactions). Hartung varied the dose of gold depending upon the patient's response and tolerance to the drug. He did not believe that further use of gold was indicated if there was no improvement following the first complete course. If the disease was arrested by the first course, a second or even a third course was given, with intervals of two or three months between courses.

Before Each Injection of Gold

1. Perform a white blood count, hemoglobin estimation and differential blood count.
2. Examine the urine for albumin, red blood cells and casts.
3. Question the patient regarding itching, sore mouth or gastrointestinal upsets.
4. Inspect the skin and mouth; note the presence of icterus (scleræ).

PRECAUTIONS DURING THE COURSE OF CHRYSOTHERAPY

The following routine is recommended in an effort to prevent or detect any complications immediately:

A thorough and detailed history including previous hemorrhagic tendencies, allergic phenomena (especially drug sensitivity), skin eruptions, bronchitis, renal or hepatic disease, diabetes mellitus, etc.

A complete general physical examination.

Urinalysis each week.

Blood count each week (leukocytes, differential pattern, hemoglobin and approximation of platelet number from stained smear, *i. e.*, plentiful, decreased, increased or absent).

Sedimentation rate each month.

Blood Wassermann reaction before treatment is begun.

These examinations are made before the onset of treatment. If even a questionable contraindication to treatment is found, gold therapy is not employed. In addition, the patient or a responsible member of the immediate family is asked to sign the following statement which is witnessed by two additional persons, whose signatures are affixed:

Date:_____

I am fully aware of the complications and reactions which may follow the use of gold salts in the treatment of rheumatoid arthritis. I realize that the mortality following gold therapy may reach, and at times even surpass 1 per cent. I know that not all patients are improved by gold therapy and that it is possible that some patients may be made worse. However, in view of the possibility of improvement following the use of intramuscular gold treatment, I am willing to assume full responsibility for any complication which might arise because of this form of therapy.

Signed_____

Witnessed:

_____

_____

In this country, until recently gold salts had been reserved for those patients in whom other forms of therapy were of no avail. This may be an error inasmuch as chrysotherapy usually results in a larger percentage of improvement in rheumatoid arthritis if given early; however, until less toxic compounds are found or until a method is devised to prevent serious complications, one is justified in a trial of conservative therapy before instituting parenteral gold. On the other hand, in a patient who is rapidly becoming worse, or in dealing with a severe arthritis not responding well to conservative measures, the cautious use of gold in proper dose is justifiable and indicated, if given by one well trained in this field.

During the entire course of gold therapy, a blood count and urinalysis should be performed each week. The appearance of a moderate amount of albumin, erythrocytes, or casts in the urine, or persistent leukopenia,

neutropenia, thrombocytopenia, or a sudden drop in hemoglobin are indications for immediate cessation of treatment. If other complications (as noted previously) occur, treatment should be suspended until these have cleared completely and the treatment may be resumed again. *With the occurrence of any serious complications (agranulocytic angina, aplastic anemia, purpura hemorrhagica, exfoliative dermatitis, acute liver necrosis, acute yellow atrophy, nephritis, etc.) the gold must be abandoned permanently.*

After a rest period of six to eight weeks following the first course of gold, a second complete course may be given. If no untoward effects are noted, this course of therapy may be repeated two or three times (following the precautions noted above).

Toxic Reactions Following Chrysotherapy.—These might frighten even the boldest physician. About 35 to 40 per cent of patients treated with gold in doses used formerly had toxic reactions. In 10 to 20 per cent of patients receiving chrysotherapy, moderately severe reactions occurred; severe reactions were seen in 6 per cent of cases and fatalities in 1 per cent or less.[63] *Almost all reports are in agreement concerning the toxicity of gold compounds.* The incidence of reaction has been decreased markedly by the use of smaller individual doses and severe toxic reactions occur very infrequently with small doses.

Reactions Following Gold Therapy

The following reactions or complications may be noted following gold therapy:

Cutaneous (*erythema, generalized pruritus, exfoliative dermatitis*, exanthemata, papular eruption, urticaria, desquamation, soreness, morbilliform rash, furuncles, hyperkeratosis of the soles, labial edema, erythema nodosum, lichen planus, leukoplakia and purpura, herpes zoster, herpes labialis, folliculitis, dermatitis pigmentosa).

Oral (metallic taste, ulcerative stomatitis, sore gums, sore tongue, glossitis, hemorrhagic gingivitis, dysphagia, loss of taste, anesthesia of the tongue).

Gastrointestinal (*nausea*, vomiting, epigastric distress, colicky pains, hiccough, *diarrhea*, rectal spasm and ulcerative enteritis).

Renal (*albuminuria, hematuria*, nephritis, oliguria, anuria, uremia).

Cardiac: precordial discomfort; hypertension.

Hematopoietic (*agranulocytic angina, purpura hemorrhagica, aplastic anemia*, secondary anemia, macrocytic anemia, eosinophilia).

Hepatic (jaundice, hepatitis, *acute yellow atrophy*).

Respiratory (cough, "gold bronchitis," epistaxis, pulmonary edema, hemoptysis).

Nervous (eighth nerve deafness, headache, dizziness, tinnitus, insomnia, herpes zoster, peripheral neuritis, crural neuritis, cerebral vascular accidents).

Ocular (conjunctivitis, keratitis, iritis, retinitis).

General (loss of weight, malaise, shock—following intravenous injections, fever, death).

Arthritic (pains in the joints, temporary increase in joint swelling and stiffness).

The *most severe reactions following gold therapy* (which fortunately are infrequent) include:

Acute yellow atrophy of the liver.
Agranulocytic angina.
Aplastic anemia.
Purpura hemorrhagica.
Severe exfoliative dermatitis.
Ulcerative enteritis.

The *mortality following gold therapy* has been as high as 3 in every 100 patients so treated; in one series of 100 patients, 3 died of severe exfoliative dermatitis following gold therapy. With the present compounds and dosage, the mortality is less than 1 per cent.

Local reactions following the injection of gold salts are rare; a short period of massage of the area of intramuscular injection will usually prevent the formation of hard nodules in the tissues. In about one-third of the cases, pains and temporary swelling of joints appear; these are more common at the beginning of the series of treatment, and are usually promptly relieved by aspirin.

If small doses of gold are employed, general reactions (as fever, malaise, etc.) can usually be avoided, although pruritus and urticaria are rather common. Both agranulocytic angina and purpura hemorrhagica have occurred in about 1 of every 200 cases so treated. Either of these two may appear suddenly and prove fatal.

In some patients, as much as 7 to 9 gm. of gold salts were given without the development of toxic reactions, supporting, to some extent, the contention that the toxicity of gold salts is probably a result of individual drug sensitivity rather than heavy metal intoxication *per se*.[62]

Less than 10 per cent of Cecil's patients had to discontinue gold injections permanently because of intolerance to the drug.

In 10 per cent of Hartung's cases, toxic reactions were severe enough to warrant stopping the drug. The incidence of all toxic reactions in one series treated with Myochrysine was 38 per cent; in order of frequency, these were pruritus, dermatitis, stomatitis, renal irritation, leukopenia, thrombopenia with or without purpura and toxic hepatitis.

Therapy was interrupted in 18 per cent of Price's series because of toxic reactions. Some form of toxic reaction was noticed in 38 per cent of those treated. In one instance, the reaction occurred after only 25 mg. of Myochrysine; all other toxic reactions followed the administration of large doses of gold.

In Cecil's series of 245 cases, 38 received inadequate gold therapy. In half of these treatment was stopped because of toxic reactions; an additional 9 patients stopped treatment because they did not show immediate improvement. Small doses of gold were given in 2 children with rheumatoid arthritis; the cause of discontinuance of therapy was not determined in the 8 remaining cases.

Frequent platelet counts have been made during the course of gold therapy but afforded very little information or assistance.

Snyder and Traeger reported an incidence of only 17 per cent of toxic reactions with sodium aurothiosulfate.[85,87] Toxic reactions occurred after as little as 15 mg. of gold and there was no period during the administration of gold in which one was certain that the patient had escaped the danger of toxic manifestations. Women were eight times more susceptible to toxic reactions than men.

Some patients who receive gold salts describe an increase in soreness and stiffness of the affected joints within a few hours after each injection; this persists for twelve to twenty-four hours.

Additional caution should be exercised in the use of gold salts in individuals with a personal or family history of allergy.

It is advisable to *stop chrysotherapy at the first sign of any untoward reaction,* however trivial this may appear. With complete disappearance of skin or oral lesions or of gastrointestinal or mild renal lesions, treatment may be resumed cautiously and with small doses.

Toxic reactions may appear at any time between the first injection up to several months after cessation of therapy.

Dawson and his associates reported the following *toxic reactions* among 100 patients adequately treated with gold salts: *pruritus*—50 patients; *dermatitis*—48; *stomatitis*—25; *gastrointestinal symptoms*—26; change in taste—21; *upper respiratory symptoms* or *hematopoietic* changes—19 each; *renal and hepatic*—2 each.[14] Moderately severe complications included 2+ *albuminuria* in 6 patients; *leukopenia* in 4; gastrointestinal symptoms in 4 and *mild hemorrhage* in 3. Five patients developed *exfoliative dermatitis* and 3 showed a marked stomatitis. One of the patients died of *aplastic anemia. The reactions were not serious in the majority of instances and it was later possible to continue therapy in most instances* after temporarily discontinuing the drug or reducing the dose. One patient developed *nephrosis,* which appeared 11 months after the cessation of chrysotherapy. The *single fatality* occurred during the second course of gold in a fifty year old patient who had previously been successfully treated with radium for a carcinoma of the cervix and who had also received large amounts of bismuth and arsenic because of a 4+ serology.

In some instances, severe reactions were observed without warning despite the most careful observation.

Cecil reported toxic reactions in 42 per cent of 245 cases. In order of frequency, the reactions in this series were: squamous dermatitis (or less frequent skin lesions such as erythema, herpes zoster, herpes labialis, folliculitis, dermatitis pigmentosa and furunculosis)—52 cases; gastrointestinal symptoms—18; stomatitis—13; exfoliative dermatitis—11; purpura and marked albuminuria—3 each; jaundice and bronchitis—2 each; agranulocytosis—1 case.

The immediate nitritoid reactions which were noted several years ago with some lots of Myochrysine have now been eliminated.

Exfoliative dermatitis was the most prevalent of the severe reactions; in 4 of these 11 patients, the lesion was generalized and required hospitalization. *Gastrointestinal symptoms* (nausea, colicky pains and diarrhea) *were usually mild* except in a woman of seventy who died following an *ulcerative enteritis* which developed after her *first* dose of Myochrysine. Both of the patients who developed jaundice made an uneventful recovery; there was a temporary remission of their arthritic symptoms during the bout of jaundice and for a few months following this, as is commonly seen following jaundice.

Purpura which developed in three instances cleared up promptly when gold was discontinued. The patient who developed *agranulocytosis* with intense angina recovered.

Large numbers of granular and hyaline casts appeared in only one case, but eventually cleared up almost entirely with no impairment of renal function.

The factors which are responsible for toxic reactions in some patients are still unknown.

Smyth and Freyberg reported that 41 of 80 cases treated with gold showed some toxicity. Albuminuria occurred in 9 cases and hematuria in 7; casts were seen in 4. Hypertension and retinitis occurred in 1 instance.

Toxic Skin Reactions.—The *most common toxic reactions following chrysotherapy* are those in the *skin.*[40] *Pruritus* is very frequent and may be localized to one or more parts of the body or generalized. *Erythema* is a frequent complication; this may also be localized or generalized. *Lesions resembling lichen planus* may be seen, confined to only one or two areas, or scattered in numerous patches over the body. These lesions vary in size from a few mm. in diameter up to patches 3 or 4 cm. in diameter. *Urticaria* occurs occasionally. *Exfoliative dermatitis,* the most serious skin complication, is *rare.* When gold compounds are stopped temporarily, the erythema, pruritus and urticaria usually clear within a few weeks but the lesions resembling lichen planus may persist for several months. Exfoliative dermatitis may become widespread; unfortunately, there is no specific treatment for this complication.

Twenty-five per cent of Price's patients had some type of skin reaction varying from a simple erythema to extensive papulosquamous eruptions involving the entire body. Fine desquamation of the eyelids was noticed in two instances. Pruritus was commonly found without a rash. In over two-thirds of the cases, the rash made its appearance before the sixth injection of gold. It usually lasted less than two weeks, if the gold was temporarily discontinued.[62] When the rash had cleared there was no residual pigmentation of the skin. The erythematous skin areas showed, on histological study, dilatation of the capillaries in the papillary layer and edema and infiltration with lymphocytes, plasma cells and histiocytes.

Although psoriasis was not considered to be a contraindication to gold therapy, the psoriatic lesions may become more marked in some instances with gold therapy.

Gold dermatitis is probably not due to a parenchymatous poisoning effect from accumulations of large amounts of gold in the skin inasmuch as Freyberg found *no consistent differences in the gold content of the skin in patients with or without dermatitis.*[14,31,84]

Freyberg believes that generalized prolonged dermatitis following gold

therapy results from a protoplasmic poisoning from the effects of accumulated amounts of gold and that this may be largely eliminated or decreased in intensity if one avoids large, frequent doses of gold.[29]

Toxic Mouth Lesions.—Second in frequency to the skin lesions are the mouth lesions, usually manifest as a *soreness,* especially *in the tongue;* numbness of the tongue may be noted. A very painful *ulcerative stomatitis* occurs occasionally and in some instances, one may note small ulcerations on the buccal mucosa. The mouth lesions usually disappear when gold therapy is discontinued although the ulcerative lesions may persist for two or three months.

In 4 per cent of cases, superficial *ulceration of the buccal mucosa* occurred; the lesions persisted for several months after stopping gold therapy in 2 of these. *Glossitis* of several months duration followed the mucosal lesions. Conjunctivitis was noted in 3 instances and facial edema in 1. Two patients showed bronchial irritation ("gold bronchitis") and 1 complained of a metallic taste.

Gastrointestinal Complications.—The frequency of gastrointestinal complications is very variable; in some series, these are infrequent. Ten per cent of patients in one series experienced abdominal cramps and diarrhea. These reactions may be mild with only *indigestion* as the symptom, or *may be severe* with abdominal cramps, nausea, *vomiting and diarrhea*—which may be bloody if an *ulcerative enteritis* occurs; this latter is a very infrequent occurrence. These symptoms usually disappear in several weeks following cessation of therapy except in the case of ulcerative enteritis, which may occasionally prove fatal.

Jaundice is an *infrequent* complication following gold therapy; this results probably from a toxic *hepatitis* and may run a prolonged course. Several fatal cases of *acute yellow atrophy of the liver* have been mentioned in early reports.[13]

Renal Complications.—*Slight albuminuria* is *seen frequently* and need not be a cause for cessation of chrysotherapy. Any degree of *hematuria* or *marked albuminuria should be a signal for at least temporary discontinuance of the gold;* hematuria and albuminuria will usually disappear in a short time after the gold is stopped.

About half of Price's series showed minute amounts of albumin in the urine at some time during the course of chrysotherapy but no other evidence of renal irritation or damage occurred in these cases. In 4 patients significant amounts of albumin were found and clinical evidence of *nephrosis* was present in these individuals.

Hematologic Complications.—Blood dyscrasias are very infrequent toxic reactions to gold, but may prove fatal. These include *thrombocytopenic purpura, agranulocytosis* and *aplastic anemia.*

Following large doses of gold, the *anemia* of rheumatoid arthritis may become somewhat worse; when small doses of gold (25 mg. of the gold salt) are used each week, the blood picture definitely improves.

Eosinophilia exceeding 5 per cent may be noted not infrequently. In 1 instance, the eosinophil count rose to 90 per cent with an associated rash and delirium when 500 mg. of gold salts had been administered. High eosinophil counts are not always associated with low sedimentation rates.

Only 1 patient in Price's series developed *agranulocytosis;* this persisted intermittently for five months with ultimate recovery.

Of the *hematologic complications,* about one-third of the cases of purpura have been fatal. In one series, every patient treated with gold showed a considerable fall in the platelet count immediately after the first dose; this decrease persisted for several days.

Hemorrhagic purpura, a rare complication, is probably the most serious. Three of Price's patients showed *thrombocytopenia;* 1 of these died (and an extensive *subdural hematoma* with multiple petechial hemorrhages scattered throughout the body were found at autopsy).

A high incidence of basophilia (70 per cent) was observed following gold therapy by some workers;[35] others were unable to confirm this.

Fatal aplastic anemia has occurred following gold therapy and about *50 cases of agranulocytosis (with 25 deaths) have been reported.* Fatalities have been reported from thrombocytopenia associated with hemorrhagic syndromes.

Vasomotor Reactions.—The *vasomotor reactions* with flushing of the face, generalized warmth, giddiness and vertigo are not often seen with present preparations of gold. *Precordial discomfort* was reported rarely following the injections and in one instance a true *anaphylactic reaction* developed immediately following an injection of gold.

Joint Pain.—Lintz reported that a large number of his patients experienced an increase in their joint pain, sometimes accompanied by a rise in temperature, during the first two to six weeks of gold therapy. In his series, most of the individuals experiencing this type of reaction showed a satisfactory response later with continued gold therapy.

A temporary increase in joint pains and stiffness occurred in a few patients following gold therapy. Transitory blurring of vision occurred in 2 instances.

Neurologic Reactions.—Toxic reactions such as *peripheral neuritis* and syndromes resembling encephalitis or cerebrovascular accidents may occur.

Nervous complications are rare following chrysotherapy.[51] However, 4 instances of severe polyneuritis were noted; all of these began during the course of treatment and resulted in severe pain in the feet with a rapid development of unsteadiness, difficulty in walking and paresthesias. In each of these 4 patients, almost complete paralysis of the feet occurred with loss of postural sense in the toes, impairment of pain sensation over the feet and lower legs and absence of ankle jerks. Knee jerks were preserved. Slight subjective disturbances were noted in the hands. These patients did not improve during observation for several months; the paralysis of the feet persisted.

Prediction of Toxic Reactions.—*There is no reliable method of predicting which patients will develop signs of toxicity or complications. The factors upon which the toxic reactions depend are unknown.* Patch tests with gold do not adequately reveal sensitivity to the metal. Certain persons may exhibit an idiosyncrasy to gold salts and may show multiple reactions after even minimal doses. In others, the question of dosage is an important factor, the greater percentage of reactions occurring with larger doses of

gold salts. It has been shown that age, sex, severity or duration of the disease appear to have no bearing on the frequency of reactions. Some of the more severe reactions seem to have followed intravenous injections; inasmuch as results following the intramuscular introduction of gold are equal to those with the intravenous route, the administration of this metal intravenously should be discouraged.

It has been shown that intoxication with gold occurs often (96 per cent of one series) in the presence of a normal sedimentation rate; in one series, dermatitis and stomatitis were common when gold therapy was continued after the sedimentation rate became normal. Ellman and his associates advised that gold therapy be stopped if the sedimentation rate falls below 12 mm., and that gold be resumed after a three or four weeks' interval in doses of only 0.02 gm. per week. Goldie, however, found no constant relationship between the occurrence of toxic complications and the sedimentation rate.[33]

Prevention of Toxic Reactions.—Toxic reactions have not been prevented by the simultaneous administration of calcium or sodium thiosulfate parenterally. Lintz suggested that large amounts of vitamin C be used in an attempt to prevent toxic reactions from gold salts. Some individuals feel that an increased vitamin C intake in the diet, particularly of citrous fruit juices, lessens the incidence of reactions.

Neither niacin nor ascorbic acid seems to significantly shorten the course of the toxic reactions, once established. Sodium thiosulfate and calcium salts have been used empirically in the treatment of exfoliative dermatitis.

Cohen and Dubbs reported less than 10 per cent of untoward reactions in patients receiving one course of gold; in those who received two courses, 18 per cent had reactions. These patients were instructed before the onset of therapy to drink large amounts of fruit juices and to eat ¼ pound of liver three times each week. These authors concluded that the reduction of untoward reactions may be due in part to the use of these measures.[10]

Treatment of Toxic Reactions Following Chrysotherapy.—*There is no specific treatment for any of these reactions; this is purely symptomatic.* Even in the very mild reactions (slight pruritus), gold therapy should be stopped until at least three weeks after the disappearance of the complicating symptoms. When chrysotherapy is begun again, minimal doses should be employed (0.010 gm.), increasing the dose very cautiously at weekly intervals until 0.025 to 0.050 gm. is given as a maximum for any single dose. *Treatment by gold should never be resumed in patients who have developed agranulocytic angina, purpura hemorrhagica, exfoliative dermatitis, marked albuminuria, aplastic anemia or severe jaundice.*

Those patients developing *jaundice* are treated as acute catarrhal jaundice and are hospitalized; an adequate dietary regimen, including a high carbohydrate and low fat intake, is given. In individuals who cannot retain food, large amounts of intravenous glucose (1000 to 2000 cc. of a 10 per cent solution) are given daily. If hemorrhagic tendencies appear with hypothrombinemia, vitamin K therapy is indicated.

Stomatitis may be relieved to some extent by the use of from 6 to 8 tablets of acriflavine each day (each tablet containing 3 mg. of the drug);[40] cevitamic acid may also be of value (100 mg. twice a day). Diarrhea and colic are usually not severe and may be checked by paregoric (4 cc. hourly for five doses in adults); occasionally, a fatal termination with ulcerative enteritis may be encountered.[1,46]

In true *purpura*, cevitamic acid should be given by mouth or intravenously (200 mg. per day).

No specific treatment is indicated if marked albuminuria appears; this is usually transient; gold therapy should be abandoned in these individuals.

Treatment of the Skin Complications of Chrysotherapy.—The mild cases of *erythema and pruritus* usually recover in a few days or weeks and are relieved by the local application of calamine lotion containing from 0.5 to 2 per cent of phenol, in addition to oral administration of small doses of bromides, 0.6 gm. (10 gr.), or phenobarbital, 15 mg. (¼ gr.) three times a day. In any patient with a serious reaction (such as exfoliative dermatitis or jaundice), hospitalization should be insisted upon.

Some of these patients are helped by sodium hypophosphite given intravenously each day as 10 cc. of a freshly prepared 20 per cent solution. Ten cc. of a 10 per cent solution of calcium gluconate may be given intramuscularly daily. Autohemotherapy has been recommended.[59] One may employ a combination of the latter two, *i. e.*, 10 cc. of a 10 per cent solution of calcium gluconate is placed in a syringe and one-half of this is injected intravenously, leaving 5 cc. of calcium gluconate in the syringe. Into this solution 5 cc. of blood is withdrawn so that a mixture of 5 cc. of blood and 5 cc. of calcium remains in the syringe. This mixture is immediately injected intramuscularly (deep into the buttocks). This may be repeated twice a day for two or three days. It has been suggested that if the involved skin area is protected from light, there may be a quicker retrogression of the rash.

Effect of Toxic Reactions on the Arthritic Prognosis.—In the majority of cases treated by gold there has been no essential difference in the end-results of the arthritis in the groups who develop toxic symptoms and in those in whom toxic reactions are absent.

Desirability of an Interval Between Courses of Therapy.—*Reactions following gold therapy may be delayed.* Many reactions occur from six to twelve weeks after the last injection of gold salts; for this reason, an interval of six to eight weeks should be allowed between courses of gold therapy. When relapses occur following gold therapy, these frequently appear about six to eight weeks after the completion of the course.

Re-institution of Gold Therapy After Appearance of Toxic Symptoms.—Following certain complications (purpura hemorrhagica, agranulocytosis, exfoliative dermatitis, marked jaundice or marked albuminuria) one should stop chrysotherapy and not use gold salts again in those patients. In the milder complications (other than those mentioned above), several weeks are allowed to elapse following the complete subsidence of reaction before chrysotherapy is begun again.

Duration of Chrysotherapy.—Treatment may be continued for several series (with rest intervals of six to eight weeks after each series) until the patient has noted relief from pain, decrease or absence of joint swelling, normal temperature and a normal sedimentation rate. The sedimentation rate is one of the best means of judging the activity of a rheumatic process. The method of performing this test and its evaluation have been described in a separate section.

In those individuals who respond well to gold therapy, the sedimentation rate is reduced, often to normal. In those who show little or no improvement under gold therapy, a striking reduction of the sedimentation rate occurs in only 5 per cent of cases (as compared with almost 50 per cent in the patients who have remissions).

Hartfall, Garland and Goldie and Cecil *et al.* report *occasional marked clinical improvement without a drop in the sedimentation rate,* although this is unusual.

In Cecil's group with remissions following chrysotherapy, *37 per cent developed a normal sedimentation rate;* in the patients who did not improve, no one developed a normal sedimentation rate after treatment.

Periodic physical examinations are made in the individual at least each three months. If the sedimentation rate becomes rapid, even though joint symptoms are lacking, another course of injections is given. The only exception to this is when the accelerated sedimentation rate is due to some other cause, such as infection, pregnancy, etc.

Continuation of Chrysotherapy if no Improvement is Noted.—Most rheumatologists agree that in approximately 10 per cent of cases of rheumatoid arthritis, in spite of all forms of treatment, improvement in the joint symptoms does not occur. In Forestier's experience with gold salts in the treatment of over 500 cases of rheumatoid arthritis, 70 to 80 per cent of all patients treated responded favorably. Of these patients, 50 per cent of the early cases and 25 per cent of the cases of more than two years' duration were apparently "cured" by from two to five series of injections, and remained well in spite of the discontinuance of all treatment during the next several years. In a meeting of the French League against Rheumatism in 1933 it was concluded that the results obtained were by far better than with any medical method previously used.[64]

"Cures" of advanced rheumatoid arthritis are rather rare. It may be possible to stop the progress of the disease and prevent further deformity and muscular atrophy, but few rheumatologists[23] will openly admit that they can "cure" cases of rheumatoid arthritis of several years' duration.

If some improvement does not result after the first series of injections, as outlined in the dosage schedule previously, it is improbable that this will occur and gold salts should be abandoned.

If one does not obtain improvement in cases of rheumatoid arthritis within a reasonable period of time (three or four months following conservative therapy) or if the course of the disease is rapidly progressive, anyone well trained in this work may cautiously employ gold therapy (if the patient or family is willing to assume the risk) after the reactions, complications and mortality of such treatment have been explained. Almost all of these patients are willing, under such conditions, to try chrysotherapy. One must explain to these individuals that every precaution will be taken (weekly urinalysis, blood count, physical examination, etc.) to detect any untoward reaction at the earliest possible moment.

Administration of Gold Salts by Mouth in the Arthritic Patient.—Absorption of gold salts given by mouth is very variable. Oral administration of gold is not advised.

Other Treatment Given Simultaneously With Gold Therapy.—*There is no contraindication to the use of other forms of therapy during the period of gold injections.* During the period of gold therapy, the patient should receive adequate mental and physical rest, physical therapy, adequate intake of calories and vitamins, and any necessary accessory symptomatic treatment (as for pain, constipation, anemia, etc.).

Indications for Gold Therapy in Arthritis.—One should attempt to treat his patients as if they were members of his family. It is much easier to come to decisions concerning treatment if this principle is kept in mind. In view of the frequency of complications and reactions, and of the mortality of almost 1 per cent attending gold therapy, some American rheumatologists have not employed gold salts unless other conservative forms of therapy (as discussed elsewhere) were of no avail.

Gold therapy should be employed in rheumatoid arthritis only when there is a rapid sedimentation rate or other very good evidence of activity of the disease. Gold salts have been of no value in burned-out inactive chronic rheumatoid arthritis. *They do not relieve contractures or produce regeneration of cartilage.*[77]

Trial of Gold Therapy in Other Forms of Arthritis.—Hartfall and his associates treated 68 cases of degenerative joint disease with gold salts but observed no cures. Some improvement was seen in over one-half of these patients but the results were in no way comparable to those in the group with rheumatoid arthritis. *Gold salts should not be used in patients with degenerative joint disease;* these individuals can usually be treated very successfully by conventional methods.

Effects of Gold Salts in Juvenile Types of Arthritis.—Gold salts have been recommended for juvenile types of arthritis (formerly called Still's disease).[36] Certain investigators have felt that in rheumatism of the juvenile type, gold salts may be harmful. Rheumatoid arthritis in children often responds well to conservative therapy.

Recent reports suggest that the results of gold therapy in rheumatoid arthritis in children are comparable to those obtained in adults. Doses should be smaller in proportion to the age and weight of the child.

Ishmael has reported good results in these children using a program of autohemotherapy, short bouts of artificial fever and sulfathiazole. This is discussed in detail under Fever Therapy.

Results to be Expected Following Therapy With Gold Salts.—If small doses of gold are used, results do not usually appear until one, two or three months after the onset of treatment. *Pain* in the joints is the first symp-

tom to be relieved definitely. This can often be judged by noting the diminution in the amount of aspirin which the patient must take for relief of this symptom. Following the relief of pain, the *joint swelling* becomes softer and tends to disappear gradually. An improvement is also obtained in the *general status of the patient;* his appetite improves, his strength returns, and the low grade fever slowly subsides.

In early cases of rheumatoid arthritis when the inflammatory process is limited chiefly to the soft tissues, improvement following injection of gold salts may be almost a cure of the process. If stiffness or muscular atrophy have occurred, these should be cared for by proper physical therapy. The results of gold therapy are not as spectacular in the more-advanced cases where cartilaginous erosion, marked muscular atrophy and contractures have occurred. However, the inflammatory process may be checked even in these patients and the pain and swelling be reduced; the addition of physical therapy will then result in marked functional improvement. Slight improvement has also been noted even in terminal stages of rheumatoid arthritis, when marked deformity and bony destruction have occurred. Some workers have reported improvement in 83 per cent of patients with active advanced rheumatoid arthritis which was refractory to other therapeutic measures.

Absolute Contraindications to Gold Therapy

Previous agranulocytic angina.
Previous purpura.
Disease of the kidneys or liver.
Severe diabetes mellitus.
Pregnancy.
Congestive heart failure.
Hemophilia.
Severe anemia or hemorrhagic tendency.
Eczema.
Colitis.

The Do's of Gold Therapy in Arthritis

Employ gold salts only in cases of active rheumatoid arthritis.
In children between six and twelve years of age, give a total of not more than 0.25 gm. of gold salts in any series.
Explain fully to the patient or a close relative the reactions, complications and the mortality.
Obtain in writing in the presence of two witnesses a signed statement by the patient or guardian (in case of a minor) releasing the physician from responsibility for any complications or fatality which might occur.
Examine the patient by history and physical examination at each visit to determine the occurrence of any reaction.
Perform a blood count and urinalysis once a week during the course of gold therapy.
Discontinue treatment at once if purpura, neutrophilic leukopenia, thrombocytopenia, cylindruria, marked albuminuria or hematuria occur.

The Do's of Gold Therapy in Arthritis—(*Continued*)

Discontinue treatment at least temporarily if any reaction occurs (whether cutaneous, gastrointestinal, pulmonary, etc.).
Attempt other types of conservative treatment for at least two or three months before using gold therapy except in rapidly progressing forms of rheumatoid arthritis. (Not all workers do this; some believe that gold salts should be given at once. We cannot yet agree with this.)
Give injections intramuscularly (preferably at least 1½ inches deep) into the buttocks; severe reactions occur less frequently than with intravenous injections.
Begin with minimal doses of gold salts and increase cautiously if reactions do not occur.
Give no more than a total of 1 gm. of gold salts as the first course of treatment.
Allow an interval of six to eight weeks between courses of treatment. This interval may be increased if improvement occurs.
Perform a sedimentation rate monthly to follow the progress of the disease.
Continue physical therapy (if indicated), the required diet, rest and other necessary forms of treatment during the periods of chrysotherapy.

The Don'ts of Gold Therapy in Arthritis

Don't insist that a patient undertake gold therapy as there may be a 1 per cent mortality attendant upon its use, as well as numerous complications and reactions.
Don't use chrysotherapy in cases of degenerative joint disease or rheumatic fever as improvement does not follow its use in these conditions.
Don't use gold therapy without explaining to the patient or a close relative in detail its dangers.
Don't promise the patient a "cure" with gold therapy, or, in fact, with any other form of therapy.
Don't neglect any symptom which the patient mentions during the course of chrysotherapy as this may be the advance notice of a reaction.
Don't give gold therapy to any individual who has had agranulocytic angina, purpura hemorrhagica, renal or hepatic damage, severe diabetes mellitus, hemophilia, eczema, severe anemia or hemorrhagic tendency, bronchial asthma, congestive heart failure or to the pregnant mother.
Don't use chrysotherapy until the patient or a legally responsible relative has given consent in writing for this treatment.
Don't forget that patients vary considerably in their tolerance to gold therapy.
Don't give large doses of gold salts to children; the dose of gold for children between six and twelve years of age should be only one-fourth of the adult dose.

Frequency of Relapses in Those Improved by Chrysotherapy.—*Relapses are common following chrysotherapy.* In one series, 34 per cent of the patients with remissions and 50 per cent of those greatly improved had relapses of their disease. Most of the relapses appeared within six to twelve weeks following discontinuance of gold therapy, but in some instances, relapses appeared as late as a year or two after remission. The symptoms were most likely to recur in those patients whose sedimentation

rates remained high despite striking improvement; the same joints were usually affected again.

In Price's series, relapses were noted in 55 per cent of all cases which showed an initial improvement; most of the relapses appeared from two to three months after therapy was stopped.

Most of the patients receive the maximum benefit from the first course of gold therapy. In those who relapse after cessation of treatment, great improvement or further remissions occur after further treatment in many, but this improvement is seldom more marked than with the original course of gold therapy. Of those patients who relapse after improvement, 50 per cent become inactive again following further gold therapy. In the group who are greatly improved following gold therapy, but then relapse, 66 per cent are improved again with another course of gold therapy.

A considerable percentage of patients who relapse following discontinuance of gold therapy have remissions or marked improvement following a second course of gold. *The relapse does not yield as strikingly to gold therapy as does the first attack.*

Freyberg has suggested that it may be possible to decrease the frequency of relapses following chrysotherapy by continuing smaller doses of gold after a series of injections has been completed so that the treatment is not discontinued abruptly. In some of his cases, at the conclusion of the regular course of therapy, he has reduced the weekly dose to half while in others he has continued treatment for several months after the termination of the usual course of therapy, giving the regular doses every two weeks in this supplemental period.

Age as a Contraindication to Gold Therapy.—Gold therapy has not been employed frequently in infants or in elderly, debilitated patients. Patients between the ages of nine and seventy-five years have been treated with gold salts. The dose must be proportionately decreased for children and treatment must be given very cautiously.

Chrysotherapy in Patients With Associated Pathologic Conditions.—Certain disease entities are distinct contraindications to gold therapy. Patients with the following associated conditions have been treated by parenteral gold without ill effects: Secondary anemias (mild, *i. e.*, as low as 50 per cent Hb.—Sahli), mild pernicious anemia, mitral stenosis, aortic stenosis, essential hypertension (without evidence of renal insufficiency), Raynaud's disease, myxedema and mild controlled diabetes mellitus. Twenty-nine of Hartfall's patients had blood pressures (systolic) over 220 mm. Hg before the onset of gold therapy, and in 75 cases the systolic pressure was above 160.

Duration of the Rheumatoid Arthritis as a Contraindication to Treatment.—Patients have been treated whose arthritis varied in duration from one month to fifty-two years. Better results are usually obtained with arthritis of short duration; this is also true of other forms of therapy in arthritis.

Acceptance of Gold Salts by the Council on Pharmacy and Chemistry.—Preparations of gold have been offered for use (orally, intramuscularly, intravenously and topically—the latter as a gargle, paste, cream, suspension or nasal instillant).[16] Gold salts have been advocated for pulmonary tuberculosis, epidemic encephalitis, bronchopneumonia, asthma, influenza, alcoholism, migraine, malnutrition, typhoid fever, septicemia, degenerative joint disease and rheumatoid arthritis. Some pharmaceutical houses have suggested their use as analgesics, antispasmodics, sedatives, hematinics, cardiac stimulants, for neurosis and to treat debility and even for acute and chronic nephritis. The Council on Pharmacy and Chemistry found a colloidal gold preparation (Colladaurum) not acceptable for N. N. R.; the preparation was promoted for therapeutic use in the treatment of cancer.[12]

The Council on Pharmacy and Chemistry has recognized the use of gold preparations only in the therapy of lupus erythematosus. In 1941, the Council was of the opinion that there was not sufficient controlled data to warrant recognition of gold salts for other than selected cases of lupus erythematosus. Others believe that gold should not be used in the latter condition.[41]

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CHAPTER XXIII

CLIMATE AND RHEUMATIC DISEASES

Cold, dampness and **chilling** *have been condemned for centuries by patients and physicians as predisposing to the onset or aggravating rheumatic diseases.*[16] However, there are very few precise data to support such a contention. Climate, weather and atmospheric conditions are made up of many variable components such as barometric pressure, humidity, temperature, atmospheric electricity, sunshine, variation in rainfall, cloudiness, storminess, etc.

It has been noted that *barometric pressure* influenced the symptoms of most arthritic patients more consistently than humidity or temperature, but it did not influence all arthritic patients in the same manner.[17] Some of these felt better when the barometer fell while others were better when it rose. *Humidity* seems to play a definite rôle in determining the symptoms of many arthritic patients. *Most rheumatic individuals feel better on hot dry days* or on cold dry days and worse on hot humid days or cold damp days.

Rheumatic pain is often increased in connection with atmospheric changes; this is noted especially with a fall of barometric pressure as occurs *before a thunderstorm.*[12] Rheumatic patients have thus become known as weather prophets. It has been suggested that such pains may be caused directly by the increased sensitiveness of the sensory nerves, arising from variation of the electrical ion concentration of the air in a negative direction.[3]

It has also been shown that there may be enormous shifts in blood volume, amounting to as high as 40 per cent, in the same city.[1] In Philadelphia, this is maximal in April and minimal in October. It has been suggested that the changes in blood volume may be the cause of symptomatic variations with weather in arthritic patients.

The *patient with fibrositis is especially susceptible to changes in temperature;* he frequently develops stiffness in the neck immediately following a draft on that area. Such stiffening from cold may be due to changes in superficial tissues, capsules, ligaments and tendons.

It is difficult to evaluate the effect of climate in ameliorating the symptoms of or in preventing a recurrence of arthritis. Most arthritics feel worse in what we consider to be bad weather. In one series in which the effects of the weather were studied on 367 patients, no consistent relationship was found between the symptoms and temperature, atmospheric electricity or humidity when each factor was considered alone.[17] Seventy-two per cent of the time, the patients felt worse as the barometric pressure fell and improved as it rose; 21 per cent of the time, the reverse occurred. Seven per cent of the time, no correlation could be noted. Storms seem to be associated with increased pains more than 90 per cent of the time, but patients vary from time to time in the response to these factors.

Segal studied his patients' subjective response to changes in barometric pressure in New York and found no confirmation of the usual impression that the rheumatic patient acts as his own barometer.[19]

Tegner found that the incidence of rheumatic disease fell off definitely and sharply as one approached the warmer climates. Northern European spas had large numbers of patients with rheumatoid arthritis, while those in southern Europe had very few of these individuals.

No one has yet definitely proved that the prognosis of rheumatoid arthritis is materially altered by a warm, dry climate, although the patient may feel relieved temporarily by a stay there. *Climatotherapy is generally not effectual by itself,* but is combined with rest, psychotherapy, physical therapy and other factors. Holbrook and Hill, who have had an extensive experience with arthritic patients in Arizona, concluded that *climate is not a panacea for arthritic patients;* in their experience, unless one employs an intensive program of therapy in addition to rest and sunshine, **chronic arthritis may continue as "a sad and crippling disease even on the Arizona desert."**[5] Unfortunately, many arthritics believe or hope that they will be cured merely by a stay of a few weeks or months in a warm, dry climate, and not infrequently make unjustified financial sacrifices to move temporarily to such a region. Rheumatoid arthritis is world-wide in its distribution even though it may be uncommon in some parts of the universe.

The effect of *ultra-violet irradiation* or *sunshine* alone is not of itself especially beneficial in the treatment of rheumatoid arthritis; during the acute stage while the patient is febrile or in markedly debilitated patients, with fever, heliotherapy may cause dizziness, further elevation of temperature and nausea and vomiting.

In the southwestern portions of this country, the barometric pressure is unusually stable and there is a low degree and low frequency of storminess, as well as a small amount of rainfall, low humidity, fairly high mean temperature and a high percentage of yearly sunshine. Because of these factors, it has been suggested that this climate may be beneficial to some sufferers from rheumatic disease.

Most individuals are in agreement as to the importance of climate in **rheumatic fever.** Mills found that attacks of rheumatic fever occurred ten to twenty times more often in the worst areas of the storm zones than in the calm warmth of the tropics. He suggested for these individuals a residence near the Mexican border which he thought would be more beneficial than the West Indies or Florida, which latter are frequented so often by storms of the tropical hurricane type, while the Southwest shows a rarity of storm changes.[9]

Paul noted a marked increase in the incidence of rheumatic heart disease in Indian school children in Montana and Wyoming as compared with those in southern Arizona; there was even a striking difference between those living in northern and in southern Arizona, where there is a difference of climate.[13] Hill and Holbrook did not observe (in a fourteen year study) a single recurrence of rheumatic fever in patients sent to Tucson from other parts of the country. Some of those who returned to their

homes in the north or east later did have recurrences of their rheumatic fever. They observed, in agreement with others, that the chance of recurrence of rheumatic fever decreases with the age of the patient, regardless of climate. It was believed that *activity in rheumatic fever subsided much faster in the climate of the Southwest* than in other parts of the country. Irradiation of the skin evidently is not a factor in this more prompt response as many of the patients received no sun baths during their therapy while convalescing; these individuals did as well as those receiving heliotherapy. Hill and Holbrook feel so strongly that the climate in certain sections of the Southwest appears to be "specific" in preventing recurrences of rheumatic fever that they advise patients with *rheumatic fever* or rheumatic heart disease to remain in the Southwest indefinitely, or at least until they have reached an age when recurrences are less likely.

Rheumatic fever has been reported to be *less prevalent in the southern than in the northern portions of the United States;* mitral stenosis is said to be twenty times as common in Boston as in New Orleans. The statistics of the Metropolitan Life Insurance Company reveal that the *mortality from rheumatic fever* (death rates per 100,000 people) *is highest* in Colorado and Utah, and in New York, Pennsylvania, New Jersey, Delaware, Maryland, Rhode Island and Massachusetts, and that the mortality rate is almost as high in Arizona as in the preceding; in the same statistical report, *the mortality from rheumatic fever was found to be definitely low in all of the southern states from South Carolina westward to and including Texas,* as well as in Kentucky, Tennessee, Oklahoma, Kansas, Nebraska, South Dakota and California. This study of rheumatic fever included rheumatic heart disease. The mortality rate was only moderate in New Mexico, but was more than twice as great in Arizona as in the states from Florida to Texas, or as in California. *The mortality rate was higher in Arizona than in Maine, Vermont, North Dakota, Minnesota, Wisconsin and Washington,* all of which are very cold regions. This is based on an accurate statistical study, and not merely on impressions.

Rheumatic fever has been considered to be uncommon in the Tropics, but *various observers have reported large series of such cases in India and Australia.* Thus, it would seem that Tegner's statement was justified—*i. e.,* that local patriotism of countries, regions or states have some influence on opinions as to the benefit of various climates on rheumatic subjects.

In Europe, countries with a high proportion of coastal area feel that "sea air" is good for the rheumatic patient; those countries with little coastal area have a disposition to recommend that rheumatic patients get away from the sea; in countries with extensive highlands, there is a tendency to attribute a great deal of importance to altitude as a favorable climatic factor.

One finds as much rheumatism on the granite hills of Sweden as on the flat islands of Holland and Denmark. Scandinavian countries usually place their sanitoria for the treatment of rheumatism by the seaside. In Great Britain, it is generally believed that cold and damp conditions, clay

soils and seaside localities are all potentially unsuitable for the rheumatic patient.[21]

Rheumatic fever is most common in the United States in the early spring and in England, in the late fall. It is common in the cold and wet months of the year.

In Hill's series, *90 per cent of patients* (with either rheumatoid arthritis or degenerative joint disease) claimed that they *were weather sensitive*—usually noting increased joint pain, stiffness and swelling when there was a marked change in barometric pressure, storminess, a damp coldness or high humidity. It is not claimed that the therapeutic results from climate (in the Southwest) are as dramatic in other forms of arthritis as in rheumatic fever. However, Hill believes that many patients with arthritis who have not been doing well on a good medical regimen elsewhere begin to improve with only the change to the dry, warm climate. Flare-ups and exacerbations were said to be less frequent in these individuals. This question will not be settled until a similar group of patients is well treated at a special spa or interested and well-organized clinic in the North (possibly in Minnesota or Wisconsin) where patients may be equally far from their homes and the worries and cares of their families, and may be equally hopeful on arriving that they will be improved.

Patients with *degenerative joint disease* and with *secondary fibrositis frequently complain of the aggravating effects of dampness and cold,* but much less consistently than individuals with rheumatoid arthritis or primary fibrositis.

Patients with degenerative joint disease and gout are apparently affected less by changes in weather than those with rheumatoid arthritis.[15] Patients with rheumatoid arthritis are said to feel better, in general on cold dry days than on cold damp days, and on hot dry days than on hot humid days. These individuals may feel even worse on hot humid days than in winter time.[2]

Patients with various forms of arthritis do not act alike in their response to changes in the weather. Some patients with rheumatoid arthritis are apparently more disturbed by changes in barometric pressure than by changes in temperature while others seem more susceptible to variations in humidity; in still others, aggravation of symptoms is thought to be caused by alterations in atmospheric electricity. Although the temperature may fluctuate as much as 50° (between winter days and nights) in southern Arizona, rheumatic patients at times feel better when they spend the winters there, possibly because of the relative constancy in barometric pressure and humidity.[16]

No certain relationship was found in a recent study in southern Australia between the incidence of rheumatic fever and the wetness of the season; no etiologic rôle could be attributed to the presence of creeks and rivers. However, in those sections in which dry heat prevailed, less rheumatic fever was noted than in the areas with combined dampness, relatively cold temperature and high rainfall.[18] Paul found that the *highest inci-*

dence of rheumatic fever in New Haven, Conn., *occurred in the two wettest districts* investigated.[14]

It has been reported that rheumatic fever is several times more common in North Dakota (latitude 47 north) than in Florida (latitude 25 north).[11,19]

After a careful study of the subject in numerous countries, Tegner concluded that local climate is a relatively unimportant factor in the incidence of chronic rheumatic disease in the temperate zone, although it may play a rôle in aggravating the symptoms of the disease.[21] Climate is probably of definite importance, however, in rheumatic fever.

It is of interest that *Lapps and Eskimos have much less rheumatic disease than those living in temperate zones* who may live indoors in poorly ventilated, artificially heated homes. *None of Scott's or Shackleton's Arctic explorers developed rheumatic disease despite the severe cold.*[15]

Hill thought that the "*microclimate*" *of the home and working place* was of greater importance than the general climate in initiating rheumatic disease.[6] In his opinion, dampness, cold, atmospheric electricity and cyclonic disturbances were unimportant, but that one should stress the use of warm, well-ventilated houses, warm porous clothing, and the consistent use of daily home methods of physical therapy, supplemented, when possible by professional physical therapy.

Mills has emphasized that atmospheric turbulence, or storminess, with its accompanying sudden changes in temperature, humidity and pressure, constitute a major disturbing factor in certain areas where cyclonic storms prevail. In his opinion, storm changes constitute a major health factor in areas where they are abrupt and frequent, but that the present evidence is too scanty to clearly understand the physiology of their effects.[10] Individuals differ very greatly in their sensitiveness to storm changes.

A high intake of vitamin B has been thought to be of great *importance in protection against severe heat;* 10 mg. of thiamin chloride per day has been shown to be more effective than tablets of sodium chloride in warding off the effects of severe heat.[10] In addition, the optimal dietary requirement for thiamine has been found to be twice as high at 91° F. as at 65° F.

It must be emphasized that in the great majority of instances, a mere residence in the southwest will not result in any marked improvement in the patient with rheumatoid arthritis;[7] in other words, **the rheumatoid arthritic should not give up his job, sell his belongings and move to the southwest with the hope that he will be cured by this procedure.** *Change in climate alone, will not result in marked improvement in rheumatoid arthritis.* If the patient moves to a warm, dry area, he should be under the care of a capable physician who has been trained in the treatment of rheumatic diseases.[8] On the other hand, *the patient with rheumatic fever will generally be distinctly improved by residence in the Tropics* or the Southwest of the U. S., Puerto Rico and similar places. Following the subsidence of his attack of rheumatic fever, it is doubtful whether the patient will have a recurrence of his rheumatic fever while living in the warm area. However, another word of caution must be added. *Tropical climate will not cure rheumatic heart disease, will not remake damaged valves and will not influence cardiac decompensation.* The moving of the rheumatic child to the Tropics or Southwest no longer assumes the importance which it did some years ago, due to the introduction of sulfanilamide. *By the prophylactic use of sulfanilamide given throughout the winter and spring months, it is generally possible to prevent recurrences of hemolytic streptococcal sore throats and of rheumatic fever in children so that there is no necessity for moving of the family in the great preponderance of cases.*

Although most arthritic patients feel worse in bad weather, and better in good weather, no one has yet determined the factor which may be responsible for such changing symptoms. It is very difficult to evaluate the effect of climate in preventing or ameliorating arthritis because of the multiplicity of factors involved.[4]

Holbrook, one of the country's leading rheumatologists, concluded that climate was not a panacea. Inasmuch as arthritis is essentially a world-wide affair, there seems to be little prophylactic value of climate in a disorder such as rheumatoid arthritis.

Hench, who has had an extensive experience in the treatment of rheumatic conditions at the Mayo Clinic, concluded that *most patients must battle their arthritic problems at home and should not be encouraged to endanger their economic status or disrupt their social ties in a vain attempt to seek a climate that cures.* It may be possible, in the future, by means of humidity and temperature control in the home, to better stabilize the environmental factors at home.[4] In a select few individuals who are made much worse during a winter in northern climates, if financial problems are *nil*, it is certainly permissible to undergo a period of intensive therapy, including climatotherapy and the advantages of a vacation.

Important lessons will undoubtedly be learned from the experience gained in the incidence and course of rheumatic diseases in various army and navy camps scattered over this country and abroad. From certain preliminary reports, several very important facts may be mentioned. It must be admitted that with the sending of troops from one part of this country to another, the troops at our northern and at our southern training bases in general come from the same parts of the country and their heredity is thoroughly mixed—*i. e.*, there should be just as many individuals susceptible to rheumatic fever in our troops in the South as among our troops in the northern camps. However, it has been noted that *only northern susceptibles get rheumatic fever, while the susceptibles in the southern camps are not similarly affected.* Furthermore, the same types of streptococci (17, 19, 30) have been cultured from the throats of troops in heavy infestations in the South—with a serious streptococcus disease rate—similar to that in northern camps, but without the occurrence of rheumatic fever among the southern troops. In other words, although the same organisms (streptococcus group A infections) are present in the throats of our troops in southern camps as in those in northern camps—*rheumatic fever is very uncommon in the southern camps.* Thus, it appears that the hemolytic streptococcus group A infection is not all that is needed to produce rheumatic fever. Although it is true that rheumatic fever is a

frequent sequel to group A hemolytic streptococcus throat infection among northern troops, this infection itself is not enough to produce the disease inasmuch as it was not followed by rheumatic fever in southern troops.

This brings up the question as to whether *meteorology* plays a rôle *in the etiology of rheumatic fever*. It has been estimated unofficially that *75 per cent of all the rheumatic fever occurring in the air force in this country was noted in a not too large area in the Rocky Mountains* (in the neighborhood of Colorado). It appears, then, that when rheumatic fever occurs in epidemic form, it requires meteorology. Among northern troops, one commonly sees the picture of respiratory infection (streptococcal), followed in approximately three weeks by rheumatic fever. It is probable that many more *severe* streptococcal infections occur among northern than among southern troops. *In the etiology of rheumatic fever,* to date, *the factors which may play a rôle include streptococcal infection, susceptibility (heredity) and meteorology.*

Florida has been proposed by some persons as an ideal location for patients with various forms of arthritis. The author has seen numerous patients return from a sojourn in that state feeling much worse than when leaving the North; the reverse has also been true. In an effort to obtain some statistics on arthritis in Florida, a compilation was made of admissions to a large hospital in Florida over a three year period. During these three years, there were 20,000 admissions to the institution. Of these, 209 were diagnosed as arthritis, giving an incidence of about 1 per cent of arthritis in a general hospital, and an incidence of approximately 0.1 per cent of the entire population in that area. Approximately half of these were diagnosed as having rheumatoid arthritis. In 15 per cent of the individuals with rheumatoid arthritis, the joint pains and stiffness became worse in that climate (in the lake region, near Orlando, Florida). In 31 patients diagnosed as having traumatic arthritis, 9 had migrated to that area after the onset of their arthritis; all of these noted aggravation of their symptoms during their stay in Florida. In 37 per cent of 65 patients with degenerative joint disease, the symptoms were aggravated since moving to Orlando. From this and similar observations. it appears that *a stay in Florida is certainly not a panacea for arthritis.*[20]

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CHAPTER XXIV

SPAS

A SPA is a health resort where one or more therapeutically valuable mineral springs are found.[12] The advantages which have been attributed to spas in the treatment of chronic arthritis include:[7,19]

Rest; the value of a vacation away from home with a complete change of scenery and occupation.

Proper exercise, hydrotherapy and physical therapy.

Psychotherapeutic value; improved morale of the patient due to his faith that he will obtain a "cure" because others have reported improvement from spa therapy.

Value of mineral baths.

Value of mineral water taken internally (this is probably of very little importance).

Improvement due to climatic conditions.

Unfortunately, American spas are not well known. It has been estimated, however, that from 40,000 to 50,000 patients take regular treatment at Hot Springs, Arkansas, and from 12,000 to 15,000 at Saratoga Spa, New York.[25] Fantus thought that many physicians in this country were disgusted with practices and quackish propaganda in some American health resorts even though more than 2000 places in this country boast of springs of some medicinal value.[12] He also stated that many of our mineral spring resorts merit distrust and skepticism because of the claims made in their advertisements. Several books have been published which describe American and European spas.[6,13,29]

The therapeutic value of spa treatment is an established feature of medical therapy based on ages of experience.

Resort therapy has, during various periods of history, been the central feature of medical care while at other times it has been reduced to the status of a superstition or a social fad or passed into the hands of the charlatan as a panacea.

The origin of health resorts probably stems mainly from the priestly healing of pagan peoples. The ancient Sanskrit Susruta distinguished six seasons of the year and described their effects upon men and animals.[14] *The rainy season from July 15 to September 15 was considered as the most dangerous to health and the change to a dry climate was strongly recommended.* The laws required that the king sojourn in such a dry locality during this season and apply suitable measures of hygiene. There was, of course, a considerable migration following the king. *Representatives of the healing art flocked to these places since bathing, massage and general care of the body were emphasized;* these localities thus developed into health resorts.

In most other instances, the origin and growth of health resorts were probably religious, with therapy centering about the sacred spring or well. The

essential purifying agent was water, which was thought to remove the spirits from the body both when applied either internally or externally. In various places, high temperatures of the waters, bubbling gases and various mineral contents gave peculiar odor and taste to the waters so that some springs and wells attained special significance through such mysterious and unusual properties.[14] Temples were built for the priests and quarters were provided for the multitude of pilgrims who made visits to the healing waters. At that time, emphasis was laid upon the divine influence emanating from the waters or some supernatural elements so that the individual was oblivious to the beneficial effect of the rest, lack of responsibilities of the home, hydrotherapy, etc., which he received.

The growth of health resorts in Babylon was political with the development of the prestige of the temples so that these became places of pilgrimage and health resorts.

As Christianity spread, these pagan healing waters became Christian healing waters and the healing effects were often attributed to some patron saint. The physical ministrations were now minimized or even abolished in some cases and mere visit and ceremony at the spring or well were thought to be sufficient for cure.

At the time of Hippocrates, the healing of internal diseases passed from the hands of the priests of Æsculapius to the rational recommendations of physicians. Following the works of Paracelsus, the mineral content of medicinal waters was stressed, but in the eighteen century, many famous waters were found to contain no more mineral ingredients than in some city water supplies.

Even in ancient times, spa therapy included the use of mineral waters internally and their application externally in the form of various baths at a great range of temperatures. The virtues of mineral waters containing copper, silver, gold, sulfur, etc., was commented upon by Hippocrates, Aristotle, Strabo, Theopompus, etc., and Galen eulogized certain of them for more specific purposes, as did Seneca and Celsus.

The early Greeks and Romans practiced underwater massage; this was carried from Rome to Egypt. At the end of the eighteenth century, Napoleon's soldiers took this back to France and Aix-les-Bains.

The famous public and private baths in Rome were skilfully designed by the greatest architects and engineers. The public baths were then often great clubs with lounges and libraries.

Later, the vapor bath with plunge, massage and rest became very important in daily life in the Mohammedan countries.

One of the earliest medical treatises on baths was written in 1489 by Savonarola of Padua; this dealt mainly with the thermal waters of Italy.[5] Charlemagne temporarily revived interest in the French mineral waters by having a bath constructed for his family at Aix la Chapelle.

Most European spas are under some form of governmental control which has maintained a high scientific interest and has aided in preventing their abuse; under this system, there is provided adequate means for the care of the poor and those of moderate means.

Spas in Europe (Great Britain, France, Germany and the Soviet Union) published financial guides and other literature which unfortunately exaggerated the recreational, scenic and climatic activities (as has been the case in some of the advertising of American spas).

Unfortunately, many English and American health resorts became fashionable watering places which catered to the paying rather than the ailing guest; this social feature has hampered the adequate development of American health resorts.[31]

Publicity for American spas often had reference to Indian patronage or to visits by early settlers and Spanish explorers.

The patrons of spas generally include middle-aged or elderly people with chronic illnesses (particularly cardiovascular, rheumatic and gastrointestinal), those convalescing from surgical operations or normal persons of middle age or older who wish to remain so.

Private corporations or individuals own most of the large spas or health resorts in this country, including Hot Springs, Virginia; Arrowhead Springs, California; Mineral Wells and Marlin, Texas; French Lick, Indiana; Glenn Springs and Sharon Springs, New York; and many others. The *Thermal Water Reservation at Hot Springs, Arkansas, is owned by the government. The Spa at Saratoga Springs, New York, is owned by the state,* while the *state of Wyoming controls the springs at Thermopolis, Wyoming. Excelsior Springs, Missouri, is a municipal development.* The watering place at Mount Clemens consists of a group of hotels or sanatoria which have grown up around the source of mineral waters in that area.[11]

Mills believes that spas of the non-stormy southwestern portion of the United States are preferable for patients with rheumatic complaints.[22]

The Federal government owns the mineral water and lands at Hot Springs, Arkansas; this water is metered to a government owned charity bath house and to many hotels and bath houses under private ownership. These latter are operated for private gain but are under strict regulations of the U. S. Department of the Interior. The springs and lands at Saratoga Spa are owned by New York State.

The *Georgia Warm Springs Foundation* was organized in 1927 mainly by the influence of Franklin D. Roosevelt to serve as a medical institution for the study and treatment of the after-effects of infantile paralysis.

During World War II, some of the larger spas in the United States were transferred to the jurisdiction of the War Department and were reorganized on a general hospital basis, being administered by the Medical Department of the Army. These *included* the large institutions at *Battle Creek,* Michigan; *Palm Springs,* California; and *White Sulfur Springs,* West Virginia.

In 1938, The House of Delegates of the American Medical Association recognized the value of those phases of medical treatment included under the general classification of health resort or spa, such as climate, thermal or mineral waters, sea water and peloids (muds). The *Committee on American Health Resorts,* with headquarters at the A. M. A., 535 N. Dearborn St., Chicago, consisted of McClellan (Saratoga Springs), Holbrook (Tucson), Smith (Hot Springs, Arkansas), Jarman (Hot Springs, Virginia) and Krusen (Mayo Clinic).

The committee has offered an opportunity to American health resorts to apply for a listing which will be compiled of health resorts which comply with the rules of the committee.

The Committee on American Health Resorts defined a **health resort** as "*an institution which gives major attention to the use of the special climatic and other natural therapeutic resources including mineral waters, peloids, etc., with which it is endowed by reason of its location.*"

Those health resorts desiring to apply for listing may do so by filling in the application blank furnished by the *Committee on American Health Resorts,* A. M. A., 535 N. Dearborn St., Chicago. The institution must furnish complete information concerning its ownership, personnel, equipment, methods of operation and method of promotion, together with copies of recent advertising, descriptive booklets, circulars, etc., or other promotional matter pertaining to the health value of the resort. The claims made for the resort must be acceptable to the committee and no resort will be listed or retained which makes unwarranted, exaggerated or misleading statements in any of its advertising. The medical supervision of the resort must meet with the approval of the committee and attendants or technicians are not permitted to prescribe treatment or to change the physician's prescription.

Jarman has defined a spa as an institution which is built around a mineral spring or springs, equipped and staffed as to utilize the water from these springs together with other therapeutic agencies for health purposes.[16] Although the springs determine the location of the spas, it requires more than a few springs to make a spa.

The *benefits from spa therapy are a result of the rest, relaxation, freedom from responsibility, baths, climate, change of environment and possibly diet.*[14] At most spas, emphasis is placed upon the triad of rest, exercise and diet.[17] Varying with their location, spas utilize various components such as sunshine, temperature, relative humidity, sea bathing, mineral springs, etc. *One of the most important secrets back of the success obtained at spas is an abundance of rest.*[38]

In an analysis of the *types of patients treated in the larger spas,* it was found that at Saratoga Spa, 31 per cent of the patients treated had cardiac and circulatory disorders while 24 per cent had rheumatic conditions; 18 per cent had gastrointestinal ailments, while smaller percentages had nervous, metabolic, skin and other disorders.

The distinguishing feature of a spa is their use of special waters for drinking, bathing or in combination. Various individuals have offered different definitions of mineral waters. In fact, in one geologic survey by the Department of the Interior, a mineral water was defined as "one sold in a bottle."[8]

McClellan has defined **mineral waters** as *natural waters with therapeutic value due to their nature and constituents.*[21]

Mineral waters have been given in several ways: (1) internally; (2)

baths; (3) inhalation; (4) hot packs; and (5) as gargles and colonic or vaginal irrigations. Drinking of the mineral waters in varying amounts is their most widespread use.

Some individuals believe that certain natural waters have properties of therapeutic value, although this has not yet been proven scientifically. Others are firmly convinced that the most important chemical in these mineral waters is water itself and that there is a great psychologic effect produced by prescribing the drinking of mineral waters according to exact instructions.

The drinking of water is almost dramatized at some spas, being done according to definite directions as to amount and time, and in some cases, to the accompaniment of music. Water is one of our best diuretics, and some waters are definite saline cathartics.

Reynolds has defined a health resort as a hospital or spa with natural therapeutic facilities for the care of chronic diseases and other institutions which offer convenient, attractive and healthful accommodations in an environment which would attract individuals requiring rest and rehabilitation and those searching for athletics, sports or even social advantages.[28]

The usual program in most health resorts is that the patient selects a regularly practicing physician of the vicinity experienced in the program of treatments offered by the spa. *Very few of these health resorts have provision for charity patients.* At *Hot Springs, Arkansas* and at the *Saratoga Spa* in *New York, the charity patient must submit proof of his inability to meet costs of therapy and medical attention is provided without charge by staff physicians.* **Living accommodations during this treatment period must be provided by the patient or his friends or by charitable organizations.**

The seasonal character of most spas aggravates their financial uncertainty inasmuch as many of these are open for less than half of the year with patronage of the recreational features naturally being much higher in the vacation months.

It has been estimated that in 1930, 100,000 Americans were treated at European spas, spending an average of $1000 each.[3]

At most spas, the majority of individuals must seek living accommodations in nearby hotels and boarding houses. Very few spas are professionally staffed; most rely in great part upon the local profession for the care of their patients.

The economic set-up of most spas is such that they cannot care for the indigent and low income classes without outside subsidies or changes in their present type of operation; *there are some provisions for low income classes and the indigent at two spas in this country* (*Saratoga Springs,* New York, and *Hot Springs,* Arkansas). In Europe, insurance and relief organizations coöperated with the spas by extensive arrangements so as to provide treatment for convalescents and the chronically ill among the low income brackets. As a basis of the loan to the Saratoga Springs Authority by the Reconstruction Finance Corporation, special arrangements are provided for the care of indigent patients.[32]

Hot Springs, Arkansas, and Saratoga Spa in New York, are the only spas

which can accommodate over 5000 patients. *Of the 103 spas in this country, 83 accommodate less than 500 patients.*

It has been estimated that nine of the most highly developed and oldest spas in the United States represent capital investments of approximately $20,000,000.

New York State has an investment at Saratoga Spa of approximately $8,800,000 in land, buildings and equipment (of which over $3,000,000 was an R. F. C. loan).

Ant and McClellan have stressed the importance of *adequate physical equipment and their intelligent use* in the successful administration of treatments in a health resort.[1] They have outlined the *requirements for such a spa,* including provisions for mineral waters, packs, massage, hydrotherapy, baths, inhalations, mechanotherapy, drinking halls, swimming pools, etc.

Elmore and McClellan have listed the *various departments which should exist for properly run large spas:* mineral water control and conservation; bath houses; hotels, boarding houses and sanatoria; recreational facilities (golf, paths, drives, winter sports, library, concerts, theatre, etc.); parks, grounds and landscaping; merchandising all by-products, including bottled waters; research and investigation; planning; maintenance and construction and publicity, promotion and advertising.

Most spas are built around a mineral spring or springs of supposed medicinal value. Before building a spa, one must be sure that the flow of such water is adequate and that its composition is rather uniform and must make proper provisions for the storage and heating of mineral waters. This is a difficult problem and *special attention must be given to the metals used for tubing of tanks, wells,* etc., which will store or distribute the waters. Copper and some of the copper alloys have been found to be the best metals for use with carbonated saline waters; if these waters are to be used internally, tin-coated red brass pipe is recommended for their conduction.[1] Copper alloy or wooden stave pipes are suggested for the handling of sulfur waters, as iron pipe lasts for short periods with such waters. *Chromium plated and nickel fixtures do not withstand the action of most mineral waters* so *that bath tub fixtures should be made of cast stainless steel wherever possible.*

One of the most bothersome and expensive problems in spa operation is the heating of mineral waters. At Saratoga Spa, the heater tanks are maintained under constant pressure and are designed so that the free gas forms a cushion at the top of the tanks. The temperature is controlled thermostatically at 135° to 140° F. by heat from acopper steam coil in the bottom of the tank. Carbon dioxide is liberated as heating proceeds; an automatic valve on top of the tank permits relief of the excess pressure. This carbon dioxide which is bled off is trapped into a sewer line and the hot water, saturated with carbon dioxide, is drawn into the bath tub; cold, highly carbonated water is added directly from storage to complete preparation of the bath.

It is important that the bath tubs be about 6 feet long and 2 feet deep and wide so that the patient may be immersed completely in a relaxed position.

Tubs should be made of porcelain; if made of iron, they should be coated with an acid resisting enamel.

Hot and steam rooms must have windows to permit observation of the patients by the attendants. The hot rooms are usually large enough so that several patients may be accommodated on reclining steamer type chairs. The heat may be provided from steam radiation; the usual temperature is from 140° to 160° F., varying with the amount of humidity.

Domed ceilings in the steam rooms prevent the condensed hot water from dripping upon the patient. Steam is usually introduced into the room as a fog through atomizing nozzles. The patients usually sit or lie on marble slabs during treatments in the small steam rooms.

McClellan has suggested that the rooms, floors, walls and equipment should be of tile, porcelain, marble or similar materials which can be readily cleaned and sterilized. Chlorinated compounds are usually employed (daily) to clean the floors in the bath and hydrotherapy rooms.

Baudisch has attempted to arouse interest in the *importance of the trace elements in biologic activity,* with special reference to the use of natural mineral water to supply these elements.[2] He has compared the effect of vitamins, which are present in our bodies in only minute amounts, with the possible effects of various minerals which are present only in small amounts in the human body; he believes that the importance of these trace elements is as yet undetermined. He has called these mineral elements "inorganic vitamins" and is firmly convinced that their insufficiency may be of much greater importance in the human economy than is at present recognized.

Mineral waters contain mainly alkaline and saline anion groups. Many thermal waters in the United States are lightly mineralized. Springs in this country most often have the sulfate anion, generally combined with calcium, magnesium or sodium.

Thermal springs are those whose temperature is above 70° F.; hot springs indicate those with temperatures above 98° F.

Mineral waters differ in their color, taste, temperature, odor, etc.

Gases are dissolved in many mineral waters; those considered of most importance therapeutically include carbon dioxide, hydrogen sulfide and radon (radium emanations).

Strong brines will assist in the venous return as well as aid the lymphatic circulation by pressure on the lower extremities.

Some mineral waters exert a laxative effect and it has been claimed that they may influence gastric motility and the circulation. Several thousand years of empiric observation is in back of their use for rheumatic disorders so that despite the lack of any scientific proof of their effectiveness, the use of mineral waters in rheumatic diseases may have more rational value than the mere effect of a warm bath and relaxation, although this is yet to be shown decisively.

The term "chalybeats" was used in former years to refer to waters with at least 10 mg. of ferrous or ferric iron per liter.

Underwater therapy is a feature of the treatment at most spas. This includes hydrotherapy, therapeutic exercise and massage. Some individuals have defined hydrotherapy as the outward application of water to the body; **balneotherapy** is used to indicate bathing at a resort or a spa.[35]

Most health resorts employ hydrotherapy to utilize the natural resources of the institution.

Hydrotherapy is one of the most useful forms of physical therapy in the treatment of rheumatic diseases. It is unfortunate that so few hospitals are equipped with facilities for such therapy.[15,24,36]

The **warm pool** relieves muscle pain and spasm in arthritis and eliminates most of the body weight by the buoyancy of the water.[27,30] In subacute and chronic rheumatoid arthritis, the increased arc of painless movements possible in under-water therapy aids in hastening the absorption of joint exudates, reduces muscular atrophy and helps to prevent adhesions.

Jarman has enumerated the various *types of baths which are available at spas:* sponge; tub; evaporation; whirlpool; carbonated (Nauheim); steam (Russian); mud; sitz; medicated; brine; sulfur water; cabinet; cold; needle douche; alternating hot and cold douche (Scotch); fan; Vichy; and Aix.

The *Hubbard-Currence tub* is a modification of the shape and construction of the Hubbard tub with the addition of a thermostatic control and an under-water douche or whirlpool. With this tub, one may begin the treatment of the patient with the water at a given temperature and gradually increase or decrease this as desired.

At the onset of treatment in the hot pool, it may be advisable or necessary to combine immobilization of affected joints in plaster while in the pool for the first few treatments in order to reassure the patient that he will not be harmed. In such cases, the plaster splints should be coated with cellulose acetate or lacquer to waterproof them.

The *optimum temperature for relaxation of spastic muscles is approximately 98° to 100° F.* Extremes of temperature are stimulating; these should not be used in the exhausted, weak, run-down individual with low blood pressure. For this latter type of patient, hydrotherapy should be applied in the neutral range.

If the temperature of the therapeutic pool is kept at approximately 98.6° F., the patient with rheumatoid arthritis (as well as other forms of rheumatic disorders) will experience a marked reduction or elimination of muscle pain and spasm temporarily while in the pool. Exercises should be administered during the period of immersion in the water, producing a hyperemia, which, together with the buoyancy of the water, results in general relaxation of the central nervous system.[35] This temperature will produce peripheral dilatation with increase in the flow of blood and lymph and increased elimination of toxic waste products. The increased temperature appears to increase the tolerance of the muscles to exercise. This continuous capillary dilatation during the muscular movements facilitates the breaking down and excretion of inflammatory exudates in and around the joints and muscles. The arc of painless motion of the

affected joints is increased markedly in some cases during under-water exercises.

Exercises under water result in reduced muscular spasm so that the patient can tolerate more motion in his painful joints. These exercises should not cause pain, but the parts should be moved in as great an arc as possible in slow, rhythmic motions. Under-water massage and exercise are very helpful in restoring body mechanics, re-educating atrophied muscles and in preventing or correcting deformities. Stretching manipulations may gradually lengthen shortened muscles. It is extremely gratifying to see arthritic patients who can stand up with great difficulty and take only a few steps even with the assistance of a walker, walk about easily and comfortably in water of shoulder depth.

It is usually advisable to *precede the under-water exercise by under-water massage.* While the body floats in water, all appreciable strain is removed from the weakened muscles or inflamed joints so that the operator or patient has no weight to lift, rendering the patient more receptive and at ease. The operator may use both hands for massage, which should be gentle and rhythmic, following the muscle groups. When hyperemia is attained, stroking (light effleurage) is begun to the muscles above and below the affected joints. When the acute stage of an arthritis has passed, one may begin gentle friction around the joints, with effleurage and kneading (pétrissage) of the surrounding muscles. Contracted tendons may be stretched gradually with gentle traction and stroking. One should not use heavy massage or sudden strong movements.[35]

Most spas utilize manual massage by trained masseurs as an important part of their program. The machines devised by Dr. Gustaf Zander of Stockholm for active and passive exercises of voluntary muscles are used very little at present because well-trained masseurs can accomplish their feats more safely, less expensively and more effectively.

Jarman believes that the spa is not the best place to treat the patient whose disease is so active as to still require splints.[16]

Singer and Phillips have summarized their concept of **Thalassotherapy** (*the utilization of ocean climate in treating or preventing disease*). Historically, sea water was given as a laxative and was used in skin diseases by the old Greek and Roman curators. Brighton mushroomed into a fashionable seaside resort when Richard Russell was able to persuade the King of England in the mid-eighteenth century to submit himself to "the cure" there. The Royal Sea Bathing Hospital was founded in Margate, England, in 1791, following which crusades for the general adoption of therapeutic ocean bathing spread to the continent of Europe.

The irregular coast line of Europe was dotted with hundreds of seashore sanatoria prior to the war. *In Belgium alone, the forty miles of seashore had more than 40 sanatoria with 3500 beds.* In contrast to this, *there are only 3 noteworthy institutions along the 6000 miles of seashore in the United States* (one each in New Jersey, New York and Connecticut). Although 20 million people utilize the American seashore as a vacation ground each year, there is no supervised daily regimen or planned climatic exposure.

Two types of climate have been distinguished: the sedative or protective climate and the stimulating climate.[22] The shores of southern California and of Florida are considered to be the sedative type, as is the summer on any seashore in this country. A winter stay at seashores other than Florida and southern California is considered to be stimulating.

Thalassotherapy offers sun, air, surf bathing and outdoor exercise; baths in heated sea water and mud packs and baths. There are practically no known contraindications to a stay at southern seashores in any season or at seashores in temperate regions in the summer and early fall. If any individual with rheumatic disease, and particularly fibrositis, is continually made worse by a stay at the seashore, it is common sense for that individual to avoid this region. *Rheumatic patients are seldom made persistently worse by a stay at the seashore during the summer months,* despite the popular concept among patients in this country that those with rheumatic disease must avoid the shore.

Climate is considered as a long range view on weather—a sum total of the meteorologic phenomena characterizing the average state of the atmosphere.[34] Climates have been divided geographically into the tropical, temperate and Arctic and geologically into the seashore, desert and high altitude climates.

The *main climatic factors at the shore* are the water, sun and air. Sea air usually is relatively free from pollen, allergens, dust and gaseous products of combustion. It is said to contain slightly more oxygen (20.99 per cent) than continental air (20.76 per cent), as well as a high barometric pressure. The *sea breeze* which is aroused and maintained by the difference between inland and ocean surface temperature is an important feature of the seashore; in the summer, this reduces the daily fluctuation of temperature, moderating the mid-day heat. *The sea breeze has been likened to a natural air conditioning mechanism at the shore,* maintaining a convenient degree of humidity, purifying the air and producing a desirable temperature range.

Although an area up to 50 miles or more inland may be influenced (as regards weather changes) by the vicinity of the ocean, the cooling effect of sea breezes can usually be felt up to 4 to 6 miles inland.

The reflection of radiation from the beach sand and from the surface of the water result in a distinct difference in the *quality of the sunshine on seashores* in comparison with sunshine elsewhere.

Although sea water contains from 2 to 4 per cent salt (mainly sodium chloride and potassium), *there is no absorption of the mineral content even following prolonged bathing.* The effects of sea bathing have been attributed to other factors, including the temperature of the water and the difference between the skin and water temperature; counterirritation (from the salt content of the water); mechanical stimulation by the surf waves, etc.

Each individual has his own comfort zone within which range he does not perspire or become chilled while resting and normally clothed. Climatotherapy is essentially supervised and planned exposure to an atmosphere whose heat absorbing capacity differs from that of the original habitat;

in addition, hydrotherapy and heliotherapy are used to reinforce this by judicious application.[34]

Among the spas in this country, one may mention the following:

Arrowhead Springs Hotel and Spa, Arrowhead Springs, California.
Bedford Springs, Pennsylvania.
Buffalo Mineral Springs and Hotel, Mecklenburg County, Virginia.
Braun Bath House and Hotel, Hot Springs, South Dakota.
Capon Springs, West Virginia.
Excelsior Springs Sanitarium and Hospital, Excelsior Springs, Missouri. (A municipal development.)
French Lick Springs, French Lick, Indiana.
Glenn Springs, Watkins Glen, New York.
Hot Springs National Park, Arkansas (a United States government project).
Manitou Springs, Colorado.
Mount Clemens, Michigan.
Mudbaden Sulphur Springs Company, Mudbaden, Minnesota.
Mudcura Sanatarium, Shakopee, Minnesota.
Mudlavia Springs Hotel and Sanatorium, Kramer, Indiana.
Our Lady of Lourdes Hospital and Sanitarium, Hot Springs, South Dakota.
Panacea Mineral Springs and Hotel, Littleton, North Carolina.
Pence Springs and Hotel, West Virginia.
Poland Springs Spa, Maine.
Saratoga Spa, Saratoga Springs, New York (an excellent spa run by the State of New York).
The Spa, Thermopolis, Wyoming. (These springs are controlled by the state of Wyoming.)
The Spa, Waukesha, Wisconsin.
Virginia Hot Springs, Virginia.
West Baden Springs, Indiana.
White Sulphur Springs, Sharon Springs, New York.
White Sulphur Springs, West Virginia.
Whitcomb Sulphur Springs Hotel, St. Joseph, Michigan.
Wyrick Mineral Springs and Hotel, Crockett, P. O., Virginia.

Other sanitaria which are especially equipped to handle arthritic patients include:

Barfield Sanatorium, Tucson, Arizona.
Battle Creek Sanatarium, Battle Creek, Michigan.
Robert B. Brigham Hospital, Boston, Massachusetts.
Desert Sanatorium and Institute of Research, Tucson, Arizona.
Florida Sanitarium and Hospital, Orlando, Florida.
Hinsdale Sanitarium and Hospital, Hinsdale, Ill.
The Leo N. Levi Memorial Hospital, Hot Springs, Arkansas.
Medea Hotel and Bath House, Mt. Clemens, Michigan.
Miami Battle Creek Sanitarium, Miami Springs, Florida.
New England Sanitarium and Hospital, Woodland Road, Melrose, Massachusetts.
Sacred Heart Sanitarium, Milwaukee, Wisconsin.
St. Francis Health Resort, Danville, N. J.
St. Joseph's Hospital and Sanitarium, Mt. Clemens, Michigan.
Torbett Sanitarium and Diagnostic Clinic, Marlin, Texas.

Before the recent war, European spas included Bath, Buxton and Harrogate in England; Vichy, Vittel, Aix-les-Bains and Royat in France; and

Baden-Baden, Bad Ems, Bad Gastein, Bad Nauheim, Carlsbad, Marienbad and Wiesbaden in Germany and Pistany Spa in Czechoslovakia.

In an effort to obtain general information concerning American spas and other arthritic institutions which might be useful to the practicing physician, inquiries were sent to many of these institutions. The answers should be of interest to physicians because they show that:

Prices at these institutions vary considerably.

Rheumatoid arthritis is not cured by a stay at a spa, but the patient may be greatly benefited in a general way.

Various forms of treatment are considered to be of value. Prices may vary considerably following the current War.

The questions and some of the representative answers are as follows (prices or other conditions are subject to change):

1. What is the Minimal Charge for Hospitalization and All Medical Fees per Week for Arthritic Patients?

French Lick, Indiana: Hotel room prices range from $8 to $14 per day (winter) and from $10 to $17 per day (summer) for single rooms; double rooms are approximately twice these prices. These figures include only hotel room and meals; baths and medical fees are additional. Baths and massage vary from $1 to $3 per treatment.

Desert Sanitorium, Tucson, Arizona: Minimum charge for hospitalization, including medical fees for an arthritic patient, $75 per week; this includes room with bath, porch, board with tray service to the room, nursing and medical care. Additional charges are made for laboratory examinations, roentgen-rays, transfusions, special medicines and considerable physical therapy.

Park View Hotel, *White Sulfur Company*, Sharon Springs, N. Y.: $25 per week per person, American plan. Medical care, baths and treatments are additional. Medical fees are $3 per visit or $10 for as many visits as are required during a patient's stay. Various baths vary from $1.35 to $3 each.

Hot Springs National Park, Arkansas: This is under *government control.* There are many hotels in the park, the largest accommodating over 1000 guests. Larger hotels operate on both European and American plan.

There are several hundred additional boarding houses, ranging in price from $7 per week upward. There are also many furnished and unfurnished cottages or apartments available, which may be rented for from $20 per month upward. Hot Springs can house 25,000 visitors at one time. Anyone desiring a list of hotels, boarding houses or other accommodations may obtain this from the Hot Springs Chamber of Commerce.

Baths may be taken without the advice of a physician. Physician's fees for examination for the baths range from $5 to $10; these physicians are licensed by the state of Arkansas and have been examined by a Federal Board of medical examiners. Seventeen bath houses are operated under the rules and regulations of the government. The water in all of these is the same. Single baths range from $1 to $1.40. Single treatments in pools, including the service of a physical therapist, are $2.25. The maximum charges for massage range from $2 for single treatments to $30 for a series of 21 treatments. There is a government *free bath house for the indigent. Applicants for free baths are required to make oath that they are without and are unable to obtain means to pay for the baths. All applicants for free baths must provide and pay their own board and lodging and have return railroad fare. The Levi Memorial Hospital admits patients free of charge.*

Greenbrier and Cottages, *White Sulphur Springs, West Virginia:* The minimum rate is from December 1 to March 1; this is $112 for a two-weeks stay. Laboratory tests and medical services are additional, such as $5 for a blood count,

$5 for urinalysis. Medical services average from $3 to $5 for an office visit. The $112 rate includes the use of the baths and the swimming pool.

St. Joseph Sanitarium and Hospital, *Mt. Clemens, Michigan:* Room and board may be obtained from $4.50 per day up; baths average $1 with $.50 additional for an attendant fee.

Battle Creek Sanitarium, Michigan: Minimal charges for arthritic patients who are not able to be up and about are $56 per week in the medical ward section; this includes room, meals, services of attending physician and dietitian, and treatments in the hydrotherapy and mechanotherapy departments. Physicians who have not previously registered are given two days stay free of all expense. Minimal weekly rates for ambulatory patients are from $37 upward. This includes the services of an attending physician and dietitian, hydrotherapy and mechanotherapy, corrective and recreational gymnastics, swimming pools, room and meals.

All individuals who enter the institution as regular patients are required to pay a $35 general entrance examination fee. This includes history, physical examination, blood sugar, B.U.N., blood uric acid, blood count, dental examination, test of vision, fluoroscopic examination of the chest, proctologic examination, urinalysis, examination of feces, etc.

The Homestead, *Virginia (Hot Springs):* The minimum charge is $130 for two weeks from December 1 to March 1 only; at other times the rate is $175. This includes room, board, medical fees, baths, massage and other physical therapy. In April and October, the rates are somewhat higher ($225 for a two weeks course).

Saratoga Spa, New York: Patients live at hotels, boarding houses or cottages in the city of Saratoga Springs; depending upon the accommodations selected, a stay of three weeks would cost the patient from $110 upward. The best hotel and living accommodations would average $300. This large and very well equipped spa is controlled by the state of New York. Prices for baths and massage vary and are low enough for almost all patients.

Poland Springs, Maine: Hotel rates vary from $5 to $10 a day. Treatments in the bath department average $3 each.

Braun Hotel, Hot Springs, South Dakota: A charge of from $25 to $30 a week includes the patient's board, room, bath and treatments. Medical attention is extra.

2. What Advantages Has Your Institution for the Arthritic Patient?

Answers to this question included: excellent medical care, scientific laboratories, suitable environment, heliotherapy, renowned mineral springs, day to day observation of the patient, sulfur baths, mud packs, special forms of massage, hydrotherapy, pleasant surroundings, etc.

3. What Special Forms of Therapy Do You Use at Present in the Treatment of Patients With Rheumatoid Arthritis?

Some replied wisely that no special form of therapy was used but that each patient was carefully studied on admission and that a regimen was designed to meet the needs of a human being who happened to be suffering from a painful, crippling, chronic disease for which no specific cure is known, but for whose comfort and well-being a great deal could be done, knowing that in time many would improve and some would get well. The main items in therapy included rest, diet, physical therapy (including exercise, mineral waters, hydrotherapy, heliotherapy, massage, etc.), transfusions and elimination of foci of infection.

4. What is the Minimum Time That a Patient With Rheumatoid Arthritis Should Remain to Obtain Benefit?

Answers ranged from two to ten weeks, averaging four to six weeks.

5. What Percentage of Patients (If Any) Have You Cured of Rheumatoid Arthritis?

About 80 per cent of the answers were to the effect that the percentage of actual cures in cases of rheumatoid arthritis was very small, and in many instances the physicians in charge doubted if any of these patients were really cured. It is true, however, that many of these patients are benefited subjectively and objectively by a suitable regimen once or twice a year at a spa as a part of their treatment. The treatment of rheumatoid arthritis should be thought of in terms of months and years and not weeks. Patients with fibrositis, degenerative joint disease and gout are often aided by a visit of several weeks to a suitable spa.

Old medico-religious ceremonies (Persian, Assyrian and Babylonian) depicted the use of *moors* and *muds*, while the Romans employed *heated sea mud* extensively in the extravagant bath houses of that city (150 A.D.). In 1935, over 300 European spas were applying *pelotherapy*,[21] particularly in Czechoslovakia, Germany, France, Italy, England, Norway, Sweden, Hungary, Russia and Rumania. Less than ten years ago, the Austrian Government registered Austrian muds and selected these as state reservations.

The term **peloid** is used to designate all types of peats, moors and muds which are employed for therapeutic purposes. *Peloids* are substances originating in Nature by geologic process which can be applied as packs or baths after artificial processing.

Singer has used pelotherapy in chronic arthritides; fibrositis; neuritis; sciatica and other conditions. Singer's classification of peloids is based upon their silicate content: (1) *Fango mud* (purely mineral); (2) *sea mud* or *liman* (mainly mineral), and (3) *moor, peat* (mainly vegetable).

Mud is composed mainly of sand, clay and kaolin. **Peats** and **moors** are made up of mosses, algæ and diatomaceæ with the débris of vascular plants. **Sea muds** show a mixture of botanical and minerogen contents. Muds are neutral in reaction while liman is alkaline and peats and moors are acid.

Fango is obtained from the beds of mineral springs of volcanic origin in the Rocky Mountains or in Italy and consists of a fine, homogeneous and nearly pulverized mud. *Liman or sea mud* is mainly mineral and is deposited on the bottom of inlets and shallow bays; it contains microscopic shells of cadavers of the one cell life of the ocean, mixed with sand, quartz and sea vegetation detritus. Liman has been obtained in Sweden, Russia and in this country in New York and New Jersey. *Peats or moors* are mainly vegetable muds and result from the moldering of marsh vegetation; they are characterized by their carbonized content of plant particles of bogs (and is obtained from Florida, southern California and Germany).

One of the best detailed descriptions of mineral waters, peloids, and climate is presented by Vogt.

The *biologic effects which have been claimed for muds are due mainly to their physical properties:*

1. Because of their specific heat which is much lower than that of water, a *50° C. temperature can be well borne in hot mud.*

2. *Mud packs grow cool very slowly* because of their poor heat conductivity.
3. Mechanical stimulation of the contained microscopic shells, spicules and quartz grains acts as a counterirritant.
4. It may be applied to practically any part of the body because of its *plasticity*.

European health resorts used mud baths extensively. A mud bath or a mud pack will cover several joints at once and can be applied at a higher temperature than water. Rectal temperatures of 103° F. can be obtained easily within thirty minutes by a mud pool.

European resorts employ more mud therapy in arthritis than American resorts. Krusen feels *that there are no particular advantages of mud packs over cleaner and simpler methods of local application of heat.*[18]

There is no convincing proof as yet that any of the constituents of mud particularly enhance its effectiveness.

The medically useful peloid should be of low cost, free of pathogenic bacteria and human contamination, easily processed, highly plastic without being sticky, uniform and should be available in large quantities.

Neuwirth used the natural mud of Pistany at the Pistany Spa in Czechoslovakia. Because of the good retention of heat, only a minimum amount was lost during the average twenty minute period of a mud pack. Mud may be applied at a higher temperature to the body than water because of the increased thermal tolerance of the skin to mud. The mud is highly plastic, has a low specific heat, a high specific gravity and is a poor heat conductor.[23]

Neuwirth believes that the general reaction produced by a warm mud pack is comparable to a foreign protein reaction. The increase in body temperature during the mud pack results in a stimulation of the general circulation, with increased metabolism.

It is claimed that the application of heated mud results in an increase in body temperature, and a lowered blood pressure.

Singer lists the *contraindications to pelotherapy* as follows:[34]

1. Acute diseases or subacute stages of chronic diseases.
2. Marked physical debility or early convalescence.
3. Marked peripheral vascular disorders.
4. Cardiac decompensation.
5. Pregnancy.
6. Advanced tuberculosis.

In some spas, **fango** is used extensively. *This is an Italian name for an extremely fine deposit which settles at the bottom of several lakes at Battaglia in the northern part of Italy.* Hot volcanic springs feed these lakes and the volcanic mineral matter comes to the surface continually with small bubbles of gas. The gray deposit of this matter is fango, which is really derived from the volcanic crater from which the water flows.

Fango is a mineral mud containing approximately 3.5 per cent water, 6.5 per cent organic matter, and 90 per cent of inorganic matter; among its constituents are calcium, sulfur, silica, iron, magnesium and carbonic acid.

It is reported that magnificent bath houses were erected by Roman emperors in what is today the town of Battaglia, and that extensive use of such mud baths was made by them.

It has been claimed that the application of such hot mud lessens congestion in deep-seated tissues by producing dilatation of blood and lymphatic vessels.[10,33] Rapid absorption of effusion, relief of pain and increased mobility of partially ankylosed joints have been reported following the use of fango packs.[4] Fango therapy produces marked hyperemia of the skin with local vascular dilatation and diaphoresis, and a reduction in capillary pressure.[20,26] The effect of prolonged heat upon the involved tissues is obtained by fango therapy with a reduction of muscular spasm from the heat and the uniform pressure exerted by the mud. Such mud treatments are known as *pelotherapy*.

Because the specific heat of mud is much less than that of water, a temperature as high as 50° C. can be borne in hot mud while a temperature of 44° C. is about the maximum borne in hot water.[33] During their protracted application, mud packs grow cool slowly because of their poor heat conductivity. The temperature drops only a few degrees in several hours. The number of calories transmitted per unit time is less with mud than with water, and it has been stated that the "heat of mud does not invade the skin vehemently, but sneaks in."[33] Thus, by use of a mud pack, one may obtain conductive heat which promotes absorption of exudates, and relieves local pain as a warm wet compress, being more soothing, penetrating and relaxing than dry baking.

The Advantages of Hot Mud Treatment: Large surfaces of several joints may be treated at once.

The heated mud may be applied at home with little difficulty and low expense.

There is a large beneficial element of psychotherapy in mud treatments.

Treatments are usually applied to one or more joints at a time. Fango is sold in this country as a dry powder, which is mixed with water before applying. The quantity to be applied is mixed with a large wooden spoon with an equal volume of water to produce a clay-like mixture. The temperature of the mixture is raised to about 180° F. (in a double boiler) with constant stirring. A portion is removed to another container and sufficient dry fango is added to bring the temperature to approximately 108° F.;[4] in subsequent treatments this may be increased to 125° F. if desired. If the material is to be applied to a knee, a woolen blanket covered with oilcloth or rubberized sheeting is placed under the knee so that the rubber is in contact with the skin. A layer of warm fango at a temperature of 108° is spread around the knee for a thickness of 1 inch. The heat and moisture is conserved by wrapping the rubber sheeting and the blanket around the mud-packed knee. This may be kept in place for thirty to sixty minutes, at the end of which time the fango is easily removed and the part cleansed. Treatments may be given daily or every second day as desired.

Good results have been reported in rheumatoid arthritis, degenerative

joint disease, traumatic arthritis and sciatica with this form of treatment. Its value is probably a result of slow heating of the affected tissues, muscular relaxation from this heat and pressure from the surrounding fango.[9,37] It may prove a useful adjunct in therapy in chronic forms of arthritis where other forms of physical therapy are not available, and where psychotherapy may be of benefit. It has not been proven that fango possesses any greater effect than ordinary clay, similarly applied.

Contraindications to fango therapy are the same as the prolonged application of other forms of heat or hot packs (advanced cardiac disease, general debility, etc.).

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CHAPTER XXV

PSYCHOGENIC FACTORS IN RHEUMATIC DISEASES

It has been suggested that *anxiety, worry and emotional shock* may play an important rôle in the etiology of rheumatoid arthritis.[15,17] Internists and psychiatrists have recently shown a renewed interest in this aspect of the patient with rheumatoid arthritis. In some cases, the arthritis has come on immediately following a severe emotional upset.[20,23] Not infrequently, these patients become worse with increased environmental stress.[3,5,9] Older writers advocate a neural theory of etiology of rheumatoid arthritis with lesions possibly in the central nervous system because of the symmetrical joint involvement. Recently, the term *"psychogenic rheumatism"* has been applied by some to indicate those states in which the stiffness, pain, swelling or limitation of motion in muscles or joints is caused, intensified or continued by mental influences. In one recent report, "psychogenic rheumatism" was claimed to be the most frequent cause of disability in 450 consecutive cases diagnosed as arthritis "or an allied organic condition" previous to admission to an Army General Hospital.

Smith and Cobb have stressed that psychogenic lesions may appear in persons who seem to be healthy and normal but may yet be extremely susceptible to emotional shock due to an inherited instability of certain organ systems. In addition, psychogenic influences may increase the extent of the lesions already present.[2,10,19]

On the other hand, chronic rheumatic infection may be the etiologic factor in some cases of long-standing mental illness even though rheumatoid arthritis is extremely infrequent in psychotic patients.

Cobb and Bauer studied carefully the psychiatric history of 50 patients with rheumatoid arthritis and entered the facts obtained on a life chart, along with the medical data from the hospital record; such life charts have been employed by Prof. Adolf Meyer at the Johns Hopkins Hospital.[4] Medical data included all past illnesses, prodromal arthritic symptoms, onset of the joint phenomena, remissions and relapses. It was concluded that *poverty, grief and family worry bore more than a chance relationship to the onset and exacerbations of rheumatoid arthritis.* These relationships may have been overlooked had they been recorded on the conventional medical record forms. In some of the men the onset of the arthritis or exacerbations of the disease coincided with periods of financial stress or lack of work. In a large number of the women with rheumatoid arthritis, the major burden was also financial because of inability of the husband to find work, often followed by his illness and the added strain of nursing, using savings, borrowing money or recourse to relief. Family worries also played an important rôle in some of these individuals, as well as loss of a parent or spouse. Other less frequent associations included marital unfaithfulness, living with in-laws and marital discord. In a group of control patients

of comparable age and sex who entered the hospital because of varicose ulcers, the interviews showed that they were dealing with a different type of person and in only 12 per cent was there any coincidence of onset of ulceration and social stress.

Individuals with rheumatoid arthritis are usually introspective, fretful, lack confidence in themselves (and often in their physician) and are often unseemingly unreasonable. *Psychotherapy, whether used consciously or unconsciously, is often essential to the proper handling of these patients, who are especially amenable to suggestion.*

Swaim has stressed the fact that *these patients must be questioned carefully about their intimate relationships with members of their family, friends and business associates.*[22] They are usually eager to unburden themselves. One must try to expose to the patient his resentments, conflicts or actual hatreds or fears. One may then explore the individual's personal beliefs or his own standards of living. People do not often like to face unpleasant truths about themselves and one must guide them to get their own convictions of where they are wrong and causing them to want to change them. Swaim believes, after long personal experiences with large numbers of arthritic patients, that if people would cultivate a mature, developed faith and believed in a super-force as part of their lives, they would not react with fear and anger the way they do under emotional strain, their health would be better and they would be less susceptible to disease. He feels that this spiritual factor may turn the tide of success or failure in some cases.

Others have shown that rheumatoid arthritis may bear a close relationship to an emotional crisis. Patients with rheumatoid arthritis are often emotionally unstable even before the onset of their illness. Psychologic factors may be of importance in producing or maintaining pain and stiffness (occasionally because of sickness benefits, sympathy, etc.). Individuals with rheumatoid arthritis have tended to be amphotonic (rather than vagotonic or sympathicotonic), demonstrating an irritability and instability of the vegetative system.[18] Emotional conflicts disturb the vegetative nervous system and some arthritic patients will not respond adequately to therapy until a proper attack has been launched against their psychologic problems.[16]

Smith and Cobb include rheumatoid arthritis as a clinical disorder which is probably related to emotional stimulation and in which *a single powerful emotional stimulus has been known to produce an attack.*[19] They stress the importance of this point in a discussion of legal cases.

Gregg commented upon the paucity of arthritis among psychotic patients. "Disabling arthritis" was found to be seventeen times greater in the community than among psychotic patients in Massachusetts State hospitals.[11] He postulated that among psychotic patients without a prolonged stimulus reaction span, because of impaired imagination and memory, there is little prolonged muscle tension or drag on the joints, little setting of the stage for prolonged ischemia, malnutrition or infection of the joints and therefore little chronic arthritis.

The term *psychoneurotic rheumatism* was used by Halliday to bring forth the idea that *emotional upsets may cause upsets in the body including pain, stiffness and limitation of motion, i. e.*, including such conditions as have been diagnosed fibrositis, muscular rheumatism, neuritis, lumbago, sciatica, pleurodynia, etc.[14] He stressed the point that attention must be given to the stiffness, aches and pains which may accompany chronic psychoneurotic anxiety states or psychotic depressions.[12,13]

The relation of psychologic factors to illness was known to physicians from earliest times, but their significance and importance were greatly lessened during the last century. The recent study of psychoneurotic illness has permitted these reactions to be formulated scientifically. In addition to the routine history and physical examination of the arthritic patient, recent research and observations in psychosomatic medicine show very clearly that a supplementary examination must include an extension backwards (in time) of the history to include psychic disturbances which preceded the onset of evident joint pathology, a careful study of his personal relationships, occupational problems, financial circumstances, quarrels and bereavement and his fears and knowledge of the disease.

One must search into the patient's facial expressions which may give a hint of an anxiety reaction—with such presenting features as a tired, worried, hunted or strained countenance. The patient's thoughts are usually dominated by his bodily symptoms and he may completely suppress, except after careful questioning, those preceding disturbances such as inability to concentrate, depression, lack of interest and feelings of futility. It is often difficult to induce the arthritic patient to speak in detail about his difficulties in life as he repeatedly short-circuits the conversation back to his treatments or symptoms.

Factors to be Determined in the Psychologic Approach to the Arthritic Patient:

1. What kind of individual was he before the development of his arthritic symptoms?
   (*a*) Previous breakdowns: nervous, gastrointestinal, etc.
2. Environmental or external events which precipitated a deep-seated emotional reaction:
   (*a*) Mental stress or strain or indecision.
   (*b*) Loss of a friend or loved one just prior to the onset of symptoms, or loss of: career, money or occupational difficulties.
   (*c*) Grief, rage, guilt?
   (*d*) Relations with his family, friends, boss, teachers, fellow-workmen?
   (*e*) Fears.
   (*f*) Financial responsibilities and difficulties.
   (*g*) Illness in the family; loss of work by husband because of illness; nursing of a sick husband or wife.
   (*h*) Marital discord? Marital unfaithfulness?
   (*i*) Strain of living with in-laws?

Halliday feels that the most common types of psychoneurotic rheumatism seen in practice are **hysterical pains**; pains, aches and stiffness

seen as episodes in chronic psychoneurotic anxiety states and in psychotic depressions; and some cases of fibrositis. It has been pointed out that many patients with "hysteria simulating rheumatism" are admitted too often to physical therapy centers so that the symptoms consequently become fixed. *Hysteria is not uncommon in men and such hysterical pains are seen most often in those individuals whose occupations expose them to excessive bodily danger.* Hysteria is known to develop often from a gradually progressive anxiety concerning one's safety. Thus, Halliday has found that hysterical pains are most common in men such as underground miners, steel erectors, window cleaners and steeplejacks (who work at heights); those concerned with the making of explosives and such sports (as football) as involve a high degree of risk of injury to the body.

Halliday found 39 per cent of 145 patients with fibrositis, lumbago, sciatica and neuritis incapacitated because of psychoneurosis.[13]

In England, a large number of patients with muscular and joint aches and pains (but without objective evidence of joint pathology) persisting for several months fall into the class of *chronic psychoneurotic anxiety states* or *psychotic depressions*. If a simple psychologic approach is used to supplement the usual medical history and examination, much of the incapacity would be preventable—which otherwise would soon become permanent and fixed.

It has been pointed out that many patients with aching and stiffness in joints and muscles who are considered to have fibrositis are victims of what has been termed by some, **psychogenic rheumatism.** It is possible that the fatigue and nervous irritability which accompanies fibrositis so often has not been evaluated properly and that the stiffness and aching are merely parts of a neurasthenia with the predominant psychogenic manifestations being these symptoms of aching and stiffness.[6,7]

True malingering is uncommon among patients admitted to army hospitals with symptoms resembling fibrositis. In malingering, there must be a conscious feigning of an illness to avoid duty; this is punishable in the army by court-martial.[20] It is more likely that the somatic symptoms (pains, stiffness, muscular aches) develop unconsciously as a mechanism of escape from some of the unpleasantries of military service.

Boland and Corr defined *psychogenic rheumatism* as those states in which muscular or joint pain, stiffness, limitation of motion or subjective sense of swelling are caused, intensified or perpetuated by mental influences.[1] "Pure" psychogenic rheumatism is such a disability without physical signs of joint or muscular abnormalities; superimposed psychogenic rheumatism is the association of incapacitating psychogenic symptoms with non-disabling organic changes.

In a study of *450 consecutive cases diagnosed as arthritis or an allied organic condition admitted to an army general hospital, psychogenic rheumatism was found to be the most common cause of disability*, suggesting that insufficient attention has been paid to the influence of emotional factors in the production of somatic symptoms of the neuromuscular system. There are, of course, many dangers and pitfalls involved in attempting to

explain physical symptoms and signs on a psychogenic basis. However, if functional states are accepted and recognized more and more frequently in other fields (gastroenterology, cardiology, neurology), certainly a concept of "psychogenic rheumatism" must be appreciated and accepted. In their series, symptoms were thought to be purely on a psychogenic basis in 20 per cent of rheumatic subjects and 15 per cent were disabled by psychogenic factors superimposed on mild organic changes. It was concluded that *a third of all of the 450 patients were incapacitated due to psychic difficulties.*

Of the 269 patients in Boland and Corr's series who had peripheral joint complaints, 22 per cent were thought to have pure psychogenic rheumatism as opposed to 21 per cent with rheumatoid arthritis, 15 per cent with osteo-arthritis, 14 per cent with acute rheumatic fever, 5 per cent with fibrositis, 3 per cent with gonorrheal arthritis, 2 per cent with gout and the remainder miscellaneous or unclassifiable types. *In the 181 with complaints referable to the back, 18 per cent were thought to be due to pure psychogenic rheumatism*, 34 per cent to unstable back syndromes (strains, scoliosis, spondylolisthesis and congenital scolioses); 18 per cent to "rheumatoid sacroiliitis and spondylitis;" 10 per cent to degenerative joint disease; 7 per cent to fibrositis, and an equal number each to protruded intervertebral disk and to juvenile dorsal osteochondritis.

Fifty cases of psychogenic rheumatism were studied in detail; 28 of these had no objective evidence of joint or muscle disease and were designated as pure psychogenic rheumatism. In 18 there were minor non-disabling structural changes; in 4 the incapacity persisted even after all physical manifestations of the observed organic process had completely subsided. The average duration of military service of the group was nine months (ranging from a day to seven years). *The sedimentation rate was normal* in all instances. There seemed to be no relation to age, rank, previous occupation, education or intelligence in the enlisted men.

Seventy-six per cent of the group had symptoms referable to the back or peripheral joints prior to military service; 14 per cent had definite inflammatory joint disease previously and 38 per cent dated their disability to an injury. More than a third of the patients gave a history of invalidism or semi-invalidism in their immediate families (such as a crippling arthritis in a brother, sister or parent). In 76 per cent, symptoms were limited to the lower extremities and back, the lower extremities being involved alone in 22 per cent of cases. *In only one instance were the upper extremities the sole site of somatic fixation;* in 8 cases, complaints were present in practically all joints. Although others have noted a striking predilection for psychogenic symptoms to involve the left side of the body, both sides were involved about equally in these patients.

Most of these 50 patients had definite psychoneurotic manifestations before entering the army; about a third of the group gave histories of disabling hysterical or psychoneurotic episodes preceding the presenting complaint—ranging from interruptions in school from "nervousness" to attempted suicides. In others, nightmares, temper tantrums, somnambulism or

other symptoms constituting behavior and personality problems were noted in the histories. In over half, an anxiety state was present at the time of examination while about a third showed a mild mental depression together with anxiety. All but 4 of this group had symptoms of an associated psychoneurosis in addition to the rheumatic symptoms.

The main symptoms manifest by the patients were *pain* (which was universally complained of)—usually *augmented by physical activity incident to military service; stiffness* and a *subjective sense of swelling* in 50 per cent; *bizarre limping* in approximately a third (often associated with abnormal postures); *limitation of motion* and *weakness* of the involved part. The patient often interpreted as pain such symptoms as tingling, weakness, burning, numbness, pricking, deadness or fulness. Pain and stiffness were present in all of the patients with backache, although on close questioning this was really a weakness or tired feeling or a dead or numb sensation. A variable degree of rigidity of the back was common, but true restriction of motion was rarely seen.

In those with "*psychogenic backache,*" *the outstanding characteristic was the persistence of the disability despite prolonged rest in bed, with continuous discomfort day and night, not responding to physical therapy, and made worse by slight physical exertion.* In one instance, it was interesting to observe a patient with an advanced active rheumatoid spondylitis pushing an individual with a pure psychogenic backache in a wheel chair. Hysterical type of anesthesia or hypesthesia was seen in 5 patients; hysterical paresis of an extremity was observed in only 2 patients.

Of this group of 50 patients who were studied intensively, at least 46 were eventually discharged from the army. In those individuals who were sent back to duty, even on a limited service status, their symptoms returned or were intensified in short periods (ranging from a few hours to three months).

It is not claimed that chronic rheumatoid arthritis results from psychic conflicts. It is often difficult to elicit the precipitating emotional cause in patients with so-called "psychogenic rheumatism." Strecker placed the musculoskeletal system last on the list as regards frequency of functional disorders in the various systems.[21] However, there are *numerous factors which may induce anxiety states in soldiers:* loss of ability to control one's personal destiny; loss of love or security with separation from home, family or wife; fear of injury; concern for the safety and well-being of dependents; resentment at authority especially when invested in one felt to be inferior; confusion from strange surroundings, competition, regimentation, etc.

Points which may be helpful in establishing a diagnosis of psychogenic rheumatism include associated psychoneurotic or hysterical manifestations; persistence of the disability despite rest in bed and physical therapy; marked incongruities between the structural changes and the severity or quality of the symptoms; bizarre limps or postures; and a normal sedimentation rate. *The possibility of psychogenic rheumatism should be considered in all patients diagnosed as fibrositis* with chief complaints of stiffness and aching in joints and muscles without obvious objective signs. *In true fibrositis, the symptoms are usually aggravated by* periods of inactivity,

exposure to cold, drafts and dampness and are relieved by heat, massage and a moderate amount of exercise in most instances. *In psychogenic rheumatism,* on the other hand, one will find associated psychoneurotic complaints, a precipitating psychogenic factor and an emotional make-up.

The only treatment which seems to be of value in patients with psychogenic rheumatism is the removal of the underlying mental conflict; in those in military service, this can be accomplished in most instances only by discharging them from the army. Prompt recognition of some of these patients in the early stages before somatic symptoms have become intensified and fixed will enable one to employ proper psychotherapy rather than physical therapy in order to prevent some of the later psychogenic rheumatism casualties.

Freyberg and his associates carried out *skin temperature studies under induced emotional stress in 25 rheumatoid arthritics* and in 25 control patients. Emotional stress was found to produce a drop in skin temperature indicative of a change in circulation. *The emotional factor appeared to have more influence in producing exacerbations or affecting the course of the illness than it did in a causative rôle.* In comparing the results in the two groups of patients (arthritics and controls), little difference was noted in the intensity of fall in temperature under induced emotional stress. Although 3 arthritics did show a drop of more than 3° C., which was greater than that of any of the controls, there was no essential difference in the amount of drop in temperature considering the groups as a whole.[18]

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CHAPTER XXVI

THE RÔLE OF VITAMINS IN ARTHRITIS

ALTHOUGH vitamin deficiency may be found in various forms of chronic arthritis, there is as yet no proof that lack of any specific vitamin causes any form of arthritis. Vitamin B deficiency occurs frequently in chronically ill patients; these individuals frequently also have lowered blood levels of ascorbic acid.

In the vitamin field much controversy exists at present over the value of massive doses of vitamin D in the treatment of rheumatoid arthritis. A few observers have noted marked improvement—even superior to that following gold therapy—in far-advanced rheumatoid arthritis; however, this is seen in most cases only when the dose of vitamin D is near its toxic level. If this dose is not maintained, the symptoms revert to their previous state.

Other equally competent rheumatologists have reported that massive doses of the same brand of vitamin D have not been of proved value in rheumatoid arthritis or that results were far from impressive. This important question is discussed in some detail (see page 498).

Vitamins are foodstuffs and under normal circumstances man obtains his supply from naturally occurring food sources. **Vitamin poor foods include** plain crackers, white bread, alcohol, pastries, candy, corn syrup, corn starch, soft drinks and polished rice.[66] *Vitamins should be considered as foods and not as drugs;* the grocery store is usually more important than the drug store in the prevention and treatment of vitamin deficiency states. Now that we can obtain some of the vitamins in the crystalline form, too much emphasis may be placed on these members of the group and not enough on other vitamins which have not been isolated as yet and of which we may, in fact, be unaware. Emphasis on a diet rich in foods containing vitamins should be the keystone of any therapeutic effort directed toward a patient who is suspected of having vitamin deficiency. The next most satisfactory method of meeting the situation is by the use of concentrates or rich food sources of vitamins still in the natural state, or if this is not sufficient, by pure crystalline preparations.

The *vitamin intake may be kept high in very sick patients by* cream, milk, fruit juices, thin whole grain cereals, ground liver and puréed legumes; feedings may be given by duodenal tube, if necessary. As the patient improves, whole wheat crackers and bread and other foods rich in various vitamins may be added.

Nutritional deficiency disease may be due to an inadequate intake, diminished absorption or utilization, increased need or increased excretion or destruction of essential nutritional substances.[133] There is an increased demand for vitamins in pregnancy, fever and in any condition associated

with an elevated basal metabolic rate. The need for some vitamins may be increased from 10 to 100 per cent during pregnancy and lactation.

Many clinicians consider certain types of arthritis to be infectious. In infections, the requirement for vitamins has been demonstrated to be increased. This is an additional reason for emphasizing a high vitamin diet in arthritis. In certain patients with arthritis, and in particular, those with chronic or rheumatoid types, there is occasionally some evidence of abnormality in function of the gastrointestinal tract, most commonly manifest by achlorhydria. Since it is well known that gastrointestinal distress from achlorhydria may interfere with digestion of foodstuffs and the absorption of vitamins, emphasis should be placed on this phase in any consideration of the problem of vitamins and arthritis. However, **there is no proof that any common form of arthritis develops as a result of a particular vitamin deficiency.**

VITAMIN A

In rheumatoid arthritis and in other chronic or infectious diseases, the demand for vitamin A as measured by the dark adaptation curve is increased.[98] In 45 of 79 patients with rheumatoid arthritis, the degree of vitamin A deficiency roughly paralleled the severity of the disease as measured by the sedimentation index. Some of these patients show various degrees of blepharitis.[145]

Race[98] and others[35,50,117] found that the carotenoid pigments, carotene and xanthophyll, and vitamin A itself, are lowered in patients with rheumatoid arthritis. Although vitamin A deficiency is not uncommon in patients with rheumatic disease, there have been no controlled experiments showing that correction of this deficiency is beneficial to the course of the disease. Monroe found no therapeutic value from concentrates of vitamin A[82] and Hall and his associates did not alter the course of the arthritis by correcting the vitamin A deficiency with doses of from 25,000 to 68,000 units of this vitamin daily by mouth.[50]

Many patients with rheumatoid arthritis need from four to ten times the amount of vitamin A present in their regular diet to maintain normal dark adaptation curves.[50] Although it is desirable to administer this vitamin in amounts sufficient to relieve any vitamin A deficiency, adequate amounts of vitamin A do not produce any clinical improvement in the arthritic picture.

The exact daily requirement of vitamin A is unknown;[157] the minimal daily requirement for an adult is approximately 4000 to 6000 U.S.P. units.[13,63] Approximately 6000 to 8000 U.S.P. units per day should be adequate for growing children, and at least 5000 units per day for pregnant and lactating women.[158] The requirement for infants is higher per unit of body weight than in older children.[71]

The yellow pigment carotene is the precursor of vitamin A (provitamin A) and is converted to vitamin A in the liver. Bile salts in the gastrointestinal tract are necessary for the absorption of carotene. *Sources of vitamin A include* milk, cream, eggs, cod liver oil, carrots, fish oil (halibut,

cod, tuna, burbot), green vegetables and sweet potatoes.[15] Crystalline vitamin A has been prepared from fish oils.[58,59]

Vitamins A_1 and A_2, although chemically different, are biologically identical.[16]

Symptoms of vitamin A deficiency include night blindness, photophobia, dry skin and conjunctiva, blepharitis and follicular hyperkeratosis.[64,65,146] Vitamin A is seldom found in the urine of normal individuals even after the administration of massive doses of this substance; however, in the presence of cancer, infections, or hepatic or renal damage, vitamin A may be eliminated in the urine.[157] It has been estimated that 95 per cent of vitamin A in the body is stored in the liver, and the remainder in the lungs and kidneys.[7]

The main pathologic changes found in tissues deprived of adequate amounts of vitamin A include atrophy and subsequent keratinization of the epithelium.

Partial vitamin A deficiency may be common in this country, if one accepts results of studies by the biophotometer.[62,75] Such a deficiency is more common in individuals of lower economic levels. *Marked vitamin A deficiency is uncommon in the United States.* There is no justification for calling vitamin A the anti-infective vitamin; however, marked deficiency of this vitamin lowers resistance to infection in man and a moderate deficiency may increase the severity of and prolong respiratory infections.[18] Where a moderate deficiency exists, an adequate intake of vitamin A may decrease the duration and severity of infection.

Tests of vitamin A deficiency include: Determination of the ability of the eyes to adapt themselves to darkness.

Examination of epithelial cells obtained from the bulbar conjunctiva (this is probably inadequate).[165]

Estimation of vitamin A content of blood plasma or serum.

The biophotometric method of measuring alterations of dark adaptation is not reliable for detecting mild forms of vitamin A deficiency.[22,87,125]

The mean value for the vitamin A content of blood plasma was found to be 20 U.S.P. units per 100 cc. of plasma (in patients over two years of age). This figure is decreased considerably during infections.[19]

In treatment of vitamin A deficiency, massive doses may be required; in some instances, best results were obtained with 70,000 units of vitamin A orally per day for fourteen days, followed by 25,000 units daily until dark adaptation returned to normal. There is as yet no satisfactory preparation for parenteral use. Toxic effects need not be feared from vitamin A. No untoward effects have followed enormous doses of vitamin A (such as 500,000 units at a single dose).

VITAMIN B COMPLEX

Compounds of the *vitamin B complex definitely known* are: vitamin B_1 (thiamin chloride), B_2 (riboflavin), niacin (nicotinic acid) and its derivatives, vitamin B_6 (pyridoxin), yeast adenylic acid and pantothenic acid; indefinite preparations are: B_3, B_4, B_5, U and W factors and others.[85,90,159,160]

Vitamin B_1 (known as *thiamin chloride*, in pure form) prevents beriberi in man and polyneuritis in animals. The name thiamin chloride (bromide, sulfate, etc.) has now been adopted in this country for vitamin B_1.[25]

Riboflavin (formerly called vitamin B_2 or vitamin G) is essential for growth in chicks and rats and prevents cataract formation in rats. *Symptoms of riboflavin deficiency include* lesions at the corners of the mouth; erosions around the eyes; a shark skin appearance of the epidermis over the nose; itchy, burning, dry eyes; photophobia; granular, red conjunctiva, especially of the lower lids, often associated with keratitis.[133] *Cheilosis* (lesions at the corners of the mouth) has been produced in women with dietary restriction of this vitamin;[116] the lesions disappeared when from 1 to 2 mg. of synthetic crystalline riboflavin were given daily for several days. Cheilosis has also responded to vitamin B_6 (pyridoxin); some cases are resistant to riboflavin and others respond to neither riboflavin nor pyridoxin.

Niacin (nicotinic acid) and its derivatives will usually prevent the occurrence of pellagra.

Vitamins B_3, B_4, B_5, B_6, U and W factor—and a filtrate factor—are necessary for prevention of various disturbances in pigeons, rats and chicks. Vitamin B_6 is also important in human nutrition.[132]

In considering therapy with vitamin B, one must emphasize (*a*) foods (especially meats) containing vitamin B; (*b*) concentrates and rich food sources (as yeast); (*c*) and crystalline preparations (*i. e.*, thiamin chloride, riboflavin, pyridoxin and niacin [nicotinic acid] and its derivatives). Clinical deficiency of vitamin B_1 *alone* probably never exists. One should supply all components of the vitamin B complex if possible in a natural food source rather than depend on crystalline products, as the latter may be incomplete and inadequate. Brewer's yeast (as tablets or in loose form) is an excellent source of vitamin B. If vitamin B cannot be given by mouth, crude liver extract given intramuscularly will supply this material.

Few plant or animal foods are potent sources of vitamin B_1.[83] *Inasmuch as vitamin B_1 is not stored in the body to any great degree*, each day's diet should furnish an adequate amount of this substance. *Good sources of vitamin B_1* are legumes, nuts, whole grains, fresh vegetables (including potatoes), fruits, eggs, muscle, organ meats, milk and yeast. *Meat is able to supply a large part of the vitamin B_1 requirement;* even 1 fried pork chop may provide an adequate total daily requirement.[81] It is believed that vitamin B_1 is preserved in stored frozen foods. *Vitamin B_1* is inactivated by heat if moisture is present and *is* therefore *destroyed in cooking*, especially in alkaline medium; 50 per cent of the vitamin is destroyed in roasting, broiling or stewing.

We have no conclusive symptom or sign of minimal vitamin B_1 deficiency.[164] The nervous and circulatory systems are mainly involved in vitamin B_1 deficiency. Such *deficiency may lead to anorexia and nausea*, which promptly disappear following administration of the vitamin.[144] *Other symptoms may*

include heaviness in the legs, tenderness of the calf muscles when squeezed, weakness of the limbs, burning of the soles, paresthesias, diminished or absent tendon reflexes, muscular weakness in the extremities, glossitis, achlorhydria, anemia and diarrhea; some of these are likely to be due to deficiency of other fractions of the vitamin B complex.

It has been found that *the average normal daily intake of pure vitamin B_1 is 1 mg.*[28,149] It has been *suggested*[133] *that 0.6 mg.* of pure *thiamin chloride is required daily for health in adults.*[36,141] The requirement for vitamin B_1 may be influenced by the level of energy metabolism,[26] and by loss of the vitamin as in diuresis or diarrhea.

The daily output of thiamin has been found to vary from 12 to 150 international units, depending on the dietary intake.[54,112] Munsell has summarized the biologic and chemical methods of assay of this substance. The vitamin B_1 content of the blood averages from 6.5 to 16.5 micrograms per 100 cc.; methods of analysis of this vitamin in the blood are in the experimental stage.[80,114]

In addition to crystalline vitamin B_1, other convenient methods of administering this vitamin include plain or autolyzed brewer's yeast and wheat germ. Brewer's yeast is somewhat unpleasant to take in bulk. It may be given stirred in cold water, or its taste may be disguised by mixing it up to 25 per cent in catsup or peanut butter.[133] In definite vitamin B deficiencies, one may prescribe from 75 to 100 gm. of brewer's yeast or 10 to 20 brewer's yeast tablets per day. These contain niacin (nicotinic acid), thiamin chloride, riboflavin, vitamin B_6 and other fractions of vitamin B, as well as enzymes, minerals, proteins and salts. In the average case of beriberi in an adult, one may give 10 mg. of thiamin chloride b.i.d.; in severe cases, this may be increased to 20 mg. from three to five times a day.

Injections of large amounts of vitamin B_1 have evidently caused some cases of herpes zoster.

Some individuals have advocated the use of *vitamin B* (in brewer's yeast or wheat germ) *in the treatment of rheumatoid arthritis.*[46,57] Fletcher has suggested that disturbances in the bowel in chronic arthritis may be associated with vitamin B deficiency.[45] There is some evidence that thiamin chloride may be used with moderate success in the prevention and treatment of roentgen sickness.[78]

Mild forms of vitamin B deficiency exist in many chronically ill patients, including those with rheumatoid arthritis. Freyberg[47] has noted evidence of vitamin B deficiency in these patients; such symptoms included a sore mouth; a red, sore tongue; and mild dermatitis over the knees and elbows. These symptoms were found most often in the patients with anorexia, malnutrition and chronic indigestion and were relieved by large doses of brewer's yeast or other vitamin B concentrates. A few patients with rheumatoid arthritis may present the typical cheilitis (of riboflavin and other fractions of vitamin B deficiency) which respond to adequate doses of the vitamin.

Neuritis as a manifestation of thiamin deficiency is not usually encountered in patients with chronic rheumatoid arthritis. Freyberg has treated many patients with rheumatoid arthritis, degenerative joint disease and fibrositis *with large doses of thiamin chloride* both orally and parenterally but *has not found any beneficial effect even in those patients with muscular and neuralgic pains.*

Freyberg concluded that **there was no direct relationship between the vitamin B complex or its components and arthritis,** but that if there is a deficiency of this food factor, supplements of vitamin B should be given to correct the deficiency and thus improve the patient's general health. *One should not expect direct benefit to the rheumatic disease from the administration of vitamin B.*

Vitamin B may be given as brewer's yeast (15 to 30 gm.) stirred into cold fruit juice, or in tablet form (3 to 6 tablets)—each given three times a day, as tolerated. Preparations of vitamin B complex in concentrated form are available on the market and may be used. Such administration of vitamin B may be helpful in improving the appetite and correcting indigestion, leading in turn to increased food consumption with a resulting improved nutrition and general health. Some patients cannot tolerate yeast in any form, as this may cause indigestion, bloating and other digestive complaints.

In the treatment of neuritis of alcoholic addicts, from 20 to 50 mg. of thiamin chloride daily was required in some cases for definite clinical improvement.[48,154]

Riboflavin is a yellow substance found in animal and plant food, especially in the green leaves of actively growing plants, and in milk, fresh liver and yeast; it is found in 5 oxidation enzymes.[151] Although the requirement of riboflavin is unknown, it has been estimated that man should receive from 2 to 3 mg. daily.[56] In riboflavin deficiency, one may prescribe 3 to 5 mg. orally as tablets or intravenously in sterile physiologic saline.

Many of the symptoms of pellagra may be relieved by niacin (nicotinic acid) amide, niacin (nicotinic acid), sodium nicotinate, the diethyl amide of nicotinic acid (nikethamide) and other compounds.[123,131,133] Niacin (nicotinic acid) may also be useful in the treatment of some cases of stomatitis.[76]

The average adult patient with pellagra should receive 50 mg. of niacin (nicotinic acid) amide orally about ten times per day; in severe cases, this must be doubled. The amide has the advantage that it does not cause an increase in skin temperature or vasodilatation of the skin which often follows niacin (nicotinic acid) and sodium nicotinate. If oral medication is not tolerated, one may use 10 to 15 mg. intravenously from five to six times per day.

Vitamin B_6 (pyridoxin) at times has a dramatic beneficial effect in relieving muscular weakness and rigidity. It lessens the rigidity in some cases of paralysis agitans (Parkinson's syndrome). The symptoms of vitamin B_6 deficiency may be adequately treated by the intravenous injection of 50 mg. of this vitamin in physiologic saline. Considerable improve-

ment has been seen following its use in pseudohypertrophic muscular dystrophy.[6]

Studies on *yeast adenylic acid* and *pantothenic acid* are still in the early stages. The latter seems to be essential to human nutrition; its function is thought to be associated with that of riboflavin.[134]

VITAMIN C

Fresh orange juice contains approximately 40 to 60 mg. of vitamin C per 100 cc.[2,70] The daily requirement for normal health in adults probably ranges from 40 to 100 mg. of vitamin C; artificially fed infants should receive 10 mg. per day.

Vitamin C deficiency may occur in some conditions even when its intake is apparently adequate;[161] these include: increased metabolism from infection, pregnancy, starvation, or other causes; interference with absorption or utilization because of achlorhydria, colitis or other intestinal disturbances and other unknown factors.[29,69] Man cannot synthesize vitamin C, and the storage capacity for this vitamin is probably limited. Vitamin C is present in the body in small amounts.[32,86,142]

Hall and his associates found that 75 per cent of 56 cases of rheumatoid arthritis showed a subnormal blood vitamin C; 59 per cent had levels below 0.5 mg. per 100 ml., but none of them presented clinical evidence of scurvy.[51] These patients could tolerate an intake of more than 100 mg. and usually 200 mg. without marked excretion in the urine, showing that they probably have a much greater demand for vitamin C than the normal individual. However, when patients with rheumatoid arthritis were placed on 200 mg. of vitamin C per day for eight months, no improvement was noted that could be attributed to the effect of the vitamin C.

It is now accepted that *the vitamin C level of blood plasma is a good index of the immediate state of the individual with respect to vitamin C.* Freyberg and his associates studied the ascorbic acid blood level of more than *100 patients with rheumatoid arthritis* and found that the majority of these patients *had abnormally low levels of plasma ascorbic acid,* which approached, in some instances, scorbutic levels (although clinical evidence of scurvy was not found). Thirty-eight per cent of patients with rheumatoid arthritis had normal blood vitamin C concentrations every time this was determined. *There was no relationship between the blood ascorbic acid content and the severity of the arthritis.* Lower values were usually (but not always) seen in those who are chronically ill, undernourished and debilitated. The incidence of low plasma ascorbic acid was found to be about the same in a group of hospital patients with chronic illnesses (excluding rheumatic diseases) as in the arthritic group. Freyberg concluded that **vitamin C deficiency was not an important factor in the causation of rheumatoid arthritis,** but that the vitamin C deficiency found in chronic arthritis is a consequence of the disease, being common to many chronic, debilitating diseases.

Large doses of ascorbic acid or fruit juices produced no marked improvement in the patient with rheumatoid arthritis.

Although some investigators have suggested that additional amounts of vitamin C may help to protect against toxic reactions from gold therapy, Freyberg found that the plasma ascorbic acid value was normal in 4 of 6 patients with gold dermatitis.[47]

A study of the ordinary hospital diet showed that its vitamin C content was inadequate to supply the increased demands of the patient with rheumatoid arthritis.[51]

Rinehart and his associates[103–110] believed that vitamin C deficiency played an important rôle in the etiology of rheumatic fever and rheumatoid arthritis. Others noted a vitamin C deficiency in rheumatic fever.[67] The cevitamic acid level of blood plasma during fasting was found to be markedly lowered in rheumatoid arthritis and rheumatic fever. Apparent deficiency of vitamin C existed in the presence of an ordinarily adequate dietary intake. They also noted the occurrence of an extremely low level of ascorbic acid in the blood in rheumatoid spondylitis and reported improvement in over 80 per cent of these patients following the administration of large doses of this vitamin. *Other workers showed that serum ascorbic acid levels, in general, are usually well below normal in patients with infections,* and that the dietary intake of vitamin C which is required to bring the serum level and the urinary output to normal values in the presence of such infection is much greater than the normal requirements.[42] *These workers feel that the effect of rheumatic fever upon the metabolism of vitamin C is similar to that of other infectious diseases.*

Rinehart reported (1934) that joint disease occurred in experimental animals deficient in vitamin C and later suggested that such deficiency may be of some significance in the etiology of some cases of rheumatoid arthritis.[104,108]

Experimental production of arthritis by vitamin C deficiency includes the following reports:

Production of changes in the heart valves related to those of acute rheumatic fever by injection of streptococci into guinea pigs brought to the scurvy level by vitamin C deprivation; prevention of such changes by addition of orange juice to the diet.[111]

Production of synovial proliferation, pannus formation and experimental scorbutic arthropathy in guinea pigs without superimposed infection.

An impartial evaluation of the rôle of vitamin C in rheumatic fever and rheumatoid arthritis points to a deficiency of this vitamin in these diseases; however, it does not appear that such a deficiency *causes* the disease.[68] *The vitamin C deficiency is probably a manifestation of the infectious state; for this reason, it is advisable to supply additional vitamin C to such patients.* In adults, 200 mg. may be given by mouth daily for several weeks. In most instances, this will result in the production of a normal blood level of ascorbic acid; once this has been raised to normal, inclusion of large amounts of citrous fruits in the diet will usually be sufficient to maintain this level. If necessary, cevitamic acid may be given intramuscularly or intravenously.

Normal adults have a daily requirement of vitamin C of approximately 60 mg.; infection increases this requirement. In individuals with various forms of infection, the amount of vitamin C in the blood may be lowered to levels found in patients with scurvy. In these patients, it is necessary to give several times the daily normal requirement of ascorbic acid to raise the blood plasma level of the vitamin to that of the renal threshold.

The estimated requirements of vitamin C for various age groups have been reported as follows: *Infants*—8 mg. (newborn) to 50 mg. per day; *children*—22 to 100 mg. per day; *adults*—28 to 100 (or more) mg. per day.[122]

The richest sources of vitamin C include oranges, lemons, raw or canned grapefruit, tangerines, raw or canned tomatoes, fresh strawberries, green peppers and raw cabbage. Other important sources are green leafy vegetables (even if cooked), apples, bananas, pineapples, potatoes, green beans and green peas. High concentrations are also found in the adrenal cortex, corpus luteum and anterior pituitary glands. Dry cereals and legumes lack vitamin C, as do cooked muscle meat, butter, eggs and cheese. *Pasteurized milk contains very little vitamin C.* There has also been noted a variation in vitamin C content of fruits and vegetables with changes in the soil, season and maturity of the food, deterioration of vitamin C content with storage and slight destruction in freezing. There is a 50 per cent decrease in the vitamin C content of milk if allowed to stand for an hour in the sunshine.[53]

Fresh orange juice retains 97 per cent of its active vitamin C even after twenty-four hours protected from air in the refrigerator;[23] it loses about 50 per cent of its ascorbic acid content if allowed to stand at room temperature for three hours.

Although some bacteria in the gastrointestinal tract can destroy ascorbic acid and prevent its normal absorption, the presence of even small amounts of dextrose (as is found in citrous fruits) deflects such microbic attacks on vitamin C.[16] *Preclinical forms of scurvy are commonly overlooked;* signs of this disturbance include heaviness, weakness, pains in the legs, nausea, dyspnea, dizziness and ready bruising.

In scurvy, the maintenance and curative dose of ascorbic acid in most cases averages from 30 to 50 mg. per day, although as much as 1000 mg. may be required. Overdosage is not harmful; toxicity is extremely low.

It has been reported that *the state of vitamin C nutrition may be determined in human beings by the following tests:* (1) fragility or resistance of blood capillaries; (2) ascorbic acid content in fasting blood; (3) excretion of ascorbic acid in urine;[148] (4) intravenous and oral tolerance tests; (5) intradermal tests, etc. The generally accepted *minimum normal value of blood ascorbic acid is 0.7 mg. per 100 cc. of blood.* Increased metabolic activity such as violent exercise and fever result in increased use or disappearance of ascorbic acid.[52,77] The requirement of vitamin C is increased in pregnancy and nursing mothers. The critical level of excretion of ascorbic acid is 1.4 mg. per 100 cc. of serum.[43]

The intradermal test for determination of vitamin C deficiency has been found to be *unsatisfactory* because of the great range of error.[92,93,113]

The *first positive clinical evidence of scurvy* may be determined by the fragility of the minute vessels of the skin.[49] The procedure recommended by Wright for this test is as follows:

Draw 2 circles each 2.5 cm. in diameter on the inner smooth surface of the forearm; the upper edges of these must be at least 4 cm. below the crease of the elbow.

A blood-pressure cuff is placed on the upper arm and the pressure is maintained midway between systolic and diastolic readings for fifteen minutes (unless an excessive number of hemorrhages occur).

Five minutes after the pressure is released, the average number of petechiæ per circle seen readily with the naked eye is counted.

Normal values: 10 or less per circle; borderline readings: from 10 to 20; more than 20 is distinctly pathologic. *A positive test is not always due to scurvy. Increased capillary fragility may be found also in poisonings* (neoarsphenamine, carbon monoxide); toxins of diphtheria, subacute bacterial endocarditis, scarlet fever, severe anemia, acetonemia, menstruation, etc.

Although a single determination of blood cevitamic acid level is the simplest procedure to give information concerning this vitamin,[36,154] it is not as accurate an index of vitamin C deficiency as is a test dose of this substance (as is done similarly in diabetes mellitus with the glucose tolerance test).[44,162] Wright has considered the best method of performing such a test to be the administration of 1000 mg. intravenously, followed by determinations of total urinary excretion of the vitamin in the next five hours. Normally, 500 mg. or more of this dose are excreted within the next twenty-four hours (80 per cent of this 500 mg. being excreted within five hours after injection).

The amount of vitamin C excreted in the urine depends upon the immediate intake of ascorbic acid and upon the degree of saturation of the tissues with this substance. As tissue saturation is approached there is an increase in ascorbic acid excretion in the urine. The technic for estimation of vitamin C in the urine or blood has been described.[10]

VITAMIN D

Vitamin D preparations of all potencies have been overemphasized and oversold to the public; the possible effects of toxic dosage must be borne in mind.[60] At least ten different sterol derivatives exhibit the properties of vitamin D; activated ergosterol (or calciferol) and activated 7-dehydrocholesterol are of importance in medicine.

Ergosterol is the parent substance of vitamin D activity in irradiated foods. Vitamin D_2 is *calciferol;* this is a naturally occurring form of vitamin D. Vitamin D_3 is dimethyl-dihydro-calciferol. The latter has been isolated in crystalline form and is the principal source of vitamin D activity in naturally occurring anti-rachitic substances of animal origin (as fish liver oils); it is probably the activable substance in human skin.[79] Activated ergosterol is found in viosterol, irradiated yeast and yeast milk.

The main action of vitamin D is to increase the absorption of calcium and phosphorus or to diminish their intestinal excretion.[118] Several types of vitamin D deficiency have been hypothecated.

Facts Concerning the Availability of Vitamin D.—Foods, unless fortified, are inadequate sources of vitamin D for infants and children.

Vitamin D is found in only a few of the foods which we normally eat;[89] these include milk and some dairy products, eggs, liver and some fish. *Milk is a poor source of vitamin D* unless it is irradiated, or unless the vitamin is added to the milk. The butterfat of the milk contains the vitamin D. *Butter contains only 1/100 of the amount of vitamin D (80 units per 100 gm.) as is found in cod liver oil.*

Vitamin D in Arthritis

Inasmuch as *the adult requirements for vitamin D are unknown,* the presence of a deficiency of this vitamin in patients with arthritis has not been determined.

Because of the small amounts of vitamin D contained in the average adult diet (in egg yolk, herring and canned salmon), one should add to the diet of arthritic patients 0.3 cc. (5 minims or more—approximately 3000 units) of viosterol (or its equivalent in vitamin D content) daily.

If tolerated, *cod liver oil is preferred in these patients,* because of its content of vitamin A and added food value; an ounce of cod liver oil supplies approximately 2500 U.S.P. units of vitamin D in addition to its caloric value. This is not tolerated by all individuals. Preparations of concentrated vitamin A and D in their natural form can be obtained.

Present evidence is controversial as to the value of *massive doses of vitamin D in altering the course*[1] *of rheumatoid arthritis.* By massive doses, one means from 100,000 to 300,000 units of vitamin D daily. *Toxic symptoms may follow such doses.* From 20 to 30 per cent of patients with rheumatoid arthritis may experience an improved sense of well-being, improved appetite and possibly lessening of joint phenomena following prolonged therapy with massive doses of vitamin D. The arthritic symptoms reappear in most of these patients when the drug is discontinued. The physician employing such doses must be on the lookout for evidence of toxicity and discontinue the drug at least temporarily at the first sign of intolerance.

Activated ergosterol of plant origin, known as calciferol (in its pure form) is now called *viosterol. One mg. of calciferol contains 400,000 units (U.S.P.) of vitamin D.*

One quart of *irradiated milk* contains only 135 U.S.P. units; ordinary cow's milk even during summer months contains only 40 units per quart.[8] The yolk contains all of the vitamin D in eggs; this is a small amount, averaging from 140 to 390 units per 100 gm.[30] Only small amounts of vitamin D are found in sardines, salmon and canned fish. There is no proof that vitamin D exists in living plant tissue; *fresh green vegetables probably contain no vitamin D.*[84]

Preparations of Vitamin D Available to the Physician.—*Cod liver oil*—contains at least 85 U.S.P. units of vitamin D per gram.

Emulsion of cod liver oil—contains at least 50 per cent cod liver oil by volume.

Viosterol in oil—contains at least 10,000 units of vitamin D per gram; this is tasteless.

U.S.P. standards apply only to the above three preparations; according to the Federal Food and Drugs Act, drugs sold under the above names must meet these specifications. In addition to the above, most drug companies manufacture their own special combinations of vitamin preparations. *Preparations of irradiated ergosterol of high potency have not been accepted by the Council on Pharmacy and Chemistry of the A. M. A.*[24] Numerous preparations of vitamin D in high dosage (50,000 units per capsule) are available—as Ertron, Deltalin, etc.

Vitamin D Requirements.—In normal full term infants, 400 international units of vitamin D in dispersed form (*i. e.,* milk) is required daily for best growth.[96] Other suggested daily requirements are (*a*) for premature infants, 600 to 800 U.S.P. units and (*b*) for adolescents, 300 to 400 U.S.P. units; (*c*) during pregnancy and lactation, 800 U.S.P. units or more. (U.S.P. and International units of vitamin D are identical.)

The exact requirement of vitamin D in adults is unknown. Many children and adults obtain large amounts of vitamin D from exposure to the summer sun; this supply may be stored in the body for weeks.[55]

Vitamin D therapy has been advised in those adults who are (*a*) deprived of sunlight;[89] (*b*) where the diet is poor and lacking in milk; (*c*) in the aged, especially if confined indoors; (*d*) following fractures or operations on bones; (*e*) during pregnancy and lactation, and (*f*) in the treatment of osteoporosis, sprue, tetany, chronic arthritis (?), psoriasis, allergy (?) and many other diseases, especially those affecting calcium metabolism and diseases of bones.

The vitamin D therapy in massive doses was begun in rheumatoid arthritis by Dreyer and Reed in 1935 when two of their arthritic patients who were receiving massive doses of vitamin D for their hay fever noted improvement in their joint symptoms.[31,95]

There is no scientific explanation as to why vitamin D may act in rheumatoid arthritis.

Reynolds claimed that "Ertron" differs in therapeutic value and safety from other forms of vitamin D.[100]

"Ertron" is made by passing an electric current through vaporized ergosterol; when cooled, the resulting vapor is collected and standardized.[130]

Ertron was found not acceptable for New and Non-official Remedies by the Council on Pharmacy and Chemistry of the American Medical Association in 1937, which concluded that *critical examination of the reports on the value of vitamin D in chronic arthritis revealed little to warrant a belief that the beneficial effects which were claimed were specific.*[27] The Council also pointed out that there was no proof that such large doses are not toxic. *In New and Non-official Remedies in 1943, the Council stated that clinical evidence did not warrant the claim that massive doses of vitamin D were of benefit in chronic arthritis.*[102]

"Ertron" is said to be a form of vitamin D_2 which is *prepared by electrical activation of heat*-vaporized ergosterol, using the Whittier process. *Vitamin D_2 may be prepared synthetically by several methods other than by ultraviolet irradiation of provitamins.* The two methods of most practical importance are the Whittier and the Milas technics.

These latter two methods rely primarily upon the use of vaporized ergosterol contained in a vacuum; this is then subjected to bombardment by electrons. The Whittier patents provide for the introduction of the electrodes *into* the chamber and the use of indium as a catalyst, with subsequent production of *rapidly moving* electrons. The Milas process provides for the production of an electronic field *surrounding* the chamber, thus activating the ergosterol by *slow moving* electrons. The intermediate products from the irradiation process are not produced in quantity in either of these methods. It is felt that *the final vitamin D* is not in pure form but nevertheless *is identical with calciferol.* By melting points, number of units per gram, and light absorption curves, all of which are substantially identical for each of the three, there is strong evidence that their chemical structure is the same.

Vitamin D_3 has been called the animal vitamin D since it is found in natural form in animal tissues and in fish liver and body oils. This is in contrast to *vitamin D_2* which *is commonly prepared from the provitamin D found in plant life.* Vitamin D_3 has been synthesized by the activation or irradiation of 7-dehydrocholesterol. Biologic assays of these two vitamins D show identical potencies in rats and humans, but in chicks, D_2 is considerably weaker so far as its antirachitic effect is concerned.

Abrams and Bauer seldom noted objective evidence of improvement from vitamin D in rheumatoid arthritis and found that the beneficial effects were only transitory.[1]

Slocumb summarized the literature up to 1940 on the effects of vitamin D in infectious arthritis.[121] He pointed out the very variable results obtained by various investigators, ranging from 20 per cent clinical improvement[57,163] to 100 per cent.[38]

Snyder wrote that the claims of certain writers[31,41] were perhaps somewhat overenthusiastic concerning the value of Ertron.[130]

Cohen felt that treatment with massive doses of vitamin D was dangerous.[20]

Steinberg found that about a third of the patients were improved on large doses of vitamin D.[139]

Improvement was reported following large doses of vitamin D in rheumatoid arthritis *by some workers.*[31,39,74,136,150]

Boots concluded that the therapeutic value of Ertron or any other high dosage vitamin D preparations was unproved, and that he certainly did not recommend Ertron as a cure for rheumatoid arthritis.[14] *In his opinion, this was a therapeutic agent of uncertain, if of any, permanent value. In 31 patients with rheumatoid arthritis* at the Arthritis Clinic of the Presbyterian Hospital who were *treated with Ertron, only 1 patient showed any objective signs of improvement.* Although 7 of the patients felt very much better, only

1 remained well after he stopped the drug and the remainder relapsed immediately after the drug was discontinued. *Boots believed that there was probably no significant difference between Ertron and any other high dosage vitamin D preparations. Eighteen of their 31 patients developed some* **toxic manifestations** *while taking Ertron,* usually nausea and vomiting, which cleared promptly after cessation of treatment.

Steck reported on his experience with Ertron in 84 patients with rheumatoid arthritis, 9 cases of degenerative joint disease and 5 cases of mixed arthritis.[137] He *concluded that only a small percentage of the cases showed complete freedom from recurrence of symptoms on cessation of treatment for long periods*—when studies were continued for five years. He found no marked correlation between the degree of improvement resulting from this drug and the duration of disability before therapy. He found no evidence of intoxication from vitamin D in his cases although he did not report whether blood calcium determinations or other laboratory studies were made in these individuals.

Freyberg and his associates followed very carefully the course of patients with rheumatoid arthritis treated with vitamin D, including 36 patients treated with Ertron.[47] Some writers had claimed that various investigators who had reported toxic results or poor clinical results following vitamin D therapy had used preparations of vitamin D other than Ertron. Freyberg's work is of extreme importance since he used Ertron and followed the results in these patients very carefully.

Following a careful analysis of his findings with vitamin D therapy (including the use of Ertron), *Freyberg concluded that* **massive doses of vitamin D should not be used with a comfortable feeling that great benefit is certain to result**; he further stressed that *Ertron,* which some claim is non-toxic, *does have real potentialities for producing undesirable effects, particularly gastrointestinal* and that increased blood calcium does result occasionally. He reported that **the course of rheumatoid arthritis was seldom favorably altered by Ertron.**

In *36 patients* with rheumatoid arthritis whom Freyberg *treated with Ertron, benefit occurred relatively seldom and was slight.* A few patients showed definite subjective and objective improvement, but the sedimentation rate was rarely reduced. Improvement often lasted only a short while after treatment had been stopped.

When improvement occurred following Ertron therapy in Freyberg's cases, *it was much more often subjective than objective* in that the patients felt better in general, had more pep, and less indigestion and constipation although they did not especially note improvement in rheumatic symptoms. This subjective improvement in general was not produced by placebos.

Other preparations of vitamin D which were evaluated by Freyberg in patients with rheumatoid arthritis included Apolarthron; Drisdol and vitamin D_2 in sesame oil. In the patients treated with Ertron, the doses commonly employed were 200,000 to 300,000 units daily for months; if this could not be tolerated, from 50,000 to 100,000 units were given daily.

The patients were observed for from one to three years before evaluating results.

Contrary to the opinions of some others, *Freyberg's results with preparations of vitamin D other than Ertron in general gave results similar to those produced by Ertron except that hypercalcemia occurred in a slightly higher percentage of cases with the other products.* His opinion was that the **results of his entire study of vitamin D therapy in rheumatoid arthritis were far from impressive** in that **the drug was of benefit in only a minority of cases** *and that when improvement did occur, this was usually temporary and only symptomatic.*

Slocumb (at the Mayo Clinic) found that large doses of vitamin D were of assistance in controlling the symptoms of 7 of 14 patients with infectious arthritis, although very little objective improvement could be seen.[121] *In his experience, vitamin D was certainly not to be classed as a specific agent for the control of infectious arthritis* as exacerbations of the disease occurred in some while the patients were taking large doses of vitamin D. *The beneficial effects in his patients were only transitory* and the arthritic symptoms returned in most instances when the drug was discontinued. He found that **there was a definite risk of renal damage from vitamin D** and that serious renal damage might occur unless the vitamin D was discontinued as soon as evidence of toxicity appeared. The most frequent sign of toxicity was gastrointestinal upset.

Slocumb concluded that **massive doses of vitamin D were of very little, if any use in producing lasting improvement in the patient with infectious arthritis.** His patients received the vitamin D for periods varying from twelve days to fifteen and a half months. When improvement occurred, it was first noticed from five days to three weeks after beginning the administration of vitamin D. All patients except 2 had a return of their symptoms within eight days to two and a half months after vitamin D was discontinued. In most cases, the symptoms returned and were as severe as previous to the vitamin D therapy. *Three individuals had transitory exacerbations of their disease while taking the vitamin D.* One patient maintained his improvement for one and a half years and another for two and a half years before having a return of their active infectious arthritis.

Snyder and his associates decided from experience that *it was necessary to use at least 150,000 units of Ertron to obtain definite evidence of improvement* in the average case of arthritis and that *from 200,000 to 300,000 units per day were required in the majority of cases for maximum benefit.*[126,128] In a few cases, these physicians used from 500,000 to 600,000 units per day for a week or ten days without producing untoward symptoms (such as nausea, vomiting and frequency of urination). In their hands, Ertron was reported as being relatively non-toxic. Mild symptoms of intolerance were controlled easily by reducing the dose or stopping the medication.

Snyder and his group did not observe following Ertron therapy any increased calcification of blood-vessels (roentgenologically or by ophthalmologic examination), impairment of renal function or of liver or gall bladder function. Although blood calcium figures were elevated occasionally following Ertron therapy, these workers did not think that there was any causal relationship between this elevation and vitamin D toxicity.

The *earliest* and *most consistent beneficial effect* in their patients *was an improved sense of well-being* with a *gain in weight* by the thin, asthenic patients. In the group of patients studied by Snyder, there was no good correlation between changes in the blood constituents (including the sedimentation rate) and progress or severity of the arthritis. Functional improvement occurred in these patients despite lack of radiologic improvement.

Snyder concluded that Ertron therapy was safe and that it could be continued for a long period without harmful effects. Many other observers disagree markedly with this.

Following Ertron therapy in Snyder's patients, the individuals felt and looked better and noted an improvement in appetite. The soft tissue swelling decreased or disappeared later and the range of motion in the affected joints increased together with an increase in functional activity with little or no pain. This improvement occurred gradually in most cases, although some individuals noted improvement within four weeks. *In most instances, two or three months of maximum doses of medication were required before the first evidence of improvement was noted.* In most of these cases, the improvement was sustained after complete cessation of the drug but in some it was necessary to renew the medication in moderate doses to keep them symptom-free. Small maintenance doses were required in some to maintain improvement. In over six years of investigation with Ertron, Snyder and his associates have never seen evidence of serious toxicity from Ertron.

When Snyder's patients were examined by an orthopedist following Ertron therapy, he concluded that of the 32 patients with rheumatoid arthritis, 5 had obtained no benefit; 2 had shown very slight improvement; 18 had shown a fair amount of improvement; 2 had shown a good improvement and 5 were from 75 to 100 per cent improved.[130] When put on a percentage basis, *only 22 per cent of the patients showed a good or excellent result while about half of the patients showed a fair result* and *another 22 per cent showed no or only poor results.* These figures are from Snyder's clinic, which clinic has probably studied Ertron more extensively than any other group. These results are inferior to the results obtained by other methods of therapy. *Snyder's results from Ertron therapy compare approximately with the results published by Short on a group* (of 300 patients) *who were studied for a period of ten years with only conservative treatment and in whom 16 per cent of cases became inactivated and 53 per cent showed a satisfactory response from only "general therapy."*[119]

Farley and his group reported the results of a five year study of 300 patients with arthritis who were treated with Ertron.[39,41] They gave each patient a capsule of Ertron three times a day and increased this dose, if tolerated, every three days by 1 capsule until the patient was receiving 6 capsules per day. Their patients were advised to drink at least a glass of milk three times a day. If signs of intolerance developed (nausea,

vomiting, frequency of urination, nocturia), the drug was stopped for about a week.

Farley and his associates reported that in practically every case, Ertron therapy was followed by a lessening of the anemia with a return of the hemoglobin and erythrocyte level to normal (although this did not always parallel the clinical improvement of the patient). They noted that the sedimentation rate remained high in many cases despite marked sustained clinical improvement and in some of the cases the rate increased during treatment.

Farley reported that the blood calcium did not rise above 10.6 mg. per cent in any case of his following the administration of Ertron. The non-protein nitrogen rose an average of 9 per cent under this treatment while the blood phosphorus remained about constant. There was no significant elevation of blood cholesterol.

Roentgen findings could not be used to measure the clinical progress of these cases as a return to normal function clinically was often associated with no demonstrable alteration in the roentgen picture, except the marked diminution of soft tissue swelling.[137]

Beneficial results were sometimes obtained as early as three to six weeks after the onset of Ertron therapy, but in some there was no demonstrable effect until after three months. Improvement began with an improved sense of well-being, increased muscle tone and then a decrease in stiffness and pain, increased mobility, a diminution in swelling and deformity, less fatigue and marked improvement in the nutritional state of the skin, hair and nails.

A few patients developed slight nausea, heartburn and anorexia; these disappeared almost immediately when Ertron was withdrawn and usually did not recur when therapy was begun later.

Unfortunately, although Farley and his associates presented a detailed article concerning the results of a five year study of arthritic patients with Ertron in 300 patients, no statistics are given of the number of patients improved but a general statement is presented in the summary which concludes that a large majority showed a general sense of well-being, improved functional activity and increased strength and that the value of Ertron was definitely demonstrated in the treatment of chronic arthritis. One of the authors (Farley) had reported previously on 87 cases of "chronic arthritis" treated with Ertron that every patient who continued treatment for two months or longer in accordance with his instructions showed considerable improvement and in most cases this improvement was so marked that these people were able to resume their business activities or occupations. Snyder, who has used Ertron extensively in chronic arthritis, later stated that Farley's claim was perhaps somewhat overenthusiastic.

Among the patients who improved following Ertron therapy, although improvement began in the first month in a few, this began in the second month in the majority of cases; in some, it was not evident until the third or fourth month.

Snyder believed that Ertron acts as a tonic, improving the patient's general resistance and sense of well-being, which is followed by a gain of weight in a large proporion of cases.

It was necessary to administer Ertron in the largest tolerated doses to obtain the maximal benefit. This dose varied with each patient. In the usual patient, treatment was begun with 1 capsule (50,000 units) three times per day. Three days later the dose was increased by 1 capsule until the patient's tolerance was reached. This was usually 6 per day, but a few patients could take 8 per day. Some patients developed anorexia, nausea, occasional vomiting and nocturia. When the drug was stopped for a day or two, the toxic symptoms disappeared in most cases. The toxic symptoms were never serious enough to necessitate discontinuing the medicine more than ten days. No patient ever developed dangerous toxic symptoms in their series and no fatalities were encountered.

Most of the cases were started on 150,000 units (3 capsules) of Ertron daily as it was necessary to give approximately this amount to produce a therapeutic effect. The average patient did better if his dose was not increased above 300,000 units per day, although a few patients could take as much as 600,000 units per day for several weeks without serious toxic manifestations.

Snyder noted that 4 patients with rheumatoid arthritis of the spine showed from 25 to 50 per cent improvement following Ertron therapy; this improvement was mainly in the muscles of the shoulders and neck.

Snyder thought that he did not observe any noticeable improvement following Ertron therapy in patients with degenerative joint disease.

Several investigators have reported *beneficial results from massive doses of Vitamin D in psoriasis.*[17,72] Freyberg treated 10 patients with psoriasis and rheumatoid arthritis with Ertron, but *did not observe very impressive benefit,* although half of the group had some subjective improvement and several noticed objective improvement; the sedimentation rate was reduced infrequently in these patients.

Snyder believed that the importance of the sedimentation rate has been overemphasized as an index of the severity of the disease or of the efficacy of treatment. He hesitated to use this test as a reliable index for diagnosis and treatment because of the discrepancies which appeared in his cases. Some of his patients who have shown marked improvement or excellent results have shown either no change in the sedimentation rate or an actual elevation of this rate at the end of treatment, while patients with poor results have in some instances shown a depression of the sedimentation rate. He believed that the sedimentation rate was sharply elevated in the acute stage of the disease but subsided as the acute stage passed.

Most writers do not agree with Snyder when he states that although the sedimentation rate is an interesting test to follow, it would be better for the average clinician to depend upon a careful clinical evaluation as to the progress of the disease. In our judgment, one should utilize his clinical judgment and attempt to correlate the laboratory findings with this. The sedimentation rate is not an infallible test for any disease or for the progress

of any disease, but, as a laboratory test, it is still the best index of activity which is available from a laboratory standpoint. One must not forget that even though a rheumatoid arthritis may be inactive, if the individual happens to have a sinusitis, bronchitis or numerous other mild infections, the sedimentation rate may be rapid; however, in the presence of a repeatedly normal sedimentation rate, one hesitates to make the diagnosis of an active stage of rheumatoid arthritis.

Snyder and his associates treated 31 patients with chronic arthritis by means of "Ertron" given intramuscularly.[129] Their preliminary injections into rats and dogs led them to believe that the dosage recommended for oral administration could be given parenterally with safety. In human beings, the first intramuscular dose employed was 500,000 units. Injections were given at weekly intervals, being increased to 1 or 1.5 million units per week. In their hands, it is reported that such treatment proved effective in a majority of the cases. None of the patients developed untoward local or systemic symptoms and no gastrointestinal symptoms developed. In some of the cases, weekly injections of Ertron did not control the patient's symptoms and these workers are now trying the effect of combined parenteral and oral therapy. Although some patients were said to have shown improvement in from two to four weeks, others showed no effect following parenteral Ertron therapy until after three months of treatment.

Toxic Symptoms Following Massive Doses of Vitamin D.—These include *nausea*, anorexia, cramps, diarrhea, *vomiting*, *frequent urination*, excessive thirst, tender teeth and gums, dryness or a sweet taste in the mouth, muscular and joint pain, dizziness, *headache*, hazy memory, muscular weakness, and at times neuralgia along the course of the mandibular branch of the fifth nerves.[12,21]

In 1 patient who was receiving only 50,000 units daily, polyuria, albuminuria, urinary casts and reduced renal function appeared. An *increased blood calcium* was noted in several instances, rising as high as 19 mg. per cent. Gastrointestinal symptoms were usually severe when the high serum calcium values were obtained. Ertron was stopped in 16 per cent of the cases because of its toxicity. There was no significant change in the blood phosphorus during Ertron therapy.

Eighty-six per cent of Slocumb's patients had toxic symptoms in at least one period of treatment with large doses of vitamin D.

In Slocumb's experience, *elevation of the blood calcium occurred frequently after massive doses of vitamin D* and symptomatic improvement was experienced by all of the patients who had such an elevation of blood calcium. *Toxic symptoms occurred with or without an elevation of the serum calcium;* an elevated serum calcium was no gage for the toxicity of the vitamin D for the patient.

Thirty-eight per cent of patients developed an elevation of the blood urea and almost all of these had toxic symptoms to warn the patient to stop taking the vitamin D; in 1 instance, however, a patient with a blood urea of 46 mg. per cent had no symptoms to warn him of the toxicity of the vitamin D to the kidneys. It was concluded that large doses of vitamin D may produce renal damage which was sufficient to result in elevation of the blood urea.

Snyder and his associates noticed no significant change in the nonprotein nitrogen during the course of Ertron therapy. Twenty per cent of their patients showed an increase in blood calcium; one-third of these reported no evidence of toxicity while others complained of nausea, vomiting or nocturia at some time during their treatment. In all of these latter, the toxic symptoms were only temporary and were controlled by discontinuing treatment or decreasing the dose. When hypercalcemia was encountered, the dose of the drug was cut in half or more. If the blood calcium was still elevated at the end of another month, the drug was withdrawn.

During a twelve month period of treatment with Ertron, no increased calcification of blood-vessels was observed; renal function was not impaired; liver or gall bladder function were not impaired.

Part of the toxic reactions observed following the administration of the ultra-violet type of vitamin D were probably due to the presence of *toxisterol*—a toxic side product which is produced in small quantities in the preparation of ultra-violet irradiated viosterol.

It was suggested that toxic symptoms might be avoided by taking a glass of milk three times a day, with each dose of medication.[127] Slight nausea was usually relieved by sodium bicarbonate or rhubarb and soda. The most severe complications following Ertron medication disappeared within ten days after discontinuing the drug.

Gastric distress was not consistently related to any increase in blood calcium. In 1 individual who had taken 8 capsules daily for four years, the blood calcium remained normal.

Snyder and his associates found *no justification for the claim that recalcification of arthritic bones occurs with improvement following Ertron therapy.*

Danger From Overdosage With Vitamin D.—This may produce symptoms resembling hyperparathyroidism. Such changes have been produced in animals resulting in extensive calcium deposits and other pathologic changes in various tissues.[138] Steinberg states that any improvement following massive doses of vitamin D therapy is a non-specific response. He believes that the toxicity of this substance has been overemphasized although he admits there seems to be no special virtue for such medication. Large doses may be dangerous or even prove fatal. Hypervitaminosis may result with a high vitamin D intake if the dietary calcium is increased or if acid substances such as ammonium chloride are given.

Adults have shown no intoxication from 20,000 units of vitamin D per kilogram per day for indefinite periods. Toxic effects of large quantities of vitamin D consist of deposition of calcium following cell injury; this process may be reversed and repaired if one promptly discontinues the use of vitamin D. Large doses of vitamin D are contraindicated in the presence of renal damage (and possibly also of arteriosclerosis).

VITAMIN E

Steinberg reported that vitamin E was found to be of value in the treatment of **primary fibrositis** but that it was of little value in secondary fibrositis.[140] This was given as wheat germ oil in doses of from 2 to 8 cc. per day, given in three divided doses in a tablespoonful of milk at meal time. In those who complained of the unpalatable taste, alpha tocopherol was given in capsules.

Vitamin E did not produce symptomatic relief in patients with sciatica of unknown origin, gout, degenerative joint disease or psychogenic rheumatism. Forty per cent of patients with rheumatoid arthritis noticed definite lessening of muscular soreness and stiffness when given wheat germ oil in doses of from 2 to 8 cc. daily for two to four months, but others experienced no relief.

Steinberg reported that *every one of 30 patients with primary fibrositis was relieved of all symptoms completely when treated with from 2 to 8 cc. of wheat germ oil daily.*

Ingham also noted remarkable improvement from vitamin E in fibrositis.[61]

Freyberg was not impressed with the results of vitamin E in fibrositis. Our results with vitamin E in fibrositis were also unimpressive although doses of 100 mg. of alpha-tocopherol were given t.i.d. for three weeks or longer.

Steinberg was stimulated to try the clinical effect of vitamin E in patients with fibrositis because of the experimental reports concerning muscle changes associated with vitamin E deficiency. He believed that the pathologic picture of fibrositis was similar to that described by Pappenheimer as occurring in ducklings on a diet deficient in vitamin E.[88] Withdrawal of vitamin E from the diets of many animals results in muscular atrophy with restoration of the muscles following intramuscular injection of synthetic vitamin E (alpha-tocopherol). Evans and Burr described a spastic paralysis in suckling young rats when the mothers were deprived of vitamin E. The disease was prevented if the mother's diet contained adequate vitamin E.[37]

Various authors have stressed the importance of diets deficient in vitamin E in causing primary muscular dystrophy.[140] Excellent results have been claimed in the use of vitamin E or the synthetic tocopherols in the treatment of muscular dystrophies; the addition of vitamin B complex increases its therapeutic efficacy.[143]

In muscular dystrophy, one may give 2 cc. of wheat germ oil mixed with vitamin B complex each day; this promptly results in increased resistance to fatigue, improvement of appetite and normal musculature replacing rubbery muscles. In long-standing muscular dystrophy with contracture deformities, 4 to 6 cc. of wheat germ oil is suggested per day; no unpleasant after effects have been noted. This dose is also of value in the treatment of primary (but not of secondary) fibrositis.[140]

Vitamin E occasionally has a beneficial effect in some patients with amyotrophic lateral sclerosis.[135,152,153] Synthetic alpha-tocopherol in doses of 500 mg. in sterile oil given intramuscularly temporarily relieves pain and weakness in the legs in these patients.

It has not been proven that vitamin E is required for normal health or reproduction in man.[156] Reports have indicated a possible therapeutic rôle of vitamin E in threatened and spontaneous abortion.[120]

VITAMIN K

Vitamin K is necessary for the maintenance of normal prothrombin concentration in the blood. The absorption of this vitamin is dependent upon the presence of bile in the intestinal tract. Human beings derive the fat-soluble vitamin K from an adequate diet and from bacterial activity in the intestine.[96,97]

Prothrombin deficiency may occur as a result of a diet inadequate in vitamin K, of inadequate absorption of vitamin K in the gastrointestinal tract (due to lack of bile salts or a decreased area for absorption) or of faulty conversion of vitamin K to prothrombin in the liver due to hepatic disease.[124] Many infants have a marked prothrombin deficiency, associated with hemorrhagic disease of the newborn; vitamin K and its esters are specific in treatment of the hypothrombinemia found in this latter disorder.

The composition of prothrombin is as yet unknown. The liver is probably the seat of prothrombin formation and of the synthesis of vitamin K.[34]

Vitamin K is widely distributed in nature and is found especially in the leafy portions of green vegetables; vitamin K_1 may be obtained from alfalfa and vitamin K_2 from putrefied fish meal. Compounds with vitamin K activity have been obtained in crystalline form.[4,47]

The most potent of the synthetic vitamin K preparations is 2-methyl-1,4-naphthoquinone.[101] In vitamin K deficiency, from 1 to 5 mg. of this substance should be given orally each day together with bile salts (or emulsified in 30 to 50 cc. of bile). If one wishes to produce a quick elevation of prothrombin, 2-methyl-1,4-naphthoquinone may be given intramuscularly in corn oil;[5] this is inexpensive, simple and effective and produces an effect within several hours.[91] Bile salts are not needed in the intestinal tract if the synthetic vitamin is given parenterally.

Vitamin K is ineffective in severe hepatitis or cirrhosis of the liver. It is of no value in hemophilia or thrombocytopenic purpura as there is no deficiency of vitamin K or prothrombin in these diseases.

Prolonged administration of salicylates may markedly lower the blood prothrombin level; when salicylates are to be given for long periods, vitamin K should also be prescribed orally.

Hypoprothrombinemia has been reported in about 50 per cent of patients with rheumatoid arthritis and in some individuals with gouty arthritis.[99] Although the administration of vitamin K restores the prothrombin level to normal, no reports are available on the significance of these observations in rheumatic diseases.

VITAMIN P

The possibility of the existence of vitamin P, a substance closely related to vitamin C, has been raised;[9] others do not believe that such a vitamin exists.[166] It has been prepared commercially from orange peel; a concentrated solution of vitamin P given by mouth proved of value in the treatment of several children with allergic, infectious and nutritional purpura.[73] The vitamin is also present in lemon juice and Hungarian red pepper. The active substance was found in pure flavone or flavone glucoside. It has been claimed that this vitamin is necessary for the maintenance of normal capillary resistance.[33]

VITAMIN T

The possibility of the existence of this vitamin results from its ability to regularly produce an increase in blood platelets in normal children.[155] This work has not been confirmed as yet. This factor is found in sesame oil but not in olive oil or cod liver oil.[115]

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CHAPTER XXVII

RÔLE OF THE ENDOCRINE GLANDS IN ARTHRITIS

INTEREST has been focused upon the endocrine glands recently with the *experimental production*, by Selye and his co-workers, *of a polyarthritis* following overdosage *with desoxycorticosterone acetate.*[77] The joint lesions produced experimentally in rats resembled histologically those seen in acute rheumatic fever. The joint lesions were produced more readily by the desoxycorticosterone acetate in adrenalectomized or thyroidectomized than in intact rats, and especially if they were exposed to cold.

The relationship between various forms of joint disease and endocrine disturbances has been commented upon by numerous authors.[55,57,88,97] Rheumatic conditions have been observed frequently in *hypothyroidism*[22,52] and occasionally in *Addison's disease*[18] and *status thymicolymphaticus.*[53] Joint lesions have also been reported associated with *hyperfunction of the adrenals* or *thyroid*[20,59,96] as well as in *Cushing's syndrome.*[50] The *arthralgias of the menopause* are well known, as well as the development of true rheumatoid arthritis at this time.[39,40,44,51,60] Joint lesions may also appear during or following the production of an artificial menopause.[42,95] *Intermittent hydrarthrosis* has been reported with reaccumulations of fluid in the joint cavity with direct time relation to the sexual cycle.[10,72,75] Rheumatoid arthritis, on the other hand, frequently lessens greatly in severity or may almost completely clear temporarily during *pregnancy*, only to recur in most instances soon after delivery.[25,35]

Numerous investigators have speculated on the *rôle of* the *pituitary* in the production of some cases of arthritis.[23,31,40,49,65] The hypertrophic joint changes associated with *acromegaly* are well known; this usually has a predilection for joints of the spinal column. Joint lesions have also been reported in patients with *hypopituitary dwarfism*,[98] *diabetes insipidus*[33] and *dystrophia adiposa-genitalis.*[76]

THE THYROID GLAND

The majority of patients with chronic forms of arthritis have normal thyroid activity.[26] In most instances, the occurrence of hyperthyroidism or hypothyroidism is a coincidental finding in arthritic patients. Bauer found no proof for support of an endocrine theory of degenerative joint disease; in his series, subnormal basal metabolic rates occurred no oftener among that group than among others of similar age.[5] Inasmuch as thyroid dysfunction and chronic arthritis are both common diseases, one is not surprised that they might coëxist in some persons.

Degenerative joint disease has been found in almost one-third of all patients with myxedema.[58] However, myxedema occurs most often in

middle-aged individuals in whom degenerative joint disease is found frequently even in the absence of hypothyroidism.

Numerous investigators[15,66,91] have studied the *basal metabolic rate* in various forms of arthritis and have reported conflicting results.[11,12] In summarizing these, the following may be said:[36,89]

Approximately one-third of patients with *osteo-arthritis* (*degenerative joint disease*) have basal metabolic rates below −15 per cent.

In *rheumatoid arthritis*, figures are variable but a normal rate is obtained in the majority.

If hypothyroidism is suspected, one should determine the *blood cholesterol* level as well as the basal metabolic rate. The blood cholesterol is usually elevated in true hypothyroidism; this serves as a check on the metabolic test. A low B.M.R. does not necessarily mean hypothyroidism.

Benefits of Administration of Thyroid Substance in Arthritis.—In cases of hypo- or hyperthyroidism complicating various forms of arthritis, the thyroid dysfunction should be treated as would be done had the patient no arthritis; one should not expect improvement in the arthritis as a direct result of treatment of the thyroid disorder.

Some workers have reported improvement in general well-being and in arthritic manifestations (both subjectively and objectively) when desiccated thyroid has been given to patients with chronic forms of arthritis associated with a lowered basal metabolic rate. In one series, 20 per cent of patients with markedly *active rheumatoid arthritis* showed improvement following its use.[71] *Over 40 per cent of patients with degenerative joint disease* and mixed forms of arthritis *improved following the use of desiccated thyroid; improvement was noted especially in the knees.* Reduced swelling, increased motion and decreased pain were observed in the knees of overweight women with osteo-arthritis following thyroid therapy.

Hall and Monroe[31] concluded that *thyroid was of benefit in almost 50 per cent of 116 patients with degenerative joint disease* in which it was used *and in only 16 per cent of 103 patients with rheumatoid arthritis.*[32] Beneficial effects included a decreased swelling and improved function of the joints, improved general well-being, lessened joint sensitivity, lessened sensitivity to cold, a change from a dry to a moist skin, and loss of weight (especially about the abdomen, hips and ankles) in obese individuals. These workers have shown the *fallacy of relying upon one determination of the B.M.R. inasmuch as some of their patients whose B.M.R. was normal at the first reading, showed a reading much more below normal on subsequent examinations.*

Rynearson and Sacasa reported a case of *hypertrophic pulmonary osteoarthropathy* which developed *coincidentally with* or secondary to the onset of *postoperative myxedema.*[74] Cushing[19] referred to a similar case in which clubbing of the fingers developed eight years after subtotal thyroidectomy had been performed for a toxic goiter. In Thomas'[94] patient with a severe toxic goiter, clubbing of the fingers developed eight months after subtotal thyroidectomy. Roentgen examination then revealed subperiosteal proliferation in the bones of the hands and feet and in the long bones of the extremities. Thinning of the subperiosteal new bone was reported with

definite subjective improvement three and a half months after the institution of thyroid medication orally (because the basal metabolic rate had dropped to −20 per cent).

One may see patients with *degenerative joint disease* with a moderate or marked lowering of the B.M.R. in whom the administration of thyroid substance seems to be of benefit. These patients are usually obese women, about the time of the menopause, manifesting arthritic changes especially in the knees and hands. It is true that a lessened load upon the knees which accompanies the reduction in weight following thyroid medication may be responsible, at least in part, for the improvement.

Indications for Thyroid Medication in Arthritis.—The main indication is the finding (by repeated determinations) of a subnormal basal metabolic rate or an increased blood cholesterol. The normal range of B.M.R. lies approximately between −10 and +15 per cent. The normal blood cholesterol ranges from 170 to 230 mg. per cent. Blood cholesterol may be elevated in pathologic states other than hypothyroidism; these include obstructive jaundice, diabetes mellitus, nephrosis, disturbance of lipoid metabolism (as in Niemann-Pick's disease, xanthomatosis, Gaucher's disease), etc. Thyroid substance may also be given to arthritic patients who have a normal B.M.R. if symptoms of hypothyroidism are noted. These include sluggishness, dry skin, loss of hair, decreased peripheral tendon reflexes, obesity, bradycardia, etc. It is most beneficial, in general, in cases of degenerative joint disease in obese women, with subnormal basal metabolic rates, and in patients with true myxedema, who complain of arthralgia.

Dose of Thyroid Employed.—One may begin with 15 mg. (¼ grain) of desiccated thyroid daily if thyroid substance is indicated. This may be increased to 15 or 30 mg. (¼ or ½ gr.) twice a day or to 60 mg. (1 gr.) or more three times a day. Basal metabolic rates and blood cholesterol determinations are performed every several weeks in these patients. Signs of overdosage include tachycardia, arrhythmias, marked sweating, precordial distress, nervousness, dizziness, a sense of nervous tension, headaches, emotional instability, pressure over the eyes, etc. After several weeks, the dose of thyroid substance may be decreased materially (in most patients).

Marked variation is found in the potency of various brands of thyroid substance. For this reason, the brand of such medication should be mentioned in the prescription and this should not be changed unless results are not obtained.

Wide variation in tolerance to thyroid substance may be found. A very small dose may be effective in one patient while a much larger dose may be required for a similar condition in another. Clinical improvement may not be noted for several months following the onset of thyroid therapy in some cases. The cardiac status should be checked carefully before thyroid medication is begun.

Relation of Hyperthyroidism to Joint Diseases.—In our experience, the occurrence of hyperthyroidism is only a coincidental finding in arthritic patients. The literature upon this subject is scanty. Duncan stated that patients with hyperthyroidism commonly complain of "rheumatism" and that there is a significant relationship between hyperthyroidism and disease of the joints.[21] He reported a series of 50 patients in whom he felt there was no question but that an intimate relationship existed between the hyperthyroidism and the joint manifestations. In some of these patients, severe and intractable joint pain was alleviated following thyroidectomy.[22] Other workers thought that the association of hyperthyroidism with rheumatoid arthritis was more than a coincidence.[28,43,85] Veiel stated that articular symptoms were usually absent in patients with exophthalmic goiter.[96]

In almost 30 per cent of a group of 292 patients with hyperthyroidism, a specific relationship between abnormality of joints and hyperthyroidism was found. Marked improvement in the arthritis was observed following removal of an adequate amount of thyroid gland. Others have described similar prompt relief in chronic progressive arthritis (associated with thyrotoxicosis) following thyroidectomy.[6,93,103]

THE PARATHYROID GLANDS

Careful study has failed to support the theory that hyperparathyroidism was responsible for some cases of rheumatoid arthritis. Parathyroidectomy should not be performed as a therapeutic measure in arthritic patients. Patients have been examined whose arthritis became worse following parathyroidectomy. Freyberg has reported the development of rheumatoid arthritis in a patient who had chronic hypoparathyroid tetany (post-thyroidectomy) for years. No definite relationship has yet been proved between arthritis and parathyroid function.

Most of the literature concerning the relationship of parathyroid bodies to arthritis deals with the effect of parathyroidectomy in chronic ankylosing arthritis.[61,78]

Chemical findings in hyperparathyroidism include: an elevation of the blood calcium and phosphatase; increase in the urinary calcium and phosphorus; and a lowered blood phosphorus.

Berard and his co-workers found the parathyroids to be macroscopically normal in 20 of 21 patients with chronic rheumatism in whom these glands were removed.[9] Histologic sections revealed no marked changes except for the presence of hemorrhagic lesions, which were evidently due to the trauma incident to operation. No hyperplasia of parathyroid tissue was noted. In their experience, hypercalcemia was rare in chronic rheumatism and signs of augmented parathyroid function were absent.

At present, *the consensus of opinion* is that *there is no proved relation between arthritis* (in any form) *and primary hyperparathyroidism* and that *parathyroidectomy is not indicated in the treatment of any form of arthritis.* This is contrary to views expressed in European literature within the past ten years.[86,92]

Helfet set forth a concept of parathyroid function in which he suggested

that parathyroid extract exerts its primary effect on phosphorus metabolism, and that the blood inorganic phosphate level was controlled by parathyroid extract.[34] He believed that an accumulation of phosphate in the blood stimulated increased secretion of parathyroid extract; this latter may result in mobilization of calcium ions from the bones to form calcium phosphate, which is inactive and may be excreted unless kept in solution in the blood by an increased amount of parathormone. In view of the generalized decalcification of the skeleton in rheumatoid arthritis, in addition to the numbness in the extremities, stiffness and pain in the joints, Helfet theorized that the mechanism in some cases of this disease may be one of secondary hyperparathyroidism. He reported that he obtained encouraging results after treatment of some cases of rheumatoid arthritis on this basis; however, he admitted that there is no definite evidence that a hyperparathyroid state exists.

In order to prevent further withdrawal of calcium from the bones in these patients, he suggested that the intake of phosphorus in the diet be reduced (in order to remove the stimulus to overproduction of parathyroid extract); such a low phosphorus diet was found to be impracticable. As an alternative method, he gave aluminum salts by mouth. Aluminum combines in the intestine with phosphates and the resulting precipitated insoluble aluminum phosphate is excreted unchanged by the bowel. Aluminum acetate was used at the onset of the experiments but in later work aluminum gluconate was substituted as a more pleasant preparation. Aluminum salts are astringent and it is necessary to add syrup, essence of cherry and other substances to the mixture to render it palatable.

No toxic reactions or gastrointestinal upsets were noted following the prolonged use of aluminum salts in small doses (4 cc. of a 2.5 per cent solution q.i.d.). If mild constipation occurred, this was easily controlled with mineral oil. All of the patients received not less than a pint of milk per day as too large a dose of the aluminum salts could produce rickets.

In a small series of patients with rheumatoid arthritis on this regimen, the appetite, color and sense of well-being improved in three or four weeks, and in some cases the subsidence of joint symptoms was dramatic; an increase in movement was noted in all joints except those ankylosed at the onset of therapy. Spindle-shaped swellings of the finger joints disappeared. These changes were seen on a program of only aluminum salts, without any physical therapy or other treatment.

ARTHRITIS AT THE MENOPAUSE

Diffuse aches and pains in muscles and joints are common at the time of the menopause. These have been termed *menopausal arthralgia*. There appears to be no objection to the use of this name to indicate such symptoms, when these are present without any joint swelling or other objective evidence of joint pathology. Such arthralgias are usually easily controlled by use of stilbestrol or estrogenic substances either orally or parenterally in sufficient doses.

However, when one employs the term "menopausal arthritis" there is usually a storm of protest. Such a term could only mean an arthritis occurring at the time of the menopause. We are opposed to the general use of the term "menopausal arthritis" because this does not clearly set forth the type of arthritis under consideration. At the time of the menopause, an individual may develop true rheumatoid arthritis, degenerative joint disease, fibrositis or other forms of rheumatic disease. Except in the case of mere arthralgias, we prefer to speak of the arthritis occurring at the time of the menopause in more definite terms, calling this either rheumatoid arthritis, degenerative joint disease, etc., depending upon the type of joint pathology which is found.

Rheumatologists agree that certain forms of arthritis are often found in women about the time of the menopause; however, many do not admit that these are related in any causative way to the menopause, insisting that such a relationship is purely coincidental. Osgood has postulated that added excretion of anterior pituitary hormones after castration may damage joint structures (resulting in pains as in so-called menopausal arthritis).[62]

In a discussion of the subject, Slocumb differentiated distinctly between arthralgias occurring at the menopause, which were benefited by estrogenic substance or stilbestrol, and rheumatoid arthritis occurring coincident with the menopause.[84] This latter group of patients was not helped by estrogenic substances or stilbestrol. Cecil[14] used the term menopausal arthritis. He noted that young women who had a hysterectomy may develop pain in the muscles and joints, with no other symptoms; this should be referred to as menopausal arthralgia.

Patients with arthritis at the menopause have no uniform clinical picture. The process has been described in the hands, knees, wrists, neck, fingers, metacarpal joints, etc., as either an arthritis or a synovitis. Many rheumatologists consider menopausal arthritis to be either degenerative joint disease and fibrositis, or rheumatoid arthritis or an admixture of rheumatoid arthritis and degenerative joint disease occurring at the menopause.[37] *Rheumatoid arthritis may begin rather abruptly in some women following an artificial menopause.* A synovitis is the predominating pathologic lesion, but degenerative joint changes have been reported.

Most of these patients seem to present a picture of mixed (rheumatoid and degenerative) arthritis. In some, the basal metabolic rate is subnormal and the blood cholesterol is elevated. These individuals often improve following the administration of desiccated thyroid gland.

Albright and his associates have shown that after the menopause, women are prone to develop osteoporosis of the pelvis and spine (so-called *post-menopausal osteoporosis*).[1] These patients could be put into a positive calcium and phosphorus balance by the intramuscular injection of estrogenic substance (Progynon B). It is not yet known whether this effect of estrogenic substance on the calcium balance in post-menopausal osteoporosis is sufficient to be of practical value.

These workers define *osteoporosis* as a condition of adult bone in which

there is a failure of osteoblasts to lay down an organic matrix, as opposed to *osteomalacia* in which there is a failure of deposition in this matrix of the calcium-phosphate-carbonate complex. They tried estrogenic substance in post-menopausal osteoporosis because of the effect of such therapy in male doves. Hens and doves have high serum calcium values when they lay eggs.[73] The bones of male doves have been found to be osteoporotic in comparison with those of the females. The administration of estrogenic substances to these male doves results in marked increase in the density of their bones due to a stimulation of the osteoblasts.[67]

Albright and his associates have discussed the clinical features of post-menopausal osteoporosis.[2] They emphasized that osteoporosis was not a disorder of calcium metabolism, but rather an atrophy of the bone matrix.

Factors in the causation of osteoporosis include disuse, senescence, and possibly faulty diet; this latter (diet) is not yet established as a factor. Albright believed that the post-menopausal state was the most common etiologic factor in osteoporosis because of the constant tendency for osteoporosis to occur in women after the menopause and the beneficial effect of estrogenic therapy on the retention of calcium in this condition.

In 42 patients under the age of sixty-five with generalized osteoporosis, 40 were women who had passed the menopause; in no instance did the condition occur in women before the menopause.

Post-menopausal osteoporosis has a predilection to involve the pelvis and spine. The long bones are less likely to be involved and the skull is practically never involved in this condition. These patients have a tendency to develop atrophy in other tissues, especially the skin.

Albright believes that stresses and strains are an important stimulus to osteoblastic activity and that atrophy of disuse is due to a decrease of this stimulus.

Repeated pregnancies are a drain on the body's supply of calcium and are a well-known factor in the causation of osteomalacia. Long-standing thyrotoxicosis also leads to increased radiability of the skeleton. Senile osteoporosis is also a well-known entity. Like other tissues, bones atrophy in old age in both sexes; fractures of the hip in the aged are a common clinical manifestation of this.

Some of the *features of post-menopausal osteoporosis* include the following:

1. Occurs almost solely in women (always past the menopause).
2. Predilection for the spine and pelvis.
3. Long bones are involved only in cases of more severe involvement.
4. No involvement of the skull (differentiating it from hyperparathyroidism).
5. Spinal lesions consist of crushed or fractured vertebræ, fish vertebræ (with expansion of the intervertebral disks into the vertebræ, converting them into biconcave disks), and herniation of the nucleus pulposus through the end-plates of the vertebræ.

The usual clinical picture of post-menopausal osteoporosis is that a woman, about ten years after the menopause, experiences a pain in the low back following a minor jolt (such as a small bump in an automobile). An *x*-ray examination is made which reveals the condition.

Kling has described 125 cases of *juxta-articular adiposis dolorosa, i. e.*, painful masses of fat near the joints.[48] This occurred only in women at the time of the menopause. These masses were most common about the knees, elbows, ankles and hips. Many of these patients had osteo-arthritis. No treatment was of special merit.

Most women who have rheumatic symptoms during the menopause exhibit no definite articular changes so that the term menopausal arthralgia should be used to differentiate this group from those who develop true rheumatoid arthritis. Various workers have reported beneficial results and control of the arthralgia with thyroid substance or natural or artificial estrogens in patients with menopausal arthralgia.[39]

Estrogenic therapy has been tried in a few cases of fibrositis occurring with hypomenorrhea in young women. Improvement was noted in some cases, but the degree of improvement was relatively slight, usually temporary and always incomplete; this therapy did not control the rheumatic symptoms in most cases.[26]

No beneficial effect could be noted following the treatment of patients with rheumatoid spondylitis (spondylitis rhizomelique) with estrogens.[26] This therapy was tried because of the predominance of rheumatoid arthritis of the spine in males and because of the definite effect of estrogens on pelvic ligaments.[39]

Freyberg's conclusions concerning the use of **estrogenic substances in rheumatic disease at the menopause** were as follows:

1. When rheumatic disease develops at the time of the menopause, estrogenic substance may often be of significant benefit on the rheumatic disease as well as on the other menopausal symptoms.
2. Oral preparations were as beneficial as injected preparations when used in adequate amounts.
3. In patients whose arthritis existed before the onset of the menopause, estrogenic therapy had little or no effect upon the rheumatic disease.
4. Estrogenic therapy had little or no effect in patients with fibrositis with hypomenorrhea or in rheumatoid arthritis of the spine.

No known hormone specifically cures arthritis. Estrogenic substances, however, may be of definite value when used in those individuals whose rheumatic manifestations begin with the menopause.

Hall and others have reported a marked decrease in arthralgic pains in menopausal arthritis following *estrogenic substance*.[31,38,45,70,79,87]

Cohen and his associates found that the striking benefits following administration of estrogenic substance to women with degenerative joint disease coincident with the menopause were of a general systemic nature; in order to produce relief of joint symptoms, it was necessary to use orthopedic and physical therapy as well as reduction of weight.[17] Little benefit was obtained from large doses of estrogenic substance in women with rheumatoid arthritis without disturbance of menstrual function. In

rheumatoid arthritis concomitant with the menopause, estrogenic substance was of benefit.

Ishmael found satisfactory results from injections of stilbestrol in 80 per cent of 48 patients with menopausal arthralgia; similar results were obtained with estrogenic hormones.[40] He obtained complete clinical remissions in the average patient with menopausal arthralgia. Ishmael used the term "menopause arthritis" to a group of cases in which the symptoms

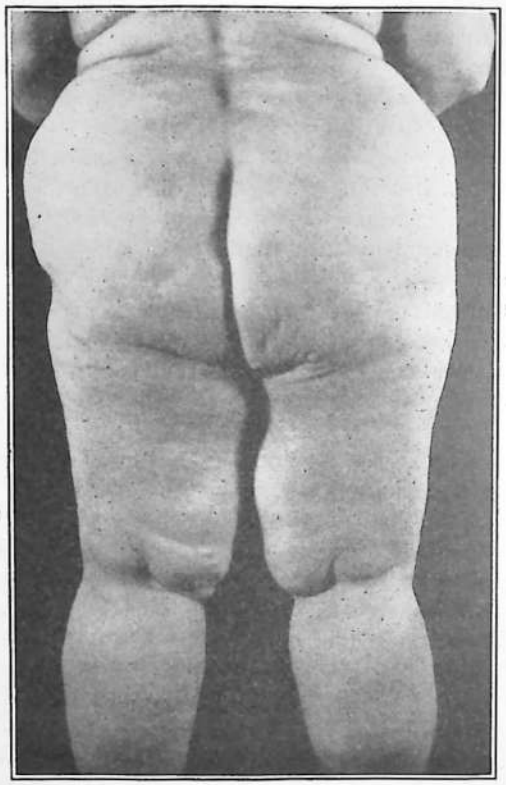

FIG. 110.—Hypersensitive bulging fat pads at the inner sides of the knees in an advanced case of juxta-articular adiposa dolorosa. Roentgenogram of the knee joint was negative. (Kling, courtesy of Am. Jour. Med. Sci.)

appeared during or soon after the natural or artificial menopause. He suggested that a lowered amount or absence of estrogenic hormone was capable of producing, by some means, rheumatic disease.

Stilbestrol is a cheap, orally effective, synthetic estrogenic preparation. It loses very little potency when given by mouth and is two and a half times as potent as estrone (theelin) by injection.[53] It may also be given intramuscularly in oil. No blood or urinary changes have occurred following its use; there has been no evidence of impairment of liver function.

When given intramuscularly, it is from one-third to one-half as potent per mg. as estradiol benzoate (Progynon B). One mg. of stilbestrol orally is equivalent to 1 mg. of estrone (theelin) given intramuscularly. Sex hormones may also be administered by pellets implanted subcutaneously through a surgical incision or through a trocar.[29]

Physicians usually give not more than 0.5 mg. of stilbestrol daily in most women at the menopause; if this dose is not exceeded, nausea and vomiting will not be frequently encountered.[102] Practically all reports have agreed as to the potency, convenience of administration and low cost of stilbestrol.

Except for gastrointestinal distress, principally nausea (which has been reported in from 10 to 20 per cent of patients), no serious damage has been reported following its administration in daily doses of 0.5 mg. or less if not continued over too long a time.[99]

Mazer and Israel presented an experimental and clinical evaluation of *stilbestrol*. They concluded that stilbestrol was a highly active estrogen whether given orally or hypodermically and that the ratio between the oral and hypodermic doses was 5 to 1. It produced all of the therapeutic effects obtainable with natural estrogens in women who tolerated stilbestrol. Compared with natural estrogens, STILBESTROL IS HIGHLY TOXIC, producing nausea, vomiting, epigastric pain and malaise in over 50 per cent of patients receiving 2 mg. hypodermically every fourth day; this is equivalent to 10,000 rat units. The incidence of toxicity is about 20 per cent when given orally at this dosage level. STILBESTROL MAY BE ADMINISTERED SAFELY IF ONE DOES NOT EXCEED 0.5 mg. daily by mouth or every fourth day hypodermically; when this dose is employed, nausea and vomiting occur in only about 3 per cent of patients. No tissue damage could be demonstrated following the use of relatively large doses over a long period of time. One may safely employ from 0.2 to 0.5 mg. of stilbestrol daily by mouth or every fourth day hypodermically; this corresponds to a potency of from 10,000 to 25,000 international units of estrone. This will not relieve all patients with the menopausal syndrome. If larger doses are required, one should not employ stilbestrol because of the high incidence of reactions.[56]

In some of these patients, improvement in the arthritic symptoms has followed the intramuscular injection of the patient's own blood (5 to 10 cc.) added to the preparation of estrogenic substance in oil.

In rheumatoid arthritis and rheumatoid spondylitis in men over forty years of age, and in older men with hypogonadism and marked neurasthenia, one may cautiously administer testosterone propionate (10 mg. or more three times a week intramuscularly or 4 mg. in an ointment rubbed into the skin daily).[4,46,90] Testosterone may also be effective orally. This drug is usually well tolerated and often results in increased vitality, lessening of fatigue and is very effective in the male climacteric (manifest by nervousness, sweating, hot flushes and melancholia).[30,47] Testosterone does not alleviate the arthritis.

PITUITARY GLAND

Chronic arthritis, together with osteophyte overgrowth in the region of the joints, is occasionally due to pituitary overactivity.[41]

The growth of cartilage may be influenced by endocrine factors.[7] The Silberbergs have injected anterior pituitary substance into young and

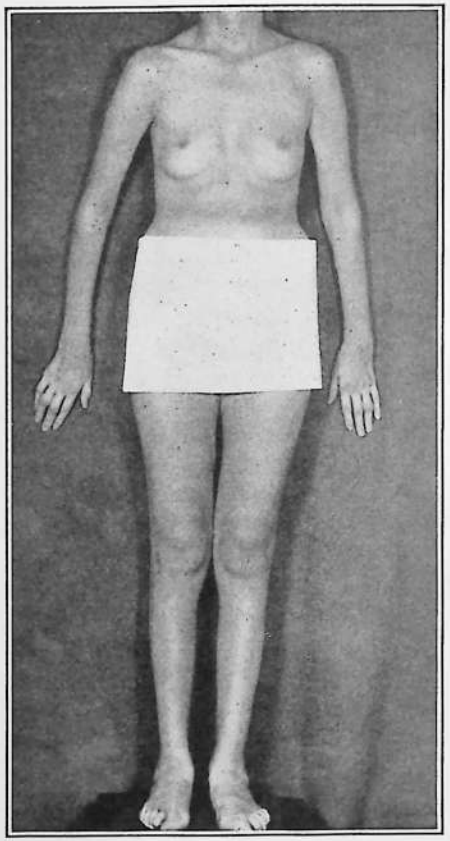

FIG. 111.—Rheumatoid arthritis in a girl, aged twenty-five years. Arthritis began three years ago, four months after the onset of amenorrhea. Patient also has a pituitary enlargement (fossa 9 by 15 mm.) and has acromegaloid features. Patient's sister also has rheumatoid arthritis and a top normal sized pituitary fossa; her arthritis also began after a premature menopause at the age of twenty-five years.

adult guinea pigs and have produced severe joint changes comparable to chronic joint disease in man.[80,82,83] They have studied the response of cartilage and bone of newborn guinea pigs to stimulation by various hormones (anterior pituitary extract, estrogens and thyroxin).[81] Anterior pituitary extract produced hypertrophy and hyperplasia in the transitional zone and later liquefaction and ulceration of the articular cartilage in young guinea pigs; in adult animals, a predominance of degenerative

changes was noted. Gonadectomy in immature subjects produced similar sequelæ. The growth and differentiation of euhyaline cartilage in normal young guinea pigs was enhanced by thyroid substance given orally for from two to thirty days.

In newborn guinea pigs, cartilage had the tendency to respond to hormonal stimuli by more proliferative and less retrogressive changes than in older animals. It was noted that the age of the animals played an important rôle in determining the reaction of the cartilage to hormonal stimuli.[81]

Injections of pituitary extract of cattle for fifteen to twenty-eight days into newborn guinea pigs resulted in proliferation and hypertrophy of the cartilage of the epiphyseal growth zones and joints; no appreciable retro-

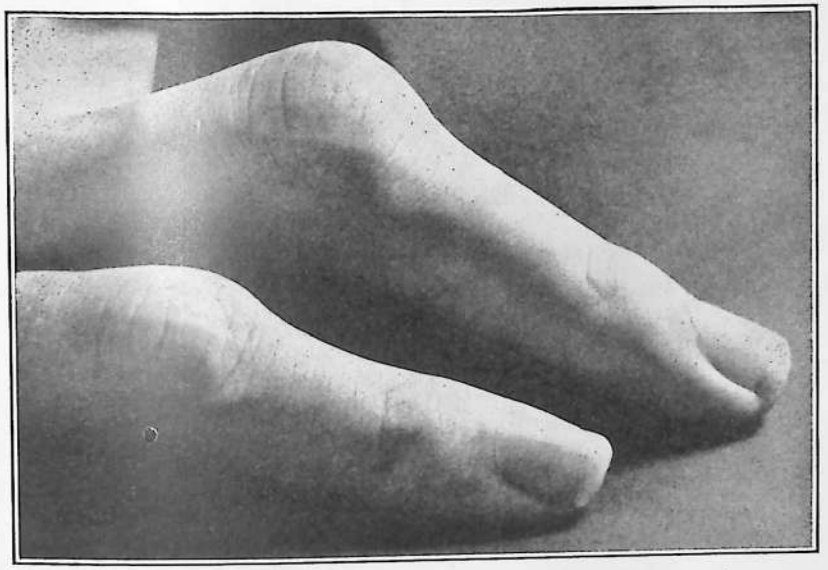

FIG. 112.—Contracture flexion deformity in young female, aged twenty-five years, with rheumatoid arthritis beginning four months after onset of amenorrhea. (See Fig. 111.)

gressive changes were seen. Other findings were an increased amount of newly formed endochondral and periosteal bone.

When estrogenic substance was injected into newborn guinea pigs, during the first three weeks of treatment there was a suppression of the growth of the cartilage of the epiphyseal disks and the joints; later, there occurred a marked proliferation of cartilage cells. There were practically no retrogressive changes. During five weeks of estrogenic administration, the deposition of bone was not greater than normal.

Thyroxin given for four weeks produced an acceleration of growth and differentiation of cartilage of the epiphyseal lines and joints, and enhancement of endochondral ossification.

Putnam and his associates produced *experimental acromegaly in dogs by*

the administration of pituitary substance; at necropsy, they found enlarged bones and *osteophytes* and flakes of bone lying in the anterior ligament of the spine.[68,69] Some of the osteophytes were indistinguishable from those of degenerative joint disease and extended into tendons and ligaments.

Alterations in the vertebral column in **acromegaly** were described by Marie in 1886.[54] These changes often gave the acromegalic a marked

FIG. 113.—Rheumatoid arthritis involving the feet in young girl (appearing shortly after cessation of menses at age of twenty-five years). Patient has enlarged pituitary fossa. The changes are most prominent at the metatarsophalangeal joints; there is marked narrowing of the joint space, some broadening of the heads of the phalanges and evidence of spur formations. (See Fig. 111.)

lower dorsal kyphosis. In a study of 584 acromegalic patients, kyphosis was found in 60 per cent.[3] Other workers have discussed enlargement of the vertebræ and an associated spondylitis with acromegaly. This *spondylitis of acromegaly* is readily distinguished in the roentgen-ray by the symmetry of the process.[16] There is a growth of new bone on the lateral and anterior aspects of the bodies of the vertebræ so that the latter are surrounded on 3 sides by new periosteal bone which has been deposited

upon the diaphysis. Fissuring is seen at the margins of this partial bony sheath; this results in a row of tooth-like processes above and below the vertebral disk. The spondylitis is most pronounced laterally.

In patients with long-standing advanced acromegaly, one may also find changes in the intervertebral disks, which may become thin and show irregular margins. Erdheim showed (by careful anatomic and roentgen-ray studies) that the altered configuration of the vertebral column in acromegaly was due to characteristic changes in the vertebral bodies and disks, often with associated specific spondylitis.[24]

FIG. 114.—Rheumatoid arthritis of the hand in a young girl, aged twenty-five years, coming on four months after the onset of amenorrhea (which has now persisted three years). There is general demineralization of the bones of the hand; the proximal interpharyngeal joints are narrowed. (See Fig. 111.)

Hypoactivity of the pituitary gland may lead to fatigue, anemia, increased susceptibility to infection and general atrophy, all of which are common findings in rheumatoid arthritis.[64] Some children with Fröhlich's syndrome have pain in the region of the hips and knees. These individuals are often helped by the administration of thyroid substance and anterior pituitary extract. The latter may be given hypodermically (such as Polyansyn, 1 or 2 cc. two or three times a week) or orally (as anterior

pituitary extract, 0.1 to 0.2 gm. [1½ to 3 gr.] per day in divided doses immediately after meals); Dunn claims that such oral administration is definitely of value.[23] Thyroid substance is given by mouth in small doses, 30 to 60 mg. (½ to 1 gr.), two or three times a day, as tolerated.

Attention has been called to the *potential importance of the pituitary in relation to rheumatic disease.*[65] Patients with arthritis often experience an exacerbation of symptoms shortly preceding the onset of the menstrual flow. This may be related to water retention which occurs at this period. Furthermore, women with rheumatoid arthritis may experience a remission of the active joint symptoms during pregnancy. In others, arthritic symptoms may appear at the menopause, leading to the so-called menopausal arthralgia. This may occur following the normal menopause or following a surgical or *x*-ray menopause. In these patients, the arthritic symptoms are usually definitely lessened following use of estrogens or stilbestrol.

It has been suggested that the symptoms of menopausal arthralgia may be due to an increased level of pituitary substances, incidental to depletion of estrogens, and that the beneficial effect of estrogenic substances is secondary to the relative depression of pituitary hormone production caused by the estrogenic substances. With this same thought, *pituitary irradiation has been suggested as a therapeutic measure in these cases.* We have seen no definite improvement following pituitary irradiation in several patients whose typical rheumatoid arthritis began shortly after spontaneous cessation of menses between twenty-five and thirty-five years of age.

In *acromegaly,* as well as in *pulmonary osteo-arthropathy,* the increase in the size of the bones is due to increase of both the periosteal and subcutaneous tissues.[27] Tufting of the terminal phalanges of the fingers and toes is also present in both diseases.

Fried has suggested that the diffuse osteo-arthropathy found in neoplastic diseases of the lungs is probably due to an endocrine imbalance (dyspituitarism) akin to acromegaly.

Brugsch reported a case of a patient with pachyderma and periostosis and suggested the name *acropachyderma with pachyperiostitis.* This is a disturbance in growth characterized by clubbing and squaring of the extremities, thickening of the skin over the face, scalp and extremities, and deformities of the long bones and their periost.[13] This syndrome has received but little attention in the United States. The face of the patient with this syndrome resembles that of a leper; his arms and legs are similar to those found in pulmonary osteo-arthropathy. The syndrome uniformly affects males after puberty and leads to extreme thickening of skin and bones.

On roentgen examination, it is found that the periosteal enlargement and apposition is strongest in the diaphysis of the metatarsal and metacarpal bones, leading to a "bottle-like" deformity. One may find only a few small irregular particles of new periosteal formation or confluent masses. The terminal phalanges are always spared. The cortical bone

is extremely thickened especially over the diaphysis, where eburnation may lead almost to obliteration of the central cavity.

The condition differs from acromegaly, pulmonary osteo-arthropathy, cutis verticis gyrata and leprosy; Brugsch discusses the differential diagnosis from these conditions.

GONADS

Except for a possible relationship between menopausal arthralgia and deficiency of ovarian secretion there is no outstanding evidence that these organs play a rôle in the production of arthritis. Benign prostatic hypertrophy and involution of the testes similarly have no known etiologic relationship to arthritis.

Hench reported the **ameliorating effect of pregnancy** upon chronic arthritis, fibrositis and intermittent hydrarthrosis.[35] He studied the effects of 37 pregnancies in 22 women with joint manifestations and concluded that *the physiologic state of pregnancy is markedly antagonistic to the continuance of the symptoms of rheumatoid arthritis.* He surveyed the literature on this subject and discussed various influences of catamenia, menopause, pregnancy, labor, the puerperal state and lactation in the development and exacerbation of articular disease.

Few individuals have commented upon the relief of joint phenomena during pregnancy. Several have noted an amelioration of a recurrent hydrarthrosis during pregnancy. Hench quoted Garrod's comment in 1890: "It is curious to observe that the occurrence of pregnancy during the course of the disease (rheumatoid arthritis, including at that time, osteo-arthritis) appears to exert opposite influences in different cases, in some cases accelerating the progress of the malady, in others acting as a temporary check upon its development." Pemberton also noted that pregnancy may be accompanied by temporary improvement or complete amelioration of symptoms in chronic arthritis.

In 20 of 22 women there was striking and generally complete relief of arthritic phenomena during and for some time after pregnancy. In these patients, when relief was obtained during a pregnancy, all subsequent pregnancies also produced a good effect upon the arthritic phenomena. *In most of the cases, relief from pains was noticed between the fourth and sixth weeks of pregnancy.* There was almost complete relief of pain and stiffness and marked reduction of joint swelling. *The period of relief from joint phenomena was approximately nine months;* symptoms did not recur in any case before parturition. In most instances, the arthritic symptoms recurred some weeks following delivery as severely (or more so) as prior to the pregnancy.

The cause of the relief produced by pregnancy in some cases of arthritis is speculative. Hench believed that neither bilirubin nor a strictly female sex hormone was responsible. These remissions are not unlike those found in patients following the development of a toxic jaundice.

Female patients with rheumatoid arthritis generally do not have an improvement in their arthritis following the use of estrogens, progestins

and chorionic gonadotropin, given singly and together. One of the most profound chemical changes in pregnancy is the marked increase in plasma lipids, which also occurs in obstructive jaundice.

The lipemia of pregnancy is due almost entirely to the increased plasma lipids. The plasma neutral fat increases in the first trimester and cholesterol and phospholipid increase in the second trimester. At term, these latter two are each increased approximately 25 per cent while neutral fat is increased more than 100 per cent over their figures in the non-pregnant woman.

Freyberg and others have shown that total serum lipids and its fractions are normal in patients with rheumatoid arthritis; he suggested that the very high levels of total lipids, phospholipids and free and total cholesterol found in obstructive jaundice may be a factor in the beneficial effect of obstructive jaundice on rheumatoid arthritis. The increase in total lipid during pregnancy is minimal when compared to the marked lipid disturbance in obstructive jaundice (in which the total serum lipids may be increased 300 per cent). Bayles and Riddell found that the lipemia of pregnancy in patients with active rheumatoid arthritis was qualitatively and quantitatively similar to that of normal pregnant women.[8]

The total cholesterol rise in pregnancy is only about one-third of that found in obstructive jaundice, with no change in the free/ester ratio; this is in marked contrast to the lipemia of jaundice, in which the increase in cholesterol is due almost entirely to the marked increase in free cholesterol (the esters remaining unchanged or below normal). The total lipid rise in pregnancy is approximately one-third that found in obstructive jaundice while the phospholipids rise only a fraction as much in pregnancy as in jaundice. Bayles suggested that it is possible that there may occur an intimate disturbance in lipid metabolism in obstructive jaundice and in pregnancy which is not mirrored by the present measurements of plasma lipids.

PANCREAS

Walker reported upon the value of pancreatin therapy in the treatment of "arthritis deformans."[100] Wolf stated that more than 10 per cent of all diabetic patients had arthritis.[104] He thought that the high blood cholesterol, acidosis and arteriosclerosis were potent factors in interfering with the nutrition of the joints, which, at best, is not very abundant; with the additional handicap of a decreasing vascular supply in the diabetic patient, the door was left open for inflammatory and degenerative changes. He found that as the diabetes was brought under control, there was marked improvement in the joint condition.

Subdeltoid bursitis is apparently much more *common among diabetic patients* than in normal individuals or persons with other chronic disorders.

SUPRARENAL GLANDS

In extremely weak and emaciated patients it has been suggested that adrenal cortical extract or desoxycorticosterone acetate may be of some

value. Watson reported clinical improvement in patients with "chronic non-specific arthritis" following weekly intramuscular injections of from 3 to 5 cc. of cortin; similar results were not obtained with desoxycorticosterone acetate.[101] No specific benefit has been seen in the arthritic manifestations following its use.

Under certain experimental conditions, overdosage with desoxycorticosterone acetate elicits a polyarthritis resembling histologically that seen in acute rheumatic fever.[77] These joint lesions were produced more readily by desoxycorticosterone acetate in adrenalectomized or thyroidectomized than in intact rats, especially if they were exposed to cold. Such joint lesions were often accompanied by the appearance of Aschoff nodules in the heart, and at times also by periarteritis nodosa. The marked similarity between these experimentally produced lesions and the manifestations of rheumatic fever were interpreted as indicating that the adrenal cortex may play an important rôle in the pathogenesis of rheumatic fever, and possibly of rheumatoid arthritis in man.

Selye and his associates showed that arthritic lesions rarely developed in the intact rat treated only with desoxycorticosterone acetate, but that such lesions developed with great frequency in similarly treated animals if the thyroid or adrenals were removed, and especially so if animals were kept in cold surroundings. Unilateral nephrectomy and sodium chloride treatment increased the sensitivity of rats to many overdosage effects of desoxycorticosterone.

Although the causative agent in these experiments seemed to be the desoxycorticosterone acetate, various factors were effective as predisposing agents in the production of the joint lesions, namely, exposure to cold, thyroidectomy or adrenalectomy—all of which greatly facilitated the production of the arthritic lesions.

Selye reviewed the literature on the relation of endocrine disturbances to arthritis.

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CHAPTER XXVIII

EFFECT OF JAUNDICE ON ARTHRITIS

THERE are numerous fascinating unsolved problems in rheumatoid arthritis. Among these, one of the foremost is the question as to why patients with rheumatoid arthritis, fibrositis and some other rheumatic disorders improve so markedly when jaundice unexpectedly supervenes. A large percentage of *patients with rheumatoid arthritis* will *note a marked lessening of the joint phenomena should* an intrahepatic or obstructive *jaundice intervene.* In some of these individuals, complete remissions may occur. Unfortunately, the rheumatic manifestations usually reappear when the jaundice has cleared. The remissions may be sudden and complete and pain may be ameliorated within a few days after visible jaundice has supervened.

This is of particular interest when it is remembered that the free and total cholesterol are greatly increased in obstructive jaundice, and that a similar rise in cholesterol is seen in pregnancy. *Pregnancy also may induce a remission in patients with rheumatoid arthritis.* Thus, we have two conditions, pregnancy and jaundice, both of which may produce more marked (temporary) improvement in patients with rheumatoid arthritis than any known therapy available at present. It is of interest in this connection to mention the close relationship between cholesterol and vitamin D, inasmuch as some workers have reported beneficial results in rheumatoid arthritis with massive doses of this vitamin (although equally capable workers could not confirm such improvement). *Unfortunately, in most instances, the improvement induced by the jaundice or the pregnancy disappears following delivery or clearing of the jaundice;* however, a further study of the changes induced in pregnancy and in jaundice may prove to be the scientific springboard from which the full scale therapeutic invasion of rheumatoid arthritis is undertaken. Unfortunately, repeated pregnancies are inadvisable in most of these individuals, and our present methods of inducing jaundice therapeutically have not been such that treatment by this means is yet feasible.

The development of jaundice may produce a remission of joint symptoms in some arthritic patients.[2,4,8–13,17,19] Hench[8] observed 45 patients who experienced this phenomenon; of these, 28 had rheumatoid arthritis; 12, fibrositis; 1, degenerative hip joint disease and 4, lumbosacral or sciatic pain. All were partially or completely relieved of joint phenomena coincident with the onset of jaundice. Jaundice of an intrahepatic or of an obstructive nature were equally effective. In his series, jaundice was caused by cinchophen, catarrhal jaundice, hepatitis, cirrhosis, gall stones and malignant growth. The effect of hemolytic jaundice was not observed. When the serum bilirubin reached a concentration of 8 to 10 mg. per

100 cc., arthritic symptoms were relieved, suggesting that the effect was more a quantitative than a qualitative one.

In general, *such remissions were sudden in appearance, often complete,* and *bore a relationship to the intensity and duration of the jaundice.* Amelioration of joint pain began within the first three days of visible jaundice. Over two-thirds of the patients noted complete relief of their arthritic symptoms. These remissions averaged several months (varying from three weeks to more than three years). *In most of the patients, the arthritic symptoms returned to their former intensity several months after the clearing of the jaundice.*

This observation again demonstrates that *rheumatoid arthritis is not a hopeless, uncontrollable disease.* The fact that the symptoms and signs of arthritis cleared during an attack of jaundice should add rays of hope to those individuals whose arthritis has not responded to previous therapy.

Hench was not successful in reproducing a harmless form of jaundice by numerous procedures, including whole bile and its constituents by various routes; decholin; bile by stomach tube or transfusions of deeply jaundiced blood.[8] Other workers attempted to induce jaundice experimentally in animals and in human beings with rheumatoid arthritis.[21–24] The administration of bile salt or of bilirubin produced no beneficial effect in these patients. However, a combination of bilirubin and bile salt had an ameliorating effect on the symptoms of rheumatoid arthritis. These workers were able to produce artificial jaundice which apparently duplicated the beneficial effect of spontaneous clinical jaundice in these patients.

The material used to produce artificial jaundice was prepared as follows: Tenth normal sodium bicarbonate was brought to the point of boiling *but not allowed to boil;* sufficient was used for solution of the bilirubin, to be added later (usually 20 to 30 cc. per 0.1 gm.). Bilirubin was then added, gently shaken, and allowed to stand for an hour. This was next passed through a Seitz filter. To the filtrate was added a sterile solution of bile salt (sodium decholin). The final solution was sterile and was protected from the light. The solution was given intravenously.

Each dose injected contained approximately 10 mg. of bilirubin per kg. and 40 mg. of bile salts per kg. From 7 to 11 daily intravenous injections were given per patient. Icterus was usually observed (first in the eyes) after the first to the fourth injection; this became more marked with subsequent doses, disappearing within two to three weeks after the last administration. In some of these patients, pain disappeared and did not recur for periods of at least five months or more.

In 6 of the 10 patients so treated, reactions occurred; local reaction was noted if the same vein was used for repeated injections. General reactions occurred immediately or within one or two hours after the injections and consisted of chills, nausea, vomiting, headache and a fever from 99° to 101° F. None of the reactions were prolonged or dangerous. Several cc. of aromatic spirits of ammonia promptly relieved the immediate general reaction. No renal or hepatic damage was found following such injections.

Freyberg and his associates found that *in* patients with *arthritis, the*

ratio of ester cholesterol to total cholesterol was normal.[1] Normal values for total serum lipids were obtained in patients with arthritis; the level of serum phospholipids was slightly elevated. *The total, free and ester serum cholesterol was nearly the same in arthritics as in normal control subjects.*

In patients with obstructive jaundice, the total serum lipids and phospholipids were very high, and the levels of total and free cholesterol were greatly increased over normal levels while the percentage of cholesterol combined in the ester form was far below that found in normal subjects.

Serum lipids were not below normal in arthritic patients. Thus it appears that *jaundice is not beneficial in rheumatoid arthritis by reason of its correcting a lipid deficiency.* It is possible, however, that the very high levels of total lipids, phospholipids and free and total cholesterol which are found in obstructive jaundice may be a factor in producing the beneficial effect of such jaundice on these rheumatoid arthritic patients.

Hench thought that bilirubin was the most likely agent responsible for the beneficial effect of jaundice on arthritis. This is of interest when coupled with Race's findings of low icterus index and serum bilirubin concentration in patients with rheumatoid arthritis.[18] Hench pointed out that other possible factors in this phenomenon included bile salts, special diet or hepatic autolysate.

Various methods have been utilized in an attempt to produce therapeutic jaundice in these patients. These included the administration of transfusions from highly jaundiced persons; bilirubin; bile salts; synthetic bile salts; human bile; diluted ox bile by stomach tube; liver extracts and toluylenediamine.[15,16,26]

Most investigations of the beneficial rôle of obstructive jaundice in rheumatoid arthritis centered about bilirubin, bile salts or bile acids. Freyberg's work was done to make a comparative study of the lipid partition of serum on patients with obstructive jaundice, on patients with rheumatoid arthritis and on normal subjects to establish the presence of any abnormality in serum lipid values of arthritic patients; to compare the serum lipids in patients with arthritis to those in persons with obstructive jaundice and to determine whether changes in serum lipids associated with obstructive jaundice could be the basis for the beneficial effect of jaundice on rheumatoid arthritis.

Snow and Hines reported the effect of obstructive jaundice on the polyarthritis of rats.[20]

Kauftheil reported high cholesterol values during the severe inflammatory stage of rheumatoid arthritis with normal values when the inflammation had subsided.[14] Bruger and his associates found a decrease in total cholesterol with a normal relationship between free and ester fractions[3,6,7] while others reported normal values for total cholesterol in arthritic patients.[5] Freyberg noted similar values for free, ester and total cholesterol in the normal control and the arthritic group, with nearly identical percentage of esterified cholesterol in the two groups. The total values for serum lipids were very high in 4 of 5 patients with jaundice; the serum phospholipids were increased to two and three times normal in the jaundiced individuals. Others[25] noted similar increases in phospholipids in most patients with obstructive jaundice.

Patients with arthritis showed no definite abnormalities in the total serum lipids or their fractions.

Advisability of Attempting to Produce Artificial Jaundice in an Effort to Cure Patients With Arthritis.—To date, jaundice is not a cure for rheumatoid arthritis, but merely one of nature's methods of temporarily ameliorating the condition. In attempting to produce artificial jaundice as outlined previously, one must remember that the preparation of the material is not simple and requires approximately two hours (the material being prepared fresh each day before injection); because of the alkalinity of the solution, thrombosis of veins readily follows. It is not advised for the general practitioner.

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CHAPTER XXIX

FEVER THERAPY

FEVER therapy (with high fever) is seldom used in the treatment of rheumatic diseases at present, except in Brucellosis and occasionally in gonorrheal arthritis and chorea. The introduction of sulfonamides and penicillin has greatly lessened the need for high fever therapy.

Although short physical fevers (thirty minutes)—raising the body temperature to 101° F. by a fever cabinet, hot tub or blanket pack may be of value in *rheumatoid arthritis* (except in greatly weakened individuals), high fevers are no longer advocated for these patients. Warm baths are of benefit in *degenerative joint disease* and heat and massage form the basis of therapy in *fibrositis.*

The results of fever therapy in arthritis may be summarized briefly as follows:

In gonorrheal arthritis—excellent results in the acute cases (of six weeks duration or less) and good results in chronic cases.

In rheumatoid arthritis—poor results, not more than 10 per cent becoming symptom free.

In degenerative joint disease and gout, almost negative results except in an occasional case.

In chorea—results are encouraging.

Tayloe has reviewed briefly the current literature on fever therapy. He concluded that fever therapy in combination with chemotherapy offered our best therapeutic weapon in the treatment of gonococcic infections and neurosyphilis, especially dementia paralytica. The introduction of penicillin will probably cause a revision in this, inasmuch as penicillin has been extremely successful in the treatment of gonococcic infections and also in neurosyphilis. Tayloe also found fever therapy to be a valuable therapeutic adjunct in the treatment of chorea, brucellosis and infectious arthritis.[38]

Heat has been used in the treatment of disease for many centuries. It has been suggested that fever serves some useful purpose or nature would not have retained it as a reaction to the invasion of harmful substances.[42] The last of the Hippocratic aphorisms suggested that heat (or fire) may cure disease: "Those diseases which medicines do not cure, iron (the knife) cures; those which iron cannot cure, fire cures; and those which fire cannot cure are to be reckoned wholly incurable."[10] The ancient Chinese, Romans and Greeks used heat extensively in the treatment of human disease.

Present day fever therapy is traceable largely to the work of Wagner-Jauregg, who found that fever produced by malaria often resulted in improvement or complete remission in dementia paralytica. Since that time, various methods of fever therapy have been made available, including

injections of foreign protein (typhoid vaccine intravenously, Coley's fluid intramuscularly, boiled milk—aolan, peptone, etc.), and physical methods such as air conditioned chambers, diathermy or short wave diathermy, hot baths. radiant heat cabinets, electric blankets, hot spray baths, hot tub baths, etc. Since a preliminary report in 1929 on the production of fever by high frequency currents, this method has been extended to the treatment of various chronic diseases.[25]

Typhoid vaccine, hot tub baths or hot packs are probably the best available form of fever therapy for the general practitioner.

Fever Therapy With Typhoid Vaccine

If typhoid vaccine is used to produce fever therapy, the technic is as follows:

1. Give from 15 to 20 gm. of sodium chloride daily for two days before therapy is begun.
2. On the day of fever therapy, patient should take a very light breakfast.
3. The *patient* is kept *completely covered in bed (lying between woolen blankets)* except for the head and neck.
4. Four *hot water bottles* (140° to 150° F.) are placed in the bed, and are replaced every thirty minutes during the treatment; *do not uncover the patient when they are changed.*[28]
5. In nervous or apprehensive patients, one may give sedation such as seconal (0.1 to 0.2 gm. by mouth) or a similar dose of nembutal. In a very restless individual, small amounts of morphine may be given (10 to 15 mg.) hypodermically (varying the dose with the age and size of the patient).
6. After the patient has been in the warm bed for an hour, give *intravenously* 50 million organisms of a *triple typhoid vaccine.*
7. Temperature and pulse should be recorded every fifteen minutes and blood pressure must be taken every thirty minutes.
8. If the temperature has not risen to 102° F. within two and a half hours, give an additional 50 or 100 million organisms intravenously. If no additional febrile response is obtained in an hour and a half, a third dose of typhoid vaccine (100 million organisms) may be given intravenously. If one is aiming to obtain a temperature of from 104° to 105.5° F., this can usually be attained by this method. When the desired peak temperature has been reached, this can usually be sustained if the hot water bottles are changed regularly without uncovering the patient.
9. If required, morphine sulfate may be given every three or four hours in small doses.
10. Liquids (at room temperature) may be given as desired. The fluid intake should be at least 5000 cc. (for adults) on the day of treatment. The individual is encouraged to drink as much 1 per cent saline solution or orange juice containing 1.5 per cent saline as possible to prevent serious salt and water depletion.
11. If the temperature rises too high, the hot water bottles are removed. If it is necessary to lower temperature quickly, cold drinks are encouraged, the patient is given aspirin by mouth (0.6 gm.—gr. x or more) and a tepid or cool sponge bath is given.
12. Care must be taken that aspirin or other salicylates are not being taken by the patient within twelve hours of the period of fever therapy.

Fever Therapy With Typhoid Vaccine—*(Continued)*

13. An ice bag may be applied to the patient's head if a severe headache occurs.
14. *In subsequent bouts of fever therapy the dosage of typhoid vaccine must usually be increased* in order to produce a similar febrile bout. This increase may be twice the original dose, or considerably more. No definite rule may be given concerning this, but sufficient vaccine must be given on each occasion to produce the desired result.
15. In those individuals who have received immunizing injections of typhoid vaccine recently, it is very difficult to produce high fevers by this method of therapy.
16. The physician should select one brand of triple typhoid vaccine and adhere to this so that he may anticipate the result which is to be expected; different brands of typhoid vaccine vary in potency. If *typhoid H antigen* is used, the dose of antigen required is usually greater and one does not usually obtain as high a temperature with the H antigen, although the chill and some of the other objectionable features of typhoid vaccine may be avoided.

Physical Methods for the Production of Artificial Fever: The Kettering hypertherm, an air conditioned chamber, is one of the instruments of choice (if available) among the physical methods for the production of artificial fever.[19,24] The apparatus requires the constant attendance of a specially trained technician.[18]

It consists of a cabinet large enough to accommodate the body of the patient, the head being kept outside of the machine. Fever is produced by heated and humidified air which is passed over the patient's body. The air is heated by an electrical resistance coil heating element at the foot of the cabinet and is circulated through the cabinet by a blower; by means of a second heating element, humidity is produced by boiling water.

There are a number of fever machines on the market which are equally acceptable. The Liebel-Flarsheim Co., of Cincinnati, Ohio, has been granted permission to construct a cabinet similar to the Kettering hypertherm.

Neymann is enthusiastic about electromagnetic induction; however, it really matters little which technic is used, provided the machine is a good one, and the personnel and general equipment of the department are well developed.[17,26]

Fever therapy is usually given in the morning following a very light, or no breakfast. The day previous to the treatment as much fluid, food and salt as tolerated should be taken. Krusen recommends a hot tub bath and ultra-violet therapy the day before treatment; he is convinced of the need of a preliminary conditioning treatment (at which time the body temperature is raised to 102° F. and maintained at this level for two hours) in order to prepare the patient for a prolonged fever on the following day. The air temperature of the cabinet is gradually raised from 125° to approximately 160° F. and the relative humidity is maintained at from 50 to 80 per cent. It requires from thirty minutes to several hours

to reach the desired body temperature. During the febrile period, an electric fan plays on the patient's face, and cold 0.6 per cent saline is drunk freely (because of the large amount of fluid and sodium chloride lost in the sweat). The intermittent administration of oxygen and carbon dioxide during prolonged sessions of fever combats anoxia. Following the termination of the treatment, the patient's temperature falls to normal within about an hour.

Some of the complications which occur from such therapy include loss of weight, nausea and vomiting (which is frequent), burns, tetany, delirium, dehydration, anoxia, heat exhaustion, herpes labialis and on rare occasions, sudden uncontrolled hyperpyrexia with mania followed by coma, collapse, and in a few cases, death.[36] This form of treatment is usually well tolerated. It has been reported that during the several hours while their temperature is about 106° F., some of these patients may "hum, sing, smoke, or amuse themselves by teasing the technician." A few beg to be removed from the chamber before the completion of the treatment.

The duration and height of the temperature is varied with different diseases. In gonorrheal arthritis, a rectal temperature of between 106° and 107° F. is usually maintained for from five to ten hours; if the five-hour sessions are used, three or four such sessions may be required for cure; if the nine or ten-hour period is tolerated, one session may suffice. It has been stated that the thermal death time of more than 100 strains of Neisseria gonorrhœæ varies from six to twenty-seven hours at a temperature of 106.7° F. (41.5° C.). For rheumatoid arthritis, milder treatments are usually given, such as five or six hours at a rectal temperature of from 104° to 105° F.; even this may be too long, and may deplete the patient.

In acute gonorrheal arthritis, 80 per cent of patients have been rendered symptom free by fever therapy and an additional 10 per cent have been markedly relieved;[4,21,23] in chronic gonorrheal arthritis, cures have been obtained in about 40 per cent of the patients with an additional 30 per cent being markedly relieved. In some cases in which sulfathiazole fails, the combination of the latter drug and fever may be more effective than either, alone.[6] **The brilliant results of penicillin in gonorrheal arthritis will make fever therapy unneccessary in these patients.** A more complete discussion of this subject appears in the sections on gonorrheal arthritis and on sulfonamides and penicillin.

Fever therapy is at best merely an adjunct in the management of rheumatoid arthritis.[33] It is generally accepted by most rheumatologists that streptococci may be in part responsible for rheumatoid arthritis; the amount of fever available is not sufficient to kill these organisms in rheumatoid arthritis.[8,9] Not more than 10 per cent of cases of acute rheumatoid arthritis have been permanently benefited by fever therapy; an additional 40 per cent were markedly relieved.[29] When "cures" are reported from fever therapy in rheumatoid arthritis, there is some doubt cast upon the correctness of the diagnosis.[34,35] Krusen has pointed out the need for greater consideration of short (thirty minute) physical fevers used in conjunction with other methods of treatment (such as massage, exercise and medical

care). These will raise the body temperature to approximately 101° F. A fever cabinet, hot tub bath or a blanket pack may be used to induce the fever. Although *most clinics have abandoned the use of prolonged high fevers in rheumatoid arthritis*, short physical fevers (thirty minutes) may be of value if given daily to all but very weak patients. In chronic stages, the percentage of patients relieved is less.

Less than 5 per cent of patients with degenerative joint disease have been rendered symptom free by fever therapy, while from none to over 50 per cent showed some degree of improvement.[2,7,16] Patients with this form of arthritis are poor subjects for fever therapy.[31,39]

Several isolated reports of good results with fever therapy in gout, traumatic arthritis and neuritis have been recorded.[13] Fever therapy has also induced remissions in Brucellosis[11] and is one of the treatments of choice in this disease.

It has been claimed that fever therapy produces symptomatic improvement in some cases of *acute rheumatic fever;* however, the number of patients so treated is too small to draw definite conclusions. In one series, 15 cases of acute rheumatic fever were treated with fever therapy (from eight and one-half to forty-six hours with temperatures of from 103° to 106° F.); 13 of these obtained complete relief from joint pain and swelling. Only 3 of these patients had recurrences. This is in general agreement with findings of other workers both with this disease and with Sydenham's chorea.[1,30,37]

Contraindications to Fever Therapy[10]

Cardiovascular-renal disease.
Old age (sixty or more years).
Active tuberculosis.
Abnormalities of the skin interfering with sweating and abnormal heat sensitivity of the skin.
Coronary artery heart disease.
Rapidly progressive late neurosyphilis.
Marked hypotension.
Aortic aneurysm.
Liver damage.
Fresh endocardial lesions (danger of embolism).

Cause of Beneficial Effect of Fever Therapy: Direct killing or static effect on bacteria, due to the heat alone.

Increased formation or mobilization of antibodies.

Vasodilatation.

Increased general metabolism from fever.

Amount of Fluid and Chloride Lost During a Session of Fever Therapy.—During a five-hour session, an individual may lose 3 or 4 liters of fluid in the perspiration and from 18 to 26 gm. of chlorides.[32] This loss of chlorides may account for most of the unpleasant symptoms occurring during fever therapy (as nausea, vomiting, exhaustion, abdominal cramps, etc.). Such

symptoms can be avoided usually by the use of cold saline solution (0.6 per cent) in liberal quantities by mouth (2 to 4 liters) during the treatment. Patients fail to absorb fluid from the gastrointestinal tract during the latter part of long sessions of fever. In such cases, physiologic saline may be given intravenously. If the systolic blood pressure falls below 90 mm. of mercury, administer 500–1000 cc. of saline intravenously. This is a very important safety measure.

Physiologic and Chemical Effects of Fever Therapy.—Briefly summarized, these include tachycardia, fall in blood pressure,[3] increased minute volume cardiac output,[41] increase in number and size of capillaries of the nail bed, and no important change in the blood urea, uric acid, creatinine,[15] sugar, calcium, plasma lipoids or serum protein. The erythrocyte count and sedimentation rate show practically no change;[27] leukocytes may be increased to 15,000 per c.mm. after an initial slight fall. Marked decrease in blood chlorides occurs but may be prevented by the oral administration of saline.[20] There is no marked change in opsonic index or complement (except possibly a slight fall in the latter).[14] The metabolic rate is increased approximately 7 per cent for each degree of fever. The electrocardiogram shows minor changes, none of which are permanent; no inference can be made from these that cardiac damage has resulted from the fever therapy.[40] There is, however, a marked drop in the ventricular filling time and a disappearance of cardiac diastasis.[22] The bacteriolytic effect has been discussed previously.

Use of Malarial Therapy in the Treatment of Rheumatoid Arthritis.—Although temporary improvement may result following malarial therapy, this is usually not maintained.[5] The production of fever by malaria has no place in the present day management of arthritis.

Autohemotherapy.—Ishmael used autohemotherapy immediately followed by artificial fever therapy in rheumatic diseases.[11,12] The purpose of the autohemotherapy was desensitization; the effects of this form of treatment were found to be greatly enhanced by the use of artificial fever. The technic was simple, consisting of the withdrawal of 10 cc. of blood from a vein and injecting this immediately deep into the gluteal muscles. This was followed at once by one or more hours of artificial fever at 101.5° produced by a short wave diathermy apparatus. An hour prior to the treatment, the patient was given the normal meal; about fifteen minutes before treatment was begun, 1 gram of sodium chloride with water was taken by mouth. The fever rooms were air-conditioned to a temperature of 79° F. and a relative humidity of 45 per cent.

In a study of over 300 patients with rheumatic disease treated by this technic, the following results were noted: Best results were obtained in patients with acute phenomena. *All patients with acute fibrositis experienced complete relief from symptoms following this treatment.* Good results were also obtained in acute infectious arthritis, gonorrheal arthritis and in rheumatoid arthritis in children (Still's disease); in the latter group, results were better when a temperature of 104° F. was maintained for four hours. Women, with rheumatic pains recurring several days prior to the menstrual

periods, were also greatly benefited. Little or no consistent benefit was found in chronic rheumatoid arthritis in adults. In "menopausal arthritis," the intramuscular injection of blood was fortified with large amounts of estrogenic substance; the addition of the latter to the autohemotherapy and fever seemed to be of distinct benefit.

In his hands, the use of autohemotherapy, and artificial fever was safe and could be carried out simply. Ishmael emphasized that this form of therapy was merely an adjunct to the routine care of arthritic patients, with common sense, careful supportive measures and other symptomatic treatment when needed. He also employed a combination of autohemotherapy, artificial fever and sulfathiazole in rheumatoid arthritis in children (Still's disease) in the following manner: 10 cc. of the patient's blood given intramuscularly, followed immediately by 104° F. of artificial fever for four hours and then sulfathiazole (varying in amount with the weight and age of the patient) at four-hour intervals day and night for three or four days. A small transfusion was then given to the patient, followed shortly by another four hours of fever therapy at 104°; sulfathiazole was discontinued about twenty-four hours after this second bout of fever therapy. This whole procedure was repeated at weekly intervals (as tolerated) for an average of three or four times. Ishmael's results with this form of therapy in rheumatoid arthritis in children were very encouraging.

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CHAPTER XXX

IONTOPHORESIS (WITH MECHOLYL OR HISTAMINE)

Most rheumatologists are not impressed with the results from iontophoresis with histamine or choline preparations in the treatment of rheumatoid arthritis.[13] In our experience, iontophoresis with mecholyl or histamine results in temporary subjective improvement similar to that following the use of hot fomentations to the joint. The psychic effect of iontophoresis is great in some instances.

Present evidence indicates that acetyl-beta-methylcholine chloride (mecholyl) exerts a systemic as well as a local effect when given by iontophoresis (ion transfer). One may expect a palliative symptomatic effect in many patients with rheumatoid arthritis following the use of mecholyl iontophoresis.[8]

The Council on Pharmacy and Chemistry of the A.M.A. has warned that:

1. Mecholyl should not be given intravenously, because of the possibility of cardiac arrest.
2. *Contraindications to the use of mecholyl include:* asthma, hyperthyroidism and any severe illness.
3. If given by injection, the area should not be massaged except when it may be necessary to determine whether a further injection is needed (especially in paroxysmal tachycardia).
4. Recumbency is advised during subcutaneous administration to avoid fainting.
5. *Iontophoresis with mecholyl should be undertaken only by those especially trained* and should not be applied over ulcers or open wounds, and with extreme caution over scar tissue.
6. *It is inadvisable to continue prolonged therapy with mecholyl by any route.*

Iontophoresis is the introduction of a drug into the deeper layers of the skin by means of a low density galvanic electric current. Opinions vary concerning its usefulness in chronic arthritis.[9,14,22,30] With the introduction of vasodilating drugs (such as mecholyl and histamine) by means of iontophoresis (also called ionization), favorable clinical results have been reported in rheumatoid arthritis, as well as in other conditions such as Raynaud's disease, varicose ulcers, etc.[5,10,18] Improvement was noted in 95 per cent of stubborn cases of rheumatoid arthritis by one author who has used this method.[16] In order that the reader may obtain an unbiased opinion concerning iontophoresis, other summarized reports concerning the effectiveness of iontophoresis of mecholyl or histamine are as follows:[9,14,22]

Fifty-nine per cent of patients with rheumatoid arthritis were helped, some becoming symptom free.[21]

Iontophoresis by choline compounds represents a valuable addition to the therapeutic armamentarium for the local treatment of the rheumatoid type of chronic arthritis.[17]

Ninety per cent relief was found in patients with rheumatoid arthritis by means of mecholyl iontophoresis.[1]

The striking therapeutic effect of mecholyl iontophoresis was the decreased fatigability and the increased endurance.[3,4]

This method would seem to have a very limited field of application. If lasting hyperemia is desired, there would seem to be better and less complicated ways of producing it.[20]

In 103 treatments with mecholyl iontophoresis given to 20 patients with rheumatoid arthritis, only 2 were improved and the treatment was abandoned as useless.[28]

Ionization with vasodilating drugs is valuable in reducing swelling and pain in small joints which do not respond to mild thermal treatment.[19]

Such treatments have been reported to be useful in early cases of rheumatoid arthritis with damp, cool, cyanotic extremities; patients with degenerative joint changes, paresthesias and sensitivity to cold; and sciatica or spinal nerve root irritation from arthritis of the spine. Elderly patients or cases of far advanced rheumatoid arthritis with marked bony changes and fibrosis showed poor results.

Two of the vasodilating drugs which have been used for this purpose are histamine and mecholyl (beta methyl acetyl choline chloride).[27] By means of a few milliamperes of galvanic current passing through a solution of either of these substances (which have been placed on asbestos paper surrounding the affected joint), the drugs may be made to pass from the solution into the deeper layers of the skin of the parts treated.

We have abandoned mecholyl or histamine iontophoresis in rheumatoid arthritis inasmuch as much simpler methods (such as hot fomentations) appear to give equally good subjective results. However, because of the favorable results reported by some authors, the technic for iontophoresis is included so that it may be available for ready reference to those physicians who may wish to employ it.[2,24,29]

EQUIPMENT NECESSARY FOR IONTOPHORESIS

A galvanic machine equipped to deliver at least 45 volts and having milliamperemeter reading up to 50.

Active electrodes of soft malleable block tin; an indifferent electrode (or moist dispersive pad).

Two insulated wire cables.

Asbestos paper.

Elastic bandage.

Solution of mecholyl or histamine

Galvanic Machine.—This can be procured from most surgical supply houses. A portable apparatus should be used. Our apparatus was obtained from the Radio Department of the M. and H. Sporting Goods Store,

512 Market Street, Philadelphia. This is priced reasonably (approximately $18–$20). Other makers of machines for this purpose include the Valverde Laboratories, 252 Lafayette Street, New York, and William Balardo, 3102 Kingsbridge Avenue, Bronx, New York.

Electrodes.—The large moist dispersive pad (indifferent electrode) which is placed over the back can be made as follows:

The strands of the terminal 4 inches of one of the wire cables are woven into copper wire screening.

This piece of fine copper screening approximately 8 x 12 inches is then wrapped with at least 3 or 4 layers of gauze bandage, covering all of the copper.

The 4 edges are bound with heavy dressmakers' tape ½ to ¾ inch wide so that no fine points of copper can project therefrom.

The wrapped copper screening is enclosed in a cover of heavy canvas; the latter must be at least 1½ inches wider and longer than the screening to allow for shrinkage when wet. The edges of the canvas are sewn together.

The electrode is ready for use, the copper screening being completely covered with gauze, its edges bound with tape, and the whole apparatus enclosed in heavy canvas. The remainder of the wire cable projects from the electrode.

The active electrode is heavy metal foil which may be cut into the size desired. The most useful form is approximately 12 inches long and 2 to 3 inches wide. All corners should be rounded. This is the regular diathermy soft malleable block tin which may be purchased as such.

Insulated Wire Cables.—These form part of the apparatus.

Asbestos Paper.—Large rolls of asbestos paper 15 inches wide and 5 yards long may be procured from Merck & Co. It is called Merck's *reinforced asbestos paper*.

Elastic Bandage.—An ordinary elastic bandage 2½ or 3 inches wide will suffice.

Mecholyl Solution.—One gram of mecholyl is dissolved in 500 cc. of distilled water. The latter is used in preference to tap water because of the presence of ionizable salts in tap water. Such solutions are stable for a period of two or three weeks. The 1 gram vial of mecholyl is the most convenient size for ordinary use; 1 gram dissolved in 500 cc. of distilled water makes a 1:500 solution of the drug. As the drug is hygroscopic, the entire contents should be placed into solution at once when the vial has been opened.

TECHNIC OF IONTOPHORESIS TREATMENT

The patient strips in a warm room and lies on the bed; the latter is covered with rubber sheeting or oilcloth.

The large indifferent electrode (moist dispersive pad) is soaked in warm water, and the excess water is allowed to drain off. The pad is placed on the bed on top of a similar sized rubber sponge knee pad (which can be procured in a 5 and 10 cent store). Before the patient lies upon the pad,

one must be sure that no fine pieces of copper wire stick through the canvas (to prevent burns of the patient). The patient lies so that his back makes good contact with the moist dispersive pad without wrinkling.

The area to be treated (such as a knee) is washed with soap and water to remove grease and is rinsed with distilled water. A strip of the asbestos paper approximately 18 to 24 inches long (and 15 inches wide) is soaked in the mecholyl solution, which has been warmed slightly. This is wrapped around the part to be treated so that all skin areas are covered. On top of this is placed in spiral fashion a double layer of asbestos paper approximately 3 inches wide and 1 foot long, saturated with the mecholyl solution. Directly on top of the spirally wrapped asbestos paper in a similar spiral fashion, is placed the metal active electrode; this should be somewhat smaller in all dimensions than the asbestos. It is held in position by an elastic bandage which has been soaked in the mecholyl solution. *No part of the active metal electrode must come into contact with the skin or a burn will result.*

The cable from the positive pole is connected to the active electrode (at the joint to be treated). *The current in the apparatus must be entirely shut off*. The cable from the negative pole is then connected to the indifferent electrode on the back.

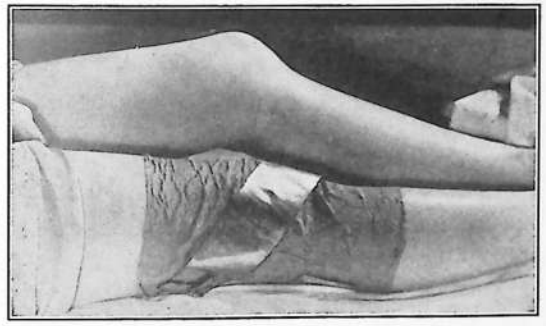

FIG. 115.—Iontophoresis with vasodilating drugs. Asbestos fabric paper saturated with solution of the drug is wrapped round the part and a smaller sized metal plate placed over it, being careful not to touch bare skin. (Kovács.)

The current is turned on very slowly and the patient notices at once a tingling sensation in the part treated and in the back. The current is increased very slowly (especially during the first several treatments), requiring at least five minutes to reach 25 to 30 milliamperes. It is advisable to permit the patient to remain quiet and warm for a period of from fifteen to thirty minutes following the treatment. If tolerated, this amount of current (25–30 milliamperes) may be continued from twenty to thirty minutes, after which the current is decreased slowly. Some workers have used as much as 40 to 50 milliamperes for twenty minutes, after a small initial treatment. The electrodes are removed when the current has been turned off.

If any persistent sensation of heat or burning is noticed by the patient during treatment, the current is too strong or the electrodes are improperly placed.

The patient must be covered from drafts during the treatment.

The resulting erythema following treatment persists for several hours.

Treatments may be given two or three times per week unless contraindicated; *a history of bronchial asthma, marked hypotension, or serious cardiac disease (other than paroxysmal tachycardia) are contraindications to therapy with mecholyl.*

Both histamine and mecholyl are vasodilator substances. Histamine is formed in the skin as a result of irritation. If histamine is used for this therapy it may be applied in solution as above or as a 1 per cent histamine ointment rubbed into the skin and covered with a moist sheet of asbestos; a current of 15 milliamperes for from three to five minutes may be used with the histamine preparation.

FIG. 116.—Moist dispersive electrode placed under back. Electrodes connected to respective terminal of galvanic source. (Kovács.)

It has been shown that *mecholyl administered by iontophoresis does penetrate the skin and is absorbed into the general circulation.* Montgomery *et al.* have shown that mecholyl iontophoresis in human beings produces a marked increase in blood flow through the hand treated.[23] Comroe and Starr found that mecholyl exhibited effects similar to those obtained by stimulation of the parasympathetic nervous system.[7] *If a patient suffers any ill effects from the administration of this drug, these can be abolished almost instantly by an injection of 0.6 mg. (1/100 grain) of atropine sulfate.*

General reactions were observed consistently by some workers following mecholyl iontophoresis, while others stated that constitutional symptoms were usually very mild and in many instances were not noticed by the patients. These included a drop in blood pressure averaging 12 mm. of mercury, an increase of 22 beats per minute in pulse rate, heaviness in the chest, marked perspiration, increased salivation, flushing of the face, neck and ears and occasionally, lacrimation. These effects came on much more gradually, caused less distress but lasted longer than similar reactions which followed subcutaneous injections of the drug. Following subcutaneous injection of mecholyl, there is a fall in blood pressure, tachycardia, sweating, flushing of the face and neck and salivation; these symptoms appear within a minute or two following injection and may persist for fifteen to twenty minutes.

Local reactions at the site of the positive electrode included an elevation of skin temperature, sweating, increased capillary flow[15] and a pinkness in this area. Boyd and his co-workers felt that the reaction was different and more lasting than that due to local counterirritation.

Mecholyl and histamine have been used *in ointments*[6] *in analgesic base balm.* **Mecholyl ointment** produces dilatation of capillaries and arterioles in the web of the frog's foot with a maintenance or increase of the circulation. **Histamine ointment** results in dilatation of the capillaries, constriction of the arterioles, and retardation of the circulation. Cohen noted symptomatic relief of joint pain in over 85 per cent of arthritic patients (with circulatory disturbances) following the use of ¼ per cent mecholyl ointment in an analgesic balm base, together with heat to the affected part. Heat increases the action of either preparation. Histamine may produce urticaria in allergic patients, and may aggravate attacks of bronchial asthma.

It has been claimed that acetylcholine given hypodermically relieved or prevented the ankylosis and muscular atrophy which normally followed the immobilization of the limbs of experimental animals.[26] However, other workers, repeating this work, found no differences between the control and treated animals.[11]

Increased endurance and decreased fatigability have been reported following mecholyl iontophoresis. It has been suggested that this may be due to the vasodilator effect of the drug. Similarly, striking results in lessening fatigue have been reported following the use of ephedrine and amino-acetic acid.[12]

Physicians interested in the use of mecholyl, histamine or other substances by iontophoresis must watch the performance of the procedure by a competent attendant before using the method themselves; it would be advisable to give the first several treatments in the presence of one trained in the use of this method. By so doing, the physician will avoid burns, electric shocks, etc. One must not merely purchase a machine and attempt to follow the directions as given; some of the finer points of technic cannot be explained adequately in writing and must be observed individually.

Iontophoresis is time consuming and difficult to continue after the patient has left the hospital; it is merely a form of symptomatic therapy.

Muether attempted to evaluate the benefits following administration of histamine.[25] He found that subcutaneous injections of histamine diphosphate (1:1000) were as satisfactory as iontophoresis and better than local applications of ointment containing the drug. Muether treated patients with rheumatoid arthritis without complete joint ankylosis.

Iontophoresis (Ionization) With Mecholyl or Histamine

Iontophoresis is the introduction of a drug into the skin by an electric current. By iontophoresis, active ions may be introduced into the body; by means of an electric current passing through a solution of a salt, the positive ion will pass to the negative pole and the negative ion to the positive pole. It must be observed that the therapeutic ion on its way to the negative pole passes through the pathologic process.[31]

Vasodilating drugs (as *histamine* and *mecholyl*) have been so used in the treatment of joints which are the seat of a chronic rheumatoid arthritis. *This is by no means a cure-all for rheumatoid arthritis* and should be looked upon merely as a means of producing peripheral vascular dilatation.

Choline derivatives stimulate parasympathetic nerves and dilate peripheral vessels.

Such drugs given by iontophoresis penetrate the skin, are absorbed by the blood stream, and produce local and, at times, general effects; with such treatment, the local effects are the ones desired.

The rationale of mecholyl or histamine iontophoresis is its ameliorating effect upon the defective peripheral circulation in chronic arthritis.

Histamine by iontophoresis must be given in a very dilute solution for only a few minutes because of the intense local counterirritant effect obtained; results are probably better in fibrositis than in chronic arthritis.

A less intense local reaction is produced by mecholyl which is used in a more concentrated solution and for a longer period.

Recently, iontophoresis with a newer cholin derivative—doryl (carbominoyl cholin chloride) has been performed using a 1 per cent solution from the positive pole. This may prove superior to mecholyl ionization.

There is no marked superiority of this method (iontophoresis) over other measures which result in vascular dilatation in the affected joints. However, some physicians are enthusiastic over their results in the treatment of chronic stages of rheumatoid arthritis with iontophoresis.

If desired, treatments may be given every two or three days.

The physician should be trained in this method before attempting to follow written instructions concerning iontophoresis.

Mecholyl or histamine iontophoresis should not be used in individuals who are subject to attacks of *asthma*, or in those with marked *hypotension* or *severe cardiac disease* other than paroxysmal tachycardia.

The histamine was kept in dark bottles, in a cool place. In hospital cases, treatments were given twice a day; patients at home were given one injection a day and ambulatory patients were treated three times a week. No cumulative or deleterious effects were noticed.

A test dose of 0.1 cc. was given subcutaneously and was increased by a similar amount until the reaction level (usually 0.4 to 0.6 cc.) was obtained; this latter was then maintained until the patient's tolerance increased.

Immediate reactions include flushing, palpitation, headache, nausea, sweating, dizziness and weakness. Histamine was not used in senile patients, or in those with marked vasomotor instability, marked arteriosclerosis, hypotension, cardiac decompensation or bronchial asthma. Seven of 10 patients with rheumatoid arthritis developed increased motion and relief of pain. These individuals became more active, cheerful and their appetites increased. The drug was also found to be especially useful in patients with bursitis and fibrositis.

It must be stressed that such therapy is entirely symptomatic and must merely serve as an additional aid in the generalized plan of treatment of these patients which includes proper mental and physical rest, adequate diet, physical and occupational therapy, orthopedic attention and other measures of importance.

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CHAPTER XXXI

VACCINES IN RHEUMATOID ARTHRITIS

Vaccine therapy is used very little in the treatment of rheumatic diseases in large arthritic clinics associated with teaching hospitals. The work of Sidel and Abrams showed that the beneficial effects which followed vaccine therapy may have been those associated with the injection of any harmless material; in their experience, injections of sterile solutions of physiologic saline were followed by equally good or better results in rheumatic diseases than those which followed vaccine therapy.[29]

They reported improvement following injections of saline in 72 per cent of patients with rheumatoid arthritis and in 86 per cent of those with degenerative joint disease.[28] In a control group treated with vaccine intravenously, 68 per cent improved. In the patients treated by subcutaneous injections of 0.5 cc. of sterile physiologic saline, no other therapy was used except salicylates by mouth; the latter were also given to the vaccine treated group. Some of this improvement is probably due to the psychologic effect of the injection alone. Unfortunately, the symptomatic improvement in both groups of patients may have been due solely to the salicylates; it would be interesting to compare the results of vaccine *versus* saline injections in patients who receive no analgesic drugs. Subcutaneous injections of saline do not cure arthritis.

On the other hand, Wetherby noted that 82.5 per cent of 80 patients with rheumatoid arthritis were clinically improved following intravenous injections of streptococcic vaccine, while only 40 per cent were improved following injections of saline solution.[36]

Although most rheumatologists agree that much of the criticism concerning vaccine treatment is justified, a few physicians who have had wide and extensive experience still believe that vaccines may be helpful in patients with rheumatoid arthritis.[12]

Recent reports in the literature have stressed that vaccines certainly are not specific in rheumatoid arthritis and that in many instances, the results of vaccine therapy have been disappointing.[11,21,24,25] Although no statistics were given, Hindley-Smith reported that following the use of autogenous vaccines of alpha hemolytic streptococci, there were but few cases in which the results were not successful.[15]

We do not advise vaccine therapy in rheumatoid arthritis. If used, however, the following general directions may be followed.

Dilutions of Vaccine.—One may prepare the following dilutions of vaccine: "1" (or 1 million organisms per cc.); "100;" "400;" "0.01" (or 10,000 organisms per cc.). Such dilutions are best performed in a laboratory where a shaking apparatus is available; the stock solutions should be shaken well for a period of fifteen to twenty minutes before dilutions are made. To prepare a 10 cc. vial containing 1 million organisms per cc. ("1") place 0.1 cc. from a bottle containing 100 million per cc. in 10 cc. of sterile physiologic saline. To obtain "0.01" (or a bottle containing

10,000 organisms per cc.), remove 0.1 cc. of vaccine from the (well shaken) bottle "1" and place this in 10 cc. of sterile physiologic saline.

Vaccine Therapy in Rheumatoid Arthritis

Patients with rheumatoid arthritis must receive a general regimen including periods of rest, splinting of acutely inflamed joints, physical therapy (heat, massage, exercise, etc.), high caloric (unless overweight), high vitamin diet, cautious removal of focal infection as a general health measure, additional symptomatic treatment—aspirin, etc., for acute pain, iron and arsenic for anemia, sedation, anticonstipation regimen, etc.

No stock vaccine produces distinctly better results than another in patients with rheumatoid arthritis. Some workers have preferred to culture all possible portals of entry of infection in the particular patient (throat, paranasal sinuses, feces, biliary drainage, cervix, prostatic secretion, etc.). The organisms found are isolated as individual strains and the patient is skin-tested to these as well as to many stock arthropathic (?) streptococci. A vaccine is prepared for that individual, containing the organisms to which he is definitely skin sensitive. Streptococcal skin tests are, however, unreliable and we may find later that they are not the most suitable indices for vaccine selection.[10,13]

Stock vaccines which have been proposed for use in rheumatoid arthritis include[12] Cecil's strain of hemolytic streptococci (Lederle); Clawson and Wetherby's strain (Lilly, V-675 or V-677); Crowe's vaccine, containing many streptococci and two strains of staphylococci (Reynolds and Branson, Leeds, England); Small's antigen from the streptococcus cardio-arthritidis (Sharpe & Dohme); Wyatt-Hicks' streptococcus vaccine (Wyeth); undenatured bacterial antigens (UBA)[27] or others.

Most bacterial antigens, at present, are given intradermally or subcutaneously; this decreases the likelihood of anaphylactic reactions.[33] There is no proved advantage of the intravenous route.[31] Subcutaneous injections are preferred. It is possible that intradermal injections may prove to be desirable.

Dosage: In acute rheumatoid arthritis, one may begin with 10,000 killed organisms subcutaneously, reducing or increasing the dose as described later; in *chronic rheumatoid arthritis,* one may begin with 1 *million* killed organisms given subcutaneously.[2,20,38]

Frequency of Injections.—Injections may be made every five to seven days, depending upon the availability of the patient.

Change of Dosage.—*In acute arthritis,* if an untoward reaction occurs from 10,000 organisms, reduce the next dose to 1000 organisms. *The aim of vaccine therapy has been to produce symptomatic improvement without local or general reactions;* if no improvement occurs with 10,000 organisms, this may be increased to 50,000; then to 100,000; 200,000; 400,000; 600,000; 1 million, 10 million, etc. *Any dose which produces improvement is not increased;* when such a dose produces no further improvement, it may be increased. *Any dose resulting in local or general reaction is reduced in proportion to the degree of reaction.* In chronic (rheumatoid) arthritis, the initial dose of 1 million may be raised to 10 million if no improvement occurs; this may be increased to 20, 40, 80, 120, 160, 200, 300, 400, 500, 600, 700, or 800 million if necessary. If improvement occurs following any dose, this dose is continued until improvement no longer follows. If untoward reactions occur, the dose is lowered so that no local or general reactions appear.

Vaccine Therapy in Rheumatoid Arthritis—*(Continued)*

Duration of Vaccine Therapy.—If improvement follows the use of vaccine, some continue the therapy until symptoms disappear or until no further improvement occurs. If no improvement is noticed in four months, vaccine therapy will probably prove of no value.

Method of Injection.—A tuberculin syringe and hypodermic needle should be used. Vaccine should be warmed to body temperature and the bottle of vaccine shaken *at least a full minute* before withdrawing its contents (to insure a more accurate measurement of organisms).

Increased Frequency of Injections.—If improvement follows vaccine therapy, but is transitory in nature (three days' duration or less), injections may be given every three days if it is impossible to produce a more lasting effect by larger doses of vaccine.

Sensitivity—Occasionally, an individual is found to be extremely sensitive to stock vaccines; because of this a skin test is performed before beginning vaccine therapy. One million organisms are injected intradermally into the flexor side of the forearm, or a similar amount of the vaccine is placed upon a scratch made in the skin. If a local reaction of any severity occurs (large area of redness, large wheal with pseudopods), the initial dose should be very small (10,000 organisms). Adrenalin (1 to 1000) should always be on hand when injections are made and should be given promptly (0.5 cc. hypodermically) if an immediate reaction occurs; such a reaction is extremely uncommon following subcutaneous injections of vaccine. However, some individuals are very sensitive to certain streptococci and may react unfavorably. In these persons, no matter how greatly the dose is reduced, reactions of an untoward nature may occur; the use of vaccine must be abandoned in such individuals.

Do not expect vaccine therapy to straighten out chronically deformed, ankylosed joints.[5]

The dose administered should be inversely proportional to the illness of the patient.[35]

Pharmacologic rules cannot be used to immunize the patient as the amount of antigen needed varies with each case.[37]

Gradually increasing small doses of vaccine may work by first desensitizing the tissues and later by building up some degree of immunity when larger doses are reached.[3,19,35]

Large initial doses of vaccine may increase tissue hypersensitivity.

There is as yet no proof that any one organism causes rheumatoid arthritis.

Storage.—These should be kept in the ice box, but should be warmed to body temperature before being used.

Sterility of Vaccine.—If cloudiness develops in a bottle of vaccine which has been clear previously, laboratory tests for sterility should be done before using it. Turbidity may be due to precipitated protein material which can be removed by proper methods of filtration. If vaccines are kept for any length of time, they should be checked every several months (even if not turbid) for sterility.

Care of Hypodermic Needles.—Rust proof needles are preferred; the points of these should be sharpened periodically. Any physician who has received a series of injections (such as in treatment for hay fever, etc.) will readily attest to the fact that a slight hook at the point of the hypodermic needle may produce marked pain on entering or leaving the tissues, depending upon the direction of the hook; if the needles are kept well sharpened, the injections may be made with practically no discomfort. Injections must be made aseptically with sterile syringes and needles.

Value of Vaccine Therapy.—At present, *we do not use vaccine therapy in the treatment of rheumatoid arthritis.* Some individuals have obtained remarkable benefit,[32] others slight[22] or no benefit and still others feel that vaccines may do harm.

Rôle of Bacterial Infection in the Etiology of Rheumatoid Arthritis.—Most observers agree that bacterial infection plays a rôle in causing arthritis.

Justification for Injecting More Bacteria or Their Toxins Into an Individual Already Infected With These Organisms or Toxins.—Vaccine therapy is used to produce a *general* response in the patient to combat the infection more adequately. As long as an infection remains localized, the infected tissues are fighting alone; vaccines stimulate the uninfected tissues to aid in this combat and produce a general antibody response to the antigen injected.[39]

How Do Vaccines Work in Rheumatoid Arthritis?—We do not know. Various methods of vaccine therapy[4,9,18,34] may immunize or desensitize the individual;[16] some of the symptomatic improvement may be psychologic, and may follow the injection of any harmless preparation subcutaneously.

Is Vaccine Therapy Justifiable in Rheumatoid Arthritis or is the Risk Involved Too Great?—Vaccine therapy is no longer employed in our clinic in patients with rheumatoid arthritis. The editorial committee of the American Rheumatism Association stated that the cautious use of vaccine therapy was justifiable in certain cases of progressive rheumatoid arthritis provided that both physician and patient were aware of the difficulties and that every care was taken to prevent harmful reactions.[14]

Long-continued Vaccine Therapy Causing Amyloidosis.—There is one such report in a young patient with arthritis who died of uremia following 41 injections of vaccine in twenty-two months.[26] However, along with other chronic infectious diseases (such as tuberculosis, osteomyelitis, etc.), amyloidosis has been found[23] to be part of the secondary pathology of this disease; thus there is no proof that the amyloidosis was due to the administration of vaccine.

Bacterial Extracts.—Small has reported good results in chronic rheumatoid arthritis following very minute doses of bacterial extracts.[30] Other workers cannot duplicate Small's results. He found that patients with rheumatoid arthritis react in a similar manner to minute doses of extracts or suspensions of all Gram-positive cocci (staphylococci, streptococci or pneumococci). He has compared his treatment to that with the minute amounts of gramicidin (the germ-killing chemical isolated from soil bacteria) required to kill virulent organisms. The sensational work of Dubos and others has revealed that a chemical from one type of bacteria may kill other pathogenic bacteria.[7,8,17]

Others have employed autogenous streptococcus bacterial filtrates in minute doses intravenously.[1]

Crowe Vaccine in the Treatment of Rheumatoid Arthritis.[6]—Crowe has written extensively concerning his views on the treatment of arthritis with vaccine and has reported rather remarkable results with this form of therapy. Dr. H. Warren Crowe has been in charge of the Charterhouse Rheumatism Clinic in South London. This clinic is devoted to the study and treatment of arthritis among the poorer classes. It is his belief that many different streptococci, serologically distinct are able to produce "rheumatism." He feels that to treat "rheumatism" successfully, all the rheumatic streptococci, as far known, should be combined in the vaccine used.

The polyvalent stock vaccine prepared by Crowe is said to contain 155 different types of streptococci in equal amounts (comprising all of the varieties found associated with rheumatic disease) *and two strains of staphylococci* (which seemed sufficient to him because of the little difference serologically between members of the staphylococcus group). The staphylococci in the vaccine include the micrococcus deformans; Crowe feels that these are just as often involved in rheumatic and allied diseases as are streptococci. He has found no deterioration of this vaccine when kept in the ice box or even at room temperature for ten years. The "Crowe" vaccine is no longer widely used in this country.

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CHAPTER XXXII

THE RÔLE OF ALLERGY IN ARTHRITIS

THE recent work of Rich and his associates has suggested that the basic characteristics of rheumatic carditis can result from anaphylactic hypersensitivity.[53] Rich has also demonstrated that periarteritis nodosa can result from anaphylactic hypersensitivity; this may serve as supporting evidence for the view that rheumatic lesions may likewise result from this type of hypersensitivity. An anaphylactic reaction may produce periarteritis nodosa in some individuals and rheumatic type of cardiac lesions in others. It is well known that acute rheumatic fever has many features in common with the anaphylactic reaction of serum sickness. Others have suggested that disseminated lupus erythematosus may be primarily a manifestation of hypersensitivity or allergy. All of this work is of extreme importance and will be watched by rheumatologists with keen interest. Urbach has suggested recently that Selye's hormonal production of arthritis in animals by desoxycorticosterone raised the possibility of an allergic reaction, *i. e.*, that the pathologic lesions encountered may be the clinical expression of an allergy to the hormone of the adrenal cortex.

It has been stated that "no one has been able to set up any one particular syndrome as allergic arthritis or to prove that any of the arthritides are chiefly or solely the results of bacterial or food allergy."[27] Undoubted clinical examples of allergic arthritis are rarely found in which provocative or therapeutic tests definitely identify a particular food or bacterial antigen.[28]

TYPES OF ALLERGIC REACTIONS RESULTING IN JOINT MANIFESTATIONS

Atopy.—This is a form of allergy which occurs only in human beings. It is characterized by a *hereditary background and positive immediate skin tests* to the offending substances. The exciting agents are known as *allergens* and the corresponding antibodies, as *reagins*. A reaction occurs when an allergen comes into contact with the reagin in the sensitized cells. It has been suggested that an allergic reaction may be prevented if there is sufficient reagin in the blood stream to neutralize all of the allergen and to prevent this from reaching the sensitized cells.[5]

Important forms of atopy include asthma, hay fever, eczema, migraine and urticaria. Atopic forms of arthritis have been reported infrequently. In true atopic arthritis, it should be possible to demonstrate the presence of antibodies (reagins) by passive transfer to a normal skin. Food allergy may be included in this group; this is discussed subsequently.

Serum Sickness.—The joint manifestations of serum sickness follow therapeutic injections of animal sera. Although this type of allergic reaction shows positive skin tests to the offending allergen and circulating antibodies capable of effecting a passive transfer to normal skin, it differs

from atopic arthritis in that there is no hereditary background. This type of joint pathology appears in a large majority of normal persons who receive injections of poorly refined horse serum. Its incidence has been lowered markedly by refinements in the preparation of such serum.

Bacterial Allergy.—The arthritic individual may become sensitized to some substance given off from bacteria harbored in a focus of infection.[36,61,67] When joint tissues have become sensitized to such bacterial products, it is possible that further exposure to this material may result in an allergic reaction with abnormal tissue changes and the development of an arthritis.

Such a theory presupposes that bacteria may enter the blood stream from some source, such as a focus or portal of entry of infection. That this supposition is true may be shown by the following:

Gonococcemia, gonococcal endocarditis and gonococcal arthritis may follow a local Neisserian infection in the genito-urinary tract.

In patients with periodontitis (pyorrhea alveolaris), the chewing of hard candy or massage of the gums results in transient bacteremias in a majority of the cases.

Transient bacteremias may follow surgical procedures (such as removal of the tonsils, teeth, etc.).

It is possible that a focus of infection may intermittently feed bacterial products into the blood stream, leading to an allergic reaction in tissues which have become sensitized to such bacteria or their products.

Bacterial allergy differs markedly from atopic and serum sickness reactions. Bacterial allergy is usually characterized by a *delayed skin reaction* to the offending substances and the *absence of demonstrable circulating antibodies* which appear to be related to the allergic reaction. Although serologic antibodies to bacteria are found in persons sensitive to those organisms, there is no consistent relationship between the presence of a delayed skin reaction to a bacterium and agglutinins or the like in the blood stream. This delayed reaction cannot be transferred passively to normal skin. It is suggested that bacterial allergy may be dependent upon an alteration of the properties of the fixed tissue cell itself and may not be concerned with any type of circulating antibody thus far identified.

Any normal individual may become allergic to bacteria, as shown by a positive delayed skin test to those organisms, without developing arthritis or other diseases. In a similar manner, most adults have positive reactions to tuberculin without having clinically active tuberculosis. *A positive delayed skin reaction to bacteria merely means* that the individual has had previous contact with those organisms.

FACTORS DETERMINING THE ROLE OF BACTERIAL ALLERGY IN ARTHRITIS

1. The intensity of the allergic response of the joint tissue (not the skin).
2. The number and frequency with which the bacteria or their products reach the joint tissue.
3. The host factor.

Factors (1) and (2) depend upon the individual host; it is this latter factor which determines how important bacterial allergy shall be in any given individual.

One may draw a parallel with the form of allergy found in hay fever a positive skin test to pollen does not necessarily mean that the individual has hay fever. If the patient has hay fever, this latter is not necessarily caused by the pollen used to obtain a positive skin test. The host factor determines whether an individual with a positive skin reaction to pollen shall have hay fever.

The concept of bacterial allergy and focal infection causing arthritis supposes a repeated exposure of joint tissue to bacteria or their products, resulting first in local sensitization, and later in a destructive allergic reaction. The acceptance of this hypothesis of the causation of rheumatoid arthritis has been prevented because *focal infection and delayed skin reaction to bacteria are common occurrences in normal individuals.* However, the real answer may lie in a combination of focal infection and concomitant bacterial sensitization on the one hand, and a susceptibility of the host tissue to these influences on the other.[35] What determines the susceptibility of the tissues in the joint to the influences of bacterial allergy and their liability to acquire it are at present, unknown. It is possible that in some cases we may first have bacterial infection of the joint (metastatic infection), with sensitization as occurs in tuberculosis, and that the severe inflammatory reactions are due to bacterial allergy rather than to the virulence of the organisms. Instances of what have been termed truly allergic articular diseases have been reported;[41] in these patients, exacerbations were extremely sudden and in some instances occurred over night and without fever.

Evidence in Favor of the Theory of Bacterial Allergy in Rheumatoid Arthritis: The production of joint lesions in animals due to local sensitization, following repeated injections of bacterial filtrates.

The higher incidence of positive skin reactions to streptococci in patients with rheumatoid arthritis than in other control groups.

Aggravation of arthritic symptoms in highly sensitive persons by minute doses of streptococci.

Flare-up of an arthritis or occasional dramatic improvement following the removal of focal infection (streptococci being by far the predominant organism in such foci).

Angevine and Cecil have reopened the subject of bacterial allergy in joint infection. The experiments of Angevine and Rothbard (discussed previously under the Etiology of Rheumatoid Arthritis) have suggested that bacterial allergy may play a definite rôle in the production of some cases of rheumatoid arthritis.[11]

Evidence Against Bacterial Allergy as a Cause of Rheumatoid Arthritis: A large number of normal individuals have foci of infection and positive skin tests.

Failure of so-called specific measures to produce improvement in some cases.

The Rôle of Streptococci in Rheumatoid Arthritis.—They are the organisms most commonly found in foci of infection, and are capable of sensitizing animals more easily than other organisms. Positive skin tests to streptococci are frequently found in these patients, and agglutinins to streptococci are present in high titer among the majority of individuals with rheumatoid arthritis.

ALLERGY IN RHEUMATIC FEVER AND RHEUMATOID ARTHRITIS

Various workers have regarded these diseases as the response of sensitive individuals to the products of infecting bacteria.[24,25,63] Rich considers both the Aschoff bodies and periarteritis nodosa to be expressions of an allergic state.[50] Freiberg produced joint lesions in rabbits following repeated injections of a bacterial filtrate; these joint lesions did not appear until several injections had been made and became more pronounced with each successive injection.[21] Positive skin reactions were found when the filtrate was given intradermally. Zinsser caused generalized bacterial allergy and was then able to produce arthritis by injection of the bacterium into the joint, while controls were not affected. Swift also produced arthritis by sensitizing joints. Freiberg and Dorst felt that they demonstrated a relationship between allergens from distant foci and the diseased joints.[22] Other investigations have indicated that the joint lesions in rheumatic fever[62,73] and rheumatoid arthritis[33,59,64] may be partly allergic in nature with local bacterial sensitization of joint tissues.

Bruun has repeated and extended the experiments of Klinge, *i. e.*, sensitization of rabbits to sterile horse serum and then, at varying periods, injecting the same serum into one or more joints.[9,34] He could produce either an acute allergic inflammation (chiefly lymphocytic) or a chronic allergic inflammation in the injected joints and in the heart, blood-vessels, muscles, liver and spleen. He considered it probable that rheumatic fever and some forms of polyarthritis are due to the action of bacteria (probably streptococci) in an allergically changed organism.

Patients with rheumatic fever are unusually hypersensitive to streptococci. Birkhaug found a large percentage of patients with acute rheumatic fever to be sensitive to streptococcus protein.[4] Specific sensitivity to a special type of hemolytic streptococcus is believed by some[14] while others consider rheumatic fever to be the result of sensitivity to various streptococci or other bacteria. Still others accept the idea of a broad streptococcal allergy.[6,7,8,54,65] It was found that 77 per cent of 40 patients with rheumatic fever showed markedly positive skin reactions to the nucleoprotein of hemolytic streptococci as compared with only 44 per cent in 207 controls.[32]

Levinthal maintained that acute and chronic rheumatic diseases were anaphylactic disorders with multiple lesions in the mesodermal system produced by continual antigen-antibody reactions in or on tissue cells.[37] In most instances, the antigen is said to consist of dissolved bacterial substances from the sites of subacute or chronic infection.

Urbach suggested that Selye's hormonal production of arthritis by

overdosage of desoxycorticosterone raised the possibility of an allergic reaction. Others (Klinge and Brunn) have shown that sufficiently sensitized rabbits will react not only to specific but also to non-specific factors such as cold with allergic manifestations of the joints, as did the animals of Selye. Urbach suggested that the pathologic lesions following overdosage with desoxycorticosterone may be the clinical expression of an allergy to the hormone of the adrenal cortex.[71]

Those individuals *opposed to the rôle of allergy as the main etiologic factor in rheumatic fever* state that it is difficult to consider valvular vegetations and thick pericardial exudate purely on an allergic basis.[58]

Skin tests are admittedly not good evidence of the etiologic rôle of bacterial allergy in arthritis because of their variation in intensity and time of appearance, and because reactions may occur in non-rheumatic persons.[20]

Some workers have reported that the percentage of positive skin reactions to hemolytic streptococci was no greater than in control groups and was less than in scarlet fever;[57,60] skin reactions to non-hemolytic streptococci are found in rheumatic fever and in control subjects.

The mere fact that patients with rheumatic fever are sensitive to streptococci does not prove that these streptococci cause the disease. We do not consider the tubercle bacillus to be the cause of glomerulonephritis merely because many patients with this disease may manifest a positive tuberculin test.

Various investigators have repeatedly suggested that the lesions of rheumatic fever may be due to the effects of a hypersensitive reaction to bacterial products rather than a direct action of bacterial toxin or a virus. The possibility that these lesions are a manifestation of hypersensitivity have long been widely discussed, particularly in Germany, and by Swift, Coburn and others in this country.[62] One of the arguments against the rôle of hypersensitivity has been the apparent specificity of the lesions as the valvular lesions and Aschoff body do not resemble the usual inflammatory-necrotic lesions of the bacterial type of hypersensitivity produced by bacterial proteins in the body sensitized by infection.

Numerous individuals have attempted to produce experimentally lesions resembling the Aschoff nodules. Rich and his associates showed that *generalized vascular lesions identical with those of periarteritis nodosa could result from hypersensitivity reactions of the serum sickness type.*[50,51,52] It was noted that *rabbits which were subjected to experimental serum sickness developed,* in some cases, *cardiac lesions that, in their basic characteristics, resembled closely those of rheumatic carditis.* They suggested that the lesions of rheumatic fever may be the results of focal reactions of the anaphylactic type. The cardiac tissue of their animals was not peppered with typical Aschoff bodies, as is so often the case in rheumatic fever.

In this study, Rich and Gregory sensitized rabbits to sterile horse serum under conditions that produced serum sickness. This can be done in the rabbit or human being by either an intravenous injection of a single large amount of serum or by a second injection following an initial sensitizing one. The rabbits were then killed at periods between one and five

weeks following the systemic reaction. Not all of the rabbits so treated developed the specific rheumatic type of cardiac lesions. However, focal lesions that were in many important respects strikingly similar to those characteristic of rheumatic carditis, occurred in the myocardial connective tissue, the mural endocardium and valves of these animals subjected to systemic hypersensitive reactions of the serum sickness type.

The lesions in these experimental animals developed as a result of hypersensitivity of the anaphylactic type. This differs in a number of important respects from the hypersensitivity of the tuberculin type.

Infection with bacteria, fungi and filterable viruses give rise to the tuberculin type of hypersensitivity, manifested in skin tests by a delayed and prolonged inflammatory reaction to the injection of the specific protein of the microörganism, in contrast with the prompt and evanescent erythema and wheal of the anaphylactic local reaction. One must remember also that anaphylactic hypersensitivity to bacterial constituents may develop during infection.

There is a marked individual predisposition which governs the site at which anaphylactic reactions will produce their effects when the sensitized body is exposed to the specific antigen. We still do not know what factors govern this individual local tissue predisposition in the sensitized body. For example, in a group of individuals sensitized to egg protein, the ingestion of eggs may be followed in one by the development of urticaria, in another by an asthmatic attack, in a third by abdominal pain and vomiting, in a fourth by purpura, etc. Similarly, some sensitized individuals may develop lesions of the rheumatic type when exposed to the antigen, while other similarly sensitized individuals may develop arterial lesions.

It is especially interesting in this connection that in those localities where the more fulminant forms of rheumatic fever are seen, the anaphylactic type of skin lesions (urticaria, erythema) occur not infrequently during rheumatic attacks. Skin lesions of this type were reported by Coburn[13] in 22 per cent of 162 carefully studied rheumatic patients. Purpura has also been recognized as a manifestation of acute rheumatic fever, being noted in 7 per cent of one series of rheumatic cases.[13] Purpura is also a recognized manifestation of anaphylactic hypersensitivity occurring occasionally during serum sickness,[48] associated with hypersensitivity to foods,[1] etc.

Tissue eosinophilia is one of the hall-marks of local anaphylactic reactions. This is more frequent in acute rheumatic carditis than is usually appreciated. Eosinophils have been described repeatedly in rheumatic hearts.[2,44]

Rich and Gregory have emphasized the many features which are common to acute rheumatic fever and the anaphylactic reaction of serum sickness. In both, one may see arthritis, fever, anaphylactic skin lesions and arterial lesions of the periarteritis nodosa type.

Very little is known of the frequency with which cardiac lesions may occur in serum sickness because of the rarity of autopsies in these cases.

It is evident, however, that serum sickness rarely, if ever, produces permanent cardiac functional damage.

Fox described a case in which there was presumptive evidence of a *relationship between the parenteral administration of a foreign protein and the development of disseminated lupus erythematosus.* He suggested the possibility that disseminated lupus erythematosus may, like periarteritis nodosa, eventually prove to be a pattern of reaction to a variety of antigens in hypersensitive individuals, *i. e.*, that disseminated lupus erythematosus may be primarily a manifestation of hypersensitivity or allergy.[19]

Intermittent Hydrarthrosis.—It has been suggested that hydrarthrosis may be a sensitization disease; a satisfactory response has been observed to intravenous peptone injections in some of these cases.[38,43,45] This subject is presented in a later chapter.

Arthritis and Allergic (Atopic) Disease Occurring in the Same Patient.—The incidence of asthma, hay fever, urticaria and eczema is no higher in rheumatic than in non-rheumatic children. Among 2000 children attending an asthma clinic and an equal number attending an associated rheumatic clinic, only 2 children gave distinct evidence of both disturbances.[30,42] Only 9 of 400 asthmatic patients had arthritis due to hypersensitiveness to infection.[26] In 500 cases of asthma of which 70 per cent were over fifteen years of age, acute rheumatism (chorea, carditis) was found in only 2.6 per cent and other forms of rheumatism in less than 1 per cent of cases.[76] Rheumatoid arthritis has been noted only occasionally in migrainous subjects although migraine is the most common allergic manifestation of rheumatic patients.[66]

In 200 patients with rheumatoid arthritis, 15 per cent gave a history of hay fever, asthma or both, as compared with 14 per cent in a control group; 31.5 per cent had allergic dermatitis or urticaria in the past as compared with 12 per cent in controls.[77] Four per cent of 50 patients with allergic dermatitis had arthritis and 2 per cent of 50 patients with urticaria showed arthritis.

Vaughan noted that 20 per cent of 1000 consecutive adults with asthma, hay fever, urticaria, angioneurotic edema, migraine, gastrointestinal allergy or allergic dermatitis complained of or had complained of rheumatic pains. Of these 200 patients with rheumatic pains, 90 showed some evidence of articular pathologic changes; only 9 of these were classified as rheumatoid arthritis. There were 32 instances of degenerative joint disease.

In the 200 patients, 143 had allergic symptoms attributable to food, 48 inhalant allergy and the remainder had only contact dermatitis.[72]

Draper believed a relationship existed between allergy and chronic arthritis because of the personal and family history which arthritic patients gave of hives, asthma and angioneurotic edema, and because of the presence of joint involvement in serum disease.[16] Young found no increased incidence of hay fever or asthma among patients with rheumatoid arthritis although these patients were abnormally susceptible to skin allergy. Pottenger noted a family history of allergic disease in 73 per cent of 150 consecutive cases of arthritis.[46]

FOOD ALLERGY

Hypersensitivity to food has been suggested as a factor in the causation of some types of arthritis.[56,68,75] Several cases have been reported which showed marked improvement in the joint phenomena following elimination of foods to which the patient was (skin) sensitive.

Probably the most optimistic report on the rôle of food sensitization in rheumatic diseases comes from Turnbull.[69,70] He concluded that cutaneous tests for protein sensitization permit the selection of a diet which will relieve most arthritic patients of the greater part or all of their symptoms. Unfortunately, other workers have not been able to duplicate Turnbull's results.

Pottenger stated there is a hereditary tendency to allergy, arthritis, longevity and a hereditary resistance to essential hypertension and glomerulonephritis.

Young found that *joint symptoms were not aggravated by ingestion of foods to which patients were clinically sensitive.* In 15 of his patients with definite skin allergy from known foods, repeated eating of these foods at no time caused the arthritis to become worse. *Bauer* stated that he *has never seen a patient with rheumatoid arthritis whose joint phenomena were traceable to hypersensitivity to food.*[3]

Although we have seen patients who repeatedly developed transient arthralgia following the ingestion of specific foods, we have never encountered rheumatoid arthritis on a food allergy basis. Arthritis due to food sensitivity is an example of a true atopic arthritis. Although it is possible that an occasional case of rheumatoid arthritis may be aggravated in this manner, instances of arthritis due to food allergy are rare.

TREATMENT OF RHEUMATIC DISEASES BY DESENSITIZATION

Bacterial allergy may play a rôle in rheumatoid arthritis. In such patients the individual strains of organisms (including streptococci) grown from the pharynx, paranasal sinuses, feces, duodenal drainage, etc., may be isolated. Skin tests may be performed with these as well as with other strains of streptococci which have been incriminated in the etiology of rheumatoid arthritis. If one accepts bacterial allergy as being an important etiologic factor in rheumatoid arthritis, the patient may be given (subcutaneously) small ascending doses of a vaccine containing those strains which cause strongly positive skin reactions. Insufficient work has been done in this field to permit final conclusions.

Buckley advised that vaccines be used for desensitization rather than for immunization in rheumatoid arthritis.[10] Wainwright attempted to desensitize patients with rheumatoid arthritis by very small intravenous doses of streptococcal vaccine.[74] Sixty-six per cent of 45 patients were improved following intravenous injections of streptococcal vaccine made from strains to which the patient was skin sensitive. Slow but progressive improvement occurred in four to six weeks, followed by an increase in agglutinins and a diminution or disappearance of the skin sensitivity.

Others feel that attempts at desensitization by streptococcal vaccines and filtrates seem worthwhile in rheumatic fever.[23,39,55] We still do not know how vaccines work.[29]

Histaminase has not been used extensively in the rheumatic diseases; further work is necessary to determine its possible value.

DRUG ALLERGY IN ARTHRITIS

If rheumatoid arthritis is an allergic disease, these individuals may be particularly sensitive to many of the medicaments to which allergic reactions have been reported. These include aspirin, phenobarbital, antipyrine, aminopyrine (amidopyrine), phenacetin, quinine, sedormid, medinal, nirvanol, phenolphthalein, insulin, argyrol, turpentine (in liniments), dinitrophenol, gold salts, neoarsphenamine, cinchophen, sulfonamide preparations, etc.[47] Symptomatic purpura has been reported following the ingestion of quinine, nirvanol, luminal and sedormid. Numerous cases of agranulocytic angina have been attributed to the use of aminopyrine.[15,17,40] "Allergic arthritis" has been reported following the use of insulin.[31]

Rawls reported relief from arthritic symptoms in 5 of 9 patients in whom urticaria followed the administration of cinchophen.[49] He again suggested that the relief obtained by some arthritic patients from jaundice may be due to other factors besides increased bilirubin.

SERUM SICKNESS

Joint phenomena have occurred in approximately 14 per cent of patients who develop reactions following the parenteral use of serum. This is a form of anaphylactic arthritis, an antigen-antibody reaction, which is unlike bacterial allergy of the tuberculin type of hypersensitiveness.

Although symptoms of this disease may follow the first dose of serum, there is usually an incubation period of from eight to twelve days; in exceptional cases, this may vary from a few minutes to more than three weeks. *Symptoms of serum sickness include* fever, vomiting, skin eruption, edema, slight albuminuria, lymphadenopathy, polymorphonuclear leukopenia with a relative lymphocytosis, neurologic complications and joint phenomena. These latter consist of pains and tenderness of various joints and muscular pains.

Joint pains following serum disease usually persist only a few days but have been known to remain more than three weeks. Despite these subjective symptoms, there is usually very little objective evidence of joint pathology. The joints may appear hot, red and swollen in a small percentage of such cases. In these, the joint fluid is turbid and horse serum may be demonstrated within the exudate by precipitation test. The joints most often involved are the knees, ankles, elbows, wrists and small joints of the hands and feet. Periarticular edema and hydrarthrosis occur occasionally.

Cherry and Prickman have reported *good results in the treatment and prevention of serum sickness by the use of histaminase.*[12] In the prevention of serum sickness, 5 or 6 tablets or capsules of histaminase (each representing 5 detoxicating units of histamine hydrochloride) were given daily, beginning at the time of administration of serum; this dose was continued daily for at least a week. In half of the cases, serum sickness did not appear; in those who developed serum sickness, the symptoms were transitory and disappeared usually within twenty-four hours. In those patients with serum sickness who had not received histaminase, this drug produced definite improvement in over 80 per cent of patients in less than twenty-four hours. This confirmed the previous report of Foshay and Hagebusch.[18]

In patients who do not respond to histaminase, one may employ repeated injections of 0.3 to 0.6 cc. (5 to 10 minims) of epinephrine (adrenalin) hydrochloride (1:1000) every several hours and ephedrine sulfate by mouth 25 mg. ($\frac{3}{8}$ gr.) three times a day for symptomatic relief. Parenteral administration of adrenalin in oil may also be useful. In about 50 per cent of cases, the symptoms of serum sickness disappear within twenty-four hours following the intramuscular injection of 10 cc. of the patient's blood. Before the introduction of histaminase, ephedrine sulfate 25 mg. ($\frac{3}{8}$ gr.), was given by mouth several times each day for several weeks following injection of serum in an effort to avoid serum reactions.

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CHAPTER XXXIII

SULFUR THERAPY

It appears that *the wave of enthusiasm for sulfur therapy* which was so popular ten to fifteen years ago in the treatment of various forms of arthritis, *has now died down to a trickle.* Although numerous articles extolled the virtues of sulfur therapy in arthritis for a period of five years or more, very few reports have appeared in the literature during the past five years concerning this drug.

Several *reasons for the abandonment of sulfur therapy* are apparent:

1. *Freyberg* and his associates, by carefully controlled experiments, *exploded the theory that there was a disturbance of sulfur metabolism in the arthritic patient,* or that there was a biochemical need for sulfur.
2. The rising popularity of gold therapy literally pushed sulfur off of the arthritic map.

It is apparent following Freyberg's work that sulfur is not indicated in arthritis due to any evident lack of sulfur or disturbance in sulfur metabolism in the arthritic patient; however, Freyberg admitted that it is possible that sulfur may have a non-specific beneficial effect.[16] Inasmuch as numerous reports have appeared in the literature concerning marked benefits following the administration of parenteral sulfur, one must remember that sulfur may have been of benefit in these individuals in some not yet understood manner. It is interesting that so careful a worker as Bauer noted that 50 per cent of a series of 14 patients with rheumatoid arthritis showed subjective improvement following only six to eight weeks of treatment with parenteral sulfur; however, these patients showed no objective improvement, but 20 per cent of this group did have significant drops in their sedimentation rate. Dawson found colloidal sulfur to be absolutely without effect when tried on 12 patients with rheumatoid arthritis.

One of the most significant facts concerning the use of sulfur in arthritis is that at present, we know of *no large arthritis clinic associated with a teaching hospital* (in Boston, New York, Philadelphia, Baltimore, Ann Arbor, Chicago, Rochester, or other rheumatic centers) which *is using sulfur therapy routinely in the treatment of any form of arthritis.* Although it is possible that sulfur may have been dropped from our rheumatic armamentarium too quickly, and without further adequate experimental trial, most rheumatologists are agreed at present that gold therapy offers a great deal more, when used correctly. It remains for some investigator to follow carefully a series of patients with rheumatoid arthritis with alternate cases treated by gold or sulfur; such work should entirely clear up any doubt which may remain in a few minds concerning the comparative value of the two drugs. This investigation is being carried out at present.

Sulfur is an ancient medicinal remedy, formerly called "Theriou," or divine, by the Greeks.[42] "Sulfur and molasses" has a long-standing reputation as a household remedy and a spring tonic.

Colloidal sulfur therapy in arthritis was first used by French and German clinicians.[33,36] Experimental evidence suggested that the cystine content of the finger nails was lowered in arthritis.[7,9,58] In recent work, no disturbance of sulfur metabolism could be found.[16]

Freyberg and his co-workers *found no fundamental difference in the amount of sulfur excreted or in the manner in which it was excreted by arthritic patients or by control subjects.* They concluded that their study revealed no metabolic or biochemical indication of need for or benefit from sulfur therapy in rheumatoid arthritis.[17]

It has been claimed that organic sulfur containing compounds (cystine, cysteine and glutathione) act as detoxicating agents in the body and that glutathione aids in cellular oxidation and reduction; the sulfur in glutathione is easily liberated and may aid in detoxication of injurious material.[55] Recent reports have disclosed no definite sulfur deficiency in arthritis; despite this, numerous individuals have reported beneficial results, especially in rheumatoid arthritis, following parenteral sulfur therapy. This may be due, at least in part, to a non-specific effect of the sulfur, or to the protein which is often combined with it; furthermore, *injections have pronounced psychologic effects.* Hypodermic injections of physiologic saline have been followed by equally good or better results than sulfur in arthritic patients.

Contradictory results have been reported concerning the urinary sulfur excretion in arthritis; some[52] have found normal excretion in patients with rheumatoid arthritis and degenerative joint disease, while others noted an increased urinary sulfur.[6,7,46]

The effects of the parenteral administration of sulfur have been studied in laboratory animals[28,30,32] and in man.[21,38,44] Fever and leukocytosis may follow such injections, although these are by no means constant phenomena. Toxic effects of the drug seem to vary with the dispersion and speed of intravenous injection, and may be due to the rate and amount of formation of hydrogen sulfide. *Anaphylactoid* or *colloid shock may follow intravenous injections of colloidal sulfur;*[25,45] urticarial or scarlatiniform eruptions have also been observed.[47] Exfoliative dermatitis may appear within twenty-four hours after the first intravenous injection of colloidal sulfur.[62] Sensitivity to sulfur is uncommon in patients with skin diseases.[34,37,40]

Sulfur and Cystine Content of the Nails.—The sulfur content of nails and hair is almost entirely in protein combination as cystine.[57] The latter is an amino-acid containing 26.7 per cent of sulfur. Normal individuals contain approximately 2.2 per cent of sulfur in the nails.[26] The sulfur content has been reported to be somewhat lowered in psoriasis, some cases of pulmonary tuberculosis, rheumatoid arthritis, inoperable carcinoma with marked cachexia and in patients with prolonged fever.

The average normal cystine content of nails has been found to be approximately 11.7 per cent, although some workers have reported much lower values in apparently normal individuals.[10] The cystine content of the nails has been reported to be decreased in rheumatoid arthritis and degenerative joint disease,[2,65] pellagra, pulmonary tuberculosis[27,59] and in chronic illness in general[49]; in many instances, normal values may be obtained in patients with either rheumatoid or degenerative joint disease.[10] The available methods of measuring cystine content of the nails are time-consuming and require skill and experience.[12,41,53,54,60] In our experience the cystine content of the nails does not indicate a sulfur deficiency in rheumatoid arthritis nor are variations in the cystine content any indication for the use of sulfur therapy in these patients.[7] The cystine content of the nails does not always return to normal in arthritic patients receiving parenteral sulfur therapy.

RESULTS OF SULFUR THERAPY IN ARTHRITIS

Definite benefits have been reported in rheumatoid arthritis following injections of colloidal sulfur. Many of the reports in the literature on the clinical results of parenteral sulfur in arthritis are favorable.[2,3,6–8,20,23,47,50,51,63–66] Others have noted little or no improvement,[19,48,61] disappointing results,[22] no definite improvement,[24] absolutely no effect[11] and improvement in about one-half of the cases.

Abrams and Bauer reported a series of 14 patients with rheumatoid arthritis treated (except in 2 instances) for only six to eight weeks with one brand of colloidal sulfur.[1] They noticed subjective improvement in 50 per cent of the patients without objective changes. Twenty per cent of their patients showed significant drops in the corrected sedimentation rate.

From reports in the literature it may be inferred that from 40 to almost 100 per cent of patients with rheumatoid arthritis have been greatly improved by parenteral sulfur therapy; this improvement has consisted of a decrease of periarticular swelling, increased mobility of the joints and diminution or absence of pain. Unfortunately, *our experience with sulfur therapy has not been this good.*

In our experience, only 20 to 30 per cent of patients with rheumatoid arthritis showed any significant drop in the sedimentation rates. When these same patients were later given gold therapy, the marked subjective and objective improvement together with the decrease in sedimentation rates left no doubt in our minds that gold therapy was vastly superior to parenteral sulfur medication; the latter did not begin to compare with gold therapy in its effectiveness upon patients with rheumatoid arthritis. In all parenteral (or other medication), one must remember that there is a great psychic effect from the injection itself or from oral medication; it is often difficult to judge the extent of this psychic effect unless carefully controlled experiments are run.

Symptomatic improvement has been reported in approximately 50 per cent of patients with degenerative joint disease during the course of sulfur therapy;[63] unfortunately, this improvement does not persist for any great

length of time following discontinuance of treatment. Part of this favorable reaction may be the result of psychologic effects of the injections.

Sulfur therapy has been reported to be of equal value in acute and chronic cases of rheumatoid arthritis.[66] Some have reported best results in younger patients with short histories and others in older patients.

Dose of Sulfur Used in Treatment of Rheumatoid Arthritis.—Just as there is no good rationale for parenteral sulfur therapy in arthritis, *there is* likewise *no good method for determining the dose of sulfur to be used,* except that employed by previous workers, or that suggested by the manufacturers of various preparations of sulfur.

Sulfur has been given intramuscularly, intravenously and by mouth to arthritic patients. Some individuals have claimed that following the oral administration of a teaspoonful of flowers of sulfur once or twice a day for several weeks, their rheumatic pains have lessened greatly; unfortunately, we have never been able to measure this improvement objectively. The old sulfur and molasses spring tonic is a well-known household remedy

Previous workers who have employed parenteral sulfur have begun with 1 or 2 mg. of sulfur given intramuscularly or intravenously. The dose was gradually increased until 25 to 30 mg. of sulfur was being given per injection. Injections were given from one to three times per week, or in some instances, daily. A total of 300 mg. of sulfur was considered to be a course of therapy, following which a rest interval of two months was permitted before repeating the course of sulfur.

If the physician desires to use parenteral sulfur, he must remember that manufacturers have prepared various solutions, some of which are to be given only intramuscularly (such as the oily solutions) and others which may be given intramuscularly or intravenously. There is no apparent reason for intravenous administration; most workers have given sulfur intramuscularly. Some of the preparations prepared for intravenous use will cause intense pain and sloughing if the drug escapes into the subcutaneous tissues.

Sulfur Therapy in Arthritis

Parenteral sulfur therapy is not a panacea for arthritis.

The cystine content of the nails is not a reliable guide as to the advisability of sulfur therapy in arthritis.

The beneficial results following sulfur therapy are probably entirely nonspecific (due to the associated protein frequently combined with the sulfur) or psychologic (due to the injection); *injections of ordinary physiologic saline subcutaneously have been followed by results equal to or better than those reported following sulfur therapy.*

Great caution must be used in sulfur administration; some manufacturers prepare separate products for intravenous or intramuscular use.

Prevent leakage in intravenous injections to avoid local pain and sloughing.

Untoward symptoms occasionally follow sulfur therapy; these may include nausea, vomiting, skin rash, abdominal pain, diarrhea, headache, chills, fever and coronary artery occlusion.

Oral administration of sulfur in arthritis has not been tried in a sufficient number of controlled cases to justify any conclusions at present.

The site of injection (usually the buttock) should be massaged vigorously after intramuscular therapy; sterile abscesses may occasionally follow the intramuscular injection of this drug. There is no particular advantage in employing simultaneous courses of intramuscular and intravenous sulfur therapy.

Untoward symptoms have occasionally followed sulfur therapy; these include urticaria, scarlatiniform eruption, nausea, vomiting, abdominal cramps, diarrhea, fatigue, headache, nervousness, muscular soreness, insomnia, loss of appetite, chills, fever and coronary occlusion. All of these phenomena are infrequent.

Indoluria in Rheumatoid Arthritis.—Indole is formed by bacterial action upon protein;[39] this action may occur in a focus of infection, or in the gastrointestinal or genito-urinary tract.[5] In normal individuals, indole is oxidized within the body to indoxyl, which is then conjugated with potassium hydrogen sulfate to form indican; the latter is excreted in the urine. The liver is supposed to be the site of the detoxifying mechanism of indoxyl.

Free indole does not appear normally in the urine. Forbes and Neale found indoluria in a variety of pathologic conditions, including rheumatic fever, rheumatoid arthritis and degenerative joint disease. They believed that indoluria might be evidence of failure of the liver in its detoxifying action. A diet containing a high proportion of sulfur amino-acids was then given to these patients. The indoluria decreased and eventually disappeared, while clinical improvement accompanied this.

Neuwirth utilized natural hot sulfur springs and mud baths and packs of sulfur containing mud at Pistany Spa, in Czechoslovakia. He found that 88 patients with rheumatoid arthritis had free indole in the freshly voided urine; his results were in agreement with those of Forbes and Neale. It has been shown that sulfur is absorbed through the skin.[4,31,35] Sulfur has also been absorbed following the application of sulfur-containing mud to the shaved skin of a rabbit. Unfortunately, Neuwirth was unable to complete his studies concerning the fate of the indoluria following the use of sulfur baths or the application of sulfur-containing muds.

Oral Administration of Sulfur.—Occasionally, a patient with a mixed form of degenerative joint disease and rheumatoid arthritis will date his improvement (lessening of joint pain and swelling) to the daily ingestion of a teaspoonful or more of flowers of sulfur once or twice a day. We have known some patients who have continued this practice (at the suggestion of friends) for several weeks or several months, without evident deleterious effects. It has been reported that sulfur increases intestinal peristalsis and produces stools which are softer and more frequent than normal.[56] Most of the sulfur which is taken by mouth passes through the bowel unchanged, being excreted with the feces. In an alkaline medium, sulfur reacts with proteins, producing sulfides; these latter are irritant, laxative and probably bactericidal.

Several workers have noted benefit from the oral administration of sulfur in arthritis.[13–15,29] Lubowe used colloidal iodized sulfur and found that 80 per cent of his patients experienced marked relief from the local

symptoms, as well as a sense of well-being, improved appetite and a decrease in fatigue. Greengard and his associates reported a prompt reduction in sedimentation rates following the oral administration of colloidal sulfur.[18] In a small number of patients with rheumatoid arthritis and in some with degenerative joint disease, we have been unable to detect any marked beneficial effect following the oral administration of capsules of colloidal sulfur.

Sulfur Baths in Arthritis.—Warm mineral springs containing large amounts of sulfur have long been used as part of the arthritic armamentarium.[43] Remarkable curative effects of such baths have been noted. It is doubtful if the sulfur content of these warm waters plays any rôle in this improvement, except perhaps a psychotherapeutic one. It is true that many patients will eagerly submit to warm mineral baths while these same individuals would rebel at the thought of mere warm water baths as a part of their physical therapy. A marked increase of blood serum sulfur has been reported following application of sulfur ointment to the skin or sulfur baths.

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CHAPTER XXXIV

BEE VENOM THERAPY

Bee venom has come and gone in the rheumatic armamentarium. *Very few workers still believe that bee venom is of any specific value in the treatment of arthritis.* Its action is probably that of a local counterirritant following its intracutaneous injection, and as such, may temporarily relieve pain in some arthritic patients. However, it has been shown that similar results can be obtained by intracutaneous injections of 10 per cent glucose.[5] Injections of bee venom are not followed by changes in the sedimentation rate in the majority of patients with rheumatoid arthritis.[5,14,20] Unless one is attempting to use bee venom as a local counterirritant, possibly as a foreign protein, or for its psychic effects, there is no good reason for bee venom therapy in arthritis or allied conditions.[24] The following material is being presented, therefore, mainly for those individuals who have some particular interest in this subject.

A large eastern arthritic clinic reported 17 per cent of patients with rheumatoid arthritis "cured" and another 56 per cent definitely improved following bee venom therapy.[15] This was all the more remarkable when it was stated that 6 of 10 patients with an advanced, active deforming type of arthritis showed moderate improvement following such therapy. Following this, physicians have inquired concerning the benefit and rationale of bee venom in arthritis. It is interesting to note that bee venom therapy is no longer used at this clinic, or to our knowledge, at any large arthritis clinic associated with a teaching hospital in this country.[2]

It has often been stated that keepers of bees did not develop arthritis and that arthritic patients were improved or cured following the sting of a bee. Bee venom supposedly provokes a strong reaction when injected into normal individuals but none in rheumatic patients.[3] The therapeutic utilization of actual bee stings has been practiced recently as well as intradermal injections of bee venom.

Bee sting was reported in the treatment of rheumatoid arthritis in 1859.[23] One of the greatest exponents of bee sting therapy was Philip Terc, a general practitioner in Austria. This physician was supposedly cured of his own rheumatism by having been stung accidentally by a number of bees. Following this experience, he continued the use of bee venom in arthritis upon a large scale. Terc is credited with having treated successfully thousands of cases with live bees.[22]

Chemistry of Bee Venom.—Bee venom is a clear liquid with an acid reaction. It is soluble in water and acids, and almost insoluble in alcohol. It is thermostable and its effect is not completely abolished by cold, even by freezing. It is destroyed rapidly by oxidizing substances and alcohol. Its action is weakened by digestive ferments, and it is destroyed quickly

if taken internally. Dried venom is said to remain potent for years if preserved from moisture or if kept in glycerin.

Pathologic Effects of Bee Venom: Destruction of the central nervous tissue by a neurotoxin.

Hemorrhagic phenomena due to an increased permeability of the capillaries.

Hemolysis of erythrocytes.

Toxic Symptoms From Bee Venom.—A list of such complications is almost legion. Such a list has been compiled and is presented below:[13]

Nervous system: Faintness, weakness, stupor, delirium, unconsciousness, restlessness, confusion, vertigo, incessant dreaming, dull throbbing headache.

Skin: Sweating, paresthesias, pruritus, local edema.

Face: Pallor, swelling, waxy appearance.

Eyes: Nystagmus, pain, congestion, corneal ulcers.

Mouth: Ready bleeding of gums, blue, swollen lips, bad breath.

Ears: Difficulty in hearing.

Throat: External and internal swelling.

Lungs: Cough, constriction in the chest.

Heart: Tachycardia, palpitation.

Gastrointestinal tract: Anorexia, thirst, vomiting, belching, heartburn, griping abdominal pain, watery foul-smelling diarrhea.

Genito-urinary tract: Pain in the loins, frequency and burning on urination, frequent long lasting erections, pains in the lower abdomen (in women), frequent abortions, and burning, swollen and hardened breasts.

Extremities: Paresthesias in the hands; severe pains in the shoulders.

Fatality From Bee Stings.—Fatal results from bee stings have been reported not infrequently.[4,7,9,11,12] Most of the fatalities caused by bees have followed stings on the face, nose, eye, neck and ear.

Pharmacologic Action of the Venom.—The action of bee venom following intravenous injection is similar to that of the saponins or the sapotoxins.[21] Investigation of the venom of bees has revealed the presence of an inflammatory irritant of a supposedly non-toxic nature; this may be the active principle of bee venom.[8]

Intracutaneous injection of bee venom produces nearly the same result[8] as that of cobra venom and histamine; the latter is present in bee venom.

Some of the pharmacologic actions of bee venom are said to include a lowering of blood pressure, acceleration of respiration, increased peristalsis, reduced coagulability of the blood and, at times, a peripheral paralyzing action.

For many years it was believed that formic acid was the important principle in bee venom. Recent work has demonstrated that bee venom contains no formic acid which can be found by chemical analysis.[17] This is of some importance as formic acid has been used recently for the treatment of rheumatic diseases.

Possible Beneficial Effects of Bee Venom in Patients With Arthritis.—The supposed curative effects of bee venom are thought to be due to the hemorrhagic and neurotoxic properties. This hemorrhagic effect is manifest by dilatation of capillaries, acceleration of the circulation, and a lytic effect on capillary endothelium permitting erythrocytes to migrate through the vessel walls into the tissue spaces. Such a process may improve the local oxygen supply and the elimination of waste products. This dilatation of arterioles and capillaries is similar to a histamine response. By its neurotoxic action, bee venom paralyzes the peripheral end-plates of the sensory nerves and releases the capillary constriction of the nerve endings of the sympathetic nervous system.

Injected Bee Venom vs. Actual Bee Stings.—By means of injectable bee venom, many factors can be controlled; these include accurate dosage, strength of the venom, asepsis, etc. When injectable bee venom is used, less pain occurs than following the sting of a bee, as the volatile oils have been removed in the former. The amount of venom which may be injected by the bee in its sting varies with the species of bee, the season of the year, the type of pollens upon which the bee has fed, and the eagerness of the bee to empty the entire contents of the poison bag.[3] Furthermore, very young or very old bees possess less venom. The virulence of bee venom is decreased by cold and by feeding of the bees upon sugar syrup rather than upon pollen; the amount of venom available is greatest in the middle of the summer. Bees can usually sting only once. Wasps may sting repeatedly but subsequent stings contain less venom.

Contraindications to Bee Venom Therapy: (1) Chronic cardiovascular disease; (2) coronary artery heart disease, including coronary occlusion; (3) angina pectoris; (4) cardiac decompensation; (5) impairment of renal function; (6) pregnancy; (7) tuberculosis; (8) syphilis; (9) gonorrhea; (10) diabetes mellitus.

Conditions in Which Venom Has Been Used: (1) Fibrositis; (2) neuritis; (3) acute rheumatic fever and rheumatic endocarditis; (4) acute and chronic rheumatoid arthritis; (5) rheumatic iritis and iridocyclitis.

The author has used bee venom only in the treatment of rheumatoid arthritis and fibrositis; no definite objective improvement was noted. On the basis of this experience *bee venom is not recommended in arthritis* except possibly for its local counterirritant or psychotherapeutic effects. Good results have been claimed, however, in these conditions by various workers.[1,10,16]

Preparations Available for Bee Venom Therapy: *"Apicosan,"* which was made in Germany by Dr. A. Wolff, and distributed in this country by A. W. Kretschmar, Inc., 396 Broadway, New York City.

Lyophilized honey bee venom has been supplied by Sharp and Dohme, Inc., Philadelphia.

"British Bee Venom" is prepared by the Antibody Products, Ltd., Watford, Hertfordshire, England.

"Apisin" is a product of the Welcda Company, Ltd., of Arlesheim, Switzerland.

"Immenin" may be procured from the State Serotherapeutic Institute, of Vienna. This should not be confused with *"Emmenin"* which is an anterior pituitary-like substance used in certain endocrine disorders. The term "Immenin" is derived from "Imme" which, in Old German, means bee.

"Ven-apis," which may be obtained from R. J. Strasenburgh Co., Rochester, New York.

Form in Which Bee Venom is Sold.—Bee venom is usually prepared as a dilution of the venom in physiologic salt solution. This is placed in sterile ampoules available for individual dosage and in larger bottles for use in clinics. Most firms prepare this venom in several strengths. For example, one of the preparations (Apicosan) is sold in four strengths. Solution I is said to contain the venom of one bee sting in each cc.; Solution II, the venom of 3 bee stings in each cc.; and Solution III, that of 9 bee stings. A fourth dilution, N, a 1 to 10,000 dilution of strength I, is used to test sensitivity before beginning therapy. Certain preparations, such as Venapis, are prepared both in solution for injection, and in inunction form.

Administration of Bee Venom.—Injections are made *intradermally.* It has been stated that if the venom is injected subcutaneously, it will be much less effective. Intramuscular or intravenous injections are not employed as too rapid absorption of this venom may result in fatality.

Skin Disinfection for Bee Venom Therapy.—Neither alcohol nor any preparation containing alcohol should be used for sterilization of the skin, ampoules, syringe or needle. *Alcohol* is said to *render the venom inert.* Syringes and needles should be sterilized by boiling and the skin and ampoules may be cleansed with acetone, ether, benzene or chloroform.

Precautions in Bee Venom Therapy

The author cannot recommend bee venom in arthritis except as a method of counterirritation, psychotherapy or possibly of mild foreign protein reaction.

A complete history and physical examination of the patient must precede all therapy; contraindications to therapy must be noted.

Injections are given *intra*cutaneously only.

Alcohol must not be used for disinfection of skin, syringe or needles.

Not more than 0.1 cc. of solution is injected at any one point; when larger doses are needed, multiple intradermal injections of 0.1 cc. each are placed at least 1 to 2 cm. apart because of possible local reactions.

Injections are made on extensor surfaces because of the proximity of blood-vessels on flexor surfaces.

Injections are given as soon as the ampoule has been opened as the venom deteriorates rapidly on exposure to air.

Sharp No. 27 gage hypodermic needles are preferable for these injections.

The patient must drink no alcoholic beverages during the course of therapy as the effect of the venom is destroyed by alcohol.

Injection sites should be observed at ten or fifteen minute intervals after injection to determine the amount of local reaction obtained; further dosage depends upon the local reaction.

Frequency of Injections.—Injections may be given twice a week. Daily injections have been used by some, and when necessary (due to limitation of the patient's time), injections have been made as often as twice a day if no untoward reactions occurred. Dosage varies with the preparation used.

Method of Determination of Dose of Each Injection.—At the time of each injection, the sites are inspected at five and ten minute intervals to determine the size of the local reaction. If large pseudo-pods develop in the wheal, the same dose is repeated at the next injection. *The dose is never increased if marked local swelling occurs.* Before each injection, the patient is questioned concerning the amount of swelling and local reaction which followed the previous treatment. There is usually no reaction; if, however, redness and swelling occurred, the same dose is repeated until this is no longer noted. The appearance of focal reactions (exacerbation of pain and swelling in the affected joints) does not call for a decrease in dosage unless the pain and swelling are maintained. Polyuria is almost always noted following these injections as bee venom is considered to be a diuretic.

The more frequent general reactions include urticaria, slight fever, headache, dizziness, nausea and diarrhea. One of the author's cases developed a severe generalized urticaria which persisted for six days despite the usual forms of therapy directed toward this complication. The occurrence of any severe generalized reaction calls for a discontinuance of the therapy, temporary decrease in the dosage or increased intervals between treatments. There have been no fatalities reported following bee venom therapy in arthritis.

Duration of Therapy.—If improvement occurs, a total of 25 or 30 treatments has been advised. If further treatment seems advisable, a second course of therapy has been used after a rest period of two months. We do not use bee venom in the treatment of arthritis.

Results to be Expected Following Bee Venom Therapy in Arthritis.—*In patients with true rheumatoid arthritis, the author has been unable to confirm the good results reported by others.*[18] In a small number of such patients (15), there has been no objective improvement in the joint phenomena with bee venom. Beck, in his book on this subject, admits that the average case of rheumatoid arthritis is much more resistant to treatment than neuritis, muscular rheumatism, etc., although he claims that the results of bee venom therapy in spondylitis are very good. He feels that patients with muscular rheumatism and myositis (fibrositis) respond quickly to bee venom therapy. Rheumatic iritis and iridocyclitis have been said to respond to bee venom therapy; in these cases, only the weakest dilution is recommended.

Beck's presentation has been criticized on the grounds that he has entirely omitted any details of his own experience with bee venom therapy.[19] No statistical record of his own results are presented and no controls are included. Beck has not offered any original proof in support of most of the statements concerning the therapeutic value of bee venom in arthritis.

Approximate Cost of Bee Venom to the Physician.—This varies with the preparation used. If a quantity of the material is purchased for the treatment of several patients, the cost of bee venom averages $.20 or less per injection.

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CHAPTER XXXV

CHAULMOOGRA OIL IN ARTHRITIS

THE *use of chaulmoogra oil in arthritis is entirely empirical.* McIlhenny, orthopedic consultant to the National Leprasarium at Carville, Louisiana, noted in 1926 that the "secondary infectious type of arthritis" was not seen in patients afflicted with leprosy. In an effort to explain why lepers did not develop rheumatoid arthritis, he concluded that chaulmoogra oil (which they received) was responsible for its prevention.[4]

Chaulmoogra oil has been used for many years in the treatment of leprosy. No deleterious effects have been noted upon any organ when the drug is given in the usual dose. Large doses administered orally may produce gastric irritation; excessive amounts injected intramuscularly may lead to a painful myositis.

Powerful bactericidal properties have been attributed to chaulmoogra oil *in vitro*.[8] Leukocytosis frequently follows its parenteral use.[10]

The preparation of chaulmoogra oil for parenteral use has been as follows:[3] 10 cc. of olive oil are mixed with 90 cc. of crude chaulmoogra oil and 0.2 gm. of benzocaine. One to two days is required for the preparation of this mixture.

Authors have reported beneficial effects in rheumatoid arthritis and in degenerative joint disease following intramuscular use of the above preparation.[1,2,5,6,9] The method of administration and its dosage as reported have been as follows:

McIlhenny[8] advised: Injections (3 to 5 cc.) twice per week in addition to from 0.3 to 1.6 cc. of chaulmoogra oil taken orally in enteric capsules at meal times.[5]

Smith and his colleagues used 5 cc. intramuscularly two times each week for the first two weeks, followed by 5 cc. weekly for an additional four weeks.[7]

Because of the *frequent production of sterile abscesses and local pain following its intramuscular injection,* it was advised that intramuscular injections be made into the gluteus medius muscle in the upper and lateral margin of the buttock. The chaulmoogra oil should be warmed to body temperature (rendering it more fluid) and the needle should be inserted at a point 1½ to 2 inches below the crest of the ilium and above the border of the gluteus maximus. When the needle touches the ilium, it is withdrawn less than ¼ inch, and the injection is made slowly into the gluteus medius.

Results Following Parenteral Therapy With Chaulmoogra Oil.—Thirteen of 15 cases of rheumatoid arthritis became symptom-free, with marked improvement in general health, decrease in joint swelling and gradual objective joint improvement.[7] Of 33 patients with a mixed form of arthritis, 18 became symptom-free and 14 were moderately to markedly

improved; of 17 patients with degenerative joint disease 6 were improved, but subsequently had recurrences of their symptoms.

The age of the patients and duration of symptoms had no bearing upon the results. Slight general reaction with fever and leukocytosis was noticed in a few.

Improvement was noted during the second week of treatment; McIlhenny found that the average patient was symptom-free within eight weeks.

Another worker reported reduction of pain and discomfort and increase in appetite, weight and general health in practically all cases of "chronic infectious arthritis" treated with chaulmoogra oil.[2]

Insufficient work has been done upon this form of therapy to recommend its use to the general practitioner. The author has not employed this drug in the treatment of rheumatoid arthritis. Local pain and sterile abscesses are not uncommon following these injections. Many drugs have been hailed as "arthritic cures," only to fall by the wayside following trial in carefully controlled cases. It is significant that, although the original paper of McIlhenny appeared in 1931, there have appeared to date very few reports on the use of this drug from any of the large recognized arthritic clinics in this country. Several drug firms (Parke, Davis & Co., Eli Lilly & Co.) have supplied this material for experimental use.

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CHAPTER XXXVI

ORGANIZATION OF AN ARTHRITIC CLINIC

THE staff of an arthritic clinic should include the following:

A chief of staff: preferably an internist, with a good working knowledge of the diagnostic, therapeutic and research problems in this field.

An assistant: also preferably an internist.

An *orthopedist:* to assume responsibility for the prevention, detection and correction of postural abnormalities, deformities, weak, pronated feet, etc., and to provide proper splints for joints as needed; he shall also perform such orthopedic surgery as may be necessary.

A *physical therapist:* a well trained physical therapist is one of the important cogs in the arthritic machine.

An occupational therapist.

Consultants: in otolaryngology; dentistry; gynecology; urology; bacteriology and pathology; roentgenology; and psychiatry.

Nurses, as necessary, depending upon the size of the clinic; they should be trained in arthritic work and must be kind and sympathetic at all times. Such nurses are very valuable in improving the morale of the patients. A dietitian is a valuable, but not an essential, adjunct.

One or more social service workers.

A laboratory technician.

One or more research fellows, if funds are available.

Representatives from reliable orthopedic supply houses and orthopedic shoe stores.

A layman's committee, composed of citizens (especially women) who will be able to aid the clinic by raising funds, disseminating knowledge concerning the prevention and care of arthritis, etc.

If it is possible, the arthritic clinic should have sufficient room available so that all of the consultants may be present at the same time in separate rooms to complete any necessary consultations at once; this obviates the necessity of asking the patient to return to the hospital on four or five different days for such consultations. If one individual in each specialty is assigned to the arthritic clinic, both the patient and the specialist will profit thereby. The patient will derive the benefit of seeing the same specialist for any revisits which may be necessary, rather than, for example, to see a variety of otolaryngologists on subsequent visits, some of whom may not be particularly interested in arthritis. The specialist will benefit by noting the abnormalities in a large series of arthritic patients, and in following the results of his therapy (if indicated) as it pertains to arthritis.

Proper *physical therapy* such as baking, massage, baths, exercises, etc.,

are a *sine qua non* in the treatment of arthritis. If funds are available, a therapeutic pool for underwater exercises should be included in the armamentarium of this department.

Occupational therapy is of great value in re-educating wasted muscles and in keeping the mind and limbs of the arthritic patient occupied.

It is especially advisable that sufficient funds be set aside, if possible, for the use of one or more *research fellows* in arthritis.

In the *orthopedic* room, apparatus must be on hand for making plaster splints, body casts and other orthopedic appliances. Arrangements should be made with a reliable orthopedic house so that an agent may be on hand during clinic hours to take orders for proper arch supports, braces and apparatus which may be required.

Close coöperation must exist between the internist and the *roentgenologist;* the latter must be interested in arthritis and must speak the same language as the internist. There should be a weekly conference between the internist and roentgenologist to go over all films taken during that period so that no misunderstanding occurs; it is preferable that the orthopedic consultant be present at such time.

A *psychiatrist* is an important addition to an arthritic clinic. By careful questioning, he can often obtain information which may be valuable in the therapy of the patient. Most of these patients are fatigued and many have complicated domestic problems which they do not care to discuss with "the first individual who comes along." A psychiatric examination should form an integral part of the examination of many arthritic patients.

The *social service worker* can take care of problems such as securing adequate relief funds for the patient (if necessary), obtaining orthopedic shoes, assisting in procuring attendants for children when the mother is confined to a hospital, etc. Many of the details which cannot be performed by the physician because of lack of time, can be accomplished by these trained workers.

The clinic must have available to it a number of *hospital beds* for special care when this is needed, as in acute arthritis, correction of deformities, operative procedures, etc.

Every arthritic clinic should have a committee of women (and men, if desired) whose function is to spread information concerning the working of the clinic, the prevention and incidence of arthritis, and to obtain additional funds for the assistance of the clinic and research in arthritis. A member of this committee should be present during the working hours of the clinic to observe its function and to assist the social service worker in any problems which may arise. Where additional medication or orthopedic appliances cannot be afforded in particular cases, it is the function of this committee to devise ways and means of obtaining these needed articles for the patients.

The clinic should have available reading matter on this subject. This may vary considerably. Some of the following are not available at present because of the European conflict.

PERIODICALS WHICH SHOULD BE INTERESTING TO RHEUMATOLOGISTS INCLUDE THE FOLLOWING:

1. Acta rheumatologica.
2. Annals of Rheumatic Diseases.
3. Archives of Physical Therapy
4. British Journal of Physical Medicine.
5. British Journal of Rheumatism.
6. Journal of Bone and Joint Surgery.
7. Occupational Therapy.
8. Physiotherapy Review.
9. Revista argentina de reumatologia.
10. Revue d'Orthopédie.
11. Revue de physiotherapie.
12. Revue de rhumatisme.
13. Rheumatism.
14. Ztschr. f. Orthop. chir.
15. Ztschr. f. Rheumaforsch.

Snyder and Traeger have traced the development of arthritis clinics in this country and have described in detail the set-up at the Arthritis Clinic of the Hospital for the Ruptured and Crippled in New York City.[1] They stressed the importance of a good *follow-up system* in such clinics.

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CHAPTER XXXVII

ETIOLOGY AND PATHOLOGY OF DEGENERATIVE JOINT DISEASE (OSTEO-ARTHRITIS)

THIS entity is as old as history. Unfortunately, it has been given many names. The term *osteo-arthritis* was first used by Garrod; Goldthwaite introduced the term *hypertrophic arthritis* because of the frequency of enlargement of the ends of the bone and the production of new bone near the joint. Other synonyms are arthritis deformans (Virchow), degenerative arthritis (Nichols and Richardson) and hypertrophic degenerative chondro-osteo-arthritis (Weil). More recently (1936) Walter Bauer and Granville Bennett suggested the name degenerative joint disease. This latter term seems most descriptive of the disease and it is hoped that it will be accepted quickly in this country and abroad; acceptance of the term, degenerative joint disease, will avoid much of the confusion which exists in arthritic nomenclature.

Throughout this book, the terms rheumatoid arthritis and degenerative joint disease are used in referring to the two common forms of chronic joint disease. Rheumatoid arthritis (formerly called atrophic arthritis; proliferative arthritis; chronic non-specific infectious arthritis) refers to that form of joint disease characterized pathologically by a synovitis with extension of an invading pannus over the articular cartilage, and in some cases, by actual bony ankylosis. Patients with rheumatoid arthritis, in general, develop the first signs of their disease at an earlier age (most often between twenty and fifty); the disease is accompanied by an increased sedimentation rate, loss of weight and strength, muscular atrophy, fusiform swelling of the proximal interphalangeal joints, as well as the knees and other joints, anemia, fever, typical roentgen changes and the development in some patients of contractures and deformities. It must be remembered that although rheumatoid arthritis most often begins between the ages of twenty and fifty, it may appear in infants, and it is not uncommon to note its onset at any age after fifty.

On the other hand, degenerative joint disease clinically does not usually produce symptoms until past the age of forty-five (except in those women who have had a premature menopause from any cause, and in those individuals whose degenerative changes may have been caused by occupational or recreational strain). Degenerative joint disease is usually accompanied by no fever, no anemia, no change in the sedimentation rate, very little or no joint effusion, no tendency to ankylosis, no loss of weight, no synovitis, but is often associated with the tell-tale Heberden's nodes (at the bases of the terminal phalanges of the fingers).

Numerous theories have been propounded in an attempt to explain the *etiology of degenerative joint disease*. These include endocrine dysfunction; chronic vascular disease with a resultant local anemia; focal infection;

metabolic disorders; aseptic necrosis of bone or cartilage; physiologic stress and strain; heredity; aging and trauma. All that can be said at present concerning the *etiology of degenerative joint disease* is that in some cases it may result from *wear and tear, trauma* or *microtrauma* and *attrition;* in many instances, the primary cause of degeneration of cartilage is still unknown. Most of the other theories concerning the etiology of degenerative joint disease have been discarded.

All workers have placed emphasis upon the *increasing frequency of degenerative joint disease with advancing age.* One must remember, however, that *this disorder is not merely an "old age ailment"* inasmuch as it may be absent in an individual of eighty and present in another of thirty-five. In Bennett and his associates' study of knee joints, evidence of the disease was found in some individuals in the second decade; clinically, the average age of onset in several hundred males in one series was thirty-one years.[58] It would be far more accurate to suggest that the *degenerative joint disease is related to the senescence of joint tissues*[13] *which in turn may be somewhat dependent upon hereditary factors.*[15]

No one yet knows what causes articular structures to age. It has been shown that comparable degenerative changes occur in tendons,[19,39] ligaments,[22] and palmar and plantar aponeuroses.[55]

Pathologically, there occur degenerative changes in the hyaline articular cartilage; these cartilages become roughened, fibrillated, ulcerated, and in some instances may be completely worn down. In addition to the primary change in the articular cartilage, other abnormalities which may occur in these joints include abnormalities in subchondral bone (such as cavities or eburnation); marginal outgrowths (exostoses, lipping, osteophytes); synovial villi, etc.

Bowie and his associates found a small but well-defined respiratory activity in articular cartilage of calves; the young cartilage cell is capable of oxidizing carbohydrate, and perhaps, protein. The respiratory power of bovine articular cartilage cells decreases by 30 per cent from infancy to adolescence and by 60 per cent from infancy to old age. They suggested that the gradual loss of respiratory power of the cartilage contributed to the higher frequency of degenerative joint disease in the more advanced age groups of man.[16]

Degenerative joint disease is often considered to be a result of aging of articular structures (especially hyaline cartilage) and the wear and tear incident to recreational, occupational or the normal strain of life.[25,43] There is no evidence that infection plays a rôle in its etiology: the sedimentation rate is usually normal, there is no fever or leukocytosis and agglutination reactions to hemolytic streptococci are usually insignificant. There is no evidence of inflammation in the synovial membrane. Inasmuch as the disease is uncommon before the fourth or fifth decades, and in view of the increased frequency of this disorder with advancing years, it is most likely that those factors which are involved in the aging of body tissues are the basis for degenerative joint disease. The excellent work of Bauer and Bennett concerning degenerative joint changes in cattle is well known.

Hypertrophic and degenerative changes seem as old as history. Hypertrophic changes have been observed in the vertebral column of a mastodon skeleton of the Pleistocene[17] as well as in the prehistoric animals in the British museum.[30] The Neanderthal man of about 40,000 years B.C. is probably the earliest known human case of degenerative joint disease; hypertrophic changes were present in the lower cervical and upper thoracic vertebræ.[64] Osteophytes were also found in the vertebræ in skeletal remains of the Neolithic area (approximately 10,000 B.C.). More recently, occipital lipping was noted in about one-fourth of more than 200 skulls from Iran;[52] Egyptian mummies showed definite degenerative joint disease.[30] Hooton found degenerative joint disease of the vertebræ in prehistoric American Indian remains.[41]

Bennett, Waine and Bauer outlined the recent advances in our understanding of this disease. Although Hippocrates noted the prevalence of joint disease in the aged and Charcot named old age and heredity as predisposing factors to rheumatic disease, it was not until the nineteenth century that the understanding of degenerative joint disease was markedly advanced by a careful scrutiny of patients and postmortem material.

Eburnation of the femoral head was described by Bell[11] in 1824 and the term *"morbus coxæ senilis"* was proposed in 1835 by the great Irish physician, Robert Smith, for the entity now known as degenerative hip joint disease.[73] Other well-known physicians who noted the high incidence of degenerative hip joint disease in the later decades of life included Adams,[1,2] Colles[20] and Todd.[74] Eckner[25] in 1843 considered this process to be a phenomenon of degeneration caused by senile atrophy, excessive wear and attrition. Later workers showed that the deterioration of cartilage often began at contact points and that marginal exostoses and eburnation were physiological sequelæ of weight bearing after primary changes had occurred in the joint cartilage.

Well-known continental physicians (Cruveilhier, Virchow, Volkmann, Von Rokitansky, Meyer and others) postulated that the primary cause of degenerative joint disease was in the synovialis and some believed that inflammation of the subchondral bone or of the articular cartilage was the initial lesion. Fortunately, Weichselbaum, the well-known Viennese pathologist (1872) disagreed with this thought; he described degenerative joint disease grossly and microscopically and related the disorder to the physiologic process of aging.[77] Later important studies included those of Beneke,[12] Nichols and Richardson,[60] Llewellyn,[57] Beitzke[10] and the excellent treatise of Pommer.[66]

Bennett and his associates concluded that *degenerative joint disease is primarily a degeneration of hyaline cartilage* with involvement of numerous species of animals in varying geographical locations, and in individuals of both sexes.[15] There are a large number of instances in which neither trauma, inflammatory joint disease nor disturbed joint mechanics seem to play a rôle. Unfortunately, articular cartilage has a paucity of cellular constituents, a very limited faculty of autogenous repair, a low respiratory rate and no adequate blood supply. In addition to this, one must consider

the undesirable effects of mechanical stress and strain which follow joint function in a tissue whose resistance to such use has been practically depleted.

Keefer and his associates made extensive gross and microscopic studies on 100 knee joints from 77 consecutive necropsies.[37] They found that between 66 and 100 per cent of all knee joints showed gross anatomic changes after the age of thirty. Abnormalities were found, especially at the points subjected to the greatest pressure, weight and movement.

In the knee, degenerative changes were most often found at the median horizontal facet of the patella and along the patellar groove of the femur. Degenerative changes varied from slight fibrillation of cartilage to erosion and thinning of the cartilage and exposure of subchondral bone.

Microscopically, early changes in degenerative joint disease were found to include splitting of the cartilage and later depression of the underlying subchondral bone which then altered the contour of the normal line of the joint. With complete destruction of the cartilage, there occurred injury to subchondral bone; this latter attempted to repair itself by new bone formation, producing exostoses by an alteration in the normal joint line from flattening and compression of the joint surface.

Keefer and his associates concluded that degenerative joint disease was due to wear and tear, strain, trauma, aging of tissue, occupation and static deformities. Key, Bauer and others have shown that mechanical insults to joints can produce the pathologic picture of degenerative arthritis.[8,49]

Bennett, Waine and Bauer studied a large series of knee joints obtained postmortem, or following amputation of all ages in individuals with no clinical evidence of joint disease. They found that **joints remain normal for only a very short time following complete maturation.** *All joints obtained from individuals beyond the second decade showed changes which were similar to those seen in degenerative joint disease.* In addition, degenerative changes soon appeared in the matrix and cells of the articular cartilage, which became increasingly marked with age.

The study by Bennett, Waine and Bauer included a careful examination of knee joints obtained postmortem or following amputation in 63 individuals varying in age from one month to ninety years.[15] Knee joints secured from individuals in the first decade of life showed no visible pathological features. One of the observations of particular interest was the finding in growing subjects that the entire bony epiphysis was surrounded by hyaline cartilage with a cover of perichondrium into which the connective tissue fibers of the outer portion of the articular capsule inserted. Such a structural arrangement permits longitudinal and lateral growth of the bony epiphysis and is an anatomical factor in the development of marginal elevations. In these individuals the synovial membranes were glistening and smooth, comprising an outer dense, fibrous layer and an inner, vascular, cellular connective tissue stratum of variable depth. In some areas, this inner layer contained many fat cells and extended in places forming branching villous projections. In other areas, no villi were present and the inner layer consisted of only one to three or four cell

layers, especially where the synovial membrane overlay ligaments and tendons.

Degenerative changes were seen as early as the second decade. In later decades, the changes were usually progressive. It is important to realize that *despite the gross pathologic changes which were seen* in the cartilaginous covering of the weight-bearing portions, *many of these joints revealed no abnormalities by x-ray examination.* This fact must be remembered: **A negative roentgen examination does not rule out degenerative joint disease!** In some of these patients, although extensive lesions were present grossly in the cartilage of all articulating surfaces, roentgen examination did not reveal any definite pathological changes.[15]

The *earliest cartilaginous changes* observed consisted of slight unevenness of the surface or a granular surface with streaking or shallow furrows. As the process advanced, these changes became more marked and *pitting, splitting and fraying of the cartilage* were observed. Small *erosions of the cartilage* followed with the development later of *extensive ulcerations* of the articular cartilage. At a later period, large areas appeared degenerated and the *subchondral bone became exposed* and *eburnated* (condensed). In addition to the evident changes in the cartilage, this study showed that with advancing years, the *villous structures* arising from the synovial lining grow in number and size with an increase in the amount and density of subsynovial tissue.

The *menisci* were also *deteriorated* with age, as manifest by thinning or shredding of these structures. *Lipping* (*marginal overgrowths*) was seen in varying degrees at the perichondrial borders of the femur, patella and tibia.

Their studies suggested that the articular lumen is a large connective tissue space with synovial fluid being an example of tissue fluid in general.

All articular tissues are of mesenchymal origin.[9] One finds no sharp and consistent line of demarcation between the intima and the tough external layer of the articular lining (synovial tissues). The intima is a highly cellular, specialized type of connective tissue with an abundant vascular supply. The collagenous matrix predominates in the outer (capsular) layer.

The intima of the synovial connective tissue extends for a short distance onto and blends with the hyaline cartilage. The outer fibrous coat of the synovialis is continuous with the periosteum. In some areas, especially behind the intrapatellar and alar ligaments, pads of fatty tissue project into the joint cavity. In addition, extending from the capsule one finds a variable number of tissue folds which extend toward the joint space. These are seen in normal joints and must be differentiated from synovial villi.

The soft tissues lining the joint spaces have been considered to constitute a physical boundary rather than a true membrane.[15] One should bear this in mind if he speaks of the synovial "membrane" inasmuch as this lining is not a true membrane such as the pleura or peritoneum.

Alterations in the joint capsule in degenerative joint disease are thought to be secondary effects. In several large series of postmortem cases, no

significant lesions in the articular capsule were seen in a thorough and comprehensive study of degenerative joint changes.[10,66] In Bennett's series, grossly visible alterations in the synovial membrane were slight and were prominent only in individuals after the age of fifty. The most obvious abnormality was villous structures hanging as threads from the synovial surface; these were very variable in size and number but usually increased rapidly after the sixth decade. These branching fringes or villi measured up to 2 cm. in length. In badly damaged joints, they resembled pedunculated lipomata because of their rounded or club-shaped endings containing fatty tissue. In a few instances, smooth, white, round and partly calcified cartilaginous nodules were found within the villus.[66]

The *synovial villi* were found most often over the fat pads and in the areas of the joint which were lined by cellular areolar tissue. The projections were composed of a connective tissue core with capillaries surrounded by synovial cells of variable thickness.

Varying degree of arteriosclerotic changes were noticed inconstantly in the synovial vessels, with hyalinization of the media and intimal thickening.

These studies showed that *fairly marked degenerative changes could be present in the articular cartilage without evident change in the epiphyseal bone.* A sharply defined layer of rigid calcified cartilage is found in the normal mature epiphysis lying between the hyaline articular covering and the subchondral bone. The subchondral bone may show striking abnormalities when profound alterations are present in the articular cartilage. Senescence, itself, is often accompanied by a certain amount of resorption of bone. Cavities (*detritus cysts*) of macroscopic size may often be seen by *x*-ray *in the subchondral bone.* These lesions are not specific for degenerative joint disease as similar microscopic or gross changes may be found in a number of pathologic conditions including rheumatoid arthritis, osteochondritis, rickets, etc., and occasionally in apparently normal individuals.[15]

The lateral and medial *menisci* are fibrocartilaginous structures of semicircular design; these are united firmly to the tibia anteriorly and posteriorly. By their arrangement as semilunar wedges between the peripheral surfaces of the tibia and femur, they leave the central weight-bearing portions of the tibial condyles uncovered so that these latter articulate directly with the condyles of the femur.

The menisci undergo the same types of attritional changes as the articular cartilage. Although the degenerative process advances rapidly in some instances, well-preserved structures are occasionally found even in the sixth decade.

Most of the interest in degenerative joint disease has been centered about the changes in the hyaline articular cartilage. This tissue is normally bluish white in normal growing subjects and is smooth, glistening, translucent and resilient. *Beginning in the second and third decades, the cartilage becomes yellowish, less elastic, more opaque and shows small pits, linear grooves or blisters on its surface,*[15] especially in those areas where weight bearing is

particularly concentrated and in the areas where shearing and twisting types of stress are evident (as in the exposed parts of the tibial condyles, patella and patellar surface of the femur). Microscopically, these early lesions appear as small elevations or depressions or slight irregularities in the surface with partially detached fibrillated shreds. Later, these narrow surface ulcers broaden into fissures and clefts and the large circular erosions with sharp edges approach the bony epiphysis; with this loss of intercellular substances and exposure of the collagenous fibrils, the articular surface shows the well-known velvety appearance and spongy texture. The splitting and fraying process extends laterally and toward the diaphysis and in some instances parts of the friable matrix are dislodged and thrown into the joint space by only slight force.

With progression of the process, the chalky, brittle calcified layer (covering the epiphyseal bone) becomes fractured and increasingly large gaps break the continuity of the bone-cartilage border.

Practically all modern investigators agree that **the initial anatomically demonstrable lesion in degenerative joint disease is a degeneration in the superficial layer of the articular cartilage.** Unfortunately, articular cartilage has a very limited potentiality for repair; this is usually accomplished by fibrous tissue resulting at times in the imperfectly formed fibrocartilage.

The **marginal proliferations** or lipping (spurs, **osteophytes,** exostoses) which occur in degenerative joint disease are the subject of much debate. Earlier workers considered them to be intracapsular overgrowths of cartilage. Others described them as fibrocartilaginous, with involvement of the periosteum in the peripheral bony excrescences. In Bennett's series, these outgrowths varied so that in some specimens, they were extensions of subchondral bone into the marginal articular cartilage; in others, they were composed of large shelves of compact bone projecting horizontally from the joint edges and in still other instances, the proliferations were composed of rounded excrescences of cancellous bone with a covering of newly formed cartilage.

Bauer and his associates believe that *no single mechanism operates in all instances in marginal proliferations* but that a part may be played by *advance of subchondral marrow, periosteal bone formation, endochondral ossification of cartilaginous matrix and compression of the devitalized articular covering.*

Bony and fibrocartilaginous lipping progressed increasingly with advancing age. It was noted that the earliest detectable proliferative changes occurred in the transitional tissue between the articular margin and the synovialis. It was especially interesting to see that large masses of cartilage often developed where the articular membrane attached to the periphery of the joint surface; these were later replaced by bony spurs or shelves. These changes developed at a later stage and less rapidly than the changes in the articular cartilage.

Articular lipping is not a prerequisite for the diagnosis of degenerative joint disease.

Pathogenesis of Degenerative Joint Disease[15]

1. *Degenerative changes* occur *in the hyaline articular cartilage* as a result of wear and tear, microtrauma and attrition. No one yet knows the exact mechanism by which joint function contributes to the development of degenerative joint disease.
2. These fibrillated, roughened cartilages produce a degree of *friction* as they slide against one another (which may be palpable and audible).
3. Motion and weight bearing, acting upon a structurally impaired joint cartilage, result in demonstrable *abnormalities in subchondral bone, menisci, synovialis* and in further damage to the hyaline cartilage.
4. The weight-bearing joints (especially the knees and hips) show a numerical and quantitative predominance of involvement with the earliest detectable lesions being located in those areas which bear the brunt of physiologic use (*i. e.*, the exposed weight-bearing portions of the tibial and femoral condyles and the patella).
5. In typical cases, the *primary disease continues until* the *original articular covering has disappeared.*
6. Epiarticular *ecchondroses* or *marginal outgrowths* almost always show signs of degeneration.
7. In those patients with marked cartilaginous lesions, *subchondral condensation of bone* appears.
8. With the decreased substance and elasticity of the degenerating cartilage, weight bearing produces more sustained traction on the peripheral margins of the joint, which stress may induce hypertrophy or hyperplasia (*marginal proliferations*).
9. In the knee, the avascular fibrocartilaginous *menisci* suffer from the attritional forces of joint function.
10. *Effusions into joints* are *rarely present* (and then are small) in degenerative joint disease. The delicate intima of the synovialis may be traumatized mechanically by the gross deformity of the articular surfaces, resulting in a few instances of limited histologic evidence of inflammation.
11. Secondarily, *villi* may appear adjacent to the articular margins: permanent folds which have undergone hypertrophy probably as the result of mechanical irritation or pinching between the irregular surfaces of the joints.

The *essential pathologic lesion* is not a synovitis but is a softening, erosion and degeneration of hyaline articular cartilage in successive stages, with hypertrophy of the neighboring cartilage and bone, tending to bring the articular surfaces into contact.[65] Granulation tissue at the margins of the joint may later be converted into cartilage or bone; detached fragments of this tissue are known as "joint mice." The *synovial membrane remains essentially normal.*

The degeneration of articular cartilage is probably a premature aging of this tissue, as this occurs from the third decade onward. The *most important factors in producing or accelerating damage to articular cartilage* are frequently repeated minor *contusions* and *strains in some occupations.* Additional accelerating factors may include the type of cartilage inherited, obesity and possibly a decreased basal metabolic rate. The primary cause of degeneration of cartilage is not yet known.

Although the exact etiology is unknown, *age and trauma are probably of prime importance;* secondary factors which must be considered include heredity and endocrine disorders. The hypertrophic bone reactions can be produced experimentally in patellæ in dogs by the ligation of patellar arteries. Increased calcification in bone occurs when its blood supply is decreased and decalcification may follow an increase in its blood supply. However, most portions of articular hyaline cartilage have no blood supply. As Hench has stated, the primary cause of the degeneration of cartilage is not yet known.[40]

In summary, *earliest pathologic changes* in degenerative joint disease consist of degeneration of the mucoid matrix of hyaline cartilage and small cracks or fibrillation of articular cartilage (changing from the normal glistening bluish-white color to a dull yellow); these probably result from wear and tear of articular cartilage.[32,48,53,61]

Degeneration occurs first *in the central portion of the cartilage* (as distinguished from the peripheral involvement which is seen in the early stages of rheumatoid arthritis).[29,63] The peripheral portion of hyaline articular cartilage may receive some nourishment from blood-vessels in the thin layer of synovial membrane found in that region; the central portion of hyaline cartilage has no blood supply and probably receives its nourishment entirely from synovial fluid.

As the process progresses, *a granular appearance of the cartilage* is produced, with the formation of pits, depressions and erosions. Coincident with this destruction of cartilage, there often occurs *proliferation of cartilage on the opposite joint surface;* the degree of proliferation is usually less than that of degeneration. Cartilaginous proliferation also occurs at the margin of the joint; this may later be converted into osseous tissue (osteophytes). Heberden's nodes are examples of this.[38]

Secondary degenerative changes may occur in other tissues (capsule, tendons, periarticular tissues).

Portions of the bone or villi may break off to form *loose bodies* in the joint.[44] Small chondromas or osteochondromas may lie within the synovial villi. Degenerative joint disease resembles traumatic arthritis pathologically so that the former seems merely to be an end-stage of the latter.[8]

Subcutaneous fibrous nodules are not found in osteo-arthritis.

The bone underlying the degenerating cartilage becomes denser and connective tissue cells fill the marrow spaces.

The *joint capsule* is usually *not particularly thickened,* and there is no inflammation of the synovial membrane (an important difference from the pathologic changes in rheumatoid arthritis where synovitis is the earliest finding).

In the absence of trauma, excessive amount of joint fluid is rarely seen in the early stages of osteo-arthritis.

The *cartilage becomes thinned and disappears from the ends of the bones;* the latter *become eburnated and shiny,* and may appear smooth and hard as a billiard ball.[24] *Cystlike cavities* may develop in the ends of the bones.

Moderate hyperplasia of synovial villi may occur but without histologic

evidence of infection. In other cases, the synovial membrane may appear normal. Round-cell infiltration of the synovial membrane and pannus formation do not occur in osteo-arthritis.

Ankylosis is rare in degenerative joint disease, and occurs only as a locking together of osteophytes of adjacent lumbar vertebra or occasionally in the sacro-iliac or terminal interphalangeal joints.

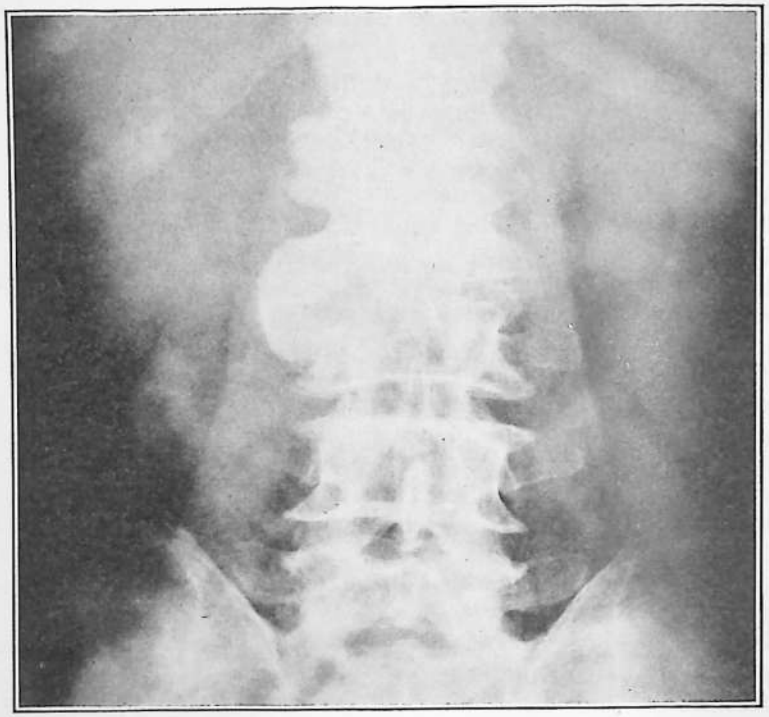

FIG. 117.—Marked hypertrophic changes involving the lumbar spine in a man aged sixty-six years. This is most marked on the right side of the bodies of the second and third lumbar vertebræ. The bony productive changes are marked and have displaced the iliopsoas muscle to the right.

The divergent claims concerning the **etiology of degenerative joint disease** have been thoroughly discussed.[16] **Etiologic theories** which have been proposed in degenerative joint disease include chronic vascular disease with resultant local anemia; dysfunction of one or more of the endocrine glands; specific metabolic disorders; focal infection (due to streptococci, amebæ, fungi or toxins from malfunctioning intestines); aseptic necrosis of cartilage or bone; and numerous others. It is apparent that these older theories have now been throughly debunked.

One of the greatest impediments to the proper understanding of the etiology of degenerative joint disease is improper generalization from single case reports or from particular instances in which this disorder may develop as a sequel to another disturbance. Pommer in 1918 thus asserted

that *occupational trauma* to joints was the single most important causative factor in etiology of degenerative joint disease based on his finding this disorder so frequently among Austrian laborers.[67] This assumption was not borne out by the authentic analysis of Heine[39] who selected from 1000 autopsies at random, 100 individuals who had done hard labor for many years and compared the autopsy findings with another 100 individuals of comparable age who had done no or only light physical work. Both groups showed the same incidence of degenerative joint disease. Pommer has been the outstanding protagonist of the mechanical theory of causation of degenerative joint disease, *i. e., "excessive use beyond physiological limits."*

Practically all students of rheumatic diseases acknowledge the *significance of physiological strains and stresses* in the pathogenesis of degenerative joint disease, but it is doubtful if this answers the question.[49] The occurrence of degenerative joint disease in the terminal interphalangeal joints, which are primarily non-weight-bearing joints, is an additional argument against the mechanical theory. Other workers[4,5,6] have shown that changes which are analogous to the degenerative phenomena seen in articular cartilage may occur in the hyaline cartilage of the trachea, ribs and in the elastic cartilage of the ear and epiglottis in which sites the functional stress of weight bearing is not a factor.

A careful analysis of the pathogenesis of degenerative joint disease leads to the conclusion that *direct or indirectly induced injury to articular cartilage, followed by degeneration of cartilage forms the common denominator in human or animal joints.*[15] Such simple functional derangements as displacement of the patella may produce in the experimental animal marked joint changes.[14] Other predisposing lesions include contusions, dislocations or fractures near or into joints, malformations and malignments of congenital, neoplastic or traumatic cause, in addition to inflammatory and specific infectious joint disease.

Degenerative joint disease has occurred in *caisson workers*[45] following the development of aseptic necrosis of epiphyseal bones; similar lesions have been found in *workers using pneumatic tools.*[28,56]

Aseptic necrosis of bone may result in degenerative changes in adjacent joints. Massive infarction of bone may result from caisson disease, injury to tissues resulting from the release of nitrogen bubbles produced by too rapid decompression. If the necrotic bone is situated at the epiphyses and borders on joints, the cartilage overlying these parts may break down and be replaced by fibrocartilage and a varying amount of secondary osteo-arthritis may result.[45]

Hypertrophic joint lesions have also occurred in *ochronosis* and *gout*, supposedly resulting from chemical injury.[7,76] Likewise, the development of degenerative joint disease *following* repeated *hemarthroses* has not been adequately explained.[31] The sensory changes which accompany *tabes dorsalis* and *syringomyelia* are said to be partly responsible for the development of the marked degenerative changes (Charcot joints) in these conditions.

Careful immunological and bacteriological studies have failed to show any good basis for the association of rheumatoid arthritis with degenerative joint disease;[21,23,33,46,62] however, some authors unfortunately group these two distinctly different diseases in the same category of chronic arthritis.

Although it has been suggested that *syphilis* plays an etiologic rôle in degenerative joint disease, the incidence of this disease is approximately the same in syphilitic and non-syphilitic individuals.[39]

A careful analysis of the etiology of degenerative joint disease shows that there is *no good evidence to support an infectious etiology* for this entity.[15]

The rôle of *vascular changes* has appealed to numerous individuals as an etiologic factor in degenerative joint disease. The great majority of competent workers who have made repeated examinations histologically of joint tissues are not inclined to consider arteriosclerosis as the cause of the degeneration of cartilage.[37,39,50,70,71] Although arteriosclerosis and degenerative joint disease frequently concur, there is no good direct evidence of a causal relationship. Degenerative joint disease is found only infrequently in the presence of even advanced thrombo-angiitis obliterans and arteriosclerosis.[51] Kling found no relationship between vascular changes in synovial vessels and the articular changes present, thus lending no support to the theory that degenerative joint disease resulted from degeneration secondary to arteriosclerosis of articular vessels.[51]

The etiologic rôle of *endocrine disturbances* in degenerative joint disease is undetermined. The changes seen in the advanced stage of the peripheral and spinal joint disease associated with *acromegaly* are grossly indistinguishable from those of degenerative joint disease.[15,26,27] Silberberg has produced somewhat comparable lesions in guinea pigs following the injection of acid extracts of pituitary substance derived from cattle.[72] Although low *basal metabolic rates* have been reported in a large percentage of patients with degenerative joint disease,[35,37,54,59] this is not true in all reports.[68,69] The association of *arthralgias* with the *menopause* and the occurrence of definite arthritic symptoms has been commented upon particularly by Hall and by Ishmael.[36,42] Umber commented upon an endocrine type of arthritis occurring rarely, but almost exclusively in women with an ovarian deficiency.[75]

Women have Heberden's nodes much more commonly than men. Although either *sex* may show a higher incidence or a greater extent of degenerative joint disease in particular articulations (due probably to environmental, constitutional or hereditary factors), if one considers all of the manifestations of the disease, the distribution between the two sexes is about equal.

The etiologic relation of degenerative joint disease to *faulty purine metabolism*[34] is without histologic evidence.[10,18,39,78] Similarly carcinoma and osteoporosis play no known etiologic rôle.[39]

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CHAPTER XXXVIII

SYMPTOMS AND LABORATORY FINDINGS OF DEGENERATIVE JOINT DISEASE (OSTEO-ARTHRITIS)

ALTHOUGH roentgen-ray evidence of degenerative joint disease is found in almost all individuals past the age of fifty, only 5 per cent of these have symptoms of the disease. Hench estimated that there are now at least 24 million Americans with roentgen evidence of degenerative joint disease (osteo- or hypertrophic arthritis) of whom over a million have some symptoms resulting therefrom.[31] This is a progressive disease and *demands treatment*, although it *rarely causes helpless invalidism.*[8]

Degenerative joint disease is rarely monarticular except where a single joint is subjected to trauma or long continued use (as in industrial workers[1,4]). Even though symptoms may occur in only one joint roentgenograms usually show similar changes in the opposite joint.[18,33]

Pain and restriction of function are the main clinical features of degenerative joint disease. Usually no marked increase in the size of the affected joints occurs, and *hydrarthrosis is uncommon, except in the knees. Friction and creaking of the joints* may be heard at a distance in some cases, while in others this may be detected only by palpation or by stethoscope; they may be the first signs of such joint disease.

Secondary fibrositis often accompanies degenerative joint disease and results in fatigue, muscular aches and pains, stiffness and loss of muscular resiliency.[9,55]

The course of degenerative joint disease varies greatly. Many of these individuals have considerable discomfort and disability for years. Others may experience only a slight stiffness in the neck or low back or an occasional Heberden's node. Pain in degenerative joint disease has been attributed to elevation of the periosteum following proliferation of marginal bony tissue and to fibrositis, pressure on exposed subchondral bone, loose bodies and to occasional pinching of synovial villi.[31]

Heberden's Nodes.—The commonest manifestations of degenerative joint disease are **Heberden's nodes.** These are small cartilaginous or bony masses situated usually at the proximal end of the terminal phalanges. They may affect any of the fingers but are *more common in the index and middle fingers.* One or all of the digits may be involved. The *thumbs are rarely involved.* The first metatarsophalangeal joint is affected occasionally, but in general the toes are exempt from degenerative joint disease. *Heberden's nodes are a distinguishing feature of this disease. They do not occur in uncomplicated rheumatoid arthritis.* Degenerative joint disease may also cause bony changes in the proximal interphalangeal joints, but in approximately only 10 per cent of cases.[14] This is in contradistinction to rheumatoid arthritis in which involvement of the proximal interphalangeal joints is the rule, and of the distal joints, the exception.

Symptoms of Degenerative Joint Disease

Usually an elderly patient with few joints involved.
Joints most frequently involved: Terminal phalangeal joints of the fingers, lumbar vertebræ, knees, sacro-iliac, lower cervical vertebræ, hips and shoulders.
Almost never involves wrists, elbows, knuckles or feet.[31]
Usually *very little or no joint effusion.*
Symptoms uncommon before forty years of age, except when due to trauma or artificial menopause.[49]
Onset insidious in most cases.
Patient usually overweight, hypersthenic type.
Practically no systemic manifestations (fever, loss of weight, anemia, subcutaneous nodules, lymphadenopathy or splenic enlargement).
Main symptoms: Stiffness, pain on motion and usually only slight deformity; *pain and stiffness* are most common in the knees and back, especially in the obese.
Pain is usually relieved by rest.
No significant muscular spasm, atrophy or flexion deformity.
Ankylosis is very rare (spine, sacro-iliac joint, finger?). True bony ankylosis does not occur.

Laboratory Tests

Sedimentation rate—usually *normal.*[3,45]
Complete *blood count*—usually *normal.*
Agglutination reactions with hemolytic streptococci—usually *negative;* antistreptolysins—generally normal; antifibrinolysins—usually absent; usually no increased skin sensitivity to streptococci.
Normal values for blood calcium, phosphorus, urea, proteins, albumin-globulin ratio, serum phosphatase and glucose tolerance test; cholesterol may be slightly increased.[38]
B.M.R.—decreased in from 10 to 30 per cent of the patients.
Main roentgen findings—narrowing of the joint space (due to destruction and finally loss of articular cartilage); sharpened articular margins; osteophyte formation and lipping of marginal bone; damaged and thickened, eburnated subchondral bone; and bone cysts. This is discussed in detail in the section on roentgenology.[16,32,34,35,66] Roentgenologic hypertrophic changes may be seen in other forms of arthritis (gouty,[63] traumatic, gonorrheal, psoriatic or in some cases of rheumatoid arthritis[30,62]).

In 1802, Heberden first described these "little hard knobs frequently seen on the fingers"; his description in Latin contained only 53 words,[28] translated later into 79 English words. Although Heberden himself did not specifically confine his nodes to the terminal phalangeal joints, current usage restricts the term to these distal phalangeal finger joints. The toes are not affected by analogous changes.

When primary degenerative joint disease affects the fingers, the terminal phalangeal joints are most commonly involved, although the proximal phalangeal joints and the base of the thumb may be involved (usually at a later date). The metacarpophalangeal joints are rarely, if ever involved in this process.

Heberden's nodes may arise spontaneously, *i. e.*, without a history of previous specific injury; these are called true, or *primary Heberden's nodes. Traumatic Heberden's nodes* may follow a baseball injury, or the catching of a finger in a door or a window, etc. Degenerative changes occur in the cartilage in both types, together with hypertrophic bony changes so that the two are usually indistinguishable. In the traumatic nodes, there is usually a history of injury followed at once by pain and periarticular soft tissue swelling; after several weeks or months, hypertrophic bony changes appear and the articular enlargement soon becomes painless and then remains unchanged throughout life.[61]

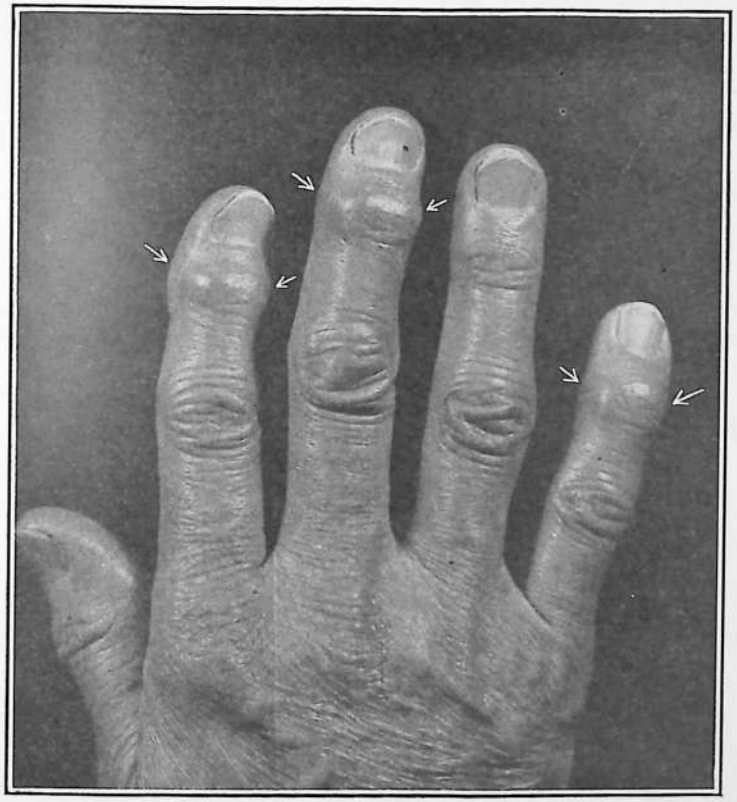

FIG. 118.—Typical **Heberden's nodes.**

The *idiopathic Heberden's nodes* usually appear insidiously, most often in individuals past the age of forty-five (but occasionally in young or middle aged persons, and particularly in women who have had a premature menopause).

In *some instances Heberden's nodes seem to appear rather rapidly, with* from slight to moderate *discomfort,* redness and swelling; the enlargement may be soft or fluctuant at times. The first symptom may be an aching and tenderness on pressure or numbness and tingling.

In the *early stage*, before the appearance of swelling or roentgen changes, the patient may complain of *tingling or numbness of the affected finger*, with or without mild *transient aching* and *tenderness on pressure*. With progression of the process, aching and tenderness become more chronic and swelling, redness and warmth appear. *X-rays* now show a beginning thinning of the interphalangeal space (due to degeneration of cartilage). The osseous reaction itself is usually introduced by slight *marginal lipping*. The bony node increases in size slowly as the *periarticular reaction* begins to subside. Later *x*-rays may reveal destruction of the interosseous cartilage and irregular destruction and hypertrophy of marginal and subchondral bone, leading often to mesial or lateral displacement of the terminal phalanx.

The nodes finally lose their tenderness and the thick, swollen periarticular soft tissues contract over the persistent bony knobs. From this point on, unless subject to further trauma, the Heberden node usually remains relatively painless, but in the eyes of most women, unsightly. Brogsitter[7] has described the pathologic histology of these nodes in detail.

Pathologically, the condition is considered to be a periostitis or osteitis with new bone formation. Histologic examination of Heberden's nodes shows the usual signs of degenerative joint disease including irregular erosion and fibrillation of cartilage, fibrotic and cystic areas in the bone marrow and spur formation.[7] There is usually an increased activity of the perichondrium with formation of new cartilage which may be transformed into bone.[53]

Small gelatinous **synovial cysts** occasionally appear as a complication of Heberden's nodes. These cysts or cystic nodules occur *on the dorsal aspects of the fingers* at or near the terminal joints or between these and the midphalangeal joints. These small cysts are usually attached to tendon sheaths; they may become inflamed occasionally. It is usually unnecessary to open these cysts surgically as they will often recede or open spontaneously. These cysts may also appear in individuals without Heberden's nodes. Weber and Freudenthal have described their pathologic reactions.[74]

Although age appears to be an important predisposing or conditioning factor in Heberden's nodes, premature idiopathic Heberden's nodes may affect individuals in the teens. Sex also seems to be an influencing factor as *Heberden's nodes* have been reported to be *found nine times as frequently in women as in men.*

Stecher reviewed the literature on Heberden's nodes and has determined their incidence in 7000 individuals. He produced evidence in support of his view that their occurrence is strongly influenced by *heredity.*[70] He divided Heberden's nodes into 2 groups: (*a*) the result of direct trauma, and (*b*) idiopathic. The incidence of *both types increased with age.* Traumatic nodes were more common in males and were found more frequently in working men than in physicians. White working men were affected more commonly than negroes. As examples of his findings, the following incidence in various age groups may be of interest:

Age:	20–29	30–49	50–69	70–79
White males	9%	10%	23%	32%
White females	0.2%	5%	20%	41%
White physicians	2%	5%	14%	

Heberden's nodes may *occur as 2 small nodules on the dorsolateral aspect of the distal phalangeal joints*, but more often as a ridge across the dorsum. They may be found on the internal or external aspect of the phalanges. As the disease advances, the swelling extends to the lateral aspects of the joint; it may occasionally be palpated on the palmar aspect. Although they are most common in individuals who have used the hands for hard work, they are often seen in the leisure class of women.

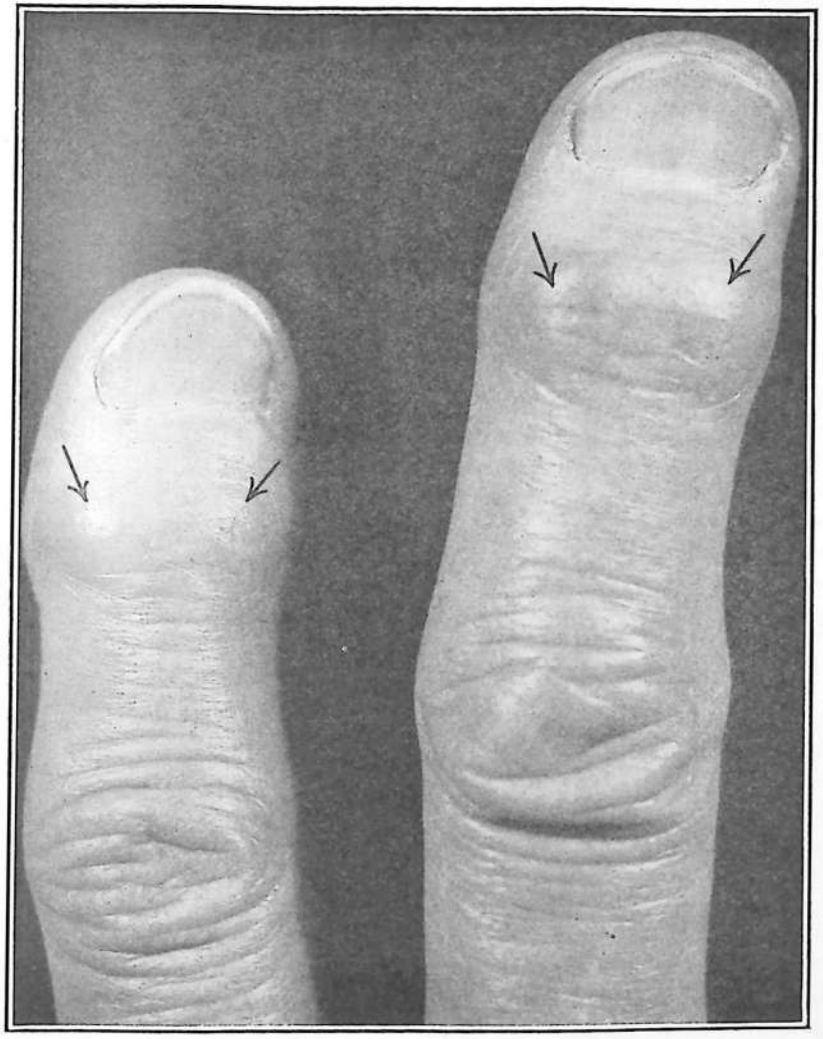

FIG. 119.—Close-up of Heberden's nodes affecting index and middle fingers.

Heberden's nodes are *frequent among* members of the *clergy;* in these individuals, they are usually found on the right and left index fingers,

possibly because of their large amount of writing, the right index finger holding the pen and the left holding down the paper.[11]

In addition to the swelling, flexion of the terminal phalanx may occur and, in a late stage, one may see lateral deviation of the terminal phalanx to either side. Heberden's nodes must be distinguished from flexion of the terminal phalanx associated with rupture of the deep extensor tendon and from hereditary symmetrical deviation of the little fingers. Clubbed fingers are not difficult to distinguish as the enlargement is confined to the terminal phalanx and is greater at the end of the phalanx than at the joint; the nail is also usually enlarged and often parrot beaked.[70]

Traumatic Heberden's nodes may follow a specific injury within several months, resulting in a painless deformity. Traumatic nodes are common in males at early ages, even in the second decade. Idiopathic Heberden's nodes are rare early in life and increase with age. They are permanent deformities, once developed.

Heberden's nodes usually worry patients more because of the unsightly deformity than any great degree of discomfort or disability.

The traumatic nodes, common in men, are usually confined to one hand or to one finger. Idiopathic nodes usually begin in one finger without trauma and spread until all fingers may be involved. Idiopathic nodes are most common after the age of sixty. Factors which have a definite influence upon the etiology of Heberden's nodes include race, sex, age and heredity; other agencies which may be influential include impairment of nerve function, as well as nutritional, circulatory, endocrine, thermal. environmental and toxic causes.[71] In a study of 68 families of patients with idiopathic Heberden's nodes, Stecher found that the mothers of these subjects were affected twice as frequently and the sisters three times as often as the general population.

They are distinguishable from gouty tophi in that the former are immovable as they are attached to the underlying bone, while tophi are usually movable; within tophi, one can usually demonstrate the presence of crystals of monosodium urate.[13] Heberden's nodes may be tender on pressure, especially if the nodes develop rapidly. Dawson has differentiated them from osteophytes elsewhere in the body in that these nodes develop just distal to the articular margin, although osteo-arthritic changes are present in the joint.

Degenerative Knee Joint Disease.—The knee is usually the most troublesome joint in the patient with degenerative joint disease. Clinically, this seems to begin in the fourth or fifth decade; it is occasionally seen in younger patients with severe trauma to the knees or excessive stress and strain to these joints. These patients are usually overweight, sometimes markedly so.

Degenerative knee joint disease may also follow occupational trauma, weak feet,[36] genu valga or vara, fracture or dislocation of the patella, tabes dorsalis or syringomyelia, traumatic hemorrhage into the joint or depressed fracture of the tibial condyle.

The *most important symptoms* are **stiffness** and **pain** and **crunching on motion** (especially flexion of the leg). Such stiffness or pain may occur on attempting to rise, particularly after sitting in one position, on arising in the morning or on ascending or descending stairs. Motion of the joint usually results in a diminution of the stiffness. There is also pain and stiffness in the structures around the joint (muscles, tendons and ligaments). Pain may be elicited by pressure upon the lower margin of the

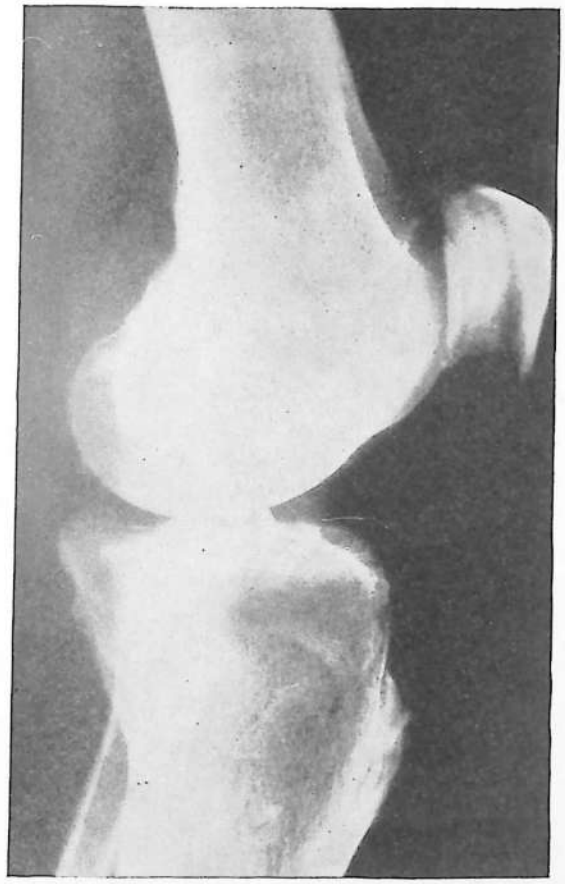

FIG. 120.—Early stage of osteo- (hypertrophic) arthritis with slight bone production. No narrowing of joint space. (Doub, courtesy of Radiology.)

patella or upon the line of the joint, especially on the mesial aspect. Pain and tenderness are common in the anterior knee region, usually centering about the patella. Passive motion of the patella is also usually painful.

Local puffiness or bogginess of the joint is often noted. A crunching sensation can usually be obtained by holding one hand over the dorsal aspect of the knee while the knee is bent; one often finds crepitation under the patella. Very little effusion is usually present. Surrounding muscular

spasm and atrophy is uncommon, and marked deformities of the joint are unusual.

There is at times a superimposed rheumatoid arthritis in these patients resulting in a mixed form of arthritis.

Osteo-arthritis of the Spine.—Hench found that the *lumbar spine is the commonest site of degenerative joint disease in men* (although second to Heberden's nodes in women).[29] Lipping of the articular margins and spur

FIG. 121.—Later stage of osteo-arthritis with more extensive bone production and beginning absorption of cartilage. (Doub, courtesy of Radiology.)

formation (osteophytes) may be seen by roentgen-ray. It has been shown that this *often follows degeneration of the intervertebral disks.* With this latter lesion, there occurs an increased motion of the vertebræ upon each other, which may be followed by stiffness of the spine and decreased motion when fibrosis of the disks has occurred.[65] *Thinning of the intervertebral disks (commonly found in the lower cervical and lumbar regions) together with marginal osteophytes, may produce a radiculitis resulting in pains radiating*

down the arms or legs. Irregular bony excrescences are formed along the borders of the vertebræ when the vertebral edges have become flattened from increased pressure upon them, subsequent to a decrease in size of the disk. Such bony excrescences have been produced experimentally by allowing a portion of the nucleus pulposus to escape following puncture of the intervertebral disks.[40]

The *main symptoms of degenerative spinal joint disease* include localized or radicular pain, stiffness, limitation of motion and muscular spasm. Coughing, sneezing or straining at stool may result in pain radiating along the nerve trunks which are subjected to irritation by the arthritic lesions as these nerves leave the intervertebral foramina. In arthritis involving the lumbar spine, the pain may be localized or may radiate down the thighs or legs.

In any patient with pain in the head, neck, chest and upper extremities, one must consider the possibility of **degenerative joint disease of the cervical spine.** The latter diagnosis is more likely if the pain is aggravated by motion of the cervical spine, especially by hyperextension, or by straining, sneezing or coughing.

Pain is produced in degenerative joint disease of the cervical spine by compression of the nerve roots which results from bilateral or unilateral narrowing of the intervertebral foramina through which these pass; thinning of the intervertebral disks is one of the common causes of such narrowing of the foramina. The foramina of the lower cervical vertebræ are small in comparison to the size of the nerves which emerge from them; in addition, the lower cervical vertebræ are the most vulnerable to trauma.[37]

Because of the varied and numerous ramifications and interconnections of the cervical nerves, pain in many regions may result from joint disease in the cervical vertebræ. The latter may cause unilateral sore throat, earache, occipital headache, pain simulating coronary artery disease, etc.

As a corollary, one must remember that *extensive hypertrophic changes may occur in the cervical spine without the production of symptoms.*

Nachlas showed that *involvement of the lower cervical spine may be associated with thoracic pains, simulating angina pectoris,* in some cases. He has pointed out that if osteophytes are present encroaching upon the cervical foramina, or if these foramina have been decreased in size for other reasons, there is such little clearance around the cervical roots passing through them that these nerves will be compressed if the neck is tilted in the proper position. In patients with severe arthritic involvement of the cervical spine, acute hyperextension and lateral bending of the head may produce a stabbing pain referred either to the precordium or beneath the scapula.

In these individuals, there may be a history of a strain, especially with the head in an unusual position, such as a sudden lurch to catch a falling child when the head is partly turned away. In some individuals, occupational strains may be responsible. These patients may show a lateral deviation of the cervical spine with frequent muscle spasm, involving the trapezius or splenius capitis; the taut muscle usually stands out prominently

when compared to the similar muscle on the opposite side. There is restriction of motion of the lower cervical spine. *Pressure over the lateral articular processes on the affected side characteristically produces pain;* some tenderness may also be observed over the spinous processes at times.

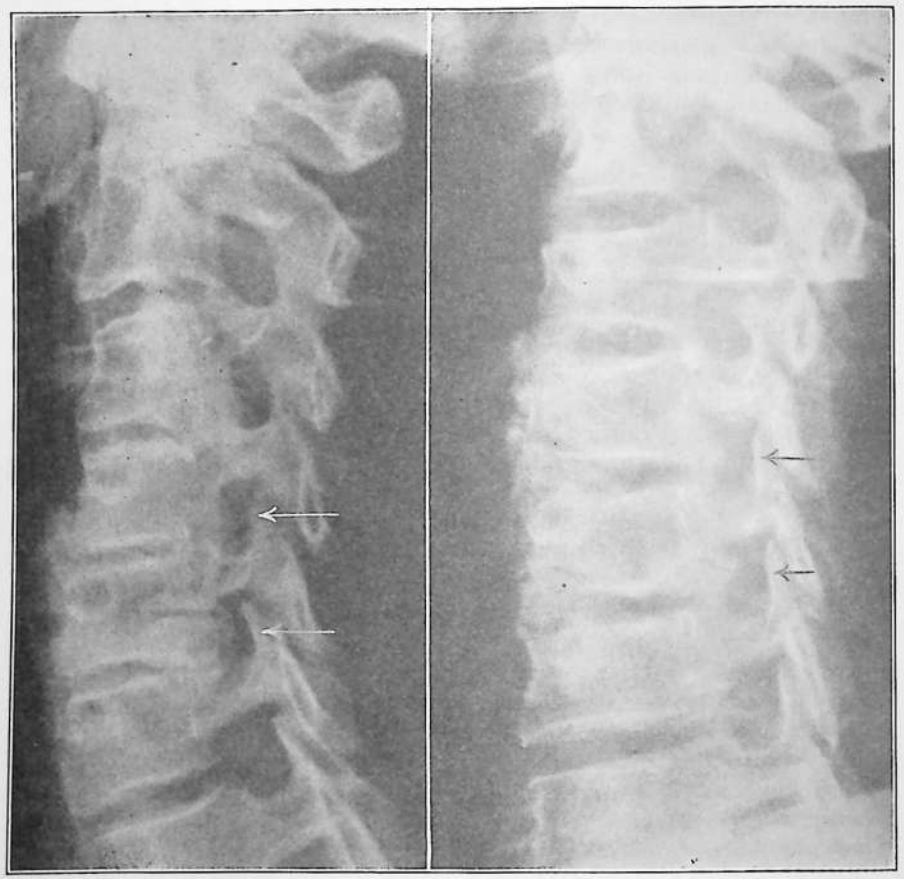

FIG. 122.—Patient aged fifty-three years. Hypertrophic changes involving the intervertebral foramina of the cervical spine, especially in the mid-cervical region. These changes show particularly in the oblique views. The hypertrophic changes are manifest on the posterior portions of the bodies of the vertebræ. Note especially the narrowing and altered shape of the intervertebral foramina.

Nachlas emphasized that pains of cervical spinous origin are not likely to be substernal, are not precipitated by emotional stress and are not brought on by dietary indiscretions.[51]

In arthritic involvement of the cervical spine, mechanical factors soon limit the range of motion of the neck. Pain may occur at first only on movement of the head and neck in certain positions; however, pressure on the cervical nerves may sooner or later produce a radicular type of pain. This pain may be more pronounced on one side than the other.

Muscular stiffness and pain about the neck and in the shoulder girdle, with or without radiation of pain down the arm are relatively frequent in individuals past forty years of age. In over 60 per cent of these patients, roentgen evidence of degenerative joint disease of the cervical spine can be found if adequate films are made, including oblique views.[48] Older writers had attributed these symptoms about the neck and shoulder girdle to arthritic changes in the vertebral column.[2,5,24,47,72] Others have recently described radiculitis with rigidity of the neck, pain and sensory and motor disturbances as a manifestation of irritation of the cervical spinal nerve roots due to cervical arthritis.[26] Turner and Oppenheimer observed roentgenologically narrowing of the intervertebral disks which they considered to be of extreme importance in this syndrome.[73]

In a few instances, bony spurs have been found on the posterior aspects of the cervical vertebræ, projecting into the spinal canal and causing pressure on the cord or encroachment on the cervical foramina.[52] It should be emphasized that **oblique views of the cervical spine are essential to demonstrate adequately the intervertebral foramina.**

Mettier and Capp examined in detail 30 patients with symptoms of **cervical radiculitis.**[48] Only 3 of these individuals were under forty years of age, most of the group ranging from forty to sixty years. The most frequent symptoms included pain, rigidity of the neck and muscular weakness of the arm or hand. In most cases, the onset of the pain was abrupt; this was often first noticed by the patient on awakening in the morning. In over half of the cases, the pain was localized in and about the shoulder, particularly about the insertion of the deltoid muscle; radiation of pain down the arm into the fingers was frequent. These symptoms usually appeared some months or even years before the patient complained of any discomfort in the neck. The pain was usually unilateral at the onset, but usually became bilateral as the disorder progressed. The pain varied in nature from a dull aching sensation to one of an excruciating character. In some, it was described as a stinging or pricking feeling while others noted sharp, jabbing, needle-like or penetrating pain. In a few, the only symptoms were a dead or drawing sensation or an aching in the muscles of the arms, or numbness and tingling in the fingers.

These individuals frequently complained of inability to comb their hair or extend the arm behind the back to adjust clothing. Some could not perform fine movements as writing or sewing; others were unable to grasp objects without difficulty.

Pain in the neck was a late development in most of these patients; a few became conscious of pain in the neck early in their illness. None of the patients noted definite restriction of movement of the neck and head, but pain in the neck was often complained of when the body was jarred. Some of these individuals described their discomfort as a muscular soreness, a feeling of congestion at the base of the brain or a headache at the base of the brain.

Reflexes of the arms were normal and there was no muscular atrophy in the shoulder girdle, arms or hands.

Roentgenograms showed *marked hypertrophic changes in the cervical vertebræ* in 18 of these 30 patients. The sites of predilection were between the fifth and sixth and between the sixth and seventh cervical vertebræ. The changes included circumferential osteophyte formation projecting into the intervertebral canals from the superior or inferior margins of the vertebræ, causing from slight to marked narrowing of the intervertebral foramina, varying with the size of the bony spurs. The changes were more common on the left side. The intervertebral disks were thinned in the region of these exostoses resulting in narrowing or in some cases, almost complete obliteration of the intervertebral spaces. When small osteophytes were seen, the disk cartilage adjacent to these was not necessarily thinned.

In an additional 7 patients, roentgen evidence of osteophyte formation was minimal and was accompanied by narrowing of the intervertebral disks in only 3 patients. Most of these patients showed some sclerosis of the articular facets.

Four patients with radiculitis showed no apparent abnormality of the articular facets by *x*-ray study and no evident new bone proliferation at the margins of the vertebræ. In these individuals with symptoms of radiculitis, there was no noticeable narrowing of the intervertebral foramina or thinning of the intervertebral disks.

The intervertebral foramina may be decreased in diameter by ingrowth of marginal osteophytes; in addition, its vertical diameter may be decreased by the close approximation of the vertebral bodies from thinning of the intervertebral disks. As a result of these two processes, the nerve trunk may be impinged as it passes through the involved foramina or one may note an effect of the sympathetic fibers controlling blood flow.

In a high percentage of cases, the sensory fibers are involved without evident involvement of the motor fibers of these cervical nerves. This may be due to the fact that the motor fibers of the anterior cervical nerves are fewer in number and form a bundle only about one-third the size of the posterior sensory fibers.

These patients with degenerative joint disease of the cervical spine are usually inadequately treated and wander from physician to physician in an effort to obtain relief. The majority of these individuals can be relieved by a program of therapy which includes some form of heat to the neck, massage, traction, postural training, and in some cases, if properly performed, manipulation.

Dorsal root pain *may simulate attacks of angina pectoris.* The referred pain or root irritation in **degenerative joint disease of the thoracic spine** may be projected around the chest or in some cases may be confined to a spot on the anterior chest wall.[25] These pains may be paroxysmal and occasionally may be noted simultaneously in one or both arms. Anterior thoracic pain may be provoked by certain movements or quirks of the neck in patients with cervical degenerative joint disease.[51]

It is very important that one differentiate between the pain of angina pectoris and the referred pain of arthritic involvement of the spine.[15,56] Dorsal root pain simulating angina may become pronounced when the patient with degenerative spinal joint disease is reclining.[76] In some instances, patients with cervical ribs or scalenus anticus syndrome may develop root pain which closely resembles angina.[64]

Smith and Kountz observed 15 patients whose pain (of dorsal root origin) simulated angina.[67] They pointed out the difficulty in recognizing pain attributable to the vertebral column as the symptoms may resemble angina very closely. In patients with dorsal root pain, the pain may occur on exertion, may be of short duration occasionally, and may be partially relieved by nitrites in some cases; these findings simulate a cardiac origin for the pain. However, in addition to the precordial distress which may occur in spinal degenerative joint disease, most of these patients also describe sharp pains in the back or elsewhere in the chest occurring together with the precordial distress. In addition, the duration of the episodes of pain are usually much greater in spinal joint disease than in angina pectoris.

Oppenheimer believed that segmental neuritis developed from crushing of nerve roots due to thinning of the intervertebral disks and collapse of the intervertebral foramina.[54] Others have also reported that osteophytes frequently occur about the transverse processes, spines and foramina, with resulting irritation in some instances of nerve roots.[12,22,44,50] However, in most instances, Key feels that there is insufficient osteophytosis in the vicinity of the nerve roots to account for the dorsal root irritation.[39]

In Smith and Kountz's series, *postural defects were evident in all of the patients.* They attempted to determine (by a study of the spinal nerves *in situ*) whether chronic deformities of the spine and thoracic cage might exert traction on the nerves with a resulting stimulation particularly of the sensory portion. In order to determine this, they used a cadaver, with the spinal cord and nerves exposed, in which they could produce abnormal degrees of spinal flexion and extension. It was observed that when the spine was either flexed or straightened to an abnormal degree, the spinal canal tended to become elongated and the cord was drawn cephalad. This imposed tension on the spinal nerves, especially at their angulations through the spinal foramina and at their attachments to the cord. They postulated that movement imposed on the nerves under such tension might give rise to irritation of the nerve fibers with the production of referred pain.

Arthritic changes in the thoracic spine may result from poor posture which is often occupational.[69] In some women, the undue strain of supporting very large, pendulous breasts may lead to undue strain on the thoracic spine.

In the lumbar region, degenerative joint disease is often related to old strains and injuries, altered mechanics in weight bearing in the middle aged (at times due to abdominal adiposity) and lack of exercise, leading to loss of tone in the back muscles. The symptoms of degenerative joint disease of the lumbar spine may be simulated by carcinomatous metastases, fibrositis, herniated disk, traumatic strains, tuberculosis, osteomalacia, spondylolisthesis or structural or referred back pains.

Degenerative Disease of the Sacro-iliac Joint.—Anatomic changes in the sacro-iliac joints, varying from alterations at the borders of the joints

to ankylosis, have been found with increasing age.[60,77] Strong ligamentous attachments normally prevent any measurable degree of motion at the sacro-iliac joints. Symptoms may appear when relaxation of these ligaments permits motion of the joints and stretching of surrounding structures. Symptoms and signs of sacro-iliac arthritis include:[36]

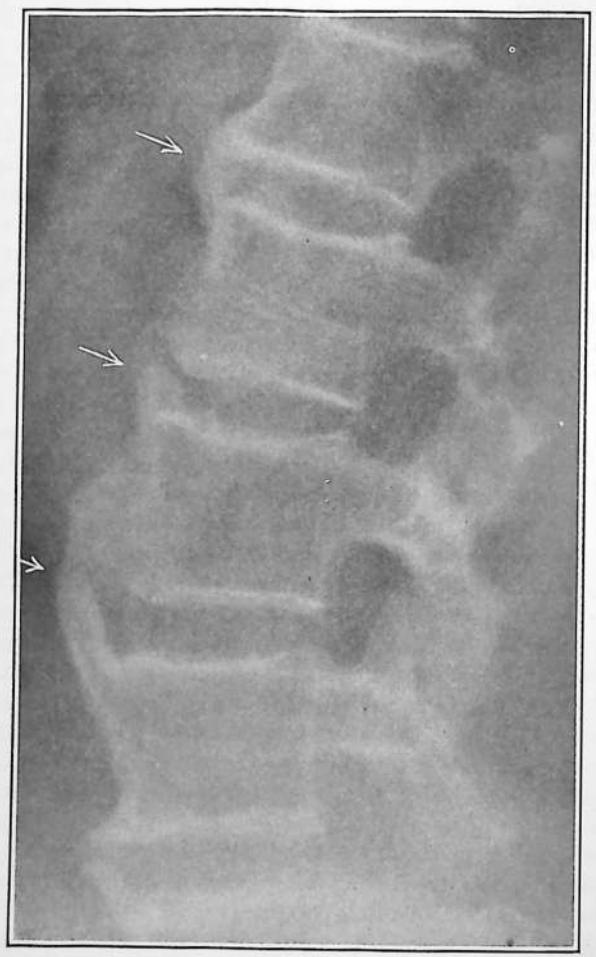

FIG. 123.—Marked hypertrophic changes in the lumbar spine.

Low backache and *pain*, aggravated by standing, by movements of the leg which cause motion in the sacro-iliac joint or by rising from a recumbent position.

Pain down the legs, in the groin or in the back.

Localized muscular spasm with the concavity of the resulting lateral spinal curvature toward the affected side.

In some cases, there is no pain on pressure or percussion over the joint.

There is usually limitation of motion and pain in the low back when forward bending is attempted from the standing position; forward bending from the sitting position (with knees semiflexed) causes no pain. This latter position relaxes the tension of the hamstring muscles. (In lumbosacral joint lesions, forward bending from either sitting or standing position causes pain.[68]) Pain is also felt in the sacro-iliac joint following straight leg raising or compression of iliac crests.

The pain of sacro-iliac arthritis may be relieved by immobilization of the affected joints, thus relaxing the ligaments.

Degenerative Hip Joint Disease (Malum coxæ senile; Morbus coxæ senilis).[21]—This condition has formerly been referred to as degenerative arthritis, morbus coxæ senilis, hypertrophic arthritis, osteo-arthritis, senile arthritis, arthritis deformans, etc. The *essential pathology* is degeneration of the articular cartilage, associated with or followed by changes in the underlying bone. The primary form of degenerative hip joint disease usually causes symptoms in older individuals, past the age of forty-five or fifty; however, it is probable that minor degenerative changes are present in the articular cartilage and other articular structures at a much earlier age. It is the most disabling variety of degenerative joint disease; fortunately, it is not one of the more common clinical sites of degenerative joint disease. It is unilateral in about two-thirds of the cases and is more frequent in men than in women, and especially in laborers and athletes.

The **main symptoms** are an insidious onset of *pain in the hip, groin or knee; stiffness in the hip* followed by *a limp on motion*, and *pain, especially on motion*, relieved by rest. It is important to remember that the *pain of hip joint disease is often referred to the groin or knee.*

Degenerative hip joint disease should always be suspected in patients complaining of sciatica, or pain in the lumbar region or even in the knees. Pain and disability are the outstanding symptoms. The pain is usually noted in the groin or along the inner aspect of the leg, although it is at times referred to the buttock or sciatic region. The degree of pain varies widely and does not always depend upon the amount of bony change.[10] There is often an increase in the discomfort following sitting or resting in bed; in some cases relief may be obtained by mild exercise. *Violent exertion may aggravate the condition.* Pain on arising from a chair is usually accompanied by restricted mobility of the hip. Pain occurring at night may deprive the patient of sleep. Severe backache may result from the compensatory lordosis which follows fixed flexion. Abduction and rotation become markedly limited.

On **roentgen examination**, degenerative hip joint disease is diagnosed by the *narrowed or absent joint space* (signifying disappearance of cartilage), *sclerosis of bone* and *marginal exostoses.* The decrease in joint space is especially evident in the weight-bearing portions of the femoral head and acetabulum. Inasmuch as *only the exostoses at the superior and inferior portions of the joint are visualized*, the abundance of these outgrowths is usually not appreciated in the roentgen study. For this reason, the roentgenologist must be especially careful in the wording of his report. A care-

fully worded roentgen report would mention the findings, but conclude with the statement that other exostoses may be present but cannot be seen roentgenologically because they may be hidden by shadows covering the anterior and superior portions of the joint. The roentgenogram will be especially helpful in pointing out a previous disorder in the hip in cases of so-called secondary degenerative joint disease.

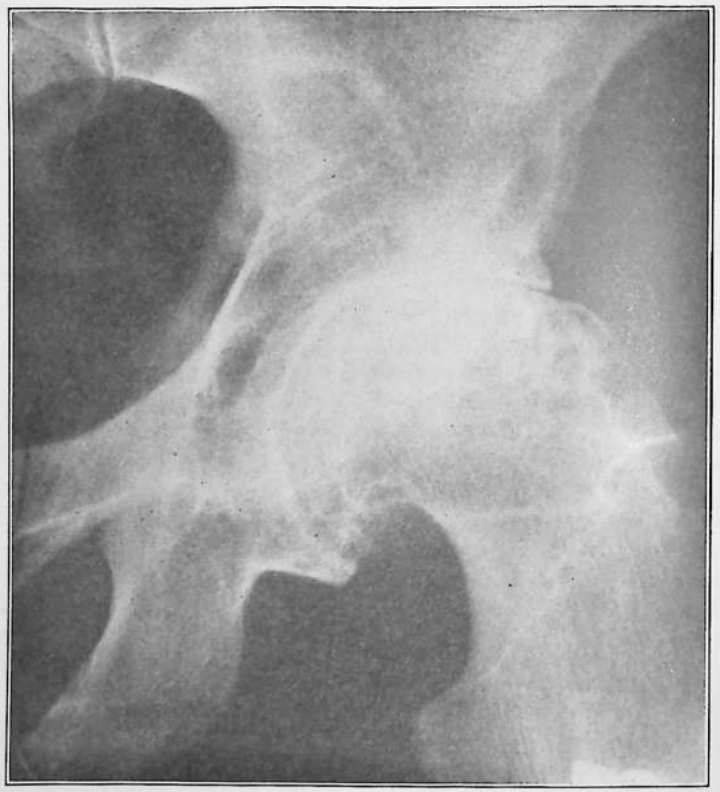

FIG. 124.—*Degenerative hip joint disease.* Note almost complete loss of articular cartilage, flattening of the femoral head and small cystic areas in the head and neck of the femur. Subchondral bone is condensed.

Exostoses are found in 45 per cent of cases; narrowing of the joint space with exostoses in 50 per cent; slightly narrowed joint space without bony atrophy or exostoses in only 5 per cent; small cystlike cavities in the juxta-articular bone, especially in the superior lip of the acetabulum and adjacent surface of head of femur.[6] Eburnation and erosion are indicated by sclerosis of subarticular bone and small areas of irregularity of the joint surface.

Flattening of the head of the femur may result in a mushroom appearance over the neck of the bone. The head of the femur may be displaced upward and outward and actual subluxation may occur.

Harmon has described a *secondary form of degenerative hip joint disease* occurring in individuals two or three decades younger. In his series of 94 patients, the mean age of the secondary cases was forty-five years, and of the primary cases, sixty-four years. There were no essential differences pathologically between the hip joints of the primary or secondary group.[27]

Various authors have enumerated the conditions that may predispose to degenerative hip joint disease Plewes studied a series of 242 cases and found preëxisting hip joint disease in over half of the cases (as trauma, adolescent slipped epiphysis, coxa plana and the Otto pelvis—protrusio acetabuli).[59] Wiberg also discussed the occurrence of degenerative hip joint disease in individuals with dysplastic acetabula and congenital subluxation of the hip joint.[75] Gilmour found that degenerative hip joint disease occurred in practically every case of protrusio acetabuli.[19,20]

Others have detailed the changes occurring in bone and joint cartilage following avascular necrosis.[41,57,58] Harmon felt that *avascular necrosis*, occurring *in the femoral head* of individuals of middle age, was the major joint insult which initiated the further progress of degenerative changes in the hip joint. The same sequence of events can be initiated by circulatory changes in the femoral head which follow traumatic dislocation of the hip.[42] Similar changes have been shown to occur (experimentally) in the hip joint after denervation.

Harmon thought that in some instances the subchondral osseous infarction was the initiating factor in the joint disease while in other instances, degeneration of cartilage occurred first. In all cases, the **end results** were similar: *degeneration of cartilage, sclerosis of subchondral bone*, and *marginal* chondro-osseous overgrowths (*osteophytes*).

Some of the causes which Harmon listed as *etiologic factors in secondary degenerative hip joint disease* were: coxa plana (Legg-Calvé-Perthes disease); slipped upper femoral epiphysis (adolescent coxa vara); congenital dislocation of the hip; congenitally inadequate acetabulum; congenital anomalies in the upper part of the femur; injury; fracture of the pelvis into the acetabulum; fracture of the neck of the femur followed by avascular necrosis of the femoral head (with and without the use of foreign body fixation); traumatic dislocation of the hip followed by late avascular necrosis of the femoral head; protrusio acetabuli; Paget's disease of the pelvis and upper part of the femur; chrondromatosis and osteochondritis dissecans of the hip joint and Charcot joints.[17] He also included some cases with a preceding rheumatoid arthritis or septic arthritis in childhood or adult life.

The majority of these patients in both the primary and secondary groups were in unusually good general physical condition.

Harmon considered degenerative hip joint disease to be a "wear and tear" effect upon one of the specialized structures of the body, with the added factors of preëxisting disease, local tissue damage and continued insults to joint structures. He stressed the rôle of trauma, either a single severe episode (sometimes resulting in fracture) or minor repeated trauma of occupational or everyday activity, in addition to a hereditary pre-

disposition to cartilage injury and/or degeneration.[46] About one-third of his patients were obese; obesity could increase the strain upon the joints of the lower extremity.

When *damage to cartilage* has been produced, a vicious cycle of *added trauma* and destruction of cartilage goes on until the *underlying bony cortex* may be *bared*, with resulting *changes in the subchondral bone*. Harmon stated that rheumatoid or purulent arthritis could produce damage to the articular cartilage, which was, in some instances, the forerunner of later degenerative hip joint disease. He commonly found wedge shaped sequestra produced in the femoral head by avascular necrosis (one-third of the cases) and suggested that this had probably initiated the joint damage in those cases.[27] No bacteria were cultured from any of the affected hip joints. There seemed to be no important endocrine abnormality, except the obesity (which was postmenopausal in some cases). Juxta-articular adiposis dolorosa was not a factor in degenerative hip joint disease; this condition has been reported in from 10 to 15 per cent of degenerative joint disease, particularly involving the knees in obese multiparous women past middle age.[43]

When the surgeon dislocates the femoral head in a patient with degenerative hip joint disease, he sees a varying degree of degeneration of the articular cartilage—with some gross ridges or pleats, alternating with or adjacent to bare spots of subchondral bone. In these hips, the surface of the *femoral head may resemble the pitted surface of a golf ball*, with similar changes being seen on the acetabular side.[27] The typical exostoses are evident immediately.

When cartilage and bone are removed, as in an arthroplasty of the hip, the amount and extent of anemic, avascular bone in the femoral head may be marked. This avascular bone is often wedge-shaped or conical with its base upward. The weight-bearing portion of the femoral head and the acetabulum show the most pronounced gross and microscopic changes.

Harmon has described loose osteochondral bodies, thickened joint capsule, free joint fluid, and hypertrophic reddish-blue synovia in these joints. It is probable that in the primary degenerative hip joint disease, very little fluid will be noted. In his patients, the fluid was clear or slightly cloudy and straw-colored, and thick. When the surgeon dislocates these hips at operation, he is struck by the *increased size of the osseous portion of the femoral head*, which occurs *at the expense of the cartilage over the femoral head and in the acetabulum.*

Grossly, the first changes noted in these joints are a change in color and increase in translucency of the articular cartilage. Fibrillation of the cartilaginous matrix then occurs, with pitting of the surface, and at a later stage, ulceration and cavitation of the cartilage. The destruction of cartilage may be so large that the subchondral bony plate is bared in some places. The bony trabeculæ become thickened under the areas of cartilaginous degeneration; the bony subchondral plate becomes thickened. Sclerosis of subchondral bone occurs in some areas. At the margins of the joint, proliferated bone pushes cartilage before it leading to the formation of chondro-osteophytes.

These **exostoses** probably are responsible for the marked limitation of motion (abduction, rotation and flexion) which is so common in the later stages of degenerative hip joint disease. Unfortunately, the exostoses are not well visualized by even stereoscopic *x*-rays as they do not have a well-developed osseous cortex.

A large exostosis is usually found on the superior and anterior portion of the femoral neck. In some cases, this exostosis of the femoral neck may be very large and may completely bridge the depression between the head of the femur and the greater trochanter, forming a ledge articulating with marginal exostoses from the acetabulum; this bony block of exostoses prevents rotation and abduction.[27]

Another large osteophyte is often found on the inferior and mesial aspect of the femoral head; as the adduction deformity increases, this osteophyte becomes larger and more irregular. It also may articulate with a projection around the acetabulum and prevent flexion, rotation and adduction.

In addition to the above mentioned osteophytes, one may see a rim of osteophytes around the acetabulum projecting anteriorly and posteriorly, arising in the insertion of the capsule. *On roentgen examination, one does not fully appreciate the abundance and complexity of the exostoses* and their effect upon motion as one visualizes only the exostoses at the superior and inferior portions of the joint, the remaining two-thirds of the head of the femur and margin of the acetabulum (the anterior and posterior portions) being covered by other shadows.[27]

The subchondral bone becomes exposed as the cartilage degenerates. The *osseous articular cortex* becomes tremendously thickened (*eburnated*) and acquires a *highly polished* surface from the effects of friction. Deeper within the bone, *cystlike cavities* containing fatty tissue may appear.[6]

Degenerative Disease of the Rib Joints.—Goldthwait has emphasized the importance of strain, irritation or disease of the rib joints as a cause of trauma to adjacent nerves resulting in referred pain around the chest or abdomen.[23] The 24 costovertebral joints are formed by the junction of the heads of the ribs with the side of the spine. In addition to these, there are 20 costotransverse joints which are formed by the articulation of the angles of the ribs with the outer parts of the transverse processes of the upper 10 dorsal vertebræ.

Each of these 44 is a true joint; all are involved in the mechanism of the thorax and may be injured or diseased. *The rib joints differ from other joints of the body in that they never rest during life* unless they are ankylosed as a result of disease. The range of motion of the rib joints is such that there is a difference in chest circumference of 3 to 5 inches between full inspiration and expiration in normal individuals.

At the level of each costovertebral joint, the intercostal nerve leaves the spine and passes along the rib. Irritation of these intercostal nerves at the costovertebral joints results in many cases of pain referred to the chest wall or around the body at different levels. The circulatory disturbances often seen in connection with irritation of these joints may be

due to the fact that the ganglia of the sympathetic nervous system lie adjacent to the costovertebral joints.

At times, one may have a true strain of these joints with actual displacement of the head of the bone following a definite wrench or injury. A rheumatoid arthritis may go on to complete ankylosis of these articulations resulting in absence of chest motion during respiration; when ankylosis has resulted, no pain will occur because there will be no motion of the joint.

The two floating ribs on each side are not supported in front by costosternal cartilage; having no costotransverse articulation to limit motion of the main joint, they may sag and move to such an extent that a greater strain is placed upon the rib joint with a greater possibility of irritation of the nerve root and trunks (*i. e.*, eleventh and twelfth intercostal nerves). Irritation of the nerve roots adjacent to these ribs is referred to the sides of the abdomen from the pubis to the crest of the ilium (the so-called intercostal neuralgia).

The best position for rest of the rib joints is midway between extreme expiration and inspiration. In the normal chest, the average downward inclination of the ribs below the horizontal is 30 degrees. In poor posture, the chest sags, the head and shoulders are rounded forward and the ribs hang low, producing a strain of the muscles and ligaments which are out of place, which, if continued, produces weakness and joint irritation.

As a rib sags, the costotransverse articulation is used as a fulcrum and the rib is twisted resulting in an abnormal rotation of the head. Long maintenance of faulty positions may lead to irritation and true arthritic involvement. A nerve leaves the spine just at the level of each vertebra adjacent to the head of the rib, so that rib joint disease can easily lead to irritation of these nerves.

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CHAPTER XXXIX

TREATMENT OF DEGENERATIVE JOINT DISEASE (OSTEO-ARTHRITIS)

ONE of the most important points in the treatment of degenerative joint disease is **reassurance to the patient that he will not progress to a stage of helpless invalidism** as may occur in rheumatoid arthritis. The condition is more of a nuisance than a calamity except when the hip joints are severely affected.[20] He must understand that, in general, degenerative joint disease is probably largely the result of the effects of age and trauma, and that there is at present no reliable method of stimulating resorption of the bony or cartilaginous projections which occur in this disease, and further that articular hyaline cartilage, when destroyed or damaged, cannot regenerate. These individuals must realize that their Heberden's nodes do not indicate an infectious process and that these will usually produce little inconvenience other than that of the cosmetic effect and slight discomfort. However, degenerative joint disease involves joints other than the fingers and much can be done to relieve this process and to prevent further damage.

It is important to differentiate this disease from rheumatoid arthritis because of the different treatment and prognosis in the two disorders. The program outlined on page 623 will, in general, aid in the handling of many of these patients; however, one must always remember that *he is treating the patient and not merely his disease.* The program must be individualized.

The **prognosis** for symptomatic joint relief is fairly good. The condition is not inevitably progressive, but when hyaline articular cartilage has been destroyed this does not regenerate completely.

Rest.—This may consist of local or general rest, depending upon the parts involved; in most instances, if large joints (such as the knee) are involved, the patient should rest in bed or upon a couch at least an hour each morning and afternoon; in a wage-earner, a period of rest should be obtained at noon, if possible. In either case, an hour's rest is prescribed after the evening meal. Most of these patients do not require complete rest in bed.

Unfortunately, many of these individuals are obsessed with the thought that they must exercise the affected parts continuously or these will become stiff; nothing could be farther from the truth. Excessive exercise results in additional trauma and damage to the involved parts; however, the joints should be moved through a full range of motion several times a day.

Avoidance of Trauma.—Any motions which tend to throw strain upon the affected joints should be eliminated insofar as this is possible. The physician must go into details with the patient concerning his mode of life, occupation, recreation, etc.; if any chronic strain is found, this must be corrected.

General Plan of Treatment of Degenerative Joint Disease

Adequate daily *periods of rest of the involved joints.*
Avoidance of trauma, strain or abnormal use of the affected parts.
Reduction in weight if obese and especially if the lower limbs are involved; adequate vitamin intake.
Physical therapy—*heat, massage* and *exercise* (properly administered).
Good Body Mechanics: elimination of faulty posture; proper support and exercises for the feet.
Orthopedic appliances: support to the back, hip, knees or feet if indicated; use of corsets, adhesive or elastic bandages, crutches, canes, caliper splints, etc., as required.
When necessary, relief of pain by analgesic drugs (aspirin and phenacetin).
Proper care of varicose veins in the legs, especially if the knees are involved; elastoplast bandaging or elastic stockings may be very helpful.
The use of desiccated thyroid substance if the basal metabolic rate is below normal or even when normal (in some cases).
Miscellaneous therapy: orthopedic operative procedures;[37,39] iontophoresis (with mecholyl or histamine); warm dry climate; roentgen therapy (?). Marked improvement does not occur in these patients following removal of focal infection, parenteral gold therapy, chaulmoogra oil, bee venom, vaccines, foreign protein therapy, artificially produced fever or sympathectomy; these measures are not indicated in degenerative joint disease.

Reduction in Weight.—Involvement of the knees is common in degenerative joint disease. Many of these patients are markedly overweight. This added weight may be a factor in causing foot strain and strain at the knee joint. Unless contraindicated, a low caloric (800 to 1500 calories) diet may be employed until the desired amount of weight is lost.

Amphetamine (benzedrine) sulfate has been used in some patients in reducing obesity from overeating. A dose of 5 mg. is usually given after breakfast; this is gradually increased to 10 mg. or even to 10 mg. after breakfast and after lunch. Benzedrine is omitted one week during each month of treatment. If mild constipation follows its use, this is usually relieved by mineral oil.[8] Benzedrine should not be given in the evening because it may cause sleeplessness.[22] Although amphetamine is used by some practitioners to aid in weight reduction, it is not employed for this purpose in our clinic.

The Council on Pharmacy and Chemistry of the American Medical Association went on record as disapproving general recognition of claims for the use of amphetamine sulfate (benzedrine) in controlling obesity. It was concluded that whatever effect the drug might have in this respect might possibly be due to undesirable properties.[4]

Physical Therapy.—Most patients with degenerative joint disease benefit symptomatically if periods of *heat, massage* and *exercise* are used. *One form of heat seems to be as effective as another* and the physician may employ baking, infra-red, short wave or conventional diathermy, hot fomentations, etc., as desired. *The effect of any of these is to produce an increased blood supply in the area treated.* Warm baths, therapeutic pools or spas often

aid by relieving pain and associated muscle spasm. **Heat** may be applied daily or several times a day if the symptoms are marked. Painful Heberden's nodes may be relieved by *bathing the hands in hot epsom salts* (approximately 1 ounce to each quart of water). *Warm paraffin applications to the hands* are often of marked symptomatic benefit in these cases. Detailed instructions in physical therapy, massage, exercises and orthopedic care have been presented in previous sections.

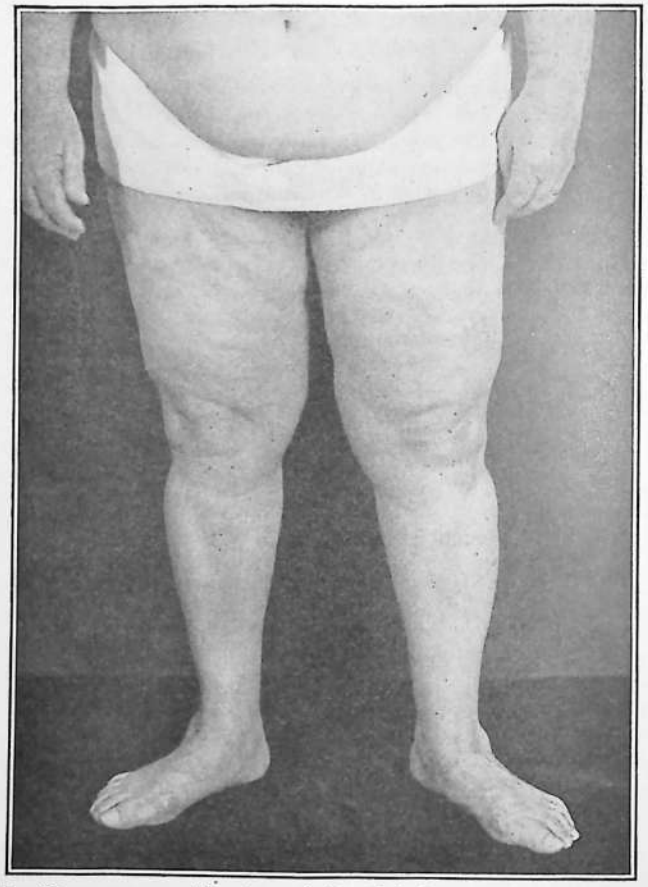

FIG. 125.—Obese woman with osteo-arthritis of the knees and pronated weak feet.

Following periods of heat varying from ten to thirty minutes, **massage** of the entire limb and of the involved joint is usually advised. Massage of the intervening muscles may be rather vigorous so that muscular atrophy does not occur; massage of the joint itself should consist of light stroking movements for the first several days. More pressure may be exerted later if pain and swelling of the joint are not aggravated. Care must be taken to avoid massage of the tender bony tissue covering osteophytes as such irritation may increase their size and sensitiveness. Most patients with

degenerative joint disease will ask for further heat and massage because of the symptomatic benefits.

Exercise of the involved joints is important to prevent the formation of fibrous adhesions in the neighborhood of the joint and to decrease the tendency to atrophy in the surrounding muscles. This must be performed in moderation and should not be so severe as to cause further injury to the joint. These patients should *not* constantly keep their joints on the move.[43] Excessive exercise may increase joint stiffness. Slight discomfort may occur during its performance. *Any discomfort which persists for more than one or two hours following exercise indicates that the motion has been too great, and must be curtailed.* Passive exercise may decrease stiffness but voluntary exercise is usually preferable in these cases in order to prevent further injury to marginal proliferations. Exercises which may be used are described elsewhere.

Snow has emphasized the rôle of physical therapy as an adjunct in the management of degenerative joint disease.[53] He employs *not too intense deep heat* to improve the circulation within the joint capsules and less penetrating forms of heat for reflex circulation and relaxation of muscle spasm. Numerous forms of heat are available, including radiant heat lamps, electric pads, hot soaks, contrast baths, paraffin baths, mud packs, etc. In addition, properly performed exercises, performed within the limits of pain and gradually increased, aid in reducing muscle spasm and in increasing joint movement. Snow believes that prolonged lack of movement in degenerative joint disease results in pain with pressure upon opposed joint surfaces where the cartilaginous insulation is gradually disappearing. If activity is carried out without weight bearing and is properly supervised, pain may be lessened.

In **degenerative cervical joint disease**, *diathermy*, *massage* and *graded exercises* are of assistance, accompanied by *manual traction or suspension* with gentle rotation to overcome the crowding of structures in the neck that causes pain from bony hypertrophy, muscular spasm and thinning of the intervertebral disks. One must always remember that if there is marked thinning of but one disk, in the presence of a history of trauma or acute snapping of the neck, one must consider the possibility of a herniation of a nucleus pulposus, requiring surgical intervention.

When the **thoracic spine** is involved, one must correct any poor posture which may be found. In overweight women with abnormally large, pendulous breasts, these structures must be supported properly to relieve some of the strain posteriorly.

Degenerative changes in the lumbar spine may be associated with altered mechanics of weight bearing in the middle aged, with abdominal adiposity, lack of exercise and loss of tone in the back muscles; in some cases, this is aggravated by old strains or injuries, or the degenerative changes may follow in a spine with asymmetrical or oblique unstable facets.

If the back is structurally weak, exercise may aggravate the symptoms and it may be advisable to prescribe a period of limitation of motion with curtailment of activities. Golf playing should be prohibited in these

individuals as it produces torsion strain to the lower dorsal spine and lumbar region. Straight leg raising exercises or passive manipulations of the lumbar spine should be forbidden if there are marked hypertrophic bony changes with marked thinning of a disk or disks.

Stout patients with abdominal sag may need high back corsets or braces, with arrangements for abdominal support. This added anterior pull must be relieved so that it is not concentrated on an overburdened, acutely angulated lumbosacral joint, but should be distributed over a wider longitudinal area of the back. In addition, a *firm mattress with fracture boards* under this, heat, graded massage and proper exercises are indicated in most instances, in addition to the back and abdominal support.

The patient with arthritis must realize that *physical therapy alone will not cure his arthritis* but that periodic courses of physical therapy, in addition to daily physical therapy at home, will aid in making possible the maintenance of the individual's activity.

In individuals with *involvement of the hip joint*, relief is often obtained by diathermy applied directly through the joint, in addition to massage (particularly of the adductors of the thighs), exercises done periodically several times per day and passive stretching of the hip joint and fascia of the thigh in an effort to maintain motion.

Patients with involvement of the hip or knee joints must be viewed as potential invalids and the physician must employ every means at his command in an attempt to avert such a catastrophe. In obese individuals, reduction of weight will lessen the burden upon the hip and knee joints. Although it is difficult to obtain the early use of *crutches* in patients with mild symptoms, if used early, they may be very valuable in relieving the strain of added weight bearing on knees or hips. Canes are not usually advised because they tend to encourage the development of flexion deformity, causing the patient to walk with flexion of the hip and lumbar spine; in addition, they throw a marked strain upon the wrist and elbow joints.

Thyroid Medication.—Some patients with degenerative joint disease improve following thyroid medication. A considerable percentage of individuals with degenerative joint disease have a subnormal basal metabolic rate. The dose of desiccated thyroid must be varied with the individual; frequent estimations of the basal metabolic rate and blood cholesterol should be done during the course of thyroid therapy. Some rheumatologists frequently give these patients with a normal B.M.R. small doses of thyroid substance and believe that it has been helpful.[26] Such a practice is not recommended routinely as thyroid substance is a powerful drug.

Analgesic Drugs.—If required, the same combinations as used in the relief of pain in rheumatoid arthritis (*q.v.*) may be employed. Local counter-irritation (by methyl salicylate or analgesic balm) may produce hyperemia and temporarily relieve pain. *Narcotics are practically never required.*

Good Body Mechanics.—*Pains in the knees and low back are due, not infrequently, to improper weight-bearing lines caused by weak, pronated feet.* Faulty posture must be corrected and proper support and exercises must be prescribed for the feet, the pendulous abdomen, the lordotic lumbar

spine, etc. These are described in detail in the sections devoted to these subjects.

Dietary Advice for Patients Markedly Overweight

Obtain a B.M.R.; if subnormal, use *desiccated thyroid* substance beginning with 30 mg. (½ gr.), twice a day, and increase to 60 mg., 0.12 gm. or 0.18 gm. (1, 2 or 3 gr.) several times a day until the B.M.R. has been brought to within normal limits, or symptoms of hypothyroidism have been relieved. From this point, a maintenance dose should be given (varying from 30 to 120 mg.—½ to 2 gr. daily). If the B.M.R. is normal, do not employ desiccated thyroid as a routine procedure.

Eat None of the Following: Potatoes, corn, fresh peas, lima beans, macaroni, noodles, spaghetti, rice, baked beans, gravy, creamed soups, candy, cake, pie, ice cream, puddings, bananas, apples, prunes, nuts, peanut butter, preserves or jellies, cream, ham, pork, bacon, cheese (except cottage cheese), mayonnaise, cocoa, chocolate.

Eat All You Want of the Following: Lettuce, cabbage, sauerkraut, cauliflower, Brussels sprouts, broccoli, asparagus, tomatoes, watercress, radishes, celery, grapefruit, plain jello, consommé or bouillon, spinach, water, tea, coffee.

Eat Ordinary (Moderate) Portions of the Following: String beans, carrots, onions, pumpkin, squash, oranges, honeydew melon, strawberries, meats except as above (roast, broiled or boiled meats preferred; if fried, drain all fat on brown paper; cut all fat from meats before eating); cottage cheese; cooked cereals.

Eat One Slice of Rye or Whole Wheat Bread Each Day—Use Very little Butter: 6 small saltines may be substituted for the slice of bread if desired.

Drink One Glass of Skimmed Milk Daily.

Plan of reduction of weight: To lose approximately 4 to 6 pounds during the first week, 2 to 4 pounds the second week, and from 1½ to 2 pounds each week thereafter. Attempt to rid the body of 50 per cent of the excess weight during the first period of dieting; when this has been achieved, additional foods (20 per cent vegetables, bread, etc.) are allowed so that the patient maintains this weight for a period of four to six weeks—thus avoiding very rapid and sustained loss of weight. Following this rest period, an attempt is then made to lose 50 per cent of the remaining excess of weight. It is usually not advisable to produce a further reduction.

If the patient does not lose weight as predicted, he must keep an accurate account of all food eaten for a period of one week and submit this in writing; often, dietary indiscretions may be discovered in this manner.

Added vitamins and minerals are given if prolonged dieting is required.

Occasionally, older individuals react poorly to marked reduction in weight; great caution must be used in these patients—in some, the low caloric diet must be discontinued.

Other Drugs.—Vitamin B (*thiamin chloride*) has been used in patients with degenerative joint disease and has occasionally been followed by a decrease of joint pain and muscular aches, and increased sense of well-being. Five mg. may be given orally one or two times a day. In patients with marked discomfort, large doses (from 10 to 100 mg. or more) have

been given subcutaneously or intravenously every other day for several weeks. Subcutaneous injections produce a burning sensation for about thirty seconds. It is uncommon to find a deficiency of only thiamin chloride in an individual. In patients with degenerative joint disease it is preferable to use the whole vitamin B complex by mouth or parenterally.

Iodides have been employed in degenerative joint disease purely on an empiric basis: "they seemed to do good." Some physicians think that they may aid in stimulating thyroid activity. In the usual case 0.3 to 0.6 cc. of a saturated watery solution of potassium iodide has been used three times a day. The drug must be discontinued if pustular eruptions, coryza, gastrointestinal upset or other signs of iodism develop. As yet, there is no good scientific basis for the use of iodides in degenerative joint disease.

Constipation is frequently present. This may be treated along the lines suggested in a discussion of this subject in the section on Rheumatoid Arthritis.

Key and his colleagues have divided patients with degenerative joint disease into four groups: (*a*) *senile* (with arthritis beginning about sixty years of age, associated with changes in the heart, blood-vessels, eyes, kidneys, brain, etc.); (*b*) *adipose* (with arthritis beginning before the age of forty in many, and affecting weight-bearing joints as the knees, sacro-iliac joints and lumbar spine, and often associated with a girdle type of obesity, hypotension, and menstrual irregularities); (*c*) *thyroid deficient* (which is rare as a pure form); and (*d*) a *menopausal* type (with arthritis beginning before, during or after the menopause, associated with vaso-motor instability, hot flashes, dizziness, etc.).[25]

Many individuals have used procaine therapy in arthritis.[57] Falta and Fenz reported encouraging results in degenerative joint disease by infiltration of ligaments and muscles around affected joints. In hip joint disease, they injected the region of the adductors or the gluteal muscles.[9] However, results are not as dramatic as those which are seen following procaine injections in acute traumatic conditions (especially sprains and contusions).

The amount of procaine to be injected varies with the site of involvement, age of the patient, and the tolerance of the individual. From 5 to 30 cc. of 1 per cent procaine hydrochloride solution has been used, being injected at the site of maximum tenderness under sterile precautions. Procaine reactions are not uncommon, but are usually harmless. Outland and Hanlon reported that such reactions could be prevented by the oral administration of from 0.1 to 0.2 gm. (1½ to 3 grains) of sodium amytal orally before injection.[41] If the procaine injection is performed in the office, it is always advisable to have the patient lie down for thirty minutes following the first several injections because of the possibility of dizziness, faintness or nausea; individuals who are sensitive to procaine should not have repeated injections unless this is absolutely essential. In such cases, very small amounts of procaine should be used and the patient should be watched carefully. It is preferable in such instances to switch to another local anesthetic, such as metycaine. In all instances, the patient should

be warned that he may have increased pain several hours after the injection when the effect of the procaine has worn off; this increased pain may be due, also, in part to an increase in blood supply to the tissues infiltrated.

Miscellaneous Treatments.—*Injections* or *iontophoresis* with *histamine* or *mecholyl* may occasionally aid in reducing the local discomfort in degenerative joint disease. These procedures have been described elsewhere. In addition to producing an increase in blood supply to the involved joints, iontophoresis may be a valuable psychotherapeutic adjunct. Simpler forms of physical therapy are usually advisable.

Some workers have considered *roentgen therapy* to be useful in degenerative joint disease;[7,11,40,59] it is said to reduce congestion and lead to absorption of fibrous tissue.[45,46] Swaim found it to be of benefit especially in mixed forms of rheumatoid and osteo-arthritis.[56] Some investigators directed the roentgen therapy over the involved regions in doses of 100 to 150 r two or three times a week (for a total of from 3 to 12 treatments).

Massive doses of vitamin D have been recommended in both rheumatoid and osteo-arthritis.[10,29] Most workers are in agreement that there is no indication for massive doses of vitamin D in degenerative joint disease.

Removal of focal infection,[48] gold therapy, sulfur,[3] chaulmoogra oil, bee venom, fever and sympathectomy are not of value in uncomplicated cases of degenerative joint disease. About 5 per cent of reported cases have become symptom-free following fever therapy.[6,18] Some workers have found vaccines to be of benefit in degenerative joint disease.[34,47,62] Sidel and Abrams obtained subjective improvement in 86 per cent of these patients by weekly injections of physiologic saline solution plus small doses of salicylates; undoubtedly any form of injection may raise the hopes of these individuals and so have a profound psychologic effect.[49] Vaccines are not recommended in cases of purely degenerative joint disease. *Vaccines are no longer used in our clinic in the treatment of any form of arthritis.*

Heberden's Nodes.—Because of the *inability* of *hyaline articular cartilage to repair itself*, very little can be done to replace the destroyed cartilage or to "cure" Heberden's nodes. The *patient should immediately be assured that he does not have rheumatoid arthritis and that his disease will not go on to crippling and helpless invalidism*, but that "his pride will be hurt more than his joints." Inasmuch as trauma aggravates the process, the symptoms will probably be more annoying if additional trauma occurs.

Pain may be relieved in the early stages by *warm soaks* in water to which magnesium sulfate has been added, or by using other forms of *heat* such as paraffin dips, dry heat, contrast baths, etc. Roentgen therapy usually does not relieve this discomfort (at least not in the dosage used at present). Although the use of estrogenic substances has been advocated for relief of discomfort, adequate reports are not available on this point. The claim that the nodes can be reduced in size by the pressure of adhesive plaster bandages or metallic bands has also not been substantiated.[44]

Cervical Spine Lesions.—McFarland and Krusen have employed the *modified Sayre head sling* in patients with degenerative disease of the cervical spine. Degenerative changes lead to narrowing of the intervertebral

foramina with compression of the emerging nerve roots, leading to inflammatory changes; exaggerated extension of the neck widens the foramina and removes the pressure upon the nerves, allowing the inflammatory reaction to subside.

Patients with degenerative disease of the cervical spine may have no pain in the neck; they frequently complain of pain over the shoulder, down the arm and over the pectoral region and upper back, *i. e.*, pain anywhere in the distribution of the lower cervical nerves or brachial plexus.[35]

Before suspending the patient in the Sayre head sling, *radiant heat* is applied from a luminous source for thirty minutes, *followed by effleurage* of the neck and shoulder. The Sayre *head sling* is then *attached to an ordinary ice scale* in order to register the number of pounds of traction which is being applied. In the average adult, *from 70 to 80 pounds of traction is applied slowly.* When this amount of traction has been reached, the head is rotated gently to the right and then to the left, following which the traction is slowly released. The whole procedure is carried out twice a day for a week to ten days, following which the patient is instructed in the use of the sling at home. When the sling is applied and attached to overhead hooks, flexion of the knees of the standing patient produces firm, strong traction on the neck.

The *treatment of degenerative joint disease of the cervical spine* includes the use of rest, heat, traction to the head, manipulation in selected cases, analgesics, orthopedic appliances to the head and neck (including plaster collars, Thomas collars, etc.) and in some cases, roentgen therapy. *Procaine injection* may result in temporary or more prolonged relief of discomfort in some instances, but must be followed by the above measures.

Hip Joint Disease.—In a study of 183 patients with disabilities from degenerative hip joint disease (osteo-arthritis), *both hips* were found to be *involved in 114* and *one hip only, in 69.* It was found that *muscular and ligamentous strain* often produced the symptoms observed in this disorder. Strain, fatigue and instability follow when the weight thrust is changed and the function of the muscles about the hip is impaired with the increased forward inclination of the pelvis and flexion of the hip. Recurrence or aggravation of the disability is prevented by use of the hip in as nearly normal weight bearing as possible.[28]

Thirty per cent of the group were *completely relieved* of symptoms and an *additional 37 per cent were greatly improved by conservative measures.* Those patients with unilateral involvement and those with extensive flexion and adduction deformities showed the least improvement; in the latter group, particularly, surgical intervention is required at times.

If there is little deformity of the head of the femur, *manipulation of the hip* is often followed by improved motion. In the aged and debilitated, *roentgen therapy* may be of assistance in relieving pain.

Normally, the *weight of the body is borne at the hip joint by the central part of the head of the femur in its contact with the strong upper portion of the acetabulum.* In degenerative hip joint disease, forward inclination of the pelvis always occurs resulting in muscle action which is less efficient both for stability of the joint and for locomotion. Following this, weakness of the gluteus and other muscles develops and adduction and external rotation follow flexion of the hip joint. The latter result in ligamentous strain with proliferation of bone and cartilage about the hip.

The *earliest roentgen evidence of degenerative hip joint disease* is an increased density in the posterosuperior border of the acetabulum and the anterosuperior surface of the head of the femur. When this is present, motion of the hip joint is limited, although subjective symptoms are often lacking. The process is often present for several years before the appearance of symptoms. *Later roentgen changes* include osseous proliferation around the posterior edge of the acetabulum and the anterior aspect of the head and neck of the femur. The head of the femur then becomes deformed and there is bony proliferation at the attachments of the rotators and adductors of the hip.

Pain and limitation of motion are the main symptoms of these individuals. The pain is usually relieved following rest or fixation; this suggests that muscular, ligamentous or articular capsular strain are probably the chief sources of the pain. There may be only slight limitation of external rotation and adduction or all motion in the hip joint may be lost. The individual notices that he is unable to tie his shoe laces or to cross his knee. In the advanced case, the leg is held in flexion, adduction and external rotation and the patient presents an awkward, shuffling gait and notices difficulty in sitting.

One cannot produce complete healing and restitution of anatomical contour of degenerative hip joint disease by any known method of treatment. Treatment should be directed first at *relieving ligamentous or muscular strain and in preventing its recurrence. Rest* in the recumbent position, and, in the more severe cases, *splinting of the hip,* is the best method of accomplishing this. In order to correct or decrease the flexion and adduction deformity of the hip, one may employ *exercises, casts, traction,* and occasionally, *manipulation.* This will aid in producing lasting improvement in function. *Postural exercises* and the wearing of a pelvic belt or *special corset* will assist in decreasing the forward inclination of the pelvis and in securing weight bearing so that the head of the femur is in contact with the strong, superior portion of the acetabulum.

Harmon has summarized the conservative treatment for degenerative hip joint disease. In his opinion, *when well-developed roentgen changes of degenerative hip joint disease have become manifest, conservative measures usually offer only temporary palliation.* **Rest from weight bearing, heat** and **sedative** and **analgesic drugs** are the conservative measures most likely to succeed. In patients with a movable hip, *extension applied to the lower limb with the patient recumbent on a firm mattress is the most effective measure in relieving pain and in correcting the flexion and adduction deformity.*[15]

If the patient must be ambulatory, *crutches* or a cane should be used and the physician should insist upon several periods of recumbency per day; this will aid in relieving pain in proportion to the amount of pro-

tection obtained from weight bearing. Patients with unilateral degenerative hip joint disease especially should experience some relief by this program. It must be emphasized, however, that in all cases, the **relief of pain is usually directly proportional to the amount of protection from weight bearing.**

Conservative Treatment for Degenerative Hip Joint Disease (Malum Coxæ Senile)[28]

1. *Rest* on a *firm mattress.*
2. If muscle spasm or flexion deformity is marked, a pillow is placed under the thigh.
3. Apply *heat* for thirty minutes three times a day over the affected hip anteriorly and laterally.
4. With subsidence of muscular spasm, the patient should retract the lower abdomen and contract the gluteal muscles at the same time in order to decrease lumbar lordosis and the forward inclination of the pelvis. These result in decreased deformity and subsidence of pain in a week or ten days; the weight thrust in walking is thus brought more nearly upon the upper and strong part of the acetabulum rather than upon the posterior portion.
5. A *plaster spica* is applied to the hip and entire leg if symptoms do not subside quickly and if flexion and adduction deformity persist. A series of plaster spicas may be required, obtaining a little more correction of the deformity at each application.
6. If there is marked muscle spasm with little limitation of motion, *skin traction* upon the hip joint is employed.
7. Patients are *allowed up for increasing periods* (with corset or pelvic belt) *when pain and muscle spasm* subside.
8. If *symptoms are not relieved* within several weeks, one should *check the diagnosis* and consider the possibility of malignancy, acute infections and Paget's disease.
9. *Gentle manipulation of the hip joint under anesthesia* may be done if there is not much change in the spherical shape of the head of the femur, *i. e.*, when the deformity and limitation of motion result chiefly from muscular spasm and soft tissue contractures. While an assistant holds the pelvis, the operator brings the hip into acute flexion, abduction and internal rotation.
10. *Roentgen therapy* may relieve pain and result in some increase in motion at the hip joint in some of these cases. It may be especially helpful in elderly patients with severe pain.
11. *Injections of procaine* or of procaine and eucupine are not as effective in relieving as high a percentage of these cases as in patients with similar involvement of the knee.[15]
12. Young persons, with extensive malformation of the head of the femur and acetabulum, are benefited least by conservative measures; in these individuals, and especially if the process is unilateral, surgical treatment is preferred.

Most forms of heat will aid in alleviating the pain in these patients whether one uses hot fomentations, diathermy, radiant heat or other measures.

If necessary, motion of the hip may be reduced to a minimum by immobilization in a plaster cast or by using a light ambulatory plaster hip spica.

Some surgeons prefer to use a short leather and steel spica to reduce motion, but in many cases, this is not effective.

Surgical Treatment of Degenerative Hip Joint Disease.—When the patient desires a movable hip, a *vitallium or leucite cup arthroplasty* is probably the procedure of choice for relief of pain.[52] *Arthrodesis* (fixation by bony union) *is the most conservative procedure* but may be indicated in unilateral hip joint disease in some individuals whose occupation may require strenuous and prolonged use of the limb, and in the aged who may not withstand the prolonged period of operation needed for arthroplasty. Arthrodesis is also indicated in those patients who insist upon a relatively certain outcome.

Ghormley and Coventry concluded that arthrodesis is the operation of choice in painful hips and is the one most certain to relieve pain.[12] Cup arthroplasty preserved motion but gave a less certain relief of pain. Among the older patients, acetabuloplasty had a definite place but offered a less certain means of treatment. Drilling operations were used in only the earliest stages of degenerative disease (osteo-arthritis) of the hip joint.

Because of the construction of the hip joint, any irregularity between the surfaces of the acetabulum or head of the femur will eventually lead to degenerative changes; these latter will usually result in pain and limitation of motion.

Following an arthrodesis, motion ceases at the joint.[42] *The patient becomes free of pain.* Supposed disadvantages of the operation have included an increased likelihood of low back pain and an unsatisfactory sitting attitude inasmuch as the hip joint can no longer be bent; Harmon found that these disadvantages were more theoretical than real.

Although best results were obtained in these patients by *arthrodesis,* the operation *cannot be used,* usually, in *patients with bilateral hip joint degenerative changes* or in those with arthritis of the lumbar spine. In addition, fixation is not obtained so easily in aged patients, who do not tolerate the long fixation required to produce satisfactory arthrodesis.

Arthrodesis at the hip joint is usually performed by dislocating the hip and carefully removing the cartilage from both the acetabulum and the head of the femur; in addition, autogenous bone grafts from the tibia are used to obtain solid bony union.

Acetabuloplasty was advised particularly among aged patients who cannot tolerate the long confinement in plaster attendant upon arthrodesis or the more drastic procedure of arthroplasty.

Other operations which have been proposed for degenerative hip joint disease include:

1. *Cheilectomy* or *exostosectomy.*
2. *Osteotomy* (to shift the weight-bearing portion of the femoral head).
3. *Drilling of the femoral neck.*
4. *Securing ankylosis by means of a metal pin.*

Most workers agree that in advanced cases, cheilectomy[51] (removal of exostoses) of the head of the femur or of the anterior and inferior rim, produces results inferior to cup arthroplasty.

Arthroplasty with vitallium cups was introduced by Smith-Petersen in 1939. This is indicated in patients with bilateral hip joint conditions or in those painful hips (from degenerative changes) in which there is an associated involvement of the lumbar spine which contraindicates arthrodesis—as well as in those patients who refuse arthrodesis. The rationale of *drilling the femoral neck* in degenerative hip joint disease is supposed to be the relief of tension within the cystlike cavities which are often seen in the femoral head. Mackenzie reported that 80 per cent of his cases showed good results following the operation,[31] but the operation has not shown such striking results in other surgeons' hands.

Mackenzie[32] obtained improvement following bone puncture in 80 per cent of 106 cases (of osteo-arthritis and rheumatoid arthritis); bone drilling of the neck of the femur has been followed in some instances by benefit.[50]

Ghormley and Coventry were not encouraged with "*forage*"—the drilling operation which has received particular attention, especially in the European literature. In some cases, a marked relief of pain was noticed following the operation but improvement followed in less than half of the cases. It may prove to be of value in early degenerative hip joint disease.

A relatively minor surgical method of obtaining ankylosis of the hip with degenerative joint disease was described by Watson-Jones.[58] He drove a long, heavy *three-flanged pin* on a guide wire, under roentgen control, through the femoral neck and head and into the pelvis. This procedure results in little evidence of shock and can be completed in fifteen to thirty minutes by a general surgeon.

McMurray reported upon *subtrochanteric osteotomy* with mesial displacement of the lower fragment in order to establish contact between the upper surface of the osteotomy and the pelvis. This operation may be considered in those patients with unilateral degenerative hip joint disease who are poor operative risks and will not submit to arthrodesis. McMurray reported excellent results in 12 of 15 cases.[37]

Skilled orthopedic surgeons have obtained excellent results in some patients with degenerative hip joint disease with the use of the **vitallium or leucite cup arthroplasty.**[14,52]

Following cup arthroplasty, relief from pain may be noticed within two or three days with the subsidence of the immediate postoperative pain; if no mechanical complication develops in the joint, this relief should be permanent. If almost total ankylosis was present prior to operation, about 30 degrees of motion in flexion will be obtained postoperatively. In those individuals who had more than 30 degrees of passive flexion prior to operation, an additional 20 to 30 degrees is gained by the surgical procedure. Some degree of rotation and abduction are regained following the operation; these motions are usually lost in the early stage of degenerative hip joint disease. Following the operation, the patient has a satisfactory weight-bearing extremity and no more hip lurch than prior to operation.

Vitallium and *leucite* are the materials most commonly used in the cup arthroplasty; *tantalum* may later be tried for this purpose. Leucite, a synthetic plastic resin, is as non-irritating and inert in tissues as is vitallium and has the added advantage of being radiolucent so that the roentgenogram can readily visualize changes in the bone within the cup. One must be cautioned not to use diathermy over the hip which has had a vitallium or leucite cup in it because of the danger of overheating (of the vitallium) or possibly, change in the shape (of the leucite).

Most orthopedists agree that *surgery is the only means of obtaining lasting relief from pain and disability in those patients who are not relieved by conservative measures.* In those individuals who are fair or good risks for major surgery, *arthroplasty and arthrodesis are the procedures of choice. In those individuals with bilateral degenerative hip joint disease, arthroplasty is the only surgical procedure which can be recommended.*

Some individuals have contended that after several years, bony overgrowths from the acetabulum may interfere with the efficiency of the vitallium or leucite cup.[6]

Orthopedic Procedures.—*Orthopedic care* is of prime importance in most of these individuals. Proper attention must be given to painful, weak feet which may produce strain on the internal lateral ligament of the knee and result in traumatic damage to this joint or strain of the low back. The care of such feet is presented in detail in a later chapter on Painful Feet; low back pain is fully discussed in the later section on Backache.

Proper postural attitudes, exercises and *supports for the low back* as well as the use of elastic, adhesive or *other supports for the knee, ankle, hip,* etc., have been detailed in previous sections. *Crutches, canes* or *caliper splints* may be of use in some of these patients. Proper weight bearing and supports for damaged joints must never be neglected in this disorder.

Manipulation has been reported to be helpful in selected cases of degenerative joint disease of the hip, cervical or lumbar spine.[19,27,60] *This must be undertaken with extreme caution and only by individuals experienced in this procedure.* In many instances, beneficial results from manipulation of the hip in degenerative joint disease are short lived.[21] Various procedures, including cheilotomy, osteotomy, arthrodesis, arthrotomy, acetabuloplasty and synovectomy[5,23] have been described. These belong to the realm of the orthopedic surgeon.

Arthrotomy is occasionally indicated in degenerative joint disease for the *removal of "joint mice"* (free bits of cartilage and bone within the joints) *or bony or cartilaginous spurs* which are producing pressure symptoms. Osteotomy may be performed in an effort to produce normal alignment of joint surfaces and proper weight-bearing lines.

Synovectomy has been recommended in some monarticular lesions affecting the knee or hip in older individuals where conservative therapy has failed to relieve pain on motion of the joint. *It is seldom performed in the presence of an inflammatory lesion.* It has been used in cases with lipping and osteophyte production or destruction of the semilunar cartilages, as well as in so-called villous arthritis, traumatic arthritis, tumors of the joint or osteochondritis dissecans.[33]

Arthrodesis is an operative procedure whereby a joint is made completely rigid. This operation is most applicable "to a single painful hip joint" which is the seat of degenerative joint disease (often called "morbus coxæ senilis"). This operation is also useful occasionally in the presence of arthritic changes about the sacro-iliac or lumbosacral joints.

Arthoplasty is an attempt to restore the range of action to a joint by removing ankylosing tissue and by reshaping the joint surfaces anatomically. This operation is often employed successfully in the knee, hip and elbow joints. In recent years cellophane, fascia, leucite or vitallium molds have been introduced into the joint to keep the bony ends apart.[17,30,36,38,54,55]

FIG. 120.—Marked mixed chronic arthritic changes in right knee. There is now almost end-to-end approximation with considerable bony proliferation secondary to the destroyed cartilage. There are numerous joint mice. The appearance suggests that this may be an old traumatic arthritis with secondary hypertrophic changes.

Haggart believed that surgery was the treatment of choice *in* selected cases with disability from **degenerative arthritis of the knee joint.**[13] In patients with pronounced disability, he obtained marked amelioration of symptoms in almost all of these patients following surgical interference with the affected knee. The operation was performed only in coöperative patients, selected on the basis of their physical fitness for surgery and their willingness to coöperate with a postoperative regimen.

All patients were given a careful medical examination preoperatively and a program was instituted consisting of reduction in weight (if obese), muscular training and development of muscular tone (the same regimen,

in general, being followed postoperatively). The operation was used only in degenerative joint disease of the knee and not in rheumatoid arthritis. Patients usually had pain and tenderness in the anterior knee region, centering often about the patella. In some of these patients there was flexion contracture and limitation of normal flexion from swelling of the soft parts and small amounts of fluid in the joint; pronounced disability was often present. Roentgenograms revealed degenerative changes about the patella; sometimes, the patella was enlarged with marked exostoses. The joint space was often narrowed.

At operation, Haggart observed erosion of the articular cartilage, especially over the femoral condyles, a thinned and fibrillated patellar cartilage, hypertrophy of the patella and hyperplasia of the synovial membrane. There was erosion and widespread degeneration of the articular cartilage with frequent exposure of subchondral bone (which was often eburnated). The most marked degeneration of the femoral cartilage was in the patellar groove.

He used a median parapatellar or preferably a vertical incision centered over the *patella*. The latter *was removed* by sharp dissection after splitting the quadriceps tendon and the patellar ligament (the patella lies behind, not in the quadriceps tendon). The patella develops as part of the quadriceps apparatus and retains a close association with the muscle in the adult; tendon fibers are attached directly to it. In its removal, it must be separated from the muscle by sharp dissection dividing the muscular and tendinous insertions.[2]

Haggart also performed a synovectomy, removed exostoses and shaved down the fibrillated and degenerated articular cartilage, removing all abnormalities as far as feasible.

The hyperplastic synovial membrane was removed from the knee joint except in the posterior compartment. He also excised the fat pad and one or both menisci (if damaged) and cut away exostoses and productive bony changes along articular margins. He closed the capsule with interrupted silk sutures, the subcutaneous tissues with interrupted plain gut and the skin with interrupted silk.

A long leg cast was applied after the postoperative dressings (including a large fluffy cotton pad about the knee) were in place; the cast was removed in five to seven days and the operator resumed muscular exercises with intensive local heat to the knee, infra-red radiation to the leg and massage to the thigh and lower leg. Early motion at the knees was encouraged by suspending the extremity at the ankle in a sling attached to an elastic cord fastened to an overhead bar of a Balkan frame, and instituting bicycling exercises while the leg was so suspended.

Gentle *manipulation of the knee under intravenous pentothal anesthesia* was performed on all patients before discharge from the hospital; this attempted to break up all adhesions in the joint and produce a more rapid return of function. It usually produced slight or no reaction, but led to a rapid increase in the range of motion.

Haggart operated upon 20 such patients. Only one knee was attacked

surgically in any patient. The average age of these individuals was fifty-six years. *Nineteen of the 20 improved, many markedly so* with an absence of severe pain and a resumption of their occupation. The one patient in whom a poor result was obtained was found later to be suffering from a rheumatoid arthritis.

Miscellaneous.—Woolf attempted to relieve *intractable pain in degenerative knee joint disease* by the *injection of the patient's fat* into the affected joint.[61] Fat was obtained from the abdominal wall or the thigh, was shaken with salt solution to wash off the blood, and was cooked for about ten minutes in a porcelain dish over a hot plate. One must continue to cut the fat as this cooks in order to allow the oil to exude. This was drained and filtered through sterile gauze. When it has cooled to 120° F., 15 cc. of the oil was injected through a wide bore needle, on each side, under the patella. The joint was moved and massaged vigorously after the injection. Of 30 injections in 24 patients, 16 were successful in relieving intractable pain. This novel procedure is as yet chiefly experimental.

There is no indication for sympathectomy in degenerative joint disease.[1]

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CHAPTER XL

SPONDYLITIS

THE most important joints of the spine are the posterior intervertebral (or apophyseal) joints. These are true diarthrodial joints. There are also 44 true rib joints made up of 12 costovertebral joints on each side where the ribs join the vertebræ and 10 on each side where the ribs touch the tips of the transverse processes. Unfortunately, the apophyseal and the true rib joints have received very little attention until recently. However, these joints are of extreme importance in rheumatoid spondylitis.[52]

The term spondylitis is derived from the Greek spondylos, meaning vertebra. Spondylitis is arthritis of the spine. One may subdivide spondylitis into traumatic, specific infectious, rheumatoid arthritis of the spine (rheumatoid spondylitis), degenerative joint disease (osteo-arthritis of the spine, hypertrophic spondylitis), etc. Of these, the two most important are the rheumatoid spondylitis and degenerative joint disease (osteo-arthritis).

RHEUMATOID SPONDYLITIS

(RHEUMATOID ARTHRITIS OF THE SPINE)

Rheumatoid arthritis of the spine is a chronic progressive disease affecting portions of the vertebral column and adjacent structures, and occurring predominantly in young adult males; it is characterized clinically by pain and stiffness in the back, with or without involvement of the shoulder and hip joints. *The small posterior intervertebral articulations and the sacro-iliac joints are involved early in the disease;* ankylosis of these joints may occur sooner or later. The bodies of the vertebræ undergo marked atrophy and the longitudinal spinal ligaments may become calcified producing the so-called *bamboo spine.* The costovertebral articulations may be severely affected. Exacerbations and remissions are frequent. In some cases, pain is absent or may be a minor feature.

In 1893, *Bechterew*[5] described what he considered to be a new neurologic disease, characterized by stiffness of part or all of the spine, kyphosis with involvement mainly of the upper dorsal spine, paresis of the muscles of the back, neck and extremities, atrophy of the muscles of the back and shoulder girdle, diminished skin sensitivity over the areas supplied by the cervical and other spinal nerves and prominent symptoms of root irritation.[6–9] Several years later, *Strümpell* (1897) described several cases with gradually developing ankylosis of the spine and hip joints.[118] *Pierre Marie* (1898) discussed 6 cases with complete fusion of the thoracic and lumbar spine, ankylosis to some extent, of the shoulder and hip joints and no involvement of the small joints of the extremities.[79] Fraenkel[30,31] in 1904 made the first complete account of the disease and used the term spondylitis ankylopoietica.

Skeletons showing typical rheumatoid arthritis of the spine had been described as early as 1700.[10] March (1895) pointed out the calcification of the longitudinal ligaments which occurs in this disease and the normal size of the intervertebral spaces. Marie is given the credit for being the first to correlate the clinical and anatomic picture in these patients. Later papers by Bechterew suggest that he was dealing mainly with degenerative changes in the spine, rather than with rheumatoid spondylitis in most instances. In some of his patients, there was a dorsal kyphosis, osteophytes on the thoracic vertebræ and marked reduction in the intervertebral spaces.

Marie and Leri (1899) revealed at autopsy in a patient with rheumatoid spondylitis that the striking feature was marked involvement of the small posterior articulations (true joints) of the spine; most of these were completely ankylosed.[80]

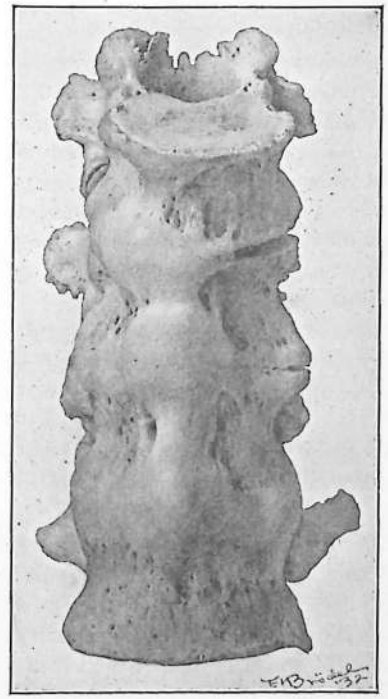

FIG. 127.—Rheumatoid arthritis of the spine with smooth bony bridging between the vertebræ. (Shands, Orthopedic Surgery, courtesy of The C. V. Mosby Company.)

The spondylitis described by *Marie* was characterized by an ascending type of ankylosis in which the cervical spine was least and last involved, and in which there was apt to be greater ankylosis in the hips than in the shoulders; the sacro-iliac joints became ankylosed early in the disease. Complete fusion of the spine occurred with only slight involvement of other joints in the body. The condition described by *von Bechterew* was characterized by a kyphosis of the cervical and upper thoracic spine, with

marked limitation of motion in the spine and without involvement of joints in the extremities. The essential pathologic lesion of rheumatoid spondylitis is an arthritis of the sacro-iliac joints followed by a primary synovitis of the joints formed by the intervertebral facets, calcification of the surrounding ligaments and finally, rigidity of the spine.[129]

The disease usually originates in the sacro-iliac joints and is accompanied or followed by inflammation of the small joints of the intervertebral facets. This inflammatory state may persist weeks, months or years, resulting finally in ankylosis of these joints and calcification of the spinal ligaments.

Excellent discussions of this subject have appeared in the European literature, especially the German[23,30,31,49,66,93,94,98,106,109] as well as a few in the older American literature.[18,24,38,83,84] Goldthwait discussed rheumatoid arthritis and osteo-arthritis of the spine.[75]

Valentini described (1899) roentgenograms in patients with long-standing rheumatoid spondylitis showing the typical bamboo spine.[130]

Simmonds[109] (1904) and Fraenkel[30] stressed the finding of involvement of the small posterior articulations of the spine in rheumatoid spondylitis and emphasized that the intervertebral spaces and the vertebræ remained intact in this disease. They also noted that degenerative joint disease of the spine (hypertrophic changes) appeared secondary to changes in the intervertebral disks, and that bony ankylosis of the costovertebral joints did not occur or was very rare in this condition, as contrasted with its frequency in rheumatoid spondylitis.

The first good discussion of the roentgen findings in rheumatoid spondylitis was presented by Schlayer (1906).[97] He was the first to demonstrate roentgenologically the ankylosis of the small posterior intervertebral articulations.

It is suggested that the confusing eponyms be dropped when one is speaking of spinal joint disease. In general, the terms Marie-Strümpell disease, or spondylose rhizomelique have frequently been used in the past as synonyms for rheumatoid arthritis of the spine (rheumatoid spondylitis); von Bechterew's disease, in the light of careful study, includes some cases of rheumatoid spondylitis, but in some instances probably described what we now speak of as degenerative joint disease of the spine (osteo-arthritis) —which is an entirely different picture. Much confusion will be avoided if we dismiss these eponyms.[72]

Incidence.—The disease is not uncommon, and large series of these cases have been reported, ranging from 98 to 260 in number.[12] Approximately 1 case of rheumatoid spondylitis has been observed to each 13 to 17 cases of rheumatoid arthritis seen elsewhere in the body.[20]

Schmorl[36] noted only *8 cases of rheumatoid spondylitis among 10,000 specimens of vertebral columns*—an incidence of less than 0.1 per cent. In a series of 2561 roentgen films of the spine, Bachman found 41 clear-cut cases.[2]

There is no question concerning the *sex incidence* of the disease. In one series of 100 cases all were males.[27] The highest incidence of females recorded has been 10 per cent.[10]

In most instances the disease begins between twenty and forty years. In one series of 100 cases, 3 began in the second decade, 44 began in the third decade, 32 in the fourth decade, 13 in the fifth decade, and 6 in the sixth decade; in the others, the date of onset was questionable.[27]

Etiology.—The etiology is not known.

There is no good evidence that rheumatoid spondylitis is *familial,* but occasionally, two members of the same family may have the disease. Although *trauma* has been discussed as a factor of possible etiologic importance, especially because of the extremely high incidence of the disorder in men,[30] trauma has not been a prominent factor in most reported series.[22] Acute or chronic *chilling* has also been mentioned as of possible significance as numerous individuals have dated the onset of their illness to war service. However, when the individual history is taken carefully, practically all of these persons had episodes of pain or previous joint disability before entering the service.[135] We have noticed, however, that in some instances, the activity entailed in amphibious operations has definitely preceded the onset of a few of our cases. One can only surmise whether the unusual activity, exposure to cold and chilling (including swimming in cold water) or other factors were of etiologic significance.

Rheumatoid arthritis of the spine has some features which are common in *infectious* diseases: the rapid sedimentation rate, low grade fever and slight anemia in some cases, tachycardia, and at times, a slight leukocytosis. The actual pathology in the small intervertebral posterior articulations is inflammatory in nature, beginning with a synovitis, similar to the picture seen in rheumatoid arthritis of peripheral joints.

In older papers, the idea was quite prevalent that rheumatoid spondylitis was in many cases a result of *gonorrhea.* Marie[79] in later papers stated that gonorrhea could cause the disease and others have held similar views.[5,114,135] Inasmuch as gonorrhea is an extremely frequent disease, it is not unusual that a certain percentage of patients with rheumatoid spondylitis will have had this disease prior to the onset of their spinal pathology. Forestier believed that the primary focus in rheumatoid spondylitis was in the genito-urinary system and that toxic products reached the pelvis and spine through the lymphatics.[28] There is as yet very little factual evidence to support either this theory or those of others who suggest that rheumatoid spondylitis is related etiologically to *tuberculosis* of the genito-urinary tract.[62]

As in most diseases of unknown etiology, the *metabolic* and *endocrine* theories of etiology are advanced with very little to support either.[116] Some years ago, it was popular to speak of the relationship of the disease to *parathyroid dyscrasia* and parathyroidectomies were done with satisfactory results being reported.[33,76,115] A careful analysis of the question fails to reveal any evidence of such relationship.[15,134]

The *high incidence of pulmonary tuberculosis* in some series of patients with rheumatoid spondylitis had led some workers to suggest that the

former disease may be of etiologic significance. It is true that pulmonary tuberculosis is a serious complication of the disease; however, in almost all instances, the tuberculosis is not discovered until a year or longer after the development of the joint disability. Scott suggested that active tuberculous infection of the sacro-iliac joints was a precursor of rheumatoid spondylitis.[101] Kienbock postulated that the joint pathology represented an exudative synostosing form of joint tuberculosis secondary to tuberculosis of the genito-urinary tract (with spread of infection through the lymphatics).[62] Recent work is in accord with the view that pulmonary tuberculosis in rheumatoid spondylitis is either a secondary or merely an incidental phenomenon.[1]

Pathology.—Krebs and Vontz[68,69] (1934) appear to be the first to recognize that involvement of the sacro-iliac joints is an essential part of the disease—and that *changes in the sacro-iliac joints are the earliest and most characteristic sign.* Very early in the disease one may find a spotty loss of contour of the sacro-iliac joint surfaces, with narrowing of the joint space.[132] In addition, one may see patchy areas of increased density in the ischia and pubes with the formation of exostoses and periosteal roughening. Fairly early in some of these patients there may be noted a bridging of the symphysis and ulcerous defects at the symphysis pubis.

The primary pathological interest in the spine in this disorder is the involvement of the true joints of the spine, *i. e.*, the small posterior intervertebral (apophyseal) articulations. In the early stage of the process there is a *synovitis* with *exudation* into the joint, roughening of the articular cartilage, *invasion of the cartilage* by round cells and production of connective tissue.[46] As the disease progresses, the *cartilage* gradually *disappears* and *bony ankylosis* may finally take place. A characteristic of this process is the generalized involvement of *multiple joints*, although the process may not involve all of these joints simultaneously. The bony ankylosis is not an isolated affair confined to one or two joints. This same process occurs in the *costovertebral articulations* with destruction of the articular cartilage and eventual bony ankylosis. One rarely finds exostoses formed about these joints in rheumatoid spondylitis.

Eltze reviewed the *pathologic anatomy of rheumatoid spondylitis.* He noted that the articular clefts were indefinite and the articular surfaces appeared as if they had been gnawed upon.[25] Inflammatory niches were interspersed with narrow bony bridges, which gradually became wider. Cloudy densifications of the bone were seen near the articular clefts. *From one to three or more years elapsed in some cases before these changes were visible by x-ray.*

The only true joints in the spinal column are those between the articular facets.[34] Rheumatoid spondylitis begins as a synovitis of the small posterior intervertebral joints, together with an osteoporosis of the bodies of the vertebræ. Scott believes that there is a close relation between infection of the sacro-iliac joints and rheumatoid spondylitis which often attacks young adults.[103] In several hundreds of these patients whom he has investigated, none have shown normal sacro-iliac joints. He has found early

changes in these latter joints several years before the onset of symptoms of spondylitis, and has suggested that chronic infection of the sacro-iliac joints may be the most important cause of spondylitis in the young adult. However the sacro-iliac joints are occasionally normal.

Others have also noted synovitis of the sacro-iliac joints in those cases of spondylitis beginning in the lumbar region. Synovitis of the costovertebral joints has been observed early in cases in which the cervical and dorsal spine were involved first.[45]

Later changes include a rigid bamboo-like appearance of the spine due to *calcification in the anterior longitudinal ligament, the ligamentum flavum* and in the lateral borders of the intervertebral disks; rarefaction and softening of the bodies of the vertebræ, and destruction and ankylosis of the intervertebral, costovertebral and sacro-iliac joints. *Exostoses are uncommon* in this disease; this is in marked contrast to their extreme frequency in hypertrophic spondylitis.

Early in the course of the disease, marked *atrophy of the bodies of the vertebræ* occurs;[99] despite this, the vertebræ do not collapse and the *intervertebral disks* are *not involved.* The *intervertebral spaces remain intact* despite the generalized involvement of the posterior articular involvement. In persons who have had the disease for many years, the intervertebral spaces may become somewhat narrowed.

An extremely common and most interesting finding in rheumatoid arthritis of the spine is the *calcification of the longitudinal ligaments* which may occur. The cause for this calcification has been the subject of much controversy. Some have contended that the calcification occurs secondary to the ankylosis of the small intervertebral joints but others do not agree with this and have reported the finding of a typical bamboo spine without demonstrable involvement of the small joints. In our experience, this latter must be extremely unusual. We have never seen calcification of the longitudinal ligaments in this disease in the presence of apparently normal small intervertebral joints.

Ossification of the periphery of the intervertebral disk may occur together with extension of the spongiosa of the vertebra through the central part of the disk, so that the involved spine may structurally resemble a long bone.[84]

Oppenheimer believes that ossification of the ligamenta flava occurs coincident with bony ankylosis of the small joints and that the longitudinal ligaments never ossify until complete ankylosis has occurred in the small joints of the affected region.[91]

The consensus of opinion at present is that as a result of a reparative process secondary to the loss of normal articular support for the spine, calcification may occur in various ligaments (anterior, ligamenta flava, capsular, supraspinous and interspinous ligaments).[22] This view is consistent with the finding of decalcification and partial absorption of previously noted ligamentous ossification in the region of operative fusion.

In those patients who have an *associated involvement of other joints, such as the shoulder and hips*, the disease in those regions is similar pathologically

to typical rheumatoid arthritis. Occasionally, however, there is seen considerable calcification and formation of exostoses about the hip joints. Very occasionally, the ankylosing process includes the temporo-mandibular joints. Rarely, ankylosis occurs in the sternoclavicular or shoulder joints.

Symptoms.—Rheumatoid spondylitis is often unrecognized and labeled incorrectly. The internist must become familiar with the diagnostic features of this disease instead of relegating it to the realm of the orthopedist.

The *outstanding clinical features of the disease* are:

1. Pain.
2. Stiffness.
3. Rigidity of the thoracic cage.
4. Tenderness locally over the sacro-iliac joints and spinous processes.
5. Systemic manifestations: loss of weight, strength and muscle tone; slight fever in some cases; tachycardia; mental depression and dejection.
6. Marked predominance (10:1) in the male sex, especially during the third or fourth decades.

The *onset* is usually *insidious* and the progress of the disease extremely slow; acute cases occur occasionally. In some, vague epigastric pains or pains in the extremities may occur for months prior to the evident onset of spinal disturbance.

Pain is the dominant symptom in most instances. This pain is very variable and even the most intelligent patients are vague in their description of it despite its severity. Pain varies in the same individual in degree, site and character from one phase of the disease to another.[53] Pain may be in the low back, sacro-iliac area, hip or shoulder; in some instances, it follows the sciatic distribution. It may be continuous, but varies in intensity. In most instances, it is worse during the early morning hours; the patient is often awakened about 3 or 4 A.M. and lies in discomfort for several hours unless he is immersed in a warm bath and then gets up and moves about for some time. The pain usually gradually subsides after the patient is up and about for some hours. Most of these individuals experience least discomfort in the afternoon and evening.

The pain is greatly increased by any violent or sudden movements, including coughing or sneezing. In some instances, progressive stiffening of the spine may continue for long periods without pain; however, sooner or later pain occurs either in the lower spine, sacro-iliac region or in the cervical area. Because of this great variability of pain and the lack of definite objective findings early in the disease these patients are *often misdiagnosed as neurotics or malingerers.*[69] However, a sedimentation rate will disclose the presence of some pathology and a roentgen examination of the sacro-iliac joints and lumbar spine may reveal evidence of the disease rather early. These films must be read by someone with considerable experience and oblique views should be taken to show the sacro-iliac joints and the true small lumbar intervertebral joints. Unfortunately, we have seen numerous patients who have been called neurotic for years because of their "vague" aches and pains in the low back and extremities,

who were typical examples of rheumatoid spondylitis. It is good medical practice to always perform a sedimentation rate and *x*-ray the low back and sacro-iliac joints when a patient complains of vague aches and pains in the back or extremities.

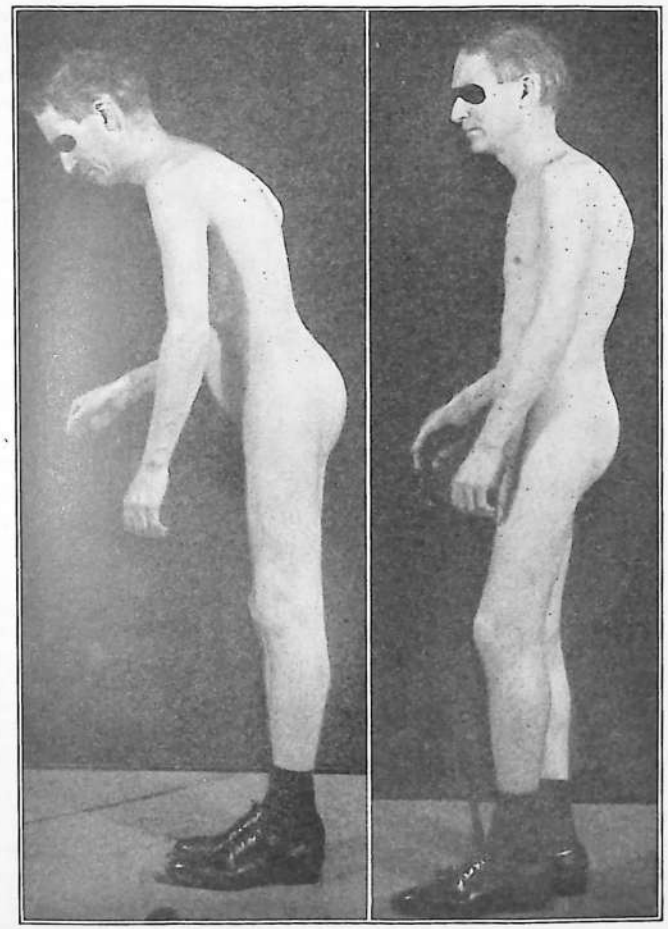

FIG. 128.—Rheumatoid arthritis of the spine. In figure at left, patient is bending forward as much as possible.

Because of the *variability of the pain*, patients have been misdiagnosed as subdeltoid bursitis, degenerative hip joint disease, pleurisy, renal colic, intercostal neuritis, acute abdominal emergency, etc.

Although most individuals with this disease have an insidious onset with exacerbations and remissions, one must remember that the disease occasionally begins acutely with agonizing lumbar or sciatic pain.

The **stiffness** results from muscle spasm (secondary to the pain) and from actual organic joint changes with fixation of these articulations. The

joints most often involved are the *sacro-iliac, small posterior intervertebral* and the *costovertebral.* The sacro-iliac joints are affected first in most instances, frequently resulting in sciatic pain.

The extensors of the spine are in most obvious spasm but motion is limited in all directions so that the flexors are undoubtedly affected similarly. The head cannot be rotated when the cervical spine is involved. One of the almost diagnostic features of the disease occurs when the individual attempts to look to the side or backward; because of the ankylosis, the head does not turn alone, but the entire body turns with the head. Flexion and rotation of the dorsal and lumbar spine may be curtailed because of rigidity of the spine.

Rigidity of the Thoracic Cage.—This is one of the most striking symptoms. In these patients, respiration becomes entirely diaphragmatic with the ribs fixed in expiration. This is a result of involvement of the costovertebral joints. This helps to account for the characteristic posture of these patients with the head carried forward and the chin depressed, together with a moderate dorsal kyphosis and flattening of the anterior chest.

As the lumbar spine becomes less flexible, the lower back is flattened and the normal lumbar lordotic curve is lessened. Although the process begins in the sacro-iliac joints and lumbar spine and works upward in most instances, occasionally, the cervical spine is involved first. Although the shoulder and hip joints are involved not infrequently, *the smaller joints of the extremities are rarely involved.*

In some instances, *tenderness* can be elicited *over the sacro-iliac joints* and the *spinous processes* of the *involved vertebræ.* In others, percussion over these areas elicits tenderness.

In the individual whose disease is well established, the dorsal and lumbar (and sometimes the cervical) spine are held rigid. The normal lumbar lordosis is absent. The thoracic cage does not move on respiration and breathing is entirely abdominal in type. With involvement of the cervical region, the head may be held rigid on the neck and bent forward. When the disease is advanced, spasm and rigidity of the dorsal muscles is observed, together with muscular atrophy in the dorsolumbar region. Signs of root irritation appear in some cases.

General Symptoms.—General symptoms may be absent early in the disease. At a later date there is loss of weight, strength and muscular tone. In some instances, *loss of weight may be extreme, amounting to 50 pounds within a year,* so that the suspicion of malignancy, tuberculosis or other disease is raised. Fever is usually slight, seldom exceeding 101° F.; in some cases, it is absent. A tachycardia (90–100) is usually observed.

These patients seldom smile and are uniformly *depressed* and *dejected.*[53] It is extremely gratifying to note the change of facial expression and mental attitude of these patients as their pain is relieved by roentgen therapy and as signs of improvement occur with proper therapy.

We have never observed rheumatic nodules subcutaneously in these patients. Hare has described in detail the symptoms of these patients.[48]

In the early stages, the patient may complain of shifting rheumatic pains for days or weeks, appearing intermittently over a course of months or years. Later, pain appears in the low back or sacro-iliac region; the discomfort is often more marked at night, especially several hours after retiring. Relief is obtained by getting up and moving about or by massage. These patients are usually stiff on awakening in the morning; this disappears after exercise. Pain in the back is often brought on by stubbing the toes, jarring heavily on the heels, walking down stairs, deep breathing, coughing or sneezing.

With progression of the disease, the patient may complain of pain along the entire spine, with stiffness, and later, rigidity (leading to the poker back). In the later stages, peripheral joints are occasionally involved.

Symptoms of Rheumatoid Spondylitis

May occur alone, or with involvement of hips and shoulders, or of peripheral joints.
Symptoms usually appear between twenty and forty years of age.
Gradual onset in about 90 per cent of the cases.
Occasional acute onset (in about 10 per cent of the cases) with fever, leukocytosis, rapid sedimentation rate, severe root pains, etc.
Marked predominance in males (10:1).
Early stage: shifting rheumatic pains in the limbs or trunk for days or weeks (intermittently for months or years).
Later: *low back or sacro-iliac pain;* marked discomfort at night made worse by lying down and *relieved by getting up and moving about,* or by massage.
Stiffness, especially in the early morning, lessening after mild exercise.
Later, may have pain, stiffness and rigidity along entire spine.
Root pains on one or both sides (with numbness and loss of power in the arms or legs); girdle pains and pains radiating down the extremities.
Spasm of paravertebral muscles, followed by muscular atrophy in the back.
Pain on motion of the spine and on deep breathing, coughing or sneezing.
Sciatica in about 40 per cent of cases; this is one of the commonest symptoms.[67]
Pain on pressure or tapping over and lateral to the spinous processes; this may be exquisite.
Areas of hyperalgesia.
Stiffness in the back, with increasing rigidity (until a poker spine occurs).
Kyphosis and a position of forward flexion of the spine as the disease progresses.
Inability to stand erect; inability to look up or to turn about without moving the entire body.
Flattening of the chest and decreased or absent expansion of the chest during respiration; reduction of vital capacity.
Characteristic slow, shuffling, bent-over gait.
Pain decreases but stiffness increases in later stages.
Characteristic roentgen findings (described in the text).

During the sacro-iliac phase, the symptoms include pain and tenderness along the course of the spine, limitation of motion, muscular wasting, decreased chest expansion and vital capacity. *Pain and spasm are the*

chief causes of limitation of motion; later, calcification in the ligamentous structures leads to complete rigidity. Dorsal bowing of the spine with rigidity of the neck is the commonest deformity; this forces the patient to carry the head in front of the body.

The early symptoms are frequently insignificant and are easily overlooked or misinterpreted.[75] Referred pain is almost always present at some stage of the disease and varies according to the segment of spine involved and the severity of the process. It may simulate the pain of renal calculus, tabes dorsalis, pleurisy, etc. These pains may radiate down the arms, legs, or around the chest or abdomen. Their intensity may vary greatly from those of a very mild nature to severe lancinating stabs.

Rheumatoid spondylitis differs from rheumatoid arthritis elsewhere in the body in that:

Rheumatoid spondylitis occurs predominantly in males (varying from 4:1 to 10:1 in some statistics).

The age incidence of rheumatoid spondylitis is less than in rheumatoid arthritis.

Patients with rheumatoid spondylitis do not show agglutination of the serum in high titer with hemolytic streptococci.

Rheumatoid spondylitis is favorably affected by roentgen therapy.

Laboratory Findings.—The only important positive laboratory findings in this disease are the *increase in the sedimentation rate* and the *roentgen findings.* The sedimentation rate is rapid even in the early stages of the disease. As improvement occurs, this tends to fall to normal in a large percentage of cases.

The roentgen changes are most important.[89,125] The earliest finding in most instances is a *haziness of the sacro-iliac joints* with *narrowing of the joint space.* In some instances, one may observe patchy areas of increased density in the ischia or pubes with the formation of exostoses and roughening of the periosteum. Ulcerous defects of the symphysis pubis and bridging of the symphysis may also occur.[22]

In addition to the haziness of the sacro-iliac joints, with narrowing of the joint space, one may note an increased bone production along the joint margins, which eventually leads to *bony fusion.* A *similar process involves the posterior intervertebral* (apophyseal) *joints of the lumbar,* dorsal and/or cervical spine at a later date; the number of joints and the areas involved varies markedly.

Very early in the disease, general *decalcification* of all bones may be noted with marked *atrophy of the vertebræ;* however, the intervertebral spaces are well preserved (although these may be slightly narrowed at a very late stage). In advanced cases, *calcification of the anterior and lateral ligaments* of the spine produces the typical *bamboo appearance,* which is pathognomonic of rheumatoid spondylitis.

The *early changes in the sacro-iliac joints are shown best by roentgen-rays* with the sacrum in a longitudinal axis (known as the 15 degree angle view) taken with the patient supine, the knees flexed and the roentgen-ray tube

tilted 15 degrees cephalad—permitting an undistorted view of the sacro-iliac joints.[48]

Oppenheimer described the technic for visualizing the apophyseal joints: 45 degrees in the lumbar region; nearly 20 degrees in the thoracic region, and 90 degree films in the cervical region.[90]

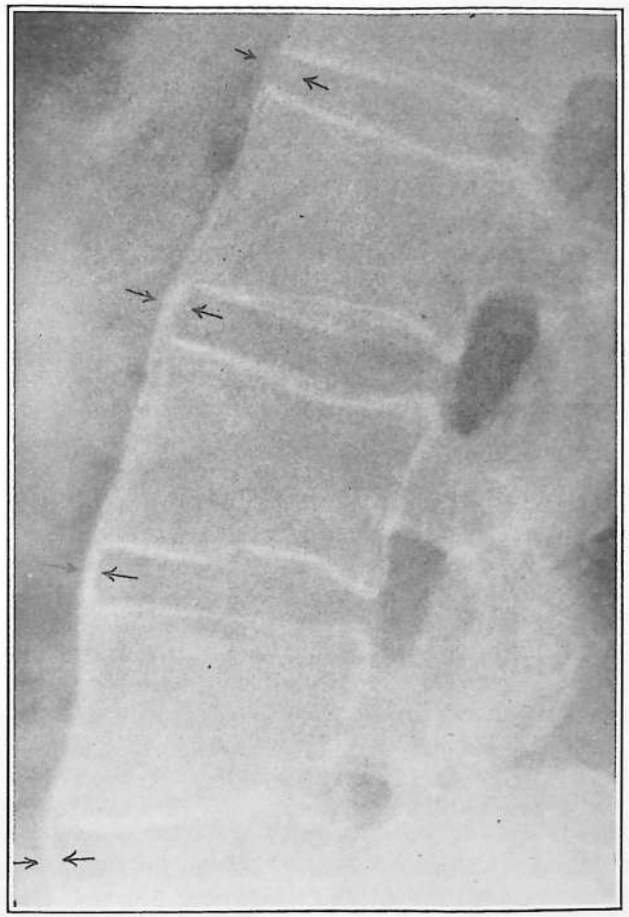

FIG. 129.—Rheumatoid arthritis of the spine showing the calcification of the anterior ligaments (note arrows). Patient is a male, aged twenty-eight, who has had low back pain for eight years. The sacro-iliac joints are obliterated.

The most important roentgenogram to be taken in young individuals with vague fleeting pains in the lower back and limbs is a three-quarter view of the spine made at an angle of 45 degrees. If one finds the loss of density in the articular processes of the lower lumbar apophyseal joints, together with blurred outlines, this is very suggestive evidence of a rheumatoid spondylitis.[14]

Roentgen Findings in Rheumatoid Arthritis of the Spine

1. *Haziness* of the *sacro-iliac joints.*
2. *Narrowing* of the *sacro-iliac joint space.*
3. *Decalcification* of the *vertebræ.*
4. Increased bone production along the sacro-iliac joint margins, eventually leading to *bony fusion.*
5. *Similar process* as above *at a later date involving the* small (true) posterior intervertebral *(apophyseal) joints* of a portion of, or of the entire spine, usually beginning in the lumbar region and progressing upward —but occasionally being noted first in the cervical area.
6. In an advanced stage, *calcification* of the *anterior and lateral spinal ligaments.*
7. *Pain, stiffness and other symptoms may precede the roentgen findings by months or years.*
8. There may occur patchy areas of increased density in the pubes or ischia with periosteal roughening and exostosis formation, ulcerous defects at the symphysis pubis and bridging of the symphysis.
9. *Peripheral joint involvement is uncommon* in association with rheumatoid spondylitis; however, the *hips* and *shoulder joints* are involved not infrequently (in what appears to be a typical rheumatoid arthritic process) and the sternoclavicular joint and temporomandibular joints may be involved rarely.

Other laboratory findings are not of significance. Streptococcus agglutinins are not increased in this disease in contrast to their presence in more than half of the patients with rheumatoid arthritis of peripheral joints.[22] Although secondary anemia is frequent, no characteristic changes are seen in the leukocyte pattern.

Ludwig, Short and Bauer studied the *cerebrospinal fluid protein* in 101 patients with rheumatoid arthritis (42 of whom had spondylitis).[78] They were impressed by the numerous neurologic manifestations of rheumatoid arthritis which, although variable, are so striking at times as to simulate disease of the peripheral or central nervous system. The only significant abnormalities observed in the spinal fluids were *increased total protein,* abnormal colloidal gold curves or a combination of the two. *Fifteen of the 16 patients with increased spinal fluid protein had spondylitis* or symptoms suggesting spinal involvement, suggesting a relationship between the elevated spinal fluid protein and the presence of rheumatoid arthritis in the sacro-iliac or spinal articulations. These alterations were seen more often in those spondylitis patients who had severe pain or sciatica or both.

Mester's test has been found *grossly unreliable* as an aid to the diagnosis of rheumatoid spondylitis.[42,136] This test was originally proposed as a specific diagnostic test for rheumatic diseases.

Complications.—Rheumatoid spondylitis and rheumatoid arthritis elsewhere in the body occur not infrequently in the same individual. In one series of patients with rheumatoid spondylitis, generalized joint swelling was observed at the onset in almost 30 per cent. The hips and shoulder joints are prone to be involved. The most frequent complications are

pulmonary tuberculosis, iritis, cardiac involvement and pneumonia.[1,62,101] *Pulmonary tuberculosis* has been reported in as many as 25 per cent of these patients, and is a serious complication.[22] This usually is noted some time after the rheumatoid spondylitis is noted.

From 5 to 25 per cent of patients with rheumatoid spondylitis develop *iritis* at some time during the course of their illness.[27,37,65,119,127,135] The occurrence of iritis in rheumatoid arthritis of peripheral joints has been presented in a previous chapter.

Pericarditis, endocarditis and *rheumatic heart disease* have been observed in a few instances. Occasionally, rheumatoid spondylitis has apparently followed an attack of rheumatic fever.[27,69] In one series, 11 of 100 patients with rheumatoid spondylitis had valvular heart disease. Occasionally rheumatoid spondylitis occurs in a patient who has had typical rheumatoid arthritis of peripheral joints.

Eltze noted that in some instances, rheumatoid spondylitis was preceded by rheumatic purpura.[25]

Differential Diagnosis.—In the frontispiece of his book on Adolescent Spondylitis, Scott illustrates a "*spondylitic wreck*" as a "horrible example" of what will happen to the misdiagnosed or neglected patient with rheumatoid spondylitis.

Scott noted that rheumatoid spondylitis attacks the young, healthy athlete with an insidious onset extending over five to seven years before spinal symptoms appear. He stresses that the only clinical sign may be the occurrence of pain in the ankle, hip or any part except the back or sacral region, but that changes are always present in the sacro-iliac joints. In his cases, *pain was not felt over the back or over the sacro-iliac joints during the most active stages of the disease,* but by the time the clinical signs of spondylitis appeared, bony ankylosis of the sacro-iliac joints was almost always complete.

It has been emphasized that the stiff painful spine so characteristic of rheumatoid spondylitis represents a late, and not an early stage of the disease.[105]

In well advanced cases, the diagnosis of rheumatoid spondylitis is usually easy. **Findings in the typical case include** roentgen abnormalities in the sacro-iliac joints and in the small posterior intervertebral (apophyseal) joints, marked restriction of spinal motion in the lumbar and in many instances in the dorsal and cervical regions, gross diminution in chest expansion with lack of motion of the thoracic cage, progressive loss of weight and disability and an elevated sedimentation rate.[112] Although involvement of the sacro-iliac joints is often the earliest roentgen sign of this disease, sacro-iliac arthritis does not necessarily signify the inevitable development of spinal involvement.

Inasmuch as several months or years may elapse before characteristic roentgen changes are demonstrable, diagnosis in the early stages should be suggested on clinical evidence.[132] In some instances, spotty atrophy may be noted about the posterior articulations in the lumbar region within a year after the onset of symptoms.[63] Unfortunately, the sacro-iliac joint

changes are not specific and characteristic for rheumatoid spondylitis alone.[74] When involvement of the posterior intervertebral (apophyseal) joints is demonstrated, the diagnosis is established.

The main differential diagnostic concern and the one which has caused most confusion in the past is between rheumatoid spondylitis and *degenerative joint disease of the spine* (*hypertrophic arthritis—osteo-arthritis*).[77] However, in the latter condition, the disturbance seldom causes symptoms

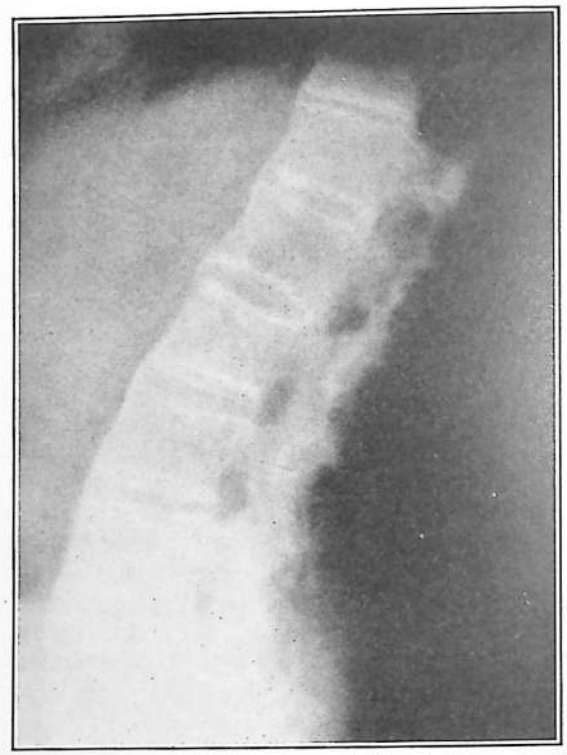

FIG. 130.—Rheumatoid spondylitis in a man aged fifty-eight years. The entire spine shows bridging across the intervertebral spaces producing a virtual ankylosis of the spine. There is exaggeration of the various curves of the spine and considerable variation in the width of the intervertebral disks. The anterior common ligaments are calcified.

before the fifth decade; the sedimentation rate is normal; anemia, fever and leukocytosis are not symptoms of the disease; and the roentgen changes are entirely different. In degenerative joint disease of the spine, the intervertebral disk degenerates and the intervertebral spaces become narrowed. Exostoses are formed about the edges of the vertebral bodies and true bony bridging may occur in advanced cases. The posterior vertebral and costovertebral small joints are not involved in degenerative joint disease. It must be remembered that *ankylosis of the sacro-iliac joints has been reported in over 50 per cent of males by the fifth decade*, so that examination of this joint offers little differential diagnostic assistance at that age.[96]

True infectious spondylitis may occur and result in a rigid spine.[117] Doub has described a syndrome consisting of a rounded kyphosis of the dorsal spine with narrowing of the intervertebral disks, with varying degrees of bone production and leading, in some cases, to a complete bony fusion of the vertebræ.[21] In his patients, there was a noticeable lack of changes in the lower spine.

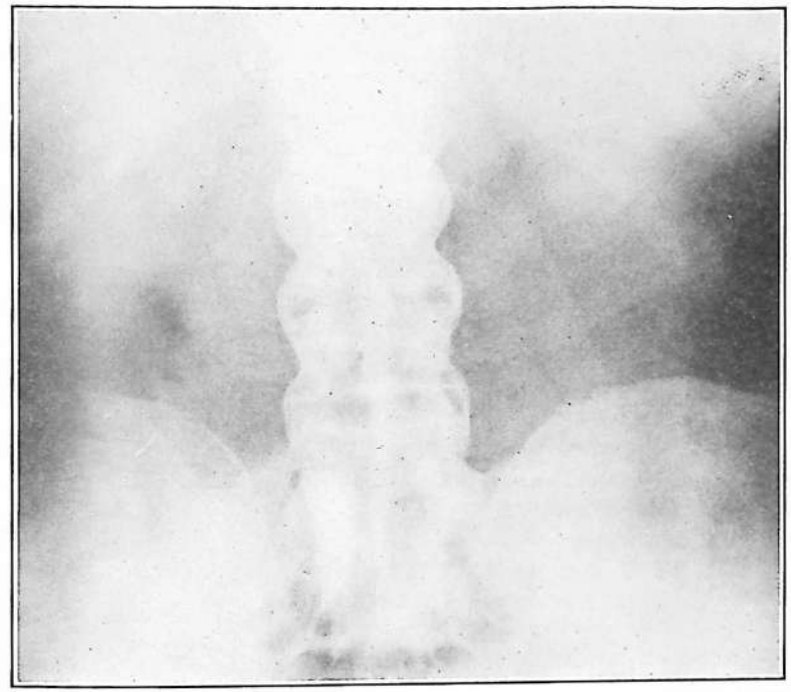

FIG. 131.—Bamboo spine of rheumatoid spondylitis. The entire spine is virtually ankylosed.

Buckley believed that rheumatoid spondylitis in the early stages may be confused with *adolescent kyphosis*.[14] *Epiphysitis of the sacro-iliac joint* in adolescents may also be confused with rheumatoid spondylitis. However, in his series the symptoms of sacro-iliac epiphysitis (low back stiffness, pain and tenderness over the sacro-iliac joint, sciatic pain and loss of straight leg raising) disappeared completely with rest and conservative measures.

Scott believes that adolescent kyphosis is not a specific pathologic entity but merely an excessive kyphosis associated with the final stages of rheumatoid spondylitis.

First lumbar and twelfth dorsal nerve neuralgia (also called causalgia or the dorsolumbar syndrome) is characterized by unilateral pain over the lower portion of the back, iliac crest and midlumbar region with hyperesthesia of the skin supplied by these nerves and a variable amount of abdominal pain and tenderness, occasionally leading to useless abdominal

operations. This pain is relieved by perineural injections or by paravertebral sympathetic procaine block.[56,57,124] Because of the root pains seen in this disease, one must consider the possibility of tabes dorsalis, primary or metastatic neoplasms of the spinal column, herpes zoster, renal calculus, pleurisy, tuberculous sacro-iliitis, tuberculosis of the hip, Pott's disease, degenerative joint disease, late rickets, Paget's disease, sciatic neuritis, protrusion of the nucleus pulposus, osteomyelitis of the spine and various acute abdominal catastrophies. Pains in the limbs may be a premonitory symptom of rheumatoid spondylitis and no complaints referable to the back may be noted for a year or more after the onset of the illness. *It is good medical practice to perform a sedimentation rate and a roentgen examination of the pelvis and lumbar spine in all patients with persistent rheumatic pains in the limbs without definite signs of specific rheumatic infection.*[11] *A rapid sedimentation rate together with loss of definition* of the sacro-iliac joints and possibly, in addition, osteoporosis of the ilium, sacrum, or lumbar vertebral bodies, suggests the presence of a rheumatoid spondylitis; only rarely does one find rheumatoid spondylitis without involvement of the sacro-iliac joint.

Differential Diagnosis

	Rheumatoid spondylitis	Degenerative spinal joint disease (*osteo- or hypertrophic arthritis*)
Sex	Males 10:1	Either sex
Age incidence	Usually begins from 20 to 40 years	Symptoms uncommon before 45 years
Sedimentation rate	Markedly elevated	Normal, in uncomplicated cases
Main symptoms	Pain, stiffness of back, tenderness over involved joints	Pain in back, root pains, areas of anesthesia and paresthesia, occasional spasm of vertical muscles
Rigidity of thoracic cage	A striking symptom	Absent
Constitutional symptoms (loss of weight, strength and muscle tone, slight fever, tachycardia)	Usually marked	Absent in uncomplicated cases
Marked *restriction of* spinal motion, especially in lumbar and dorsal regions	Present	May be slight
Intervertebral spaces	Normal	Usually narrowed
Osteophytes	Usually none	May be marked
Narrowing of sacro-iliac joints	Characteristic	May occur in some cases
Narrowing of small posterior intervertebral joints	Characteristic	Absent (except to a slight extent in occasional cases)
Bony ankylosis of the *sacro-iliac* joints and spinal posterior intervertebral joint	Characteristic late in disease	Absent
Calcification of longitudinal ligaments	A late characteristic	Absent
Results of roentgen therapy	Excellent in early cases; definite benefits even in later cases	Subjective improvement in some cases but no dramatic results as in rheumatoid spondylitis

Prognosis.—*In acute cases, especially in young individuals, an ankylosed (poker) spine may be noted within several months after the onset of the disease.* However, rheumatoid spondylitis does not usually shorten life, although marked deformities may occur if proper treatment is not instituted promptly. Death, when it occurs, results from the serious disturbances in function when the chest cage is rigid. In untreated patients, the symptoms usually subside after a variable number of years leaving the individual with the bamboo (stiff—immovable) spine, and the rigid thoracic cage, but with very little or no pain. Before the use of roentgen therapy, the disease usually progressed, subject to remissions and exacerbations, so that the patient was given little peace until bony ankylosis was established at the end of ten to twenty years[22] at which time pain ceased. The amount

FIG. 132.—Rheumatoid spondylitis in a young man aged twenty-two years. This patient presented changes involving the articular facets and the sacro-iliac regions; haziness and narrowing of the small intervertebral joints can be seen above. There is narrowing of the articular spaces and demineralization of the spine (which often accompanies rheumatoid spondylitis).

of permanent crippling varied with the therapy and attempts to minimize deformities. In a few instances symptoms were minimal and therapeutic results were apparently excellent.[64] It has been suggested that the rigid thorax favored the development of pulmonary tuberculosis as a cause of death in these patients.

Treatment.— *The physician must realize that he has a sick patient with a systemic disease who must be treated, as well as his spine.* The *general treatment* for this disease is similar to that of rheumatoid arthritis elsewhere, including rest; high caloric, high vitamin *diet* (if underweight); removal of definitely infected foci; the use of a *non-sagging mattress* with boards placed under this if necessary; sleeping without a pillow; *deep breathing exercises* to prevent marked change in thoracic respiration; a special spinal *brace* worn during the day; *physical therapy*, and if available *roentgen therapy*. It has been our experience that **roentgen therapy is the treatment of choice** *in the early stages* (before ligamentous calcification) of this disease.[48] Gold has recently been found to be of value in some cases of rheumatoid spondylitis.

FIG. 133.—Rheumatoid arthritis of the spine with complete ankylosis in faulty position. (Shands, Orthopedic Surgery, courtesy of The C. V. Mosby Company.)

Rinehart has emphasized the extremely low blood vitamin C level (under 0.2 mg. per cent) in rheumatoid spondylitis.[95] The capillary strength is also low; this latter could be elevated in 70 per cent of these patients by *large doses of vitamin C*. In his practice, 18 of 21 patients experienced lessening of their severe fatigue and 19 of 21 noted less pain following the administration of large doses of cevitamic acid. He reported complete "cure" of 2 of these patients in whom the disease was of less than one year's duration. Others believe that the lowered vitamin C is merely a symptom of the disease along with the lowered serum protein, hemoglobin, etc.

Swaim and Kuhns have found that the improvement was hastened by rest, relief from strain, increase of vital capacity and increased circulation.[122] During the acute stage of the disease (with associated pain, stiffness and muscle spasm) they advocate recumbency in bed to rest the muscles and inflamed ligaments. *Prolonged rest for the spine and limbs together with mobilization of the costovertebral joints are important factors in the treatment of arthritis of the spine.* They advise no sitting or walking

until pain and muscle spasm have disappeared; in severe cases, they do not permit the patient to rise for meals or bathroom privileges for a month or more. A summary of the essential orthopedic points in their treatment of these cases is as follows:

Orthopedic Suggestions in the Treatment of Rheumatoid Spondylitis[122]

Absolute *rest in bed* in severe cases, for periods of a month or more.
Gradually *remove pillows from under the head.*
After several days, attempt to correct or prevent forward bowing of the chest and fixation of ribs in downward inclination.
The patient lies on his back on a *firm mattress* with a small firm pillow placed under the most prominent part of the spinal deformity, for thirty minutes several times a day.
Gradually use a larger pillow or small sand bag.
If there is difficulty in producing motion of the ribs, use a posterior molded shell with ridges placed to exert pressure upon the angles of ribs posteriorly.
Hot fomentations to the entire spine for twenty minutes twice a day.
Frequently change the *plaster shells* or jackets as improvement occurs.
Breathing exercises to increase thoracic capacity.
When the active disease becomes quiescent, use exercises with the patient sitting on the edge of the bed and later in a chair; *avoid muscle spasm.*
If the spine is not straightened within several weeks, apply a light plaster jacket, and renew this at regular intervals.
When maximum correction has been obtained, apply a waxed leather or celluloid jacket; this should be worn for several years.

One of the most important steps in the treatment of rheumatoid spondylitis is to *support the spine adequately* and at the same time to provide for maintenance of the mobility of the costovertebral joints, the hips and the shoulders. It may be impossible to prevent ankylosis at the hip or shoulder joint; this problem needs much further investigation.

Swaim has reported excellent results in rheumatoid spondylitis with the use of *plaster and leather jackets.*[120] He expressed the belief that the stiffness of the back was due to severe muscular spasm of spinal and abdominal muscles which served as a constant protection against any painful motion. He believed that *ossification of the spinal ligaments was nature's method of permanently immobilizing the spine in order to avoid motion, strain and inflammation of the ligaments.* Accordingly, he advocated recumbency with attempts to hyperextend the spine by removing pillows from under the head, and by using plaster half shells for the back, hot fomentations to the whole spine for twenty minutes twice a day and corrective exercises. If the spine was not straightened within two weeks by this program, he employed a light plaster jacket, extending high in front to the symphysis pubis below, and reaching in back from just under the shoulder blades to the tip of the spine. Abdominal breathing was limited by molding the jacket tightly over the abdomen. Sufficient padding was used over the sternum, anterior superior iliac spines and over the spine. The pelvis was firmly gripped by the jacket. This was not removed until

all spasm had disappeared (usually several weeks). New plaster jackets were applied until the maximum correction had been obtained. A waxed leather jacket (laced in front and steeled in back) was then worn (usually day and night) for several years.

Rest in Bed in Rheumatoid Spondylitis[123]

Improper posture in bed leads to poor results in this disease.
The mattress must not sag, and, if necessary, large boards must be placed longitudinally under this.
The patient should not be placed in a semi-Fowler position but should remain flat in bed with at most only a small pillow under the head (no pillow is preferable).
For thirty minutes, two or three times a day, attempts should be made to hyperextend the spine by removing the pillow from under the head, placing a small firm pillow under the upper thoracic spine and having both hands under head, with the patient lying on his back. This position tends toward fullest expansion of thorax.
Complete rest in bed for some weeks may be necessary if deformity has already occurred.[19]
Use infra-red radiation or hot fomentations to the spine for several short periods (twenty minutes each) daily; this aids in relaxing muscle spasm and relieving pain.
If ankylosis is inevitable in spite of treatment, the optimal position for this is full expansion of the chest, and extension of the spine without increase in the normal curves.[100]

During the period of rest in bed in the acute stage, exercises of the extremities of a mild nature must be encouraged so that muscular wasting does not occur. Periods of faradism followed by massage to the muscles of the back may be given before the patient is allowed out of bed. Pain or cramp in the back due to muscular spasm or fatigue may be relieved by dry or moist heat, *i. e.*, infra-red radiation or immersion baths, together with the under-water douche at a high temperature.[17] The use of heat and light massage to the muscles of the back, shoulders and hips will often relieve pain and muscle spasm and prevent wasting. Deep breathing exercises must be encouraged from the onset of the disease to promote chest expansion. Active movements of the hips and shoulders and gentle lateral bending exercises for the trunk will also prove valuable. Deep pool baths, if available, will assist in relieving muscular spasm.

Howorth has suggested a **spinal osteotomy** as a simple, safe procedure in late cases of rheumatoid spondylitis in which considerable calcification or ossification has occurred.[55] The operation should be reserved for this stage and is used merely as a last resort to correct deformity. Postoperative pain usually subsides in four or five days; after this time, pain is negligible and there is no pain in that part of the spine after the fusion. Howorth has emphasized that it is very difficult to determine by *x*-ray how much ankylosis is present and which joints are ankylosed in the rheumatoid spondylitic patient. In some of these patients, it is necessary to perform osteotomy through areas in the lumbar region, removing as much bone

as is necessary to correct the deformity. Spinal fusions have been performed especially in the lumbosacral region to prevent pain and reduce deformity. Following the osteotomy, the patient is placed in plaster in bed for three months, and then may be up and about in a plaster jacket for another three months. The ideal time, in Howorth's opinion, to perform the operation is when it can be done posteriorly without having to do anything anteriorly.

Breathing Exercises in Rheumatoid Spondylitis.—Proper breathing exercises are an important aid in therapy. *Forced deep breathing exercises may be done with benefit* for periods of five minutes several times a day. The main purpose of such exercises is *to promote thoracic respiration* and to keep the costovertebral joints supple. Abdominal breathing must be discouraged as much as possible; this may be decreased by a tight belt or broad muslin band worn around the abdomen during the periods of breathing exercises. By voluntary contraction of the abdominal muscles during inspiration, the tendency for thoracic expansion will be increased because of partial immobilization of the diaphragm.

With the patient lying on the back and the hands clasped under the head, a deep breath is taken and the chest is raised without lifting the lower back. The chest is now held up, and during expiration, the lower part of the abdomen is drawn in. The important points in this exercise consist of the lifting of the chest which tends to be pushed higher with each breath, and the contraction of the lower abdominal muscles during exhalation.[40]

Roentgen Therapy.—Although the results of roentgen therapy in rheumatoid arthritis of peripheral joints are unreliable, unpredictable and less satisfactory than is usually claimed, *significant symptomatic improvement is obtained in over 70 per cent of cases and objective improvement in 50 per cent of cases when roentgen therapy is used in rheumatoid spondylitis. In the early stage of the disease,* Smyth and Freyberg reported *significant,* sustained, *subjective and objective improvement in over 90 per cent of the cases,* some of whom were completely relieved of all clinical evidence of the disease.[111,112] A significant reduction of pain and stiffness, and in some instances, objective improvement was seen following roentgen therapy even in later stages of the disease, after the occurrence of ligamentous calcification. In our experience, *roentgen therapy has been the best method of controlling the symptoms of rheumatoid arthritis of the spine.* Although a significant decrease in the sedimentation rate occurs after roentgen therapy in almost half of the cases, it is not yet known whether the roentgen effect is symptomatic or whether the course of the disease is actually affected favorably.[112]

Kahlmeter reported improvement in 3 patients with rheumatoid spondylitis following roentgen therapy (1930).[58] Scott later claimed remarkable results in these patients following wide field roentgen irradiation.[102,104] He has reported his results in more than 400 patients treated by roentgen therapy; some of his patients in the early stage of the disease were said to become symptom free within a few months. Kemen noted improvement

in 67 of 77 cases following roentgen therapy.[61] Recently, Hare treated the entire spine and sacro-iliac joints and found relief of pain in 80 per cent of 35 patients with rheumatoid spondylitis.[48]

Smyth and Freyberg found a large percentage of patients who improved significantly following roentgen therapy. In the early stages of the disease, *relief of pain often occurred soon after the first series of treatments* and continued gradual improvement followed until all clinical evidence of the disease disappeared. Some patients with partial calcification of ligaments showed a significant increase in motion and chest expansion when their back pain was relieved. In most of these individuals, the partial or complete beneficial effects persisted. In those whose symptoms recurred several months after therapy, additional roentgen treatments were usually effective in producing relief. In most cases, improvement was reported within the first week after treatment was begun and some patients insisted that pain and stiffness were decidedly reduced within a few hours after therapy. In those in whom no change was evident until after the second series of treatments, the improvement was generally less and slower. The possibility of benefit with additional radiation was slight when no improvement was obtained after two series.

In some instances, lead screens were used to protect the parts from irradiation to determine the psychogenic effect from radiation. None of the patients who were protected by lead screens showed significant improvement, but when they were later treated with roentgen rays, significant improvement occurred without exception. In other patients, one area of the spine was treated with irradiation and later, a different area was treated, but with a lead screen; no beneficial effects were noted when the screen was used. Thus, it appears that *the effect of roentgen therapy in rheumatoid arthritis of the spine is definitely not a psychogenic one.*

Despite the definite symptomatic and objective improvement in these patients, no evidence of anatomic improvement was noted when these individuals were re-examined roentgenologically from six months to two years after therapy. Significant clinical improvement occurred with reduction in the sedimentation rate despite an increase in the calcification of the paravertebral ligaments. This again suggests that the increased calcification may be actually a part of the healing process and not a result of continuing active inflammation.

Dosage of Roentgen Therapy.—Most workers have employed a form of local high voltage roentgen therapy.[36,48,58,61,121,126,133] There is as yet *no recognized standardized technic of roentgen irradiation for rheumatoid arthritis of the spine.* At present, factors including dosage, size of portals and the frequency of radiation are under investigation. In their earlier work, Smyth and Freyberg used 200 kv. (175 kv. constant potential equivalent) with 0.5 mm. copper and 1 mm. of aluminum filter, a half value layer of 0.9 mm. of copper, a 50 cm. skin target distance and an output of 50 r (measured in air) per minute; the size of the field approximated 200 to 300 square centimeters. A series of treatments consisted of a total dose of 600 r per field, each field being given 200 r every other day for 3 treatments. More recently, because of the occurrence of marked leukopenia (temporary) in a few instances, this dose was reduced to a total of 300 r per total field dose per series, 150 r being given every other day for 2 treatments. This series of 2 treatments per field was usually given at monthly intervals (for three months) to the same area. Additional treatment was given if symptoms recurred or if they developed in new areas. Any portion of the spine, or all of the spine (cervical, thoracic, lumbar or sacral) and the sacro-iliac joint, or any combination of these regions were irradiated. The symptoms and physical signs determined the parts selected for treatment. The hips and shoulder joints were also treated in a few cases with far advanced disease.

Scott has used what he terms *wide field therapy* in these patients and has reported excellent results. Scott used a special wide angle x-ray tube to deliver a "*roentgen ray bath*" to the whole trunk, front and back, which are covered by two overlapping 12-inch fields. The factors were 140 kv. 3 mm. aluminum filter, and 60 to 100 r, with a standard distance of tube to the patient of 17 inches. Treatments were given twice a week in the first series for a total of 8 sessions; following a rest interval of six weeks, treatments were again given twice a week, but for 6 sessions; after another free interval of six weeks, a third series of treatments were given—once a week for 6 sessions.[112]

Roentgen-ray therapy should be employed as an integral part of the treatment of this disease. In addition, all of these patients must obtain adequate rest, caloric and vitamin intake, splinting, heat, massage, breathing and other exercises and good orthopedic care including braces or spinal jackets as needed, a firm, non-sag mattress, etc.

Badgley has suggested that if vitallium cup arthroplasty is required in some patients with rheumatoid spondylitis with hip joint involvement, excellent results will be obtained in those with bony ankylosis and a low sedimentation rate. Thus, roentgen therapy may be valuable preoperatively in those patients where surgery may be required.[4]

Forestier reported excellent results in 25 patients with rheumatoid spondylitis following the subcutaneous injection of radon.[29]

Ill Effects From Roentgen Therapy of Rheumatoid Spondylitis.—*Leukopenia* is the only potentially serious ill effect encountered to date. It is not uncommon to find transient decreases in the number of leukocytes following such therapy but significant leukopenia is not seen commonly. If marked leukopenia is noted, the leukocyte count usually returns to normal within three or four weeks if the roentgen therapy is stopped. A reduction in the dosage usually greatly lessens such a tendency to leukopenia.

Various workers have commonly found *anorexia, nausea and vomiting* when the thoracic and lumbar spine were irradiated. Such symptoms began from four to six hours after therapy and usually subsided in two or three days. When larger amounts of irradiation are used, these symptoms are usually more severe.[112] In our experience when treatments are given posteriorly only, gastrointestinal symptoms are seldom encountered in

this dosage in male patients.[16] In one female patient, nausea was severe enough to interrupt treatment for a week.

If it is necessary to treat the *cervical spine*, the patient must be warned that *temporary depilation* may result in the area treated. It is usually more prudent, in women, to avoid irradiation above the hairline if this can be done.

Because of the *possibility of sterilization in the female* following large doses of roentgen irradiation, Smyth and Freyberg suggested that not more than a total of 300 r be given over the female pelvis in a series of treatments.

Mode of Action of Roentgen Therapy.—There is as yet no adequate explanation of the ways in which *x*-rays act to produce these changes. It has been shown that following roentgen therapy there occurs a marked dilatation of the capillaries, subcapillary plexuses and arterioles under the area treated, which persists not only for a few hours (as occurs with other forms of heat) but for at least several days. This active hyperemia may be part of the answer to the improvement from roentgen therapy in these patients.[16]

INFECTIOUS SPONDYLITIS

Acute infectious lesions of the intervertebral disks, sometimes called infectious spondylitis, have been reported following numerous diseases, such as measles, scarlet fever, influenza, smallpox, typhus fever, malaria, *Brucellosis, typhoid* or paratyphoid fever and infections with pneumococci, staphylococci and streptococci.[3] The characteristic finding is narrowing of the involved intervertebral disk; only a portion of the disk is usually involved. The rapid appearance of bony proliferation developing into marginal pads and bridging bands is especially typical.

The disease has been reported in patients from nine to fifty-nine years of age, averaging about thirty-five years. Symptoms include pain in the back, fever, spasm of the lumbar or psoas muscles, tenderness over the affected area and limitation of motion.

In the acute stage of the disease, *x*-rays are usually negative; when followed over several months, one will note thinning of the involved disk with marginal bony proliferation. Solid bony fusion may occur in some cases.

This disorder must be differentiated from osteomyelitis, tuberculosis, post-traumatic thinning of the disks, congenital absence of the disks, osteochondrosis of the disks, or specific infectious processes. It can usually be differentiated from tuberculosis by its more sudden and violent onset or preceding acute infectious process elsewhere in the body and rapid healing usually with fusion of adjacent vertebræ.

Prognosis is very good as to recovery with minimal or no disability, and as to mortality. In the presence of severe symptoms with fever, marked pain and prostration, the patient is treated symptomatically and the spine is fixed by means of a plaster body cast. With subsidence of pain and evidence of healing (within several months), a corset or steel brace may be used until symptoms have entirely cleared (within a year or so).[35]

SYPHILITIC SPONDYLITIS

Freedman and Meschan discussed 3 cases of **syphilitic spondylitis.** Involvement of other bones in addition to the vertebræ was present in all cases. In one instance, the diagnosis was substantiated at autopsy by histologic findings. In the other two, both had a positive Wassermann reaction and some of the bone lesions responded to antisyphilitic therapy.[32]

In the *Charcot spine*, there is bizarre destruction of bone and extensive new bone production. On the other hand in *syphilitic spondylitis*, the periosteum is invaded by the treponema pallidum. Syphilitic spondylitis has a marked *predilection for the cervical vertebræ;* as in syphilitic osteitis elsewhere, the bones most affected are those exposed to slight trauma and pressure. Syphilitic spondylitis may occur in congenital syphilis, or during the secondary or later stages of acquired syphilis. It rarely develops before secondary skin changes occur.

Syphilitic spondylitis is characterized by localized pain (which may be more marked at night); stiffness and tenderness on palpation. It is common to find coëxisting lesions in the sternum, clavicle, tibia or radius.

Diagnostic criteria which have been proposed for a *diagnosis of syphilitic spondylitis* include: (1) the presence of active syphilis; (2) rapid improvement under antisyphilitic treatment; (3) strongly suggestive roentgen findings.

Syphilitic spondylitis may involve only one vertebra or two or three adjacent ones. The vertebræ may show bone destruction or new bone production, or a combination of both. Eburnation of the vertebral body occurs occasionally. In most cases, the vertebræ become surrounded by spur formation.

DEGENERATIVE JOINT DISEASE OF THE SPINE

(Hypertrophic Spondylitis, Osteo-arthritis of the Spine)

Oppenheimer has been foremost in stressing the importance of the apophyseal joints.[90] He showed that both rheumatoid arthritis and degenerative joint disease (osteo-arthritis) could occur at the apophyseal (small posterior intervertebral) joints and that such changes were entirely different from the average physician's concept of "arthritis of the spine" by which latter he usually means only the exostoses at the edges of the vertebral bodies, with in some cases, thinning of the intervertebral disks. The diarthrodial joints (apophyseal joints) differ anatomically from the disk synchrondroses. Oppenheimer correctly states that the reactions at the edges of the vertebral bodies are certainly osteitic, but should not be called arthritic. True arthritis of the spine is really localized in the apophyseal joints.

Degenerative disease (osteo-arthritis) of the spine is not an inflammatory process but is usually a reaction to primary changes in the intervertebral disks. Because of age, trauma and other factors, the disks become dehydrated, fibrillated, fissured, distorted, thinned, less elastic and less firm; incident to this, and probably because of the loss of the buffer effect of

the disks and the increased mobility of the vertebræ and impingement of the rims of the vertebræ on each other, there occurs sclerosis of adjacent vertebral borders, strain on the vertebral ligaments, and marginal bony proliferation with the formation of osteophytes.[1] *Degenerative joint disease (osteo-arthritis) of the spine is present in practically all patients over the age*

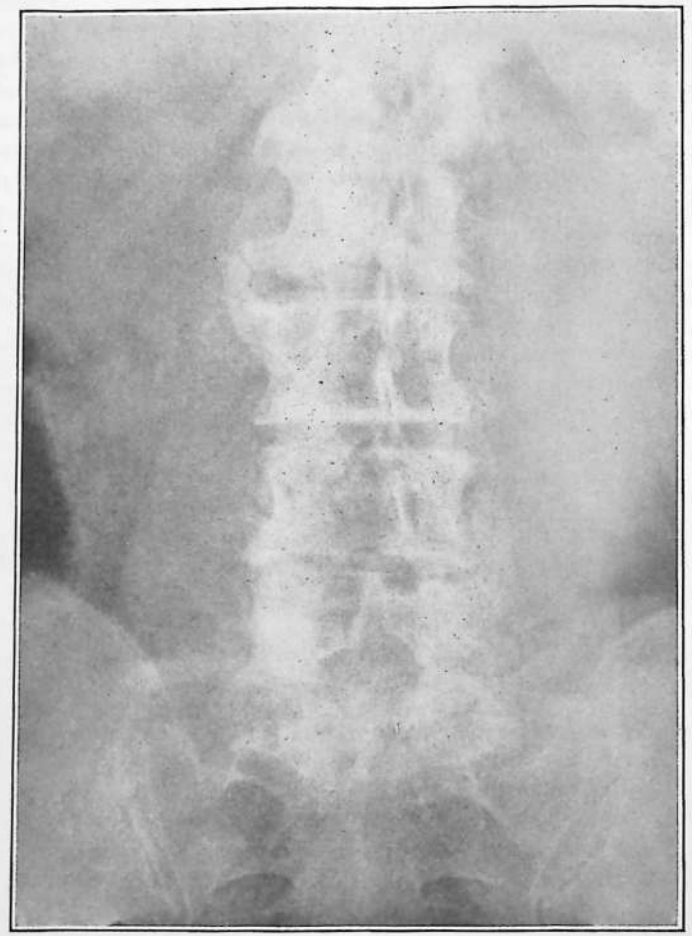

FIG. 134.—Degenerative joint disease (hypertrophic arthritis) of the spine in a patient aged seventy-three years. There are marked hypertrophic changes involving the bodies of the lumbar vertebra.

of fifty; in only a few of these is the condition severe enough to cause symptoms.[50] It is probably a degeneration accompanying age, with an added traumatic factor, especially in view of the erect posture of man.

Schmorl studied 10,000 spines during postmortem examinations and found that *osteophytes were not present in the first twenty years of life, but that they increased in frequency with age following this period.*[99] There were

11 per cent with osteophytes in the third decade, 36 per cent in the fourth, 78 in the fifth and 93 per cent in the sixth decade. *In spite of the fact that degenerative disease (osteo-arthritis) of the spine is so commonly found in postmortem examination, it seldom causes marked symptoms.*

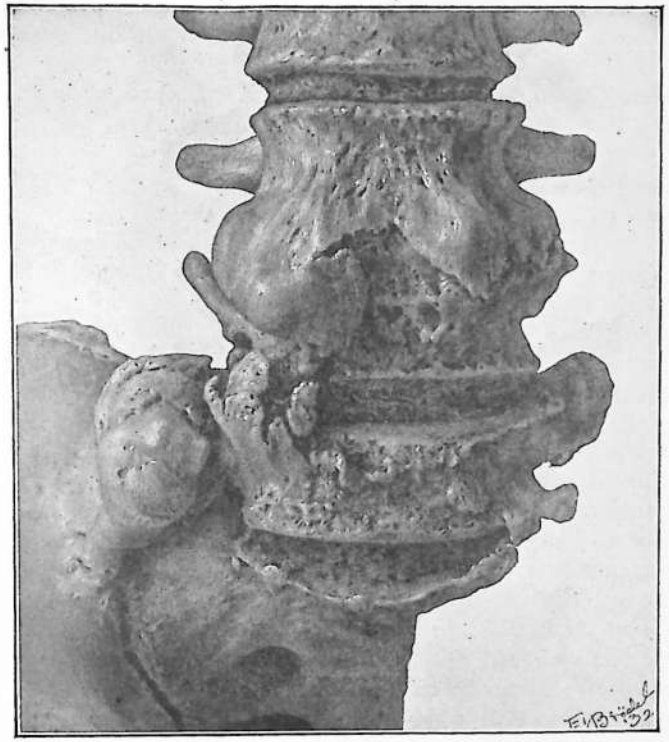

FIG. 135.—Hypertrophic (osteo) arthritis of the spine and sacro-iliac joint; note the extensive formation of bony spurs and bridges. (Shands, Orthopedic Surgery, courtesy of The C. V. Mosby Company.)

Osteo-arthritis of the spine is also called hypertrophic spondylitis (or osteo-arthrosis of the spine). These names are incorrect inasmuch as no inflammatory lesions of the spine occur in this disease. This is a degenerative process as distinguished from the inflammatory changes seen in rheumatoid spondylitis. It is similar to degenerative joint disease (osteo-arthritis) elsewhere in the body.

Bony ankylosis does not occur except through fusion of osteophytes. The **main pathologic findings** include *marginal exostoses* and *osteophytes,* preceded by *flattened margins of the bodies of the vertebræ, narrowing of the intervertebral disks and eburnation. The most commonly involved portions of the spine are the cervical and lumbar regions.*

Increased mobility of the bodies of the vertebræ on each other (due to degenerative changes in the disks) results in strain of the intervertebral

ligaments; this may stimulate the formation of new bone by the periosteum, leading to osteophyte formation.

Osteo-arthritis of the cervical spine may be the result of increased trauma along the margins of the vertebral bodies which follows thinning of the intervertebral disks (discogenetic disease).[91,128] *Degenerative disease of the spine does not result in ankylosis* except in instances where the exostoses may be of sufficient length to impinge upon each other and become united by bone.

Symptoms (Absent in Many Cases): *Root pains*, increasing *limitation of spinal motion*, and occasionally, muscular atrophy.

Areas of *anesthesia* and *paresthesia*.

Occasional *spasm of vertebral muscles*.

Headache, pains in the neck, shoulders and arms.

Horner's syndrome (enophthalmos, narrowed palpebral fissure and miosis on the affected side) if the lower cervical or upper thoracic vertebræ are involved.

Precordial pains (occasionally) with involvement of the lower cervical and thoracic vertebræ.

Nervous phenomena.[51]

In any patient with pain in the back of the head, neck, shoulder or chest which is aggravated by movements of the cervical spine, by coughing, straining or sneezing, the basic pathology is probably a degenerative disease of the cervical spine (osteo-arthritis). Because of the varied interconnections and ramifications of the cervical nerves, it is possible for an earache or unilateral sore throat to be derived from an arthritis of the cervical spine. Early morning headache may be caused by cervical spinal arthritis in as many as 40 per cent of the cases.[60]

Horwitz has analyzed the **degenerative changes in the cervical portion of the spines** of 50 adult male cadavers and has summarized the features of discogenetic disease in this region.[54] Various stages of degeneration of the cervical intervertebral disks were found in 76 per cent of specimens. Changes were most common in the lower cervical segments, involving the disk between the fifth and sixth cervical in 76 per cent, that between the fourth and fifth in 56 per cent, that between the sixth and seventh cervical in 36 per cent, etc.

The *pathologic findings* included fibrillation, fissuring, narrowing, brown degeneration and ossification of the disks associated with proportionate sclerosis of adjacent vertebral spongiosa and marginal bony proliferation, with the formation of exostoses in 70 per cent of specimens. The latter was most marked in the anterolateral part of the vertebral body and involved especially the lower four cervical vertebræ. In only one instance was there a protrusion of the intervertebral disk into the spongiosa. In three instances, the disk was ossified and was associated with fusion of the adjacent vertebral bodies.

Horwitz noted that *narrowing of the intervertebral foramina* accompanied loss of the posterior portion of the disk and exaggerated cervical lordosis; these foramina were actually widened when the entire disk was narrowed

and the normal anterior cervical curvature was lost. Forty per cent of the specimens exhibited encroachment of the lumen of intervertebral foramina anteriorly by posterior lipping of adjacent vertebral bodies, and posteriorly by marginal proliferation of articular facets covered by normal ligamenta flava.

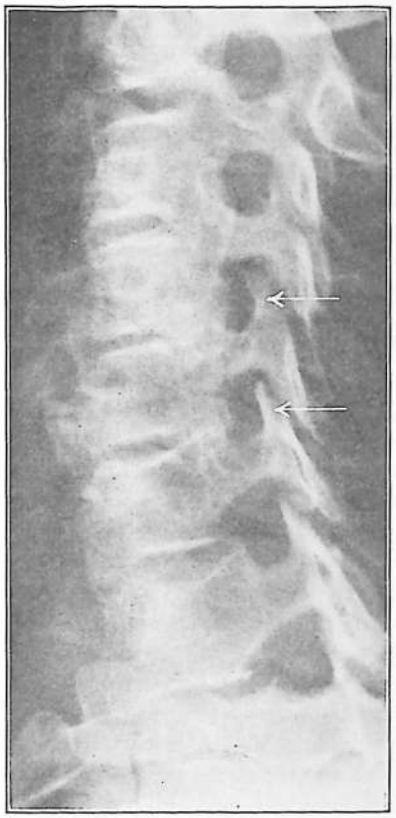

FIG. 136.– *Degenerative joint disease of the cervical spine* with narrowing and deformity of the intervertebral foramina.

The size and shape of the cervical intervertebral foramina are best visualized by *roentgenograms of the 45 degree oblique views;* few anatomic variations occur in this region of the spine. When direct lateral views are made of the cervical spine, the rays penetrate parallel to the articular surfaces, and reveal the presence of any clouding, narrowing or obliteration of the joint space, irregularity of articular margins or osteophyte formation, sclerosis of adjacent bones, etc.

If the cervical vertebræ are involved, symptoms may include *headache, sore neck, painful shoulders and arms, areas of hyperesthesia and anesthesia* and flattening of the thenar and hypothenar eminences without actual muscular paralysis.[43] On physical examination, one may find *decreased*

mobility of the cervical spine and *crepitation* (audible or palpable) *over the back of the neck* on motion of the head. If the anterior cervical roots are involved, muscular atrophy of the upper extremity and shoulder girdle may occur.[88] *The pain, soreness and aching in the cervical region may be aggravated* by movement of the spinal vertebræ, raising the head on awakening, coughing (Dejerine's sign), sneezing and straining at stool.

Thinning of the disks with unilateral or bilateral narrowing of the corresponding intervertebral foramina may lead to compression or irritation of nerve roots, resulting in pain, weakness, muscular atrophy and paresthesias in the neck, shoulder girdle, arm or precordium. The pressure of osteophytes or thickened soft tissues on the nerve roots may lead to similar symptoms. *The discomfort resulting from osteo-arthritis of the cervical spine may simulate angina pectoris,* cervical rib or scalenus anticus syndrome, neuritis of the brachial plexus, subdeltoid bursitis, arthritis of the shoulder joint and other lesions. In some cases, the overgrowth of soft tissue or bone in cervical osteo-arthritis may cause pressure on the spinal cord and produce symptoms resembling extramedullary cord lesions.[85]

Although the pain is quite variable in these cases, one of the most common complaints is pain radiating down the radial side of the arm to the forearm, together with paresthesias in the fingers. Many of these patients are apt to complain of pain in one or both shoulders or between the scapulæ rather than in the neck. It is of anatomic and diagnostic importance that the fourth cervical nerve provides sensory fibers to the top of the shoulder, while the fifth supplies the outer aspect of the arm as far down as the elbow.[59]

The motor fibers of the anterior cervical roots are fewer in number and comprise a bundle only one-third the size of the posterior sensory fibers. This may partly help to explain why cervical degenerative arthritis is able to pick out the sensory fibers in such a high percentage of cases and not involve the motor fibers at the same time.[82]

Kelly has reviewed the literature on **chronic degenerative joint disease** (hypertrophic—osteo-arthritis) **of the cervical spine, with radiculitis.**[11] He stressed the fact that many patients with pains in the shoulders and arms had a radiculitis from osteo-arthritic changes in the cervical spine, which changes may or may not be demonstrable in the *x*-ray film. Sensory symptoms are very common in these cases; almost all have some pain or paresthesias. Root pains associated with aching and soreness and aggravated by motion of the neck are usually due to degenerative joint disease of the cervical spine. This process *may produce no symptoms in many patients,* while in others it may cause localized or widespread neurologic signs.

The *outstanding characteristic* of this condition is *aggravation of all root symptoms by movement of the spine and by anything tending to increase the pressure within the meninges:* coughing, sneezing or straining (Dejerine's sign). Some of these patients are unable to sleep in a recumbent position because of the pain that may be brought on. In other individuals, pain

may be aggravated by riding, walking, raising the head from the pillow, getting out of bed, lifting, bending, or prolonged sitting.

In the cervical spine, the size of the intervertebral foramina diminishes from above downward. The sixth and seventh are the largest cervical nerves but pass through the smallest exits from the spine; thus it is not surprising that symptoms referable to the lower spine are twice as common as those of the upper because of the greater liability to mechanical irritation in that region.

Extensive marginal bony proliferation and soft tissue thickening of the costovertebral articulations may impinge upon the stellate or second thoracic ganglia, leading to peripheral vascular phenomena in the upper extremities.[54]

If the thoracic vertebræ are involved, symptoms may include:

Precordial discomfort or pain simulating angina pectoris, especially if the second to fifth dorsal vertebræ are affected.[92]

Aggravation of the pain by movement of the spine.

Indigestion (burning, heaviness or pressure in the epigastrium) if the sixth to the ninth dorsal vertebræ are affected.

Pain in the abdomen between the umbilicus and the inguinal ligaments if the tenth to the twelfth dorsal roots are involved.

Symptoms resembling appendicitis, gall bladder disease, carcinoma of the breast, pleurisy, tuberculosis, angina pectoris, etc.

Smith and Kountz and others have focused attention upon the possibility of *dorsal root pain simulating attacks of angina pectoris.*[86,110] Root irritation from degenerative joint disease of the thoracic spine may be projected around the chest or confined to one spot on the anterior chest wall; the pains may be paroxysmal or may be noted occasionally in one or both arms. Anginoid dorsal root pain may become pronounced when the patient with hypertrophic changes of the spine is reclining. In some instances, cervical rib discomfort or scalenus anticus syndrome may simulate angina. More difficulty is encountered when it is remembered that these anginoid pains from dorsal root irritation may in some instances be relieved by nitrites.

Smith and Kountz made an *experimental study of dorsal root pain using a cadaver,* in which, with the spinal cord and nerves exposed, an abnormal degree of spinal flexion and extension could be produced. When the spine was flexed or straightened to an abnormal degree, the spinal canal tended to become elongated so that the cord was drawn cephalad. This produced tension on the spinal nerves, particularly at their angulations through the spinal foramina and at their attachments to the cord. It was postulated that movement imposed on the nerves under such tension might cause irritation of the fibers with the production of referred pain.

The symptoms resulting from damage to the costovertebral or costotransverse joints have been described in detail in the section on Degenerative Joint Disease. Goldthwait has called attention to the importance of disease of these 44 small joints.[39]

In any painful progressive spondylitis, vertebral malignancy must be ruled out. The differentiation of such root pains from angina pectoris is occasionally difficult. However, the following points may prove useful in this connection:

Root Pains of Degenerative Joint Disease (Osteo-arthritis[44])
These are closely related to motion of the spine (as in bending, getting out of bed, raising the head on awakening, walking, lifting or sitting in one place for long periods).
Relief of pain by removal of aggravating mechanical factors and by the use of corsets, braces, and a firm, non-sagging mattress.
Pains are delineated by broad belt-like zones corresponding with spinal root dermatomes.
Bilateral sensory changes are usually present.

With **lesions of the lumbar vertebræ,** one may have pain in the back, radiating down the thigh, and sensory changes. Low back pain and lesions of the lumbar intervertebral disks are discussed in detail in later sections on Backache and Sciatica.

The main symptom of degenerative joint disease (osteo-arthritis) of the spine is pain of root origin radiating over one or both sides of the body. Symptoms most often follow sudden trauma or over-use. These pains are probably caused by pressure upon nerve roots in the spinal canal or as they emerge from their foramina; such pressure may be caused by osteophytes.[13,41] In addition, decreased motion of the spine and root sensory changes also occur.

Roentgen Findings in Degenerative Joint Disease (Osteo-arthritis) of the Spine.—These include *narrowing of the intervertebral spaces, flattened vertebral bodies, lipping* and the *formation of osteophytes.* Marked osteoporosis and calcification of ligaments (which are usually observed in rheumatoid spondylitis) are absent. The sacro-iliac joints are often free from roentgen changes, although anatomic changes in the sacro-iliac joints have been found accompanying increasing age.

In order of frequency, the *main sites of osteophyte formation in the vertebral bodies* are: (1) the third to the fifth lumbar, (2) the third to the sixth cervical, and (3) the fourth to the tenth thoracic vertebræ.[107] The areas in which the small intervertebral joints are especially involved in the osteo-arthritic process are the third and fourth cervical, the cervicothoracic joint, the fourth and fifth thoracic vertebræ and the second and third lumbar vertebræ.[108] It has been observed that in right-handed individuals, the largest lumbar osteophytes are on the left side and *vice versa.*[50]

Treatment.—The treatment in general is similar to that of degenerative joint disease elsewhere in the body and includes:

Avoidance of strain (to prevent recurrences).

Attempts to *improve posture* and *correct weak, painful feet.*

Rest and the liberal use of *heat* to the back (as an electric pad, hot fomentations, infra-red radiation, short wave or conventional diathermy, etc.); intermittent heat decreases muscular spasm. Snow has emphasized the rôle of physical therapy in degenerative joint disease, stressing the importance of heat (including diathermy), massage and graduated exercises.[113]

Massage (following the application of heat).

Supports to the back when necessary (including a firm, non-sag mattress or a plaster shell for the back, back brace, corsets, cervical collar, etc.).

Spinal traction and casts may be required.

Deep *roentgen-ray therapy* occasionally may give relief of discomfort with a decrease in muscular spasm.

Correct body mechanics (*i. e.*, to lessen the lumbar lordosis, forward bowing of the dorsal spine and the forward position of the head).[71]

Temporary use of analgesics for control of pain if necessary (aspirin and phenacetin, 0.3 gm. [5 gr.] of each, from two to four times a day).

Paravertebral nerve block by procaine has been required for the relief of pain in some cases.[131] If pain persists after several weeks of complete rest, search for other causes of the pain.

Kelly found some patients with osteo-arthritis of the cervical spine to be distinctly benefited by *roentgen irradiation.*

Spinal fusions are not indicated in degenerative joint disease (osteo-arthritis) of the spine.[70] There is practically always a widespread involvement of the entire spinal column. In cases in which spinal fusion has been done, pain migrates to the area above or below the fusion.

If the patient has such marked involvement that jars are carried to the spine in ordinary walking, the patient should be confined to bed.

Codeine or other narcotics are not required in degenerative joint disease of the spine. Most patients can be made comfortable with heat, rest and recumbent support to the back in a plaster shell or on a firm bed; as the muscular spasm subsides, these individuals again become comfortable when in the erect position.

In patients with persistent pain, it may be advisable to completely immobilize the spine by means of a plaster jacket with the thoracic spine in hyperextension and the lumbar spine flat.

A very satisfactory **soft support for the neck** when the patient with osteo-arthritis of the cervical spine first begins walking is a cardboard covered with sheet wadding and pulled through stockinet or a *soft collar made of thick felt,* 4 inches wide at one end, 2½ inches at the other and about 1 yard long. This is wrapped around the neck and is held in place by a tape.

Intermittent traction to the head, while it gives immediate temporary relief, is not of permanent value. Constant slight head traction in recumbency through a sling on the chin and occiput is advised when there is marked muscular spasm about the cervical spine; it is discontinued as soon as the spasm disappears. A head halter may be used to straighten the cervical spine, widen the intervertebral foramina and lessen cervical and shoulder pain. One may begin with a weight of 2 pounds and work up to 4 or 6 pounds if tolerated.

The method of gentle stretching and manipulation recommended by Hanflig *in cases of cervical arthritis* is as follows: The patient is seated on a chair under an overhead hook to which is attached a block and tackle Sayres sling suspension apparatus.[47] This sling is then adjusted, care being taken that it is well padded at the chin and occiput. Upward traction is applied until the patient's buttocks can be swung just above the seat. The patient's shoulders are now held by an assistant and the head and Sayres sling are forcibly rotated to the right and left several times; the entire procedure is repeated after the patient has been lowered and rested for a moment. A Thomas collar is made and applied; this is removed only for hot fomentations to be applied to the neck after each treatment. This treatment is performed three times on the first and second day and twice a day thereafter for several treatments. The frequency of treatments is then varied according to the progress noted. The Thomas collar may be dispensed with as soon as pain begins to disappear. When pain is decreased, active rotation, flexion and extension of the head are performed.

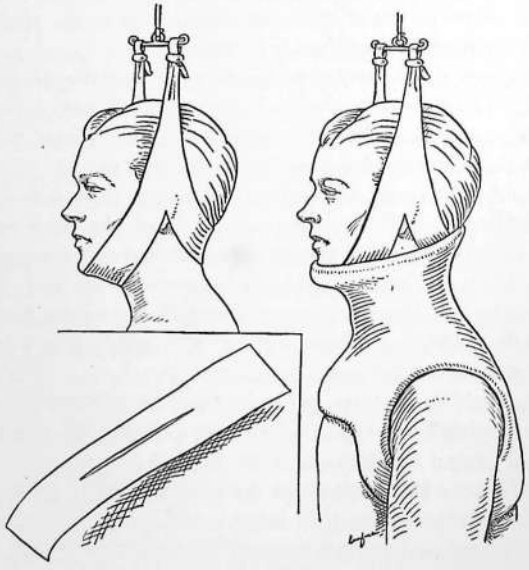

FIG. 137.—Muslin for head sling traction or suspension of head in application of cast (Lewin, Backache and Sciatic Neuritis).

McFarland and Krusen attached the Sayre head sling to an ordinary ice scale to register the number of pounds of traction which is being applied.[81] Before suspending the patient in the sling, they applied radiant heat from a luminous source for thirty minutes, followed by effleurage of the neck and shoulder. From 70 to 80 pounds of traction was then applied slowly by the Sayre head sling. When this had been reached, the head was rotated gently to the right, and then to the left, following which traction was released slowly. This was carried out twice a day for a week or more in patients with cervical degenerative joint disease, following which the patient was instructed in the use of the apparatus at home.

The rationale for head traction in degenerative joint disease of the cervical spine is that the intervertebral foramina become narrowed with resulting compression of the nerve roots emerging therefrom. This pressure results in inflammatory changes in the nerves. Exaggerated extension of the neck by traction widens the foramina and removes the pressure from the nerves. Following the release of this pressure, the inflammatory reaction of the nerves subsides.

Neviaser has used a traction manipulation for stiff and painful necks in cases of trauma, muscular involvement and chronic arthritis (ruling out first any organic vertebral disease such as fracture).[87] Following the application of heat to the neck, the patient stands on his tip toes and places his head in a halter; head traction is applied by pulling on a rope running through an overhead pulley. The slack is taken up and the patient comes down gradually, and gently until his heels barely touch the floor. Following a few minutes of head traction, the head is turned in a direction away from the involved muscles to relieve spasm. When the point of maximum rotation is reached and the chin points toward the shoulder, one may hear a click or snap.

The neck is next manipulated in forced hyperextension. One hand pushes forward at the level of the seventh cervical vertebra and the chin is pushed up and back as far as possible while maintaining traction. Finally, the head is returned to a straight position for two minutes. In acute cases, relief has been afforded within several days; in chronic cases, three to six weeks of treatment has been necessary to relieve pain and to increase the range of motion of the cervical spine.

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CHAPTER XLI

FIBROSITIS

The name fibrositis has been given to the syndrome characterized by pain, stiffness, aching or soreness, often appearing abruptly in any area of the body containing fibrous tissue (especially in muscles and around joints), more pronounced after a period of prolonged inactivity, relieved usually by mild exercise, heat, procaine injections or massage, and exhibiting practically no important physical signs except the occasional occurrence of fibrous nodules of varying sizes in the affected region. All laboratory tests are negative. The significance and even the frequent existence of such nodules has been questioned by many in this country, as well as by some in England where the syndrome seems most prevalent.

This commonly discussed condition has been the subject of very little original work. Although the term fibrositis may not be correct pathologically, there can be no question that a great many patients fall into this category. Unfortunately, most discussions of the syndrome are merely recitals of or quotations from previous papers.

Prior to World War II, some rheumatologists were not convinced that fibrositis could be classed as an entity. With the experience gained by many of our competent medical observers in the British Isles during World War II, there can no longer be any doubt concerning the existence of such a condition. It is evidently encountered much more frequently in the British Isles than in this country, and may produce symptoms which are extremely bothersome, painful and in some cases, temporarily incapacitating.

World War II has also shown us that a *large percentage* of our soldiers entering the medical service of an Army General Hospital with symptoms simulating fibrositis have developed these on a *psychogenic basis* and that these symptoms cannot be relieved by heat, massage and exercise, but are abolished by discharge from the Army which represents the only means of removing their underlying mental conflict.[10,55]

Much more convincing work than has been presented to date is necessary to portray the pathology of fibrositis. At present, this syndrome is the subject of an intensive investigation.[60] Bauer has pointed out that *we do not yet have adequate evidence that fibrous tissue is affected primarily.* It is possible that the muscle and muscle proteins may undergo *chemical change,* thus producing these symptoms.[68] *No one has yet explained why sitting in a draft should be followed by the sudden onset of a lumbago or a stiff neck.* Although we speak glibly of the fibrous tissue or muscles "jelling" following such an exposure, this is purely hypothetical.

Fibrous thickenings in "chronic muscular rheumatism" were first described by William Balfour, an Edinburgh practitioner, in 1816. His book on this subject described numerous nodular tumors and fibrous thickenings

which caused pain on pressure and referred pain. Gowers' introduction of the term fibrositis did not occur until 1904.

Stockman was the first to describe any pathologic lesions in fibrositis.[71] In Stockman's book on "Rheumatism and Arthritis" published in 1920, there are excellent chapters on fibrositis, panniculitis and Dupuytren's contracture. Llewellyn and Jones also wrote a book on fibrositis, published in 1915.[50] Telling later (1930) stressed the clinical importance of fibrositis in general practice to the English physicians and Slocumb (1936) discussed the disease in this country.[77]

The term fibrositis has been applied loosely to those individuals with vague pains, shifting from one part of the body to another and with very little or no important findings on physical examination. Some American physicians doubt the existence of this disorder, because of the paucity of pathologic findings.[27] *No pathognomonic pathologic lesions have been found.* Hench stated that the term fibrositis is still on probation in this country.[38]

Steinberg believes that fibrositis is no more an entity than is anemia, but that secondary fibrositis is part of the picture of a general infection as influenza, rheumatic fever, syphilis, rheumatoid arthritis, gonorrhea or systemic disturbances such as alcoholism, gout, plumbism, degenerative joint disease, etc. The etiology of primary fibrositis is unknown.

Halliday has pointed out that the commonly accepted theory of fibrositis is a top-heavy superstructure erected on a few simple clinical observations, namely that in certain patients with fibrositis (non-arthritic "rheumatism"), thickenings and nodules can be felt on palpation in or near the region complained of and that these may be very tender to touch and pressure.[33] Also, in certain instances, sections of tissue removed from the thickening or nodule may show minor pathologic changes of a non-consistent and indeterminate nature.

Fibrositis is considered by some to be an inflammatory reaction of fibrous tissue anywhere in the body.[18] It has been defined vaguely as a swelling and proliferation of white fibrous tissue due to injury and toxic agents; pressure upon arterioles and nerve filaments is a secondary effect.[63]

Collins has defined fibrositis as an acute or chronic painful state of muscles, subcutaneous tissues, ligaments, tendons or fasciæ, arising independently of gross anatomic disease from which pain might be referred. This pain may be felt locally at tender points or may be referred according to segmental distribution. He believes that fibrous tissue is not the only tissue concerned in the process of fibrositis, nor inflammation the only process involved. He states that factors which may initiate or aggravate fibrositis include infection, psychoneurosis, climate, trauma, occupation, endocrine disturbances and the "rheumatic diathesis."

Collins is of the opinion that a large percentage of cases of fibrositis are non-infective and arise by abnormal strain on ligaments and tendons, from trauma, exercise, fatigue or faulty posture; endocrine diseases and varieties of muscular cramps due to local vascular disturbances.[16]

"Muscular rheumatism" and "non-articular rheumatism" are usually included in the term fibrositis.

The Common Sites of Fibrositis.—Fibrous origins, insertions and aponeuroses of muscles; deep fascia; subcutaneous tissues; fibrous portions of the capsules of the larger joints, and related ligaments; muscle sheaths; supporting tissue of certain nerves. Classified according to location, fibrositis is known as:[79]

Panniculitis—fibrositis of the subcutaneous tissue.

Intramuscular fibrositis—also known as myositis or muscular rheumatism.

Periarticular fibrositis—involvement of the joint capsule.

Bursitis—bursal fibrositis.

Tendinitis—involvement of the fibrous tissue of tendons.

Interstitial neuritis—or perineural fibrositis—involvement of large nerves such as the sciatic.

The Etiology of Fibrositis.—This is unknown. Among the various agents incriminated are: Lowered general resistance, *exposure to cold and damp weather*, *trauma*, focal infection,[41] mental anxiety, worry, fatigue,[2] *chilling of the body*,[57] intestinal intoxication, chronic strain, bacterial or metabolic toxins, etc. Chilling of an area of the body which has been previously overheated is one of the commonest clinical causes of fibrositis. *Injury*, as a single or repeated chronic strain, *plays an important rôle* in its etiology (as in the lumbago of miners and gardeners). *A secondary type of fibrositis* may accompany rheumatic fever, rheumatoid arthritis, degenerative joint disease, gonorrhea, acute attacks of gout, influenza, syphilis, tuberculosis, etc.[77] It has been claimed that nearly all gouty patients have fibrositis;[19,52,54] however, there is considerable debate as to the prevalence of gouty fibrositis. It is rarely found in the chronic form of gout, but it appears during acute gouty involvement of a bursa or tendon.

The main *etiologic factors* include mental and physical trauma, occupation, climatic changes and metabolic and endocrine changes (especially *diabetes* and *hypothyroidism*). The vague pains diagnosed as fibrositic have disappeared at times following the removal of a definitely infected focus.[27] Focal infection has been shown to be a cause of fibrositis in only a few cases. Acute focal infection, such as acute streptococcal tonsillitis, may cause fibrositis.

Stockman stated that most of the original lesions resulted from bacterial infection such as "acute rheumatism" and influenza although he admitted that overstrain and cold were also capable of initiating the process. These new formations of fibrous tissue are said to be well supplied with chronically inflamed nerve twigs and are very sensitive to various disturbing elements such as cold winds and drafts, changes in weather, unusual sudden strain or muscular exertion or febrile illnesses.

Pathology.—It is doubtful if fibrositis can yet be defined on pathologic terms; however, it is a common clinical condition.

There are no convincing pathologic studies in this disease. The editorial committee of the American Rheumatism Association stated that American clinicians have hesitated to recognize fibrositis as an entity

because of its ill-defined pathology, its subjective symptoms and the lack of any abnormal chemical reactions.[36]

There are no Pathognomonic Pathologic Lesions Found in This Disease.—There is no typical microscopic appearance of the fibrositic nodule whereby it can be identified histologically.[58] Biopsies have not been performed in large numbers and, when made, the pathologic picture is vague. Stockman first described the inflammatory hyperplasia of connective tissue in this disorder.[74] Slocumb discussed three stages of the process: (1) a low grade inflammatory serofibrinous exudate with proliferation of fibroblasts and blood-vessels; (2) local tissue thickenings or gross subcutaneous nodules; and (3) fibrous contractures or capsular thickenings in the shoulders, palms, etc.[66]

Stockman described the *pathology of fibrositis* as an inflammatory hyperplasia of the white fibrous tissue of muscles, nerves, fasciæ, etc., in small patches so that the affected areas become swollen and edematous. There is an outpouring of serofibrinous exudation, rapid proliferation of fibroblasts, appearance of numerous minute new blood-vessels and absence of polymorphonuclear leukocytes in this exudate. There is no formation of pus. The resultant swelling is usually soft, small and ill-defined. These inflamed excrescences may then resolve spontaneously or from therapy or more often increase in size because of proliferation of connective tissue cells so that the mass becomes tough and fibrous.[75]

The larger of these lesions are said to be the size of an almond and can be felt as ill-defined swellings along the edges of muscles or in subcutaneous tissue and fascia. These new formations may be scattered all over the body in large numbers or may be confined to one or a few parts; much of this changed tissue is too minute to be detected except by its tenderness on pressure.

Steinberg suggested that the pathology of primary fibrositis and that of nutritional muscular dystrophy of young rats is very similar. It has been shown that alpha tocopherol will prevent or cure this disturbance in rats.[71]

Fibrositis is found most often in the middle-aged individuals in positions of the body most subject to strain: the lumbar region (of gardeners), the shoulders (of miners, etc.).[31]

Symptoms.—Clinically, fibrositis is characterized by pain and stiffness, particularly on movement, occurring in the subcutaneous tissues, in fibrous origins, insertions and aponeuroses of muscles, in fascia, ligaments or tendons, in the fibrous portions of the joint capsule and in the supporting tissues of some nerves.[27]

The *main features of fibrositis are pain, stiffness, aching and limitation of motion with little or no effect on the general health.* The process may be acute, subacute or chronic.[20,32,46] The resulting incapacity varies from mild stiffness after a period of rest to inability to move any muscle of the body because of pain consequent to the motion. Pain on motion of the part and muscular stiffness are often the only symptoms. Others may complain of sore, tender areas with occasional spasm of the surrounding muscles.

Symptoms of Fibrositis

Pain, stiffness, aching or *soreness*, often appearing abruptly in any area (especially in muscles or around joints) where fibrous tissue is present; *more pronounced* on awakening or *after periods of prolonged inactivity;* relieved by mild exercise.

Practically *no physical findings except* the occasional appearance of *fibrous nodules* (tender or painless) of varying sizes in the affected region. These are composed of fibrous tissue and may occur in non-painful regions. They do not necessarily indicate a recent bout of fibrositis but may be the result of previous attacks. Some clinicians ignore these as they may be found in "normal individuals" (possibly due to former bouts of the disease). The detection of such nodules has probably been stressed too much.

Intense headache resulting from cervical fibrositis; pain in the chest from intercostal fibrositis; abdominal pain from involvement of rectus or other muscles; pain low in the back (lumbar fibrositis—lumbago), etc. *Lumbago* is merely fibrositis of the muscles or ligaments of the low back, often appearing after chilling of the low back following previous overheating of this area.

May resemble neuritis because of occasional occurrence of perineural fibrositis.

Attacks of fibrositis are characterized by pain apparently induced by wear and tear, sudden strain, chilling or worry, or various combinations of these.[31]

The pain of fibrositis may be dull or sharp, continuous or intermittent.[37] It is occasionally knifelike or burning like a hot poker. *The pain and stiffness are usually worse on awakening or after any period of inactivity for several hours; both symptoms are usually decreased following mild exercise.* The pain is usually a dull ache, most marked in the morning; it is usually associated with stiffness. The pain decreases and the stiffness lessens as the part is exercised. It frequently recurs throughout the day following any prolonged period of inactivity, such as sitting in a theatre, sitting for long periods at a desk or table, etc.[27] It may recur in the evening and may be marked after several hours of rest in bed at night. Some of these individuals are so stiff and achy after a full night's sleep that they deliberately are awakened by an alarm clock two or three times a night so that they can get up and walk around for a few minutes, to lessen their stiffness and soreness; after doing this, they do not feel nearly as bad on awakening.

Special movements or pressure may cause sharp twinges of pain. Recurrences are frequently caused by local change in temperature, trauma, mental stress or exposure to dampness. In some cases, it is probable that the pain is psychogenic as an outward manifestation of emotional disturbance. The common varieties of fibrositis are the intramuscular and the periarticular forms, both being present together in about 50 per cent of the cases. In most cases, "myositis" or "muscular rheumatism" is actually fibrositis.

Periarticular fibrositis denotes involvement of the capsule and ligaments surrounding various joints. Perineural fibrositis denotes involvement of a nerve sheath (such as the sciatic). One frequently notes involvement of the palmar fascia, fascia lata and inguinal ligament, as well as various tendons, fasciæ, aponeuroses and bursæ. Involvement of the subcutaneous fibroareolar and adipose tissue also occurs.

The *muscles most frequently involved* by fibrositis include the trapezius, sternocleidomastoid, intercostals, latissimus dorsi, serratus muscles, semispinal dorsi and cervicis, splenius capitis and cervicis, sacrospinalis and semispinalis capitis and the small deep muscles of the back as well as the muscles of the scapula (supra- and infraspinatus, rhomboids, teres major) and the deltoid. The muscles of the anterior abdomen are occasionally involved.[71]

No age group is exempt; the acute form is more common in young individuals and the chronic is seen more frequently in middle age. Involvement of the lumbar region (lumbago) and sciatica occur chiefly in men; panniculitis is more common in women at the menopause and is often associated with hypothyroidism.

Fibrositis may involve various areas of the body, resulting in local symptoms.[11] It has produced severe occipital and vertical headache, stiff neck (acute wryneck or torticollis), pain in the chest (pleurodynia, devil's grip), abdominal pain (with involvement of the rectus or other muscles), pain and stiffness around the joints (often misdiagnosed arthritis), pain in the distribution of large nerves (brachial plexus, sciatic nerve[1,17,24]), lumbago, intercostal neuralgia, etc. Lumbago is a fibrositis affecting especially the lumbar muscles. It is one of the commonest causes of pain in the low back. It is seen very commonly in the early spring or summer in the amateur gardener who perspires markedly and is then exposed to the chilling of the evening air. Some have considered *Dupuytren's contraction* to be fibrositis of the palmar aponeurosis.

Severe headache is frequently caused by fibrositis; this occurs chiefly at the occiput. If the great occipital nerve is affected, a perineuritic pain may be noted along the course of the nerve up the back of the head. In other cases, the scalp may be involved. Fibrositis of the frontal fascia, often involving the supra-orbital nerves, results in frontal headache.

Ellman has suggested that a further test which is useful in some cases is to increase the tension in the suspected structure—as by contraction in a muscle, or stretching in the case of a tendon, ligament or band of fascia; if the structure contains the fibrositic area, such a maneuver will reproduce the pain.

Although muscular atrophy appears very slowly, if at all, in fibrositis, it may occur following repeated attacks in the same area when loss of power also follows.

In Ellman's[50] patients, pain was confined to a group of muscles in half of the group; 14 of these had accompanying pain in various parts of the body and in another 6 there was also pain which traveled or shifted from one part to another. In 4 individuals, pain was noticed "all over the body." Adjectives used to describe their pain included shooting, twisting, tearing, piercing, burning or gnawing; in the emotional individuals, the adjectives used included excruciating, agonizing, nagging and depressing.[27]

In about two-thirds of the patients, the pain was intermittent, being continuous in the remainder. Exertion, exposure to dampness and drafts and cold aggravated their comfort in most of the patients.

Practically all of these patients experienced minor degrees of *stiffness.* In about half, actual limitation of motion was noticed. In 3 of the individuals, straightening was very difficult after marked forward bending.

In 40 per cent, some weakness of the affected part resulted with limitation of motion and general lassitude.

Crepitus which is often felt in the neck and which is so often attributed to arthritis of the cervical spine, is often due to friction between muscles and adjacent structures—much like the crepitus of the knees in villous arthritis.

Attacks are often initiated or aggravated by dampness, cold or drafts and either warm weather or the application of warmth usually gives relief of discomfort. Overexertion aggravates the symptoms; slight to moderate exertion produces comfort and a limbering up of the stiffness.[71]

Temporary deformities, such as traction of the head to one side or lateral curvature of the spine may be caused by fibrositis.

Young individuals do not often have symptoms of primary fibrositis. Its peak incidence is in the fifth decade although it is usually manifest first in the latter part of the fourth decade. Both men and women are affected. It usually affects apparently healthy individuals.

Attacks are usually acute; however, the process may be chronic. The acute attacks usually persist for several weeks.

Pain or tenderness may be localized or generalized involving a very small area of a muscle or whole group of muscles. Generalized stiffness is usually a concomitant symptom.

In elderly patients with fibrositis, muscle spasm may completely obliterate the indurated areas. The diagnosis of fibrositis in the elderly is based on the presence of stiffness in the muscles with pain on stretching, muscular spasm and in some cases, the presence of nodules or indurated, tender areas, without signs of rheumatoid arthritis or other causative factors.[28]

Nodules of Fibrositis.—These have probably been overstressed as a diagnostic aid; obvious visible nodules are rarely found. Krusen questioned whether fibrositic nodules were as important as many writers believed and whether they could be rubbed away as consistently as some stated.[49] Buckley believes that the importance of fibrositic nodules has been overestimated, as these are often asymptomatic. They can be felt often in the superficial fibrous tissue of the buttock, just below the iliac crest.[13]

They vary in size from a pea to an almond, and can be dissected out only with difficulty.[76] Some have stated they are "only accessible to the finger of faith."[37] Various observers have reported these as large nodules

in fibrous aponeuroses, small nodules usually in muscle bundles, and small, sharply localized hard areas in muscular tissue.

They may be tender or painless, and seem to occur most commonly in severe cases.[30,35]

They present no definite microscopic picture, at times containing fibrous tissue and at others not capable of differentiation from the surrounding structures.

Physical examination usually shows no important findings which can be attributed solely to the fibrositis. The importance of the fibrositic nodules has been disputed. Some feel that these nodules may be the scars of previous trauma or inflammation. These vary from small round areas to large plaques or thick fibrous sheaths or septa. In some instances they may be too deep to be palpable or muscular spasm may prevent their detection. They are often felt in individuals who have never complained of fibrositic symptoms.

These nodules may or may not be tender. If the area of thickening is responsible for the patient's symptoms, palpation of this area should reproduce his pain and infiltration with procaine should temporarily relieve it. Although enthusiasts for fibrositis claim that they are able to palpate nodules in a large percentage of these patients, many physicians are able to detect nodules in only a very small percentage of such sufferers even after the most careful examination.[33] On the other hand, extremists assert that this is because sufficient patience and practice have not been given to the examination. In many instances, nodules are found in patients who have no complaints of fibrositis whatsoever.

Copeman discussed the rôle of **"myalgic spots"** or **"rheumatic nodules"** in fibrositis. In his opinion, the myalgic spot is an early stage of what may later become a nodule, if left untreated.[21] When the nodule is definite enough to be palpated, Copeman believed that this was a sign of chronicity of fibrositis. The myalgic spot is an area of hyperesthesia in a muscle or tendon sheath which produces local or referred pain when stimulated. The spots are generally multiple and occur most often in the powerful muscles of the back and gluteal region, often toward the edge of the muscle where it blends with the more fibrinous portion. It occurs much less frequently in the peripheral muscles and in the anterior muscles of the trunk.

Although many observers believe that trauma plays a large part in the development of the myalgic spots or nodules, their etiology and pathology are still unknown or very poorly defined.

Copeman noted that in an epidemic of influenza in which lumbar and other pain was a prominent feature, pain was also referred from small hypersensitive areas in the muscles (indistinguishable from "rheumatic myalgic spots."[21] On re-examination of these patients one and two months later, these spots were still present, but in diminishing numbers. The patient was usually not aware of this until pressure from the examining finger disclosed them. These patients did not have a previous rheumatic history. Copeman believed that myalgic spots could arise in the

course of acute influenza and that some of these persisted, and could later be reactivated by other types of infection. It was suggested that the activity of some of these myalgic spots was maintained by the common cold.

Copeman suggested that the *myalgic spots* which *are a legacy from acute infections of earlier years* may form the basis of many rheumatic syndromes of later life. These victims are usually unaware of the presence of such spots until they may become activated by trauma, chilling or systemic infection.

Gordon considers the fibrositic nodule to be the hallmark of the fibrositic subject but the tombstone of the fibrositic attack.

Laboratory Findings.—These are usually normal (including the complete blood count, sedimentation rate, blood serum proteins, calcium, phosphorus, etc.).[61] In occasional cases, the sedimentation rate may be somewhat increased and in some acute cases, a slight leukocytosis occurs.

Roentgen-rays of the involved areas are negative. Cultures of nodules removed or of biopsies show no bacterial growth.

Differential Diagnosis of Fibrositis From Rheumatoid Arthritis

	Fibrositis	Rheumatoid Arthritis
Joint pain	Pain is periarticular; usually worse on awakening; improved following gentle exercise; made worse by rest	Definite joint pain; often increased following exercise; improved by rest
Joint swelling	No synovial exudate	Synovial exudate frequent
Joint tenderness	Absent or very mild	May be marked
Stiffness of joints	Rarely objective; usually subjective only	Commonly subjective and objective
Muscular atrophy	Absent	Common
Remissions	Frequent	Uncommon
Constitutional phenomena	Usually absent; usually no loss of weight	Fever, tachycardia, *loss of weight*, etc.
Sedimentation rate	Normal; occasionally elevated	Markedly elevated in most cases
Anemia	Rare	Common
Roentgen-rays of joints	No bony changes	Definite findings
Prognosis	Good, from the standpoint of the joints; recurrences are frequent	Very uncertain
Response to therapy	Very variable; occasionally prompt	Slow

Differential Diagnosis.—Periarticular fibrositis is often confused with rheumatoid arthritis. It is important to attempt to separate the two because of the difference in treatment and in prognosis. The subjective symptoms of stiffness, aching and limbering up are much the same in primary fibrositis as in the secondary fibrositis which accompanies rheumatoid arthritis (as periarticular, intramuscular, tendinous, etc.). However, in true rheumatoid arthritis, the pain produced by intra-articular disease gradually increases in the damaged joints; on the other hand, real joint pain is absent in fibrositis.

Many common diseases are evidence of primary fibrositis but masquerade under such names as myositis, torticollis, lumbago, tendinitis, muscular rheumatism, periarticular fibrositis, panniculitis, etc.

Epicranial and cervical fibrositis often give rise to headache; pectoral fibrositis may simulate anginal attacks and intercostal fibrositis may be confused with pleurisy.[56]

Fibrositis of the muscles and ligaments of the back may simulate angina, appendicitis, cholecystitis and duodenal ulcer.[31,34]

Pain may occur in muscles, joints and fascia frequently in the early stages of various acute febrile diseases such as meningitis, influenza, typhoid fever, typhus fever, scarlet fever, relapsing fever, etc., and may last during the entire course of others, as brucellosis, dengue, etc. *Epidemic myalgia* (devil's grip, epidemic pleurodynia, Bornholm disease) is an acute febrile illness whose major symptom is muscular and tendinous pain; although its cause is unknown, recovery is complete. Non-articular skeletal pains also occur in rheumatic fever, trichiniasis, dermatomyositis, etc. Painful conditions of muscle and connective tissue may result from bacterial septicemia or toxemia or from pyemia and some specific infections.

A *febrile relapsing nodular non-suppurative panniculitis (Weber-Christian's disease)* has been described.[4] Crops of subcutaneous nodules appear during episodes of fever; pathologic examination reveals that there is an associated peculiar type of fat atrophy. The disease is most common in women. Nodules are found most frequently on the thighs. Death has been reported occasionally in this syndrome.

Binkley reviewed the syndrome of relapsing febrile nodular non-suppurative panniculitis (Weber-Christian disease).[15,81] This process is characterized by recurrent attacks of malaise and fever of varying degrees, associated with subcutaneous nodules which appear to be localized inflammation of the panniculus adiposus.[59] The nodules may show varying degrees of tenderness, erythema and mottled brawny pigmentation.[8] They do not suppurate and may undergo spontaneous regression, resulting frequently in localized areas of atrophy and depressions in the contour of the skin.[23,6] The syndrome may be confused with migratory phlebitis, erythema nodosum, erythema induratum and nodular syphilis. Less than 20 cases had been reported in the literature until recently. The great majority of these were in females.

Obesity has been a common feature. Some of the patients have been seriously ill during the febrile attacks, but this was unusual.[4]

The excised nodules show fat necrosis and liquefaction with a mild degree of fibrosis. In most instances, the nodules were found in the subcutaneous tissues of the trunk and extremities. Binkley reported a case in which the nodules appeared first in the panniculus adiposus of the breasts, where they remained predominant.[8] Aspiration of these nodules produced a thick, greenish yellow sterile mucoid material; similar material was obtained from nodules irrespective of their location and was reported as liquefied fat containing rare leukocytes. The nodules regressed in three to four weeks, but sporadic new nodules made their appearance on occasions, usually associated with mild malaise. There was some residual evidence of thickening and persistent cutaneous attachment in some instances. During a follow-up of a year, two minor episodes occurred. Each was associated with areas of nodular panniculitis, chilly sensations, relapsing fever and mild pains in the joints.

Some cases of fibrositis have been called neuritis or sciatica. Many of these have made pilgrimages from physician to physician. In one of Ellman's patients aged fifty-three, symptoms had been present since the age of thirteen and had resulted in numerous wrong diagnoses including chronic nephritis, cardiac dilatation, appendicitis and uterine displacement.

Disabling changes in the hands resembling sclerodactylia may *follow myocardial infarction* in as many as 20 per cent of cases.[44] Pain and stiffness of the fingers are usually the first symptoms. These appear from three to sixteen weeks after the acute myocardial infarction. Although the swelling of the hands and fingers subsides in time, the stiffness and pain persist. Some of these patients develop marked contractures of the fingers but others have few residual deformities and little decrease in motion.

Kehl reported 6 cases of *Dupuytren's contracture* occurring *after myocardial infarction.*[45] The palmar changes which appeared were typical of Dupuytren's contracture, beginning usually as a firm nodule in the palmar aponeurosis, most often in the ulnar area near the metacarpophalangeal joint, and progressing slowly. It extended gradually to involve the whole palmar fascia, excluding the tendons. In some instances, these changes may suggest at their onset the diagnosis of fibrositis.

Halliday has stressed the *differentiation between true fibrositis and* **"psychoneurotic rheumatism."**[33] He suggested that a psychologic approach be a supplement to the routine medical examination in these patients—which might point out a diagnosis of hysteria, chronic anxiety state or psychotic depression. This supplementary examination may show that the fibrositis emerged during a period of intense emotional stress and point out why the fibrositis is often "cured" when emotional stress is relieved.

The ungainly term, psychoneurotic (or **psychogenic**) **rheumatism** indicates the thought that emotional upsets may bring about upsets in the body that include subjective symptoms of pain, stiffness and limitation of motion which are often diagnosed as fibrositis, lumbago, etc. It has long been accepted that pain may be a symptom of hysteria, but until recently, due attention has not been given to the pains, aches and stiffness that may accompany chronic psychoneurotic anxiety states or psychotic depressions.[33]

The relevance of psychologic factors to illness was recognized by physicians from earliest times, but with the development of the mechanistic outlook during the nineteenth century, their significance and importance were greatly subordinated or forgotten. Fortunately, the modern study of psychoneurotic illness has enabled these reactions to be formulated in a scientific way.

Ellman and his associates performed a routine clinical and a special psychologic examination on 32 military and 18 civilian patients diagnosed

as fibrositis. Only cases of over three months' duration were included. Physical abnormality was found in about half of these individuals; among the abnormalities noted were: (1) tender, localized areas or nodules where the specific pain was produced by pressure and abolished by procaine infiltration; (2) focal infection; (3) postural spinal abnormalities; (4) a history of sacro-iliac strain with radiologic irregularity of the articular surfaces and accompanying tenderness, wasting and spasm on movement of the surrounding muscles. About half of the patients showed some manifestations of hysteria; 7 had anxiety states and 3 had depressive states. There was only 1 malingerer.[27]

In their experience, a strictly localized pain indicated the presence of organic change while *vague shifting pains were suggestive of an underlying emotional disorder.* Very few of those with gross organic changes had significant psychologic abnormalities while the majority of individuals with very little organic change had significant psychologic disorders.

Halliday has urged that a *supplementary examination he made of psychologic factors in all cases of fibrositis.* This includes an etiologic investigation of the patient's personal relationships, occupational problems, financial circumstances, quarrels and bereavement and fears and knowledge of disease; "looking upon the man"—*i. e.*, a consideration of his face and manner; and finally an extension of the history-taking backwards in time to include disturbances in psyche which preceded the disturbances in the soma. The patient's facial expression may be of extreme significance—*i. e.*, strained, tired, worried, hunted, placid, smiling, restful, etc. A psychologic approach will determine what kind of a person the patient was before he developed his symptoms; what external events preceded this response; why he developed these symptoms and why the patient took these symptoms in that particular way.

It has been pointed out that many individuals with symptoms of fibrositis have been unduly independent, conscientious, rigid, stiff or touchy before they became ill and have suffered from psychoneurotic illness in the form of breakdown or ill-defined gastritides.

Hysterical pains have been found to be common in persons engaged in occupations which expose the individual to excessive bodily danger, such as underground miners, steeplejacks, window-cleaners, steel-erectors, etc., as well as in those manufacturing explosives or in those sports which may entail a high degree of bodily risk such as football.[33] *Pains, aches, stiffness and limitation of motion are very common as episodes in chronic psychoneurotic anxiety states and in psychotic depressions.* In some series, as many as one-third of patients labeled fibrositis fall into this category. Unfortunately, in the absence of a simple psychologic approach as a supplement to ordinary physical examination, much incapacity (which should have been preventable at the beginning) becomes permanent and fixed. Although the term fibrositis may be of value to the pathologist, this name is somewhat misleading to the general practitioner who immediately believes that he is dealing with an inflammation and subordinates all of his therapy to this line of thought.

Boland and Corr reported that **psychogenic rheumatism** *was the most frequent cause of disability in 450 consecutive cases diagnosed as arthritis or an allied organic condition* previous to admission to the medical service of an Army General Hospital. By the term psychogenic rheumatism is meant those states in which pain, stiffness and a subjective sense of swelling or limitation of motion in joints or muscles are caused, intensified or perpetuated by mental influences.[10] In the absence of structural joint or muscle abnormalities, the condition has been designated as pure psychogenic rheumatism; the psychogenic rheumatism is considered to be superimposed when the psychogenic symptoms are associated with nondisabling organic changes.

Although functional disturbances have long been recognized in cardiology, gastroenterology, neurology and other fields, the concept of psychogenic rheumatism has not been fully appreciated.

In Boland and Corr's series of 450 patients with rheumatic symptoms, pure *psychogenic rheumatism was more often responsible for peripheral joint and muscle symptoms than were rheumatoid arthritis, rheumatic fever or degenerative joint disease.* It was also of interest that psychogenic rheumatism was one of the most frequent causes of complaints referable to the back. In 1 out of every 5 rheumatic patients, the symptoms were purely on a psychogenic basis and in 1 out of 7 the individual was disabled by psychogenic factors superimposed on mild organic pathologic changes.

Fifty cases of psychogenic rheumatism were studied in detail. In 38, there was a history of peripheral joint or back symptoms prior to entry into military service. In 38 of the 50 cases, symptoms were limited to the back and lower extremities. The upper extremities were the sole site of somatic fixation in only 1 instance. In 8 cases, complaints were present in practically all joints. The majority of these patients had definite psychoneurotic manifestations before entry into military service. In one-third of the group, there was a history of hysterical or disabling psychoneurotic episodes prior to the onset of the presenting complaint (ranging from attempted suicide to interruptions in schooling because of nervousness). Others gave a history of nightmares, temper tantrums, enuresis, somnambulism or other personality or behavior problems. At the time of examination, 28 of the 50 had a greater or lesser degree of anxiety state. Sixteen showed a mild mental depression.

In 46 of the 50 patients there were definite associated psychoneurotic symptoms in addition to the rheumatic complaints. The most frequent symptoms in the peripheral group were pain, stiffness, limitation of motion and a subjective sense of swelling and weakness of the involved part. All complained of pain, which was usually aggravated by physical activity incident to military service. The patient often interpreted as pain such symptoms as numbness, tingling, pricking, burning, weakness, deadness or fulness. About half of the group noted stiffness and a subjective sense of swelling. In 15, bizarre limps were seen, some of which were associated with abnormal postures which were sufficient in themselves to cause pain and muscle spasm.

Pain and stiffness were encountered in all of the patients with backache. After close questioning, this often became a tired feeling, weakness, a dead or numb sensation or localized burning in the back, especially in the lumbosacral area. True restriction of motion of the back was rare, but a variable degree of rigidity of the back was common. It was often evident during the examination that the apparent immobility was due to pain on forced motion and muscle spasm and not to structural change.

One of the most important characteristics in the group with back pain was the persistence of the disability despite prolonged rest in bed. *Typical of this psychogenic backache was the continuous discomfort night and day which was refractory to physical therapy or rest in bed and was aggravated by slight physical exertion.* Five of the patients showed anesthesia or hypesthesia of the hysterical type; there were 2 cases of associated hysterical paresis of an extremity. One of the unusual sites seen was a patient with advanced rheumatoid spondylitis pushing another with purely psychogenic backache to the post exchange in a wheel chair!

When an attempt was made to return these patients to duty, they returned to the hospital in from four hours to three months with the same or intensified symptoms. *At least 46 of this group of 50 patients with psychogenic rheumatism were eventually discharged from military service.* Trials at limited service were unsuccessful.

Disabling pyschogenic complaints as observed in an Army General Hospital are seen frequently in the muscles and joints and are comparable in occurrence to similar complaints in the cardiovascular and gastrointestinal systems.

The predominant localization of psychogenic musculoskeletal symptoms in the lower extremities and back in soldiers may be their unconscious attempt to solve the desire for separation from military service as these structures are synonymous with marching and soldiering. When a psychoneurotic predisposition is present, it is not surprising that such a group might substitute bodily distress for the emotional conflicts growing out of the severe stresses incident to military service and to the transition from civil to army life.

It is often difficult to elicit the immediate precipitating emotional causes in some cases. Manifold factors induce anxiety states in soldiers; the loss of security caused by separation from home; the resentment of authority, especially when this is invested in those who are considered inferior; fear of bodily harm; confusion associated with strange surroundings, crowding, regimentation; loss of control of one's personal destiny; concern for safety and financial well-being of dependents, etc.

Psychogenic rheumatism may be suggested by certain observations:[10] (1) The persistence of disability despite rest in bed, physical therapy and other therapeutic measures; (2) association with other psychoneurotic or hysterical manifestations; (3) gross incongruity between the severity or quality of the symptoms and the structural changes; and (4) bizarre limps or postures.

Boland and Corr diagnosed primary intramuscular or periarticular

fibrositis in only 5.8 per cent of their medical admissions to an Army General Hospital (which is in sharp contrast to the figure of 70 per cent observed by Copeman and Hutchison in French or British military hospitals). Boland and Corr ventured the diagnosis of fibrositis only when a typical history was elicited in an emotionally stable individual. They used as criteria for the diagnosis of fibrositis aggravation of the symptoms by periods of physical inactivity, dampness or weather changes and relief by moderated exercise or heat. In the absence of significant organic changes, it is extremely difficult to evaluate symptoms such as aching and stiffness, and particularly when these are located in the back. They believe that the nervous irritability and fatigue which accompany fibrositis so frequently have not been properly evaluated and that in many instances these symptoms are part of a neurasthenia in which the stiffness and aching serve as the predominant psychogenic manifestations. *Psychogenic rheumatism may be separated from fibrositis by an evaluation of the patient's make-up, finding a precipitating psychogenic factor and recognizing any associated psychoneurotic complaints.*

These patients are not malingerers; in Army practice, true malingering has been very uncommon when compared to the frequency with which somatic symptoms develop unconsciously as an escape mechanism from the unpleasant associations of military service.

In most of these patients with psychogenic rheumatism in the Army, Boland and Corr concluded that the only solution was separating them from military service, which in turn was the only means of removing their underlying mental conflict. In many, the underlying emotional make-up was so poor and the associated psychoneurotic manifestation so definite it was neither advisable nor feasible to attempt rehabilitation for military service. It is hoped that *more prompt recognition and proper psychotherapy instead of physical therapy will prevent some of these casualties from psychogenic rheumatism.*

Bornholm disease is the only condition characterized mainly by muscle pain which occurs in epidemic form. In Bornholm disease, one commonly sees high fever, prostration and pleural and abdominal pain. The terms Bornholm disease and epidemic myalgia are used synonymously by many individuals.[6]

Fibrositis commonly involves the trapezius muscle and is probably one of the most common forms of rheumatic disease seen in general practice.

The common condition of **"stiff neck"** or **acute torticollis** may be another form of **epidemic myalgia.** In 1941, stiff neck was prevalent in southern England. Beeson and McNair Scott accumulated data on a group of factory workers in this region.

During a period of two months, these workers found 18 per cent of 163 factory workers to present a **"stiff neck."** This was more common in women employees (most of whom were in the third decade) than in the males (most of whom were in the fifth decade). Some of these individuals were incapacitated for two weeks or more, although the disease was usually mild. The clinical features resembled those of acute fibrositis with

localized areas of muscular tenderness, tingling sensations in the extremities and in which systemic manifestations were lacking. This was in contrast to the usual features of Bornholm disease in which marked systemic reaction is common.

These patients usually give a history of a fairly sudden onset of pain in one or both shoulders which frequently shoots up the neck or down the arms and may simulate brachial neuritis.

The clinical picture noted by these workers in the typical patient with acute torticollis was as follows:

1. The head was held stiffly and the neck was not moved.
2. Pain was noted in some part of the trapezius muscle although the sternomastoid or deltoid muscles were also involved in most instances.
3. The most frequent sites of pain in the trapezius were the (*a*) occipital attachment; (*b*) the superior margin of the muscle midway between the acromion and the occiput; and (*c*) the portion adjacent to the lower cervical spine.
4. Occasionally the pain extended to other regions as the lumbar, gluteal, forearm and arm regions; multiple sites of pain were the rule.
5. The pain was usually described as an ache, being sharp in a few and a sensation of stiffness in several.
6. Most of these individuals were unable to turn the head from side to side.
7. General symptoms were usually absent—the patients feeling well except for a pain in the neck or shoulder.
8. The duration of symptoms was usually a few days in most instances, although these occasionally persisted for several weeks.

Fibrositis in Industrial Workers.—Fibrositis of the muscles or the neck is common among taxi drivers and market porters and those carrying heavy weight on their heads. Fibrositis of the arm and hand muscles is seen often in gardeners, locomotive firemen, bus conductors and dustmen. Fibrositis of the back and gluteal muscles includes the largest group of occupational fibrositis: bus drivers, miners, policemen, gardeners, stokers, iron workers, women engaged in lifting and milkmen.[69]

Trauma and muscular fatigue are common factors in these individuals, as well as exposure to cold, humidity and heat. The trauma is often slight, but recurrent.

Muscular and ligamentous strains are important predisposing causes in industrial fibrositis. Most of these individuals are *sensitive to cold*, and have bluish limbs and frequently have had chilblains. Postmen, who maintain a good circulation by constant walking, do not develop fibrositis often, despite their exposure to inclement weather.

The occupational fibrositis of the neck usually begins with discomfort in the neck muscles. The patient may complain of a fairly severe headache for a day or two, in the occipital region. The pain may later become localized in the trapezius or other muscles, so that motion of the head becomes restricted and painful. Pain may be continuous and burning and may radiate through the neck. This may be caused by frequently repeated stretching and slight rupturing of muscles or attachments in the neck with smaller or larger hemorrhages present in some of these cases.

Slot feels that heat is the cardinal medicament—especially hot moist heat—such as a hot tub bath (105° F.) into which has been placed ¼ pound or more of magnesium sulfate. The patients soak in this, and attempts to move the painful portion gently under the water. Other useful methods include infra-red rays, diathermy and gentle massage.

Injection of procaine (1 per cent) into the affected muscles is probably the most valuable single agent.

In sciatica, Slot has had best results from epidural therapy and manipulation.

Rest in bed for a day or two, with aspirin, 0.6 gm., every three or four hours, is very helpful. Many of these patients are tired and physically exhausted.

The animal body contains at least *434 muscles*, constituting about 45 per cent of the body weight. Muscles are subject to wear and tear during a large part of the day.[29]

Good discussed myalgia in detail. Its symptoms are pain and local tenderness. Myalgia is a muscular disease in muscles or their appendages: tendon, ligaments, fascia or perimyium. It may affect one or several muscles. The pain may be dull and aching or sometimes agonizing. The pains may be superficial and deep. It is usually aggravated by contraction and relieved by relaxation of the muscle affected. It may increase during sleep to such a pitch as to awaken the patient. There may occur a diminished or temporary loss of power in special muscles, such as a tendency to drop objects or giving way of the knees. Stiffness is a frequent complaint.

The muscle groups most commonly involved are the trapezius (in the neck); the shoulder muscles; the elbow; dorsal and lumbar myalgias; precordial pain; rectus myalgia; sciatica (hip muscles)—with myalgic spots usually in the quadratus lumborum, glutei or tensor fasciæ latæ; knee; feet.

As a working hypothesis, he advanced a diminished blood supply confined to the myalgic areas, leading to oxygen-want producing pain, paresthesias and loss of power. A rapid, effective and permanent cure follows procaine injection—1 per cent into the myalgic spots.

It is evident that clinically myalgia and fibrositis are merely different names for the same disease.

Prognosis.—This is usually good as regards joint phenomena ("the most tractable of all the rheumatic affections)."[79] Acute cases may respond readily to treatment but recurrences are common. Chronic cases may persist for years; however, there is usually not much incapacity to the patient except in those with fibrositis around the shoulder joint or in the palmar fascia. However, these usually respond well to the judicious use of heat and exercises.

Treatment.—In general, during the *acute stage*, the affected part is put at *rest* by immobilization (as by strapping, sling, etc.) and is protected from chilling. Pain may be relieved by *aspirin* and *phenacetin*, beginning

with 0.3 gm. (5 gr.) of each four times a day; if pain persists, codeine sulfate, 15 to 30 mg. (¼ to ½ gr.) may be added to this mixture. *Heat* is useful in the acute stage and may be employed as medicated plasters, electric pads. infra-red therapy, etc.[78] Stiffness from fibrositis may be relieved by a *hot mustard bath.* To prepare the latter, from ½ to 1 ounce of dry mustard is mixed with cool water to make a paste. This mixture is added to bath water at a temperature of from 100° to 102° F.

Treatment of Fibrositis

In the *acute state, rest* of the affected part, with protection from cold or chilling. Procaine injection into the painful spots may abolish the discomfort.

Following subsidence of the acute stage, *heat and firm massage* are *important factors in therapy.*[9]

Analgesics for relief of pain: aspirin and phenacetin, āā 0.3 gm. (gr. v), every three or four hours p.r.n.; codeine sulfate, 15 mg. (¼ gr.), if necessary.

In acute cases, relief of pain may be obtained by autohemotherapy and fever therapy[40,48](typhoid vaccine),[67] needling or *local injections of procaine,*[73] *counterirritants* (such as dry cupping), and methyl salicylate ointment locally; cupping, however, may interfere with massage which may be required at a later date.

In generalized fibrositis, improvement may occur with *hot cabinet baths,* steam cabinet baths, hot mustard baths, epsom salt tub baths and *generalized massage.*

For localized areas of fibrositis, following subsidence of the acute stage, diathermy or short wave diathermy is useful; during the acute stage, histamine by injection or iontophoresis has been found to be of value.[26,65]

Other forms of physical therapy which have been used following the subsidence of the acute stage include infra-red radiation, hot baths, whirlpool baths, contrast baths, and, in the case of the extremities, paraffin baths.

Postural defects must be corrected (including weak, painful feet, chronic low back strain, etc.).

Fatigue and exhaustion must be avoided and the patient's general mode of life must be sensibly regulated.[46]

Consider the possibility of psychogenic rheumatism.

Protect the patient against an undue amount of chilling, dampness, sudden changes in temperature and acute respiratory infections.

If these methods fail to produce results, other measures which have been suggested but which have not been of value in our experience include removal of focal infection, large amounts of vitamin B or E, autogenous or stock vaccines,[37] bee venom[12] or acidophilus milk (gold salts proved useless[37]).

Ishmael reported excellent results in acute fibrositis (complete remissions in every patient) following *autohemotherapy and artificial fever.* This consisted of the withdrawal of 10 cc. of the patient's blood from one of the veins in the arm and injecting this deep into the gluteal muscles before clotting occurred. This was followed immediately by an hour of artificially induced fever (by short wave diathermy) at 101° F. The procedure was repeated, if required, two times per week for 8 or 10 treatments.[43]

Hench[39] discussed the ameliorating effect of jaundice on primary fibrositis. Bee venom has not been of value in fibrositis.[72] Manipulation has been recommended in cases of old fibrositis of the back muscles.[5,20]

Lumbago is treated in Great Britain mainly by infra-red rays; in Amsterdam, ultra-violet rays are used; in Leyden, long wave diathermy; in Denmark, cold wet packs; and short wave therapy in other regions.

Stimulating movements (at the correct time) are valuable in rheumatic diseases. In the United States and Great Britain, forced movements are not considered beneficial except in a limited number of cases.

Technique of Dry Cupping

Materials needed: From 3 to 5 small medicine cups (1 or 2 ounce), alcohol sponges, several large pieces of gauze, methyl salicylate ointment (or petroleum jelly), matches.

Cover the painful area with a thin layer of ointment.

Wet the inside of a small medicine cup with an alcohol sponge; invert the cup and pour out the excess alcohol. Dry the outside of the cup thoroughly.

Light the alcohol within the cup by a match.

As soon as lighted, place the cup on the affected area so that the open end is in contact with the patient's skin. A vacuum will be created within the cup, pulling the skin and subcutaneous tissues into this; the cup adheres firmly to the involved area.

Repeat this with from 2 to 4 additional cups, as needed, depending upon the size of the involved area.

Remove the cups in from eight to ten minutes, when redness of the skin has occurred; these are removed by pulling up on cup with one hand and pushing down on the skin adjacent to the cup with the other.

Cover the reddened area with a thin layer of ointment and gauze.

Caution: Be sure that the excess of alcohol is poured out of the cup before this is lighted; otherwise, burning alcohol may fall upon the bed or rug and cause fire. The outside of the cup must also be wiped dry or burning alcohol may injure the fingers.

Counterirritation from dry cups or local injection of procaine may be needed to relieve the pain. Moynahan and Nicholson obtained *relief in 74 of 80 patients with fibrositis by infiltration of the painful areas with procaine hydrochloride.*[56] Each tender spot is injected with from 2 to 3 cc. of procaine, giving immediate relief of pain, tenderness and muscle spasm. Every tender area is infiltrated and the pain completely eliminated at the first sitting. *Procaine may be injected* directly into the involved area as a sterile 1 or 2 per cent solution; a total of from 5 to 30 or more cc. may be used as necessary, taking into consideration the anatomy of the affected part.[14,22,80] Steinbrocker has employed local injections and regional analgesia with procaine for intractable pain in chronic arthritis and related conditions. He uses up to 20 cc. of a 2 per cent solution of procaine hydrochloride in physiologic saline, or from ½ to 5 cc. of a solution of procaine base in sweet almond oil, dissolved in the hot oil after it is sterilized. Regurgitation of the solution along the needle tract is avoided by using

a small gage needle (usually 22), by rapid withdrawal of the needle after injection, and by pressure over the site with gentle massage for several minutes following this. Care must be taken (aspiration) to be sure that the needle is not within a blood-vessel. Although the aqueous solutions may be injected subcutaneously, the oily preparations should always be given deeply. Such injections have been used in traumatic, rheumatoid or degenerative processes of joints, bursæ, muscles, nerves and periarticular tissues.

Ray has discussed the use of isotonic glucose in fibrositis.[62]

Unfortunately, the technic of dry cupping has been relegated largely to the wastebasket. This may prove useful in relieving fibrositic pain; in addition, it may have a marked psychic effect upon the individual.

Use of Histamine in Fibrositis.—This may be of value especially in cases of widespread fibrositis, with poor peripheral circulation and with marked stiffness on awakening in the morning.[51,53] This has been used both subcutaneously and by iontophoresis in fibrositis. It has been recommended that this drug be given as follows:[25]

0.05 mg. histamine acid phosphate hypodermically.

Increase the dose daily by 0.05 mg. until improvement is noted; this usually occurs at between 0.1 and 0.5 mg.

This dose should then be given every two or three days; the dose may be increased if response decreases.

The patient must recline for thirty minutes following injections because of subsequent headache and dizziness.

The injected histamine produces a flushing in superficial areas of the body. Following the injection, flushing of the head and neck, thumping in the head, relief of pain and disappearance of stiffness are noted within a few minutes. This effect of the histamine lasts for only several hours, although in some cases benefits have been noted for as long as three days following the injections. It *should not be used in* debilitated patients or *in those with a history of bronchial asthma.*

Heat and Massage.—Following subsidence of the acute stage of the disease, deep heat and massage are said to be beneficial. As soon as tolerated, massage may be rather vigorous. Numerous individuals have expressed grave doubt that deep, painful massage could "rub away" fibrous nodules and tissue; usually trauma increases the amount of fibrous tissue present. If available, heat may be supplied by diathermy, short wave diathermy[7,47] or by infra-red radiation. Following improvement, the individual must avoid mild trauma which might aggravate the lesion. Exercises for the involved part should be begun as soon as possible.

In occipital fibrositic headache, relief may be obtained by holding the neck muscles at the nape of the neck or by throwing the head back to relax muscular spasm; rotation of the head to stretch the muscles of the neck may also give relief.

Vitamin E.—Steinberg has reported on the use of vitamin E in fibrositis. He concluded that it was of value in the treatment of primary fibrositis but that it resulted in very little relief to individuals with secondary

fibrositis.[71] Thirty patients with primary fibrositis were completely relieved of all symptoms by the oral administration of from 2 to 8 cc. per day of wheat germ oil (taken in a tablespoonful of milk in divided doses three times a day). However, the latter is unpalatable, and may cause skin rashes and gastric disturbances.[71]

Mixed tocopherols were found to be equally effective whether given orally or parenterally. Steinberg believes that primary fibrositis is a metabolic disorder associated with deprivation of vitamin E.

In his patients, good results were obtained with a daily dose of 300 mg. of mixed tocopherols daily for a week and 150 mg. orally each day for the next two weeks. Maintenance doses of 100 mg. per day were then continued indefinitely.[70]

Unfortunately, we have not been able to duplicate the good results reported by Steinberg following the use of mixed tocopherols in fibrositis.

Hutchison encountered *78 cases of "non-articular rheumatism" in seven months in a military hospital.* Among these, there were *54 cases of fibrositis* and *6 cases of sciatica.*[42]

Fibrositis usually affected the fibrous tissues of the neck, the trapezius muscles, the lumbar and sacro-iliac areas, the hamstring and gluteus muscles and the intercostal muscles. The main symptoms were pain and stiffness. Active fibrositis usually showed definite tenderness, spasm, and in some instances, the formation of nodules. The onset was gradual or sudden. The average period of hospitalization for the 54 patients with fibrositis was twenty days; all patients were returned to duty. Hutchison stressed that if properly diagnosed and treated, fibrositis should rarely cause prolonged invalidism. He believed that soldiers with non-articular rheumatic manifestations were kept off duty too long because of incomplete diagnosis and treatment.

The precipitating causes in fibrositis were sudden strain, wear and tear, sepsis and infection, chilling and psychologic factors; in 17 cases, no cause could be obtained.

His patients with fibrositis were treated as follows: (1) Rest. (2) Heat —for the relief of pain. (3) Massage, in conjunction with heat. (4) Analgesic drugs to relieve pain—in every acute case. (5) *Local injections of procaine subcutaneously and intramuscularly into the tender spots;* this produced dramatic relief of pain—much more rapidly and completely than could be obtained with any analgesic. When pain was relieved by procaine injection, motion was regained. Pain and stiffness were lessened in chronic cases by procaine injection. Further heat and massage are always given after the procaine injections. The procaine injections also serve as a diagnostic aid inasmuch as *pain of psychogenic origin was not relieved by such injections;* in these individuals, the injections were painful in themselves and often intensified referred pain. In most instances, the patients were kept in bed for an hour after procaine injection, following which time active movements were encouraged.

In sciatica, Hutchison cautioned that massage should be employed cautiously and only in the later stages. Sciatica was much less common

but more serious than fibrositis. True sciatic neuritis was differentiated from *pain of sciatic distribution due to fibrositis* in the gluteal muscles by diminution or absence of the ankle jerk on the affected side, hypotonia and wasting of the muscles of the calf and thigh, and in some cases, analgesia of the upper and outer side of the foot in sciatic neuritis; these findings are not present in sciatica associated with fibrositis in the gluteal muscles. The sciatica was mild in Hutchison's 6 cases; the patients spent an average of thirty-nine days in the hospital.

Sciatica.—Sciatica may occur from spinal cord lesions, protruded disk, pelvic tumor, bony overgrowth in the sacro-iliac or hip joints or more commonly to chronic fibrositis of the muscles and nerves.[75]

In Stockman's experience, *sciatic pain* begins most often because of rheumatic nodules and thickenings, especially in the upper two-thirds of the buttock. The inflamed hypersensitive twigs from the sciatic nerve which traverse the fibrositic areas reflect the pain down the sciatic nerve trunk. In his experience, he can usually feel numerous little swellings in the fibrous sheaths of the gluteus medius and maximus, especially along and just under the crest of the ilium and lower down. If they cannot be felt, they can be localized by their tenderness. Massage of these sensitive fibrositic areas in the buttock forms the basis of treatment as employed by Stockman in most cases of sciatica.[75] Local application of heat for ten minutes prior to massage renders this less painful and more effective. Anodynes may be applied locally and analgesics may be given internally to assist in lessening the discomfort. Stockman feels that other measures are usually useless or unnecessary.

Gentle and firm *effleurage* is used early to get rid of the effusion and congestion in the inflamed areas. The amount of pressure applied varies with the patient's tolerance, as this maneuver is always painful. The bruising or discoloration of the skin which may result from ready bleeding of the congested tissues does not deter Stockman, who continues the *massage daily for twenty to thirty minutes.* After three or four weeks, the massage becomes much less uncomfortable and this can be given more firmly and hard pressure may be made on the individual thickenings by circular movements with the finger tips and knuckles, preceded by a few minutes of effleurage to decrease congestion and remove fluid.

Fibrositis of the nerve trunk is another common cause of sciatica, usually in the buttock but less frequently in the thigh. The nerve sheath is attacked primarily, with a spread of the inflammation into the finer connective tissue bundles and fibers producing severe pain and disability in the limb. This may result in numbness and muscular wasting and altered reflexes. Rest in bed, heat, local anodynes and analgesics internally are indicated if there is marked local reaction in the nerve sheath. Very gentle effleurage may be given at this stage. In some of these cases, Stockman advises careful surgical dissection at a later date to clear the nerve sheath and bundles from their fibrositic tissue. If sciatica occurs following gross injury to the nerve, it is best treated by an operation to remove adhesions or cicatrix along the nerve.

Brachialgia.—In patients with brachialgia, painful fibrositic spots may be found in the fibrous tissues connected with the deltoid, trapezius, supra- and infraspinatus, pectoralis major and other muscles; nerve twigs traversing these spots may refer the pain to the main nerves of the arm. Massage of the painful areas in the muscle or nerve will usually suffice, in Stockman's opinion, to relieve the discomfort.[75]

Similarly, he believes that dull or acute pain in the frontal, occipital or temporal regions is frequently due to perineuritis of nerves supplying these regions. The inflamed, tender area is often confined to the point at which the nerve emerges from deep cover: *i. e.*, the temporal nerve as it crosses the temporal fossa; the great occipital as it runs superficially in the scalp and the supra-orbital at the supra-orbital notch.

In occipital pain and headache, the discomfort often arises from fibrositic areas in the shoulder or neck muscles or in the fibrous tissue around the cervical vertebræ—at some distance from where the pain is felt. The pain may be reflected along larger nerves from small twigs in the inflamed patches. The whole area must be searched for tender spots and these must be massaged; this can be done by the patient or his family five or ten minutes each day.

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CHAPTER XLII

RADIOLOGY IN ARTHRITIS

Roentgen findings may be negative in the early stages of an arthritis. Even though articular pain, tenderness and slight swelling may be present, the roentgenologist may find no osteoporosis, decrease in the articular space, change in bony contour or increased periarticular density; in other words, it is too early to find alterations in the roentgen appearance. Such negative roentgenograms are often seen in early acute gout, serum sickness or acute rheumatic fever (even though the joint is red, tender and presents a slight swelling).[13]

The roentgenologist may later find the following *basic alterations in the roentgenogram:*

1. Periarticular swelling.
2. Osteoporosis.
3. Decrease in the interarticular (interosseous) space.
4. Changes in bony contour (proliferative or destructive).

Periarticular Swelling
1. May be the only roentgen sign in early cases. 2. This is visible as an increased density of the soft tissue shadow around the joint or an increase in size of the periarticular shadow. 3. Increased density of the soft tissue nearest the intra-articular space suggests synovial involvement.

Diminution of the interosseous (intra-articular) space and irregularity of the opposing surfaces is roentgen evidence of cartilaginous invasion—seen in rheumatoid arthritis, or degeneration of cartilage as in degenerative joint disease.

Roentgen Evidence of Bone Involvement
1. Osteoporosis. 2. Lipping of bone. 3. Marginal or subchondral proliferation of bone. 4. Destruction of bone (marginal or subchondral areas of bony erosion).

Various forms of arthritis may manifest the same basic alterations as seen in the x-ray. *The roentgen and pathologic response of joints to various insults is too similar to permit dogmatic interpretation of the roentgenograms.*

There is no dependable roentgen differentiation of the various types of cartilaginous destruction except suggestive data obtained from the speed and extent of the cartilage destruction. Articular cartilage reacts to insults by

disappearance (whether due to degeneration, autolytic dissolution as in some septic joints or inflammatory invasion). *There are no characteristic distinguishing roentgen features of any form of synovitis;* insult to the synovial membrane is followed by inflammatory exudation, villous proliferation or fibrosis.

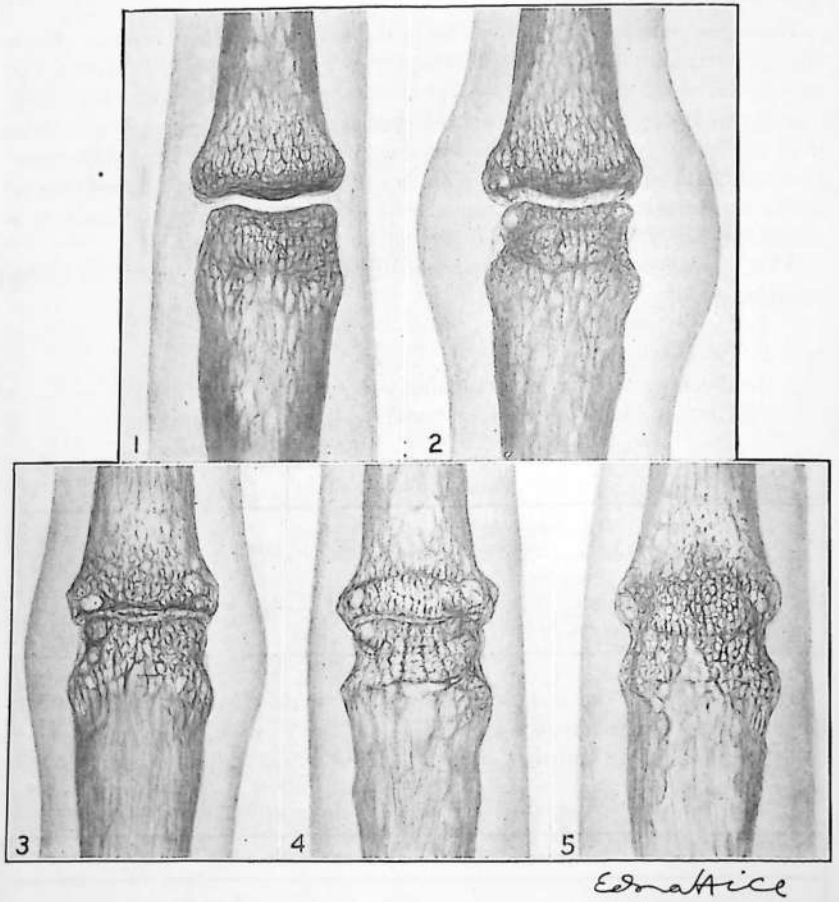

FIG. 138.—Roentgen findings in **progressive rheumatoid arthritis.** Note the *normal proximal phalangeal joint* of finger in *1*. There is no periarticular swelling. The joint space is preserved indicating that the articular cartilage has not been obviously damaged Note normal density of bone and absence of cystlike cavities. *2*. Indicates *early rheumatoid arthritis.* Note the *fusiform soft tissue* (periarticular) *swelling* with some haziness of the joint space. *3*. Further progress of the disease with *narrowing of the joint space* indicating destruction of cartilage, *demineralization* of the bone and beginning formation of *small bony cysts* near the joint margin. *4*. As the disease advances, *further demineralization* occurs, with further narrowing and *almost complete obliteration of the joint space*, with diminution of the soft tissue swelling. *5*. In the *late stage* of rheumatoid arthritis, the joint space may entirely disappear (indicating complete destruction of cartilage); there is *bony ankylosis* with some increase in the mineralization of the bone as compared with *4*. The soft tissue swelling may disappear as in this instance.

It is surprising that the roentgenologist can distinguish some forms of joint disease as often as he can in view of the fundamental limitations in roentgen and pathologic response.

Periarthritis may occur in acute gout, early gonorrheal arthritis, early rheumatoid arthritis, periarticular fibrositis, rheumatic fever, etc. The roentgenologist cannot distinguish these unless additional changes are present, such as atrophy of bone, loss of articular space and abnormality of bony contour.

FIG. 139.—**Rheumatoid arthritis** in a woman of thirty-six. Multiple joints were involved (feet, ankles, wrists, hands, elbows, shoulders and neck). Arrows point to *fusiform periarticular swelling* around proximal phalangeal joint of middle finger. This *joint space* is *narrowed. Bony trabeculæ are hazy.* This represents a fairly early stage of rheumatoid arthritis.

Many difficulties arise which *may confuse the roentgenologist.* The presence of osteoporosis of the epiphyses with slight narrowing of the interosseous space will suggest rheumatoid arthritis; however, if considerable bony lipping is present, he must also think of degenerative changes (osteoarthritis)—a mixed arthritis. The osteoporosis frequently seen in the shoulder joint following long immobilization for traumatic periarthritis

might suggest to the younger roentgenologist an early rheumatoid arthritis. When there is definite destruction of bone and cartilage, it is very difficult for the roentgenologist to distinguish a gonorrheal arthritis from severe rheumatoid arthritis, or even a degenerative hip joint disease (malum coxæ senilis) from the roentgen picture, alone.

FIG. 140.—Rheumatoid arthritis in young male, aged twenty-four years. There are marked changes involving the carpus of the right hand; the *joint spaces* are *almost entirely obliterated*. The radiocarpal joint space is indistinct. The *proximal interphalangeal joints* show evidence of *collapse* and there is marked *spotty atrophy* throughout the hand and wrist.

Hypertrophic changes are usually the dominant ones in degenerative joint disease (osteo-arthritis); however, atrophic changes may be modified by weight in the weight-bearing joints (such as the ankle) so that these may show more hypertrophy than atrophy—even in rheumatoid arthritis. In addition, *hypertrophic changes may be seen in rheumatoid arthritis, gout and in traumatic joint disease.* Hypertrophy of bone may be present in the terminal phalangeal joints in psoriatic arthritis. Hypertrophic changes

are sometimes seen in the spinal column involved by tuberculosis, or in typhoid arthritis of the spine.

Hypertrophic Changes in the Roentgenogram May Occur in:

1. Degenerative joint disease (osteo-arthritis).
2. Rheumatoid arthritis (especially in weight-bearing joints).
3. Gout.
4. Traumatic arthritis (or postural arthritis).
5. Psoriatic arthritis.
6. Typhoid arthritis of the spine.
7. Tuberculosis of the spine—some cases.

Destruction of Bone and Cartilage May Occur in:

1. Advanced rheumatoid arthritis.
2. Septic arthritis, such as gonorrheal arthritis.
3. Tuberculous arthritis.
4. Severe psoriatic arthritis.
5. Severe arthritis accompanying some cases of ulcerative colitis.

What the Clinician Wants to Know From X-rays of Involved Joints

1. He does not want only a differential diagnosis; however, the *x*-ray plays an important rôle in estimating the *severity* of the process or the *stage of the arthritis*. Thus question No. 1 from the roentgenologist is: *what is the stage of the arthritis?*
2. Are the ends of the bones covered with cartilage? The joint space indicates, in general, the thickness of the cartilage. Degenerative changes in a joint are roughly inversely proportional to the thickness of the cartilage. How much narrowing of the joint space has taken place?
3. Are the articular parts in normal alignment? This is important in determining a plan of reconstruction and in preventing further deformity.
4. What is the condition of the bone on the articulating surfaces and adjacent thereto? What can be said of the zone of provisional calcification? Ground glass atrophy and the width of the joint space can be followed, giving an idea as to progression of disease. Also punched out areas should be noted.
5. Are there any changes in contour of the bones—especially in such joints as ball and socket and weight-bearing joints—such as flattening of the head of the femur?
6. What are the conditions within the joint space? villi? joint mice? soft tissue swelling?
7. Do abnormalities exist in the periarticular soft tissues? Calcification in bursæ or in ligaments of the spine? Does tenosynovitis exist? Are the swellings concentric or eccentric? Is there subperiosteal hemorrhage? Are bone tumors or fractures present?
8. The internist would like an estimate, if possible, of the general bony texture and structure—the mineral content of the bone. The structure and mineral content of a bone should be determined at a distance from the involved joints—and preferably where the bone is superficial and not hidden by surrounding fat, etc., such as the skull, mastoid, alveolar process, etc. This might help in estimating the severity of the infection—the general infection in rheumatoid arthritis.

The roentgenologist should avoid the diagnosis of hypertrophic arthritis whenever hypertrophic changes are present, because these occur in a variety of joint abnormalities. He should report his findings, and then attempt to form a diagnosis on the basis of his roentgen findings and the clinical history, if this is obtainable.

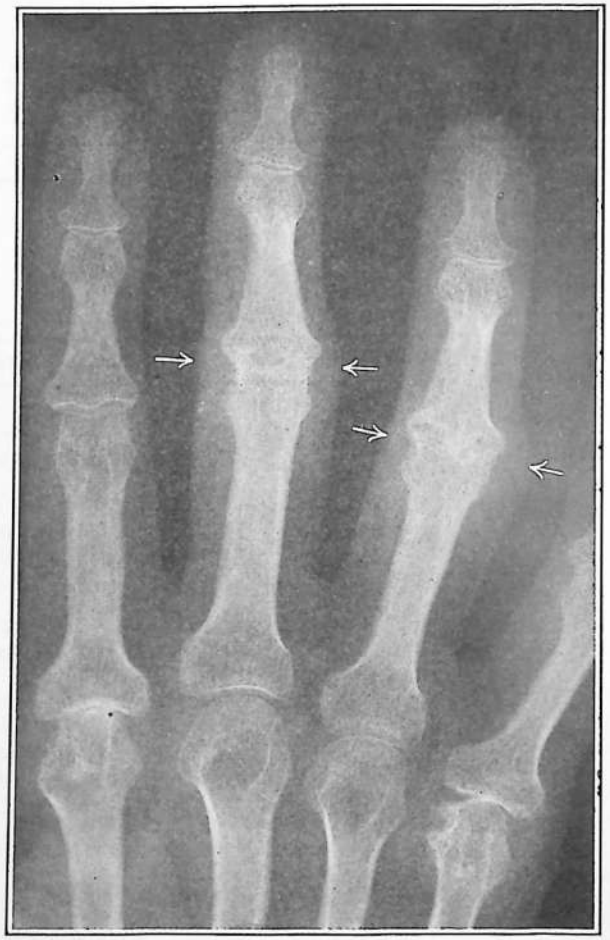

FIG. 141.—Close-up of moderately advanced rheumatoid arthritis of the fingers in a woman aged twenty-five years, whose arthritis began three years after the onset of an amenorrhea. Her sister also has rheumatoid arthritis (with onset following childbirth). The soft tissue swelling is evident at the proximal phalangeal joints, together with the narrowed joint spaces and ground glass appearance of some of the bones.

A frequent mistake seen by x-ray is the clinical diagnosis of lumbago or sciatica when the symptoms are being caused by malignancy, usually secondary carcinoma of the pelvic bones or vertebræ. *Paget's disease* (osteitis deformans) is undiagnosed frequently, or misdiagnosed as arthritis.

Patients diagnosed clinically as arthritis may be found on *x*-ray to have these symptoms resulting from myeloma, carcinoma, Charcot joint, tuberculosis of a kidney, calculi in the urinary tract, etc.[103]

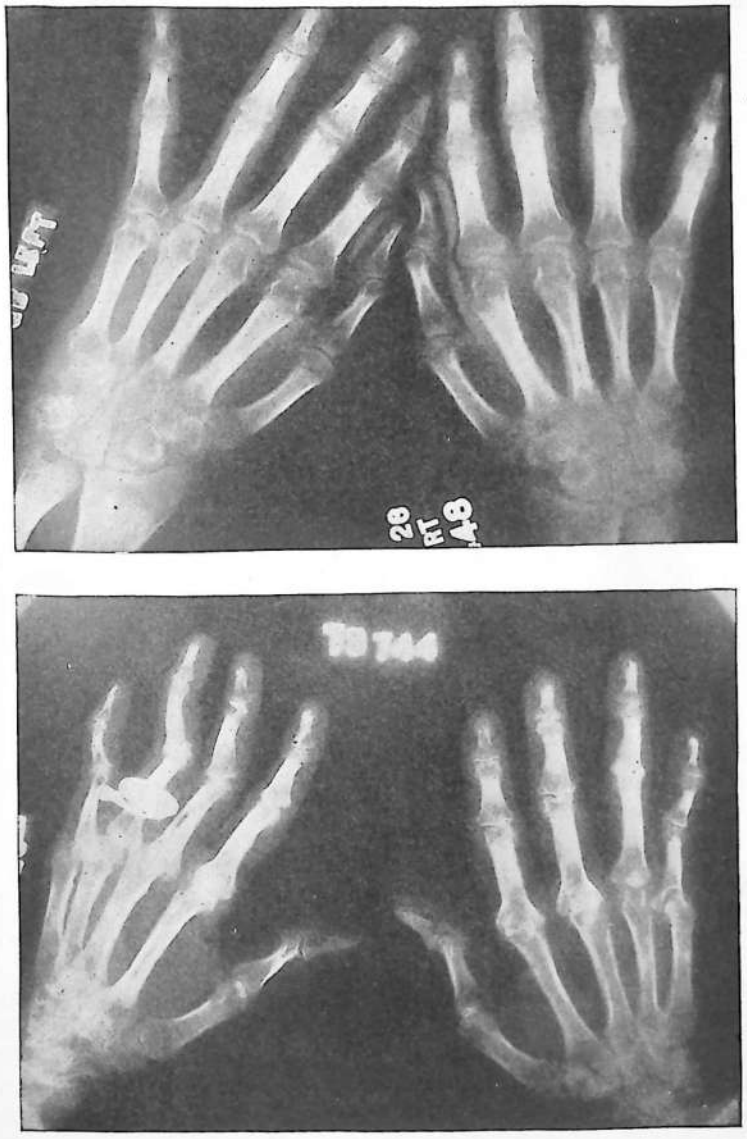

FIG. 142.—Above: Early stages of rheumatoid arthritis: osteoporosis with soft tissue swelling; very little destruction of cartilage and no destruction of bone. Below: More advanced stage of rheumatoid arthritis with destruction of cartilage and bone and beginning luxation of fingers. (Doub, courtesy of Radiology.)

The roentgenologic features of arthritis have been made the subject of separate volumes.[10,62,73] Roentgen-rays are useful in the diagnosis and in the treatment of some arthritic conditions. The roentgen-ray will show only those changes which are sufficiently well advanced to produce a recognizable change in the density and contrast of tissues.[114]

FIG. 143.—*Rheumatoid arthritis* in a woman aged thirty-three years. Note narrowing of joint spaces, especially in proximal phalangeal joints of third and fourth fingers. There is subluxation at the metacarpophalangeal joint of the thumb.

If the roentgenologist uses the terms atrophic or hypertrophic to describe changes which he sees in the roentgenograms, he must be sure that the clinician does not wrongly interpret his findings and think that this denotes either a "hypertrophic" or an "atrophic" arthritis. This is one reason for discarding these former diagnostic terms in favor of degenerative joint disease and rheumatoid arthritis, respectively. *If the roentgen diagnosis does not agree with the clinical impression, the clinician must not feel that the roentgen diagnosis is final by any means* and should go over the films with the roentgenologist and correlate these with the clinical data.[12]

Degenerative joint disease may closely simulate the picture of the reparative process of various forms of arthritis (tuberculous, gonorrheal, pneumococcal and syphilitic[31]). In one series of roentgenograms of more than 500 joints in 60 patients with chronic polyarthritis, 25 per cent showed no roentgen findings although pain or deformity was or had been present.[98]

FIG. 144.—Involvement of the hands, wrists, feet and knees; numerous subcutaneous nodules over forearms, knees and buttocks in a patient aged fifty-nine years. There are marked atrophic and hypertrophic changes and demineralization of all of the bones of the hand and wrist. The picture is predominantly that of rheumatoid arthritis.

It is the duty of the clinician to furnish his roentgen colleague with a brief history of the case, noting the age of the patient, the sex, the joints affected, the appearance of the joints clinically, the course of the disease and other salient features. It is the duty of the roentgenologist to report his findings in detail and in addition to give his diagnostic impression as determined from the roentgen findings and the history of the case. When the internist and roentgenologist disagree, a consultation should be held.

Roentgen Examination of a Joint.[120]—This will show: A positive picture of the bones.

Some indication of the soft parts (by proper technic).

No visualization of cartilage except by special methods;[79,100] the width of the joint space is a guide of its pathology. The true articular space has been demonstrated roentgenographically on several occasions.[30,126]

No visualization of the capsule or synovial membrane except by the use of very soft rays in the larger joints.

FIG. 145.—**Rheumatoid arthritis** of twelve years duration. The changes present are indicative of an *advanced degree* of rheumatoid arthritis in which areas of *bony ankylosis* are already present. There is *subluxation* of the distal interphalangeal joints of both thumbs and *partial subluxation* of the proximal interphalangeal joint of the left little finger. The most marked *loss of joint cartilage* has occurred in the carpus, where there is evidence of bony ankylosis. There are numerous *scattered juxta-articular cystic areas* and *spotty areas of demineralization.*

In normal bone, the cortex or outer layer, is the most dense; this is well defined along the shaft and fades away toward the cancellous extremities. The medullary canal is less dense and also fades into the cancellous ends of the bones. The least dense portions of the bone are the cancellous ends.[103]

The joint spaces represent the thickness of the cartilages between the articular surfaces. Thinning or destruction of the cartilages is seen on the *x*-ray by a diminution of the joint space.

Roentgen Findings in Rheumatoid Arthritis

1. No changes may be apparent very early in the disease.
2. The *first roentgen sign* is usually *periarticular soft tissue swelling*—a fusiform swelling of the soft tissues about the joint, indicated by increased density of the soft tissues (due to proliferation of synovial membrane, joint effusion and thickened joint capsule).[113,114,127]
3. *Generalized skeletal decalcification* is an early sign.[18] The decalcification is usually more marked in the articular ends of the bones. This demineralization cannot be distinguished roentgenologically from that of hyperparathyroidism, hyperthyroidism, rickets, osteomalacia or senile osteoporosis. There is *rarefaction of the trabeculated ends of the bones,* followed by atrophy of the ends of the bones, thinning of the articular cortices and a *ground-glass appearance* and atrophy of disuse in the shaft. Generalized decalcification of the bony skeleton is one of the most characteristic roentgen findings.
4. Definite *effusion within the joint* presenting a dense, sharply outlined and fusiform appearance.
5. Progressive *narrowing of the joint space* (indicating thinning of the articular cartilage).
6. Destruction of the cartilage and the presence of cortical erosions with *punched out areas of bone* along the articular surfaces; these are usually smaller than those of gout (and in gout, there is no alteration in the bone density no matter how advanced the stage).[24]
7. The *articular surfaces become irregular and may be destroyed;* this may be followed in some cases by subluxations and dislocations.
8. Marked *transparency of bones.*
9. Hypertrophy of bone may appear in the late stage; this may be seen relatively early in weight-bearing joints.
10. In the *very late stages* of severe rheumatoid arthritis, very little if any periarticular swelling remains, but there is marked *atrophy of bone,* destruction of cartilage, moderate *bony lipping* and *osteophytes* (in some cases), marked destruction of bone, *disarrangement of the joint and deformity.*[12] *Fibrous or bony ankylosis* may occur, resulting in continuity of the trabeculated ends of adjacent bones. Bony ankylosis is most common in joints with a narrow range of motion, such as the sacro-iliac joint or carpus. Surrounding muscles become atrophied and occasionally small calcified deposits may be seen about the joint margins. One may see deviation, luxation or subluxation, thickening of the bones and deformity of the bony ends at the joints, due to mechanical forces. If the disease becomes quiescent, there may be an increase in bone density. Occasionally, telescoping of the ends of the bones occurs with the development of the opera glass hand (*la main en lorgnette*).[17]

Lupo's report of the radiology of chronic joint diseases was based on a study of several thousand patients.[38] He noted the following radiologic changes in rheumatoid arthritis:

1. Conspicuous and early increased bone transparency, differing from the atrophy of disuse, because the former is more homogeneous, is confined to spongy tissue and stands out in contrast to the relatively increased density of the cortex.
2. Progressive narrowing of the joint spaces, extending uniformly along the whole line of the joint.

3. Plastic, waxlike deformation of the epiphyses, due to shrinking of the joint capsule.
4. Lastly, destruction of the articular extremity and disappearance of the joint line and bony ankylosis.

FIG. 146.—Advanced rheumatoid arthritis, aged seventy years. Pain and stiffness of multiple joints, especially in the hands and feet since menopause thirty years ago. Hand and wrist show a moderate amount of *demineralization* of the bones. There is *narrowing of all* of the *joint spaces* of the metacarpals and phalanges. Similar changes are present in the carpal bones to a more marked extent. Here all the normal architecture of the wrist bones is lost and there seems to be *ankylosis* present between these bones and the radius and ulna.

Taylor and his co-workers noted *systemic decalcification, atrophic bone destruction, joint effusion, narrowing and obliteration of the joint space and evidence of bone production* (lipping and osteophytes) *in more than 75 per cent of all cases of rheumatoid arthritis.* Systemic decalcification was most marked in the bones of the hands and feet, although it was noted throughout the skeleton. One should always obtain lateral views of the small joints of the hands or one may miss large areas of destruction.[117]

Decalcification of bone will aid greatly in identifying the rheumatoid arthritic. As the disease becomes more quiescent, there is increased density of the shafts of the bones. These workers pointed out that *punched-out areas of bone are a prominent feature of rheumatoid arthritis and occur as often in this disease as in gout* (although such areas are usually somewhat larger in the latter disease). In rheumatoid arthritis, these appear as though bone were removed with a punch; no reaction is noted in adjacent bone. Kuhns found the *shoulder* to be *involved in only 9 per cent* of 1765 patients with

FIG. 147.—**Rheumatoid arthritis** of the hands and feet since fourteen years of age with marked residual deformity. At present this fifty-one year old woman has leukopenia and splenomegaly. The small joints of the hands and feet show advanced destructive changes. The *cartilage* has been completely *destroyed* and the articular ends of the bone are motheaten as well. Dislocations occurred between many of the metatarsophalangeal joints.

rheumatoid arthritis. Roentgen changes were rarely marked in these individuals and symptoms referable to the shoulder were frequently marked. Involvement of the shoulders was usually bilateral, progressed slowly, usually without the exacerbations which affect the weight-bearing joints and in some cases, almost painlessly. Unless early vigorous treatment was instituted, marked limitation of motion occurred.

Oppenheimer has shown that the only true joints between spinal units are the intervertebral joints formed by the articulating facets.[82–86] The *intervertebral space* (between the vertebral bodies) is not a joint space inas-

much as it is filled with the disk and its membranes and has no synovial membranes or articulating surfaces.[75] The roentgen technic of visualizing the apophyseal intervertebral articulation and the intervertebral foramina has been described.[46]

FIG. 148.—Marked **rheumatoid arthritis** with involvement of multiple joints, particularly the proximal phalangeal joints (where flexion contractures have occurred) and in the wrist.

Scott[106] has repeatedly stressed the *importance of early recognition of* **rheumatoid arthritis of the spine.** He states that the so-called *adolescent kyphosis* is really an excessive kyphosis associated with the final stages of rheumatoid spondylitis. Roentgenologists must familiarize themselves with the early roentgen findings in this disorder. Clinically, the only symptoms for months or years may be ill-defined pains in the extremities, with a gradual loss of weight and rapid sedimentation rate. In any such symptoms occurring in a young adult over a period of years, one should always make a roentgen study of the sacro-iliac joints as changes occur in this area early in the disease. **The sacro-iliac joint space is narrowed or obliterated even in the early cases.**[118]

OSTEOPOROSIS

Formation and resorption of bone are going on continuously in adult bone. In *hyperparathyroidism*, the mass of bone is deficient because resorption is too great; in *osteoporosis* or in *osteomalacia*, there is too little formation of bone. In osteoporosis, the osteoblasts do not lay down sufficient osseous matrix; in osteomalacia or in rickets, the osseous matrix is not calcified.

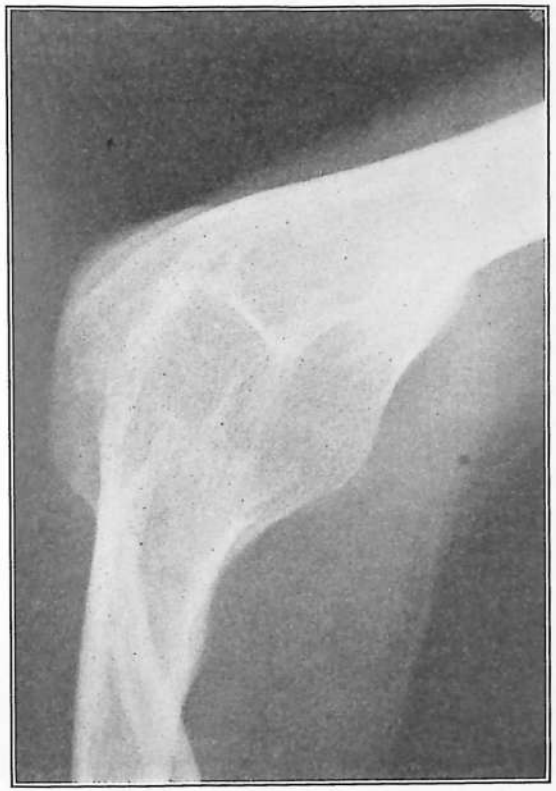

FIG. 149.—**Advanced rheumatoid arthritis** in a woman of thirty-nine whose rheumatoid arthritis began at the age of eleven. Joint involvement progressed so that now many joints show *bony ankylosis*, as is the case in this elbow. *Marked generalized demineralization* was present. *Joint spaces have been obliterated* and considerable *deformity* was present.

Stresses and strains are thought to be an important stimulus to osteoblastic activity; the atrophy of disuse may be due partly to a decrease of this stimulus.[1] Considerable evidence has accumulated to show that osteoblasts manufacture *phosphatase. In the absence of hepatic disease,* the *serum phosphatase level is an index of osteoblastic activity.* If the skeleton is rarefied, the serum phosphatase should be increased unless the rarefaction is due to primary hypoplasia of the osteoblasts (osteoporosis). Albright

has stressed that an important diagnostic feature of osteoporosis is the absence of an increased phosphatase level.

Serum calcium and *phosphorus* levels are normal in osteoporosis as this is not a disease of calcium metabolism. If, however, young individuals have a large proportion of their body skeleton suddenly immobilized by a cast or by infantile paralysis, one may then encounter a hypercalcemia.

In osteoporosis, there may occur an increase in the urinary excretion of calcium and phosphorus until the skeleton becomes demineralized, at which

FIG. 150.—**Mixed arthritis** of the knee in a woman of fifty-eight; marked destruction of articular cartilage on the mesial side. *Rheumatoid arthritis and degenerative joint disease.* Sedimentation rate was moderately rapid.

time this hypercalcinuria and hyperphosphaturia disappear; in hyperparathyroidism, these latter persist regardless of the amount of decalcification.

Albright and his associates have reviewed the literature on the **connection between ovarian function and calcium metabolism.** Various workers have shown that: (1) administration of estrogens to pigeons results in hypercalcification of the long bones in males and females, and raises the serum calcium level; (2) following the giving of estrogenic material to mice, the marrow cavities were almost completely replaced by compact bone of

coarse bony spicules. Other workers noted that the long bones of male pigeons are filled with marrow while those of females were solid.

The most common etiologic factor in generalized osteoporosis in women is the *postmenopausal state.* Other factors concerned in the causation of osteoporosis include senescence and disuse; it has not been definitely established that faulty diet is a factor.

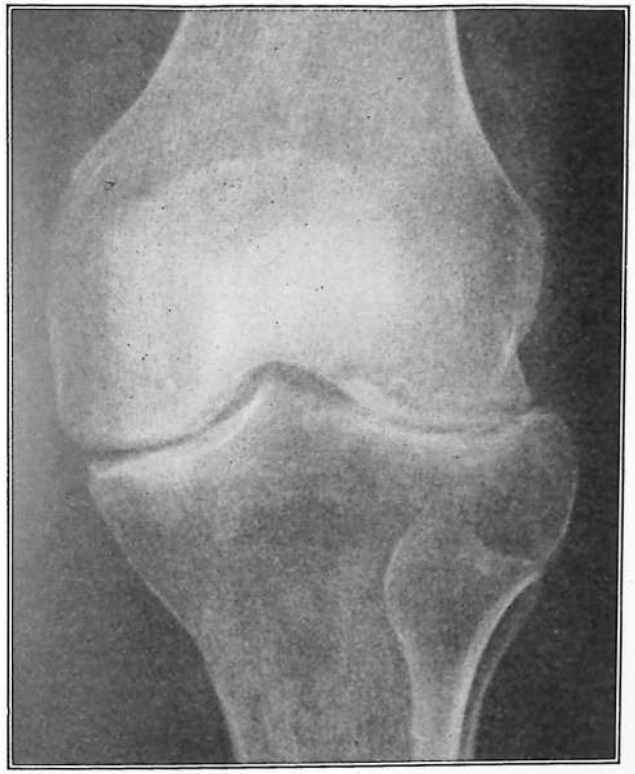

FIG. 151.—**Advanced rheumatoid arthritis** of the knee. Note *narrowing of the joint space* indicating destruction of cartilage. Also note homogenous (hazy) appearance of ends of bones.

Post-menopausal osteoporosis involves particularly the *spine* and the *pelvis*, with less involvement of the long bones. *The skull is almost never attacked* (in contrast to its frequent involvement in hyperparathyroidism). Patients with postmenopausal osteoporosis tend to develop atrophy of other tissues, particularly the skin.

The *atrophy of disuse* is well recognized clinically. It is caused most frequently by flaccid paralysis in a limb or by immobilization in a plaster cast. As individuals age, their sedentary life is undoubtedly a factor in leading to generalized osteoporosis.

A *deficient diet* is the main factor in the causation of *osteomalacia; repeated pregnancies* probably further drain the body's supply of calcium

and are another factor in the etiology of osteomalacia. Albright does not believe, however, that the diet or pregnancy play an important rôle, if any, in the etiology of osteoporosis.

Thyrotoxicosis may be associated with an increased secretion of calcium in the feces and urine. In patients with long-standing thyrotoxicosis, there is an *increased radiability of the skeleton.*

FIG. 152.—Long history of **rheumatoid arthritis.** There are extensive changes involving the articular surfaces and adjacent portions of the bones of the knee joint. There are also changes in the soft tissues. On the mesial side of the knee joint, there are some hypertrophic changes. Patient is a woman aged sixty years.

The *osteoporosis* occurring in *Cushing's disease* has many of the clinical features of postmenopausal osteoporosis. In Cushing's syndrome, the increased conversion of protein into sugars leads to a negative nitrogen balance; this is probably the most important factor in the causation of osteoporosis in this syndrome.

In Cushing's disease, estrogen formation is decreased or absent and the urinary androgens are increased. The presence of an excessive amount of androgens may be responsible for the severity of the osteoporosis in this syndrome as testosterone inhibits the effect of estrogen on the formation of bone (in doves). This increased androgen content may also be the cause for the ineffectiveness of estrogens in producing a positive calcium balance in these patients.

In *old age,* bones atrophy in both men and women. Fracture of the hips in the aged is a common manifestation of this. This has been known as *senile osteoporosis.*

The main arguments in favor of an entity such as **post-menopausal osteoporosis** are (1) the frequent occurrence of osteoporosis in women after the menopause and (2) the beneficial effects of estrogen on the calcium metabolism in these patients.

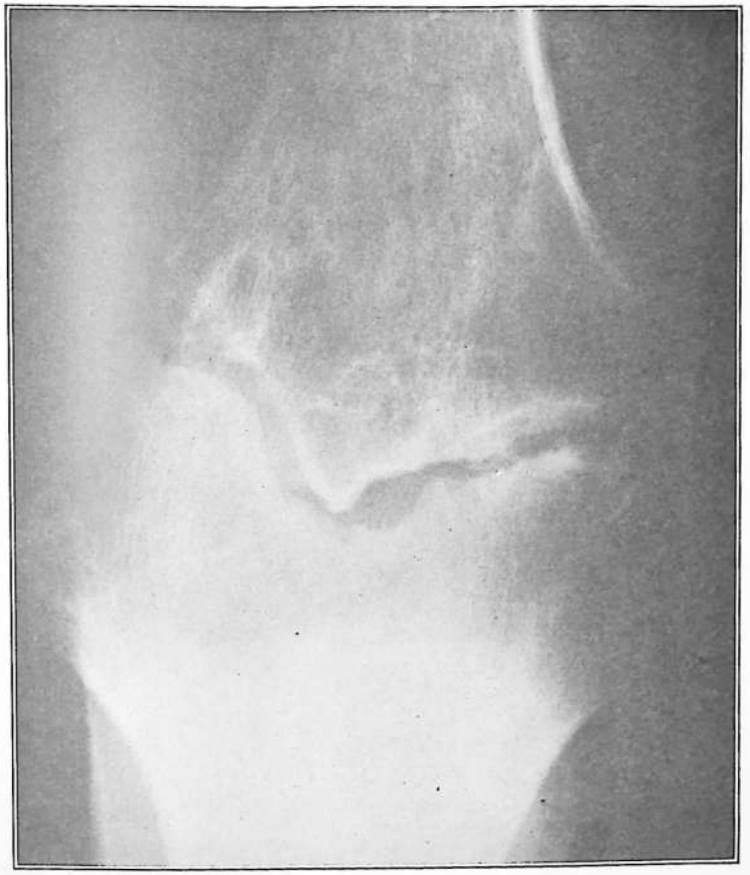

FIG. 153.—*Extensive, advanced* **rheumatoid arthritis** *in a diabetic* aged sixty-one years. Arthritis is of thirteen years' duration. There is marked *erosion of the articular surfaces* of the femur and tibia with considerable *destruction of bone.* (Multiple joints were involved in an extensive rheumatoid arthritis: fingers, wrists, etc.) Sedimentation rate was very rapid.

Albright and his associates reviewed 42 patients that were admitted to the hospital over a ten-year period with a diagnosis of generalized osteoporosis without an obvious cause; they excluded *osteoporosis due to infantile*

paralysis and all patients over sixty-five because of the possibility of senescence being a factor. Of these 42 patients, it is remarkable that 40 were women past the menopause. Two were men. The condition was not found in any women before the menopause.

The *average time of onset* of symptoms was nine and a half years after the menopause in 27 patients with physiologic menopause and 13.4 years in 10 cases of artificial menopause.

FIG. 154.—Same patient as preceding figure. Lateral view of this knee shows marked patellar changes also.

The *spine and pelvic bones are involved predominantly* in the process; in cases with more severe involvement, long bones may be involved. Lesions in the spine consisted of crushed or fractured vertebræ and herniation of the nucleus pulposus.

In the average case, a woman about ten years after the menopause receives a minor trauma and then experiences back pain; a roentgen examination then reveals the condition.

ADDITIONAL ROENTGENOLOGIC AIDS

The roentgenologist can be of further assistance to the physician in his study of the arthritic patient by finding bony anomalies, foci of infection (teeth, paranasal sinuses, gall bladder, gastrointestinal tract, pulmonary pathology, renal infection, old osteomyelitis), or other pathologic bony

lesions.[25] These may include evidence of *subdeltoid bursitis; hyperparathyroidism; neoplasm* in various viscera or *metastases to bone; psoas abscess; Paget's disease;* pituitary tumors; *spinal anomalies; scoliosis; multiple myeloma;* scurvy; rickets, etc.

FIG. 155.—**Psoriatic arthritis.** The *involvement of the terminal phalangeal joints* in the fingers or toes, *with destructive changes in the terminal phalanges,* is not uncommon in rheumatoid arthritis associated with psoriasis. This woman of thirty-eight has had a long-standing psoriasis, with joint changes (in multiple joints) for the past five years. Rheumatoid arthritis, itself, usually exempts the terminal phalangeal joints, concentrating more on the proximal phalangeal joints; however, the terminal phalangeal joints may be involved in some cases of rheumatoid arthritis.

PNEUMOROENTGENOGRAPHY[77,108]

Various investigators[65,68,69,107] have injected air (or oxygen, or carbon dioxide) into joints, to aid in the differential diagnosis of injuries and diseases of the soft tissue structure of the knee.[5–8,14,26,43,51,56,63–65,67,80,92–94,96,108,119,125] Others have injected various gases into joints therapeutically.[11,41,91,95,96,97,99]

A simple method of injecting air into the knee is as follows:[94]

With the knee relaxed in slight flexion on a roentgen-ray table, aspirate any fluid from the joint under local procaine anesthesia.

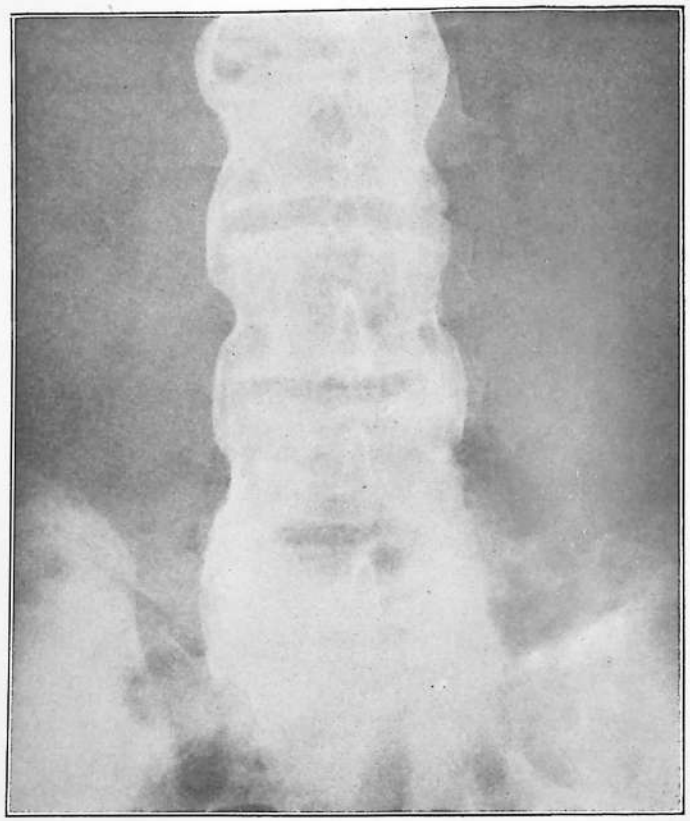

FIG. 156.—**Ankylosing spondylitis** involving the entire thoracic and lumbar region and the sacro-iliac joints. The anterior and lateral ligaments were ossified in the lumbar region and there was ossification of the posterior longitudinal and lateral ligaments in the thoracic region. This is an advanced rheumatoid arthritis of the spine in a man aged thirty-nine years.

Insufflate the joint with 100 to 150 cc. of air from a Luer syringe with needle and stop-cock.

Make stereoscopic AP and plain lateral roentgen-rays, using soft tissue detail technic.

Release the air, withdraw the needle, and apply a sterile dressing and elastic bandage.

This procedure is not to be undertaken without considerable experience upon cadavers and should always be performed with extreme caution.

The *technic of injection of gas into a joint* employed by Savage and Taylor

was as follows: The knee joint was flexed to 45° and the site of entry of the needle (usually in the recess between the lower, outer edge of the patella and the lateral condyle of the tibia) was infiltrated down to the capsule with 0.5 per cent procaine.[101] The gas was injected through a

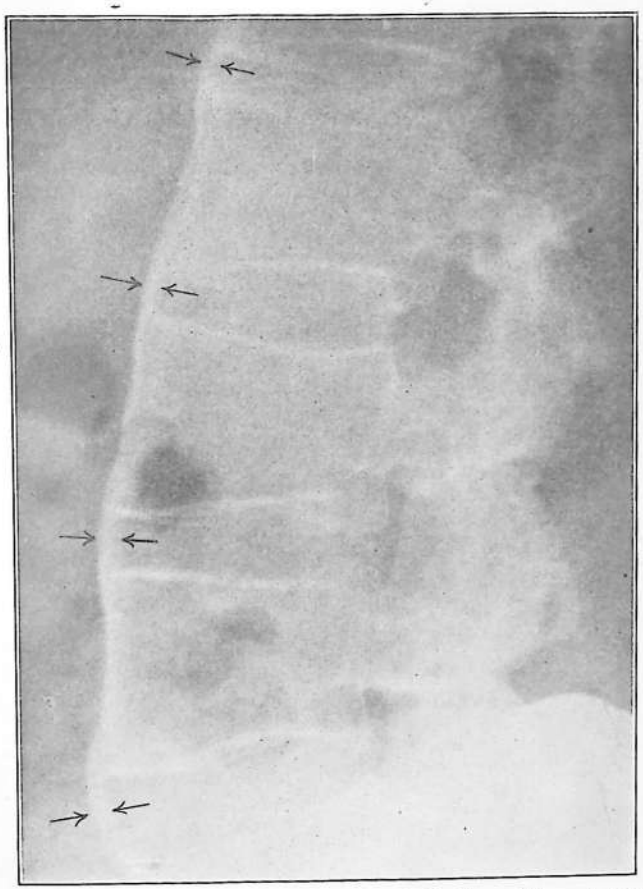

FIG. 157.—**Rheumatoid spondylitis.** Note the *calcification of the anterior ligaments.* This male of twenty-eight years has had low back pain for eight years, with known ankylosis of the spine for two years. The sacro-iliac joints were obliterated and there was marked involvement of the small true posterior intervertebral joints.

needle attached to a pneumothorax apparatus. The joint pressure as recorded on a manometer before injection was not greater than 1 cm. of water. It was usually necessary to raise the pressure to more than 25 cm. of water before any appreciable amount of gas could be forced into the joint. The injection may be carried to the point at which discomfort begins. About 300 cc. could be injected without causing undue pain.

The gas could usually be felt within the joint for at least four days. The patients did not complain of discomfort during or after the injection.

Savage and Taylor studied the oxygen and carbon dioxide gas tensions in normal and abnormal knee joints and noted in a few cases that the oxygen tension was higher and the carbon dioxide tension lower than normal in acute rheumatoid arthritis.

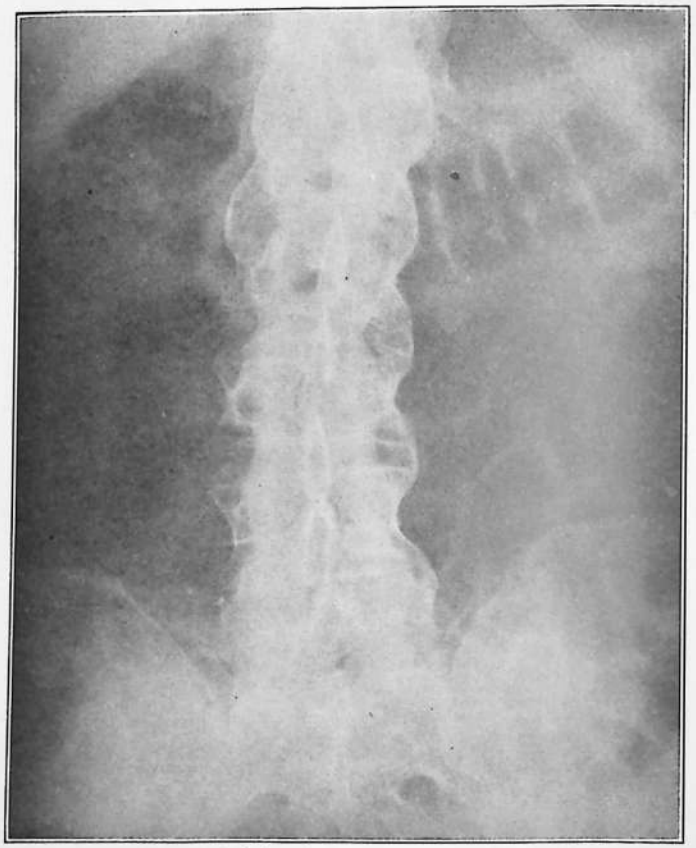

FIG. 158.—Rheumatic spondylitis. Anterior-posterior view of same patient as in preceding figure. *Sacro-iliac joints* are *obliterated* and the ligamentous calcification is evident.

VISUALIZATION OF KNEE JOINTS

Boyd observed no serious reaction in a roentgen-ray study of the knee joint visualized with 30 per cent iopax, dissolved in 0.5 per cent procaine.[9] The iopax was quickly absorbed. Keller produced satisfactory opacity in joints with four dyes (hippuran, arthropsin, neoiopax and skiodan).[58] Such dyes are contraindicated in patients with liver or kidney damage. Keller has visualized adhesions in joints following the intra-articular injection of "arthropsin," a 10 per cent solution of the disodium salt of tetra-iodo-ortho-sulfo-benzoic acid.[57]

Dunham and Kautz have reviewed the literature on rheumatoid spondylitis.[23]

Bone Density.—It is possible to evaluate bone density in the roentgenogram; Stein has used ivory wedges for this purpose.[115] These are especially valuable in comparing serial films taken at intervals of several months or years.

FIG. 159.—Late stage of rheumatoid arthritis with marked deformity and ankylosis. (Doub, courtesy of Radiology.)

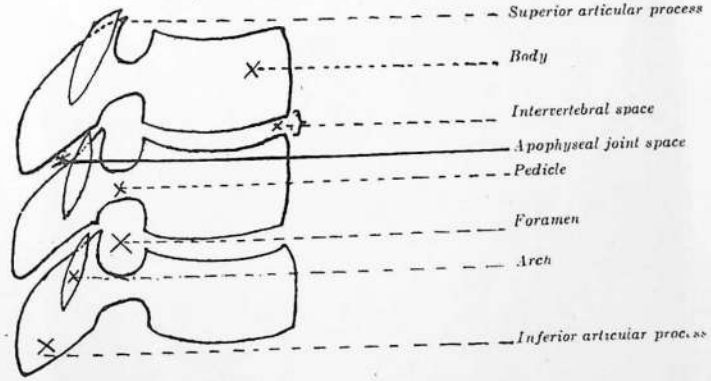

FIG. 160.—Diagram showing true intervertebral joints and foramina. (Oppenheimer courtesy of Radiology.)

Progression of Roentgen Findings.—Morrison and Kuhns *rarely observed improvement* in the roentgenologic picture in serial roentgenograms in cases of rheumatoid arthritis or degenerative joint disease followed from four to ten years.[78] They noted that roentgenologic changes usually progressed for long periods following clinical quiescence.

Roentgen Findings in Rheumatoid Spondylitis

1. Changes in the sacro-iliac joints (narrowing or obliteration of the joint even in early cases). The anterior and posterior fissures of the sacro-iliac joints lose definition and later become obliterated; sclerosis of bone may appear around the obliterated sacro-iliac joints, followed by bony ankylosis of these joints.
2. *Narrowing of the apophyseal joint spaces.*
3. Irregularity of the outlines of the facets.
4. Bony ankylosis of the articulations.[33,104]
5. Marked increase of bone transparency, with accentuation of the borders of the vertebræ, which appear like reels, with lipping at their edges.
6. *Demineralization* of the vertebral bodies in the early stages; normal density may be regained after bony ankylosis has occurred.
7. No involvement of the intervertebral disks; the intervertebral spaces are of normal width.
8. The ligamenta flava and *longitudinal ligaments* may become *calcified or ossified.*[6]

FIG. 161.—Mixed form of arthritis in a woman aged seventy-four years. *X*-ray of the knees shows considerable hypertrophic and some atrophic changes. The patient also had involvement of the fingers and spine; this began shortly after the menopause (at the age of fifty).

Degenerative Joint Disease (Osteo- or Hypertrophic Arthritis).—In degenerative hip joint disease (osteo-arthritis of the hip), there is a loss of cartilage at the maximum pressure point with formation of osteophytes on the edge of the acetabulum and around the head of the femur. Cysts

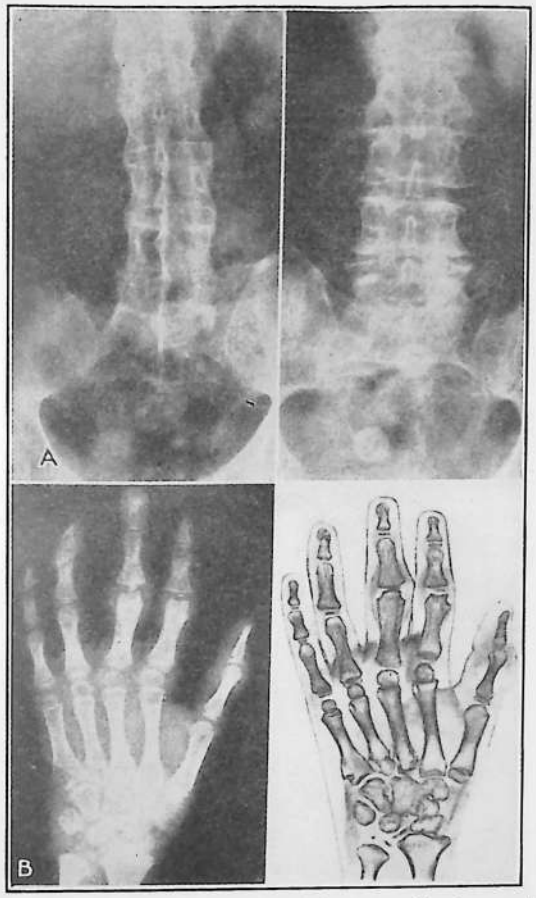

FIG. 162.—*A* (left): Rheumatoid arthritis of the spine showing systemic decalcification, narrowing and obliteration of the spaces between the facets of the articular processes, calcification of the longitudinal ligaments and obliteration of the sacro-iliac joints. *A* (right): Degenerative joint disease of the spine, showing absence of decalcification, lipping and osteophytes and relatively good preservation of the sacro-iliac joints. *B:* Rheumatoid arthritis (Still's disease) in a child showing systemic decalcification, atrophic destruction of bone, narrowing of the joint spaces, swelling of the soft tissues, and effusion, symmetrical and especially well-marked at the proximal interphalangeal joints. (Taylor, Ferguson, Kasabach and Dawson, courtesy of Arch. Int. Med.)

may occur in the femur and acetabulum. Sclerosis of the bone surrounding the joint is noted. In the late stage, one may find an upward and outward displacement of the head of the femur.

The conventional antero-posterior roentgen films do not give a full clue

to all of the gross and microscopic pathologic alterations in degenerative hip joint disease. *On roentgen examination, the hip with degenerative joint disease* (osteo-arthritis) *shows a decrease and finally a loss of joint space* (due to erosion of the cartilage) particularly in the weight-bearing portions of the head and acetabulum, *osseous sclerosis* and *marginal exostoses.* Roentgenograms never permit one to fully appreciate the abundance and

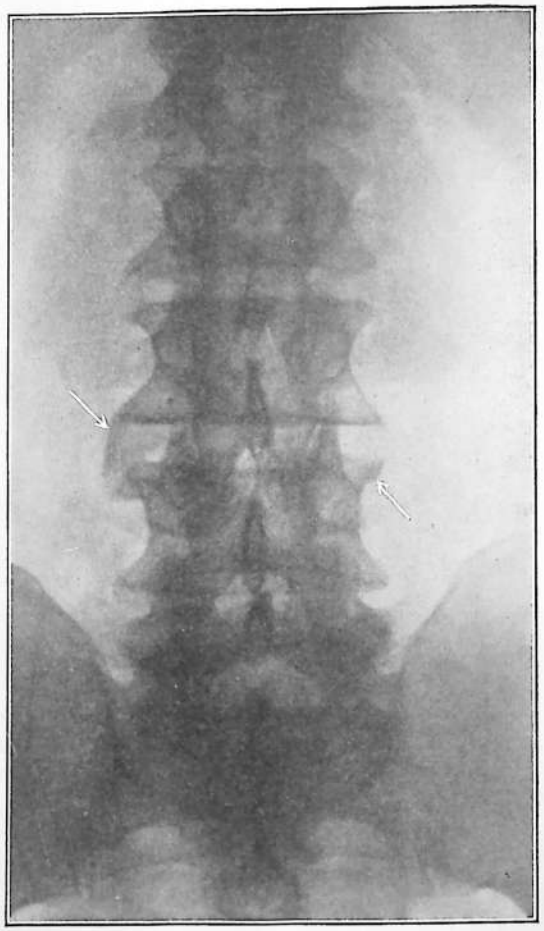

FIG. 163.—Hypertrophic changes in the lumbar vertebræ. (Holmes and Ruggles.)

complexity of the exostoses and their effect on motion as one visualizes only those at the superior and inferior portions of the joint. The remaining half or more of the femoral head and acetabular margins which are in the anterior and posterior portions of these structures are covered by other shadows.[36] This is discussed more fully in the chapter dealing with the symptoms of degenerative joint disease. In degenerative knee joint disease (osteo-arthritis of the knee) osteophytes at the upper and lower

margins of the patella may be the earliest manifestation. Osteophytes may then be noted on the head of the tibia and the condyles of the femur. The joint cartilage is usually well preserved until late in the disease.

Roentgen Findings in Degenerative Joint Disease (Osteo- or Hypertrophic Arthritis)[13,29,114]

Early stage:
- *Narrowing of the joint space* (due to thinning of the joint cartilage).
- Sharpening of the articular margins.
- Formation of bony spicules and *osteophytes* at the margins of the joints.
- Slight tilting of one bone on another (changes in the alignment of bones) because of the irregularities along the bony joint surfaces (one fitting into the other).
- Atrophy of disuse at the ends of the bones in some cases (usually with preservation of the bony trabecular pattern—as differentiated from the ground glass atrophy usually seen in rheumatoid arthritis).

Later stage:
- Increase in the size of the osteophytes.
- Further narrowing of the joint space which may appear jagged or saw-toothed or almost obliterated.
- Subluxations of joints may occur.
- Formation of *cysts at the ends of the bones* and punched-out areas in the borders of the bones.
- Increase in circumference of the joint due to new bone formation.
- *Condensation of subchondral bone.*

Advanced stage:
- Osteophytes may appear quite large and irregular.
- Deformities and deviation of the bones from pressure and traction.
- *Eburnated* (very dense) *ends of the bones* following disappearance of the cartilage (demonstrated as obliteration of the joint space).
- Atrophy of disuse extending throughout much of the shaft of the bone.
- Marked irregularities of the ends of the bones, but ankylosis only very rarely (except as mechanical interlocking of exostoses as may occur between adjacent vertebræ).
- Joint mice in some cases.
- *Joints most affected:* distal interphalangeal joints of fingers, lumbar and cervical spine, knees; shoulder joint rarely involved.[10]

In the hand, the earliest roentgen evidence of degenerative joint disease is a narrowing of the joint space (loss of cartilage) between the terminal phalanges, and lateral displacement of the terminal phalanges, with the formation of Heberden's nodes. *In the foot,* one may occasionally note similar loss of cartilage and the presence of osteophytes.

Jostes[50] stressed the value of *body section roentgenography* as an aid in diagnosis of *occipitocervical lesions.* These are especially useful in patients with pain in the neck or occipital region radiating to the shoulder in whom the ordinary roentgenograms of the cervical and shoulder regions were repeatedly pronounced negative. The advent of the *laminograph* opened up a new field of pathologic possibilities.

Fractures in Degenerative Joint Disease.—In 11 fractures occurring in individuals with degenerative joint disease (between fifty-six and seventy-

five years), the vertebræ were involved in 3, the ribs in 3, the radius twice and the neck of the femur, phalanx of a toe and ulna once each.[1] In all of the long bones, a simple fracture of the shaft occurred (except for the fracture of the neck of the femur). A compression type of fracture occurred in the vertebræ. In 5 of these, major trauma had brought about the fracture, while muscular effort was responsible for one (ribs). Union occurred in all in a normal period.

FIG. 164.—Typical **Heberden's nodes**, the *signposts of degenerative joint disease* (osteo-arthritis). Note their typical *characteristic position* at the bases of the terminal phalanges, while the proximal phalangeal joints are not usually involved in this process.

In dealing with a fracture in a limb with arthritic involvement, one is justified in immobilizing the part only long enough to permit sufficient callus formation to hold the fragments in good alignment. The cast is then removed and a splint is used for immobilization to permit passive motion of the affected joint in order to prevent marked limitation of motion or ankylosis.[1]

The presence of osteophytes is not pathognomonic of degenerative joint disease, but is probably the result of chronic irritation of bone in the immediate neighborhood of the joint.

Degenerative Joint Disease in the Fingers

1. Loss of cartilage, usually to one side of the terminal interphalangeal joint with slight lateral displacement.
2. Complete loss of cartilage; formation of osteophytes, Heberden's nodes.
3. Irregularity of joint surface with formation of excessive osteophytes.[103]

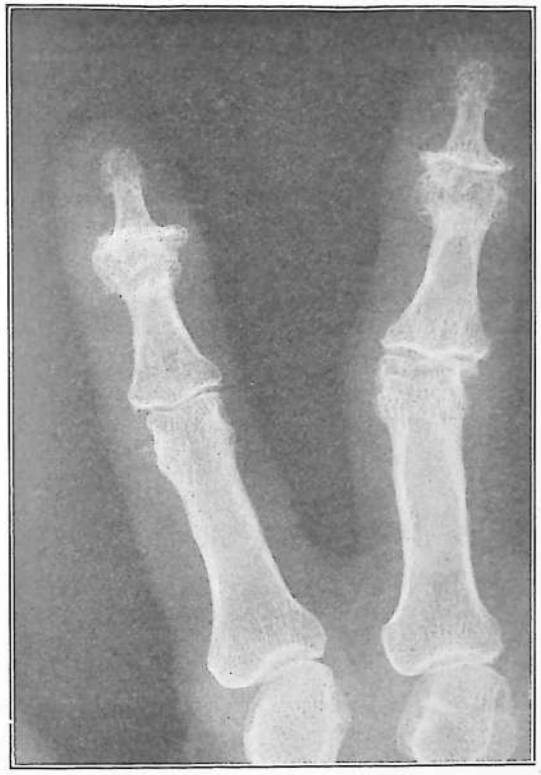

FIG. 165.—**Degenerative joint disease** with formation of **Heberden's nodes** at terminal phalangeal joints. Note also some involvement of proximal phalangeal joint (which occurs in a smaller percentage of cases). *Terminal phalangeal joints* are involved characteristically in *degenerative joint disease* (osteo-arthritis). Note *loss of cartilage* in terminal phalangeal joints as evidenced by narrowed or obliterated joint spaces. *Proliferation of bone* (hypertrophic changes) are evident at terminal phalangeal joints.

The *earliest manifestation of degenerative joint disease in the fingers is the decrease or loss of cartilage,* usually limited to one side of the terminal interphalangeal joint, leading to a falling together of the articular surfaces and a slight lateral displacement of the terminal phalanx.

Loss of cartilage is probably one of the earliest roentgen indications of the onset of any type of arthritis. Following this, small thornlike *osteophytes* appear, which seem to originate from the edges of the articular surfaces; they may become quite large. *Heberden's nodes* are caused by a marked splaying out of the base of the terminal phalanx at times asso-

ciated with an actual alteration in bone formation. The splayed-out appearance of the bone at the terminal joint is unlike that of osteophytic formation.[103]

With destruction of the cartilage, the *joint surfaces become irregular*. Tailors and seamstresses frequently show degenerative joint disease in the fingers, especially in the index and middle fingers, suggesting that these changes are in some way associated with overstrain and trauma.

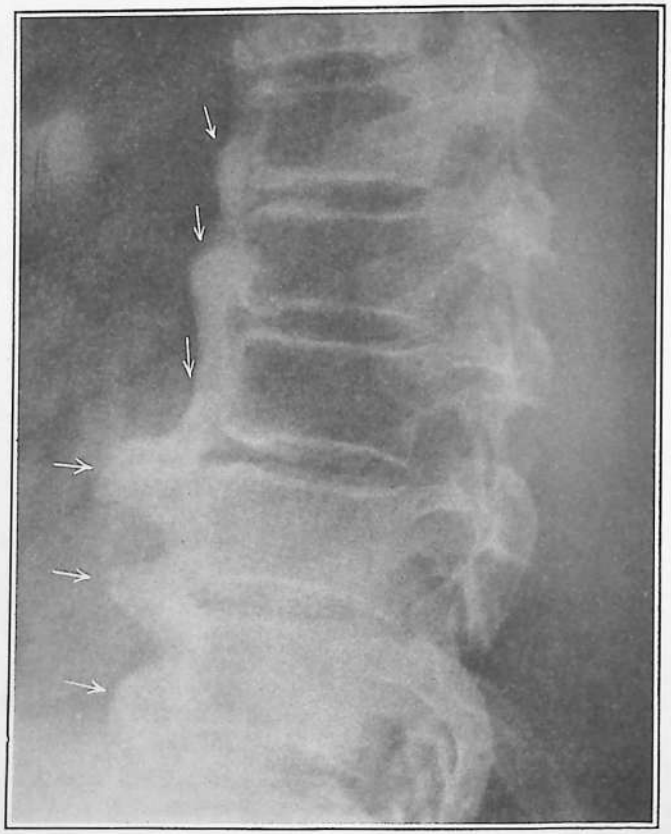

FIG. 166.—**Degenerative joint disease** seen here involved the *thoracic vertebræ* (in a patient aged sixty-seven years). There was *marked lipping with complete bridging* across the intervertebral spaces of some of the thoracic vertebræ. The appearance resembled that of a *flowing hyperostosis*. These changes represent an *advanced stage of degenerative joint disease*.

Even in the most advanced cases, one does not see bony ankylosis, fusion of carpal bones or extensive subluxation of joints.

In some cases of degenerative joint disease, one may find small opaque bodies in the capsule or periarticular tissues, unattached to bone. These seem to be islands of true bone developing in tissues which normally should contain no bony elements and are not broken off portions of osteophytes. These are found only in relation to the finger joints and do not seem to occur in any other pathologic condition. They vary in size from a grain of sand to a grain of wheat.

Roentgen Differentiation of Rheumatoid and Osteo-arthritis

	Rheumatoid Arthritis	Osteo-arthritis
Earliest signs	Fusiform swelling of soft tissues and rarefaction of trabeculated ends of bones	Formation of osteophytes and narrowing of the joint space
Soft tissue involvement	Fusiform swelling about joints, especially in phalanges	Usually none
Lipping and osteophytes	In about 75 per cent of cases	In every case
Heberden's nodes	None	Very frequent
Punched-out areas of bone at articular margins	Very common	Usually absent
Systemic decalcification	In almost every case; may be the earliest roentgen sign	Mild and consistent with age of patient
Narrowing of joint space	In almost every case	In about 50 per cent of cases
Ankylosis (fibrous and bony)	In approximately 25 per cent of cases	Very rare; only in spine

Roentgen Differentiation of Gout and Rheumatoid Arthritis

	Rheumatoid Arthritis	Gout
Punched-out areas of bone at articular margins	Common (over 80 per cent of cases)	Equally common; areas usually larger
Systemic decalcification	Common	*Absent* except to mild degree consistent with age of patient
Thinning of articular cartilage	In almost every case	In about two-thirds of cases
Periarticular swelling	Symmetrical, fusiform	Asymmetrical swelling about joints
Bony ankylosis	In about 25 per cent of cases	Very rare
Involvement of metatarsophalangeal joint of great toe	May occur—not characteristic	Very frequently involved
Joints involved	Proximal interphalangeal joints of hands, metacarpo-phalangeal joints, wrists, knees, spine; others less often	Especially hands and feet; may affect any joint but is rare in spine, hips and shoulders

Hereditary Multiple Exostoses.—Jaffe discussed in detail **hereditary multiple exostosis** as a congenital anomaly of skeletal development.[48] In its more severe form, multiple exostosis is a rather conspicuous skeletal disorder with characteristic knobby protrusions, especially in the metaphyseal regions of the long bones. Other names for the disorder have included "multiple cartilaginous exostoses," "multiple exostoses," "heredi-

tary deforming chondrodysplasia," "diaphysial aclasis," "dyschondroplasia" and "multiple osteogenic exostoses."

In the cases recognized clinically, the patient or a relative accidentally discovered one or more "bumps" on the skeleton. These are usually

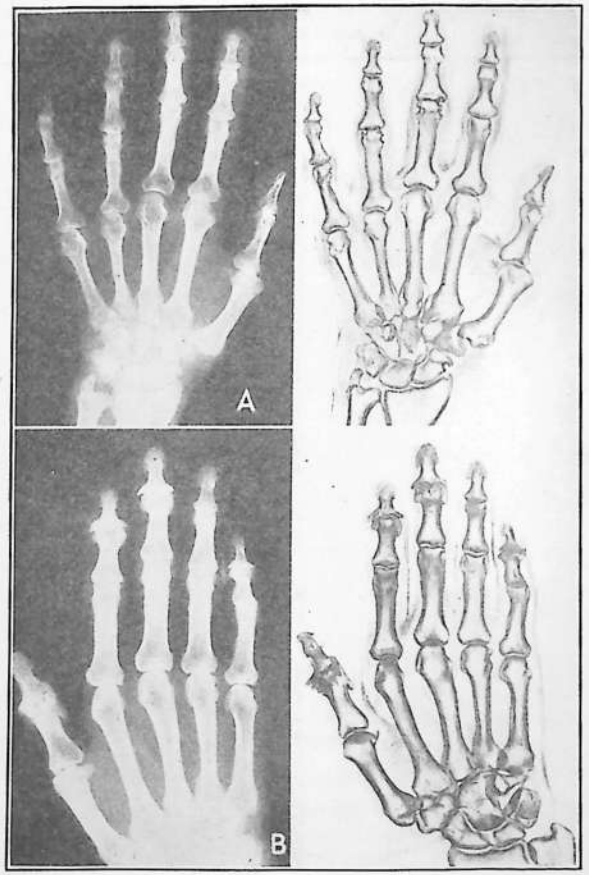

FIG. 167.—*A*, Roentgenogram and drawing showing some features of rheumatoid arthritis: systemic decalcification, small areas of atrophic destruction of bone, narrowing of the joint spaces, swelling of the soft tissues and effusion, which is symmetrical and is especially marked at the proximal interphalangeal joints. *B*, Roentgenogram and drawing showing some features of degenerative joint disease: absence of decalcification, marked production of bone, especially at the terminal interphalangeal joints, and comparative absence of destruction of bone or changes in the soft tissues (Taylor, Ferguson, Kasabach and Dawson, courtesy of Arch. Int. Med.)

noticed between the ages of two and ten. Later, similar lesions may appear on other bones. Months or years may elapse between the discovery of the first lesion and the seeking of medical aid. Growth of the exostoses slows down after adolescence and no new lesions form during adult life.

The presenting protuberance is most commonly felt on the metaphysis of some long bone, especially near the knee, ankle or shoulder. The long bones are involved more heavily as more exostoses appear. In severe cases, exostoses may also be found on the juxta-epiphyseal regions of the metacarpal and metatarsal bones, phalanges, ribs, innominate bones and vertebræ. They are seldom found on the skull.

The exostoses tend to be *bilateral* and *symmetrical* in distribution, but one side of the body may be more severely affected than the other.

Severely affected individuals may show shortness of the limbs in relation to the trunk, resembling a mild achondroplasia.

Subjective complaints are *rare* in the early stages of the disease. Minor difficulties appear later especially from pressure of a large exostosis on neighboring tissues. If the large exostoses are near joints such as the knee, hip or ankle, full articular function may be hindered. In some instances, a bursa develops between the growth and the overlying soft parts. Peculiar curvature and deformity of the forearm often develop. In Jaffe's series of 28 patients, chondrosarcomatous transformation occurred in one or another of the exostotic areas in 3 instances.

The exostotic area represents an out-pouching of the cortex of the affected region.

Approximately *half of the offspring of an affected subject manifest the disorder*. The defect may be transmitted through unaffected females in a family tree.

Multiple exostoses are supposedly due to defective modeling of the bone with perverted periosteal and perichondral activity.

If an exostotic area must be removed because of local difficulty, it should be excised intact with its fibrous periosteal coat to prevent recurrence.

GOUT

The characteristic feature of gouty arthritis is the deposition of sodium biurate crystals (which are transparent to *x*-rays) in the bone in the immediate neighborhood of the joints. These are seen on the film as punched-out areas on the surface of the articular bone, just beneath the cartilage or they may appear as holes in the articular end of the bone. They vary greatly in size from minute areas to ones large enough to resemble a bone cyst.

Roentgen Signs of Gout

1. Roentgen examination *may be negative* for years.
2. *Asymmetrical periarticular swelling* is usually the first roentgen sign.
3. Minute localized destruction or disappearance of bony trabeculæ may occur in the immediate neighborhood of a joint.
4. In chronic cases, small *punched-out areas* may appear on the bony articular surface or cystic areas may appear in articular bone. Cartilage and bone are destroyed and small or large punched-out areas may appear.[13]
5. In later stages, one may see irregular, asymmetrical *topi* (in the soft tissue), bony hypertrophy and destruction of the articular ends of bones and cartilage with areas of erosion of varying size.

Roentgen Signs of Gout—(*Continued*)

6. Gouty changes are not often seen in larger joints; they do not occur in the shafts of the bones.
7. A number of joints of the hands or feet may show gouty changes while the remainder appear quite normal.
8. Always *examine the great toe* if gout is suspected as it is affected sooner or later in about 80 per cent of cases.
9. **Do not rule out gout if the x-ray is negative.**
10. Most suggestive roentgen findings: *punched-out areas* 5 mm. or more in diameter in the subchondral bone of the base or head of the phalanges of the hands or feet.

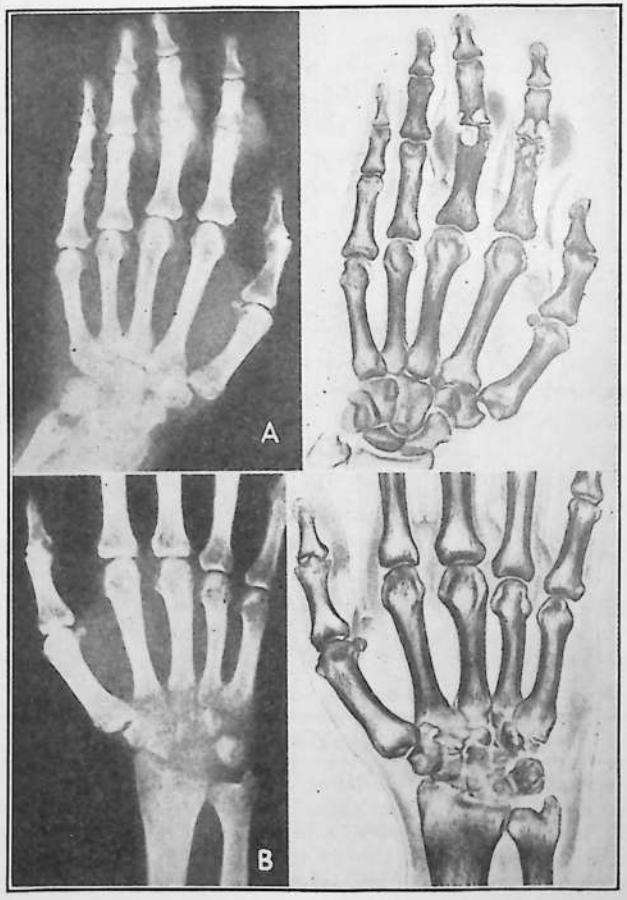

FIG. 168.—*A*, Gout: Roentgenogram and drawing showing large areas of atrophic destruction of bone with changes in soft tissues centered on the areas of destruction of bone rather than on the joint itself. *B*, Gonorrheal arthritis: Roentgenogram and drawing showing early stage of local and regional decalcification, swelling of the soft tissues and effusion, active destruction of bone and narrowing and obliteration of the joint spaces. (Taylor, Ferguson, Kasabach and Dawson, courtesy of Arch. Int. Med.)

A negative roentgenogram does not rule out gout in acute cases, as there are often no roentgen findings in the early stages. In one series, no roentgen evidences of gout were seen in 19 patients who had their disease more than twenty-five years.[39] *In all suspected cases of gout, the feet should always be x-rayed for punched-out areas;* these are usually 5 mm. or more in diameter and are found in the subchondral bone of the base or head of the phalanges of the hands or feet.[74] These are the most suggestive roentgen findings of gout. Marginal bony hypertrophy is also commonly found. Scott has stated that there are no diagnostic radiographic findings of gouty involvement of a large joint. (See section on Gout.)

FIG. 169.—Male aged twenty-two years. Attacks of joint pain for four years; pain in left great toe twelve years before this. At the time of this examination, there was marked soft tissue swelling around the proximal phalangeal joint of the right middle finger, with partial obliteration of the joint space. No marked demineralization was seen. Some soft tissue swelling was noted about the wrist, but the carpal bones were not affected. The metatarsophalangeal joints of both great toes were affected. See next figure.

FIG. 170.—*X*-ray of great toe in *same patient three and a half years later.* The changes are marked in this picture. *Tophi* are now present in the ears and on the mesial side of each great toe. Blood uric acid was markedly elevated. (The changes in the hands were also progressive with numerous large punched-out areas). Diagnosis: **gout.**

GONORRHEAL ARTHRITIS

Roentgen Findings in Gonorrheal Arthritis
1. May be negative early.
2. Periarticular swelling is usually the first sign.
3. Bony atrophy occurs as disease progresses.
4. In severe cases, the cartilage may be destroyed and bony ankylosis may appear early.
5. Marked local osteoporosis may occur in severe infection.

There are no pathognomonic roentgen signs of gonorrheal arthritis.[87] Suggestive changes include a spotty ground-glass atrophy of the neighboring bones, early acute localized atrophy, bony erosions and a punched-out appearance of the bone. Early diffuse atrophy of bone and rapid marked bone destruction may occur in acute gonorrheal arthritis. *Systemic decal-*

FIG. 171.—**Gonorrheal arthritis** showing *marked destructive changes* in the wrist.

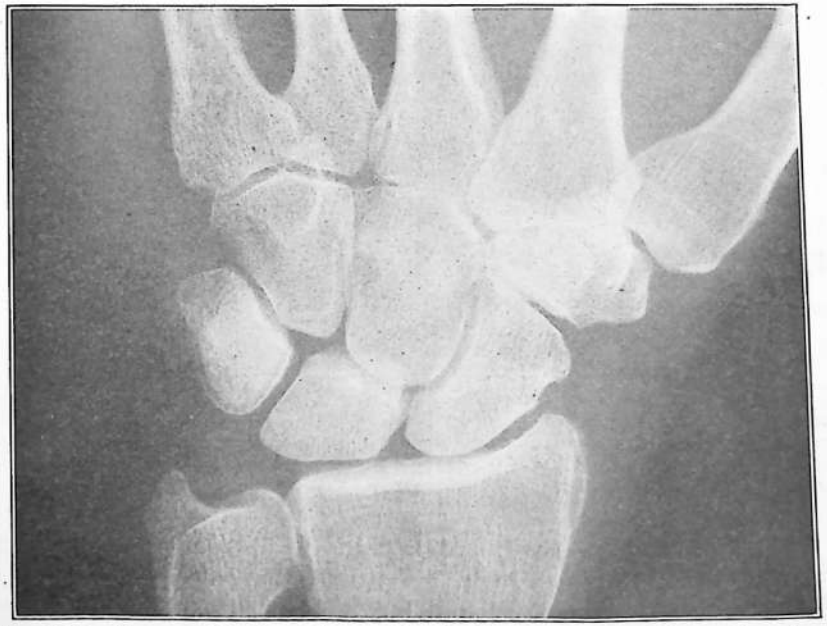

FIG. 172.—Bony architecture of normal wrist for comparison with previous figure. Note normal joint spaces and trabeculation of bones.

cification does not occur and lipping and production of osteophytes occur in less than 20 per cent of cases. Narrowing of the joint space, fibrous or bony ankylosis and soft tissue changes are common. Gonorrheal arthritis may simulate rheumatoid arthritis, but the roentgen changes are usually more marked and appear within a much shorter period of time.[107] (See section on Gonorrheal Arthritis.)

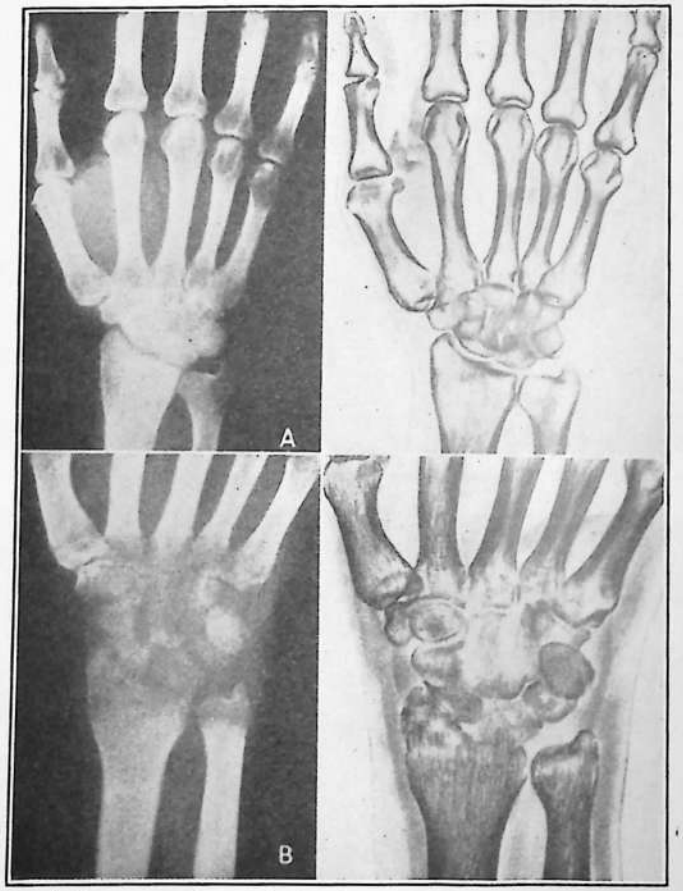

FIG. 173.—*A*, Later stage of gonorrheal arthritis. Note the fibrous and bony ankylosis and decalcification. *B*, Tuberculous arthritis, showing local and regional decalcification, a marked degree of active destruction of bone, narrowing and obliteration of the joint spaces, swelling of the soft tissues and effusion. (Taylor, Ferguson, Kasabach and Dawson, Arch. Int. Med.)

Lupo has described what he considered to be a characteristic roentgen sign of early gonorrheal arthritis which consists of the appearance of a thin line of increased transparency closely beneath the thin line of increased opacity that limits the bone toward the joint cavity. The length of this line is variable and it may extend along the whole joint border. This

corresponds to the laminar sequestration which he describes as characteristic of the pathology of gonorrheal arthritis. This sign is less evident in advanced cases.

Pyogenic Arthritis (Roentgen Aspects) (Pneumococci, Streptococci, Staphylococci, Gonococci)

1. Small, more or less circular *zones of atrophy*—sometimes confluent in polycyclical areas of decreased opacity. These areas are in an epiphysis or in the spongy bone. The lesions are sometimes surrounded by a thin halo of diminished transparency.[38]
2. Confluence of the above areas may result in a large break-down of bone.
3. In acute septic arthritis, the first roentgen sign is *destruction of bone in the articular border*, surrounded by a blended halo of demineralization, with atrophy of disuse appearing later.
4. Soft tissue swelling early.
5. *Destruction of bone and cartilage* as the disease progresses.
6. Later, hypertrophic changes.
7. Ankylosis, as a terminal feature.

TUBERCULOUS ARTHRITIS

Roentgen findings may be absent in the early stages of this disease. There are *no findings which are entirely typical of tuberculosis of joints in any stage.*[40] Although the findings vary greatly with the stage and intensity of the process, the *usual roentgen signs include:*[88] Synovitis, periarticular swelling, atrophy or sclerosis of bone, production and destruction of bone in the epiphysis and metaphysis, destruction of cartilage as seen by narrowing of the joint space, sequestration and the finding of cold abscesses or sinuses. Pomeranz advised that one *suspect tuberculosis in the presence of* any of the following: wedge or cone-shaped lesions; kissing sequestra; narrowing of a joint space occurring late in an infection, despite associated destructive changes; complications such as kyphosis, sinus formation or cold abscess.

In the absence of a history of trauma or gonorrhea, chronic monarthritis should lead one to suspect tuberculous arthritis. The joint damage in tuberculous arthritis requires much more time for its development than in other forms of arthritis. Tuberculous arthritis in old age may resemble hypertrophic or septic arthritis.[45] (See section on Tuberculous Arthritis.)

Rheumatic Fever (and Serum Sickness)

The radiologic findings in rheumatic fever are usually negative, the only features being:

1. *Periarticular swelling* in some cases.
2. *No bony or cartilaginous reaction.*
3. Slight increase of articular spaces, probably due to joint effusion.
4. Just appreciable bony atrophy—present only in cases of very long duration and due to inactivity.
5. Inflammatory thickening in the periarticular tissues.[38]

HEMOPHILIC ARTHRITIS

In the early stages of *acute hemarthrosis*, the roentgen-ray reveals the distended capsule, widened joint space and shadow of blood (which is denser than synovial fluid).

In *chronic hemophilic arthritis*, the joint space becomes narrowed and one sees irregular indented articular bony surfaces, deformity of the ends of the bones, bony cysts, increased density of synovial tissues and a map-like destruction of cartilage. In some cases there is atrophy of the bones.

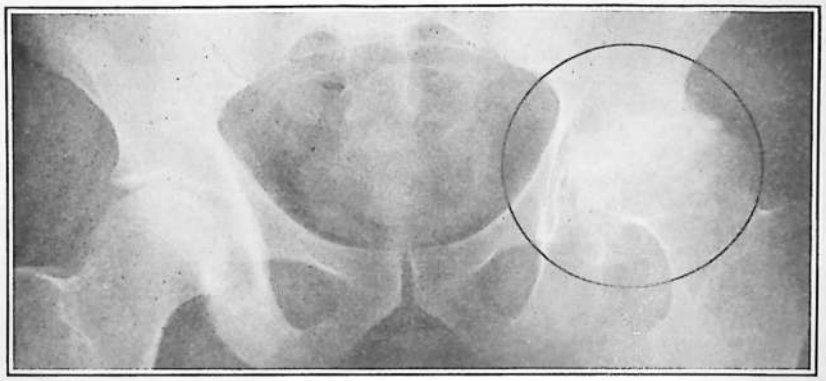

FIG. 174.—Patient, aged thirty-four years, with swollen joints and limited motion. *Hemophiliac.* Patient had severe discomfort in the right hip for twenty years. Late stage of **hemophilic arthritis** of the hip. (Knee joints have also been involved.)

Hemophilic arthritis presents no diagnostic roentgenologic picture and in the chronic stages may resemble gout, hypertrophic or tuberculous arthritis.[19] (See section on Hemophilic Arthritis.)

McDonald and Lozner studied the roentgenograms of joints of 15 adults with hemophilia.[76] In every patient they observed by *x*-ray pathologic changes in one or both knees, and in all but one patient, in one or both elbows. The degree of change generally increased proportionally with the age of the patient.

In the stage of acute hemarthrosis (whether spontaneous or traumatic), the joint is distended with blood. Key has shown that marked cellular reaction may occur in the synovialis following the instillation of blood into a joint cavity.[60,61] Fibrous ankylosis may eventually occur with thickening of the capsule. *The broken-down iron pigment deposits may cast a dense roentgen shadow and are occasionally mistaken for calcification.* The joint space is narrowed and the cartilage becomes irregular and thinned.

Subarticular cystic absorption may be accentuated by decalcification of the bone by disuse. These cysts may vary in diameter from a millimeter to more than a centimeter; they may be *single* or *loculated* and round or irregular. Key believed that intra-osseous hemorrhage occurred in bone which had been decalcified by limited use, and the hemophilic hemorrhage progressed to cause an area of aseptic pressure necrosis in the bone.

Not all hemarthroses result in joint changes. Gocht reported a patient with 45 bouts of hemarthrosis into one joint without permanent cartilaginous or bony changes.[32]

The *acute hemophilic hemarthrosis may be confused roentgenologically with* any traumatic, serous or purulent synovitis with distention of the joint space by fluid. The joint is usually partly flexed as the capsule has the greatest capacity (with least tension) in this position. One cannot differentiate these conditions roentgenologically. There is seen a generalized soft tissue clouding which is most marked along the lines of the distended capsule.

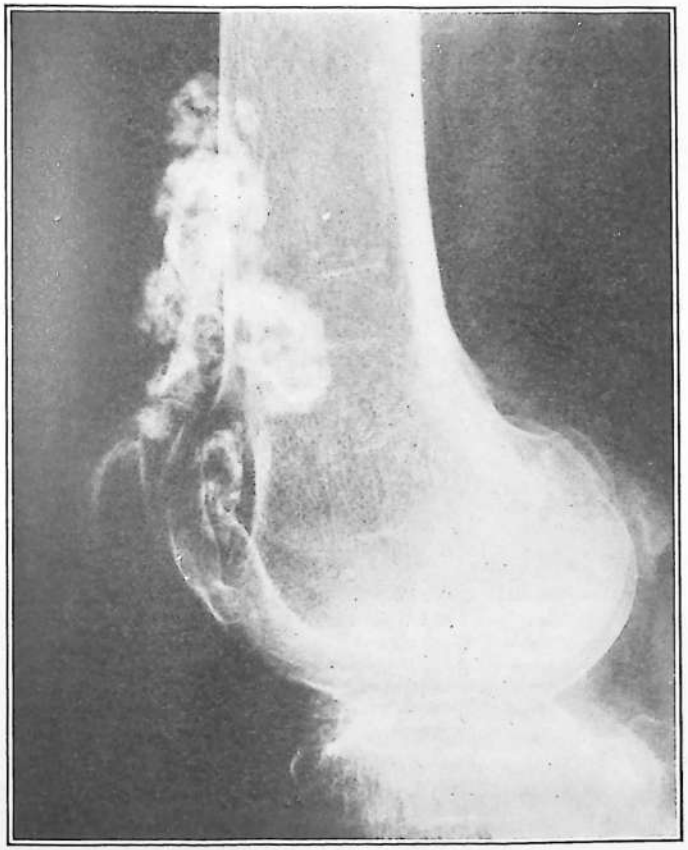

FIG. 175.—*Synovial calcification in the suprapatellar bursa;* slight hypertrophic changes involving the knee joint in a patient aged sixty-six years.

In the *chronic stage of hemophilic arthritis*, one may see generalized decalcification, cystic changes in the subchondral bones, narrowing and irregularity of the joint space, increased density of the synovial tissues resembling calcified areas and deformity of the ends of the bones. As *differentiated from degenerative joint disease*, the findings in hemophilic arthritis usually

are evident at an early age (before the age of twenty-one frequently); cystic changes are more common in hemophilic arthritis and generalized decalcification is more common and usually more severe than in degenerative joint disease.

FIG. 170.—Aged sixty-five years. Increasing *painless instability*. Marked *deformity* of the left knee, consisting of a marked increased density of the bones forming the joint, marked narrowing of the joint space and much *osseous débris* in the surrounding soft tissues. The femur and tibia are in rather *poor alignment*. The appearance is characteristic of a **Charcot joint.** The characteristics of syphilitic joint disease are presented in a later chapter.

Hemophilic arthritis can be *differentiated roentgenologically from rheumatoid arthritis* by (1) the common involvement of the proximal phalangeal- and metacarpophalangeal joints in rheumatoid arthritis (which is uncommon in hemophilia); (2) true bony ankylosis may occur in rheumatoid arthritis (but is very rare in hemophilic arthritis); (3) subchondral cystic changes are seen much more often in hemophilic than in rheumatoid arthritis.[76]

Miscellaneous.—Roentgen findings have been described in **alkaptonuric ochronosis.**[87] The articular manifestations of **leukemia** [34] and of **leprosy**[112] have also been discussed.

TRAUMATIC ARTHRITIS

Traumatic arthritis may resemble degenerative joint disease radiologically.[20] Traumatic arthritis from injury results in thickening of the synovial membrane, narrowing of the joint space, with necrosis of the cartilage and proliferation of bone.

Traumatic Arthritis

1. *Periarticular swelling* is the earliest sign.
2. *Bony hypertrophy* may follow severe trauma (as baseball fingers).
3. In very severe trauma, *destructive changes* may appear (especially in the hip), with narrowing of the joint space, necrosis of cartilage and proliferation of bone.
4. In *chronic traumatic arthritis* due to recreation or posture, the early findings are usually negative except for a small swelling occasionally (as the tennis wrist); later, hypertrophic changes usually appear (as in prize-fighters).[13]

Camp has classified the malacic diseases of bone. Because of the large number of diseases which affect the structure of bones, roentgen diagnosis may be difficult. Some of these *malacic disorders* include atrophy of disuse or of arthritis; congenital defects such as osteogenesis imperfecta and fragilitas ossium; dietary insufficiency as rickets, osteomalacia; renal dwarfism; hyperparathyroidism; hyperthyroidism; Cushing syndrome; diabetes mellitus; suprarenal disease; senile osteoporosis; postmenopausal osteoporosis and *osteitis deformans* (*Paget's disease*).

ROENTGEN THERAPY IN ARTHRITIS

There has been a recent wave of enthusiasm and interest in roentgen therapy in rheumatoid arthritis. Results to date include mainly symptomatic improvement, with at least *temporary relief of pain* (and in some instances, swelling) *in about a third of the patients with rheumatoid arthritis of peripheral joints*, and *excellent results* both subjectively and objectively *in treatment of rheumatoid arthritis of the spine* (rheumatoid spondylitis).

We have not seen marked permanent improvement in rheumatoid arthritis of peripheral joints with the doses employed at present. It must be remembered that these doses are merely experimental; it may follow that larger doses to peripheral joints will be of more definite value.

In an experimental study of the effects of roentgen therapy on rheumatoid arthritis of peripheral joints, we gave from 50 to 75 roentgens per treatment. Treatments were given from three to five times per week and the total over any one portal did not exceed from 450 to 600 r per series. Not more than two portals were usually treated per day, and in some instances only one was treated each day.

Factors were: 135 to 140 pkv, 8 ma, 0.25 to 0.5 mm. Cu filter, 25 to 30 cm. skin target distance (25 for small and 30 for larger joints). When the spine was treated, a 40 cm. skin target distance was used. In most

instances, three joints were selected which caused the patient most discomfort. Two of these were then irradiated while a third received the same treatment (but with a lead filter in place, to determine any possible psychic effect). *About 30 per cent of the individuals had some gastrointestinal upset* (mainly nausea, and occasionally, vomiting) and in *about 15 per cent, this was severe enough to interrupt or temporarily alter treatment.* White blood and differential counts and Hb determinations were made twice per week. The interval between each series of treatments was one month. When patients with rheumatoid spondylitis were treated, 3 portals were used over the spine, including the sacro-iliac joints, and in some instances, the hips.

Children tolerate roentgen therapy well and in general were given approximately the same dose. *In men, the testes were protected by lead. The dosage used was insufficient to produce sterilization in women* (when the lower spine was treated). In general, 800 r or more are needed front and back to produce sterilization in the majority of women. Women have rheumatoid arthritis of the spine infrequently (only about 10 per cent as often as in men); however, *when women were treated, only half the usual dose was employed in the lowermost portal over the spine.*

In the treatment of *subdeltoid bursitis*, 4 portals are used: posterior and anterior, lateral and top; in some of these cases, the cervical spine was also irradiated.

Unfortunately, our work with roentgen therapy was interrupted by World War II, so that statistical analyses are not significant as yet. However, our impression was that *improvement did not persist when roentgen therapy was given to rheumatoid arthritic peripheral joints*—although about a third of these individuals did have definite subjective amelioration of discomfort and some decrease in joint swelling—as a temporary phenomenon.[44] It is hoped that variation in the dosage employed in the future may be of more benefit. *In the patients with rheumatoid spondylitis, roentgen therapy* in our hands has been of the *utmost value* in producing subjective and objective improvement with lessening of pain and stiffness, and in reducing the sedimentation rate in about half of these patients. The improvement in rheumatoid arthritis of the spine following roentgen therapy in our cases persisted; if discomfort recurred, this was usually relieved by further *x*-ray therapy. One must not get the impression, however, that roentgen therapy will straighten the spine in a long-standing chronic spondylitic wreck with marked ligamentous calcification. Orthopedic measures, physical therapy and general sensible care must be given to these patients in addition to the roentgen therapy.

Our results of *roentgen therapy in subdeltoid bursitis* have been very *good.* In acute (non-traumatic) subdeltoid bursitis, from 60 to 75 per cent or more of the patients are freed of pain, many of these dramatically following 2 or 3 treatments. It must be remembered that there may be a definite aggravation of pain following the first treatment probably due to the active hyperemia which is produced. *X*-ray therapy is not of definite value in chronic adhesive bursitis.

Smyth and Freyberg reported that *results with roentgen therapy were unpredictable and unreliable in rheumatoid arthritis of peripheral joints, but marked improvement was noted in rheumatoid arthritis of the spine.* Local high voltage roentgen irradiation over the spine resulted in significant improvement in 72 per cent of all patients treated. In the *early stage of the disease,* results of roentgen therapy were excellent, *92 per cent showing significant, sustained, objective and subjective improvement.* In the late stage, even after calcification of spinal ligaments, significant reduction in pain and stiffness occurred in a high percentage. In 41 per cent of cases, there was a significant decrease in the sedimentation rate.

Most rheumatologists now consider **roentgen therapy the best method of controlling the symptoms of rheumatoid arthritis of the spine;**[35,109] this should be combined with proper orthopedic management, physical therapy and general care. It has not yet been determined whether the course of the disease is favorably affected by roentgen therapy or whether the benefit is entirely symptomatic. Others have commented upon the favorable results following roentgen therapy in these cases.[35,52,59,102,105,124]

Smyth and Freyberg studied the *possible psychic effect of roentgen therapy,* using a lead screen in some cases to prevent the roentgen-rays from reaching the diseased part. *None of the patients protected from irradiation by lead screens showed significant improvement,* but without exception, when they were later treated with roentgen-rays, significant improvement occurred. Other patients were treated first with irradiation directed to one area of the spine, and improvement was noted only in the area treated; later, a different area of the spine was treated, using a lead screen, with almost uniformly no beneficial effect following.

In patients with early disease (rheumatoid spondylitis), *striking relief of pain occurred soon after the first series of treatments,* with continued gradual improvement to the point of complete disappearance of all clinical evidence of the disease. When pain in the back was relieved, some patients with partial calcification of ligaments showed a significant increase in motion and chest expansion. The beneficial effects persisted in most patients throughout the period of observation. In those whose symptoms recurred several months after therapy, relief was usually obtained by additional treatments. Most individuals reported some improvement within a week after the onset of therapy and some individuals insisted that pain and stiffness were decidedly reduced a few hours after therapy. In others, improvement was less and slower and no change was evident until after the second series of treatments. If improvement did not occur by the time of completion of the second series, it was unlikely that benefit would follow additional radiation.

Anatomic improvement did not occur following symptomatic benefit from roentgen therapy. Roentgenograms made from six months to two years after therapy showed no evidence of anatomic improvement. In some there was an increase in the calcification of the paravertebral ligaments despite significant clinical improvement and reduction in the sedimentation rate.

Ill-effects included *leukopenia* in a few instances (as low as 1100 W.B.C.), *gastrointestinal upsets* (anorexia, nausea and vomiting) and *temporary depilation over the neck* when irradiation was given above the hair line. Leukopenia occurs uncommonly with the dose of roentgen therapy employed, but it is advisable to perform a leukocyte and differential count at least each week, or just prior to each treatment session.

The reduced dose employed by Freyberg was a total of 300 r per series to each field, 150 r being given every other day. Three series of treatments were usually given to the same area at monthly intervals. If symptoms were not completely relieved, or recurred, or if symptoms appeared in new areas, further treatment was given. Irradiation was given to any or all portals of the spine (cervical, thoracic, lumbar, sacral) and the sacro-iliac joints, or any combinations, selecting those parts for treatment by means of symptoms and physical signs. In some instances of far-advanced disease, hips and shoulder joints were also treated. *Factors included:* 200 kv (175 kv constant potential equivalent), with 0.5 mm. copper and 1 mm. aluminum filtration, a half value layer of 0.9 mm. of copper, a 50 cm. skin target distance and an output of 50 r (measured in air) per minute. The usual size of the field was 200 to 300 sq. cm.

Because of the *possibility of sterilization in the female* from large amounts of roentgen irradiation over the pelvis, one does not give a total of more than 300 r over the female pelvis in a series of treatments.[28]

Scott reported excellent results with **"wide-field" roentgen therapy** in patients with rheumatoid spondylitis. He used a special wide-angle *x*-ray tube to deliver a **"roentgen-ray bath"** to the whole trunk, front and back, which are covered by two overlapping 12-inch fields. The *physical factors* were: 140 kv, 3 mm. Al filter and 60 to 100 r, with a standard distance of tube to the patient of 17 inches. The first series of treatments consisted of 2 per week for 8 sessions; following a rest interval of six weeks, a second series included 2 treatments per week for 6 sessions and following another interval of six weeks, a third series of weekly treatments was given for six weeks.

The mechanism of action of roentgen therapy in rheumatoid spondylitis is unknown. Following roentgen therapy, there occurs a marked dilatation of the capillaries, subcapillary plexuses and arterioles under the area treated, which persist not only for a few hours (as occurs with other forms of heat), but for at least several days. This active hyperemia may be part of the answer to the improvement from roentgen therapy in these patients.[16]

Roentgen therapy has been used in practically every form of arthritis.[21,22,29,33,42,53,71,121,122] Although most rheumatologists agree that it is of marked benefit in patients with bursitis (especially acute or subacute subdeltoid bursitis with or without calcification) or with rheumatoid spondylitis, its use in other rheumatic disorders is the subject of much debate. *Kahlmeter* (of Stockholm) *has given more than 25,000 roentgen treatments to over 4000 cases of all kinds of rheumatic diseases over a period of ten years.*[54] He has pointed out that although roentgen therapy was used as early as 1897 in

cases of neuralgia and arthritis, reports concerning its use have been very few, although usually enthusiastic. Kahlmeter believes that roentgen therapy is useful in acute bursitis and tendinitis and in some cases of chronic arthritis and neuralgia.[55]

Horwitz and Dillman found roentgen therapy to be of value in the control of joint effusions which were seen in certain non-specific synovial lesions of knee joints in human beings.[47] They found no ill-effects on articular or para-articular structures of dogs when their more vulnerable articulations (in the extremities) were treated with much larger doses than were humans. No immediate or remote ill-effects could be found in these dogs.

No heat or plaster should be applied to the skin for at least a week preparatory to or following roentgen therapy; this includes short and long wave diathermy, hot air, ultra-violet radiation, ointments, dressings, heating pads, plasters, etc. By such precautionary measures, the danger of skin reactions will be minimized.

Best results in roentgen therapy have been obtained in acute bursitis and tendinitis (including subdeltoid,[15,37] trochanteric, patellar and Achilles bursitis), rheumatoid spondylitis and gonorrheal arthritis. Roentgen therapy has also been used in psoriatic arthritis. Roentgen therapy has been used successfully in the treatment of coccygodynia and sciatica.[2,3] Some workers[70–72] feel that roentgen therapy of the heart is of definite therapeutic value in some cases of carditis in rheumatic fever; however, such work is experimental and is not recommended.

Because the subject of roentgen therapy in arthritis is in its infancy, the question of dosage is still one of trial and error. The dosage employed by Kahlmeter is as follows:

	Single dose		No. of doses per series	Days between applications in one series
Rheumatoid arthritis	100–150 r		2–3	2 3
Acute gonorrheal arthritis	75–100 r		3–4	2 3
Subdeltoid bursitis	200 r		1–2	2–3
Lumbago	150 r		2–3	2–3
Osteo-arthritis of hip	150–200 r		2–3	2–3
or spine	...	or	5–6	7

Roentgen therapy will produce no effect on cartilaginous destruction, ankylosis, intra-articular exudates or contractures in rheumatoid arthritis. Kahlmeter reported good results in rheumatoid arthritis such as decrease in periarticular swelling and tenderness, and less pain along tendon sheaths and tendinous and muscular attachments. Langer employed roentgen therapy to the sympathetic ganglia in the side of the neck and lumbar spine in addition to local therapy.[68]

Smyth and Freyberg treated patients with rheumatoid arthritis of peripheral joints by means of local high voltage roentgen irradiation and noted objective improvement in only 26 per cent of cases; 44 per cent showed no subjective or objective benefit.[110,111] The sedimentation rate remained unchanged in 85 per cent of cases. Results of roentgen therapy were unpredictable and unreliable when peripheral joints were treated and

were so discouraging, that roentgen therapy was abandoned in rheumatoid arthritis of peripheral joints, except in rare, obstinate cases or cases in which a psychic effect was desired.

Swaim has reported much better results following roentgen therapy.[116] *In 85 patients with rheumatoid arthritis, subjective improvement occurred in more than 90 per cent of* those whose articular pain and swelling was of six months' duration or less; objective improvement was also noted. In those whose rheumatoid arthritis was from six months to two years in duration, objective improvement occurred in 60 per cent and subjective improvement in 85 per cent. In *those with arthritis of more than two years' duration, objective improvement was seen in 52 per cent* and *subjective improvement in 72 per cent.* In mixed forms of osteo- and rheumatoid arthritis, subjective improvement occurred in about two-thirds of the patients and objective improvement in one-fourth. *Swaim concluded that the earlier roentgen therapy is begun in rheumatoid arthritis, the more rapid is the benefit; children seemed to improve more rapidly than adults. In his experience, frequent small doses over a long period of time resulted in more improvement than larger doses given infrequently or at longer intervals.*

Although Swaim began roentgen therapy in rheumatoid arthritis for the relief of pain, he noted a gradual reduction of swelling and an increase in motion. He believed that the chief effect was on the soft tissues; these seemed to be less swollen following roentgen-ray treatments.

Swaim and his colleagues gave their roentgen treatments approximately every two weeks. They believed that the **roentgen formulas**, which they gradually developed after years of trial, were of importance. These were as follows: *For knee joints or smaller joints:* 118 kilovolt peak, 5 milliamperes, 1 mm. Al filter, 14 inches distance, total of 225 r (given at the rate of 90 r per minute); *for spines:* 130 kilovolt peak, 5 milliamperes, 3.5 mm. Al filter, 14 inches distance, total of 195 r (39 r per minute for five minutes); *for degenerative joint disease of the knees and hips:* 130 kilovolt peak, 5 milliamperes, 3 mm. Al filter, 14 inches distance, 107¼ r (39 r per minute for two and three-quarter minutes). They concluded that this form of therapy relieved pain in degenerative joint disease and has been of benefit to hips in rheumatoid spondylitis, and has been helpful locally in early cases of rheumatoid arthritis if given adequately.

No radiologic technic has been developed which will cause dissolution of bony deposits. Extensive osteoporosis has been reported following high voltage therapy.[27] Leddy believes that the beneficial effects of roentgen therapy in arthritis are due chiefly to destruction of leukocytes in the inflammatory process.[69]

Superficial irritations of the periosteum (such as epicondylitis and styloiditis) and bursitis usually respond well to roentgen therapy.

Ochsner and Mumford noted marked improvement following roentgen therapy in rheumatoid arthritis.[81] Weinberg reported on the results of roentgen-ray treatment in 160 cases of arthritis and painful joint conditions.[124] Most of the patients were relieved of pain and remained symptom-free; the results were particularly good in cases of rheumatoid spondylitis.

Scott divided the *rheumatic conditions* amenable to roentgen-ray therapy into two groups: *those amenable to local roentgen therapy* such as brachial neuritis, neuralgias, acute or chronic bursitis, tendinitis, myalgia, sciatica, acute or chronic gonorrheal arthritis, local and infective arthritis, degenerative joint disease (except spinal involvement), acute and chronic gouty arthritis, and as a palliative measure, in rheumatoid arthritis.

Wide field roentgen-ray therapy was advised in fibrositis (back tissues), lumbago, periarticular arthritis, polyarticular arthritis, osteo-arthritis of the spine and rheumatoid arthritis. His *results* may be summarized as follows:

Degenerative hip joint disease—20 per cent did well.

Degenerative knee joint disease—very good results.

Degenerative joint disease of smaller joints—very slight alteration (if any) in the size or the shape of joints; satisfactory results—*i. e.*, relief of symptoms, which is permanent in some cases.

Gonorrheal arthritis—brilliant results (roentgen-ray therapy is no longer used in this disease because of the effectiveness of sulfonamides or penicillin).

Gouty arthritis—immediate relief of acute attacks by local roentgen-ray application; chronic gouty arthritis—relief of symptoms and decrease of joint swelling. Effects are merely palliative in either case (colchicine is so effective in relieving the pain of acute gout that roentgen treatment is usually unnecessary).

Acute infective arthritis—rapid and sometimes spectacular results; very satisfactory results in chronic arthritis.

Bursitis, neuritis, sciatica, myalgia—excellent results.

Rheumatoid arthritis—effects may be delayed several months; *best results are often obtained if treated early. These patients are very radiosensitive, especially during the active stage of the disease.*

Fibrositis—definitely *satisfactory* results.

Degenerative joint disease of the spine—relief of symptoms.

The **technic of roentgen therapy which is utilized in our patients** under the direction of *Dr. Eugene P. Pendergrass* is as follows:

1. **In Thin Patients.**—Factors are 135 kv.; 8 ma.; ¼ mm. copper filtration; H. V. L., 9.0 mm. Al.; distance 30 to 40 cm. Fifty r is given and repeated every second day for 3 doses—with a total dose of 150 r per field treated.

If the spine is being treated, two fields are used. Only one portal is treated each day, except in emergencies.

When peripheral joints are being treated, the same factors are used, treating at the most three joints at any one session. In the treatment of peripheral joints one does not have as much back scatter and reaction as when irradiating over the trunk.

2. **In Large Patients or When Treating Patients With Body Casts.**—Factors are 200 kv.; ½ mm. copper; 15 ma.; H.V.L., 1.0 mm. Cu.; distance 50 cm. Individual dose—50 r.

In any series of treatments, the course of therapy is repeated in from

six to twelve weeks, varying with the response of the patient. If necessary, one could repeat the series for 6 entire sets of sessions without disturbance of testicular or ovarian function.

3. **In Bursitis** (acute or subacute).—Fifty r is given over the hips or shoulders; distance 25 to 35 cm. Treatments are given daily for four days; after a week, another single treatment is given, followed again in another week by another single treatment if necessary—giving a total of 6 treatments. If there is not some definite improvement following this, no further therapy is given.

In chronic subdeltoid bursitis, 2 treatments are given per week for six weeks, with a total dose of approximately 600 r. In these patients, the dosage is modified to fit the case. When *bilateral subdeltoid bursitis* is present, one may desire to give 50 r to one shoulder and 75 to 100 r to the other.

Factors in treatment of subdeltoid bursitis are 140 kv.; filtration—$\frac{1}{4}$ mm. copper or less; 8 ma. Treatments are given anteriorly, posteriorly, laterally and on top of the shoulder; in some cases, the back of the neck is also treated.

Summary of Roentgen Therapy in Arthritis

Subdeltoid Bursitis.—Excellent results in acute or subacute cases; very poor results in chronic adhesive cases.

Rheumatoid Spondylitis.—Very good results; probably the best single therapeutic agent available.

Rheumatoid Arthritis of Peripheral Joints.—Results are questionable. Some reliable authorities report subjective and objective benefits in a large percentage of patients (the more rapid benefits occurring in the group treated early in the disease); others have obtained very little improvement. Frequent small doses over long periods of time are advised.

Degenerative Joint Disease.—Results are questionable. Relief of pain is reported in some. Others report no benefit.

Gonorrheal Arthritis.—Very good results; however, sulfonamides or penicillin are the treatment of choice.

Gouty Arthritis.—Acute attacks of gout demand colchicine; symptoms of gouty arthritis have been relieved by roentgen-ray treatment.

Fibrositis.—Good results reported, especially in bursitis, *q. v.*

RADIO-ACTIVE SUBSTANCES IN THE TREATMENT OF RHEUMATIC DISEASES

Radium probably produces a stimulating effect on metabolism. Radio-active bodies are elements which may change into other elements by emitting energy as radiation, such as alpha, beta and gamma rays. There are three families of such substances: (1) Uranium—radium group; (2) thorium group; and (3) actinium group. The latter provides no substances which are of value in medicine at present.

Uranium is obtained from an ore, pitchblende (uranium oxide); after passing through six stages, uranium turns into radium.

Radio-active substances, such as radium, radon, mesothorium, radiothorium, thorium X and thoron, have been used in the treatment of rheumatic diseases.[123] Radium is employed as soluble salts, such as the bromide or chloride. Radium chloride and atophan have been combined for parenteral use.

Thorium is extracted from another ore, monazite (thorium oxide). Radon is a gas and is present in various mineral waters. Radiothorium has been used in this country in radio-activated drinks. Mesothorium bromide has been used for injection.

Radio-active substances may produce late untoward effects such as: necrosis of the jaw and progressive anemia, radio-dermatitis, pernicious anemia or myeloid leukemia.

Thorium X has been used in rheumatic and gouty conditions. It is said to be effective in psoriatic arthritis and in ankylosing spondylitis (in the later stages). It must be used with great care; complete blood counts should be made at frequent intervals.

Thorium X is never given subcutaneously but always intravenously or intramuscularly. It has been injected weekly for a series of 8 increasing doses not exceeding a total of 800 microgrammes (if the products of disintegration are left behind in its manufacture, or 1000 if these have been removed). At least eighteen months interval must be allowed between each series.

Radio-active substances should not be employed by the general practitioner. Their use in arthritis is still in the early experimental stage.

Radon ointment was credited with relief of pain in a majority of patients with "chronic arthritis," using alpha-ray therapy, with a fatty carrier (neutral anhydrous lanolin) as a vehicle for the radon.[49] No injurious effects were noticed even after prolonged use in proper dosage. Such work is still experimental.

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CHAPTER XLIII

RHEUMATIC FEVER

THE frequency of rheumatic fever in the armed forces, its occurrence several weeks following hemolytic streptococcal upper respiratory infections in many cases, its infrequency in the southern camps and concentration in the northern camps and Rocky Mountain area (especially Colorado and Wyoming), the use of sulfonamides as a prophylactic measure, and the recent suggestion that large doses of salicylates (by mouth or intravenously) may inactivate the disease more quickly or may prevent the occurrence of carditis in some cases, have all combined to place rheumatic fever in a very important position among the rheumatic diseases today.

Rheumatic fever is an important military disease.[31] Epidemics of hemolytic streptococcus infection and rheumatic fever have been reported in the armed forces.[154]

It has been estimated that there are about 170,000 new cases of rheumatic fever per year in the United States in times of peace, with an estimated total of 840,000 cases of rheumatic heart disease (active and inactive) per 100,000,000 people.

Coburn believes that *heredity* and *environment* combine to produce *individuals who do not handle hemolytic streptococcal respiratory infections in a normal manner.* In these persons, there is an inadequate immune response to the primary infection resulting in sensitization of the reticuloendothelial cells. Later, contact of antigen and antibody within these sensitized cells produces an abnormal reaction leading to the characteristic inflammation of rheumatic fever.

The incidence of rheumatic fever has increased in the Army since the outbreak of World War II. Special interest has been aroused in rheumatic fever in the Army because of its concentration in certain regions in the United States, its uncertain etiology, its definite relation to certain common acute infections and its disabling sequelæ.[120,122]

Recent research indicates that *the characteristic symptoms of rheumatic fever may be allergic manifestations on the part of sensitized body tissues to invasion with hemolytic streptococci or their products.* Among United States troops, several significant epidemiologic features have been observed: (1) The peak incidence tended to occur in May. Scarlet fever and other streptococcal diseases usually reached their peak in March. (2) There has been a *markedly higher prevalence of rheumatic fever in the Rocky Mountain region* of the Seventh Service Command, particularly in *Colorado* and *Wyoming* than in other sections of the country. This agrees with civilian data which show that civilian rates tend to be lowest south of the 35th parallel of latitude. Although exceptions have occurred, *tropical and subtropical regions have far less rheumatic fever than areas in the North Temperate Zone* where low winter temperatures are encountered. (3) In some posts,

a high incidence of rheumatic fever followed a high prevalence of common respiratory diseases, not identified as streptococcal; it is possible, however, that unrecognized or unreported streptococcal infections may have constituted a considerable part of the total respiratory admission rate. In certain posts, a high incidence of rheumatic fever often followed a winter during which scarlet fever or streptococcal sore throat were very common.

Most of the cases in the Seventh Service Command developed in men who had been in the service command several months. It was not shown that men from any particular section of the country were especially susceptible. Although the articular manifestations yielded promptly to the usual medical therapy, *20 per cent of cases at one hospital developed evidence of at least transient cardiac damage.* Individuals with cardiac damage were separated from the service and a specified period of close observation was required in all cases before returning them to full duty.

The Commission on Hemolytic Streptococcal Infection and the Commission on Air-Borne Infection, operating under the Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army, appointed by the Secretary of War, has coöperated with army hospitals in the study of the *chief medical problem* which *is the prevention of relapses in the convalescence of rheumatic fever patients.* Recent published investigations suggest that systemic administration of sulfonamides may be effective in preventing recurrences; the avoidance of relapses will probably reduce the incidence of late cardiac sequelæ of rheumatic fever.[111]

Prior to World War II, it was generally believed that rheumatic fever was essentially a disease of childhood. This is not the case during war time, as *rheumatic fever constitutes a serious problem in branches of the armed forces.* In some branches of the service, from 100 to 200 new cases of rheumatic fever have been reported per week—*i. e.*, an incidence of 100 per 100,000 per year. At some bases, the incidence of rheumatic fever in World War II has been about ten times the rate of gonorrheal infection there. *Young adults in the service have been attacked in large numbers in certain areas.*

Various measures have been used in an effort to combat rheumatic fever in the service. *Oiling of blankets and of floors* reduced the hospital incidence of respiratory disease 30 per cent. In some areas sulfadiazine was given as a prophylactic measure to all new troops coming into an epidemiologic area (4 gm. in twenty-four hours). In addition, a sharp lookout was kept for a rise in streptococcic disease and a rise in carrier rate; all of these were treated with sulfadiazine. Other troops were given sulfadiazine daily for four days, with a repetition of the sulfadiazine after a rest interval of a few days. Still others received sulfadiazine daily for months.

It was found that the incidence of streptococcal disease was reduced only slightly by a dose of 4 gm. of sulfadiazine (in twenty-four hours) to all new troops coming into the area. Although this was effective in meningococcic infections, this was not of marked value in hemolytic streptococcal diseases. *Treatment of carriers and of individuals with streptococcal infections by sulfadiazine reduced the number of admissions for respiratory*

diseases and the number of streptococcic infections for about five days.[54] *Treatment of all troops with 1 gm. of sulfadiazine daily for four days, followed by a repetition of this dose after a rest period of four days, reduced the hospital admissions for respiratory diseases and the number of positive cultures for hemolytic streptococcus as much as 40 per cent; this also reduced the incidence of rheumatic fever almost proportionately.*

It has been found that the same type of streptococci (17, 19 and 30) occurred in the southern camps as in northern camps and were present as heavy infestations with a serious streptococcus disease rate, but without the occurrence of rheumatic fever in the southern camps. Thus, *although the same organisms*—the Type A *hemolytic streptococci were found in the South* as in the camps in the North, the fact that *rheumatic fever did not occur in those infected with hemolytic streptococci in the southern camps* indicates that we need something more than a hemolytic streptococcus Type A infection to produce rheumatic fever. Incidentally, the boys at both the northern and southern camps come from approximately the same groups of the country and their heredity is thoroughly mixed so that there should have been just as many susceptibles at the southern camps as in the northern ones; however, only the susceptibles at the northern camps developed rheumatic fever. It appears, thus, that a Group A *hemolytic streptococcus infection alone is not sufficient to produce rheumatic fever, even in a person who is susceptible* (with proper hereditary factors).

The third factor (in addition to hemolytic streptococcal Type A infection and hereditary) is whether *meteorology* plays a rôle. It has been estimated that a very large percentage of all rheumatic fever in some branches of the service occurs in a not too large an area in the Rocky Mountain district.

In the army camps in the North, the **typical picture** which has been seen *has been a respiratory (hemolytic) streptococcal infection, followed about three weeks later by rheumatic fever.* It was found that there were many more severe streptococcal infections in the northern than in the southern areas.

In conclusion, it appears that **important etiologic factors in rheumatic fever** include:

1. *An infection of the upper respiratory tract* with Group A *hemolytic streptococci.*
2. A *susceptible hereditary background.*
3. The influence of *meterology* in some instances and
4. A still *undetermined x factor.*

Rheumatic fever is the most serious chronic condition originating in childhood and is one of the foremost problems in public health. *Rheumatic fever is the leading cause of death in children from ten to fourteen years of age,* is second only to tuberculosis in the fifteen to twenty-four year group and is third in the five to nine year olds (in whom communicable diseases rank first and pneumonia second as the causes of death). The introduction of sulfonamides and penicillin should greatly lower this mortality from pneumonia.[168] Rheumatic fever and heart disease cause almost 13 per cent of all deaths in white persons from five to twenty-four years of age.

Rheumatic Fever

1. **Etiologic agent:** present evidence is that the *hemolytic streptococcus* is of great importance in instigating exacerbations and recurrences. Other factors probably of importance: *climate, heredity* and an *unknown x factor.*
2. **Pathology:** *focal injury to collagen* with *fibrinoid swelling* of the mesenchymal ground substance, necrosis of collagen fibers, proliferation of primitive mesenchymal cells forming the *rheumatic granulomata* and finally *scar* formation. *Exudation* is an *early* manifestation, seen clinically as periarticular edema, increased synovial fluid, pericardial and pleural effusion. Exudates disappear quickly following the administration of antirheumatic drugs; proliferative changes are not similarly affected.
3. **Epidemiology:** seasonal incidence in the north temperate zone follows that of the streptococcal carrier rate with about a month's lag between peaks of the curves.[250] The geographic distribution is likewise parallel with the streptococcic carrier rate. *There is very little rheumatic fever in areas with very few streptococcal carriers* (Puerto Rico, Alabama, Labrador, the Virgin Islands and Spitzenberg). The *high incidence* of rheumatic fever *among children of poor, crowded families* is probably due to the greater probability of spread of streptococci under such conditions.[78]
4. **Recurrences:** a large percentage of rheumatic subjects exposed to an epidemic of streptococcal disease will probably have relapses. *Recurrences are unlikely in patients with rheumatic fever so long as they are free from upper respiratory infections with streptococci.* Careful nose and throat bacteriologic studies of individuals during epidemics in institutions have confirmed this.[250] In some cases, the relation between rheumatic fever and streptococci is frequently missed unless frequent careful examinations are made.
5. **Prophylaxis:** *sulfanilamide* used to prevent recurrence of hemolytic streptococcal respiratory infections during months when these are prevalent will usually prevent recurrences of rheumatic fever. In susceptible subjects (those children with a family background of rheumatic fever and with frequent sore throats), it is possible that sulfanilamide may be used prophylactically to prevent original attacks.
6. **Treatment:** *salicylates* to control fever, pain and pulse rate (and possibly to prevent cardiac damage, although the latter is still experimental—see section on therapy); *rest* as long as activity of rheumatic infection persists; *symptomatic* care; *protection against relapse.* This is discussed in detail later in this chapter.
7. **Prognosis:**
   (*a*) Some have only a single attack with no obvious cardiac involvement and live an expected normal life span.
   (*b*) Others have signs of valvular insufficiency which may disappear in a year or two, permitting a normal course in life.
   (*c*) This group shows persistence of valvular disease with a diminution in life expectancy.
   (*d*) A few may die during the initial attack from pancarditis or hyperpyrexia.
   (*e*) This group apparently recovers, but has repeated recurrences of rheumatic fever with increasing cardiac damage from each until cardiac failure occurs.
   (*f*) The last group runs a course similar to the previous for a year or two and then may overcome his infection, but be left with a badly damaged heart.[250]

Marked decreases in mortality have occurred from both the acute and chronic phases of rheumatic fever in the age group from five to twenty-four; the death rate from chronic heart disease has declined 62 per cent and from acute rheumatic fever—69 per cent in the past thirty years.

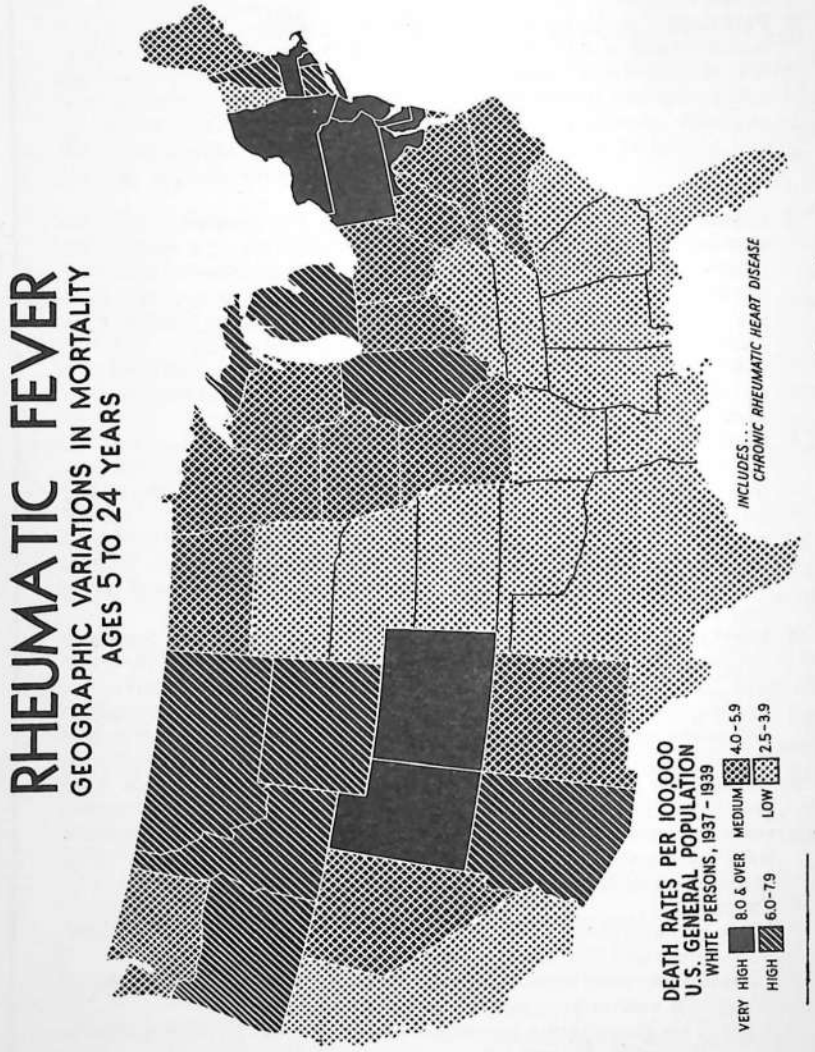

FIG. 177.—(Courtesy of the Metropolitan Life Insurance Company.)

Acute rheumatic fever takes its greatest toll among children of primary school age while chronic rheumatic heart disease becomes more important after the age of ten.[168] Between the ages of five and twenty-nine, most deaths from heart disease are rheumatic. From the Metropolitan Life Insurance statistics, the *death rates from rheumatic fever in the age group from five to twenty-four are greatest in* Wyoming, Colorado, New York, Pennsylvania, Massachusetts, Rhode Island, New Jersey, Delaware and Maryland and are *lowest in* the southern states from South Carolina to Texas inclusive (including Tennessee and Kentucky), as well as in California, South Dakota, Iowa, Kansas and Arkansas.

Rheumatic fever is responsible for more deaths of children from five to fourteen years of age than any other cause and accounts for a large number of deaths in older age groups as well. At present, 14 *states* have *programs* in operation *for the care of children with rheumatic fever or heart disease,* while others are submitting plans for similar programs. Such a program consists of education of parents in recognition of early symptoms, provision of hospital facilities and convalescent wards, public health nurses for home visiting and similar measures.[124] Federal funds have been used to include aid to state agencies for the development of such services for children affected with rheumatic fever.

Murphy has discussed the evolution of our knowledge of rheumatic fever with a historical survey of rheumatic heart disease.[171]

Incidence.—Rheumatic fever is an extremely common disease. Hedley has estimated that there are more than 1,000,000 Americans with rheumatic arthritis.[105,111] In England it was found that 1 out of 20 school children suffered from "rheumatism" (which is the name commonly applied to this disease on the Continent).[49] It is the commonest cause of organic heart disease in this country in individuals under the age of forty.

It is reported to be *less* prevalent in the southern than in the northern portions of the United States;[220,223,282] mitral stenosis is said to be twenty times as common in Boston as in New Orleans.[116] Rheumatic fever has been considered to be uncommon in the tropics, but various observers have recently reported large series of such cases in India and Australia.[69,145,157,273]

Bungeler believed that rheumatic fever was found just as frequently in the Tropics as anywhere and suggested that the incidence of this disease was not dependent on climatic factors. *He found a high incidence of rheumatic fever in tropical Brazil,* suggesting that the tropical climate had no therapeutic effect on the disease. He pointed out that variable climatic conditions may prevail in the same latitude as a result of differences in altitude and other factors. He exemplified this by the different climatic conditions in the state of Sao Paulo in Brazil, this state being partly tropical and partly subtropical. He studied the incidence of rheumatic heart disease in more than 600 autopsies there and found signs of rheumatic disease in 54 (Aschoff nodules or signs of rheumatic endocarditis). He believed that the elimination of focal infection was of primary importance in the treatment of rheumatic fever.[42]

Nichol found rheumatic heart disease to be only one-third as frequent in native-born children in southern Florida as in children born in the northern states but living in the South.[175] Paul and Dixon found that in the northwestern states, 4.5 per cent of Indian children had signs of rheumatic heart disease while in the southwestern states only 0.5 per cent were so afflicted.[179]

Although Coburn found that *stubborn rheumatic infection quickly became quiescent and remained so in 10 children who were transported to Puerto Rico,* Jones and his associates were less enthusiastic with their experience of transporting 26 rheumatic children from Boston to a subtropical climate.[129] Although some of their patients did well, others continued to have active infection, 3 died while in the South and 2 others showed increased activity of the infection.

Present evidence suggests that unless there is no hardship or financial sacrifice, it is better for rheumatic children to remain in their northern environment and to receive sulfonamide prophylaxis.[34]

Rheumatic fever is uncommon in Panama, southern China and the Malay States, but is common in northern United States, England, South Africa and Australia.[175]

Hyman analyzed the contributory rôle of heat, humidity and other environmental factors of the tropics on the development of cardiac disorders in the armed forces. This study is based on a large series of cases observed in the South Pacific areas. Over a third of the cases had neurocirculatory asthenia. The cardiac neurosis was only one manifestation of the general psychosomatic neurosis characterized by gastrointestinal, central nervous system and psychic abnormalities. He concluded that *exposure to heat and humidity of the tropical areas played an insignificant part in the production and precipitation of cardiovascular disturbances, with the possible exception of neurocirculatory asthenia.*[126]

In 90 per cent of cases of rheumatic fever, *the onset occurs before the age of fifteen,*[72] and frequently between the age of five and seven years;[11,15,73] however, cases may be found at any age. Rheumatic fever rarely occurs under three years of age. First attacks of the disease have been reported in infants less than a year of age and in adults over fifty or sixty years.[71,75,84] Rheumatic fever is slightly more common in girls than in boys.[48,92]

Its commonest seasonal occurrence in the United States is in the early spring,[238] and in England, in the late fall.[224] It is common in the cold and wet months of the year.[275] The disease is more common in cities than in the rural districts, and more prevalent among the poor than among the well to do.

There is a strong familial factor in rheumatic fever.[22,153,244] A history of rheumatic infection has been found in only 15 per cent of families of non-rheumatic children, but in almost 50 per cent of families of rheumatic children; others have found a family incidence of rheumatic fever varying from 18 to 70 per cent.[43,186] Some investigators have reported no significant difference in the frequency of rheumatic fever in families of varying economic levels (from poverty to middle class comfort[280]); in addition there is no good evidence that a rheumatic child or parent constitutes a hazard *per se* to non-rheumatic or quiescent members of the family.[276]

Morris and Titmuss studied the epidemiology of rheumatic fever *in England* and concluded that *poverty was the main predisposing factor.*[170] In their cases, environment influenced the death rate only if and when conditioned by poverty. There was a *consistent rise in the mortality from valvular heart disease with deepening poverty.* They found that the mortality was significantly higher in the poverty-stricken villages of South Wales than in any of the large towns. Their analysis showed also that overcrowding did not increase the mortality from heart disease, suggesting that acute rheumatic fever is not spread by droplet infection.

Hedley summarized his findings in a study of 4653 cases of rheumatic heart disease, rheumatic fever, chorea and subacute bacterial endocarditis admitted to Philadelphia hospitals over a period of fifteen years (1920 until 1935).[112] He found that rheumatic fever, Sydenham's chorea and rheumatic heart disease among hospital patients in Philadelphia tended to occur with greatest frequency in the sections of the city occupied to a large extent by the poor; this relationship was not invariable. A low rate of admissions and deaths was noted in every city ward inhabited for the most part by persons living under reasonably favorable economic circumstances. He obtained the impression that *these diseases occurred with greatest frequency in those sections of the city occupied by poverty-stricken persons.* He did not find that proximity to a watercourse was an important factor. His study also suggested that the more acute or fulminating forms of rheumatic infection occurred with relatively greater frequency among the extremely poor.

Rheumatic heart disease often occurred without clinically demonstrable rheumatic fever; not infrequently, the signs of heart disease developed prior to joint phenomena.

Wilson believes that susceptibility of the host determines the development of rheumatic fever. May Wilson and her associates[277] integrated genetic and epidemiologic methods of analysis in rheumatic fever. She had previously *concluded that hereditary factors were primarily responsible for the familial concentration of rheumatic fever* and postulated that the genetic susceptibility for rheumatic fever was transmitted as a single autosomal recessive gene.[281] Using age and genetic factors in combination, it was possible to estimate the number of case onsets expected at various times during the life experience of a group of 109 rheumatic families, including 456 rheumatic and non-rheumatic siblings. They found good agreement between the expected and observed incidence over a period of twenty years (from 1920 until 1939).

Some observers suggested that in rheumatic fever an active case constituted a risk for other siblings in the family.[90,206] Wilson and her colleagues showed that the risk for susceptible children to develop rheumatic fever was no greater before or after the onset of a case.

Wilson and Schweitzer found that 49 per cent of 112 rheumatic families had parenteral rheumatism while the parents and pedigree on both sides of the family were apparently negative in only 28 per cent of the families.[278] In 4 pairs of identical twins, all were alike in having rheumatic fever.[169] In 12 pairs of fraternal twins, 5 pairs had similar incidence and 7 pairs had dissimilar incidence. These workers believed that hereditary susceptibility seemed to determine the familial incidence of rheumatic fever but was not necessarily the sole condition essential for the development of

the disease. Read and her co-workers analyzed the family histories of 33 white children under fifteen years of age with rheumatic fever and a similar number of children admitted to the tuberculosis clinic as a control group. The parents of the rheumatic patients showed some rheumatic manifestations in 30.8 per cent in contrast to only 7.7 per cent of the parents in the control group.[188]

Other workers investigated the records of 8500 twins, among which rheumatic fever was reported in 72 sets.[133] The latter were studied very carefully. Among the 27 sets of uniovular twins, concordance of acute rheumatic fever was observed 5 times and discordance 22 times. In the 45 sets of binovular twins, concordance was observed only once. This much higher incidence of concordance of rheumatic fever in uniovular twins suggests that hereditary factors play a rôle in its etiology.

Etiology and Pathogenesis.—Paul has detailed the epidemiology of rheumatic fever and some of its public health aspects.[180] *The cause of rheumatic fever is unknown.*[247] The rôle of hemolytic streptococcal upper respiratory infection in initiating rheumatic fever and in inciting relapses is well known.[248] At present, evidence suggests that the hemolytic streptococcus is extremely important in initiating exacerbations and recurrences; other important etiologic factors include heredity, meteorology or climate and some as yet unknown factor. The symptoms of rheumatic fever may be allergic manifestations on the part of sensitized body tissues to invasion with hemolytic streptococci or their products. Schultz concluded that increased allergic irritability, as demonstrated by the development of secondary reaction following the intracutaneous injection of small amounts of rabbit serum, occurred more frequently in patients with rheumatic fever and nephritis than in controls.[215] The incidence of this increased allergic irritability was greater in individuals who had had recent focal infections.

In 2 milk epidemics in Denmark (1926 and 1935) during which beta-hemolytic streptococcus sore throat occurred in large numbers, rheumatic fever was not an uncommon complication. In one such epidemic, 2500 of the inhabitants were attacked by the severe sore throat and 30 developed rheumatic fever as a complication. The articular symptoms of rheumatic fever made their appearance in from one to five weeks after the onset of the sore throat, usually in a period in which the patients considered themselves well, although some were still tired, achy and noted general malaise. After this interval, they suddenly became ill with fever and joint pains and gradually developed typical symptoms of rheumatic fever.[160]

Rich and Gregory produced experimental serum sickness in rabbits and noted in some cases the development of cardiac lesions that, in their basic characteristics, resembled closely those of rheumatic carditis.[194] They postulated that *the lesions of rheumatic fever may be the results of focal reactions of the anaphylactic type.*

Various individuals have repeatedly suggested that *the lesions of rheumatic fever* may be due not to a direct action of bacterial toxins or invisible viruses, but *may represent the effects of a hypersensitive reaction to bacterial products.*[51,243] However, the Aschoff body and the valvular lesions do not resemble the usual inflammatory necrotic lesions of bacterial type hypersensitivity produced by bacterial proteins in the body sensitized by infection; this apparent specificity of the lesions has been repeatedly advanced as an argument against the rôle of hypersensitivity.

Rich and his associates showed that generalized vascular lesions identical with those of periarteritis nodosa could result from hypersensitivity reactions of the serum sickness (anaphylactic) type.[191–193]

Rich and Gregory found focal lesions in the myocardial connective tissue, the mural endocardium and the valves of animals subjected to systemic hypersensitive reactions of the serum sickness (anaphylactic) type; these lesions were strikingly similar to those characteristic of rheumatic carditis. Their nature and situation corresponded in fundamental ways to those of the peculiar tissue reaction characteristic of rheumatic carditis. The cardiac tissue in these animals was not peppered with typical Aschoff bodies as occurs so often in rheumatic fever. They emphasized that *these lesions in the experimental animals developed as a result of hypersensitivity of the anaphylactic type,* which differs in a number of important respects from hypersensitivity of the tuberculin type. The anaphylactic local reaction gives rise to a prompt and evanescent wheal and erythema as contrasted with the tuberculin type of hypersensitivity (from infection with bacteria, fungi and filterable viruses) which is manifest in skin tests by a delayed and prolonged inflammatory reaction to the injection of the specific protein of the microörganism. However, anaphylactic hypersensitivity to bacterial constituents may also develop during infection.

A fundamentally important manifestation of rheumatic fever pathologically is focal injury to connective tissue, characterized by edema with swelling and degeneration of the collagen fibrils. These alterations are precisely the primary and characteristic effects of the anaphylactic reaction upon the connective tissue.

Acute rheumatic fever has many features in common with the anaphylactic reaction of serum sickness. In both, there may be fever, joint manifestations, anaphylactic cutaneous lesions and arterial lesions of the periarteritis nodosa type. In addition, the recent studies of Rich and Gregory have shown that a type of carditis similar to that of rheumatic fever may occur in experimental serum sickness. It is interesting in this connection that in localities where the more fulminating forms of rheumatic fever are seen, anaphylactic types of skin lesions occur not infrequently during the rheumatic attacks (in more than 20 per cent in some series). Purpura, another manifestation of anaphylactic hypersensitivity, is also a recognized manifestation of acute rheumatic fever. Tissue eosinophilia appears more frequently in acute rheumatic carditis than is ordinarily appreciated. There have been reports of extensive infiltration of eosinophils in rheumatic hearts containing Aschoff bodies.[8,271] As an added link in this picture, Boots and Swift studied the character of the synovial exudate aspirated from affected joints and concluded that this exudate from joints in serum sickness is microscopically indistinguishable from that seen in joints of

patients with rheumatic fever.[33] Salicylate therapy usually definitely shortens the period of arthralgia in serum sickness.[194]

True rheumatic fever has not yet been reproduced in animals nor has it been observed in animals in the natural state.

The question of non-specific infection, bacterial allergy, or synergism between infection and vitamin C deficiency have been the subject of much controversy; some have attempted to show that this is a virus disease, but such studies have failed.[80,199,214]

Angevine and his associates made careful and extensive cultural studies on 22 patients with rheumatic fever; no organism of significance was found in the synovial fluid, pleural exudate, pharyngeal washings, heart valves and verrucous vegetations, pericardium or myocardium.[4] Swift[251] reported the cultivation of filterable pleuropneumonia-like microörganisms from arthritic exudate and from an erythema nodosum nodule in patients with rheumatic fever, but later found that these organisms probably originated from the mice inoculated with the exudates; he has been unable to grow these germs subsequently directly from exudates or tissues of patients with rheumatic fever.[246] Sabin has produced a progressive, migratory, proliferative polyarthritis in mice by intravenous or intraperitoneal injection of this organism.[211]

The *pathogenesis of rheumatic fever* includes three phases: (1) an *acute upper respiratory infection*, of several days duration; (2) a *symptomless period* lasting from a few days to five or six weeks (during which time it is decided whether or not a rheumatic attack will materialize); and (3) the *actual attack of rheumatic fever*.[115,118] The factors which seem to be essential for the development of rheumatic fever include infection with hemolytic streptococci (Lancefield Group A), a hereditary background of susceptibility, proper meterologic or climatic conditions, a disease pattern which is peculiar to rheumatic individuals, and an increase in antistreptolysins, indicating a marked immune response.

Symptoms.—*No one clinical sign or laboratory test is pathognomonic of this disease.*[117] The onset of an attack of rheumatic fever may be insidious or abrupt. It frequently follows a sore throat or an acute tonsillitis. Within a short time (averaging three weeks but varying from a few days to five or six weeks) following the latter, the patient develops *pain* or *stiffness* in one *joint* or another, a moderate degree of *fever*, excessive *perspiration*, and *tachycardia* out of proportion to the degree of pyrexia.

In the average patient with rheumatic fever, an acute infection of the upper respiratory tract is followed by a silent period without symptoms for about seven to twenty-one days after which the acute febrile migratory polyarthritis appears. This is initiated only occasionally by a chill; chills are found more often at the onset of the early polyarthritis of gonorrheal arthritis.[120] The joints may be red and swollen and pain and tenderness are usually out of proportion to the objective findings. Subcutaneous nodules around the joints appear in from 5 to 10 per cent of cases.

Laboratory findings include anemia, leukocytosis and an elevated sedimentation rate.

Common Symptoms of Rheumatic Fever

Frequently overlooked prodromes: marked pallor out of proportion to the anemia, ready fatigue, joint and *muscle pains*, "*growing pains*," failure to gain weight, abdominal pain and epistaxis.
Preceding *sore throat* or acute *tonsillitis*.
Migratory, symmetrical *polyarthritis* in adults; *in children, joint symptoms may be minimal or absent.*
Fever—101° to 104° F. (oral), irregular; often between 99° and 101° F.
Excessive perspiration during acute attacks.
Tachycardia out of proportion to the degree of fever.
Subcutaneous nodules (occasionally).
Cardiac involvement, to some extent, in practically every case.
Migratory, pulmonary hemorrhagic solidification and hematuria.

Loss of weight may be marked. Transient pulmonary symptoms may occur and renal involvement appears occasionally. Other symptoms may include pallor, vague muscle and joint pains, fatigue and epistaxis.

Patients with acute rheumatic fever do not usually come to an arthritis clinic. Most attacks of rheumatic fever begin in the early spring, the season of most frequent respiratory infections. In more than half of the cases, the attack is preceded by tonsillitis, sinusitis or an acute sore throat. Rheumatic fever is a disease mainly (but not solely) of temperate climates and is apparently associated in some way with hemolytic streptococcal infection.

The *larger joints are most often affected first*, and those of the lower extremity somewhat more frequently than the upper. The knees, ankles, shoulders and wrists are most often involved, but the hips, elbows and small joints of the hands and feet may be affected. *No joint has seemed to escape.* In the average case, several joints are affected, simultaneously or successively. *One of the striking features of rheumatic fever is the tendency of the inflammatory process to flit from joint to joint*, often subsiding and entirely disappearing in one joint as another is affected. Frequently, symmetrical large joints are involved simultaneously, and the same joint may be involved several times before the infection has been overcome.

The **affected joint** is hot, swollen, red and tender; motion produces marked pain. Joint pains are often more marked at night. *The joint involvement may occasionally be so persistent and marked that it simulates acute rheumatoid arthritis.*[245] After a period of from two to ten days of treatment, the inflammatory changes disappear from an involved joint and the appearance of that joint becomes normal, full motion being achieved without discomfort.

In addition to the joint phenomena, other major manifestations of the disease may include sore throat, upper respiratory tract infection, tonsillitis, fever, tachycardia, subcutaneous nodules, cardiac involvement, skin eruptions, abdominal pains and epistaxis.

The **fever** is often high in those patients with severe joint manifestations, ranging from 100° to 104° F. It is irregular and atypical and persists for a variable period of time; when an antipyretic is used, the temperature

usually falls slowly by lysis with subsidence of the joint phenomena. In untreated cases, the fever persists for about thirty days. Together with the fever there occurs **copious sweating.** This may drench the patient so that the bed clothes become soaked with perspiration. Rheumatic fever is *one of the so-called sweating diseases* along with undulant fever, malaria and septicemia. The **pulse** is markedly elevated and remains high even after the temperature falls to normal. This tachycardia may persist to some extent for weeks or months.

Rheumatic fever may simulate rheumatoid arthritis and only the passage of time and subsequent developments permit a correct diagnosis in some cases.[17] Rarely, it may mimic polyarticular gout or early acute specific arthritis.

Symptoms in Children.—*In children, the onset of the disease is often much more insidious than in adults,* and is frequently associated with upper respiratory tract infections.[130] The younger the child, the less apt are the joints to be involved. In a large series, the sole initial manifestations of rheumatic fever were growing and joint pains, chorea and polyarthritis —each in about 25 per cent of cases. Active carditis was the sole manifestation in only 3 per cent.[275] In 15 per cent, active carditis was present at the onset.

Symptoms in children may include fatigue, malaise, growing pains, intermittent fever, tachycardia, pallor, anemia, night sweats, epistaxis, chorea, subcutaneous nodules and cardiac murmurs. *Epistaxis* is frequently observed without trauma. It is usually controlled by cauterization; occasionally repeated epistaxis necessitates transfusion. **Joint pains and swelling may be entirely absent in children with rheumatic fever.** Rheumatic joint pains are more common in damp and rainy weather. In children the pains are often relieved by lying in a warm bed, and may recur if the patient becomes cold. **In some children the only evidence of the disease may be vague muscular pains, abdominal discomfort, fever, epistaxis and a general feeling of sickness.**[86]

In childhood the onset of rheumatic fever may be very difficult to recognize in the presence of respiratory infections. The affected joints may be somewhat painful, but there is little swelling. Children often show no redness, swelling or heat in the joints. Stiffness is more marked than tenderness; joints are only rarely hot or red.

Wilson found joint pains in 67 per cent of rheumatic children; in 26 per cent of cases, the joint pains were the first manifestation of the rheumatic process. The most frequently involved joints in children were the ankles, knees and wrists; the chief signs were pain and stiffness.

Fatigability and failure to gain weight in rheumatic children may be the only evidence of a carditis.

In children and young adults, rheumatic fever is often polycyclic with severe inflammatory reactions taking place in vascular tissues during each febrile phase of the cycle. Such reactions continue to occur as long as there is antigen liberated from the respiratory pathogen—the hemolytic streptococcus Group A; this induces an abnormal antibody response in the rheumatic patient.[53]

The excellent monograph of Dr. May G. Wilson on "rheumatic fever" during the first three decades is recommended to anyone interested in this disease in childhood and young adults.

In one series of more than 400 cases of rheumatic fever in children, early symptoms included: arthralgia, 97 per cent; arthritis, 28 per cent; sore throat, 23 per cent; rash (usually morbilliform), 14 per cent; vomiting, 9 per cent; abdominal pain, 8 per cent; cough or cold, 8 per cent; stiff neck, 3 per cent; pyelitis, 2 per cent; choreiform motions, 1.5 per cent; nodules, 1.5 per cent.[159] This low incidence of rheumatic nodules (as seen in Australia) is unusual. In another series of children with rheumatic fever, the first symptoms were—chorea (44 per cent), arthritis (37 per cent) and primary carditis (19 per cent).[265]

In typical cases of rheumatic fever the diagnosis is relatively simple. In children, the so-called **"growing pains"** may be a manifestation of this disease. These vary greatly but may consist of *vague, dragging, fleeting pains in muscles and occasionally in joints,* frequently accompanied by easy fatigability, loss of weight and anorexia.[5]

Most often, "*growing pains*" are noted in the *hamstring tendons* behind the knee; there may be swelling, tenseness and tenderness of these tendons. *The pains may be fleeting and mild, or may persist for days and be severe enough to produce a limp.* Wilson has emphasized that growing and joint pains in childhood may be seen during infections (especially respiratory) or after excessive exercise or in association with foot strain; she has cautioned against interpreting all such pains as minor manifestations of rheumatic fever.[275]

Recent studies have questioned the rheumatic nature of growing pains (vague muscular pains and aches, especially in the legs). Hawksley concluded that growing pains did not provoke rheumatic carditis and that they were not related to rheumatic fever.[104] Cardiac "rheumatism" developed in less than 1 per cent of a large series of these children. In a follow-up study of 200 patients who complained only of leg pains, Shapiro found that none of these children developed rheumatic heart disease. He concluded that such leg pains, coming on characteristically at the end of the day and often awakening the patients during the night, were due to normal growth and not to rheumatic fever. Others believe that "growing pains" are merely examples of foot strain in most instances.

Many acute and chronic diseases must be differentiated from rheumatic fever in children; these all may produce joint involvement (including leukemia, syphilis, tuberculosis, undulant fever, osteomyelitis and rheumatoid arthritis—Still's disease[275]).

CARDIAC INVOLVEMENT

The chief manifestation of rheumatic fever is carditis; the latter causes 40,000 deaths annually in this country. *About one-third of patients with rheumatic fever develop active carditis;* in the others, cardiac involvement remains subclinical. It may be difficult in some cases to ascertain whether

the heart is affected (*i. e.*, to an extent noticeable to the clinician), and whether transitory or permanent damage will result. At times, this cannot be determined definitely for some months or years.[28] During the acute stage of the disease, signs of cardiac involvement include subjective symptoms (dyspnea, edema), the development of murmurs or other adventitious cardiac sounds, disturbances in conduction of the heart (including electrocardiographic evidence) and changes in the size of the heart.

Marked dyspnea, peripheral edema and *signs of decompensation* (including an enlarged liver) *are uncommon during the acute stage of the disease;* their presence would indicate cardiac damage. A prolonged elevation of temperature with a disproportionately high pulse rate is the rule and does not necessarily denote damage to the heart. Soft systolic murmurs may be heard over the heart in many diseases accompanied by fever and tachycardia; however, if such a systolic murmur becomes loud during the course of the disease, it is more likely to be permanent and to indicate actual cardiac damage. Too much stress has been placed upon cardiac murmurs and too little upon heart tones.[135]

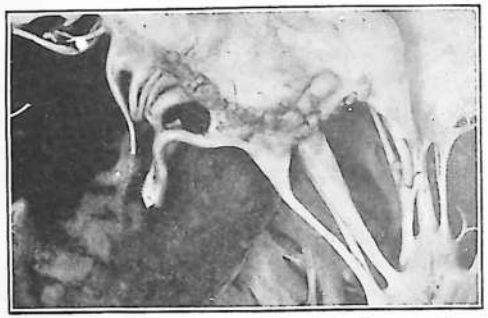

FIG. 178.—Rheumatic endocarditis. A bead-like row of vegetations runs along the line of contact of a cusp of the mitral valve (Boyd, Textbook of Pathology.)

Systolic murmurs do not always indicate cardiac damage. Soft systolic murmurs may be associated with fever, anemia, hypertension, nervous excitement, tachycardia and hyperthyroidism, without any demonstrable evidence of organic heart disease. *A systolic murmur may appear in normal individuals following brisk exercise. The occurrence of a diastolic murmur practically always indicates organic cardiac damage.* The mid-diastolic blow which appears early during active carditis (when the mitral ring is dilated rather than stenosed), tends to disappear as the active infection subsides.

In an occasional patient, *electrocardiographic* changes may be the only evidence that the illness is rheumatic. A slight degree of delayed or disturbed conduction (which may not be evident clinically) can be detected by the electrocardiogram. Further delays of conduction may lead to actual dropping of ventricular beats.

Another factor of importance during rheumatic fever is the *size of the heart.* If the heart increases in size during an attack, it is probably damaged; however, the converse is by no means true. *A normal sized heart may be the seat of marked disease.*

A pericardial friction rub indicates cardiac involvement. If the pericardium is involved, the myocardium is damaged in most instances.

Following subsidence of an attack of rheumatic fever, *the amount of cardiac damage may vary in different patients as follows:*

Some individuals may continue throughout life without developing signs of rheumatic heart disease (unless a recurrence of rheumatic fever occurs).

The patient may develop a slight valvular lesion which is carried throughout life to a normal age expectancy (with proper care and the good fortune to escape a recurrence of the acute rheumatic attack).

Permanent heart damage results in at least one-third of these patients.[153] The more frequent the recurrence of rheumatic fever, the greater is the likelihood of cardiac involvement. *The younger the patient, the more likely is the possibility of cardiac damage;* this is apt to be more severe in young individuals.

In rare instances (1 to 4 per cent of cases), *death may occur during an acute attack* of the disease from diffuse cardiac involvement (including auricular fibrillation and emboli).

Subacute bacterial endocarditis occurs in about 4 per cent of the cases; this may develop at any time but especially past ten years of age. It may be seen at times in cases with little cardiac involvement.

Rheumatic fever tends to recur. A patient who may have escaped valvular damage during his first attack may succumb to it in his second or third. Wilson believes that about 15 per cent of children escape some recurrent manifestation of activity of their rheumatic fever. She has also found a diminished tendency for recurrences starting at about puberty. The most frequent form of recurrence is usually the same as the initial manifestation of the disease.

The mitral and aortic valves are attacked most frequently. These may be involved separately or together. *Mitral insufficiency, alone, or in combination with aortic insufficiency may occur as early as forty-eight hours after the onset of an acute rheumatic attack.*[25] However, it requires months, and usually years, for sufficient scar tissue to be produced to result in valvular stenosis. This is important, inasmuch as stenosis of the valve may appear clinically several years following an attack of rheumatic fever. This increases the prognostic difficulties.

If one were asked for statistics, it might be stated that:[153]

Approximately one-third of the patients with rheumatic heart disease will suffer no consequences clinically of their cardiac damage.

Another third will die within the first twenty-five years of life.

The remainder will die within the age limits of twenty-five and fifty years.

The average age at death of patients with rheumatic carditis is thirty years.

In 325 rheumatic children, involvement of the heart was found in 85 per cent within a year of the onset of the rheumatic fever.[275]

In children with rheumatic myocarditis, symptoms include fever (100° to 104° F.), anemia, loss of weight, leukocytosis, rapid sedimentation rate and an engorged tender liver. Cyanosis and edema are not common in the first attack in children. This exudative stage persists from one to four weeks, and is followed by a subacute proliferative stage (which may persist for months).

In patients with active carditis, a marked decrease (30 or more per cent) of vital capacity may be seen. With progressive cardiac involvement, one notes poor quality of the cardiac sounds, widely transmitted murmurs and possibly gallop rhythm. In children, as the heart dilates with cardiac failure, the diastolic murmur of mitral stenosis often disappears.

Fulminating cases of myocardial involvement are unusual but may result in cardiac dilatation, within one or two days, producing marked cyanosis, edema, orthopnea and a markedly engorged liver.

SUBCUTANEOUS NODULES

These are usually seen in the subacute stage of rheumatic fever.[85] They may vary in size from a pin-head to an almond and are usually about the size of a split pea. Their *most common location* is about the elbows, fingers, knees, palms, neck, wrists, ankles, carpometacarpal joints, toes, occiput or iliac crests. They vary from 2 mm. or less to more than a centimeter in width, and are usually discrete, but occasionally become conglomerate.[275] Inasmuch as similar nodules may occasionally occur in rheumatoid arthritis, their presence is not pathognomonic of rheumatic fever.

They are usually firm and painless and are not, as a rule, attached to the skin. They may rapidly increase in size and persist for several days, weeks or months. These nodules are noted more frequently in England than in this country.

The frequency of such nodules is variable. They have been reported in from 2 to 37 per cent of these patients.[9,10] *The presence of nodules is considered to be indicative of a serious infection* as they are often associated with severe and progressive cardiac damage.

Bauer and his associates have carefully studied large numbers of these nodules.[24] They attributed the pathologic changes in the nodules of rheumatic fever chiefly to injury of small blood-vessels, followed by an exudation of plasma and blood cells into the connective tissue; small focal lesions similar to myocardial Aschoff nodules were also seen. Because of the predominantly exudative process in rheumatic fever, these nodules appear and resolve more quickly than those in rheumatoid arthritis. (The findings of these workers are discussed in detail in the section on Symptoms of Rheumatoid Arthritis.)

Other Cutaneous Lesions.—Over 10 per cent of patients with acute rheumatic fever manifest cutaneous lesions; these are often overlooked.[275] They include *erythema nodosum, erythema multiforme, small purpuric,*

papular or urticarial lesions and erythema annulare rheumaticum. This latter is a transitory rash which appears following the endocarditis; it is characterized by pale red or bluish-red rings, semicircles or polygons rom 1 to 3 cm. in diameter. These are not raised but are macular, and do not result in scaling or pigmentation; there is no associated itching, swelling or hemorrhage. The lesions are noted[3] most often on the chest, abdomen, back and thorax, and are uncommon on the extremities; they have not been reported on the face or in mucosæ. Erythema annulare rheumaticum may be an initial symptom of rapidly fatal rheumatic fever.[2]

Rheumatic erythemas appear and disappear irregularly with wide fluctuations in intensity. Keil has presented a thorough review on rheumatic erythema.[136] The increased permeability of the capillary walls may be the pathologic basis for the skin lesions in rheumatic subjects. During a recent epidemic of rheumatic fever, crops of bluish, non-tender, maculopapular purpuric spots were noted as an outstanding symptom; these were most frequent upon the arms and legs, and persisted from one to six months, disappearing without desquamation or scar formation.[47]

A rheumatic nature has not been proved in the following controversial skin lesions: erythema nodosum, Osler node, psoriasis, scleroderma, dermatomyositis, systemic lupus erythematosus and erythema diutinum.[135]

The pathogenesis of erythema nodosum is open to question. Some workers have found a causal relationship between Streptococcus hemolyticus infections and erythema nodosum, as well as between this skin lesion and rheumatic fever.[233] Others do not believe that erythema nodosum is of rheumatic origin, even when preceded by sore throat and joint manifestations. Keil found no good evidence that erythema nodosum was rheumatic.[134] He observed that many individuals labeled these cases as tuberculous when the tuberculin test was positive and as rheumatic when the test was negative.

In a small school epidemic of erythema nodosum involving 14 cases, all of the boys were found to be highly sensitive to tuberculin and 12 of these showed definite or suggestive lesions of tuberculosis.[37] Eight of the cases occurred in one cottage. A boy with open pulmonary tuberculosis had been removed from the school several weeks after the onset of the epidemic.

Wallgren[266,267] and von Henkelom[263] believe that erythema nodosum is a non-specific allergic skin eruption which appears especially in tuberculosis but occasionally in other infections (such as rheumatic fever).

INVOLVEMENT OF THE LUNGS AND PLEURA

Rheumatic fever occasionally causes pleurisy and pneumonia. *Pulmonary complications* include bronchopneumonia, massive collapse of the lungs, pleural effusion and acute pulmonary edema.[139,184] It has been suggested that rheumatic pleurisy is more common than is generally believed;[235] interlobar pleural thickening was demonstrated roentgenologically in almost 50 per cent of children with other manifestations of rheumatic fever (as contrasted with only 9 per cent in normal children). Others have found

pleurisy to occur in from 2 to 20 per cent of patients with rheumatic fever; the hemorrhagic nature of this fluid and its ready formation have been described.

In patients with "*rheumatic pneumonia,*" physical signs may persist for only a few days as the consolidation is rather transient. Some individuals have found that the pulmonary lesions are identical pathologically with those of rheumatic fever in other areas (including focal perivascular lesions, Aschoff type lesions, vascular injury, etc.).[96,172]

Coburn has described striking pulmonary changes in a series of 35 rheumatic patients; these have been in the nature of definite but *rapidly shifting pulmonary consolidations* which he believes are a part of the rheumatic disease, because in every instance they occurred almost simultaneously with recognized rheumatic phenomena.[51] Because the consolidation is transient, it may be missed on physical examination. In his series, the pulmonary signs were rather typical, with fine râles heard at the onset, and later, bronchial breathing, dulness and egophony. This began as often in the upper as in the lower lobes and the extent of the involvement varied from "loose hemorrhagic consolidation in a part of one lung to complete solidification of the five lobes." Two of the characteristic findings were *their migratory nature and the rapid disappearance of the lesions within a few days.* Purulent sputum was not noted; in some instances the sputum was frothy and blood tinged while in others there was a hacking, non-productive cough. Roentgenologically the appearance was either that of pulmonary edema or broncho- or lobar pneumonia.

On the other hand, the possibility of a rheumatic pneumonia has been questioned. Mallory states that he has no clear conception as to whether there is such an entity and that he "cannot make head or tail" of the descriptions.[161] He noted pulmonary lesions which were midway between pneumonia and infarcts in their appearance.

PERICARDITIS

Pericarditis has been reported to be much less common in rheumatic fever than myocarditis or endocarditis, while some have said that the pericardium was almost always involved when the disease attacked the heart.[97] Acute pericarditis has been detected clinically in from 12 to 19 per cent of these patients.[203] *Rheumatic fever does not often cause extremely large pericardial effusions and paracentesis of these is practically never indicated.* Large doses of salicylates will usually lead to prompt relief and resorption of these effusions.[29]

A pericardial effusion in rheumatic fever may produce a chill with high fever, dyspnea, precordial pain, cough and a rapid elevation in blood pressure;[275] the liver may become enlarged rapidly and severe abdominal pain may be present. The temperature ranges from 102° to 104° F.; there is usually a leukocytosis and a rapid sedimentation rate.

Pericardial involvement is often overlooked during life. The physical signs of pericardial effusion will be present, including an enlarged area of cardiac dulness at the base of the heart and to the right of the sternum, distant heart sounds and a leathery to and fro friction rub. Roentgen examination will usually reveal the pericardial effusion, appearing as a suspended paper bag, filled with water;[275] the pulsations of the heart are greatly decreased as seen fluoroscopically.

Excluding tuberculous pericarditis, heart failure due to an adherent pericardium is usually the result of rheumatic infection.[50]

ABDOMINAL SYMPTOMS

Abdominal symptoms in rheumatic fever may be marked and may simulate acute appendicitis.[100,284] The literature on this subject has been reviewed recently.[283] Mild abdominal pain is so common in rheumatic children that it has been considered to be a typical rheumatic phenomenon and a sign of rheumatic activity.[181,236] Recurring acute or chronic attacks of abdominal cramps may occur. These are usually in the epigastrium (but occasionally generalized or peri-umbilical) and do not radiate; they may come on in the left upper quadrant only while walking and be relieved by rest.[77] Nausea and vomiting may be noted. There may or may not be abdominal tenderness but there is usually no rigidity. The episode often runs its course before the next meal. The temperature may rise to 101° F. or above. These abdominal cramps may be the only subjective manifestation of rheumatic fever, or there may be a history of muscular or joint pains or slight epistaxis. Abdominal pain is also common in children in non-rheumatic infections (such as upper respiratory infection).

Rapid marked enlargement of the liver is often noted in children with rheumatic carditis. usually associated with congestive failure of the kidneys.

Clinical Picture of Rheumatic Peritonitis[284]

A young patient, with *previous rheumatic history* (as a rule).
Signs of rheumatic valvular damage often found in cardiac examination.
Abdominal pain and signs of peritoneal irritation appearing during or just prior to the onset of an attack of rheumatic fever.
Severe general reaction, *high fever and leukocytosis* (often above 20,000).
Diffuse abdominal phenomena with less tendency for localization in the right lower quadrant than in appendicitis.
Most common findings at operation or necropsy: clear fluid in the abdomen, hyperemia, edema and infiltration of the peritoneal and subperitoneal tissues without evident intra-abdominal cause.

These attacks may recur for several years, but each succeeding attack generally becomes milder. In the chronic condition, only mild fleeting abdominal pain may be noted. *If there is any serious question of the possibility of appendicitis, exploratory operation should be performed.*

CHOREA

Sydenham's chorea is a nervous manifestation whose morbid anatomy is very imperfectly understood and whose exact relationship to other rheumatic conditions is still disputed by some.[112] *Osler found acute endo-*

carditis with great frequency in chorea and stated that there was no known disease in which endocarditis was found so constantly postmortem as in chorea.[177] Various authors have reported from 3 to 77 per cent of cases of Sydenham's chorea to manifest signs of rheumatic heart disease. Hedley has summarized these.[112] In recent years, some writers[91,128,261] have questioned whether chorea without clinical or laboratory evidence of infection should be regarded as part of the rheumatic syndrome, or at least more than a rather mild rheumatic manifestation accompanied very infrequently by cardiac damage. Hedley concluded that heart disease was less likely to develop in Sydenham's chorea unattended by other signs of rheumatoid infection. He found that 20 *per cent of children with rheumatic heart disease have histories of chorea.*

In a large series of cases studied by Hedley, approximately *90 per cent of the admissions for chorea were confined to the five to fourteen year age group.* Only 2.5 per cent were among persons under five years and 7.5 per cent occurred among persons older than fifteen years. Most authors are in general agreement as to the age incidence of chorea; it is also generally agreed that *chorea is infrequent under the age of five.*

A child with almost any form of Sydenham's chorea stands a much greater chance of developing rheumatic heart disease than a child who has never had any form of Sydenham's chorea. From 10 to 15 per cent of cases of rheumatic heart disease gives histories of chorea, many with frank attacks of rheumatic fever.

Rheumatic fever, Sydenham's chorea and rheumatic heart disease are relatively uncommon under five years of age. *Very few cases of rheumatic fever occur under the age of two.* The peak of onset of rheumatic fever, Sydenham's chorea and rheumatic heart disease occurs in the five to nine year age period.

In Great Britain, most writers have indicated that rheumatic fever and chorea occurred with greatest frequency in the fall and during December and least often in the spring and early summer. Most American writers suggest that these conditions are most common in this country during the late winter and spring months and least common during the summer and fall. In Philadelphia, the greatest number of admissions involving rheumatic fever was in April, the fewest in October.[112]

Wilson found that chorea was the initial manifestation of the rheumatic process in about 25 per cent of 699 rheumatic children. The earliest age of onset of chorea was twenty months and the oldest patient with recurrences of chorea was eighteen years. *Recurrences of chorea over the age of twenty are very uncommon.*

Prodromal symptoms of chorea include personality changes, frequent emotional upsets, enuresis, restless sleep, etc. A very obedient child may become irritable, inattentive, clumsy and sulky, or may act silly. These individuals may then show the incoördinate, irregular, objectively purposeless movements which may become severe enough to interfere with walking, talking and swallowing. They are usually relieved by sleep, rest, hot baths, sedatives and aggravated by emotional upsets.

It is often difficult to distinguish between true choreiform movements and habit spasm. The former may also be seen in non-rheumatic infections, as poliomyelitis, birth injuries, encephalitis, tic and psychogenic disturbances.

Chorea has been noted as a symptom of rheumatic fever in from 5 to 34 per cent of cases.[272] The commonest cause of chorea in childhood is a rheumatic infection. *Chorea is nearly twice as common in females as in males.*[111] Sydenham's chorea is relatively uncommon among negroes. Children with rheumatic fever are no more nervous than those with other diseases. Some workers[54] feel that chorea and rheumatic fever are independent while others maintain that chorea is merely a symptom of rheumatic fever.[128,178,209]

In uncomplicated cases of chorea (*i. e.*, without other evidence of rheumatic activity) *the sedimentation rate has been reported by some to be normal*[117] *while Bauer found a rapid rate in 100 per cent of 70 choreic patients upon admission.*[119]

Jones and Bland found that only 3 per cent of 134 patients experiencing chorea alone had heart disease, but Wilson found no significant difference in the degree of cardiac involvement or mortality whether chorea, polyarthritis, or growing or joint pains were characteristic in the course. *Children with chorea may be treated as potential rheumatic subjects.* They should be removed from school and placed at *rest in bed* for several weeks with mild *sedatives* and salicylates. Chorea appears to be a self-limited disease and is rarely fatal.[275]

The recent trend in therapy is toward *artificial fever*, although this does not influence the subsequent occurrence of carditis. Various workers have reported good results with this method.[16,138,232] These physicians produced artificial fevers of from 104° to 106° for an average of three to four hours at each session. Others obtained successful results from fever produced by short wave, intravenous injections of typhoid vaccine[210] or hot tub baths[156] (giving an average of 12 daily baths, raising the temperature of the water from 85° to 110° F. during the course of two to three hours). Some physicians prefer to use nirvanol.[99]

NEPHRITIS

Acute nephritis has been reported in from 10 to 20 per cent of cases of rheumatic fever.[98] Diffuse glomerulonephritis has been found in 22 per cent of cases of rheumatic endocarditis by one individual,[21] while others stress its infrequency (1.3 per cent). Uremia has occurred in rheumatic fever, with recovery following appropriate therapy.[222]

There have been numerous observations of the *infrequency of clinical evidence of renal involvement during the active stage of rheumatic fever;* this varied from 0.67 to 7 per cent. Bell found rheumatic disease and nephritis frequently in autopsy material (with renal involvement in 25 per cent of 104 cases at necropsy which showed rheumatic valvulitis with active lesions).[21] In one institution, although 1622 patients entered the hospital

with the admitting diagnosis of "acute rheumatic heart disease," the clinical diagnosis of rheumatic nephritis was not made.[125] Three thousand necropsies were performed at this institution, among which 153 presented rheumatic cardiac disease as the principal cause of death. In 2 cases, renal involvement was probably the most significant cause of death.

Numerous observations have been made at autopsy of the frequency of glomerular and vascular changes in patients who have had rheumatic fever. Klotz emphasized the frequency of visceral involvement.[142] He described a nonsuppurative perivascular infiltration around the interlobular arteries with healing by fibrosis, with the final production of a granular contracted kidney. Fahr stated that rheumatic fever may be an etiologic factor in some cases of malignant nephrosclerosis.[82] Others also described renal lesions in patients with rheumatic heart disease, with the nephritis preceding, occurring simultaneously with or following the onset of rheumatic manifestations.[141,208,212,262] The glomerular lesions were usually of the focal embolic type or acute diffuse glomerulonephritis. Longcope has denied the specificity of the renal lesions and stated that they may be caused by inflammatory factors of diverse etiology.[155] It is possible that they may be the result of anaphylactic hypersensitivity.[194]

INFANTILE LEUKEMIA RESEMBLING RHEUMATIC FEVER

Joint symptoms may be prominent in *infantile leukemia.* The possibility of *leukemia must be borne in mind in all cases of joint (and bone) pains in young children,* and a careful examination of the blood smear must be made for immature cells. *Diagnosis of the leukemia may be difficult early in childhood* because of the physiologic lymphocytosis, the palpable spleen in some normal young children, and the tendency of children to respond to an infection with a lymphatic reaction.[88]

Joint pains have been noted so frequently in infantile leukemia as to cause confusion with the diagnosis of rheumatic fever.[66,230] If a polyarthritis is found in a child under three years of age, the absence of signs of acute carditis is good presumptive evidence that this lesion is not rheumatic.[239,240] *Fever and joint pains may occasionally disappear even in acute leukemia following administration of salicylates.*[231]

Smith has explained the bone and joint pains in infantile leukemia on an anatomic basis due to the invasion and multiplication of leukoblastic cells within the bone marrow. During the first several years of life, actively functioning red marrow fills all of the long bones. The conversion of the active red marrow to non-functioning fatty marrow begins at about seven years of age and is complete at about twenty-one years (except for areas of red marrow in the upper ends of the proximal long bones). The bones become rarefied and leukemic cells spread beyond the bony cortex and may elevate the periosteum. Increased pressure by the rapidly multiplying leukoblastic cells within the bones of these young children may produce pain.

LABORATORY FINDINGS

1. *Rapid sedimentation rate.*
2. Leukocytosis with shift to the left in the differential white blood count in the acute stage of the disease.
3. Electrocardiographic signs of myocardial involvement in some cases.
4. Presence of antifibrinolysins, antistreptolysins and precipitins.
5. Streptococcus agglutinins found by some.
6. Blood cultures usually negative.
7. Skin tests to hemolytic streptococci—usually positive.
8. Formol-gel reaction positive in most cases in the presence of active carditis.

In a comparison of several tests upon patients with rheumatic fever, the corrected sedimentation rate was found to be the most sensitive, the formol-gel test the least sensitive and the Weltmann reaction intermediate in sensitivity.[140]

An increase in the rapidity of the sedimentation rate is the best index of persisting infection. In general, the patient should be kept in bed until the sedimentation rate has returned to normal or nearly normal.

Some workers have found electrocardiographic abnormalities in every case of rheumatic fever when daily tracings were made.[164] Others have stated that no information was added which was not obtained by physical examination, although confirmatory evidence was furnished.[36] Some of the electrocardiographic changes in rheumatic carditis which may occasionally be of assistance in differential diagnosis include: an increased height and breadth of the *P* wave, notching, increase in the *P-R* interval, inversion of the *T* wave in 2 or more leads, notching of the *QRS* complex and right axis deviation.

Antifibrinolysins.—Hemolytic streptococci will liquefy normal human fibrin clot, but the plasma clots from the blood of patients convalescent from acute hemolytic streptococcal infections resist this fibrinolytic action;[166,258,259] the reaction, however, is not specific.[264] *The presence of antifibrinolysins indicates a recent hemolytic streptococcal infection* and is shown by the fact that the fibrin in blood serum shows resistance to lysis when exposed to cultures of hemolytic streptococci, *i. e.*, its clot is not liquefied by these organisms. It is of academic interest only that such antifibrinolysins are present in the blood stream of patients with rheumatic fever, but are absent in those with rheumatoid arthritis.[101,173]

Boisvert found the fibrolysin test positive 68 times in 76 children with rheumatic fever.[30] The test usually becomes negative with the onset of convalescence. The fibrolysin test may also be positive in streptococcic cervical adenitis and in hemorrhagic nephritis. The behavior of this reaction is added evidence of the etiologic significance of hemolytic streptococci in rheumatic fever.

Antistreptolysins.—Synonyms for this include antihemolysin and antistreptohemolysin. A streptolysin, which is a soluble product of hemolytic streptococci, produces hemolysis of red blood cells. It is an antigen, and

as such leads to the formation of an antibody known as antistreptohemolysin or antistreptolysin, which latter may neutralize the streptolysin. The titer of antistreptolysin in a given serum may be determined by titrating with streptolysin. *Antistreptolysins are present only following hemolytic streptococcal infections.* Patients with active rheumatic fever usually show a high antistreptolysin titer. Again, this is of academic interest only, as the test is not simple enough to be utilized generally.

Precipitins.—In acute rheumatic fever, the patient develops precipitins to protein fractions of hemolytic streptococci. Hemolytic streptococci have been found to contain a nucleoprotein (P)[59,147–150] which is not specific as it gives cross-precipitation with other Gram-positive cocci, a group specific carbohydrate (C) and, in addition type specific protein and carbohydrate fractions (M and S).

Streptococcus Agglutinins.—Positive agglutination tests with hemolytic streptococci are reported by some to be uncommon in patients with rheumatic fever,[167] while others find agglutinins to hemolytic streptococci in a titer of 1 to 100 or more in a majority of these patients.[27,95]

Coburn and Pauli have reported the demonstration of secondary antigens and antibodies in rheumatic fever.[61] If substantiated, this is clinical confirmation of the theory of secondary anaphylaxis and immunity and may pave the way for further diagnostic and therapeutic methods in various infectious diseases.

Blood Cultures.—A survey of the literature tends to show that positive blood cultures are found infrequently in rheumatic fever, and in no greater percentage than in other febrile diseases which are not characteristically accompanied by bacteremia.

Skin Tests.—Skin reactions to hemolytic streptococci are positive in the majority of patients with rheumatic fever, but in severely progressive cases, skin tests are usually negative (as usually occurs with tuberculin tests in miliary tuberculosis).

The **formol-gel reaction** may be of assistance in the diagnosis of active carditis in patients with rheumatic fever.[89] Experimental observations indicate that a strongly positive reaction in children or in adults, in the absence of arthritis, suggests an active carditis.[216,218] If the reactions are repeatedly negative (even though the sedimentation rate is accelerated), a severe carditis is probably not present.

Blood Phosphorus.—The phosphorus content of whole blood of children with rheumatic fever was found to be about 15 per cent less than patients convalescing from other acute and chronic illnesses. There is normally a high requirement of phosphorus for optimal growth. Phosphorus also plays a rôle in muscle metabolism.[81]

Cholesterol.—Offenkrantz and Poindexter noted hypocholesterolemia in acute rheumatic fever and rheumatic carditis in some cases.[176,183]

Capillary Resistance.—Brown and Wasson tested 150 children with rheumatic fever and found that their capillary resistance averaged 6.6 cm. lower than in normal children.[39] The highest capillary readings were obtained in July and August, with a drop beginning in October, and continuing through February. A correlation was noted between capillary resistance and the sedimentation rate, suggesting that possibly the capillary damage and lowered resistance may be produced by streptococcus toxins.

E.C.G.—Reyersbach and Kuttner concluded that prolongation of the *P-R* interval was not in itself a reliable index of myocardial involvement inasmuch as they found an abnormal prolongation of the auriculoventricular conduction time in 5 per cent of 150 normal girls whose electrocardiograms were studied.[190]

Heffer noted abnormalities in 39 per cent of electrocardiograms made on 181 patients in whom Sydenham's chorea was the first sign of rheumatic fever.[114] These changes occurred in the absence of clinical or other laboratory findings of cardiac involvement.

Treatment of Rheumatic Fever

Rest in bed until signs of activity have disappeared, even after salicylates have been discontinued; this may require weeks, months or years.[254]

Salicylates or aspirin in doses sufficient (if possible) to relieve painful joints and to attempt to lower temperature; in children, even massive doses may not appreciably reduce the fever.

Coburn has advised the use of large amounts of salicylates daily to quickly modify the activity of the disease. His work suggests that in some instances they may prevent the development of carditis in rheumatic fever. If this work is confirmed this will be an extremely important addition to therapy.

Local therapy to joints: splinting; local analgesic applications.

Removal of focal infection (especially tonsils) after the acute attack has subsided; there is no proof, however, that this lessens the severity of the cardiac lesion.

Repeated small *transfusions* if severe anemia occurs; large amounts of iron if only moderately anemic.

Adequate intake of calories and of all *vitamins*, but particularly of vitamin C (including 200 mg. of cevitamic acid orally each day in adults and half of this amount in children).

Residence in a tropical climate if possible and use of sulfanilamide in children (daily) for at least several years to prevent recurrences of hemolytic streptococcal respiratory infections and rheumatic fever.

TREATMENT

Rest in Bed.—Most cardiologists agree that the patient with rheumatic fever should remain at rest in bed until he is clinically definitely improved and until activity of his infection has subsided, *i. e.*, until practically normal results have been obtained for the sedimentation rate, white blood count, differential picture and temperature. Unfortunately, this may, in some cases, be economically impossible and may result in the loss of several years of schooling in children. Whenever it is possible, however, the individual should be kept in bed until all joint symptoms and precordial distress have disappeared and until the above laboratory standards have been satisfied.

Martin has outlined *convalescent care* in rheumatic fever.[162] Inasmuch as the first five years following the initial rheumatic infection are the critical ones, proper treatment should be instituted at once in these individuals. As many as two-thirds of the deaths have occurred within these first five years of the initial rheumatic infection.

If the heart is slowed down only 10 beats per minute by rest in bed, it contracts nearly 15,000 times less in twenty-four hours.

Salicylates.—Salicylates seem to have a beneficial effect on joint manifestations and pericardial effusion (*i. e.*, exudative reactions) and until recently there was no evidence that they stemmed the progress of endo- or myocarditis. Given in adequate amounts, this drug results in lessening of joint pains and lowering of the temperature.

In children, as much as 4 to 8 gm. (60 to 120 gr.) of salicylates together with sodium bicarbonate may be necessary in twenty-four hours to control pain and reduce the temperature to normal. Some clinicians prefer aspirin in doses of from 2 to 3 gm. (30 to 45 gr.) daily in adults; aspirin is usually given in approximately one-half the salicylate dosage. Others state that the toxic effect of salicylates is minimal if combined with magnesium oxide and that 6 gm. (90 gr.) of sodium salicylate daily, together with 4 gm. (60 gr.) of magnesium oxide are equivalent to 10 gm. (150 gr.) of sodium bicarbonate and an equal amount of sodium salicylate. Some prefer the administration of salicylates by rectum; 8 to 12 gm. (120 to 180 gr.) of sodium salicylate in from 2 to 4 ounces of warm water or milk may be given from one to three times a day through a catheter and syringe (the tip of the catheter being from 6 to 8 inches inside of the anus). Salicylates may be continued until joint pain and fever are controlled without their use; the dose may be decreased markedly as the patient improves.

Other substances which have been used include aminopyrine,[6,41,83] cinchophen[185] and neo-cinchophen, intravenous injections of emulsified olive oil (to adsorb toxins by the minute fat globules), intravenous colloidal charcoal[234] (with the same idea), etc.; *aminopyrine or cinchophen are not advised because of the possible danger of granulocytopenia*[64] *or severe liver damage*, respectively. The use of olive oil or charcoal injections intravenously cannot be advocated.[65]

Derick, Hitchcock and Swift found that *aspirin* (or neo-cinchophen) *usually prevented the arthritis of serum sickness* and that in these individuals the production of precipitins against horse serum was inhibited.[76] Sheldon was not convinced that a course of aspirin begun at the time of onset of a pharyngitis in patients with inactive rheumatic fever was beneficial but suggested further trials.[225] Leech gave 1.3 gm. (gr. xx) of aspirin daily for six months to 65 children with inactive rheumatic heart disease.[152] Rheumatic recurrences appeared in 7 per cent of the treated group, and in 11 per cent of a control group; however, the children taking the aspirin had better general health, had fewer minor complaints and gained more weight than the controls. Schlesinger reported fewer rheumatic recurrences in children and milder recurrences when these appeared in those rheumatic children who were given from 0.5 to 0.8 gm. of aspirin three times a day at the onset of pharyngitis.[213] *Coburn and Moore gave from 4 to 6 gm. of sodium salicylate daily for a month to rheumatic subjects who developed pharyngitis and in whom Group A hemolytic streptococci were cultured from the throat.*[58] *Only 1 of 47 patients so treated developed rheumatic fever.*

One of the most interesting recent developments in the treatment of rheumatic fever is the *use of large amounts of salicylates daily*, while following the plasma salicylate level, in an effort to quickly modify the activity of the disease. It had been previously considered that salicylates merely relieved the pain and fever associated with the disease, and had no effect upon the development of cardiac lesions. **Recent work by Coburn suggests that large doses of salicylates may (in some instances) prevent the development of carditis in rheumatic fever.**[53]

Salicylate is the only drug which has survived many years in the treatment of rheumatic fever. There is no evidence that salicylate has any effect on the infectious agent or that it modifies the immune response of the rheumatic fever patient.

Kapp and Coburn studied the metabolism of sodium salicylate and showed that rheumatic fever patients who showed the best clinical response, including those who received salicylates intravenously, excreted a larger proportion of the drug in the form of salicyl than patients who did not respond satisfactorily; this suggested that some of the salicylic acid reacted with the inflammatory process and was conjugated.

In 43 patients admitted to a hospital with an acute attack of rheumatic fever, intermittent doses of from 3 to 6 gm. of sodium salicylate were given only for the relief of symptoms and pyrexia. About 40 per cent of this group had polycyclic rheumatic attacks and all but 3 developed cardiac valvular disease. *Eighteen patients who were treated with daily doses of 10 gm. or more of sodium salicylate by mouth or vein all showed rapid clinical recovery and a progressive fall in the blood sedimentation rate.* This study suggested that although 3 to 6 gm. daily was adequate to relieve pain and fever, it failed to suppress the inflammatory reaction.[53] *Doses of 10 gm. daily gave symptomatic relief and also modified the rheumatic infection. These effects were obtained most rapidly in patients receiving sodium salicylate intravenously.* In exceptionally severe attacks, larger intravenous doses were required at the onset. These patients did not develop evidence of renal irritation and prothrombin determinations were normal in 6 of the patients who were tested.

Brodie, Udenfriend and Coburn developed a method for measuring the *concentration of the salicyl radicle in plasma.*[90] It was found that the salicylate plasma content is readily brought up to 400 gamma per cc. in four hours by intravenous administration, while it requires two days to develop this level with oral doses. It is possible to attain a plasma level of 600 gamma per cc. when 20 gm. of sodium salicylate are given intravenously during a period of eight hours. When necessary, one can build up a plasma level of 650 gamma per cc. in a few hours without causing symptoms other than mild tinnitus.

Coburn's Technic for Salicylate Therapy in Acute Rheumatic Fever.[53]

First day: *10 gm.* of *sodium salicylate* in 1000 cc. of 0.9 per cent NaCl *by intravenous drip* in four to six hours.

Second day: If rheumatic symptoms persist or if the temperature has not reached normal, give 20 gm. of sodium salicylate in 2000 cc. of 0.9 per cent NaCl in eight hours.

Third day: Repeat second day's treatment if necessary, but if the patient is symptom-free and afebrile, give 10 gm. as on the first day.

Fourth to sixth day: *Continue sodium salicylate infusions of 10 gm. daily until the sedimentation rate has dropped appreciably* (about 20 per cent).

Seventh to thirtieth day: Give a total of 10 gm. of sodium salicylate and 4 gm. of sodium bicarbonate daily by mouth in divided doses; *i. e.*, 1.6 gm. (25 gr.) of sodium salicylate and 0.6 gm. (10 gr.) of sodium bicarbonate every four hours day and night.

After the thirtieth day: *After the sedimentation rate has remained normal for two weeks or more, permit the patient a trial week in bed without salicylate.* If the sedimentation rate remains normal and symptoms do not recur, allow him up progressively more and more. If rheumatic symptoms, fever or a marked rise in sedimentation rate occur during this test week, give another two weeks' course of therapy beginning with one intravenous infusion of 10 gm. followed by oral salicylate medication as outlined previously.

Additional Therapy for the Patient With Acute Rheumatic Fever Includes:

1. *Good nursing care.*
2. *Rest in bed* during the acute stages of the disease.
3. *Relief of cardiac pain* by an ice-bag to the precordium, or by codeine or morphine if needed.
4. Liquid or semisolid *diet* in the acute stages.
5. *Fluids* should be *forced* to replace water lost by sweating; in the presence of severe heart failure, limit fluids to 800 to 1500 cc. in twenty-four hours.
6. Cooling baths to reduce fever are unnecessary because salicylates are so effective, and local applications for the joint phenomena are usually unnecessary.
7. *Soft pillows* may be used *as splints* under and around inflamed joints in the acute stages.
8. *Aspiration of pericardial effusion* is *seldom required* but may be needed occasionally if the amount of fluid is large enough to interfere with cardiac function; thoracentesis may be needed if pleural effusion produces circulatory or respiratory difficulty.
9. *If congestive heart failure* occurs, *digitalization* is required, together with restriction of salt and fluids and the use of diuretics in some cases.
10. *Chorea* usually requires only *sedation* (as phenobarbital from 0.015 to 0.030 gm. ($\frac{1}{4}$ to $\frac{1}{2}$ gr. four times per day), *rest* in a *quiet room and sympathetic handling* of any emotional instability.[165] If acute or violent manifestations occur, *careful nursing* is required to guard against personal bodily injury from the uncontrolled movements and *fever therapy* should be given (aiming to produce a temperature of from 104° to 105° F. for three to five hours; the fever therapy must be repeated for an average of six sessions, as needed). Milder forms of chorea which do not respond to conservative care may require fever therapy.

Transition From Bed Rest to Activity in Patients With Rheumatic Fever:[165]

1. When the sedimentation rate has returned to or nearly to normal, and when the rectal temperature, pulse and leukocyte count have remained normal for ten to fourteen days in the absence of antirheumatic drugs and when there is no evidence of joint pain, subcutaneous nodules or rash, with a satisfactory increase in weight and hemoglobin and erythrocyte count and the patient's general condition has improved satisfactorily, the patient may *sit in a chair* for twenty to thirty minutes morning and afternoon and increase this by thirty minutes per day until most of the day is spent out of bed.
2. *Walking on the level* is permitted next and finally stair climbing, if necessary.
3. *Rest in bed must be resumed temporarily if signs of increased rheumatic activity appear at any time.*
4. *Mild sports* (but no strenuous competitive activity) *are permitted and may be encouraged in individuals without evidence of cardiac embarrassment. Too close restriction may lead to the development of cardiac neuroses.*
5. *Additional general items of therapy* include *occupational therapy* or *home schooling*, especially for children confined to bed at home; *iron* (as ferrous sulfate 0.3 gm. t.i.d.) or *blood transfusions* for anemia, and *sulfanilamide as a prophylactic measure.* **Sulfanilamide is useless during the active stage of the disease and may be harmful.** Children tolerate it well as a prophylactic measure. *Climatotherapy* is useful in cases of long standing, low grade activity; the disease is definitely less common and less acute in the southern United States. Arizona, southern California and Puerto Rico have been equally popular as a climate of choice for these subjects.
6. Statistical studies show *no evidence that eradication of foci of infection favorably affects the course of rheumatic fever.* Some believe that tonsillectomy is dangerous if done early in the acute stage of rheumatic fever. In rheumatic individuals who are susceptible to sore throats and tonsillitis, it is usually agreed that tonsils should be removed during an inactive stage of the disease.

Oil of wintergreen is extremely poisonous and constitutes a particular hazard in the home with children (because of its pleasant odor).[132] All oil of wintergreen when dispensed should be labeled "poison" and the purchaser warned of its danger. Death may follow poisoning from oil of wintergreen.

Local Therapy to Joints.—The swollen and painful joints may be protected by pillow splints, or molded plaster, castex or leucite. *Relief following salicylates is usually so prompt that splints are seldom required.* If the lower limbs are involved, *a cradle should be placed over the legs* so that the weight of the bed clothes does not rest upon the involved joints. Applications of methyl salicylate ointment to the involved joints, followed by wrapping in cotton or wool, may be useful in lessening joint discomfort. Fatal cases of methyl salicylate poisoning have been reported.[18] In one child, fatal toxic symptoms (dyspnea, vomiting, cyanosis, delirium, acidosis and death) occurred, following 4 gm. (60 gr.) of salicylates (orally) and a total of 90 cc. (3 ounces) of oil of wintergreen to the knees during a period

of five days.[151] In very occasional cases, even with local splinting of the involved joints, codeine or morphine may be required.

Tonsillectomy.—*If the tonsils are definitely infected, they should be removed after the acute infection has subsided;* however, if, after an adequate period of rest in bed (four to eight weeks), the disease shows no signs of abating or is becoming worse, one may risk tonsillectomy (if the tonsils are definitely infected).

Swift believed that tonsillectomy seemed indicated if the tonsils remained diseased because of the frequency with which rheumatic fever followed tonsillitis.[249] He believed that the tonsils might be the site of production of sensitizing substances or areas of infection from which bacteria were discharged into the blood stream. However, *the removal of tonsils is not absolute insurance against recurrences of the disease* inasmuch as patients may have other sources of sensitization or may incur reinfection with hemolytic streptococci.

Kaiser summarized the *effect of tonsillectomy in 48,000 rheumatic children.*[131] He concluded that: *"Tonsillectomy is justified in practically every rheumatic child until other factors that influence this disease are better understood."*

1. Tonsillectomized children are as likely as others to develop muscular rheumatism and chorea.

2. *Tonsillectomized children are as likely to develop subsequent rheumatic fever and carditis as non-tonsillectomized ones.*

3. *The incidence of recurrent attacks of rheumatic fever is uninfluenced by tonsillectomy performed before or after the initial attack.*

4. *The mortality was nearly twice as high in children with tonsils at the initial attack as among those tonsillectomized before the initial attack.*

Miscellaneous Forms of Therapy.—1. **Fever Therapy.** Fever therapy is not advised for rheumatic manifestations, other than chorea, and its use should be considered experimental.[55]

There are but few reports of fever therapy in rheumatic fever.[226] This must be employed only as a hospital procedure under the careful supervision of one especially trained in the field. In one series of 15 patients so treated (with the Kettering hypertherm for periods of eight and a half to forty-six hours with temperatures of 103° to 106° F.), 13 individuals obtained complete relief from joint pain and swelling and only 3 had recurrences (from two weeks to twenty-one months later).[79] In many cases, the sedimentation rates and leukocyte counts became normal after fever therapy. The symptomatic improvement was usually evident after the first treatment. In another small series of patients, similar results were obtained;[240] the same type of improvement has been noticed in the fever therapy of Sydenham's chorea, many being cured, and the majority being markedly improved (even some cases previously unrelieved by typhoid vaccine).[115] *This is still an experimental procedure in rheumatic fever.*

2. **Streptococcus Hemolyticus Filtrate.**—Wasson thought that immunization of children who have had one or more attacks of rheumatic fever with a stock filtrate of Streptococcus hemolyticus *appeared to prevent further attacks and improved the well-being of the individual.*[268] Only 2 of 34 children developed the disease in its acute stage during the course of therapy in contrast with 15 and 43 per cent in two control groups.

Wasson and Brown concluded after nine years of experience with immunization, first with crude and later with attenuated hemolytic streptococcus toxin, against recurrences of attacks of rheumatic fever that *the children were much benefited by the prophylactic treatment and that they suffered no harm.*[270] They used a *tannic acid precipitated toxin of strain NY 5 intradermally in 4 injections* of 5000, 8000, 10,000 and 12,000 S.T.D. at three week intervals, following which 10,000 S.T.D. was given every six months. *Forty-two subjects were so immunized and during a nine month period there were no rheumatic attacks while the incidence of attacks was 33 per cent in 33 controls.* This treatment called for only 5 visits during the first winter and for 2 a year thereafter. Wasson and Brown suggested that this short method of immunization may confer what may be a lasting immunity against acute rheumatic reinfections. Coburn and Pauli, however, found no evidence that the course of immunization increased resistance to streptococcal infections or rheumatic fever.[34]

3. **Vaccines and Sera.**—Small has reported good results in the treatment of rheumatic carditis with an aqueous extract of streptococcus cardioarthritidis.[227–229] He states that "hyperergy" or marked reactivity or hypersensitiveness to streptococci or their derivatives is an outstanding feature of the rheumatic state. Some individuals[94,158,279] have favored attempts at desensitization by streptococcal vaccine and filtrates,[269] while others have reported this procedure to be useless.

4. **Sulfanilamide.**—Swift and his colleagues concluded that the *toxic action of sulfanilamide in active rheumatic fever so far outweighed the beneficial therapeutic effects that its administration to patients with active manifestations of this disease did not seem justified.*[253]

The symptoms may be intensified and marked toxemia may occur following the administration of the drug during the active course of the disease. Sulfanilamide was of no value in the treatment of chorea.

Sulfanilamide is ineffective in treating acute attacks of rheumatic fever and may aggravate the disease.[163,253] Following a trial of sulfonamide therapy in active rheumatic fever, toxic reactions with high fever occurred in some cases and congestive heart failure was precipitated not infrequently. It is not known whether this latter was due to an intensification of the rheumatic state, to an effect of the sulfonamide on the myocardium or to a toxic reaction of another type.

Thomas has emphasized that it is better to give sulfanilamide as a preventative than to give digitalis for irreparable heart disease.[255–257] She began the use of sulfanilamide in convalescent rheumatic (fever) patients in 1936 with a group of adolescents and young adults who had had acute rheumatic fever within the immediately preceding three years. The patients were ambulatory, although 69 per cent had chronic rheumatic carditis and 33 per cent had definite cardiac enlargement. The rheumatic states were quiescent when sulfanilamide was begun.

The cause of rheumatic fever is still unknown. However, pharyngitis or tonsillitis from the beta-hemolytic streptococcus often precedes an acute attack of rheumatic fever by from one to three weeks. In the acute stage of the disease, the antistreptolysin titer rises.

In the group of the patients treated prophylactically, *0.6 gm.* (gr. x) *of sulfanilamide was given morning and evening daily from October or November until June. None of the treated patients suffered from beta-hemolytic streptococcus infections during the period of sulfanilamide treatment and no major attack of rheumatic fever occurred in any patient during the time sulfanilamide was being given.* On the other hand, major rheumatic episodes were seen in 15 of the control patients.

Thomas gave sulfanilamide prophylactically to some of her patients for from three to six years. *In this six year period, only two rheumatic recrudescences were seen in patients receiving sulfanilamide. All of the individuals who received sulfanilamide for long periods are in vigorous physical condition.* Patients who tolerate the drug for two weeks usually have no further trouble.

If toxic reactions appear, the drug is withdrawn and then resumed in a dose of only 0.3 gm. (gr. v) a day, gradually increasing to 0.6 gm. twice a day. If a sudden drop in leukocytes occurs (which may be expected after eighteen to twenty-one days of treatment), sulfanilamide should be discontinued.

Coburn and Moore gave prophylactic doses of sulfanilamide to children for three years.[56,57] At the end of this time, 100 of the patients, mostly adolescents, had the sulfanilamide withdrawn. None of these 100 patients had streptococcic pharyngitis or manifestations of rheumatic activity when they received sulfanilamide. Thirty-two of these 100 patients contracted hemolytic streptococcic pharyngitis during the first twelve months following the withdrawal of sulfanilamide, and in 40 per cent of these untreated, infected patients, rheumatic fever developed. These follow-up observations indicated that *rheumatic children who escaped streptococcic infection and rheumatic activity while receiving sulfanilamide prophylactically for three years were still susceptible to both streptococcic pharyngitis and rheumatic fever.* This study also shows that *the prophylactic effect of sulfanilamide does not exert any beneficial effect beyond the period of treatment.*

Kuttner noted that only 2 of 108 children receiving sulfanilamide contracted streptococcal pharyngitis and only 1 of these developed rheumatic manifestations.[144] Among the 104 children serving as controls, 48 contracted streptococcal pharyngitis and 23 of these developed rheumatic relapses. No rheumatic recurrences were observed in children who escaped streptococcal upper respiratory infections. These children were kept at a sanitorium for rheumatic children. They ranged in age from seven to fifteen years. These studies were carried out for two successive winters. Beginning in October and continuing until June, sulfanilamide was given in a dose of 1 gm. (in three divided doses) per day to children weighing 75 pounds or less, and from 1.3 to 2 gm. in those weighing more than 75 pounds.

This dose was increased slightly during the second year in order to maintain an average blood level of 1.5 mg. per cent.

About *15 per cent showed toxic manifestations from the sulfanilamide: fever, nausea, urticaria, erythema* and *abdominal pain. No serious reactions were encountered.* Three children showed a gradually developing *leukopenia* accompanied by a fall in neutrophils, but the blood picture returned to normal within a few days when the drug was discontinued. *Those children who did not develop toxic reactions within five weeks tolerated the drug well.* In most instances, the hemoglobin fell slightly in those receiving sulfanilamide. No evidence of sensitization was noticed when the drug was started after a lapse of five months following two winters of treatment.

Thomas *advised that sulfanilamide be begun before the patient with rheumatic fever is discharged from the hospital because of the danger of exposure to respiratory infections in the home.*[255] In her opinion, if the leukocyte count and temperature were normal after the withdrawal of salicylates, there was no need to wait until the sedimentation rate fell to normal before beginning sulfanilamide prophylaxis. She further advocated that *sulfanilamide prophylaxis be continued throughout the entire year* since some recurrences appear in the summer and fall. She has advised that *sulfanilamide prophylaxis be continued for five years or longer after an attack of rheumatic fever.*

Prophylaxis in Rheumatic Fever

1. *There is no specific agent for the prevention of rheumatic fever.*
2. There is no simple test to separate those susceptible to rheumatic infection from the non-susceptible.
3. The present view of the pathogenesis of rheumatic fever is that some individuals, because of hereditary or environmental factors, or both, develop abnormal tissue reactions to hemolytic streptococci;[34] thus, *the prevention of rheumatic attacks is based mainly on the prevention of hemolytic streptococcus throat infections.*
4. The general resistance should be maintained by proper nutrition and avoidance of chilling and fatigue.
5. The patient and his family should be educated concerning the rôle of respiratory infections in rheumatic attacks and the necessity of protecting rheumatic subjects from such infections.
6. According to Wilson, if both parents had rheumatic infection, nearly all the offspring will become afflicted; if one parent is positive and a grandparent on the other side of the family is positive, there is a 50 per cent expected incidence in the offspring. If parents are negative, but one grandparent of each line is positive, the expected incidence is about 25 per cent. Therefore, one should closely observe offspring of families in which rheumatic fever is prevalent to detect the disease at its onset. It is doubtful if advice will often bear fruit but patients should be informed of such possibilities when advised as to marriage and child-bearing.
7. If permanent *residence in tropical or subtropical climates* can be arranged without hardship or financial sacrifice, such a prophylactic undertaking is reasonable. Although streptococcal infections and rheumatic fever were formerly thought to be rare in the tropics, recent

Prophylaxis in Rheumatic Fever—(*Continued*)

studies have shown the presence of such diseases.[44,146,221,274] However, *rheumatic fever is much less frequent and is usually milder in southern United States than in the northern temperate zone.* Transportation to southern climates is not the sole answer to rheumatic infection because of the economic status of most of these individuals and because the disease may remain active or may recur in such climates. Residence in a tropical climate should not provide a false sense of security against recurrences.

8. **The use of sulfanilamide daily seems to be the best form of prophylaxis against recurrences of hemolytic streptococcal respiratory infections and recurrences of rheumatic fever.[46] Sulfadiazine prophylaxis on a mass scale greatly reduces the incidence of rheumatic fever.[122]** Sulfanilamide is given in a dose of 0.6 gm. (gr. x) morning and evening from October or November until the end of May. A blood level of 2 mg. per cent appears to be adequate.[34,165] It may be advisable to continue the drug during the whole year (for at least several years). *Toxic reactions* may appear between the fourteenth and fiftieth day (most often). If marked leukopenia occurs, the drug is stopped, and begun again later in smaller doses. Nausea and vomiting cause withdrawal of the drug in some instances.
9. Slum clearance projects and similar sociologic advances may help materially in reducing the incidence of rheumatic fever.
10. Visitors and attendants with respiratory infections must be kept away from rheumatic fever patients. Other members of the family must understand that their "simple sore throat" may be a potential attack of rheumatic fever to the patient and isolation technic must be taught.
11. The prophylactic value of *tonsillectomy* is still open to question. Most recent work suggests that there has been no striking influence upon the course of rheumatic fever by removal of the tonsils. All are agreed that if tonsillectomy is indicated as a general health measure, it should be done (just as in non-rheumatic patients). There is also general agreement that *tonsillectomy should not be done until the rheumatic fever is quiescent* or it may result in an acute exacerbation of the disease. At present, we advise the removal of tonsils in children with rheumatic parents, or in rheumatic children in whom there are frequent bouts of pharyngitis or tonsillitis. However, available statistics do not show that tonsillectomy reduces the incidence of recurrence of rheumatic infection.
12. Available data concerning the value of *hemolytic streptococcus toxin* given intradermally are conflicting. Coburn and Pauli found no evidence that the course of immunization increased resistance to rheumatic fever or streptococcal infections.[60] Wasson and Brown reported results with a short course of immunization which compared favorably to those obtained following sulfanilamide prophylaxis. This question must await further work.
13. Recent work has also suggested that *salicylates may be of value prophylactically;*[58] in 47 quiescent rheumatic subjects who were given from 4 to 6 gm. of sodium salicylate daily for a month beginning at the onset of hemolytic streptococcus pharyngitis, only one developed rheumatic fever, although 15 others had a brief rise in sedimentation rate after withdrawal of salicylates. In a control group, 40 per cent developed recurrences of rheumatic fever. Others have tried the effect of a course of aspirin in the prevention of rheumatic fever, giving from 1.5 to 2 gm. daily in divided doses over periods ranging from six to twelve months.[152,182,225] Results of this attempted prophylaxis with aspirin were questionable but suggested that there was definite benefit in most instances.
14. The specific value of other vaccines or antisera[262] in the prophylaxis of rheumatic fever is very questionable as equally good results were obtained following small doses of typhoid vaccine.
15. Neither vitamin C nor any other vitamin or foodstuff has been shown to have a specific beneficial action in the prophylaxis or treatment of rheumatic infection. All essential foodstuffs should be ingested in adequate amounts.

Prophylaxis in Rheumatic Fever—(*Continued*)

Stowell and Button discontinued sulfanilamide permanantly in 25 per cent of the patients treated prophylactically because of fever, rash, leukopenia or nausea; *one of their children died of agranulocytosis twenty-nine days after onset of sulfanilamide therapy.*[237] Others have had entirely different experience and toxic reactions were encountered in not more than 15 per cent of the subjects; these were not serious.[103,143,255]

Holbrook has reported that a **50 to 75 per cent reduction in the incidence of respiratory diseases and streptococcic infections followed sulfadiazine prophylaxis** in a large troop population.[122] **There was a parallel reduction in rheumatic fever.** Coburn reported that **continuous ingestion of 1 gram of sulfadiazine daily was effective in checking advanced streptococcic epidemics, checking an epidemic at its onset and in protecting 85 per cent of susceptible recruits from implantation with bacterial respiratory pathogens.**[54] As little as 0.5 gm. of sulfadiazine daily is almost 85 per cent effective in preventing implantation by Streptococcus hæmolyticus. This has been given continuously for as long as three months.

Recently, small series of cases have been reported using *sulfathiazole* or *sulfadiazine* instead of sulfanilamide in prophylaxis. Boyer has had to discontinue the prophylactic use of sulfadiazine in only 1 case because of a toxic reaction (fever and rash); during the early weeks of therapy, no lowering of the leukocyte count was observed as occurs with sulfanilamide.[34] Others have expressed the opinion that the toxic reactions from sulfadiazine (mainly urinary) have prevented them from continuing further experiments with this drug and they have now returned to the use of sulfanilamide.[204]

It has been found that the possibility of exacerbations following tonsillectomy can be decreased markedly by the daily administration of aspirin for a month following the operation.[7] The frequency of exacerbations following tonsillectomy may be decreased if sulfamerazine is given for twenty-four hours prior to and following operation. Insufficient data are available at present on the value of such therapy. Sulfonamides should not be given for any prolonged period during the active stage of rheumatic fever as these patients do not tolerate the drugs well at

that time; the symptoms during the active stage of the disease may be made markedly worse by the administration of sulfonamide preparations.

Open Air Environment.—Hubbard and Griffin treated patients with rheumatic fever in a carefully controlled, **open air environment.**[123] *It was possible to keep them free of respiratory infections.* The children slept outdoors, even in the most severe weather, and were kept in the open air as much as possible during the day. Throughout the entire period, the children had no respiratory infections and no recurrences of rheumatic fever. With freely circulating air that is rapidly diluted with the outer air, there is relative freedom from bacterial contamination in comparison with the usual hospital wards. Bacterial determinations made during the winter, spring and summer compared favorably with similar counts obtained in a carefully supervised, air-conditioned, infection-free premature infant nursery and in infant cubicles protected with ultra-violet radiation. Hemolytic streptococci and other pathogenic bacteria were found in air samples from hospital wards, but only staphylococci, molds and a variety of non-pathogenic organisms were obtained at the sanitorium.

Anemia.—Almost all patients with rheumatic fever manifest some degree of anemia; this can be treated with adequate daily doses of iron (such as ferrous sulfate, 0.2 gm. [3 gr.], four times a day or ferrous gluconate, 0.3 gm. [5 gr.], five times a day) or, if severe, with small multiple transfusions (150 to 200 cc. each).

Vitamin C.—Rinehart believed that vitamin C deficiency was of etiologic importance in rheumatic fever.[195–197,199–202] No harm is done by including a large glass of orange juice in the daily diet of these patients, or by giving tablets of ascorbic acid (100 mg. twice a day).

Rheumatic fever may be associated with poverty—which suggests that nutritional inadequacy may play a rôle in its etiology. Recent surveys have shown that vitamin C undernutrition is frequent in the poorer classes. Recurrences of the disease are not prevented, however, by maintenance of rheumatic patients on adequate doses of ascorbic acid.[198]

Others have noted no improvement following vitamin C therapy in these patients[217,285] The vitamin C deficiency is probably an associated phenomenon as is so common in many chronic infections.

Climate.—In some parts of the United States rheumatic fever is said to be comparatively rare.

It has been suggested that *a prolonged stay in a subtropical climate may be of almost specific value to most of these individuals.* Marked improvement or remissions have been found in a majority of patients with active carditis who have been sent to Florida or Puerto Rico.[175,205] However, transportation of individuals with rheumatic fever to tropical or subtropical climates involves many difficult problems and is not to be entered into lightly. Southern Florida, Arizona, southern California and Puerto Rico have been equally popular as a climate of choice for these subjects.

Sulfanilamide.—Although sulfanilamide seems to be of great prophylactic value in preventing recurrent attacks of hemolytic streptococcal upper respiratory infections and of rheumatic fever, the physician must be warned

that according to our present information sulfanilamide should not be given to patients as treatment of an acute attack of rheumatic fever because of the untoward effects which may follow its use in this active stage of the disease.

Penicillin.—Penicillin has been tried in a series of 30 patients with acute rheumatic fever without any influence upon the course of the disease.

Splenectomy.—This has not resulted in any favorable modification in the course of the disease.

Foreign Protein.—*In general, foreign protein such as typhoid vaccine is not advised in the treatment of rheumatic fever;* however, in an occasional obstinate case, attacks have been stopped by its use.[26,45]

Special Care of Children With Rheumatic Heart Disease.—Various workers have stressed the importance of early and continuous supervision of rheumatic infection in childhood in an effort to minimize later effects of the disease.[70] When the disease has become inactive, every effort should be made to minimize exposure to upper respiratory infections and parents and teachers must be discouraged from making a chronic invalid out of the child; school authorities should be informed that the vast majority of those who attend regular schools when their disease is inactive can and should engage in normal school life. Physical activity should be encouraged within the limit of the child's capability. *Only a small per cent of adolescent children have permanent heart disease of sufficient severity to preclude normal activity.*

Hedley studied the **facilities in the United States for the special care of children with rheumatic heart disease.**[113] He concluded that in the larger cities in the North, "all-purpose" rheumatic heart disease hospitals would probably be more efficient and economical than mere convalescent institutions. He suggested that initial attacks of rheumatic fever could be treated in a general hospital and the patients be admitted to the cardiac sanitarium when their activity had subsided sufficiently.

Hedley reported that in 1942 there were less than 1000 beds devoted exclusively to the care of rheumatic heart disease patients in the United States, 300 of these being in institutions in Greater New York. Prior to World War II, London alone had 900 beds set aside for prolonged hospital and convalescent care of children with rheumatic heart disease.

In 1940, there were *in the United States, one rheumatic heart disease hospital, seven convalescent institutions devoted exclusively to the treatment of children with heart disease, and one organization with a unit furnishing foster home care to children recovering from rheumatic infection.* Several additional rheumatic heart disease sanitaria have been opened since 1940, and in the fall of 1940 a general convalescent institution in Florida was to be devoted entirely to the treatment of rheumatic heart disease. In some places, convalescent sanitoria furnish care to rheumatic cardiac patients and some of these have wards set aside for these individuals. With the coming of World War II, special attention has been paid on a large scale to the institutional care of service men with rheumatic fever and rheumatic heart disease.

The *House of the Good Samaritan in Boston, Mass.*, has been devoted exclusively to the treatment of rheumatic heart disease since 1921, being *the only civilian rheumatic heart disease hospital in the United States.* This institution had a capacity of 80 beds and treated about 175 patients per year. Until recently, all patients were admitted free. Now, a maximum charge of $10 per week is made to patients who can afford to pay, but strictly private patients are not accepted. The average duration of hospitalization is about four months. A previous tonsillectomy is necessary for admission. This institution has a splendid follow-up system and has been responsible for numerous research studies on rheumatic fever.

Cardiac Convalescent Sanatoria are located as follows:

1. *Saint Francis Sanitorium for Cardiac Children, Mineola, Long Island, N. Y.* Since 1937, this has been used exclusively for the care of children with rheumatic heart disease. The building is located in the suburbs on a 15 acre tract. It has a capacity of 50 beds. Admission is restricted to girls aged six to twelve years.

2. *Irvington House, Irvington-on-Hudson, N. Y.* This was established in 1920 and has a capacity of 150 beds. It is located on a knoll 500 feet high on the east bank of the Hudson River about 20 miles north of the center of New York City. This is a private institution. Applications are made through the New York Heart Association by the chief of a recognized heart clinic. Tonsillectomy is not required. No private cases are accepted and all treatment is free of charge. Children of both sexes seven to fifteen years of age are accepted. Admissions are limited to patients who have had rheumatic fever or chorea and who offer reasonable good prognoses.

3. *Pelham House, Pelham Manor, N. Y.*, was established in 1914 and is the oldest cardiac convalescent institution in this country. It is owned by a private organization (The Pelham Heart Association). It admits girls from five to twelve years. Most of the cases are admitted from and followed by the clinics of the Presbyterian Hospital (in New York).

4. *Martine Farm, White Plains, N. Y.*, was established in 1921 and consists of a large farm house with 25 beds on a 45 acre tract. Boys and girls aged six to sixteen are treated.

5. *The Children's Heart Hospital in Philadelphia, Pa.*, is actually a convalescent sanitorium, containing 60 beds and located on a 14 acre tract. The institution is privately owned and is operated under the auspices of the Philadelphia Heart Association. A charge of $3 a week is made which is met either by the parents or by referring social agencies. It admits white and colored girls aged three to thirteen, white boys aged three to twelve and colored boys aged three to eleven. This institution is used by the University of Pennsylvania for undergraduate and graduate instruction.

6. *The Lymanhurst Cardiac Convalescent Home in Minneapolis, Minn.*, was organized in 1935 and is under the joint control of the city health department and the school system. Its capacity of 40 patients is limited mainly to children of school age less than sixteen years.

7. *The La Rabida Jackson Park Sanitorium in Chicago* has been devoted to the treatment of heart disease since 1922. It has a capacity of 100 beds and is financed by private donations.

Newer sanatoria have been established as follows:

1. Victoria Foundation, Inc., Morris Plains, N. C.
2. Heart House, Pittsburgh, Pa.
3. The National Children's Cardiac Home in Miami, Florida.

General Convalescent Institutions which furnish convalescent care to rheumatic cardiac patients include the following:

1. New Haven Community Center, Hamden, Conn.
2. Ridge Farm Sanatorium, St. Louis, Mo.
3. Children's Convalescent Hospital, Rochester, N. Y.
4. Milwaukee Children's Convalescent Hospital, Milwaukee, Wis.
5. Theresa Grotta Home for Convalescents, Caldwell, N. J.
6. Happy Hills Home, Baltimore, Md.

Other convalescent institutions which furnish care free or at reasonable cost to children with heart disease are the Willow Crest Home, Willow Crest, Pa.; Ivy Craft Convalescent Home, care of Jefferson Hospital, Philadelphia, Pa.; Dunwoody Home, Newtown Square, Pa.; Broomall Convalescent Home, Broomall, Pa.; Children's Hospital and Convalescent Home, Wellesley, Mass.; Christ Child Home, Rockville, Md.; Sarah Schermahorne Home, Milford, Conn.; Seashore Home, Atlantic City, N. J.; Children's Hospital of Michigan Convalescent Home, Farmington, Mich.; Loet Home, East View, N. Y.; and Children's Convalescent Home, Westfield, N. J. At present, the increased interest in this problem is leading to the formation of many more institutions of this type.

Some general convalescent institutions have been prejudiced against receiving rheumatic cardiac patients because of the fear of sudden death. Such fear is groundless; *at the Children's Heart Hospital in Philadelphia, there has not been a sudden death among more than 1000 admissions over a period of fifteen years.*

The Speedwell Society, 20 East Forty-second Street, New York City, *is devoted exclusively to furnishing prolonged care to 30 children aged six to twelve years with rheumatic or potential rheumatic heart disease.* These children *are placed in foster homes* within a radius of about a mile. Not more than 3 children are sent to one home. They are examined periodically by a local physician and are contacted daily by a visiting nurse. A special school is maintained in the center of the area. The duration of this foster care is two winters.

PATHOLOGY OF RHEUMATIC FEVER

The two basic inflammatory changes occurring in rheumatic fever are *exudation* and *proliferation*, both of which represent response of tissue to injury. The exudative process is responsible for the swollen joints and rheumatic pleurisy and the proliferative reaction for the Aschoff bodies and subcutaneous nodules.

The joint process is peculiar in that it almost never results in suppuration or in permanent damage to the joints. During the acute stage of the disease, joint cavities become distended with a turbid, yellowish fluid, with an accompanying injection and slight swelling of ligaments, capsule and synovial membrane.

A *pancarditis* is usually present; *involvement of the endocardium is the outstanding lesion in most cases.* Myocarditis occurs in practically all cases and pericarditis is also frequent. The endocardium covering the mitral valve is most often affected although the aortic and tricuspid valves may also be involved in the process.

The mitral valve is damaged in practically all cases of rheumatic carditis, while the aortic is injured in 50 per cent, the tricuspid in 33 per cent and the pulmonary, only rarely.[68] Mitral stenosis and aortic insufficiency become more frequent with an increasing number of attacks.

Rheumatic verrucæ are small, firm wart-like excrescences most often seen at the line of closure of the valve leaflets on the left side of the heart, especially the mitral and aortic valves.[275] They average 1 to 2 mm. in size and may become confluent.

Active carditis occurs more frequently when polyarthritis is present at the onset and less often when the initial signs are chorea or growing or joint pains.

The outstanding pathologic lesion in the myocardium is the **Aschoff nodule.**[8] These lesions are found most often in the posterior wall of the left ventricle and basal part of the interventricular septum and consist of large, irregularly shaped, round wandering cells often placed concentrically around a small blood-vessel. They have been seen in from 30 to 90 per cent of hearts with a history of rheumatic infection.[275] Aschoff bodies have also been seen in the endo- or pericardium, adventitia of blood-vessels and in other organs. The subcutaneous nodules found in fascia, tendons, periosteum, etc., throughout the body have been described previously.[67,242] Other pathologic details are not of special interest to the general practitioner.

Rheumatic Fever and Rheumatoid Arthritis.—Boots has been interested in a group of patients whom he considers to have a *chronic form of rheumatic fever*, including many cases formerly diagnosed as rheumatoid arthritis.[32] In these individuals, the joints have the typical appearance of rheumatoid arthritis *with symmetrical fusiform swellings*, but the blood sera of these individuals do not agglutinate hemolytic streptococci and they uniformly exhibit a high antistreptolysin titer. He considered these patients to have adult rheumatic fever.

Points of similarity between rheumatic fever and rheumatoid arthritis include: (1) The tendency to occur in the same families, chiefly in temperate zones, often preceded by an upper respiratory tract infection; (2) similarity of the subcutaneous nodules; and (3) the possible etiologic rôle of hemolytic streptococci in both.

At times, it is very difficult to distinguish between these diseases; the passage of considerable time may be required for a definite diagnosis.

Bayles found that 6 of 23 autopsied cases of rheumatoid arthritis showed changes in the heart valve leaflets and myocardium similar to those that usually follow rheumatic fever.[19,20] Others have found similar cardiac lesions in 16 of 30 autopsied cases of rheumatoid arthritis.[207]

Bennett presented his observations on autopsy findings in 48 cases of rheumatoid arthritis and 101 of rheumatic fever subjects.[23] He concluded that the usual anatomic changes observed in rheumatoid arthritis differed so markedly from those of rheumatic fever that one must infer that the pathogenesis of the observed lesions was different.

Baggenstoss and Rosenberg studied at necropsy the anatomic changes in the viscera of 30 patients with rheumatoid arthritis.[14] Rheumatic heart disease was present in 16 and other cardiac lesions in 8. They concluded that the high incidence of rheumatic cardiac lesions in this series was suggestive of a relationship between chronic rheumatoid arthritis and rheumatic fever.[13] The relationship of rheumatic heart disease to rheumatoid arthritis is discussed in a previous chapter (Visceral Lesions in Rheumatoid Arthritis).

Other observers found cardiac lesions at autopsy in 20 of 25 patients with chronic infectious arthritis; in 14 of these, the cardiac lesions were indistinguishable from those of rheumatic fever. In 10 of these cases, the rheumatic lesions seemed to be active and progressive at the time of death. Among the 14 patients with rheumatic cardiac lesions, there were 10 cases with mitral valvulitis, 5 with aortic valvulitis, 2 with tricuspid valvulitis and 5 with pericarditis. In each of the 3 subacute cases, typical Aschoff bodies were identified in the myocardium.[12]

	Rheumatic Fever vs.	Rheumatoid Arthritis[46]
Age incidence	Practically 90 per cent of cases begin under 15 yrs.	Practically 90 per cent of cases begin over 15 yrs.[72]
Permanent joint damage	Uncommon; almost never	Very frequent
Response to salicylates	Usually dramatic	Slight, only temporary relief
Electrocardiogram	May occasionally show evidence of cardiac involvement	Usually negative (unless patient is of middle age or above)
Involvement of pleura or lungs	Not uncommon	None
Roentgen-rays	Usually negative except soft tissue swelling	Characteristic; may be negative early; later generalized osteoporosis, etc.
Agglutination reaction with hemolytic streptococci	Usually negative	Strongly positive in most cases after several months
Antistreptolysin titer	Increased	Normal

PROGNOSIS OF RHEUMATIC FEVER

Recurrences are so numerous in rheumatic fever that they are *almost the rule.* The younger the patient the more likely he is to have recurrences, and the higher is the mortality. On the other hand, death rarely occurs during an acute attack of rheumatic fever; in a few cases, pancarditis, often associated with nephritis, leads to a fatal outcome in the acute phase of the disease. In at least one-third of the patients, permanent heart damage results.

Exacerbations of the disease are *frequently preceded by upper respiratory infections;* however, other factors which may provoke attacks include tonsillectomy, injections of typhoid vaccine, operation, injuries, emotional shock, high fever, vaccine[63] and scarlet fever.

The joint phenomena invariably disappear within several weeks or months. The cardiac damage, however, is permanent and is often progressive and increases with subsequent attacks. In general, greater cardiac damage occurs in younger individuals with rheumatic fever than in older patients.

Cohn and Lingg made a *statistical study of the natural history of rheumatic heart disease.*[62] They followed 3129 patients with rheumatic cardiac disease until death over a period of fifteen years. There was no important difference between the sexes in the age at onset. The mean age of onset for males was 14.5 and for females 15 years.

Rheumatic fever usually began in childhood in these cases, especially between the ages of five and ten years. The mean duration of the disease was about thirteen years. *Fifty per cent of the patients died within nine years after onset;* about 25 per cent lived more than seventeen years and *10 per cent lived for thirty or more years. When the disease began in childhood,* 69 per cent survived childhood, 35 per cent survived adolescence, 18 per cent reached the age of thirty and *5 per cent went on beyond forty-five years.* When the disease began in adolescence, 85 per cent survived this age period, 55 per cent reached thirty, and 21 per cent reached forty-six years or more. When the onset was in the twenties, 23 per cent (and when it was after thirty, 44 per cent) survived the age of forty-five.

Sixty-five per cent of all cases of rheumatic cardiac disease were found in childhood; only 16 per cent were found in the age group past forty-five years (of whom one-fifth were first afflicted after the age of forty-five).

In youth, the onset of the disease was characterized by polyarthritis in about half and by carditis or chorea in a third. In adults, the onset was with polyarthritis and with advancing age by valvular lesions alone.

Keith found the *chief manifestations of rheumatic fever in children* to be joint symptoms, chorea and nodules. The *nodule* was the least common, but *was associated with the most severe forms of the disease* as 99 per cent of those individuals also had heart disease, which was usually progressive.

Heart disease developed in about 80 per cent of rheumatic fever patients with joint phenomena, in 50 per cent of those with pure chorea, in 15 per cent of those with chorea but no other rheumatic manifestations, and in

70 per cent of those with chorea and other manifestations of rheumatic fever.

In individuals who had rheumatic manifestations but no heart disease, rheumatic heart disease developed subsequently in 25 per cent of children and in 5 per cent of adults. *Seventy-five per cent of children had one or more recurrences of rheumatic infection within five years of its onset when sulfanilamide prophylaxis was not used.* In 70 per cent of adults with rheumatic heart disease, the lesion was slowly progressive whether it was obviously or obscurely active.

In children with rheumatic heart disease, 4 per cent died in the first attack, 12 per cent in the first five years and from 20 to 30 per cent within ten years of the onset of heart disease. In the adult clinic patients, 40 per cent died in ten years and 75 per cent in twenty-three years. In about 10 per cent, clinical signs of rheumatic valvular disease disappeared over the subsequent ten years.

Factors which influenced the prognosis were progressive disease, increase in the size of the heart, progressive valvular damage, pericarditis, valvular lesions and nodules. In the past fifteen years, there has been a marked reduction in the mortality.[137]

Almost 50 per cent of all patients (and 85 per cent of children) with rheumatic fever have recurrences within a period of several years. **Evidences of activity of the disease** which should be watched for in patients who have had rheumatic fever include long-continued fever, muscle or joint pains, epistaxis, subcutaneous nodules, failure to gain weight or persistent underweight, pallor, increased sedimentation rate, leukocytosis, lassitude, tachycardia, abdominal pain, nausea and electrocardiographic findings. It must be remembered that **an active stage of the disease may be present with a normal temperature.** Any patient who is sick and has had rheumatic fever or rheumatic heart disease previously has reactivation until proved otherwise.

Important Signs of Activity of Rheumatic Fever in Children
Pallor.
Failure to gain weight.
Rapid sedimentation rate.
Leukocytosis.
Fever.
Symptoms: sore throat, joint pains, etc.
Cardiac murmurs.

Active carditis appears to be the only manifestation of rheumatic activity of prognostic significance.[275] Ungerleider has discussed the relation of heart size to prognosis.[260] In 246 children with active carditis, 36 per cent died and 50 per cent showed mitral stenosis with or without aortic insufficiency with at least moderately enlarged hearts; active carditis included any active rheumatic process in the heart, clinically recognizable. In another group of 427 children with rheumatic fever, active carditis was

never recognized; only 2 of these died (1 of subacute bacterial endocarditis and 1 during childbirth). Forty-three per cent of this group developed moderate cardiac enlargement and 30 per cent, mitral stenosis. Whether polyarthritis, chorea or growing and joint pains characterized the course caused no significant difference in the degree of cardiac involvement.

In view of the ameliorating effect of pregnancy on rheumatoid arthritis, it was interesting to observe that Coburn reported that *pregnancy seemed to protect rheumatic subjects against recrudescences of rheumatic activity after hemolytic streptococcal infections.*[52]

Boyer and Nadas have discussed *the ultimate effect of pregnancy on rheumatic heart disease.*[35] Hamilton and Thomson have surveyed the immediate risk of pregnancy in women with rheumatic heart disease.[102] It is agreed that the woman with rheumatic heart disease faces a *definite immediate risk in pregnancy.*[87,93,127,189,219] However, *if the mother survived single or multiple pregnancies,* Boyer and Nadas concluded from a statistical study that *the pregnancy had no delayed deleterious effect on the course of the rheumatic heart disease.* Postmortem data did not reveal that the increased load of pregnancies and motherhood produced any appreciable increase in cardiac hypertrophy. There was no evidence to show that multiple pregnancies tended to lower the cardiac efficiency permanently.

Hoffman and Jeffers studied *61 fatalities in rheumatic heart disease complicating pregnancy* and found that the fatalities resulted principally from congestive heart failure following delivery at or near term.[121] The most important factor amenable to control was the cardiac status of the patient at the time of delivery.

Wilson found a *mortality rate of 20 per cent in those with only one attack of active carditis, increasing to 64 per cent after four or more attacks.* The severity of active carditis has been found to be more important in prognosis than the severity of other rheumatic manifestations.

In one large series of rheumatic children, congestive failure occurred in 26 per cent; the mean duration of life following the attack of cardiac failure was 2.8 years. *After an attack of congestive failure the rheumatic child has less than one chance in three of living to twenty years of age.*

Stroud and Twaddle did not find that prolonged hospitalization materially decreased the death rate nor did it increase the number of patients who were able to lead active lives in later years; this conclusion was reached after reviewing fifteen years' experience at the Children's Heart Hospital of Philadelphia.[238]

Wilson found that of patients afflicted with rheumatic fever, less than 2 per cent died during the first year; 5 per cent by the fourth year; 10 per cent by the seventh year, and nearly 20 per cent had died by the eleventh year.

In about 33 per cent of children with rheumatic fever, cardiac damage was very slight; 67 per cent never had circulatory symptoms. Most of the children who survived puberty had no symptoms during adult life.

Coombs found the average expectation of life in rheumatic heart diseases to be as follows:

	100 patients who, when first seen, show definite signs of rheumatic heart disease	100 patients, including also those whose early signs are suspicious, but not definite
Recovery in	31	44
Death under 21 years in	28	19
Death under 31 years in	38	28
Death under 41 years in	50	39
Surviving 40 years, though crippled	19	17

Brown and Wolff recently reported that 50 per cent of 175 consecutive unselected cases of acute rheumatic fever (with or without chorea) escaped permanent organic valvular damage, even though half of these had multiple attacks of rheumatic fever.[40] Their observations did not substantiate the belief that permanent cardiac injury was more apt to develop in patients with severe infections or in those with a greater number of recurrences of rheumatic fever. In their series, those who recovered without permanent cardiac damage from a first attack had an excellent chance of doing so from subsequent attacks.

In a study of over 600 patients who died of *rheumatic carditis,* it was found[74] that the *average age of infection was seventeen years* and *that the period of freedom from symptoms was approximately eleven years after the infection;* during this period, the patient could carry on normal physical activity. Death occurred on an average of five years following this, or sixteen years following rheumatic infection. During the last three years of his life, the patient was almost completely a cardiac invalid. In this series it was uncommon to find even terminal stages of rheumatic heart disease after fifty years. More than 40 per cent developed auricular fibrillation. The *duration of life was not determined by the location or number of valves involved except that the prognosis was less favorable when the pulmonary or tricuspid valves were affected.* In 8 per cent of 100 young patients with rheumatic carditis observed for a period of ten years, total disappearance of clinical signs of carditis occurred.

Subacute bacterial endocarditis occurs in less than 5 per cent of patients with rheumatic fever; however, over 50 per cent of patients with subacute bacterial endocarditis have had rheumatic fever.

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CHAPTER XLIV

GONORRHEAL ARTHRITIS

In the Navy, about 2 per cent of those with gonorrhea developed gonorrheal arthritis. In one series, the gonococcus was responsible for about one-third of all of the cases of tenosynovitis.[30]

More than a million acute gonorrheal infections occur each year in the United States. Almost half a million persons with gonorrhea are constantly under observation and treatment in this country.[87] *From 1 to 5 per cent of these individuals developed gonorrheal arthritis*[34,72,92,93] prior to the institution of sulfonamide or penicillin therapy; it is expected that this number will be greatly decreased by the newer chemotherapeutic agents. Gonorrheal arthritis occurred in only 0.5 to 0.8 per cent of women with gonorrhea.

In view of the devastating nature of the disease if untreated (the marked bony destruction), and because of recent advances in the therapy of this disorder (sulfonamides and penicillin), it is important that every effort be made to diagnose it in the earliest stage.[2,19] **Treatment of gonorrheal arthritis is now a fairly simplified procedure; diagnosis is often faulty.**

DIAGNOSTIC FACTORS

1. *History.*
2. *Physical examination.*
3. *Laboratory tests* (smears and cultures for gonococci, gonococcus complement fixation test and roentgen examination).

Diagnostic Pointers in Gonorrheal Arthritis

History of gonorrhea (urethritis or involvement of the cervix, conjunctiva or rectum).
Onset of joint symptoms *usually* within ten to twenty days after primary gonorrheal infection.
Predominance in males (3:1).
Usually a *polyarthritis at the onset.*
Involvement of *knees*, *ankles* and *wrists* in order of frequency.
Frequent occurrence of *tenosynovitis* about the wrists and ankles.
Occasionally associated with conjunctivitis and iridocyclitis.
Gonococcus complement fixation test of blood positive in over 80 per cent of cases (if properly performed and repeated); may remain negative for the first four weeks of the disease.
Isolation of gonococci from joint fluid (by special culture methods).

Joints Most Commonly Involved in Gonorrheal Arthritis.—In large series of such cases, the order of frequency of involvement was: (1) *knee*—77 per cent; (2) *ankle*—38 per cent; (3) *metatarsophalangeal joints*—28 per cent;

(4) *wrists*—23 per cent; (5) *fingers*—22 per cent; (6) *shoulder*— 20 per cent; (7) *metacarpophalangeal joints*—17 per cent; (8) *toes*—14 per cent; (9) *other joints:* hip—10 per cent, elbow—8 per cent, sacro-iliac—6 per cent, intervertebral—5 per cent, heel—5 per cent, temporomandibular—2 per cent, sternoclavicular—2 per cent, olecranon bursa—0.7 per cent, acromioclavicular—0.7 per cent.[46,52]

Gonorrheal Arthritis Usually Affects Multiple Joints.—*In approximately 80 per cent of the cases, the disease is polyarticular* at its onset. This is not generally appreciated; unfortunately, the thought is prevalent that gonorrheal arthritis is a monarthritis. This is not so. It is true that in some cases, after an acute polyarticular onset, one joint may be affected much more severely than others and the disease may seem to persist particularly in one joint, such as a knee, ankle or wrist.

Incubation Period.—The disease most commonly begins from ten to twenty days after the onset of an acute gonorrheal urethritis (or at times a gonorrheal cervicitis, conjunctivitis or proctitis), although gonorrheal arthritis has been noted simultaneously with the appearance of an attack of urethritis; in other individuals, Neisserian arthritis has been reported occurring twenty years after the onset of an attack of gonorrhea, being induced in this instance by instrumentation or pelvic surgery.[36] In 80 per cent of several hundred cases of gonorrheal arthritis the joint manifestations appeared within the first five weeks after the primary infection.

Age Incidence.—Gonorrheal arthritis may occur at any age but is most common between the ages of twenty and forty. It has been reported in individuals seventy years of age.[44] The simultaneous occurrence of gonorrheal arthritis in a mother and newborn infant has been recorded,[37] but this is rare.[64] Gonorrheal arthritis is uncommon in infants, occurring in less than 2 per cent of children with gonorrheal vulvovaginitis.[28,85] In infants who develop a Neisserian arthritis following a gonorrheal ophthalmia, the former may occur between the fifth day and the fifth week of the disease, but most commonly during the second week.

In one instance, gonorrheal polyarthritis developed in a child of eight months shortly after it was given blood transfusions from its mother who had had arthritis for nine years (a sequel to gonorrhea). The child's synovial fluid contained gonococci.[23]

Gonococcic arthritis has been reported in a prematurely born infant; the organisms in this case probably entered *via* the gingivæ.[25]

Usual Clinical Picture.—An individual about thirty-five years of age contracts an *acute gonorrheal urethritis. Two or three weeks* later, acute *polyarticular arthritis* develops. There is a dull aching pain at the onset which is exaggerated by motion. With involvement of the synovial tissues, an *accumulation of synovial fluid* results; as much as 165 cc. of fluid may be aspirated from one of these joints.[58] *Pain* later becomes *severe* and may be excruciating so that the affected part may appear paralyzed because of the desire of the patient to splint it. There is *swelling, redness, tenderness* and *marked limitation of motion of several large joints,* such as the knees, ankles, elbows, etc. A marked *tenosynovitis* may be noted

about the ankles and wrists. The *temperature varies* between 100° and 104° F., and there is a moderate neutrophilic *leukocytosis* (up to 25,000) and a *rapid sedimentation rate.* As a general rule, the joint disturbance does not tend to become migratory unless the primary focus (urethra, etc.) is subjected to trauma. *After the first several days of the disease, the multiple joint phenomena become less intense and the process may become most marked in one of the affected joints.* Thus, the acute polyarthritis may develop into a stubborn monarthritis.

The acute joint affection may persist for several weeks or months if not properly treated. The surrounding muscles undergo marked atrophy, because of the immobility of the affected parts due to extreme pain on motion. The outcome of the disease depends upon the severity of the infection, the resistance of the patient, the promptness of adequate therapy, reinfection with gonococci and the ability to clear up the original focus of infection.

Any stage of joint involvement may be encountered from mild arthralgia to severe purulent arthritis with bony ankylosis. The disease may be acute, subacute or chronic. The acute joint involvement may be divided into several groups: (*a*) arthralgia of one or several joints; (*b*) acute synovitis; (*c*) acute arthritis with involvement of cartilage, synovial membrane, joint capsule and periarticular tissues with the presence of a serofibrinous exudate; or (*d*) purulent arthritis. A purulent gonorrheal arthritis may occur without the presence of organisms other than the gonococci within the joints.

Fever is seen in most patients with gonorrheal arthritis; it may be slight, persisting only a few days, or it may be marked (to 104° F.). In some of the patients, a prolonged fever varying from 99° to 103° F. may be observed. The febrile response in this disease is usually not markedly affected by salicylates.

Leukocytes range from normal to a marked elevation (25,000) with a predominance of neutrophiles (from 75 to 90 per cent). In some of the prolonged cases, secondary anemia is also a feature.

Laboratory Tests Available for the Diagnosis of Gonorrheal Arthritis.—Gonococcus complement fixation test.

Smears and cultures for gonococci (in joint fluid and in the original focus of infection).

Roentgen examination of the affected joints.

Skin tests(?).

Examination of the Joint Fluid.—The cell count of synovial fluid in gonorrheal arthritis may vary from 1600 to 236,000 per c.mm.; 45 to 100 per cent of the cells may be polymorphonuclear neutrophils. In general, in those effusions from which the gonococci can be isolated, the total cell count and neutrophil percentage is higher than in sterile fluids. In about one-fourth of these cases, gonococci may be isolated by culture. *Special cultural methods will reveal gonococci in many cases in which they were not observed in the stained smears.* One of the best available cultural methods for gonococci is that of McLeod which utilizes the oxydase reaction with

tetramethylparaphenylenediamine hydrochloride and the growth of the organisms in an atmosphere of carbon dioxide.[57] This method has been simplified by supplying carbon dioxide in jars for these cultures.[73,86]

For further identification of the gonococci, fermentation reactions and agglutination with antigonococcus diagnostic serum may be performed.

FIG. 179.—Gonorrheal arthritis of the wrist in a boy of twenty-one. There is very little motion in the wrist. The joint spaces between the carpal bones have been entirely destroyed; there is almost complete bony union of all these bones.

Gonococcus Complement Fixation Test.—If properly performed, this is one of the most useful aids in the diagnosis of gonorrheal arthritis. Unfortunately, the test cannot be done by an untrained technician and requires proper materials for its performance. If the reports of the test are routinely returned from a laboratory as negative in many cases of suspected gonorrheal arthritis, the test is probably not being performed properly or the reagents are ineffective. Carefully performed, *the test (if repeated) should be positive in at least 4 of every 5 cases of Neisserian arthritis* at some stage of the disease; in some clinics, a positive reaction has been obtained in 98 per cent of patients with gonorrheal arthritis.[56] Certain important points concerning the test are listed on the following page.

Roentgen Examinations in Gonorrheal Arthritis.—*There are no pathognomonic roentgen signs of gonorrheal arthritis;*[43] however, the roentgenologist may be of assistance in diagnosis and occasionally in treatment.[67] A *spotty ground glass atrophy* of the neighboring bones and *early acute localized atrophy* have been mentioned as suggestive of gonococcal disease.[50,63]

Early diffuse atrophy of bone, and *marked, rapid bony destruction* are often seen in roentgenograms in acute gonorrheal arthritis. Other changes include bony erosions, punched out appearance of the bone, osteolysis and chondrolysis, fibrous or bony ankylosis, and osteophytes or exostoses.

Gonococcus Complement Fixation Test

Usually negative for the first ten days of gonorrheal infection.

May remain negative for six weeks following infection; may never become positive if the case is mild or if there is a freely draining discharge (with little of the antigen absorbed). A localized chronic gonorrheal infection may never show a positive reaction.

If gonorrheal arthritis is suspected, the test should be repeated at least three times at intervals of ten days unless a positive result is obtained. *Three negative tests (ten days apart) in a patient with suspected gonorrheal arthritis throw grave doubt upon the diagnosis.*

A weakly positive test, followed in several weeks by a more strongly positive reaction is good evidence of gonorrhea.

The test may remain positive for several years following clinical recovery, although a *negative test is usually obtained within one-half to one and one-half years following clinical recovery.*

These patients may develop non-specific forms of arthritis.[89] Thus, arthritis plus a positive complement fixation test does not always indicate gonorrheal arthritis; the arthritis may be coincidental and non-gonorrheal.

Positive reactions are obtained more often in gonorrheal arthritis than in uncomplicated gonorrheal urethritis.

The test is negative in approximately 20 per cent of cases of gonorrheal arthritis; a positive test is thus of great diagnostic value; *a negative test does not rule out gonorrhea. Ten per cent of positive tests are false positives.*[29]

Positive reactions in patients whose histories are not consistent with a diagnosis of gonorrheal arthritis may not be significant.

The test may be persistently positive in less than 2 per cent of patients with rheumatoid arthritis; negative results are usually obtained in acute rheumatic fever, degenerative joint disease, gout and spondylitis.

Positive tests may occur as long as four months following injections of gonococcus vaccines in normal persons.

If the test is positive with synovial fluid, it is usually positive using the patient's blood; thus, it is unnecessary to perform this test upon the synovial fluid except when the blood gonococcus complement fixation test is negative and the clinical history is very suggestive of a gonococcal arthritis. Positive reactions may be obtained with blood in patients whose joint fluid is negative.

Skin Tests for Gonorrhea.—There is no general agreement concerning the value of skin tests with various products of the gonococci. Such tests are not considered valuable diagnostic aids at present. A gonococcus filtrate (Corbus-Ferry) has been introduced recently for diagnostic and therapeutic use in gonorrhea. It is a bouillon filtrate of the gonococcus which is said to contain the soluble specific gonococcus toxin. Normal individuals gave cutaneous reactions when injected with dilutions of this material as low as 1:1500. When this filtrate was detoxified by fractional

heating, intradermal injections of 0.1 cc. of this material gave a positive reaction only in gonorrheal subjects.[13] Ninety-eight per cent of 50 patients with gonorrhea demonstrated skin reactions averaging 2.3 cm. as compared to reactions averaging 0.2 cm. in control cases following skin tests with Corbus-Ferry filtrate.[12] Further work has been done with this material indicating that it is an aid in diagnosis of gonorrheal infections.[14]

GONORRHEAL TENOSYNOVITIS

Gonorrheal tenosynovitis is common in association with gonorrheal arthritis. In one series, it was noted in 48 per cent of the patients. *Tenosynovitis is more frequent in patients with gonococcic arthritis than in any other type of arthritis* and is a valuable diagnostic sign. Occasionally, the tenosynovitis is present without evidence of arthritis. The most commonly affected tendon sheaths are those of the extensors of the palm and wrist; other sites are the sheaths about the internal and external malleoli, the dorsum of the hands and feet, the long head of the biceps at the shoulder, and the radial and ulnar bursa at the wrist.

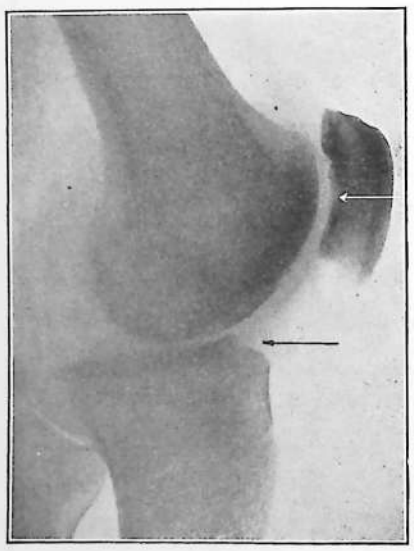

FIG. 180

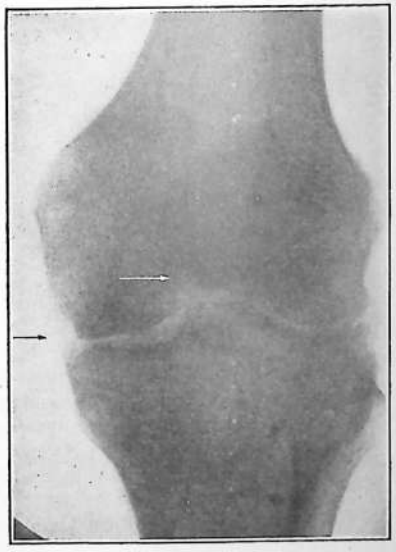

FIG. 181

FIG. 180.—Gonorrheal arthritis; lateral view. Areas of erosion indicated by the arrows. (Holmes and Ruggles.)

FIG. 181.—Gonorrheal arthritis, antero-posterior view. (Holmes and Ruggles.)

In acute gonorrheal tenosynovitis, the pus is greasy, gray and translucent.[30] *Suspect this diagnosis in any acute tenosynovitis with no history of antecedent trauma and especially if there is a focus of gonorrhea.* The diagnosis is made by smear or culture of the pus in the tendon sheath (aspirated by a syringe and hypodermic needle under aseptic technic).

The skin over the affected tendons is hot, tense and swollen, and severe pain occurs on movement. This may be acute or chronic. Systemic reaction is usually slight. *Acute suppurative gonococcic tenosynovitis is rare.*[4] The clinical manifestations are similar to those of ordinary pyogenic type of infection except that the tendon is usually not destroyed. Good results are obtained by splinting and adequate dosage of sulfonamides or penicillin.

SUPPURATIVE GONOCOCCIC MYOSITIS

Linner described a case of acute gonorrheal urethritis complicated by suppurative myositis and purulent arthritis.[54] *Suppurative gonococcic myositis* is an infrequent complication.[31,41,59,74] In the reported cases, the interval between the genital infection (which usually preceded the abscess) and the suppurative myositis varied from several weeks to several years. Primary multiple abscesses were less common than abscesses by direct extension from an infected joint. These abscesses occur most frequently in the thigh, calf, posterior axillary fold, psoas major, latissimus dorsi, posterior tibial and masseter muscles.[76,88,94] *Surgical incision and open drainage* with daily irrigations have been the most successful treatment to date.[71] In Linner's case, *daily doses of sulfathiazole did not prevent the development of suppurative myositis and arthritis.* These patients with suppurative myositis have not yet been treated with penicillin so that it is not known whether penicillin will be of value in this complication (suppurative myositis) or whether its use in the treatment of gonorrheal urethritis will prevent the occurrence of this complication.

Until the introduction of penicillin, the commonly accepted dosage of sulfonamide for the treatment of acute gonorrheal urethritis was 4 grams of sulfathiazole or sulfadiazine daily (in divided doses) for five to seven days.

It is of interest that penicillin given intravenously did not cure the gonorrheal arthritis in Linner's case although 10,000 units was given every three hours intravenously for a total of 16 doses. There was considerable improvement in the knee joint with a resulting normal temperature, but there was a recrudescence of the knee infection in a week with gonococci found in the thin yellow pus obtained at aspiration at this time. Following the *daily injection* (for three days) of 8700 units of *penicillin* in 15 cc. of sterile physiologic saline solution into the knee joint, the affected knee slowly decreased in size and the patient was apparently cured.

ASSOCIATED FEATURES IN GONORRHEAL ARTHRITIS

Conjunctivitis is more common in gonorrheal arthritis than in other forms of joint lesions; (it may occur also in postdysenteric arthritis, Reiter's disease and rarely in acute rheumatic fever). Ocular signs may assist in diagnosis; these may be found in from 10 to 20 per cent of cases of gonococcal arthritis. The most common is a *catarrhal conjunctivitis* which may occur simultaneously with or may precede the joint phenomena. This conjunctival exudate is sterile. The ocular lesion may disappear before the onset of the arthritis and usually results in no permanent damage. In some

cases, one observes an *irido-cyclitis*, which is a serious complication; permanent impairment of vision may result. The latter complication is found in approximately 3 per cent of cases of gonorrheal arthritis, while a conjunctivitis may be noted in as many as 15 per cent.

FIG. 182

FIG. 183

FIG. 182.—Lesions on the sole of a foot in a patient with *Keratodermia blennorrhagicum*.
FIG. 183.—Penile lesions in a patient with *Keratodermia blennorrhagicum*. This is an uncommon form of skin eruption occurring in about 3 per cent of patients with gonorrheal arthritis. The lesions are most common on the soles; they are composed of thickened skin with sharply demarcated edges; necrosis or sloughing may occur in the center of the lesion. This young man's skin lesions and gonorrheal arthritis cleared promptly following the institution of sulfonamide therapy.

Keratodermia blennorrhagicum, a rare form of skin eruption, has been seen in approximately 3 per cent of patients with gonorrheal arthritis.[11] The lesions, which are most common upon the plantar surfaces of the feet, may also occur on the glans penis. They are composed of thickened skin

with sharply demarcated edges; necrosis or sloughing may occur in the center of the lesion.

	Gonorrheal arthritis	Rheumatic fever	Rheumatoid arthritis
Complement fixation test (Gc)	Positive in over 80 per cent of cases if repeated every 10 days for 3 examinations	Almost always negative	Usually negative.
Synovial fluid	Gonococci demonstrable by smear or culture (in at least 25 per cent of cases)	No gonococci	No gonococci.
Electrocardiogram	Usually no changes	Evidence of cardiac involvement in almost every case if serial examinations are made	Usually negative.
Response to salicylates	Not dramatic; some relief of pain and some reduction of temperature	*Usually dramatic* with complete relief of pain and return of temperature to normal if adequate doses are used	Temporary relief, but no dramatic response.
Involvement of pleura or lungs	None	Not uncommon	None.
Presence of serous conjunctivitis	In about 20 per cent of cases	Very uncommon	Absent.
Response to sulfonamides or penicillin	Excellent	Very poor or none; may be made worse in acute stage by sulfonamides	Very poor or none.
Response to fever therapy	Usually dramatic	May also be dramatic (in some cases)	May be dramatic in only about 10 per cent of cases.
Preceding upper respiratory infection	In about 10 per cent	In most cases	Frequent.
Roentgen-rays	May show rapid bony destruction	*Usually negative except for soft tissue swelling*	*Characteristic:* may be negative early; later generalized osteoporosis, etc.
Permanent joint damage	May be marked	*Usually none*	May occur.
Age at onset	Most often 20 to 40 years; may occur at any age	*First attack under 15 years in 90 per cent;* may occur at any age	*90 per cent of cases after age of 15.*[16]
History of gonorrhea	*Usually within several weeks preceding joint pains*	Usually negative	Usually negative; possibly coincidental
Agglutination reaction with hemolytic streptococci	Usually negative	Usually negative	*Strongly positive in most cases.*
Antistreptolysin titer	Normal	Increased	Normal.
Subcutaneous nodules	Absent	Present in about 20 per cent of cases	Present in about 20 per cent of cases; may persist for weeks.
Tenosynovitis	Present *in almost 50 per cent of cases*	Usually absent	Usually absent.

The clinical features of keratosis blennorrhagica are *gonorrhea, polyarthritis* and *cutaneous keratosis*. This occurs once in from 5,000 to 7500 cases of gonorrheal urethritis and not more than 100 such cases have been reported.[11,70] *The joint involvement usually precedes the skin lesions.* In most instances, there is simultaneous involvement of several joints (especially the knees, ankles, wrists and acromioclavicular joints). There is usually severe joint pain.

Acute hemorrhagic glomerulonephritis is also a rare complication.

Cutaneous Manifestations.—Keil[48] and Lichtman[53] have emphasized the *frequency* and *diagnostic significance of cutaneous manifestations* which may accompany gonorrheal arthritis. These include *petechiæ* and *small red sore spots* (at times with a central white necrotic region) and *hemorrhagic purulent vesicles* or bullæ, surrounded by an erythematous areola.

Frequency of Bacteremia in Gonorrheal Arthritis.—Gonococci have been recovered from the blood stream in 4 per cent of these patients; the mortality in the group with bacteremia approximated 60 per cent in the era before the use of sulfonamides and penicillin. In about 40 per cent of the patients with bacteremia, endocarditis is present.

One of the striking features of patients with such bacteremia is the maculopapular eruption, which may become hemorrhagic, vesicular and then pustular. This appears on the trunk but is more intense on the extremities. Enlargement of the spleen and liver, jaundice and thrombocytopenia have been seen occasionally in these individuals.

Gonococcus Carriers.—Carpenter and Westphal studied the problem of the **gonococcus carrier** and showed conclusively that *a person may have a low-grade carrier state for years which is difficult to overcome;* such a focus for the possible development of gonorrheal arthritis can smolder indefinitely.[8]

Spink and Keefer have shown that *gonorrheal arthritis may occur without any symptoms referable to the genito-urinary tract.*[82] They have reported several cases of gonorrheal arthritis as a complication of *pregnancy;* the latter *may cause an increase in the severity of a latent gonorrheal infection.* One patient whose history included a gonorrheal urethritis at the age of twenty, developed an acute exacerbation of his gonorrheal arthritis fifty years later, following a prostatectomy.[24] In 70 patients with gonorrheal arthritis, 13 instances of acute polyarthritis were observed without symptomatic evidence of localized gonococcic infections of the genito-urinary tract. Because of a preceding acute upper respiratory infection in several of these, acute rheumatic fever was at first considered. Of these patients, 10 were women who knew of no gonococcal infection before their admission to the hospital.

DIFFERENTIAL DIAGNOSIS

Acute gonorrheal arthritis is *often confused in its early stage with rheumatic fever*, as the early differentiation between the two is difficult in some cases. *A sudden polyarthritis following an upper respiratory infection is most often rheumatic fever;* however, if the patient does not improve with adequate doses of salicylates, the joint manifestations may be gonorrheal in nature. If there is any question concerning the diagnosis, fluid should be aspirated from the affected joints and cultured for gonococci.

Spur Formation on the Os Calcis May or May Not be Due to Gonorrheal Infection.—Teaching some years ago led one to believe that spur formation on the os calcis was usually the result of gonorrheal infection. This has been shown to be untrue.[35] *Gonorrheal spurs are uncommon*, having been seen in less than 10 per cent of cases of gonorrheal arthritis. Several other popular misconceptions may be mentioned. It was taught that involvement of the spine, sternoclavicular joints and temporomandibular joints by gonorrhea was more common than by other forms of arthritis. As a matter of fact, gonorrheal spondylitis is uncommon; the sternoclavicular joint is involved in less than 3 per cent of cases of gonorrheal arthritis,[33] and the temporomandibular in only 2 per cent of cases.

TREATMENT OF GONORRHEAL ARTHRITIS

Recent innovations have revolutionized the treatment of gonorrheal arthritis. By the use of penicillin or sulfonamides most cases of gonorrheal arthritis may be controlled very promptly.[10,55] However, one must remember that auxiliary treatment is of importance; this includes rest of the affected parts, splinting, sedation, physical therapy, etc.

Treatment of Gonorrheal Arthritis
Rest of the affected joints by splints and prevention of deformity during the acute phase of the disease.
Penicillin and/or *sulfonamides*.
Analgesics (aspirin, codeine and morphine if necessary).
Aspiration of affected joints and introduction of penicillin into the joint cavity.
General care of the patient, careful follow-up physical therapy.

Rest of the Affected Joints and Prevention of Deformity.—The affected joints should be placed at absolute rest as motion is extremely painful. In some cases this may be accomplished by cotton padding and wood or pillow splints. When possible, one may use Castex or plaster splints. These are well padded with cotton so that no pressure points exist. The splints are held in position by adhesive strapping, light gauze bandage or tapes.

The patient notices relief of local pain as soon as splints are applied because of the absence of motion of these joints. Such splints should be used day and night if possible, removing them once each day; at this time very gentle passive movements of each affected joint are performed through the full range of painless motion. Adhesions which form within twenty-four hours can be broken up easily by gentle passive motion. Some individuals feel that they can be broken up if the splints are left in place more than twenty-four hours; however, it is safer to remove these daily to permit joint motion and inspection of the affected parts.

Penicillin and Sulfonamides.—Oard and his associates[62] used penicillin sodium in the treatment of 411 patients with gonorrheal urethritis. It was found that *sulfathiazole and penicillin appeared to enhance the effect of each other against the gonococcus.* It was found that the use of moderate amounts of sulfathiazole and of penicillin was a safe, rapid, efficient and economical method of treating gonorrheal urethritis. They prescribed 8 gm. of sulfathiazole on the first day (in divided doses) and 4 gm. of the drug on the second day in divided doses. On this second day, 10,000 units of penicillin was given intramuscularly every three hours for a total of 5 injections. The treatment failed to clear the gonorrhea in 8.5 per cent of the white patients and in 2.5 per cent of the colored. However, it has been shown that **in almost all cases in which a total of 80,000 units of penicillin was given, the symptoms and urethral discharge cleared and bacteriologic cure resulted within twenty-four hours or less** (although a slight mucoid discharge persisted for a few days). **When a total of 100,000 units of penicillin was used, all patients were promptly cured by penicillin alone.**

All patients with gonorrheal arthritis should be given (at once) adequate dosage of sulfonamides and/or penicillin (unless the patient has been found previously to be sensitive to any of the sulfonamide preparations, or unless there is evidence of renal damage or other contraindication to its use). A full discussion of this subject, including dose, precautions, reactions, etc., is presented later. (See Sulfonamides and Penicillin.)

Fever Therapy in Gonorrheal Arthritis.[21,77,83]—It has long been known that the occurrence of some intercurrent febrile disease might result in a cure of the primary ailment. It has been shown that high temperatures exert a bactericidal and bacteriostatic effect on certain germs *in vitro.*

Raising the body temperature to 105° or 106° F. for periods of from five to ten or more hours is a serious undertaking and requires utmost vigilance. Such a procedure is not an office treatment. Because of the severity of the procedure, this should be employed only by individuals with special training in this field.

Among the methods used for administering fever by physical means are radiant or luminous heat cabinets, short wave diathermy cabinets, electric blankets, sleeping bags, hydrotherapy, hot water baths, blankets, etc. The Council for Physical Therapy has felt that the most acceptable device is the cabinet type, so arranged that the patient may be observed during his treatment, and so equipped that rectal temperatures may be read conveniently.[20]

Artificial fever is of marked benefit in the treatment of gonorrheal arthritis. Cures have been reported in from 50 to 100 per cent of the cases with relief in over 90 per cent. The introduction of the sulfonamides and penicillin has replaced fever therapy in this disease except in a very few patients who may not respond promptly to such therapy.

The air conditioned chamber known as the *Kettering Hypertherm* is generally agreed to be one of the most effective forms of physical apparatus for fever therapy. With it, any desired degree of fever may be maintained while the patient's body is free within the chamber, the head being outside.

Fever is induced by a simple air heater to raise the temperature of the air within the chamber; an equally simple means humidifies this air to any desired degree.[17] This air is circulated around the patient ten times per minute. Thermostatic control of the temperature is maintained as well as humidostatic control of the relative humidity. In the case of any emergency, the patient can be removed from the apparatus within several seconds. The patient lies upon an air mattress, with his head resting upon a pillow outside the cabinet. The electrical apparatus which heats, humidifies and circulates the air is placed in a compartment in the foot of the cabinet. This cabinet is approximately 6 feet long, 30 inches high and 36 inches wide.

The patient is usually given a sedative such as sodium amytal, 0.4 gm. (gr. vi) before entering the cabinet. The air temperature in the cabinet which approximates 125° F. at the onset of treatment is raised as rapidly as possible to 160° F., the relative humidity being maintained at from 50 to 80 per cent.[84] When the desired body temperature is produced (after about one and one-half hours), the patient is covered with a blanket and the cabinet air temperature is adjusted to maintain the desired fever. During the ordeal an electric fan blows on the patient's face and ice water or saline is given in large quantities. Hypodermics of morphine may be required, because of marked distress or restlessness. A trained technician must be in constant supervision as the patient's T.P.R. and the air temperature and humidity are checked at least every fifteen minutes. Following the treatment, the temperature of the body returns to normal (or its previous level) within one or two hours.

Among the *physiologic reactions produced by fever therapy* are:[40] increased pulse rate, circulatory rate and cardiac output; no marked change in blood volume; marked drop in chlorides (unless supplied during the treatment) with the loss of from 18 to 26 gm. of sodium chloride in from 3 to 4 liters of sweat. *Contraindications to fever therapy include* old age; cardiac, vascular or renal disease; chronic alcoholism; pulmonary tuberculosis; extreme weakness or emotional instability.

Complications may also occur *following fever therapy.* These include burns, erythema, nausea, vomiting, diarrhea, headache, herpes labialis, dehydration, tetany, circulatory failure, auricular fibrillation, heat stroke, hematemesis, shock, epileptiform convulsions, delirium and death. Most patients tolerate the fever very well.

The aim of this treatment is to destroy the gonococci by subjecting them to a sufficiently high temperature for an adequate length of time. Strains of gonococci vary in their ability to withstand degrees of heat; some can withstand the same degree of heat for longer periods than others. Inasmuch as gonococci are generally killed by a temperature of 41.5° C. (106.7° F.) in from five to seventeen hours, it is generally agreed that the main aim of fever therapy in gonorrheal arthritis is to sterilize the joints by a direct bacteriolytic effect.[32] The thermal death time of 130 strains of gonococci *in vitro* at 106° to 107° F. is about six to twenty-seven hours.[7,90]

More than one strain of gonococci may infect an individual. Strains of gonococci obtained from the patient and his consort usually have the same

thermal death time. Ninety-nine per cent of gonococci cannot withstand a temperature of 106° F. for more than five hours. The other 1 per cent are destroyed only by the use of high temperatures over prolonged periods of time (41.5° C. or 106.7° F. for from five to twenty hours). Fever has been given in several sessions (2 to 6) of five to six hours each at a temperature of from 106° to 107° F. In some cases, if the thermal death time of the strain of gonococci present can be determined, one long session of fever (from five to seventeen hours as may be required) equivalent to the thermal death time of this strain, has been suggested. Some workers have substituted one ten-hour session of fever for the four or five shorter sessions of from five to six hours each.[51] By thus lengthening the initial session from five to ten hours, it has been possible to decrease the number of sessions from an average of almost five per person to slightly over one. As a result of this routine (ten-hour fever session), over 95 per cent of a series of 84 patients with gonorrhea were dismissed as clinically cured.

Some strains of gonococci can tolerate a higher temperature than can the individual; fever therapy fails in these instances. Fever therapy may cure gonorrheal infection of joints, urethra, uterus, or tubes, but infection of Skene's glands with the gonococcus is resistant to such treatment; it is sometimes necessary to cauterize these structures.[18]

In some resistant cases, fever and sulfonamide therapy have been combined.[38,39,49]

It is likely that **penicillin** will replace all other therapy in gonococcal infections except the **sulfonamides.**

Results of Fever Therapy.—Eighty to 95 per cent of patients with acute gonorrheal arthritis may be clinically cured by fever therapy.[60,61,79] In some cases of chronic gonorrheal arthritis (*i. e.*, more than six weeks' duration) the results are only about half as good. Because of their simplicity (in most instances), penicillin and/or sulfonamides have now supplanted fever therapy in gonorrheal arthritis.

Criteria of Cure of Gonorrhea.—Spontaneous remissions have been reported in cases of gonorrheal arthritis without specific treatment. Recovery has also been observed in patients with blood stream invasion by the gonococcus with symptomatic treatment only.[1] The following points must be observed before a cure of genito-urinary gonorrhea can be accepted: cessation of the disease clinically; clear urine without shreds, or absence of gonococci in these shreds if present; several negative smears and cultures for gonococci; a normal prostate whose secretion contains no excess of leukocytes; absence of adnexal tenderness; no cervical irritation in women, no urethral flare-up following alcohol, sexual excitement, instrumentation or menses.[37]

Roentgen Therapy in Gonorrheal Arthritis.—Kahlmeter, of Stockholm, obtained recoveries in at least 90 per cent of these patients by roentgen therapy in the early stage of the disease.[42] Others felt that roentgen therapy was equal or superior to fever therapy in these individuals, in addition to its simplicity of performance without exhausting the patient;[26] still others were not impressed with its results.

Fever Therapy in Gonorrheal Arthritis[29,68,69,80]

Replaced now (almost completely) by penicillin and/or sulfonamides.

Method of choice for production of fever: Kettering Hypertherm; other methods: short wave diathermy,[3] general radiothermy, hot water baths, heated air currents, etc.

Thermal death rate of 130 strains of gonococci *in vitro* at 106° to 107° F. varies from six to twenty-seven hours;[5] a patient may be infected with several strains of gonococci.

Dose: ten hours of fever (at one session) 106° to 107° F.; repeat only if necessary (usually not more than one such session is required); with former method (five to six hours at a session) four or five sessions were usually needed for clinical cure.

Clinical cure results from properly given fever therapy in from 80 to 95 per cent of patients with acute gonorrheal arthritis, and in about one-half this number in chronic gonorrheal arthritis (of over six weeks' standing).

Should be given only in a hospital under the careful supervision of specially trained individuals; one of the latter must be present constantly during the entire therapy.

Cure is due to direct bactericidal effect of high temperature upon gonococci with (probably) some assistance of defense factors within the body.

Complications of fever therapy include death. Because of the marked sweating, large amounts of sodium chloride are lost during treatment (which must be supplied during the period of fever).

The *treatment* advocated by Kahlmeter *in cases of acute gonorrheal arthritis* included single doses each of from 75 to 100 r, repeated three to four times at intervals of from two to three days. The presence of pus in the joints is said to be a contraindication to roentgen-ray treatment.[78] The use of penicillin and/or sulfonamides has supplanted roentgen therapy in this disease.

Treatment of Primary Gonorrheal Focus.—This is beyond the scope of this work.[65,66] However, attention must be called to the successful treatment of most forms of gonococcic involvement with sulfonamides or penicillin.

Analgesics.—Relief of pain in mild cases may be obtained with 0.3 to 0.6 gm. (5 or 10 gr.) of aspirin every three or four hours as required. In the more severe cases one may need 30 mg. ($\frac{1}{2}$ gr.) of codeine phosphate or 10 to 15 mg. ($\frac{1}{6}$ to $\frac{1}{4}$ gr.) of morphine sulfate or dilaudid hydrochloride 2 to 3 mg. ($\frac{1}{32}$ to $\frac{1}{20}$ gr.). The latter does not produce as much gastric distress as occasionally occurs with morphine; in addition, it is not so habit forming as morphine. If the pain is so severe that the patient requires morphine for relief (although the part has been placed at physiologic rest) this drug should be given.

Aspiration of the Affected Joints.—Aspiration of the joints may be done to relieve pain, to protect the intra-articular tissues from contents which would destroy cartilage, for diagnostic purposes, to reduce the formation of adhesions, to prevent undue stretching of the joint capsule and prior to the introduction of penicillin directly into the joint space.

General Care of the Patient.—The patient should be kept at rest in bed until several days after the temperature has subsided and the joint phenomena have greatly improved. During the febrile stage, a liquid diet may be required. As soon as the temperature has returned to normal, nourishment should be forced.

If the individual is anemic, iron is given by mouth (such as ferrous sulfate, 0.2 gm. [3 gr.], four times per day); transfusions may improve the general condition but are seldom needed. In adults, 400 or 500 cc. of blood may be given every several days if required.

After the acute stage of the disease has passed, physical therapy (including heat, massage, baking, baths, etc.) will be required (see Physical Therapy). The responsibility of the physician does not cease at the end of the period of penicillin or sulfonamide administration. Proper support for the affected joints, rest, heat, exercise and massage must be prescribed as required. The progress of the infection may be followed by means of the sedimentation rate.

It is of interest to note that Culp reported that mercurochrome given intravenously compared favorably with sulfanilamide in the treatment of gonorrheal arthritis.[15]

If weight-bearing joints are affected, walking should not be permitted until all evidence of inflammation in the joints has subsided.

PATHOLOGY OF GONORRHEAL ARTHRITIS

The pathologic process in gonorrheal arthritis is *similar to that in other pyogenic forms of arthritis.*[27] One sees thickening, edema and infiltration of the synovia with polymorphonuclear leukocytes in the early stages, while later there is a production of fibroblasts, capillaries and infiltration with neutrophils and plasma cells. If the disease progresses to an advanced stage, synovial fibrosis occurs.

Depending upon the virulence of the organisms, their numbers and the resistance of the host, *various degrees of joint damage* may occur. These may range *from a mild arthralgia to an acute purulent arthritis; inflammatory reactions in tendons and muscles* have not been infrequent.

Postmortem examinations have been made upon joints which were the seat of an acute gonorrheal arthritis at the time of death of the patient.[45] In one of these in which gonococci were found in the joint fluid, the synovial membrane was completely destroyed and was replaced by granulation tissue which contained neutrophiles, lymphocytes, macrophages and plasma cells; a perivascular infiltration of lymphocytes was noted in the deeper layers of the synovia. Such inflammatory lesions in the synovial membrane appear to be much less intense in gonorrheal arthritis with sterile synovial fluid.

The *degree of destruction of articular cartilage varies markedly* in gonorrheal arthritis. It appears to be greatest in the wrists, phalanges, hips and ankle joints. The lessened damage in the knee joint may be due in part to the large amount of fluid containing antitryptic substances which may collect

there. The articular cartilage of the knee joint may be protected from the tryptic activity of the white blood cells (in their attempt to digest cartilage) by the antitryptic content of the synovial fluid. *Destruction of cartilage is most marked when the exudate is thick and purulent;* such an exudate contains a large number of leukocytes and a relatively small amount of synovial fluid.

Ankylosis is not uncommon in improperly treated cases of gonorrheal arthritis. This results from periarticular fibrosis or adhesions between the articular cartilages. Adhesions between the patella and the femur result in stiffness of the knee joint. True bony ankylosis is uncommon except in the wrists.

Synovial fluids infected with gonococci do not show active bactericidal antibodies but the blood from such persons is frequently bactericidal for the same organism.[81] However, sterile synovial fluid in cases of gonorrheal arthritis is actively bactericidal for the homologous strain of gonococci due to the presence of antibody and complement.

The presence of mucin in synovial fluid may aid some strains of gonococci in surviving, particularly when the antibody content of the fluid is low. Mucin may be a factor in favoring the localization and survival of these organisms in the synovial fluid and tendon sheaths[47] as *gonococci seem to localize and survive in areas containing large amounts of mucin* (urethra, cervix, conjunctiva, joints, etc.).

Although gonococci may be obtained by culture (special methods, *q. v.*) from the joint fluid in about one-fourth of the cases of gonorrheal arthritis, demonstration of the offending organisms in the blood stream in these cases is very infrequent.[47]

A chemically defined medium has been described for the cultivation of the gonococcus.[91]

Antibodies appear in the blood during the course of a gonococcic infection. The bacteriolytic titer of the blood increases during the course of the disease in patients with gonorrheal arthritis.

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CHAPTER XLV

MISCELLANEOUS FORMS OF ARTHRITIS

PNEUMOCOCCAL ARTHRITIS

PNEUMOCOCCAL arthritis occurs infrequently; it is still less common since the use of sulfonamides and penicillin in the treatment of pneumonia. When pneumococcal arthritis occurs, it usually responds promptly to adequate sulfonamide and/or penicillin therapy.

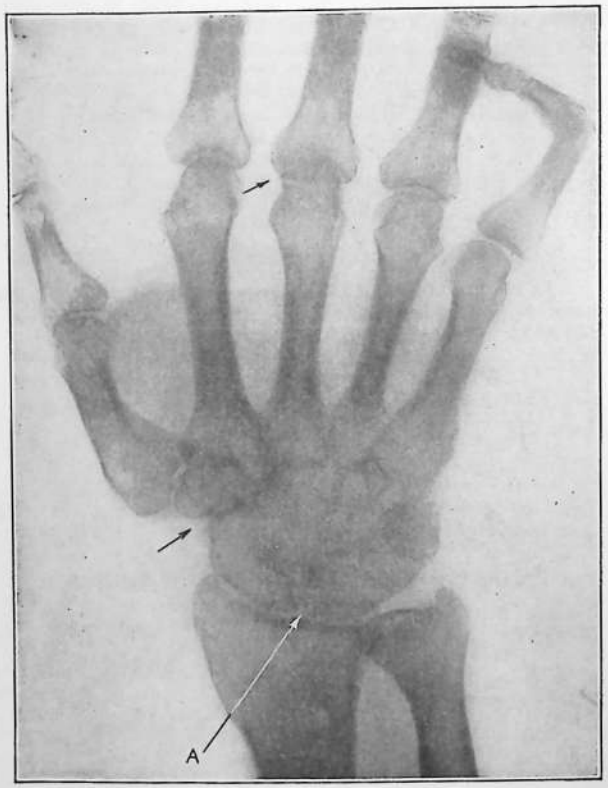

FIG. 184.—Infectious arthritis. Note the loss of articular cartilage at points indicated by the arrows and about all of the carpal bones. Characteristic area of erosion at *A*. General rarefaction of the carpus and articular ends of the long bones. (Holmes and Ruggles.)

In a series of 1298 patients with lobar pneumonia, arthritis was so uncommon that it was not mentioned as a complication of the disease.[197] Only 2 cases of pneumococcal arthritis were seen among 1800 patients with pneumonia in twenty-five years at the Hospital of the Rockefeller

Institute.[42] Others found arthritis in from 5 to 6.5 per cent of patients with lobar pneumonia and considered it a fairly common complication in pneumonia.[124,188,254] Few cases of primary pneumococcal arthritis have been reported.[27] In one series of 185 collected cases of pneumococcal arthritis, this was primary in only 26 cases.[207] In Guy's Hospital in London, only 7 cases of pneumococcal arthritis were found in a period of fifteen years. A review of the literature on this form of arthritis is presented by Fagge.[65]

Important Points in Pneumococcal Arthritis[113]

Incidence varies from 0.1 to 6.5 per cent of patients with pneumonia; more than 75 per cent of cases are associated with pulmonary infection.

Primary pneumococcal arthritis seldom occurs.

Order of frequency of involved joints is: knees, shoulders, wrists, elbows. Children are more frequently affected than adults.

Polyarticular involvement occurs *in only 25 per cent* of cases.[33]

Pathology: Usually a *suppurative arthritis*, involving one joint only in most cases; pneumococci can usually be recovered from the joint fluid. The process is occasionally non-suppurative. It usually occurs after the stage of resolution of the pneumonia. Mild arthralgia occurs occasionally.

Prognosis—good if the patient survives the primary pulmonary infection. Joint function is usually preserved without ankylosis.

Diagnosis is made by the occurrence of an acute arthritis in a patient with pneumonia or pneumococcic septicemia, or by finding the organisms in the joint fluid in an acute arthritis.

Treatment: Immobilization of the affected joint with molded plaster splints (removed once daily for motion of the joint if possible). Sulfonamides or penicillin should be administered; see chapters on Sulfonamides and Penicillin for dose, administration, contraindications, reactions, etc.

Adequate support to the affected joints, heat, massage and exercises following subsidence of the acute infection. If a purulent collection forms within the joint, this should be aspirated early. Open surgical drainage is usually unnecessary now with the use of sulfonamides or penicillin. In some cases it is advisable to inject penicillin directly into the affected joints daily for two or three days.

Blankenhorn and Grupen reported 11 cases of **pneumococcic pyarthrosis,** in all of which the pneumococcus was recovered either from the blood or from the joint; in 7 cases, the organism was recovered from both.[25] The diagnosis was usually made by the appearance of an *acute, painful involvement of one or two large joints.* Occasionally, pneumonia is not even suspected in these patients. The appearance of the affected joints may be similar to gonorrheal arthritis and the differential diagnosis is made mainly by easily growing and identifying the pneumococcus (before sulfonamides are begun). *None of these 11 patients showed a migrating polyarthritis.* Roentgen examination usually showed only slight demineralization of bone and soft tissue swelling.

In *8 of the 11 cases, a single joint was involved* and in the other 3 cases,

only two joints were involved; none of the cases showed involvement of more than two joints. The most commonly affected joints were the knee and ankle.

This group of patients was treated by conservative measures, including *sulfonamides*, serum and repeated aspiration. *Surgical drainage was found unnecessary.* The joints were aspirated for diagnosis as soon as the disease was suspected and repeated tappings were done to provide drainage as often as fluid reaccumulated. The joints were immobilized with splints as long as fever was present. Sulfonamides were given orally and serum was added to the therapy if the patient was not doing well on sulfonamide alone, or if there was a positive blood culture late in the course of the pneumonia. All of the patients who received sulfonamides with or without serum recovered with excellent functional results.

JOINT INVOLVEMENT IN UNDULANT (MALTA) FEVER (BRUCELLOSIS)

Brucellosis frequently exhibits joint pains, pains in the limbs and "rheumatic pains." Arthralgia is common in brucellosis but actual arthritis is rare; in occasional cases, the joint symptoms may resemble those of rheumatic fever or rheumatoid arthritis.[112] Hydrarthrosis occurs occasionally.

BRUCELLOSIS

Green and Freyberg studied the incidence of brucellosis in 25 patients with rheumatoid arthritis and in 25 patients with other rheumatic disorders.[94] None of the patients with rheumatoid arthritis had evidence which was diagnostic of brucellosis. In the second group, 3 had convincing evidence of active brucellosis and 6 others had "possible brucellosis." They concluded that *although arthralgia and other rheumatic symptoms are common in brucellosis* and in some cases non-purulent joint inflammation occurs as a temporary phenomenon, *brucellosis is seldom, if ever a cause of chronic non-purulent joint inflammation.*

More than 50 per cent of patients with brucellosis may have rheumatic complaints such as myalgia, arthralgia, non-suppurative or suppurative arthritis, spondylitis, osteomyelitis or osteoperiostitis.[118] In some cases (up to 10 per cent) joint symptoms may appear as the chief complaint.[242]

Arthralgia was reported in 32 per cent of one series of patients with brucellosis, but evidence of joint inflammation was found in less than 2 per cent.[100] The incidence and prominence of joint pains is greater in California where the bovine strain of Brucella is the predominant type than in Iowa where the porcine strain predominates.[118,257] The bovine strain causes actual joint inflammation only rarely while the porcine strain produces arthritis more often (but not as commonly as the melitensis strain).[100]

Diagnosis of Undulant Fever.—In most instances, chronic brucellosis is diagnosed by the history, clinical findings, specific laboratory tests for brucellosis and exclusion of other diseases. The diagnosis may be suggested

by a history of a possible source of infection (such as a laboratory worker, drinking unpasteurized milk), recurrences of wave-like *bouts of fever*, loss of weight, *marked sweating*, weakness, general malaise, arthralgia, headache, depression, secondary anemia, leukopenia with relative lymphocytosis or normal leukocyte count, positive agglutination test,[20,257] positive intradermal test,[15,62,103] cultivation of organisms from blood[143,277] or joints, opsonophagocytic test[71,87,129] and exclusion of other diseases.[45] *Because of the multiplicity of symptoms and paucity of physical signs, the disease is often overlooked or is labeled neurasthenia.*[63] Occasional cases may resemble pulmonary tuberculosis[162,167,269] or encephalitis.[175] Brucellosis should be suspected in any long continued, undiagnosed illness, especially if fever is present.[10]

In patients in whom the diagnosis is strongly suspected, complete tests should be performed for brucellosis. Available tests include *Br. abortus agglutination test*, an *intradermal test with brucella antigen*,[185] an *opsonophagocytic index* and *blood culture;* in some instances the organisms have been obtained by *culture of joint fluid.* Chronic cases may show an agglutination no higher than a 1:25 dilution (or less).

Laboratory tests for brucellosis are frequently confusing and may be misleading and permit different interpretation. The most reliable is the *agglutination test*, but this is not infallible. In one series, a negative agglutination test (titer less than 1:40) was found in 46 per cent of patients with chronic brucellosis.[64] It must also be remembered that the titer may remain high after recovery from the disease.

There is no general agreement as to how strong a Brucella abortus agglutination must be before the condition is considered active. Most clinicians required a positive reaction in a titer of 1:80 or more as evidence of active infection. The agglutination reaction is usually negative in those patients with positive skin tests to brucella antigen. The opsonophagocytic index, although theoretically of value, is a complicated procedure and much controversy has arisen regarding its interpretation.[51,275]

The *brucellergin skin test* is *less specific* and therefore *less reliable* than the agglutination test. Positive brucella skin tests have been reported in almost 14 per cent of patients ill with diseases other than brucellosis and in more than 10 per cent of apparently healthy subjects.[64] These may be false positive tests or may be due to repeated contacts with Brucella from the handling of infected animals or drinking infected milk with the resulting production of a state of hypersensitiveness (with a positive brucella skin reaction) although the patient had never noticed symptoms of the disease. The allergic state may persist for some years following complete recovery from the disease.

Darley found that 15 per cent of "ailing" and 9 per cent of "well" persons gave positive intradermal tests with brucella antigen. In 124 patients with degenerative joint disease, 14 per cent showed positive reactions, while 18 per cent of those with "infectious arthritis" had positive skin tests.

The least specific and least reliable test for brucellosis is the *phagocytic index.*[94] This *may be positive in more than 30 per cent of patients ill with*

chronic diseases other than brucellosis.[64] Freyberg and Evans found no consistent correlation between the phagocytic power of the leukocytes and the state of infection. In some cases, brucellosis, proved by recovery of the organism, may occur even though the agglutination and skin tests as well as the phagocytic index are negative. Unfortunately, recovery of Brucella by culture or animal inoculation can be accomplished in only a few cases with brucellosis.

The average duration of undulant fever is *three months; individual cases may persist for years.* There is no pathognomonic type of fever;[58] chronic cases may be almost afebrile.

Synonyms: Malta fever; Brucellosis; Bang's disease (in animals).

Frequency of undulant fever: about 15 per cent of all cows and 10 per cent of all milk goats in this country have or have had this disease.[265] Several thousand cases are reported each year in human beings in this country.

Incidence of joint involvement: About 25 to 50 per cent of cases.[5,14,251]

Types of joint involvement:[191,274] Arthralgia, non-suppurative or suppurative arthritis, intermittent hydrarthrosis, infectious or suppurative spondylitis.[80,160] Although arthralgia is common, arthritis is rare; joint pains are often migratory.[117] *The arthritis may be acute or occasionally chronic.*[84] Spindle shaped swellings of the fingers may resemble rheumatoid arthritis[183] and migratory joint pains may be mistaken for rheumatic fever.[241,263] Bursitis,[111] neuritis and neuralgia have also been reported.

Joint phenomena may occur during the height of the disease or during the convalescent period.[82,83,128,159] Neuralgic pains and transient attacks of arthritis are not uncommon. *In about 10 per cent of the patients, the arthritic involvement may dominate the clinical picture. Arthralgia occurs in practically every case.* The actual arthritic involvement may include the formation of a serous or purulent joint effusion. *Any joint in the body may be involved* although the shoulders, knees, hips, ankles and joints of the hands and feet have been affected most commonly. Severe pain may be noted in the sacro-iliac joint.[49] A burning type of pain may occur in the wrists, ankles and elbows.[273] Hydrarthrosis may be intermittent.[98,99,236]

Harris reported 6 instances of *popliteal bursitis* of varying size in chronic brucellosis. All but one of these individuals noted improvement or disappearance of the bursal involvement when treated with Brucella abortus vaccine.

Phalen, Prickman and Krusen reported 3 cases of **Brucella spondylitis** in two of which destructive lesions of the spinal column were present.[204] *All of the patients recovered and the bony lesions were repaired spontaneously following a course of physically induced hyperpyrexia.*

Spondylitis is not uncommon in undulant fever.[23,158,160,198,247,252] Palagi collected 29 cases of brucella spondylitis from the literature in 1934 and added 17 of his own.

As a complication of undulant fever, spondylitis is most likely to occur several months after the beginning of the febrile state but localization in the spinal column has occurred as early as three weeks and as late as a year after the original infection. In some instances, the patient apparently recovered from the systemic disease but was later affected by the spinal complication. One should always suspect the possibility of brucellosis with spondylitis in backache occurring in farmers, veterinarians, butchers, dairymen, packing house employees and those with a history of ingestion of raw milk.

Destructive lesions of the vertebræ may occur in brucellosis with wedging of the vertebræ and narrowing of some of the interspaces. Such lesions are usually limited to a few vertebræ in the lumbar region.

In some cases of Brucella spondylitis, surgical intervention may be required, such as incision and drainage of an abscess or spinal fusion. In Phalen and his associates' cases, 3 sessions of fever were given, separated by an interval of three days with a sustained rectal temperature of 105° to 106° F. for five hours.

In most cases of Brucella spondylitis, the diagnosis is based upon the finding of vertebral lesions or clinical signs and symptoms of spondylitis in individuals with a history of remittent fever, a rapid sedimentation rate and the presence of a high titer of specific agglutinins for Brucella organisms in the patients' blood.

Treatment.—Early reports indicated that undulant fever responded rather promptly to adequate sulfonamide therapy;[18,28,253,260] this is now doubted by many observers. At present, the consensus is that acute cases of brucellosis occasionally[179] respond to sulfonamides, but usually do not.[69] Fever therapy or treatment with Brucella vaccine remain the most satisfactory forms of therapy.

Recovery from undulant fever has been reported in from four to nine days after the onset of sulfanilamide therapy.[29] Other forms of treatment include the use of foreign proteins (such as *typhoid vaccine,*[153,196,238] sterile milk, etc.); *artificially induced fever;*[278] stock or autogenous *vaccines* of *Br. abortus* or *Br. melitensis* or both; human convalescent *serum* and various animal antisera prepared against strains of Br. abortus and Br. melitensis; toxic filtrates from broth cultures of these organisms; various chemicals including dyes intravenously and by mouth; neoarsphenamine;[267] methylene blue; metaphen;[1] mercurochrome; acriflavine; methyl violet and fouadin.

In chronic brucellosis, fever therapy (physically induced or by typhoid vaccine) has produced good results. If typhoid vaccine is used, approximately 10 to 25 million dead organisms are given intravenously (to adults). Injections are given at intervals of four or five days; the dose is increased at each injection in an attempt to produce a fever reaching a peak of from 102° to 104° F. In some cases it is necessary to reinforce the injection by a second or even a third injection several hours after the first. A total of four or five bouts of fever are usually given over several weeks. If an individual has a severe reaction to the injection of typhoid bacilli, it may be advisable to employ the same dosage (without increase) or to increase this very slightly for subsequent injections (unless febrile reactions do not occur). In some patients, it may be necessary to double each dose to obtain the desired reaction. A standard brand of killed typhoid organisms should be used for all of the injections.

The *mortality in undulant fever* approximates 1 to 4 per cent although it may reach 13 per cent in severe outbreaks.[116] Prophylaxis of the disease consists of boiling or pasteurizing all milk, and blood testing of all cows and goats with destruction of the infected animals.[93]

ARTHRITIS WITH CHRONIC ULCERATIVE COLITIS

Arthritis is an uncommon complication in patients with chronic ulcerative colitis, occurring in about 4 per cent of cases.[10,36] It is more apt to accompany subsequent exacerbations of the colitis than the first attack.

The joint involvement may be monarticular or polyarticular and varies in severity (usually in proportion to the intensity of the ulcerative colitis). Joint phenomena resemble those of true rheumatoid arthritis; periarticular involvement is common early in the disease and intra-articular changes at a later stage. Hydrops of the joint has been reported but suppuration has not been noted.[111]

Some of these patients have had marked improvement in their arthritic phenomena following treatment of the chronic ulcerative colitis with a low residue, high vitamin diet, Bargen's specific serum and later with vaccine. In other instances, deformities have developed.

Sulfasuxidine or sulfadiazine have produced slight improvement in the colitis in a few of these patients. One of our patients with a regional ileitis noted marked improvement following the intramuscular injection of 100,000 units of penicillin daily (in divided doses every three hours) for two weeks.

ACUTE SUPPURATIVE ARTHRITIS

Joint disease from pneumococci, meningococci and hemolytic streptococci is becoming very uncommon since the introduction of the sulfonamides and penicillin.[112]

Acute suppurative arthritis *may be caused by* several mechanisms: (1) *direct wounds* of the joints; (2) *blood borne infection* (metastatic); and (3) *extension from neighboring areas of infection* (as from an adjacent osteomyelitis).[48] The metastatic form of suppurative arthritis is the most frequent and may follow pneumonia, gonorrhea, septicemia, osteomyelitis,[32,218] meningitis, subacute bacterial endocarditis, sore throat,[235] scarlet fever,[30] measles, infected wounds, peritonsillar abscess, otitis media,[76] mastoid infection, infected varicose ulcers,[192] furuncles, infected burns,[264] pyelitis, infection of the umbilicus and sinusitis.[209]

Bacteria which may produce a purulent arthritis[105,157] include the *staphylococci* (aureus and albus), *hemolytic or green producing streptococci,*[244] *gonococci, pneumococci,* and less common invaders such as the meningococci, influenza bacilli,[203] typhoid bacilli, Bacillus pyocyaneus, micrococcus tetragenus,[211] Salmonella suipestifer,[268] etc.

The *pathologic changes* vary in intensity with the type of organism and the resistance of the individual;[79,81,146] there may be a mild synovitis or a markedly destructive lesion of the joint which may prove fatal. The damage is produced in large part by the proteolytic enzymes of the white blood cells (in the pus), and to a lesser degree by mechanical pressure and erosion.

Early symptoms include the acute onset of fever, chills, sweats, leukocytosis, local pain, swelling, redness, limitation of motion (due to muscular spasm) and tenderness around the joint.[70,109,149,164] The *most commonly affected areas* are the large joints such as the hip,[8,35,72] knee or shoulder, and less commonly an ankle, elbow, sacro-iliac region or wrist.[115] The disease is *usually monarticular.* Purulent arthritis is more common in children than in adults. Such patients have often been diagnosed as having rheumatic fever or a mild form of infectious arthritis.[155]

Nicholson has reviewed the subject of **septic arthritis of the hip.**[189] Prior to the introduction of the sulfonamides, crippling deformity occurred in 87 per cent of these patients. The pyogenic infection was usually blood borne due to staphylococci or streptococci most often, but also to pneumococci, mixed infections and rarely the pyocyaneus, colon, typhoid or melitensis organisms.

Symptoms include a history of primary infection of several days' duration and often local trauma the preceding day; mastoid infection in children is an especially important primary focus. These patients develop a fever of from 101° to 103° F., moderate leukocytosis and pain; the latter renders weight bearing impossible. *Pain in the knee is the most common complaint* (due to reference along the obturator nerve). The thigh is usually held in flexion, adduction and internal rotation; after a few days, this is in abduction, flexion and external rotation. The acute pain permits only a few degrees of motion of the joint. The joint fluid causes bulging and thickening of the capsule; the thigh appears edematous and the inguinal crease is obliterated. Marked tenderness is experienced on local pressure.

Aspiration may decompress the joint, temporarily relieve pain and aid in diagnosis. Lateral aspiration is usually employed; the needle is introduced above the tip of the trochanter and is tilted down along the femoral neck. If the infection is permitted to progress, the joint cartilage undergoes autolysis and the distended capsule may rupture at its inferior mesial portion. Spontaneous dislocation of the hip may occur (posterior and upward with the head anterior).

Roentgen examination is of little aid in early diagnosis unless there is a nearby osteomyelitis. Roentgen-ray findings are usually present after the first week or two of the disease. The amount of narrowing of the joint space is a measure of the damage to the articular cartilage. Reliance must be placed upon aspiration of the joint fluid for examination.[145] The latter may contain several hundred thousand leukocytes per c.mm. in hemolytic streptococcal arthritis; almost all of these cells may be polymorphonuclear cells.[148] This should be considered in any case of acute monarthritis with severe symptoms in which moderate or marked constitutional symptoms are present.[182,195]

Hefke and Turner have called attention to obscuring or widening and curving of the border of the obturator shadow as seen by *x*-ray in some types of hip joint pathology; this is called a **positive obturator sign.**[110] It

was thought that this sign reveals the presence of septic hip joint disease by x-ray at an early stage, and is of some importance in the early diagnosis of tuberculosis of the hip. It is absent in fractures about the hip, osteochondritis and other pathologic conditions of the hip.

The obturator shadow is a soft-tissue shadow seen in the A-P roentgen examination of the pelvis; it is just medial to the acetabulum on the inner aspect of the pelvis. The upper border of the shadow usually begins lateral to and below the lower pole of the sacro-iliac joint and follows the line of the pelvic inlet downward over the acetabulum, disappearing behind the upper ramus of the pubis. It is believed that the substratum of the shadow is the obturator internus, whose tendon is adjacent to the capsule of the hip joint.

Pyogenic arthritis of the sacro-iliac joint is very unusual. Avila reported 7 cases of primary pyogenic infection of this joint with recovery in all. In all of these cases but one, the abscess formed anteriorly.[6,130,166]

Seven cases of **septic spondylitis** were reviewed, 5 of which were caused by staphylococci, one by streptococci and one possibly by the influenza virus. Six cases were acute, with abscesses and one was chronic, without an abscess. The x-ray revealed the abscesses and edema early. Treatment consisted of incision of the abscesses and immobilization of the affected part. Healing occurred in every case, but some stiffness of the back persisted.

In cases of marked infection, the sulfonamides cannot be expected to control infection alone, but surgical procedures may still be necessary.[24,75] Penicillin will probably prove to be of far greater value.

Traumatic arthritis may produce severe monarticular phenomena but in such cases the constitutional symptoms are usually absent or mild. *Acute purulent arthritis may follow injury of a joint.* In acute gouty arthritis, constitutional reaction is also usually mild. The existence of only a periarthritis must be ruled out.

Prognosis.—This is often poor unless treatment is instituted within the first week of the disease. The outlook for preservation of function in acute suppurative arthritis is fairly good if early diagnosis, chemotherapy, and if necessary arthrotomy and drainage have been instituted; if bone destruction has occurred before treatment is begun, the prognosis is poor.[132] The primary site of infection and the causative organisms have been stated to be the main determining factors. The mortality reached 20 per cent in the pre-sulfonamide era.

Treatment.—Sulfonamides and/or penicillin should be used when the invading organism is the hemolytic streptococcus, meningococcus, gonococcus, pneumococcus[121,184,266] or staphylococcus. Although 9 serologic groups of hemolytic streptococci have been identified, most cases of acute or serious hemolytic streptococcal disease in man result from the group A (Lancefield) organisms; the main source of the latter is the human nasopharynx.

Sulfonamides and/or penicillin are effective against this group of bacteria.

The method of administering these drugs and the possible untoward reactions have been described elsewhere. (See chapters on the Sulfonamides.)

Further treatment for suppurative arthritis includes immediate aspiration of the infected material and introduction into the joint (when possible) of 20,000 units or more of penicillin (dissolved in physiologic saline) daily for two or three days. If a primary focus can be found, it should receive adequate treatment. In some instances, when aspiration of the purulent fluid is impossible, it may be necessary to incise and drain the joint, suturing a rubber tissue to (but not inside of) the capsule; motion of the joint should then be instituted. At the time the joint is incised, one may introduce 20,000 units or more of penicillin in solution into the joint. If ankylosis is inevitable, the joint should be splinted in its most favorable position. Prior to the introduction of sulfonamides and penicillin, the joints were washed with bichloride (1:15,000) in saline.[140] In occasional instances, amputation was necessary.[219] Supportive therapy with transfusions, high caloric diet and analgesics or narcotics is usually necessary.[60] Corrective orthopedic surgical procedures are required in those instances in which deformity has occurred. With the further use of sulfonamides and penicillin, surgical intervention (except aspiration of the joint and instillation of penicillin) should become unnecessary in most of these cases.

Suppuration within the hip joint runs a benign acute course during the first two years of life compared with this disease in later childhood and adolescence; this is due in great part to the large amount of cartilage in the component structures of the hip in young children.[101] Recognition may be delayed until the child begins to walk, when one observes pathologic dislocation of the hip. This condition is often misdiagnosed an abscess. In the presence of a pyogenic exudate, the cartilage (which makes up the greater part of the upper femur at this age) is dissolved. Although the child apparently recovers from the infection with rapid closure of his wound (if the general physician mistakes this for an abscess and drains it), the damage produced is not evident until the child becomes ambulatory at the end of the first year of life when one notices the hip lurch produced by the dislocation.

Harmon has described a reconstruction operation which may be done during the second and third years of life.[101]

Epiphyseal pseudarthrosis at the hip may be seen after operative fusion of the hip, following inflammatory ankylosis of the hip or as a sequel to adolescent epiphysiolysis.[182]

Heberling reviewed the *results of treatment of 201 patients with acute suppurative arthritis.*[109] Surgical therapy consisted of incision of the joint, under general anesthesia and suturing an appropriate sized soft rubber drain to the capsule—not permitting this to intrude into the joint. Active motion was begun as soon as possible and was repeated every three hours. If it is occasionally necessary to replace extruded drains and reopen pockets, this can usually be done without anesthesia.

Early walking and weight bearing did not hasten recovery. In those

with involvement of the knee or hip, adhesive traction with a small amount of weight was used.

Sites of incision were as follows:

1. *Shoulder:* anteriorly through the deltoid.
2. *Elbow:* posteriorly over the radio-humeral region.
3. *Wrist:* dorsal into the snuff-box.
4. *Hip:* anteriorly between the sartorius and the tensor fasciæ femoris.
5. *Knee:* mesial anterior and occasionally a second incision on the lateral side of the patella.
6. *Ankle:* posterolateral and anteromesial.

After opening the joint, it is gently flexed and extended a few times to express the contents before suturing the drain in place. Fluffed gauze is piled on loosely and secured, permitting complete joint motion. When the discharge becomes thin and scanty and the thickening and bogginess about the joint begin to disappear, the drains are removed and the opening is allowed to close gradually.

In *septic arthritis of the hip joint,* rest of the joint should be accomplished by a double hip spica including the thigh of the opposite leg to prevent adduction and abduction, and the foot on the involved side to control rotation. Windows may be provided in the cast for aspiration of the joint. In some cases, surgical drainage may be necessary (if the femoral head is destroyed, if thick purulent material is present, etc.). Dependent drainage may be obtained by opening the hip joint posteriorly. Weight bearing must not be instituted too early; follow-up orthopedic care and physical therapy are extremely important.

Inge and Liebolt have reported in detail upon 36 patients with acute suppurative arthritis treated by operation before the sulfonamide era and have found that:[132]

Best results followed in those cases where early arthrotomy was done.

The use of small, soft rubber drains into the joint cavity (withdrawn as rapidly postoperatively as subsidence of the infection permitted) was one of their most helpful adjuncts in the treatment of suppurative arthritis.

Early active and passive motion was invaluable.

Aspiration with injections of antiseptics have been found to be inadequate in the treatment of severe cases of this disorder.

Fever therapy will not be routinely beneficial in the treatment of staphylococcus suppurative arthritis as these organisms are not destroyed by a temperature of 106.7° F. for one hundred hours.[114] An occasional instance of good results with fever therapy in such a case has been reported.[106] The use of staphylococcal bacteriophage is probably of little, if any, value.

It would appear at present that the chemotherapy of choice in suppurative (non-tuberculous) arthritis includes:

1. Penicillin—100,000 units (or more) daily, intramuscularly (in divided doses, every three hours).

2. Aspiration of the affected joint and introduction of 20,000 units of penicillin directly into the involved joint daily for several days.

3. If penicillin is not available, use sulfamerazine (orally). Adults may receive an initial dose of 2 grams followed by 1 gram every eight hours. This should be continued for at least three or four days before reducing the dose unless marked leukopenia or other untoward signs appear. Sufficient alkali should be given to keep the urine alkaline.

4. If the infection does not subside promptly both penicillin and sulfamerazine may be employed.

MISCELLANEOUS (UNCOMMON) FORMS OF ARTHRITIS

Actinomycosis.—Forty-seven cases of *actinomycosis of the spine* have been reported;[144,180] this may be mistaken for Pott's disease. It is usually secondary to a focus in the respiratory or intestinal tract. Clinical diagnosis is very rarely made. *Multiple sinuses* may be present and there is a more destructive invasion than in Pott's disease. The mortality rate is about 90 per cent. Dobson and his associates reported cures in 3 cases of actinomycosis by therapy including sulfanilamide, iodides and roentgen-rays.

Anthrax.—Joint complications are rare.[55,57] Migratory arthralgia and serous exudates have been noted.

Bacillary Dysentery.—Serous effusions containing Shiga's bacillus have been found in approximately 3 per cent of cases of bacillary dysentery.[88] This may involve one or multiple joints[186,190,226] and appears most often from one to three weeks after the onset of the diarrhea, although it has also been noted several weeks after cessation of the diarrhea.[17] Such involvement (usually of the knee or ankle) persists from four to six weeks, and is usually followed by complete recovery of joint function.[13,91] Suppuration and resultant ankylosis have occasionally been noted.[108] Kruspe has discussed the rôle of dysentery in the etiology of Reiter's disease.[156]

Blastomycosis.—Meyer and Gall have collected 12 cases of blastomycosis of the spine. The findings are similar to those of actinomycosis.

Brucellosis (Undulant Fever).—Discussed previously (page 830).

Cerebrospinal Meningitis.—Discussed later. (See Meningococcal arthritis.)

Cholera.—Serofibrinous synovitis has been reported; general aches and pains in joints and muscles may occur.

Coccidioidal Arthritis.—Rosenberg and his associates reported a case of *coccidioidal arthritis* with involvement of the ankles, *which did not respond to either sulfanilamide or roentgen therapy.*[223] The main reservoir for this infection lies in the Sacramento-San Joaquin Valley in the southern agricultural valley of California. About 90 per cent of reported cases have concerned patients observed in California. The disease is caused by the fungus C. immitis.

Joint involvement may occur in the acute benign phase of the disease or in the more dangerous chronic granulomatous stage. The disease (also called *valley fever, desert fever, desert rheumatism*) is usually mild and nonfatal and is characterized by an *acute onset with general aches and pains, malaise, fever, sore throat, toxic erythema* and in some instances, conjunc-

tivitis and bronchopneumonia. *Lesions of the erythema nodosum type appear*, especially on the shins from eight to fifteen days after the onset of the disease. Pulmonary lesions may appear at this time.

Joint involvement is *relatively insignificant*, although it may be distressingly painful; *the arthritis subsides completely*. If the joints are involved in the granulomatous phase of the disease, this is serious and may be an indication of a disseminated infection which will lead to death.

About a third of the patients with valley fever develop signs of acute arthritis. This usually appears simultaneously with the lesions of erythema nodosum. The *affected joints are tender, painful on motion and may be slightly swollen.* No one has observed effusion into these joints nor has suppuration taken place. In some instances, arthritis, conjunctivitis and erythema nodosum appear together, persist approximately a month and then disappear at about the same time. There is no residual damage or deformity in the affected joints.

One should suspect the presence of valley fever especially in those with acute arthritis which is accompanied by an acute pulmonary infection with or without the presence of erythema nodosum. If this patient has lived or has been visiting in southern California, the disease is much more likely. A definite diagnosis may be made by identifying the C. immitis in the sputum by direct examination, culture or inoculation into guinea pigs.

In patients with the acute type of valley fever, survival is the rule. *The chronic granulomatous phase develops in only about 1 in every 400 patients with valley fever.* In these chronic granulomatous stages (coccidioidal granuloma) the mortality may be as high as 50 per cent.

In this chronic granulomatous phase of coccidioidomycosis, *bone and joint lesions are fairly common.* In one series of 256 cases, the joints were affected in 79 (with involvement of the ankle in 33, knee in 21, foot in 16, elbow in 11, wrist in 10, shoulder in 8, finger in 7, vertebræ in 6, hip in 5, sternoclavicular in 1, with others unspecified). *The roentgen findings in these joints may mimic those of tuberculous arthritis* with regions of destruction in articular surfaces early which is often accompanied by swelling of the overlying soft tissues. Articular cartilage may be destroyed with narrowing of the joint spaces. The joint spaces may completely disappear in later stages and extensive areas of destruction may be noted in articular surfaces. *Ankylosis may occur* in some cases.[177] The arthritis is predominantly destructive both in tuberculosis and in coccidioidal granuloma with little tendency to heal by production of bone; pulmonary involvement is frequent in both. Coccidioidal granuloma usually attacks joints by extension from adjacent bony lesions.[39] The destructive process in bones in coccidioidal granuloma develops more rapidly than does tuberculosis.[258]

Coccidioidal arthritis may be suspected if progressive mono- or polyarticular arthritis occurs in individuals who have lived in or visited the San Joaquin valley region in southern California; if serial roentgenograms show an unusually rapid progress of the destructive bony lesions, the diagnosis is more likely.[223] The final diagnosis is made by finding the organisms in sputum, tissues or pus from an involved area.

About 50 per cent of individuals with coccidioidal granuloma die. In these individuals, the disease becomes generalized and the terminal phase shows a septic high fever, chills, emaciation and severe anemia. No specific treatment has been of value in these patients. Although one patient was markedly improved by sulfanilamide,[131] the drug was not of value in others.

It is sometimes difficult to differentiate coccidioidomycosis, blastomycosis and torulosis.

Dengue.—Simmons reported arthralgia in 45 per cent of cases of experimental dengue.[240] Severe aches and pains in bones and joints are common symptoms of dengue. Joint swelling with redness and pain have been observed.[4]

Dermatomyositis.—Keil has commented on the resemblance of this disease to lupus erythematosus and has reported several "transitional cases" and considered the possible etiologic rôle of lead in these patients. These subjects may have vagrant articular pains, muscular atrophy, smooth, atrophic, glistening oval patches over the finger joints and contractures about the large and occasionally the small joints.[147]

Grossman reported 9 new cases of dermatomyositis in adults.[137] Because of the variability of the manifestations, he doubted whether this is a single clinical entity. The cause of dermatomyositis is unknown. The disease may occur at any age but is most common between the ages of ten and fifty. Both sexes are involved equally.

The onset of the disease may be either abrupt or insidious and exacerbations and remissions are frequent. The illness may last from weeks to years, with a *mortality rate* of from *50 to 60 per cent.*[178]

The *most common complaints* are muscular pains, tenderness, stiffness and fatigue which may be localized or generalized. *Involvement of muscles usually produces the earliest symptoms.* As the disease progresses, there is tenderness, weakness and atrophy of skeletal muscles. The muscles of the shoulder, pelvic girdle and proximal muscles of the extremities are affected most often; however, the extrinsic orbital muscles, tongue, and muscles of the jaw, pharynx and larynx, intercostal muscles and the diaphragms may be affected. The most common cause of death is respiratory paralysis from diaphragmatic involvement or pulmonary infection. Muscular contractures may occur late in the disease.

Other common complaints are *fever, headache* and *loss of weight.* Paresthesias or muscular cramps may be noted. In some instances, purpuric spots, epistaxis and bleeding into the kidneys and intestines have been seen.

Cutaneous lesions usually appear during the course of the illness. These are usually erythematous and may simulate numerous dermatologic conditions. One may also see loss of hair, pigmentation, sclerodermatous changes, subcutaneous calcification, hyperhidrosis and telangiectases. Lesions of the mucous membranes may be encountered. A soft or brawny edema with erythema has been seen frequently. *Raynaud's phenomena are present frequently* and may be the earliest sign of the disorder.

Arthritic pains may occur but actual joint disease has not been noted. These pains have usually been ascribed to periarticular or tendinous involvement.

Other findings in some cases include *splenomegaly* and *hepatomegaly, adenopathy, effusions into body cavities,* retinal changes and decreased tendon reflexes.

Diseases which have been confused with dermatomyositis include scleroderma, disseminated lupus erythematosus, periarteritis nodosa, epidemic myalgia, polyneuritis, trichiniasis and early stages of rheumatic fever or rheumatoid arthritis. Most physicians know little more than the name when one mentions such conditions as periarteritis nodosa, dermatomyositis, lupus erythematosus and scleroderma.

In advanced cases, it is usually easy to split off scleroderma from this group. The butterfly facial rash, with extreme sensitivity to light, frequent finding of leukopenia, signs of renal irritation in many cases, effusions into serous cavities and actual joint pain and swelling (in a large percentage of cases) will usually help to remove *disseminated lupus* from this mix-up.

The multiplicity of symptoms in both periarteritis nodosa and in dermatomyositis makes diagnosis extremely difficult. The recent work of Rich and his associates in suggesting that *periarteritis nodosa may be the result of an allergic reaction* should stimulate further work along this line in dermatomyositis and in lupus erythematosus. Periarteritis nodosa cannot usually be diagnosed with certainty without a biopsy which reveals the characteristic collections of small round cells surrounding a small blood-vessel; in most instances the diagnosis is made when the histologic sections are examined following an autopsy. The diagnosis of periarteritis nodosa is not suspected and is certainly not made in most individuals with this disease. For this reason, we *must suspect this group of diseases* (periarteritis nodosa, lupus erythematosus, dermatomyositis and scleroderma) *in individuals with bizarre muscular pains and aches, loss of weight, irregular periods of fever, muscular atrophy and joint pains* so that these patients can be studied more carefully during life in an attempt to find some possible etiologic factor in these disorders and to establish whether or not all of them are separate entities.

Sedimentation rates in dermatomyositis vary but are usually normal or only slightly increased. Total serum proteins are approximately normal although the globulin fraction has been increased in several of the patients.

The only constant laboratory finding in dermatomyositis is *spontaneous creatinuria;* this varies from 53 to 633 mg. per twenty-four hours. It must be remembered that healthy women may excrete considerable amounts of creatine in the urine particularly during the menstrual period.[137]

Electrical stimulation of multiple muscle motor points does not show reactions of degeneration, but it is necessary to use from 25 to 40 per cent more current to produce contractions than in normal controls.

The *pathologic changes* seen in muscles histologically are not specific. Similar changes have been observed in numerous diseases such as pneumonia, toxic goiter, rheumatoid arthritis, etc. These include hemorrhage into muscle, edematous interstitial connective tissue (which might be increased in amount), infiltration of the interstitial tissue with lymphocytes, the occasional appearance of lymphoid follicles, proliferative and degenerative changes in the muscle fibers (with edema of the fibers, loss of striation, atrophy, fragmentation, necrosis, and hyaline, fibrinous, granular or vacuolar degeneration). In some instances, walls of arterioles were thickened or showed extensive changes.

There is no satisfactory treatment of dermatomyositis. In many cases, salicylates do not relieve the muscular symptoms. Vitamin E has been used in a few cases with reported improvement. Other therapy has been symptomatic, including rest, blood transfusions, physical therapy, etc.

Diphtheria.—Acutely painful, swollen joints occur occasionally during the early stage of diphtheria; diphtheria bacilli have been isolated from these. Arthritis and chorea minor have been reported accompanying diphtheria in children.[127]

Drugs.—Bone and joint pains may occur following the administration of such drugs as arsenic, mapharsen, iodobismitol, gold, sulfonamides (sulfatoxic arthritis), phenobarbital.[172,208]

Erysipelas.—Complications of erysipelas may include arthralgia, hydrarthrosis, arthritis, and rarely, suppurative arthritis.

Gaucher's Disease.—Schein and Arkin studied *8 cases of Gaucher's disease with hip joint involvement.* Gaucher's disease is a rare, familial disorder of lipoid metabolism, characterized by deposition of kerasin in the reticulum cells of the reticulo-endothelial system; these are called Gaucher cells. Most of the patients are Jewish. The *symptoms* are *most often* encountered *in childhood and adolescence.* The disease may involve any part of the reticulo-endothelial system: spleen, liver, lymph nodes, bone marrow, etc.

Cushing and Stout in 1926 reported the first case of joint involvement with Gaucher's disease, in which surgery was done in the hip.[47] The bone and joint lesions in Gaucher's disease have been reviewed by Welt, Rosenthal and Oppenheimer in 1929[270] and by Reiss and Kato in 1932.[212] Only 16 cases of hip joint involvement in Gaucher's disease have been reported to date.

In children, the *hip lesions in Gaucher's disease include:* lesions of the femoral head, often suggesting Legg-Perthes disease or early tuberculosis; lesions of the femoral neck with simple infiltration seen by *x*-ray; pathological fracture of the neck or coxa vara deformity, with broadening and irregularity of the epiphyseal line.[233] In adults, the hip lesions may present symptoms and signs of degenerative joint disease (osteo-arthritis) and roentgen evidence of Gaucher's disease without symptoms or signs pointing to the hip.

The onset of the disease is often associated with a low grade *fever* and intermittent *pain* in various bones and joints and one finds the *enlarged spleen, liver* and *secondary anemia,* often with leukopenia and thrombocytopenia, brown pigmentation of the skin and hemorrhages. *Puncture of*

the sternal marrow will nearly always reveal the diagnosis by the finding of the large typical Gaucher cells.

The disease may persist for years.

During the acute symptoms, rest is essential with or without traction or immobilization of the hip in a spica. In childhood, prolonged rest is indicated to minimize deformity. Physical and roentgen therapy have been of very little, if any, benefit. In children and adults with involvement of the hip joint, rest in recumbency with traction has provided at least temporary relief. Cushing and Stout resected the head and neck of the femur in a woman of thirty-three with this disorder and removed 6 loose sequestra from the partly destroyed acetabular wall; this patient made an uneventful recovery from the operation.

Glanders.—Joint swelling with muscular and joint pain are not uncommon. Joint suppuration occasionally occurs.

Haverhill Fever.—The main characteristics of this syndrome include an abrupt onset, fever, a rubella-like or morbilliform rash, chiefly on the extremities, and a polyarthritis.[206,272] Mild or marked joint redness and swelling may occur and may persist from several days to several weeks. Joint phenomena usually appear during the first several days of the illness. Although marked limitation of joint function may occur, this usually clears within several months.

The organism (Haverhillia multiformis) has been demonstrated in the blood[107,200] and joint fluid[231] of some of these patients and specific agglutinins for this organism are present in the blood stream; serum is required for its growth in artificial media. The disease has been reported following rat bite[66,234] and following the drinking of (possibly) infected (raw) milk. Farrell and his co-workers have reviewed the literature on this disorder.

In addition to the epidemic of 86 cases, 14 sporadic cases have been reported. Although some cases may follow a rat bite, this disease should not be called "rat bite fever" as this term is also used to describe *sodoku* (or Spirillum minus infection). The latter is caused by a highly motile spirillum which cannot be cultivated outside of the animal body. Articular involvement is not a characteristic of sodoku; the disease responds to arsenical therapy. Neo-arsphenamine may occasionally be of value in Haverhill fever.

Hemochromatosis.—Dekkers reported a case of hemochromatosis combined with multiple joint anomalies.

Histoplasmosis.—Histoplasmosis is a generalized disease with variable clinical characteristics; it is caused by the histoplasma capsulatum of Darling.[150] The four *main clinical pictures* of histoplasmosis include: (1) a febrile illness with enlarged liver and spleen, anemia, leukopenia and septic temperature; (2) predominance of lymphadenopathy, simulating Hodgkin's disease, leukemia or lymphosarcoma; (3) predominance of pulmonary symptoms, often complicated by tuberculosis and (4) onset with a small skin lesion which may develop into a generalized ulcerative cutaneous lesion.[178]

Key and Large reported the occurrence of *involvement of a knee joint by histoplasmosis,* which closely resembled tuberculosis of the knee joint clinically. The patient had a dull ache and some disability in the left knee for seven months. There was no history of trauma. The knee gradually became swollen and stiff. The pain became constant, being dull with more intense pain on movement.

The knee was considerably enlarged, and excess fluid was present in the joint. The periarticular tissues were thickened. No redness was noted but there was a moderate increase in local heat over the joint.

Roentgen examination of the knee showed thickening of the soft tissues and moderate bony atrophy with spotty atrophy of the bone structure.

A thick purulent material was aspirated from the knee joint; culture of this was negative. Inoculation of the material into guinea pigs failed to show tuberculosis.

X-rays taken four weeks later showed increased atrophy of the bone, narrowing of the joint space and marked broadening and erosion of the margins of the intercondylar notch.

The extremity was amputated as the patient did not respond to sulfonamides or other therapy.

On gross examination, the knee was markedly enlarged. The synovial cavity resembled a large abscess cavity, filled with thick, gray pus; the abscess extended into the calf. There was considerable erosion and necrosis of the articular surfaces of the bones and *the specimen resembled advanced tuberculosis of the knee grossly.* There was marked thickening of the synovial tissues, which were covered with a gray-white exudate.

Microscopic sections resembled a tuberculous synovial membrane but no tubercles were seen and the giant cells were of the foreign body type. No acid fast organisms were seen. The synovial membrane was transformed into a mass of granulation tissue, infiltrated with giant cells and macrophages. Monocytes or mononuclear phagocytes were the predominating cells in the inflammatory exudate. *Many of the phagocytic cells contained the histoplasma capsulatum of Darling.* These parasites were present in enormous numbers in some places.

Almost all recorded cases of histoplasmosis have died (with only several exceptions). Full doses of sulfathiazole had no effect in Key's case. Meleney has suggested the use of antimony preparations.

Infectious Mononucleosis.—Although arthralgia is unusual in this disease, joint pain and fever may occasionally be the symptoms at onset and may simulate rheumatic fever.[19]

Influenza.—Monarticular or polyarticular joint involvement may complicate influenza;[26,77,174] this usually appears early in the course of the disease, but may not be noted until several weeks later. Suppuration has occasionally occurred.[203] Although joint involvement has been reported in as many as 25 per cent of such patients,[276] the present concept is that such joint pathology is much less common now.[108]

Leprosy.—Lepers are apparently immune to arthritis. Partially because of this, the use of chaulmoogra oil has been tried in the treatment of rheumatoid arthritis.[176] The pseudo-arthritis of lepers is mainly an osseous

destruction; disintegration of epiphyses and diaphyses follows destructive changes in the phalanges and metacarpal and metatarsal bones, due to trophic changes.

Lupus Erythematosus.—Lupus erythematosus is now believed to be a sensitization phenomenon to a bacterial allergen, the product of activity of the streptococcus, the tubercle bacillus or perhaps other organisms. The **acute disseminated type** is uncommon. The victims of this form are usually *women*. A bright *erythematous rash* appears beginning usually on the face, but often spreading rapidly to the trunk and limbs by enlarging, coalescing red, erythema multiforme-like lesions. These patients are seriously ill and are often prostrated. *Fever* is extremely variable from a low grade irregular type to an intermittent irregular septic type. The *prognosis* in these patients is usually *very poor*. These individuals frequently have *aches and pains in multiple joints* and may show *transient swellings of various joints*. The joint swelling is not usually persistent. In addition, *effusion* may occur *in* any of the *serous cavities* (pleura, peritoneum).[9]

The **chronic, fixed type of lupus erythematosus** is much *more common*, with the lesion showing a predilection for the face—with a patch on each cheek, often connected by a bridge of rash across the nose forming the so-called butterfly or bat's wing eruption.

Most writers have emphasized the *importance of avoiding actinic therapy* (sunshine or ultra-violet light) and the *danger of attempting to eradicate foci of infection during the acute phase of the disease*. Either of these procedures may cause a severe exacerbation of the disease which may prove fatal within a short period. In individuals in whom the diagnosis is in doubt, some physicians have exposed a very small area of the skin (3 by 3 inches) to ultra-violet irradiation; the occurrence of a marked local reaction or the development of a systemic reaction following this is good presumptive evidence that the diagnosis is lupus erythematosus. Some of these individuals notice the appearance of their skin lesions only after exposure to the sun. One must emphasize to these patients and their families the dangers associated with such exposure.

Many of the features of disseminated lupus erythematosus suggest that it is primarily a manifestation of allergy or hypersensitivity. In view of the recent observations of Rich and Gregory[215,216] periarteritis nodosa, whose pathologic lesions resemble those of some cases of disseminated lupus, is considered by most individuals to have a fairly well established allergic basis.[74] The tendency at present is to regard periarteritis nodosa as a type of reaction pattern rather than as a disease with one etiologic basis. The offending antigen in periarteritis nodosa may be a foreign protein or a compound formed by conjugation with sulfonamide drugs.

Fox reported a patient in whom there was presumptive evidence of a relationship between the parenteral administration of a foreign protein and the development of disseminated lupus erythematosus.[74] He discussed the similarity in general symptomatology between serum sickness and disseminated lupus and suggested that it may prove eventually to be a pattern of reaction.

Disseminated lupus erythematosus has been characterized as a *diffuse collagen disease* inasmuch as the alterations of the disease process are localized primarily in the collagenous tissues of the body.[152] The numerous organic changes may be understood as local manifestations of widespread damage of collagen.

There have been very few reports on the pathology of the affected joints in disseminated lupus. Hypertrophy of the synovial villi has been reported with perivascular infiltration of inflammatory cells of the subsynovial and capsular tissues.

Acute disseminate lupus erythematosus is associated with widespread visceral lesions involving predominantly the kidneys, blood-vessels, lymph nodes, serous and endocardial surfaces and skin.[44] The disease is found most often in women in the second and third decades. The prognosis is grave, with an average duration of about eighteen months.[9]

Prominent clinical features include the skin lesions, prolonged irregular fever, joint involvement, secondary anemia with leukopenia and signs of renal involvement.

The clinical picture is extremely variable. In some individuals, the only symptoms are joint pain and swelling, of a very variable nature, with or without a low grade intermittent fever. If these individuals develop a rash on the face with or without signs of renal involvement, the diagnosis of lupus erythematosus disseminata must be suspected.

Cluxton and Krause recently reviewed the literature on acute disseminated lupus erythematosus.[44]

Arthralgia is usually present at one time or another in these patients. Some workers have reported the occurrence of joint deformities late in the disease with involvement of the smaller joints of the extremities;[78,261] however, *it is very unusual to see permanent changes in the involved joints in lupus erythematosus.*

Arthralgia and arthritis are rather common in lupus erythematosus.[222] Joint disease is present in 20 per cent of the chronic disseminated types, 57 per cent of the subacute disseminated types and 63 per cent of the acute disseminated cases.[243] Polyarthritis with prolonged fever, anemia, erythematous cutaneous lesions, polyserositis, endocarditis, nephritis and a fatal termination has been described.[210] An acute arthritis, with myocardial damage may simulate acute lupus erythematosus.[86]

This group of symptoms has also been known variously as the *visceral erythema group* (Osler), *atypical verrucous endocarditis* (Libman-Sacks), fever of unknown origin (Christian) and diffuse peripheral vascular disease (Baehr *et al.*). The Reifensteins analyzed the reports on 19 such cases with postmortem examinations but could adduce no definite diagnostic criteria; however, every patient had pleuritis and articular manifestations. More than one-half had nephritis or arteritis. No clue as to an etiologic agent was obtained.

Slocumb summarized his findings in 10 patients with lupus erythematosus who had joint manifestations.[243] In 2 of these, joint symptoms followed

skin changes. In the other 8, skin lesions followed from one and one-half months to five years after the appearance of the joint symptoms.

Joint changes varied considerably, resembling rheumatic fever, subacute or chronic infectious arthritis and fibrositis. Attacks of fibrositis recurred at intervals of from two to four weeks in 3 patients. Symptoms included pains in the muscles and joints which persisted for several hours or one or two days, involving one or two regions at a time. With progression of the disease, the attacks of fibrositis became more frequent and more extensive and resulted in aching and subjective stiffness.

In all of Slocumb's patients there occurred *an acute inflammation of several joints, persisting a few days and occasionally leaving some aching and stiffness.* Six of the patients developed chronic periarthritis and arthritis with acute and subacute attacks, and with residual periarticular swelling. Joint deformities were not seen except in one case; there was no roentgen evidence of bone disease.

The articular symptoms of lupus erythematosus are rarely diagnosed properly until the skin lesions have appeared. Joint symptoms may precede cutaneous lesions by months or several years, leading to the false diagnosis of fibrositis, rheumatoid arthritis or rheumatic fever.[119]

In cases of fibrositis with elevation of the sedimentation rate and leukopenia or in patients with atypical rheumatic fever which has not responded to salicylates (and may be associated with leukopenia instead of the more common leukocytosis), one should bear in mind the possibility of *lupus erythematosus;* if signs of renal irritation are evident, this presumptive diagnosis is even more likely. This is of importance because severe or fatal exacerbations of unrecognized lupus erythematosus have been precipitated by chrysotherapy, fever therapy or even heliotherapy for treatment of a supposed rheumatoid arthritis or other disorder.

The *Libman-Sacks syndrome,* described first by these authors in 1924, includes polyarthritis, leukopenia, a characteristic mural and valvular atypical verrucous endocarditis and pleurisy with effusion; lupus erythematosus was present in 2 of their 4 patients.[169]

Severe and even fatal exacerbations of lupus erythematosus have been precipitated by chrysotherapy, heliotherapy or fever therapy in which the diagnosis was mistaken for rheumatoid arthritis or rheumatic fever.

There is *no specific treatment* for the acute disseminate type of the disease. Transfusions and general care seem to offer as much hope as other measures. Inasmuch as most instances of the acute disseminate form occur in women during the menstrual ages, attempts have been made to alleviate the disease by surgical or roentgen castration. These attempts have usually been unsuccessful. *Penicillin* may prove to be of value.

Injections of bismuth or of gold have been employed in treatment of the chronic discoid form of lupus.[245]

Heavy metals, in general, are contraindicated in the treatment of acute disseminated lupus as leukopenia and thrombocytopenia are commonly present. Salicylates have been used for the fever and arthralgia and in some instances, temporary remissions have followed blood transfusions. The ultimate results of sulfonamide therapy have not been encouraging.[44]

Lymphogranuloma Venereum.—Lymphopathia venereum is distinctly a venereal disease affecting all sexes and races.[255] The primary sore, which usually appears from two to five days after infective coitus, is usually asymptomatic and heals spontaneously. Joint involvement may occasionally resemble rheumatic fever or purulent arthritis.[138]

The arthritis which may occur with lymphogranuloma venereum runs a variable course. In occasional cases, one may encounter tender, painful, acutely swollen *joints;* however, the process is *usually chronic* and *persists for weeks or months.* Polyarticular involvement is common, with frequent involvement of the knees, ankles and wrists. Intermittent hydrarthrosis occurs occasionally. The Frei reaction is positive in all cases; cultures of synovial fluid are sterile.[112]

Dawson and Boots have reported a series of 24 patients (16 females and 8 males) who showed an arthritis associated with lymphogranuloma venereum.[53] In most of these individuals (usually colored), there occurred a *chronic indolent serous arthritis,* often with intermittent hydrops; acute flare-ups were not uncommon and there was a tendency to relapse. The disease was usually polyarticular, involving especially the knees, ankles, shoulders and wrists; occasionally, migratory joint involvement or generalized arthralgia occurred. Bilateral joint involvement was noted in 50 per cent of the cases. Patients varied from nineteen to fifty-five years of age, the majority being in the third and fourth decades.

This type of joint involvement *persisted for weeks or months without destruction of bone or cartilage;* swelling was usually confined to the periarticular tissues. The joint fluid was sterile on culture, and was never purulent. Cell count of the synovial fluid varied from 900 to 5000 per c.mm.

Other findings in these patients included: *A positive Frei skin test;* other evidences of lymphogranulomatous infection (especially *rectal strictures*); marked elevation of the sedimentation rate; and a positive gonococcus complement fixation test in 33 per cent of cases.

Grace and Rake have employed a new yolk sac antigen consisting of a suspension in isotonic solutions of sodium chloride of the elementary bodies of the virus of venereal lymphogranuloma; this can be prepared with any desired concentration of virus.[89,90] In performing *complement fixation tests* with this antigen, it was found that these tests were more sensitive than the Frei reaction.

Dawson and Boots believed that the arthritis was a clinical entity associated with lymphogranuloma venereum; however, the specific virus was never demonstrated in the joint fluid.

This disease responds to sulfonamide therapy.[97,202,230,259] Visceral and joint manifestations have usually disappeared following sulfonamides. Despite this, *a positive Frei test may persist for many years after apparent healing* has occurred (as is the case with the tuberculin test in tuberculosis); *in the early stages, the Frei test is negative.*

Malaria.—Acute inflammatory involvement of multiple joints occasionally appears during the illness or convalescence. Most of these respond to quinine.

Measles.—Although articular complications have been reported occasionally, their association with measles has not been definitely accepted. Infectious spondylitis has occurred occasionally after an attack of measles.[7]

Meningococcal Arthritis.—Gwyn in 1898 reported the first authentic case of meningococcus septicemia in a patient with meningitis and arthritis.

Articular lesions have developed in from 5 to 20 per cent of meningococcus infections in various epidemics.[136,225]

Meningococcus infections increase in number in war times. The conditions at large training stations are ideal for the production of a state in which these infections are endemic: men live in barracks and new recruits are constantly arriving. Bush and Bailey discussed an epidemic of meningococcus infection at a Naval Training Station.[34]

Jaeger found 37 cases of meningococcic arthritis among 1000 cases of meningococcic infection.[135] It is very likely that the true incidence of meningococcic joint involvement was much higher inasmuch as when postmortem examination was made in 40 cases, 9 showed purulent synovitis which was not diagnosed clinically. Instances of purulent *meningococcic bursitis* and *purulent tenosynovitis* were described. This study suggests that most cases of meningococcus arthritis are instances of *purulent synovitis.* The *wrists* were involved most often; *knee joints* were also frequently attacked. The onset of the disease was either as a *mono-* or a *polyarticular involvement,* the cases being about equally divided in the two groups. The prognosis was good in all cases.

During non-epidemic periods, from 2 to 10 per cent of troops may be meningococcus carriers[66] while in periods of epidemics, the rate in healthy troops may reach 80 per cent.[41] The organism probably gains entrance to the body from the nasopharynx with subsequent invasion of the blood stream, and if not prevented by spontaneous resistance or therapy, localizes in the meninges, joints, skin, eyes or other body tissues.[122] The idea has been abandoned that meningitis is due to direct infection of the meninges through the cribriform plate except in a few cases where known trauma, with fracture, is present.

Meningococcus sepsis usually begins with *prodromal symptoms of disease of the upper respiratory tract.*[246] From one to many days later, acute symptoms appear such as a *chill,* with *fever;* the latter may rise suddenly or this may be gradual. *Other symptoms* then include weakness, malaise, muscular aches, *headache,* nausea, *vomiting,* and *pains* or actual inflammation *in joints. The rash is the most characteristic manifestation;* the most common manifestation of this is *petechial* or *purpuric* with lesions varying from 1 to 15 mm. in diameter. Many other forms of skin lesions may appear.

The articular complications of cerebrospinal fever were first reported by Welch. Osler[193] reviewed this literature and Rolleston[220] and Herrick and Parkhurst added valuable information gained during World War I.[124] Schein reported personal experience with 23 cases.[232]

This may occur as pain and tenderness in multiple joints, usually without much effusion.[12] Hemorrhages may occur into articular or periarticular tissues (usually causing only transitory involvement).

Keefer feels that meningococcic arthritis is a metastatic lesion.[146]

Pyarthrosis due to the meningococcus most commonly appears at the end of the first week of the disease. This usually involves only a single joint. Meningococci may be found in the joint fluid. Intermittent migratory arthritis may occur in chronic meningococcal septicemia.

Articular involvement in meningococcus infections is of several varieties.[73] *One may see arthralgias involving multiple joints* during the first few days of the disease as a part of the meningococcemia—occurring simultaneously with the purpuric manifestations. Herrick has described intra- and periarticular hemorrhages of the joints in these cases. The involvement is usually symmetrical and is often transitory in nature, producing marked pain, redness and tenderness of the joints, but a minimal amount of joint swelling. Occasionally, joint manifestations may precede the meningeal signs by days or weeks (or occasionally months), rendering diagnosis difficult.[37]

In others, the arthropathy begins after the fifth day of the disease and is usually *monarticular*—involving particularly the *knee;* if polyarticular involvement occurs, one joint is usually involved to a much greater extent than the others. Joint effusion may be marked; the amount of pain, redness and tenderness over the joint varies greatly. Meningococci have been found in these joints in about one-third of the cases. The *joint fluid varies from a frankly purulent effusion to a serous, mucinous or sero-purulent affair.*

With the passing of serum therapy in meningitis, one no longer sees the arthralgias and arthropathies associated with serum sickness in patients with meningococcic infections.[123] However, joint pain may occur following the use of sulfonamides (*sulfatoxic arthritis*). This disappears following withdrawal of sulfonamides.

Most observers question the existence of the so-called postmeningitic spondylitis described by Epstein and by Billington.[21,61] Inasmuch as it is usually reported in the lower lumbar spine, Fox and Gilbert suggest that this is very likely a postpuncture affair, with cellulitis or spinal osteomyelitis or herniation of the nucleus pulposus from an injury of the annulus fibrosis by the spinal puncture needle.[73]

Adams has discussed the *arthritis* which occurs *with meningococcemia.*[2] In the non-meningitis case, there is usually a history of respiratory symptoms and joint pains with or without redness and swelling. Large joints, such as the knees are affected most often. These individuals will usually also have deep muscle aching in the arms and legs, and a macular or petechial eruption. In chronic meningococcemia, one or more joints may be tender. Early treatment with sulfonamides is almost certain to effect a cure except in acute fulminating septicemia and in those with signs of meningitis who are seen late in the course of the disease.

Seventy-six consecutive cases of meningococcus meningitis recovered satisfactorily with sulfonamide therapy, as did 50 consecutive cases of meningococcemia. The joint manifestations all responded to sulfonamide therapy.[34]

In one series, 80 patients with meningococcus meningitis and 32 with meningococcemia have been treated with sulfadiazine with one death in the former group and none in the latter.[50]

It has been suggested that the initial dose of sulfonamide be administered intravenously to patients with meningitis even though they appear only moderately ill when first seen.

Hill and Lever[125] reported 2 cases of unilateral arthritis of the knee joint among 68 patients with meningococcic infection seen in an Army Station Hospital in this country. Both resolved completely within two weeks without tapping; sulfadiazine therapy was used routinely.

These patients should be treated by splinting of the affected joints, repeated aspirations if necessary, and in all cases sulfonamides and/or penicillin should be given. The prognosis is good when these drugs are used. After the acute stage of the disease has passed, adequate physical therapy (heat, massage and exercises) will be of great value and should be continued until maximum function has returned.

Mumps.—Slight joint swelling and effusion occur occasionally. Joint pains are not uncommon and usually disappear within a week. Acute arthritis has also followed suppurative parotitis.[45]

Palindromic Rheumatism.—Hench and Rosenberg have described an unusual, oft recurring disease of joints and adjacent tissues in a study of 34 cases seen at the Mayo Clinic.[120] The outstanding features of this syndrome are *multiple afebrile attacks of acute arthritis and periarthritis* (and sometimes also of para-arthritis) with *pain, swelling, redness and disability usually of only one, but sometimes of more than one small or large joint in an adult male or female. These attacks appear suddenly and develop rapidly. They usually last only a few hours or days and disappear completely,* but recur repeatedly at irregularly spaced intervals (which may be short or long). *Little or no constitutional reaction* or abnormality is seen by laboratory tests. Even after years of disease and scores of attacks, no significant functional, pathologic or roentgen residues occur despite the transitory presence in at least some cases of an acute or subacute inflammatory polymorphonuclear exudate in the articular tissues and cavity.

The patients had their disease from a few months to twenty-five years before being seen by these observers. In 2 cases, the disease had been present from twenty-one to twenty-five years. *The great frequency of irregularly recurring short attacks, each with a complete symptomatic remission was the striking feature of the disease.* Most of these patients had scores of attacks during their illness and some had hundreds of attacks. There was a general average of about 23 attacks per year.

Although the frequency and regularity of attacks varied greatly, the character of the individual attacks was somewhat similar. In one instance, attacks occurred almost daily for two years, following which only 4 or 5 attacks occurred per year. In others, the frequency of attacks greatly

increased with the passage of time. The pace of the disease was relatively unchanged, however, in most cases.

In more than three-fourths of the cases, *the attacks lasted from a few hours up to three days.* Some of these were extremely painful despite their short duration. The minimal length of attacks was three hours. *Attacks lasting more than a week were exceptional.*

The *intervals between attacks* varied markedly in different cases and from time to time in the same case. In most of the individuals, it was impossible to predict when the next attack would occur. There was *no regularity of attacks* such as may occur in intermittent hydrarthrosis. The interval between attacks might vary from a few hours up to six years.

In most attacks, only one joint was involved, although from 2 to 4 or more joints were involved occasionally. Almost any joint in the body might be involved in an attack. The proximal phalangeal joints were involved most often, the metacarpophalangeal joints less commonly and only occasionally the terminal phalangeal joints. The knees were involved at one time or another in most cases as well as the feet, wrists, shoulders and ankles. The dorsum of the hand was frequently affected. Other joints affected included the elbows, jaws, sternoclavicular joint and cervical vertebral joints. The thoracic and lumbar spine were not specifically involved. Although any joint might be affected in the individual patient, most patients noted several favorite sites, particularly the fingers, wrists, shoulders, knees, toes and elbows.

The attacks began at any time in the day or night although in a large number, most of the attacks came on in the late afternoon or evening. *In most instances, the onset was sudden* with pain and swelling developing within a matter of minutes or an hour or so. The maximum severity of the attacks was reached rapidly (within a few hours in most instances).

Although the pain varied considerably from case to case and from one attack to another in the same patient, the *pain was generally severe* in most cases. In almost all of the patients, a variable degree of articular redness appeared. Articular swelling was of a moderate nature in most instances.

There was usually a *considerable degree of disability with the attacks* in most cases.

Little or no constitutional reaction was evident during the attacks. *Fever did not occur* despite the frequent severity of the attacks. None of the cases had loss of appetite. Seventy-four per cent of the group had lost no weight despite hundreds of attacks so that significant loss of weight is not a characteristic feature of this syndrome.

About a third of the patients experienced *recurrent swellings of para-articular soft tissues.* These attacks of para-arthritis usually appeared suddenly. The swellings varied considerably in size from the size of a dime to swellings considerably larger. Redness was usually present and pain was often severe. These bouts of para-arthritis usually lasted from six to twenty-four hours and then entirely disappeared. These lesions resembled somewhat those of urticaria or angioneurotic edema.

The para-articular swellings were seen most commonly at the malleoli,

bottoms of the heels, finger pads or distal phalanges, and at the flexor surface of the forearm just proximal to the wrist and thumb pads.

Small subcutaneous or intracutaneous nodules developed in a few patients and were usually very transitory.

Red blood counts and hemoglobin percentage were usually normal; the total and differential leukocyte counts showed a tendency toward relative lymphocytosis between attacks and either relative lymphocytosis or polymorphonuclear leukocytosis without eosinophilia during attacks. Blood uric acid was normal, as were serum calcium and phosphorus and phosphatase. Blood cholesterol and cholesterol esters were often somewhat elevated as were the fatty acids. The sedimentation rate was normal in 40 per cent of cases, slightly increased in 50 per cent and moderately increased in 10 per cent. The sedimentation rate was generally but not always slightly increased during or just after attacks. Urinalyses and blood Wassermann reactions were negative. *Roentgen examinations showed nothing significant* in over 90 per cent of the cases.

Unfortunately, histologic studies were inadequate to provide a complete picture of the pathologic process. It was concluded from biopsy studies that during an attack involving a joint, there is an acute inflammation with periarticular edema and gross capsular thickening. Large numbers of polymorphonuclear leukocytes appear within the synovial membrane, but eosinophils are not frequent. A fibropurulent exudate may appear within the joint cavity. With subsidence of the attack, the exudate is absorbed from the synovial fluid and the inflammatory cells disappear from the synovial membrane. Fibroblasts proliferate actively in the synovial membrane and capsule for a brief period, but the signs of inflammation in the synovia and capsule clear rapidly with subsidence of the attack and the joint structures eventually return to normal.

Similar sudden inflammation may affect *tendon sheaths*. The latter shows a non-specific inflammatory process with excess fluid in the tendovaginal space. The tendon sheaths return to normal with subsidence of the attack.

In palindromic rheumatism, the *articular cartilage was not destroyed* and *no pannus* was formed.

These attacks seemed to have *no seasonal relationship* and *season and weather bore no important effect* (in contrast to the condition in rheumatoid arthritis). A few of the women thought that their attacks were worse during the menses. In most instances, neither work nor trauma seemed related to attacks. Although about half of the group thought that a particular food acted as an offender, it was not possible to prove this. About half of the patients had no clinical signs of allergy. In most instances, cutaneous tests were found to be negative to various antigens including the inhalants and foods.

Some *points in favor of a possible allergic etiology of the attacks* included their sudden appearance and disappearance without residues; the thoughts of some of the patients that attacks occurred after eating particular foods; the presence of allergic manifestations in about half of the group and the occasional occurrence of hives or angioneurotic edema and arthritis in the same patient. However, the allergic history was negative in half of the group; skin tests to allergens were negative in about four-fifths of the patients; eosinophilia was not found in the blood or affected tissue; *epinephrine was without effect* when given during an attack and large doses of histamine would not provoke an attack; furthermore, histamine desensitization was without therapeutic effect.

There was no good evidence to suggest that the disease was related in any way to an infection.

Among the diseases which must be *differentiated* from palindromic rheumatism are rheumatoid arthritis, periarticular fibrositis, intermittent hydrarthrosis, rheumatic fever, gout, angioneural arthrosis (as described by Solis-Cohen[249]) and Kahlmeter's[142] group of patients with "allergic rheumatism." These two latter groups present striking similarities to the findings in palindromic rheumatism and may represent the same disease.

The te m palindromic is derived from the Greek word meaning "to run back," "recurring" or "returning." This term, palindromic rheumatism, has been approved for inclusion in the "Standard Nomenclature of Disease."

Various measures have been used in *therapy* of this condition, including removal of focal infection, vaccines, bee venom, heat, analgesics, epinephrine, ephedrine, amphetamine, ergotamine tartrate, various diets (low in purine, free of supposed food antigens), histaminase and sulfanilamide. *None of these, except heat and analgesics, have apparently given much relief.* Some of the patients who received *histaminase* thought that their attacks were less frequent and less severe. Temporary relief was noted in one individual after *typhoid vaccine* therapy, or *fever* therapy. Two of the patients had less severe and less frequent attacks when their *nervous system was relaxed* with phenobarbital or change of occupation or other forms of mental relaxation. One physician mentioned that his attacks disappeared temporarily while taking from 5 to 6.5 grams of *calcium gluconate* daily.

In 3 individuals, the attacks stopped spontaneously. In 5 others, attacks became shorter and less frequent. In 3 others, the attacks are as frequent but are shorter. In another 4, attacks are of the same duration but occur less frequently. In 7, the condition has remained unchanged, and 3 patients became worse; others could not be followed.

Thus, the *prognosis* for cure either spontaneously or by therapy is fair. Despite the great frequency and inconvenience and handicap during the attacks, there have been *no instances of permanent crippling.*

Periarteritis Nodosa.—In 101 cases of periarteritis nodosa, *27 per cent showed arthritis.*[102] Boyd summarized the clinical features in 395 reported cases of periarteritis nodosa. Seventy per cent of the patients were males. *Articular* and *neuromyositic symptoms* tended to appear early and were often the first or second symptom to be noted. Any joint may be involved. *As the syndrome progresses, the articular symptoms usually fade into the background.*[31] Actual *articular swelling* has been noted *in over 10 per cent* of these patients. *Diagnosis* is confirmed *by muscle biopsy*, although a

negative result does not absolutely exclude periarteritis nodosa. Unfortunately, most cases are diagnosed at autopsy.

Because of the widespread vascular involvement, clinical manifestations are bizarre and protean.[92]

Symptoms that are encountered frequently include *weakness, loss of weight, tachycardia, fever, anemia, leukocytosis, eosinophilia, hematuria, gastrointestinal symptoms, nephritis and polyneuritis.* In individuals with bizarre symptoms, the presence of an eosinophilia, especially with leukocytosis, should suggest the possibility of periarteritis nodosa. Unfortunately, eosinophilia has been reported in only about 12 per cent of these patients.[165]

Although the disease is not always fatal within a short period, these patients live an average of only eleven months after the onset of symptoms.[139]

Recent work by Rich and his associates suggests that this disease is probably a type of reaction pattern with the offending antigen probably being a foreign protein.

Pneumococcal Arthritis.—Discussed elsewhere (page 828).

Puerperal Septicemia.—Joint involvement is usually the result of metastatic infection via the blood stream, most commonly from hemolytic streptococci. Very mild or severe joint damage may ensue. If due to hemolytic streptococci, good results may be obtained with sulfonamides or penicillin.

Rat Bite Fever.—Arthritis has been reported as an uncommon complication of rat bite fever.[161]

Beeson reported 2 cases of rat bite fever in which Spirillum minus was isolated from the blood by mouse inoculation and blood cultures for Streptobacillus moniliformis were negative.[16] Although neither patient showed arthritis during the period of observation, one of the patients gave a history of swelling around the wrists, elbows, knees and ankles prior to admission.

Jeffers and his associates have discussed the etiologic and clinical relationship of Haverhillia multiformis septicemia to Haverhill and rat bite fevers.[3] The Spirillum minus (Spirochæta morsus muris) has been thought to be the causative agent in most patients stricken after the bite of a rat; an entirely different organism, Haverhillia multiformis (Streptobacillus moniliformis, Streptothrix muris ratti) has been recovered from a few of these patients.

Haverhillia multiformis is also the organism recovered from patients with Haverhill fever, although there was no history of rat bite in the individuals in the epidemic at Haverhill in 1926.

Sodoku is the name given by the Japanese to infection with Spirillum minus. Joint involvement (arthritis) has been mentioned in only a few patients with this disease. Fevers due to Haverhillia multiformis are characterized by septicemia with metastatic arthritis and morbilliform and petechial cutaneous eruptions. Arsphenamines have been effective in the therapy of rat bite fever due to either organism.

It has been suggested that the original epidemic of Haverhill fever was milk borne.

Reiter's Disease (Spirochætosis Arthritica).—This is a triad of *urethritis, conjunctivitis* and *arthritis* (usually occurring in this order). There is a severe inflammation of the anterior and posterior urethra, often associated with cystitis. The urine is cloudy and contains shreds. The abundant purulent discharge disappears quickly in most cases. No gonococci have been demonstrated in the urethral discharges.

The *conjunctivitis* is attended with abundant pus and may lead to a keratitis or an iritis. Conjunctivitis may be lacking in some cases.

The *joint involvement* is usually *multiple,* involving especially the knees, feet, ankles or hips. The joints become painful and swollen but there is no permanent stiffness or ankylosis.

Although Reiter's original cases were severe, most other reports were of a milder disease with a moderate degree of fever (which may be of a septic type). There is a tendency to recurrence but these patients recover spontaneously in a few weeks. Treatment is entirely symptomatic.

Scarlet Fever.—Healey reported that 3 per cent of young adults being immunized against scarlet fever developed multiple joint pains as a reaction to one or more injections of the sterile filtrate of broth cultures of scarlet fever streptococci.[108] The majority of individuals with this reaction had noted joint pains previously associated with rheumatic fever or streptococcic infections of the nose and throat. Such individuals appeared to develop rheumatic affections such as heart disease, polyarthritis and erythema nodosum more often than other persons not similarly sensitized.[214]

From 1 to 5 per cent of patients with scarlet fever develop joint complications,[221] beginning about the fourth to the tenth day of the disease. These may include a mild, migratory polyarthritis resembling rheumatic fever;[201] purulent arthritis with hemolytic streptococci cultured from the joint fluid; or recrudescence of a rheumatic fever. The use of sulfonamides and antitoxin is discussed in the chapter on Sulfonamides.

Scleroderma.—The stiffness and disability resulting from scleroderma has been described in a previous section. In the early stage, it is often mistaken for rheumatoid arthritis. The lack of fusiform joint swellings, normal sedimentation rate, *x*-ray findings and tightly drawn skin and tense subcutaneous tissues aid in differentiating the two.[134,194]

Septic Arthritis.—Discussed elsewhere (page 834).

Smallpox.—Serous or purulent arthritis has been reported occasionally.[151]

Spirochetemia.—Williams reported the finding of *fusiform bacilli and spirochetes* (of the type found in Vincent's angina) *in the cultures of blood from 2 patients;* in one of these patients, the organisms were recovered from the blood eleven times, and successful subcultures of the organism were made.[271] These organisms were slightly pathogenic for rats. In these 2 patients with *fusospirochetemia, myalgia and a migratory arthritis was observed,* together with the sudden development of fever, headache and prostration. In one of these splenomegaly appeared, with jaundice, pleurisy

and a hemorrhagic eruption. The source of the infection was considered to be the bite of a rat in one instance and the tonsils in another.

Causative organisms have been cultured from the blood in but a few of the spirochetal diseases.[163] Reiter cultured spirochetes from the blood of a patient with cystitis, arthritis and conjunctivitis.[213] Palmer demonstrated many times the presence of spirochetes in the blood of a patient with a prolonged illness with vague muscle and articular pains, relapsing fever, a rash resembling that of German measles, pericarditis and arthritis.[199]

In Williams' patients, the initial symptoms resembled those of rat bite fever, relapsing fever and Weil's disease. Fever developed suddenly, associated with chilliness, prostration, headache and muscular and joint aching, followed subsequently by a migratory arthritis. The first of these patients had an intermittent type of arthritis for two years, while the second was free of arthritic symptoms after a month. In both of these patients, *clinical improvement followed treatment with arsphenamine* and fusiform bacilli and spirochetes apparently disappeared from the blood.

Striatal Syndrome.—De Haas reported the history of a man aged forty-five who underwent a gastric resection under *nitrous oxide anesthesia.* He was cyanotic during the anesthesia and there was arrest of breathing for two or three minutes. Artificial respiration and cardiac massage brought about first arterial pulsation which was followed by return of respiratory movements. *Following the recovery from operation,* the patient *developed a typical striatal syndrome* and mild signs of a pyramidal tract lesion. This patient, who had never had articular complaints previously, developed *four to five weeks after the operation, redness and swelling of various joints of the arms and legs, associated with fever, and was classified as a rheumatoid arthritis.* It was suggested that there may be some connection between lesions of the corpus striatum as found in paralysis agitans and rheumatoid arthritis.

Subacute Bacterial Endocarditis.—A non-suppurative arthritis may occur, due to emboli lodging in or around the joint. The inflammation usually subsides in a few days. Goodyear reported purulent arthritis due to streptococcus viridans in an infant seven weeks old.

Sulfatoxic Arthritis.—With the use of prolonged sulfonamide therapy, one must be on the alert for the development of the so-called *sulfatoxic arthritis.* Long observed the occurrence of painful joints during sulfanilamide or sulfathiazole therapy.[170] Glicklich and Sherman reported a patient with chancroidal infection who developed tender swollen elbows and knees during sulfathiazole therapy. The fluid was sterile and contained only a few polys per high power field. Other manifestations of *drug toxicity* were also present.

The joint pathology subsided with cessation of sulfonamide therapy, and recurred again a week later after administration of a test dose of 3 grams of su'fathiazole; the joint swelling again promptly disappeared within twenty-four hours after stopping the sulfonamide. We have observed this same phenomenon in several patients with meningococcic meningitis who received sulfadiazine; the swollen joints (elbow and knee) contained

a sterile fluid and the joint swelling disappeared promptly after withdrawal of sulfonamides, leaving no detectable joint pathology.

Syphilis.—Discussed elsewhere. (See Chapter XLVII.)

Typhoid Fever[187,228,229] and Paratyphoid Fever.[168,217]—Typhoidal arthritis is uncommon. Monarticular or polyarticular arthritis may occur in as many as 12 per cent of cases.[38,133,227] Although this is usually serous, suppurative arthritis and ankylosis have been noted.[154] In some instances, typhoid spondylitis has appeared several months or years after convalescence from the typhoid fever.[173,239,256] In these cases, the spine must be fixed in a plaster shell or by a brace until the disease becomes quiescent. The lower portion of the spine is most often affected.

Typhus Fever.[237]—Joint pain may occur in typhus fever. Purulent joint effusion has been observed in one instance.

Ulcerative Colitis.—Discussed elsewhere (page 834).

Whooping Cough.—Joint involvement is very rare.

Yaws.—Joint pains may occur. The skeleton is affected in 20 per cent of cases. Articular surfaces may be destroyed.

Miscellaneous.—Davidson reported a case of "infectious" polyarthritis associated with chronic bilateral bronchiectasis.[52]

Arthritis has also been caused by micrococcus tetragenous infection;[211] B. suipestifer[95,248,262] (during the first year of life in a prematurely born infant); filariasis; dracunculosis;[141] B. pyocyaneus[22,67,205] (with pyarthrosis); micrococcus catarrhalis[46,250] (only 2 case reports in the literature—a purulent arthritis resembling gonorrheal arthritis clinically); and tularemia.[68] On several occasions B. tularense have been recovered from bursal fluid Echinococcus involvement of bones and joints has been reported.[219]

Salmonella Schottmülleri has been isolated from a sacrolumbar lesion of twenty-four years' duration.[59] This organism has been recovered from a patient with purulent arthritis.

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CHAPTER XLVI

TUBERCULOUS ARTHRITIS

An analysis has been made of the *bone and joint tuberculosis seen in a general tuberculosis hospital* during fifteen years.[78] The following points were noted:

1. Three and seven-tenths per cent had single or multiple tuberculous bone and joint lesions, usually secondary to a tuberculous focus in the lungs.
2. Of those with bone and joint tuberculosis, *74 per cent had chronic pulmonary tuberculosis* and 26 per cent had no pulmonary involvement.
3. The *frequency of joint involvement* was: spine—33 per cent; hip—15 per cent; fingers, toes and long bones—11 per cent; knee—8 per cent; sacro-iliac joints—8 per cent; elbow—4 per cent; shoulder—3 per cent; ankle—3 per cent; wrist—2 per cent with other regions involved in a small percentage.
4. The bone and joint involvement was most prevalent in the second and third decades.
5. The outcome is serious and the complications are most frequent when moderately advanced pulmonary tuberculosis is present; conservative measures should be employed in these. The prognosis is also more serious when tubercle bacilli are present in the sputum, in those with a draining sinus or multiple abscesses or in those with extrapulmonary tuberculosis.
6. Almost half of the 160 patients with bone and joint lesions died; 17 per cent were well; 21 per cent were improved, and 13 per cent were unimproved.

Tuberculosis in man is now due almost entirely to the human type of bacilli as bovine tuberculosis affects only 0.4 per cent of cattle.[6]

Repeated roentgenograms and synovial biopsies may be needed in doubtful cases; guinea pig inoculations and microscopic examination of synovial tissue may be necessary for diagnosis.[33] *Early tuberculous arthritis is still extremely difficult to diagnose;* there is no infallible diagnostic method in such cases. In a study of *100 cases of bone and joint tuberculosis in individuals under sixteen years, 55 per cent* of the lesions *involved the spine* and 21 per cent the hip. In a similar group in adults, 52 per cent involved the spine and 18 per cent the hip. The younger group showed a much larger incidence in the small tubular bones of the hands and feet, while the adults showed more sacro-iliac and knee-joint involvement.[65]

The *most commonly involved vertebræ* were from the sixth dorsal to the fifth lumbar. Multiple involvement was common.

Osseous and pulmonary tuberculosis occurred in 42 per cent of the children and in 55 per cent of the adults.

A paravertebral abscess was found in 49 per cent of the spinal involvement in the pediatric group and in 71 per cent of the adult group. At times, it was the only roentgen evidence of underlying bone disease.

Diagnosis of Tuberculous Arthritis

Slow, **insidious development** *of a* **monarthritis;** several joints involved occasionally. About 85 per cent are monarticular.

Family history of tuberculosis, or previous personal history of *pleurisy with effusion, suppurative cervical adenitis or fistula in ano.*

Associated visceral tuberculous lesion (lungs, lymph nodes) in almost 50 per cent of cases.[48]

Order of frequency of joint involvement: *spine, knee, hip,* elbow, ankle, sacro-iliac, shoulder and wrist (temporomandibular joint is rarely involved).

History of preceding trauma in 50 per cent.

Symptoms: Local: Presence of one or more of the following: *Doughy swelling of the joint* due to enlarged synovia rather than to fluid; *pain; stiffness; heat; limp on walking;* muscular spasm and limitation of motion; *tenderness* to palpation; muscular atrophy (sometimes early and marked). Pain and tenderness are late symptoms.[21,44]

General: Slight or no fever; loss of weight.

Present in approximately 4 per cent of all cases of tuberculosis.[15]

Most common in the first fourteen years of life, but may occur at any age. Tuberculous arthritis has been reported appearing in men over fifty years of age.[25]

Additional aids in diagnosis: Biopsy of joint tissue; guinea pig inoculation of joint fluid; microscopic examination of joint fluid for tubercle bacilli; positive tuberculin test; normal serum phosphatase.[61]

Evidence against tuberculous arthritis: Presence of considerable amount of joint fluid; arthritic involvement of four or more joints.[45]

Four clinical stages have been described.[79]

Invasion: physical, but no roentgen signs are present. Duration: weeks or months; occasionally several years.

Tissue destruction—duration, several months; roentgenograms show soft tissue involvement with absorption and cavitation in cartilage.

Quiescence—absence of joint swelling; no further bony destruction shown by serial roentgen-rays.

Healing—joint is not painful; no swelling is present. Fibrous tissue develops from tuberculous granulation tissue; fibrous ankylosis occurs. Tubercle bacilli may remain for years in such fibrous tissue.

With erosion of the cartilage and exposure of the ends of bones, there is increased pain and stiffness and muscular relaxation, especially in sleep; this latter permits the roughened ends of bones to come into contact, awakening the child with severe pain, and causing cries and jerks at night.

Progressive destruction (with remissions and exacerbations) unless properly treated.

Preservation of articular cartilage (joint space) until late in the disease.

Etiology.—The decrease in the incidence of tuberculous arthritis may be due to the more rigid control over dairy herds; most cases of surgical tuberculosis were attributable to the bovine strain.[37] Tuberculous arthritis

may result from extension of a tuberculous process in adjacent bone, or may be a primary involvement of the synovial membrane. Primary synovial tuberculous arthritis is probably more common in children than in adults. Although it was formerly thought that most instances of tuberculous arthritis were secondary to tuberculosis of bone, it is often difficult to determine the site of the primary focus.[73] Henderson has stated that tuberculous arthritis is always secondary to tuberculosis elsewhere in the body; primary sites include the mesenteric and bronchial lymph nodes.[48]

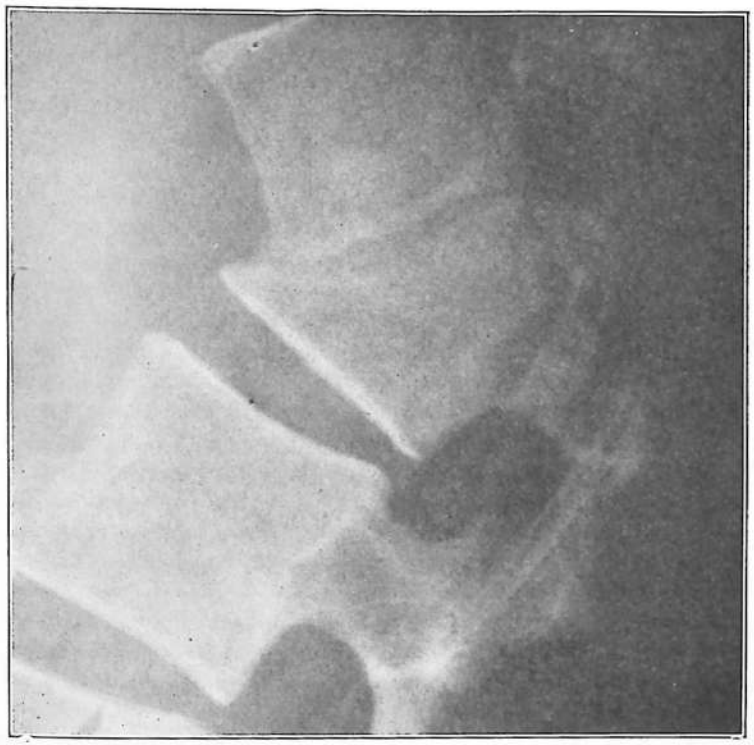

FIG. 185.—Old Pott's disease with deformity in a patient aged fifty-four years. There is a lesion involving the eleventh thoracic vertebra due to a healed Pott's disease. There is no cold abscess formation. The intervertebral disk between these vertebræ has disappeared. There is no encroachment on the spinal canal.

Pathology.—The pathology of tuberculous arthritis has been well described by various workers.[4,31,54,66] Allison and Ghormley believe that all cases of joint tuberculosis should be regarded as involvement of the synovial membrane, bone and cartilage.[5]

Whether the tuberculosis is primary in the synovial membrane or in the bone there develops *a diffuse tuberculous synovitis with granulation tissue* which *attempts to spread over and to destroy the articular cartilage.*[72] In those joints in which the articular cartilages are in close contact over a

large area of their surface, the granulations are prevented from spreading over the surface of the cartilage, but erosion occurs at the margins. In other joints in which large surfaces of the cartilages are not in apposition, the granulation tissue spreads over and erodes the cartilage.

Subchondral infiltration of granulation tissue is one of the earliest changes in tuberculosis. Subchondral granulations absorb the bony articular cortex and lower portions of the cartilage, and in some cases, result in detachment of the cartilage. Because of the absence of proteolytic ferments in tuberculous exudates, this loosened cartilage may persist for months, but is finally destroyed. Grooves may be formed on the articular surfaces of the bone due to invasion and absorption by the tuberculous process.

Massive bony invasion at pressure points may occur in the knee and hip, producing large bilateral areas of necrosis. True osteophyte formation does not occur at the margins of the articular cartilage in tuberculous arthritis.

In one series of 224 cases, the frequency of joint involvement was as follows: *spine*, 49 per cent; *knees*, 20 per cent; *hips*, 11 per cent; sacroiliacs, 7 per cent; tarsus, 6 per cent; ankles, elbows, wrists, each 2 per cent; shoulders and symphysis pubis, under 1 per cent.[15] Involvement of an ankle, knee, elbow and spine has been reported in the same individual.[80]

Although the tuberculous process may be quiescent, it rarely heals. If secondary sequestra are present and the process remains active, bony ankylosis almost never occurs although fibrous ankylosis may be noted when the articular cartilage is completely destroyed.

Roentgen Findings in Tuberculous Arthritis.— *The roentgen picture is not pathognomonic* and may include osteoporosis, areas of rarefaction (due to abscesses) and narrowing of the joint space. *Active early tuberculosis may be present in bones and joints with negative roentgen findings. Increased radiability and soft tissue swelling are usually the earliest roentgen signs. Later* one notes *marginal erosion with marked irregularities of contour along the joint surfaces, marked haziness of the joint,* evidence of *subchondral invasion* with apparent separation of the cartilage and *narrowing of the joint space.*

In joints with extensive contact of articular cartilage, there may occur: regional atrophy of bone, decreased density of the bony articular cortex and preservation of the normal cartilage space.

In joints with extensive areas of non-contact of articular cartilage (as the knee), one may see: regional atrophy of bone, narrowing or complete obliteration of the cartilage space, and decreased density of the bony articular cortex where the cartilaginous surfaces are not in contact, with preservation of bony articular cortex and cartilage space in the regions of contact (condyles and tuberosities).

If secondary invasion of the bone occurs at the traumatized points of contact, large bilateral areas of necrosis occur at opposing points in the bones; detachment of such areas forms "kissing sequestra."

TUBERCULOSIS OF THE HIP JOINT

The *primary lesions* are usually in the *neck or head of the femur;* these may result in complete disappearance of the head of the femur and destruction of the acetabulum. Other important points include:[33]

Early synovial invasion.

Marked rarefaction of bone with obliteration of landmarks.

Misshapen or entirely absorbed femoral head.

Occurrence in children, in most cases, under the age of ten.

Early symptoms: *limp; pain in the knee and thigh* from pressure on the obturator and anterior crural nerves; *muscle spasm; fulness in the groin; tenderness on pressure over the hip;* slight flexion and abduction of the thigh; flattening of the buttock and disappearance of the gluteal fold on the affected side; *limitation of all motions at the hip,* especially of rotation and abduction; at a later stage, adduction deformity of the thigh and cold abscesses or sinuses in some cases, pointing most often on the outer side of the thigh.

Treatment:[9] Rest, high caloric diet, heliotherapy, fresh air, etc.; *recumbency in bed with extension* in the axis of the deformity for several months until evidence of activity has disappeared, followed by cautious ambulatory treatment with no weight bearing for a long period. This is aided by the use of a high shoe on the healthy side, braces and crutches allowing the affected leg to swing free.

The assistance of an *orthopedic surgeon* should be enlisted in all of these cases. *The best results in tuberculous arthritis of the hip follow the production of solid bony ankylosis.*[1] In tuberculous involvement of the hip, immobilization may be procured by a plaster splint with the addition of traction to secure separation of the diseased surfaces. Months or years are required for fusion which is the ideal treatment. Motion in the hip joint will result in relapses and deformity. The procedure of choice in the hip, even in children, is surgical fusion; this heals rapidly.

In a series of 76 patients with proved tuberculous arthritis in 77 hip joints treated by arthrodesis, good end results were obtained in 52 per cent, fair results in 22 per cent, poor results in 8 per cent, and a mortality of 18 per cent.[10] It was noted that *the outlook was more favorable if* the arthrodesis was not done before eight years of age or after thirty years, if there was no active pulmonary tuberculosis or sinus or cold abscess about the hip at the time of operation and if there was no secondary pyogenic infection about the hip.

TUBERCULOSIS OF THE KNEE JOINT

Tuberculosis of the knee is a *common type of joint tuberculosis in adults.* It occurs in 11 per cent of all tuberculous bone and joint lesions. Synovial tuberculosis progresses to bony changes in 45 per cent of cases.[64] *Tuberculous arthritis of a low grade may persist in a knee for years with very little discomfort. Local signs include* heat, swelling, deformity, stiffness, pain, limping, muscular spasm, restriction of motion, and occasionally an intra-articular

effusion. Flexion of the knee and contraction of the hamstring muscles develop. Slight jarring of the joint may produce excruciating pain.

Early diagnosis of tuberculous arthritis is aided by use of synovial membrane biopsy (which may show tubercles in as high as 82 per cent of cases) and aspiration of joint fluid for special cultural methods of tuberculosis and guinea pig tests.

Key summarized his findings in *adults with tuberculosis of the knee joint.*[51] *Pain* was the first symptom in 70 per cent of cases; this was usually insidious in onset. In 22 per cent of cases, *swelling* of the knee was the only initial symptom. *Stiffness* was the initial symptom in 10 per cent. *Local heat* was always present and there was usually *gross limitation of motion*. About 6 per cent had abscess formation at an early stage.

In 87 per cent, the patient's general physical condition was good. Diagnosis of early cases was often extremely difficult; the *most valuable laboratory aid* was bacterial examination of the pus from an abscess (including guinea pig inoculation). Key thought that biopsy of synovial membrane was not without risk. *This disease may be confused in adults with* rheumatoid arthritis, gonorrheal arthritis, syphilis, hemophilia, traumatic lesions and neoplasm.

At present, most individuals treat tuberculous arthritis in children conservatively for a time, but treat adults surgically unless an upper limb is involved—when one may temporize for a while. Tuberculous arthritis is a serious disease with a high mortality. Although operation alone will not cure the individual, it may turn the tide in his favor.[16]

Haas performed successful arthrodesis of knees for tuberculosis in children as young as two and seven-tenths years, without loss of growth (or in some cases, not more than $\frac{1}{2}$ inch).[39]

Ober reported some cases of tuberculosis of the knee in which complete recovery occurred without operation, but pointed out that in these cases the tuberculous process did not extend into the knee joint or through the epiphysis, but extended laterally and decompressed itself through the side of the bone medially or laterally. Fusion is not done in these patients as healing occurs without any disturbance to the knee joint.

Ober believes that *operation on a tuberculous joint is contraindicated if there is any indication of active pulmonary tuberculosis; a chest x-ray should be taken in all patients with tuberculosis of joints.*[67]

Attention must be paid to the *general treatment of the patient* and not only to his affected joints. Rest of the joint with splints, extension by weights and rest in bed is usually the treatment of choice in children. *In adults, conservative treatment has been abandoned because the results are poor.* If the knee is involved, *resection of the affected articular ends of bone with bony ankylosis of the remaining parts is the ideal treatment in adults.* The patient can often return to work within a year following such an operation. Immobilization in a brace or cast for months or years has been used in children, with the knee in complete extension to prevent flexion deformities. Results of conservative treatment in children are usually not good.

The **operative procedure of choice** *is arthrodesis by surgical resection.*[86] As contraindications to operation, Henderson listed debilitated patients, short expectancy of life, very old or very young patients or the presence of draining sinuses. At the time of operation, it is advisable to remove as much of the tuberculous tissue as possible; this will produce quicker fusion and will lessen the tendency to abscess formation later. Henderson has described the technic of this operation in 274 cases of tuberculosis of the knee. There was no fatality in his series. Good bony fusion was obtained in almost 90 per cent of those patients who could be followed.

Skeletal lesions seen in the roentgenogram include decalcification and have almost no differential value; bone degeneration appears too late to aid in diagnosis. In the soft tissues, the *x*-ray may show a dirty opaqueness, particularly when both knees are contrasted.

It is rarely possible to identify the tubercle bacillus directly from the intra-articular fluid. Culture for tubercle bacilli or guinea pig inoculation is much more likely to reveal the organisms. Although synovial biopsy is not characteristic macroscopically, the microscopic appearance may be very suggestive of tuberculosis.[23]

In individuals from young adult life to middle age, **orthopedic surgeons are agreed that the treatment of choice is surgery to produce femorotibial synostosis.** *In the aged,* tuberculosis of the knee joint is usually treated (in most instances) by *amputation* at the thigh. It is possible that prolonged use of newer chemotherapeutic measures (sulfonamides, penicillin and other drugs) may result in an arrest of some tuberculous processes; the effect of these agents on tuberculosis of joints has not yet been reported.

Tuberculosis of the knee joint is most common in infancy and childhood. There is still a *lack of general agreement as to the advisability of surgical measures in treatment during the years of growth* while epiphyseal cartilages are still present. Most surgeons prefer not to resect the knee in individuals during the period from birth to epiphyseal closure because some have claimed that: (1) tuberculosis of the knee heals under conservative treatment in the child, and (2) that the resulting deformity and shortening following resection in the child are too crippling to justify this procedure.[53]

Unfortunately, no reliable statistics are available to show that proved tuberculosis of the knee in the child or infant heals spontaneously or that its course differs clinically from tuberculosis of the knee in later years. McKeever employed conservative treatment in 46 patients for an average of three years per patient; not one knee joint was found which was free from clinical signs of active disease or which permitted the patient at the end of this time to be up and about without apparatus of some type. In 39 of these 46 patients after conservative therapy, it was possible to recover tubercle bacilli or to demonstrate these organisms microscopically when they were subjected to resection later.

McKeever found that in 47 patients in the first ten years of life suffering from proved tuberculosis of the knee joint, the most frequent age of onset was within the first four years.[59] In 46 of these patients, no satisfactory results were accomplished by conservative therapy or by surgical procedures

short of economical resection. The latter procedure resulted in healing of the tuberculosis in 32 of 34 patients.

Following resection, the knee joint of a child ankyloses very readily and the percentage of successful fusions following a single operation is high. In his series, marked shortening occurred in only 30 per cent of the infants and children treated. *McKeever concluded that economical resection of the knee was not contraindicated prior to epiphyseal closure* and that this procedure should not be withheld until the age of fifteen from children with tuberculosis of the knee joint. *He advised that it be practiced early after a period of conservative treatment* which is adequate to cause an abatement of signs and symptoms of the acute disease.

Hallock agreed that *tuberculosis of the knee should be operated on even at an early age* and that *fusion is the only method which will give a large percentage of cures* in these cases. He has never seen a recurrence of tuberculosis in a knee that became fused in an experience of more than twenty-five years in a large orthopedic hospital.[41] He believed that shortening following the operation was probably caused by delaying the operation too long inasmuch as the disease had already produced disturbances or destruction of the epiphyseal lines before fusion was performed. In his opinion, one of the methods of preventing marked shortening was to perform arthrodesis before the disease had partially or completely destroyed the epiphyseal lines. He advised fusion of the knees in these children at 180 degrees to avoid flexion deformities through the distal epiphyseal line of the femur, followed by braces for a number of years in very young children to prevent slipping through this epiphyseal area.

Erlacher advised early radical extirpation of the tuberculous focus in the capsule or region of the joint;[27] this was possible in approximately 25 per cent of cases and produced firm and rapid healing, often preserved normal motion and greatly decreased the duration of treatment. Short wave therapy,[85] fever therapy[26,71] and gold[30] are of very little or no value in treatment.

TUBERCULOSIS OF THE SPINE

(Pott's Disease; Tuberculous Spondylitis)

This is seen most often between the ages of two and ten. It is the *most common form of tuberculous arthritis.* Trauma may be a predisposing factor in this disease.[81]

Approximately 1 per cent of all deaths from tuberculosis in the United States are due to Pott's disease.[6] Pott's disease is considered to be a local manifestation of a chronic general tuberculous infection.[50]

The lesions occur most commonly in the bodies of the vertebræ although the intervertebral disk may be affected first;[13] in more than one-half of these cases, the thoracic vertebræ are affected. The anterior border of the vertebral body is most commonly involved. A number of vertebræ (6 to 8) may be damaged in the dorsal area. Collapse of these results in angulation of the spine with shortening of the trunk. Deformities are produced by

caseous softening of the bodies of the vertebræ with the added pressure of weight bearing.

Isolated tuberculosis of a vertebral spinous process has been reported.

Cold abscess formation and *paralysis* are the two *commonest complications.* Such an abscess in the cervical region may be found laterally in the neck or in the retropharyngeal space. A dorsal abscess may remain localized, or may travel along the anterior aspect of the spine forming a psoas abscess; in some cases, these point in the thigh. Psoas abscesses have been reported to be so large as to cause bulging or filling of the lower portion of the abdomen.

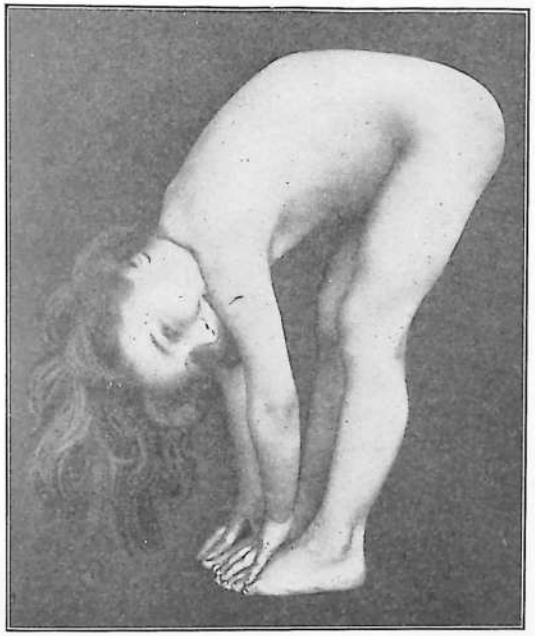

FIG. 186.—Incipient Pott's disease. Showing the break in the contour of the spine of which the normal flexibility is but slightly impaired. (Whitman.)

Symptoms.—Slight *abnormalities in gait;* awkwardness in walking; listlessness.

Rigidity and *upright position of the spine* even when bending.

Pain and weakness in the back, and pains and crying at night.

Abdominal pain (referred along the intercostal nerves); occasionally referred to the buttocks or knees.

Spinal deformity (*kyphosis*).

Tenderness and *muscular spasm* over the affected area.

Occasionally paralysis of the legs may occur from pressure upon the spinal cord by granulation tissue or cold abscess.

Treatment.—In children, treatment consists of *rest in bed in recumbency on a Bradford frame with extension of the head and feet.* Rest of the affected

parts in children with Pott's disease is secured by the position of hyperextension on a convex frame or sand bags, or a plaster bed or plaster jacket. Fresh air, heliotherapy and a high caloric diet are essential in these youngsters.

Ambulatory treatment may be begun after several months of freedom from symptoms. A plaster jacket is used for support when the patient is out of bed. Spinal support must always be used when the patient is up. When ambulatory, a light brace may be used for spinal support and for prevention of dangerous movements of the spine.

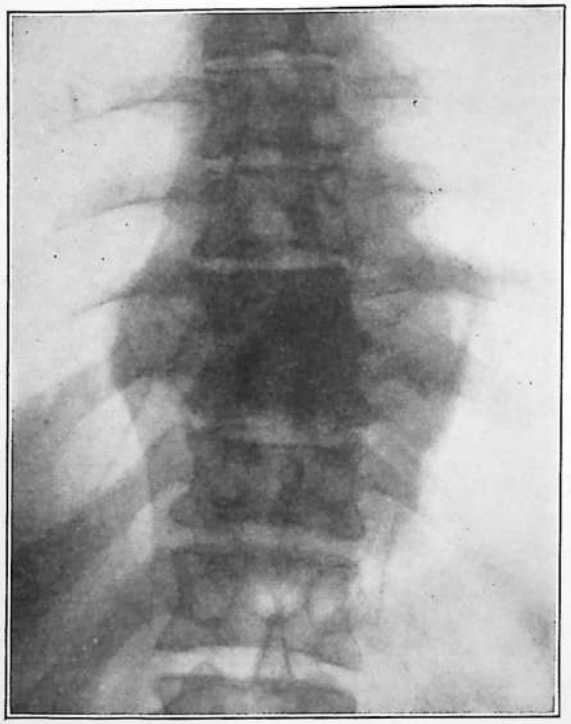

FIG. 187.—Tuberculosis of the thoracic spine in an adult. (Holmes and Ruggles.)

Harris and Coulthard studied the bone and joint lesions of 296 patients who were followed for a period of from one to ten years. Only 8 per cent of these were in children (as their hospital accommodated chiefly adults). Their *best results were obtained by* treating bone and joint tuberculosis first by *rest* and controlling other tuberculous foci in the body; *then, by operation to obtain bony ankylosis* of the affected joint. Fibrous ankylosis or unstable joints often marked the end result of non-surgical treatment; the results of those with bony ankylosis were superior to those treated non-surgically.

In 88 per cent of patients, tuberculosis of the spine was arrested by spine bone graft.[43]

Over 40 per cent of the patients had *pulmonary tuberculosis;* this *is the most serious of the tuberculous complications.* Prognosis is affected favorably by the adequate treatment of abscesses (to prevent secondary infection of bone) and by freedom from multiple tuberculous lesions in the body. Secondary infection of the spine or of the sacro-iliac joint or hip rendered the prognosis poor. Secondary infection of the knee, ankle or foot required excision or amputation. It will be interesting to see how greatly the introduction of the sulfonamides and penicillin will aid in those patients with a complicating secondary infection.

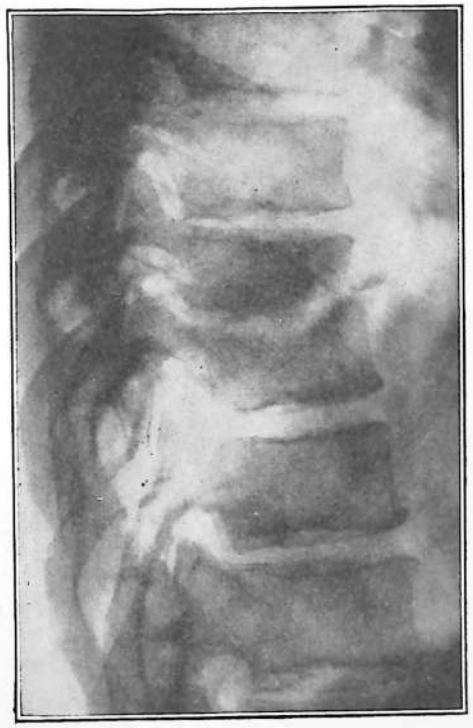

FIG. 188.—Lateral view of the same patient as in Figure 187. (Holmes and Ruggles.)

About a third of the patients were free from multiple tuberculous foci.

Treatment of tuberculous spondylitis must include relief of pain; healing of the spinal lesion; prevention or cure of deformity, of complications and of relapses as well as prolongation of life.[36]

Prolonged general care is always advisable in Pott's disease; surgical fusion should be postponed until the resistance of the individual is increased and until the lesion becomes stationary or begins to heal.[14,58,60] Surgical fusion of the vertebræ should be considered only as an aid and not the cure of Pott's disease.

When active tuberculosis is localized to the spinal column, the mortality

rate in Pott's disease may be less than 10 per cent;[17] if active tuberculosis is present elsewhere in the body, the mortality may surpass 50 per cent. It is evident that *fusion operations should not be attempted until tuberculosis of the lungs or other organs is controlled.*

In older children and in adults, arthrodesis is advised in order to fuse the bodies of the vertebræ. Unfortunately, it is not possible in this case to remove the diseased bone. In adults, following spinal fusion, an apparatus to support the spine should be worn for at least a year. Haas favors early spinal fusion even in children.[38]

Spontaneous healing of tuberculous spondylitis is rarely seen.

Abscesses complicating Pott's disease may be treated as follows:

If small and causing no symptoms—no treatment.

If large, aspirate with a small bore needle; repeat, if necessary.

If necessary, incision and evacuation with immediate suturing of the wound to prevent sinus formation.

Mayer reported that if operation was performed during active tuberculosis, fusion did not alter the course of the tuberculous process, but that if it was done during the reparative stage, it was beneficial in that it strengthened a weak spinal column. *In most reported series, mortality rates are lower among patients who have been operated upon than in those treated without operation.* Meyerding preferred conservative treatment for children but noted that results following fusion operations were better for adults.[60]

Swett reported that healing rarely occurred in the presence of an abscess and accordingly advised early aseptic evacuation of all abscesses. On the other hand, Meyerding suggested that the abscesses be left alone unless they cause symptoms of pressure or absorption of bone.

Paralysis of the legs complicating Pott's disease usually results in recovery within several months following the use of a convex frame.

Finkelstein made a careful ten year study to determine whether fusion operations shortened the course of tuberculous spondylitis or whether such operations prevented deformity.[29] Twenty-six patients had Albee type fusion operations while 17 others of similar age group, severity and situation of disease were treated by conservative measures only (rest on frames, sunlight, good food, fresh air, etc.). *The non-surgical group was cured in 25 per cent less time* than the surgical group so that the operation in their hands seemed to prolong, rather than shorten the course of the disease. The disease in the vertebræ seemed uninfluenced despite firm fusion. *Approximately the same number of recurrences, spinal deformities, abscesses and paraplegias developed in the conservatively treated and the surgically treated groups.*

Swett and his associates discussed the end results in 350 cases of Pott's disease, evaluating the *merits of operative and non-operative treatment.*[82] They found that *healing rarely occurred when an abscess was present,* especially if there was any evidence of calcification near the lesion. Healing usually followed disappearance of the abscess. In this series, *healing occurred in about 35 per cent in both the operated* (posterior spinal bridging) *and non-operated cases.* They believed that the dominant factor in the

healing of Pott's disease was concerned with the presence of and the care of the abscess. Meyerding agreed that surgical fusion was only an aid and not a cure for tuberculosis of the spine, and believed that fusion could be done without aggravating the disease. *Rest,* a *high caloric* diet and *heliotherapy* are the paramount requirements for these patients.[60] A symposium on the subject of Pott's disease which appeared in *The Journal of Bone and Joint Surgery* in 1940 should be of special interest to workers in this field.[2,3,6,14,17,36,42,50,58,83]

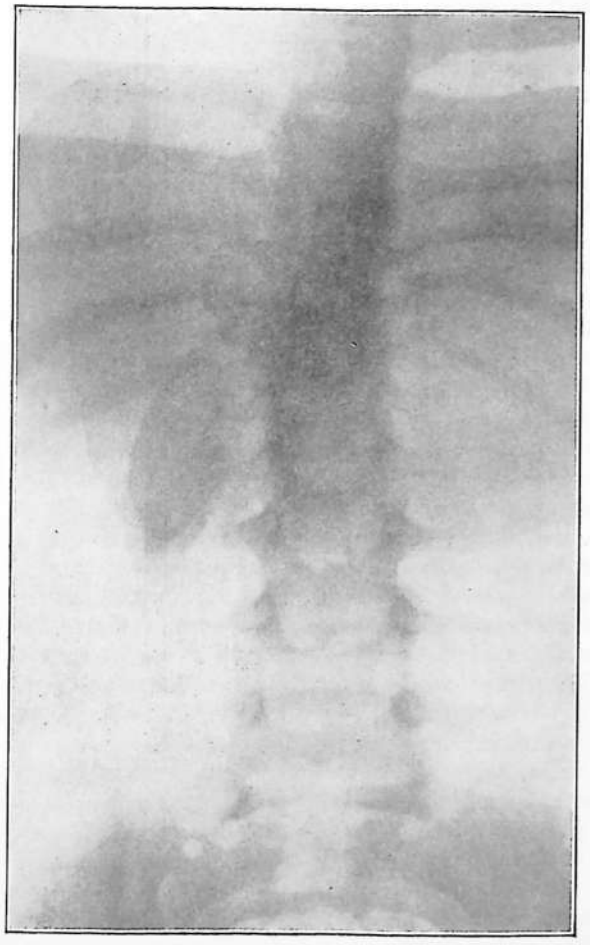

FIG. 189.—Tuberculosis of the spine showing calcification in a small psoas abscess. (Holmes and Ruggles.)

MISCELLANEOUS JOINTS

Involvement of the *shoulder or ankle joint*[49,88] presents no unusual symptoms. In the shoulder, arthrodesis is the treatment of choice in adults, and immobilization in abduction in children for months or years.

Tuberculosis of the shoulder must be differentiated from other chronic disabilities such as traumatic and rheumatoid arthritis, degenerative joint disease, and periarticular calcification and bursitis.[63] In uncomplicated cases, the prognosis after treatment is usually good unless active pulmonary tuberculosis or local abscess is present. In uncomplicated cases, the operative procedure of choice is extra-articular fusion by an osteo-periosteal graft from the acromion to the neck of the humerus. If postoperative splinting is proper and of sufficient duration, bony ankylosis and intra-articular healing almost always occur in four to eight months. The arm is held slightly forward and abducted to 70 degrees.

Rest and immobilization are also advised in involvement of the **elbow joint** in children. Tuberculosis of this joint often tends to result in extensive early bony destruction with abscess formation. In adults, an arthrodesis is preferable, with fusion in flexion. If strenuous work is not required of the individual, a pseudarthrosis may be advisable with removal of large areas of the diseased tissue in the humerus, radius and ulna.

Destruction of a finger joint without new bone formation in the region should be suspected as being of tuberculous etiology.

Peritrochanteric bursæ may be involved alone by tuberculosis.[47]

The **wrist** is rarely involved in tuberculous arthritis. Support by a brace or splint is used in children and arthrodesis, if possible, in adults.

Liebolt has found that lack of operative or natural fusion of a tuberculous joint is often a predisposing factor to tuberculous meningitis; surgical intervention in a tuberculous joint does not produce tuberculous meningitis.[56]

Although arthrodesis is generally considered to be the treatment of choice in tuberculous joints, recovery with function has been reported.[22]

Sulfonamides.—Feldman, Hinshaw and Moses showed that 4,4′-diaminodiphenylsulfone was an effective agent in inhibiting or combating experimental tuberculosis of guinea pigs. Continuous prolonged administration yielded cumulative benefits which were not attained in short term experiments.[28] The drug was not excessively toxic for guinea pigs in the dose used. They have encouraged efforts to modify this drug to obtain a compound more suitable for clinical application.

Unfortunately, experiments to date have not shown *penicillin* to be of therapeutic value in tuberculosis.

TUBERCULOUS TENOSYNOVITIS

This condition is rare, occurring only thirty-six times in the past forty-five years at a large general hospital.[1] The average age incidence was thirty-six years (ranging from fifteen to seventy-five years). The right hand was involved in 22 cases (volar surface in 15, dorsal in 6 and both in 1) and the left hand in 14 cases (volar—8, dorsal—5 and both—1). None of the patients were laborers, although 30 made hard use of their hands. Thirteen of the individuals had tuberculous lesions elsewhere in the body.

The *diagnosis* was made by the finding of a *gradually developing painless*

or slightly tender mass (most often on the volar aspect of the hand), with partial stiffness and inability to completely flex or extend the fingers. Grasping strength was decreased. In the later stages, finger motion occasionally produced creaking or grating due to the accumulation of rice bodies (fibrinous deposits within the tendon sheaths).

Treatment consists of rest by splinting, and if necessary, incision and drainage or resection of the involved tendon sheath.

TUBERCULOUS RHEUMATISM

This is the name applied by various observers, especially in Europe, to a type of polyarticular joint involvement, simulating rheumatoid arthritis, which may be of tuberculous etiology, but in which the tubercle bacilli cannot be demonstrated in the joints by smear or culture.[18,19,21,34] The existence of such an entity is doubted by many individuals in this country. Most American rheumatologists believe that this condition is merely the occurrence of rheumatoid arthritis in an individual who happens to have an associated visceral tuberculous lesion.[35]

The clinical picture of tuberculous rheumatism was described by Poncet in 1897; it is now known by some as **Poncet's disease.**[74–76] Some individuals believe that tuberculous rheumatism may be caused by: (*a*) toxins from tubercle bacilli at a distant focus; (*b*) attenuated tubercle bacilli in the joints; (*c*) hypersensitivity (allergy) to primary evanescent tuberculous or other foci;[54] (*d*) an ultravirus of tuberculosis not yet demonstrable microscopically; or (*e*) tuberculous involvement of the nervous system with trophic joint changes.

The protagonists of the disorder point to the similarity between the habitus (thin, undernourished, readily fatigued) in tuberculosis and rheumatoid arthritis, and to various experimental, laboratory, therapeutic and roentgenographic evidence. The excellent résumé of this subject by *Brav* and *Hench* gives in detail the pros and cons of this argument.[11] These investigators *can find no incontrovertible proof to support the existence of an entity such as tuberculous rheumatism.* They have shown that: (1) the family incidence of tuberculosis is no higher in polyarthritis than in other non-tuberculous diseases; (2) polyarthritis is very uncommon (from 0.2 to 5.6 per cent) in tuberculous patients;[12,24,77] (3) active pulmonary or other visceral tuberculosis is very uncommon in arthritic patients;[20,40,70] (4) the signs of so-called tuberculous rheumatism are not distinguishable clinically from other types of polyarthritis; (5) there are no pathognomonic roentgenologic[62,69,87] signs of tuberculous rheumatism; (6) no specific tuberculous lesions have been found in the joint tissue on pathologic examination;[8,57] (7) it is difficult to reproduce tuberculous lesions of joints experimentally;[32,68] (8) laboratory tests do not provide adequate proof of the tuberculous nature of the lesion; (9) most workers have been unable to recover tubercle bacilli from culture of these joints or from the blood stream.

It has been pointed out that *it is impossible to distinguish clinically*

(including roentgen and physical examinations) *between tuberculous rheumatism and other forms of joint involvement* such as rheumatic fever or rheumatoid arthritis.

Criteria for the diagnosis of tuberculous rheumatism, as enumerated by some workers include:[46,52,55] absence of endocarditis; no response to salicylates; presence of non-articular tuberculosis; positive tuberculin test; positive results by animal inoculation from synovial fluid and predominance of mononuclear cells in the synovial fluid.

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CHAPTER XLVII

SYPHILITIC JOINT DISEASE

TRUE syphilitic arthritis is not common. With the use of penicillin in the treatment of syphilis, joint disease should become even more rare. Joint involvement however, may occur at any stage of congenital or acquired syphilis.[3] Syphilitic joint disease may be classified as follows:

1. **In congenital syphilis:**
   - A. *Parrot's syphilitic osteochondritis.*
   - B. *Clutton's joints* (symmetrical serous synovitis—hydrarthrosis).
   - C. Less common forms: gummatous synovitis, syphilitic dactylitis; suppurative joint disease.
2. **In the secondary stage of syphilis:**
   - A. Arthralgia.
   - B. Synovitis.
   - C. Hydrarthrosis.
   - D. Tenosynovitis.
   - E. Bursitis.
   - F. Arthritis resembling rheumatic fever.
   - G. Arthritis resembling rheumatoid arthritis (F and G may also occur in congenital syphilis).
3. **In tertiary syphilis:**
   - A. Gummatous arthritis (with involvement of soft tissues, cartilage or bone).
   - B. Charcot joints (of tabes dorsalis).
   - C. Chronic syphilitic arthritis.
   - D. Juxta-articular gummata.
   - E. Juxta-articular nodes.

In a large series of patients with chronic forms of arthritis, Kuhns and Feldman found a positive serology in only 1 per cent of more than 1000 persons.[27] In another series of 10,000 syphilitic patients, chronic lesions of the joints of late syphilis were seen in only 0.6 per cent (although syphilitic lesions of bones occurred in about 9 per cent).[33]

About 5 per cent of syphilitic children are born dead and only 25 per cent of these congenitally affected survive the first year.[29]

All bone and joint disease in children should be carefully investigated for evidence of congenital syphilis. The commonest syphilitic joint disease in children is a periarticular infiltration with marked enlargement of the joints, with limitation of motion but very little or no pain on manipulation.

Parrot's Syphilitic Osteochondritis (Parrot's Pseudoparalysis).—The commonest type of syphilitic joint lesion with objective findings is seen in the early months of congenital syphilis.[31,34] This occurs as an *osteochondritis* in a *juxta-epiphyseal* location, which is at times accompanied by considerable periarticular swelling. It is seen in approximately 5 per cent of children with congenital syphilis.

It is *most common in the first three weeks of life* and rarely begins after three months of age. The upper limbs are affected more commonly than the lower. The pathologic lesion is a gelatiniform change in the bone and cartilage which breaks down to form a greenish yellow fluid. Complete epiphyseal separation (Parrot's pseudoparalysis) or fracture of the epiphysis may occur.

On roentgen examination, one sees widening of the joint space, an irregular epiphyseal line, periosteal thickening, decalcification of bone just proximal to the cartilage, cupping of the diaphysis and an irregular density and streakiness of bone adjacent to the cartilage.[9,12,25,45]

If proper treatment is given in time, the joint may undergo complete resolution; shortening or deformity will occur if the growing line has been greatly damaged.[20]

Clutton's Joints (Symmetrical Synovitis in Congenital Syphilis).—Clutton first described a symmetrical synovitis of the knees in congenital syphilis in 1886.[8] This is *one of the most common forms of syphilis of the joints.* Clutton's joints occur in congenital syphilis, usually appearing *between the ages of eight and sixteen.* They are characterized by a *marked painless symmetrical swelling* of the periarticular tissues with effusion into the joints.[26] This develops insidiously.

The involvement is usually limited to the soft tissues. The swelling produces some mechanical impairment to motion. The chief sites of involvement are the *knees* and elbows; the knees are involved in about 85 per cent of the cases. These patients are able to walk well despite the effusion. There is usually *no fever* and there is *very little local heat, pain or tenderness.* Interstitial keratitis is often an associated finding.

The *fluid reaccumulates rapidly if aspirated* and may persist for years without permanent damage to articular tissues.[28] From 10,000 to 45,000 leukocytes (chiefly lymphocytes) have been found per c.mm. of this fluid. A similar form of synovitis may occur in early or late acquired syphilis. Polyarticular joint involvement has also been reported.[6,7]

Roentgen examination is usually negative except for the separation of the ends of the bones due to the synovial effusion. **Syphilis should always be suspected in all cases of bilateral painless hydrops of the knees in children.**[45]

Although relapse is common, the condition usually responds well to treatment, resulting in complete cure.

Syphilitic Dactylitis.—This occurs chiefly in children and is uncommon. Syphilitic dactylitis may appear in the fingers or toes in children from one to three years of age with congenital syphilis.[22] A characteristic spindle shaped swelling appears and may result in deformities and marked shortening; in some cases, it leads to absorption of bone.

Miscellaneous.—Gummata of bone, Charcot joints and spondylitis are uncommon manifestations of late congenital syphilis. Gummatous lesions are rare under twelve months of age.[20] In congenital syphilis, one may see joint lesions which are identical with those of the tertiary stage of the acquired form. Aseptic necrosis may occur in congenital syphilis, resulting in the formation of "pus" within the joints.

SECONDARY STAGE OF ACQUIRED SYPHILIS

Bone and joint syphilis is rare in the secondary stage of the disease and presents no characteristic signs or symptoms.[3] Vague aches in muscles or bones—*arthralgia*—occur in about 5 or 10 per cent of patients in the early stage of acquired syphilis; these are most common during the height of the systemic reaction. They are usually more of an *ache* than a severe pain, are often *nocturnal*, and are not increased by gentle motion of the joints.

Syphilitic Arthritis.—This is very uncommon during the secondary stage of acquired syphilis.[19] Stokes has not often seen true syphilitic arthritis. Despite this, syphilitic arthritis is said to have been recognized as an entity by the ancients and to have been well described in the Middle Ages. Efficiently treated cases of syphilis do not develop specific arthritis.

Syphilitic arthritis usually involves *multiple joints* and is seen most frequently in the *large joints* (especially the knees);[37,38] involvement of the sterno-clavicular joints may occur.[14] An acute or chronic synovitis may be seen; this is *frequently bilateral*. The joints may appear swollen and may show a fusiform appearance, with thickening of the synovial membrane and surrounding tissues and some muscular spasm and atrophy. In other cases, there may be no swelling of the joints. There is very little limitation of motion, local heat or muscular spasm. Muscular wasting is usually of minimal degree.[18] The physical and roentgen manifestations are usually far greater than the pain, muscular spasm and disability. Although bony ankylosis does not occur, impairment of function and deformity may be found. The Wassermann reaction is positive in most cases.

The disease usually responds promptly to efficient antisyphilitic therapy, but not to salicylates.

Hydrarthrosis.—Transient hydrops may occur in early secondary syphilis; this may be polyarticular but usually affects *the knees*. An abundance of fluid is present. There is a moderate amount of pain but usually no redness of the joint. Treatment with arsenicals is usually effective. In some cases, a more persistent form of hydrarthrosis may occur with slight disability and very little muscular wasting.

Tenosynovitis.—This is usually a *mild, symmetrical, painless* swelling of tendon sheaths, most often occurring bilaterally.[42] Infiltration of the tendon sheath may occur in the later stages.

Bursitis.—Syphilitic bursitis is usually painless or only slightly painful; there is swelling, bursal effusion and a tendency to break down with sinus formation. It may also occur during the tertiary stage.

Syphilitic Periostitis.—This does not usually cause joint pain; the process is *common in early and in late syphilis*. Periostitis adjacent to a joint may be responsible for articular pain.

Syphilitic Spondylitis.—Syphilitic spondylitis is caused by an invasion of the periosteum and bone by the treponema pallidum. Syphilitic spondylitis has a marked *predilection for the cervical vertebræ* as syphilitic osteitis

affects chiefly those bones exposed to trauma and pressure. Syphilitic spondylitis may occur in congenital syphilis or in acquired syphilis.

Symptoms of syphilitic spondylitis include localized pain which may be more marked at night, stiffness, tenderness to palpation and avoidance of motion of the head. One commonly sees coëxisting lesions of the sternum, clavicle, tibia or radius.

The *diagnosis is substantiated by:* (1) evidence of active syphilis; (2) marked improvement under antisyphilitic therapy; and (3) suggestive *x*-ray findings. The disease may be limited to only one vertebra or may involve several adjacent vertebræ. One may see destruction of bone or new bone production, or a combination of the two. In most instances, more or less spur formation occurs around the vertebræ simulating the appearance of a non-specific inflammatory spondylitis.

Freedman and Meschan reported 3 cases of syphilitic spondylitis with substantiation by necropsy findings in one.[15]

Pseudorheumatic Form of Syphilitic Arthritis.—This has been described by Todd as occurring *in children* with congenital or acquired syphilis, or in adults. Multiple joints may be involved, especially the knees, ankles, wrists or elbows. In children, there is little pain, redness, heat or muscular atrophy even though the affected joints may be distended; the temperature rarely rises above 99° F. The joint phenomena do not respond to salicylates. *Keratitis* is present in about three-fourths of these patients.

In *young adults*, there may be involvement of several joints in succession with tender, swollen, red painful joints and with fever of 103° F. or more. Marked sweating and anemia may occur. Salicylates do not result in improvement. In most instances, the Wassermann reaction is strongly positive. Treatment with arsenicals results in rapid improvement. These cases are uncommon. **Suspect syphilis in any case of "rheumatic fever" which does not respond promptly to salicylates.**[45]

Rheumatoid Form of Syphilitic Arthritis.—Very rarely, syphilis of the joints may produce the clinical picture of rheumatoid arthritis with fever, sweating, pain, disability, fusiform swelling of joints, etc. These patients have a positive serology and usually respond to antisyphilitic therapy.

TERTIARY SYPHILIS

Syphilitic joint lesions are more common in tertiary syphilis than in the secondary stage. Chronic syphilitic arthritis with synovitis, gummatous arthritis or Charcot joints may be found at this stage.

Synovial Form of Gummatous Arthritis.—This is most common in the early stages of tertiary syphilis but may also occur in children with congenital syphilis. Larger joints are affected most frequently. Considerable amounts of painless effusion are characteristic of the process; occasionally, severe pain may occur. Other features include the presence of an irregular lumpy, localized swelling about the joint with very little restriction of motion or muscular wasting. The gummatous process may affect the whole joint or may remain localized. This may resemble tuberculosis of the joint.

In children, these have been known as *van Gies' joints*. In severe gummatous arthritis in tertiary syphilis, suppuration may be the end result.

Osseous Form of Gummatous Arthritis.—This usually involves large joints. Extensive gummatous involvement may occur with very little impairment of motion. Although such joints may be large and tense, there is *very little evidence of inflammation*, muscular spasm, wasting, limitation of movement, constitutional reaction or ankylosis (unless secondary infection occurs).

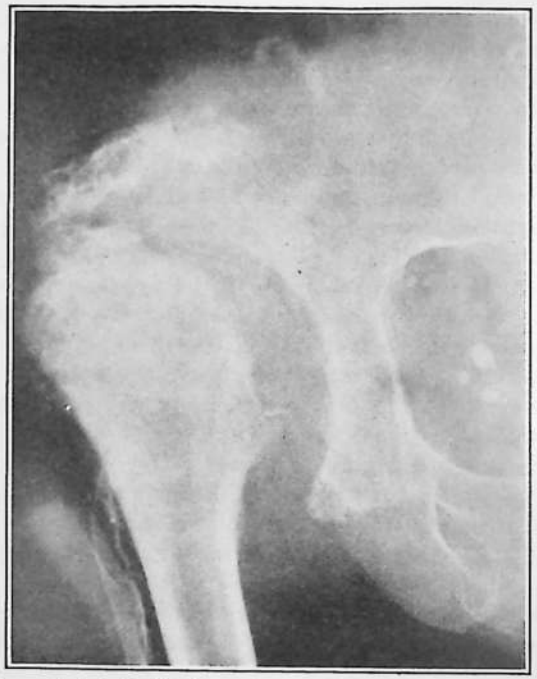

FIG. 190.—*Charcot joint of right femur* in a patient, aged sixty-nine years, who contracted syphilis thirty-eight years ago. The Charcot hip has been present for thirteen years. The patient has Argyll Robertson pupils, bilateral deafness, absent knee jerks and had a right-sided hemiplegia three years ago.

Gummatous deposits may be found in perisynovial tissues and may occur in the cancellous ends of the bone and later spread to cartilage.[30,32] Although the bony changes are most marked, all of the joint structures are usually involved in the process. In young individuals, the process may begin as an epiphysitis. One may see erosion and irregularity of the joint surfaces, cavitation within the bones and bony outgrowths. In adults, the condition may resemble degenerative joint disease. When the spine is affected, the picture simulates that of tuberculosis.

In adults, one large joint is usually affected. Extensive bony changes may be seen; these may be confined to the joint or may extend the whole length of the bone. Osteophytes are common. Gumma of the patella has been reported.[2] If specific therapy is not instituted, the joint may become ankylosed. **Syphilis and tuberculosis should always be suspected in a monarthritis or deforming arthritis at any age.**

Juxta-articular Gummata.—These may occur in tertiary syphilis and lead to symptoms in the joint.[16] Roentgenogram may show considerable destruction while clinically there is little heat or pain in the joint.[46] The lesion may heal completely if there has not been complete destruction of joint surfaces.

Juxta-articular Nodes.—These have been reported in patients with syphilitic infection; they may resemble the subcutaneous nodules of rheumatic fever or rheumatoid arthritis.[21]

Charcot Joints.—Mitchell first referred to tabetic arthropathies in 1831.[35] Charcot described these joints in 1868.[5] About 5 or 10 per cent of tabetic patients and 25 per cent of those with syringomyelia develop arthropathies; they are much more frequent in males than in females. *Charcot joints may occur* in tabes dorsalis, syringomyelia[11] and occasionally in paresis, psoriasis, leprosy, poliomyelitis, compression and destruction of areas of the spinal cord or following peripheral nerve section.[41] Although Charcot joints begin most commonly after forty years of age, they may occur as early as the age of twenty. The disease may be produced in animals by section of the posterior (sensory) roots.[13]

Kernwein and Lyon commented upon the three *theories advanced for neuropathic joints:* traumatic, trophic and the mechanical.[23] In numerous individuals with classical tabes dorsalis (including ataxia, hypotonia and loss of sensibility deep in the joint) neuropathic joints never develop. Only unilateral neuropathy may develop in others with bilateral sensory changes; in these, a history of trauma can usually be elicited, the severity of the injury being overlooked because there is no pain. It is apparent that loss of articular sensation alone does not cause the development of a neuropathic joint.

Various observers have repeatedly shown that neuropathic joints may be produced in dogs, cats or rabbits by destroying the articular sensation by cutting the posterior roots. In a case reported by Kernwein and Lyon, severance of the sciatic nerve was followed by atrophic changes in the muscles of the leg; the ankle became relaxed because of a loss of motor nerve supply. There was an ataxia and anesthesia below the knee. The trauma associated with walking on such a limb was frequently excessive because of the absence of the protective sense of pain. A neuropathic arthropathy of the ankle developed with disintegration of the joint. Roentgen examination showed changes typical of neuropathic arthropathy.

Loss of deep sensation with an added factor of *acute or chronic trauma* in some cases leads to *effusion, relaxation of ligaments, chronic strain, erosion of cartilage,* sclerosis of subchondral bone, flattening and condensation of the heads of the bones, parosteal bone formation, marginal fractures and loose bodies in the joint, exostoses and calcification of muscular and ligamentous structures—leading to disorganization of the joint—the end stage of neuro-arthropathy.[40,48] The changes are similar in either tabetic

or syringomyelic arthropathy, except that in the latter they are more common in the upper extremity, develop more slowly and do not often go on to the extreme destruction as is seen in the lower extremity with tabes dorsalis (probably because weight-bearing joints are not affected). Charcot joints have been reported in the hip,[10] knee, shoulder, elbow, hand, foot, ankle, spine and rarely in the sacro-iliac joints.[47]

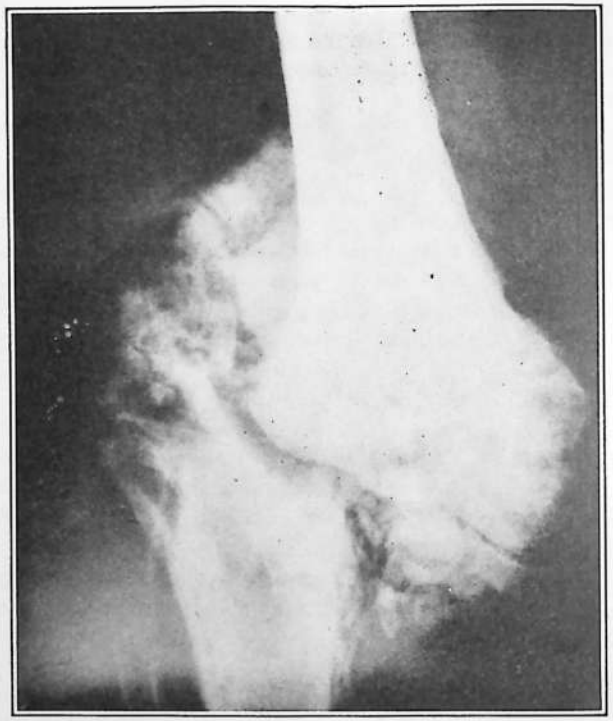

FIG. 191.—*Charcot joint of a knee* (both knees were similar); patient is sixty years of age. There is marked destruction of the knee joint with marked proliferation and bony fragmentation as is typical of a neurotrophic joint.

The disease usually begins in the cartilage and the zone of calcification between the cartilage and the underlying cancellous bone.[36] *Earliest changes include* erosion of the cartilage, chip and compression fractures and marginal proliferation of bone. Later one may see ossification in muscles and spontaneous fractures. Spontaneous transverse fractures are most commonly seen in the femur, tibia and forearm, but may also occur in the tarsal bones.

In the *hips*, there is early erosion of the acetabulum with irregularity of the joint line. Later, one sees enlargement and destruction of the acetabulum and absorption of the neck and head of the femur. In the *knee*, roentgenograms reveal early breaking down, condensation and flattening of subchondral bone in the condyles; as the process continues, genu

varum or valgum appears and dislocation may occur. Free bodies are found in the joint in about half of the cases. In more advanced cases, exostoses are common, in addition to ligamentous and intramuscular ossifications.[43]

The *pathologic process* in general is one of decalcification of bone with gradual destruction of the joint surfaces, followed by an overgrowth of bone at the margins of the joint.[39] It has been mistaken at times for degenerative joint disease.[17] The primary pathologic change is an invasion of the articular cartilage by fibrous connective tissue from the pannus on its surface. Some effusion is usually present in the joint at the onset; marked edema and infiltration of periarticular tissues may occur.

FIG. 192.—Pathologic fracture with ossification in a large hematoma at the left shoulder in a man, aged fifty-two years, with tabes dorsalis. The lesion caused very little pain.

Charcot joints may develop gradually or with great suddenness. Although the larger joints are more commonly involved, the small joints of the hands or feet are occasionally destroyed.[24] The process is frequently bilateral. The disease process may remain stationary for years or may gradually progress until complete disintegration of the joint has occurred. Although these lesions are usually painless, *pain may appear at some time in their course.* These joints may be diagnosed by "the loss of contours, the projecting knobs, the bag-of-bones feel and the extraordinary hypermobility, permitting in some cases the most amazing distortions without the slightest

distress."[44] Massive formation of bone and cartilage occurs with swelling, laxity of ligaments and finally complete disintegration of the joint surfaces resulting in a flail joint. **The occurrence of joint disease without heat, pain or tenderness must always make one consider neuroarthropathy.**[1]

Roentgen-ray examination of a Charcot joint reveals an enormous destruction of the articular surfaces and of the bones with a chaotic bony and cartilaginous hyperplasia in the surrounding tissues—out of all proportion to the clinical findings.[41]

The positive clinical evidence of rigid pupils and absent knee jerks is of much more importance than blood or spinal fluid Wassermann tests in the diagnosis of tabetic arthropathy. The relaxed, disorganized, swollen, comparatively painless joint should always suggest neuroarthropathy; the absence of pain (in most instances) despite such an advanced pathologic process is probably the most striking clinical feature. In tabetic cases, one may also note the Argyll Robertson pupils, the positive Romberg, absence of knee jerks and decreased sensation in the lower extremities.

The blood Wassermann has been negative in as many as 55 per cent of cases in some series of tabetic arthropathies, while the spinal fluid Wassermann was negative in one-third of these cases.

Specific therapy has no effect upon the articular picture. Charcot joints are treated by rest and immobilization with splints or braces, physical therapy and stabilization and alignment of the joint by conservative or surgical measures. Fusion operations are successful at times if the destructive process is not too great. Amputation may be necessary occasionally due to instability of the joint; however, poor healing in the stump is not uncommon. *Penicillin is not of value in Charcot joints.*

DIAGNOSIS OF SYPHILITIC JOINT DISEASE

This diagnosis is made by a *careful history* (including a personal or family history of syphilis, repeated miscarriages, interstitial keratitis, iritis, lymphadenopathy, chancre, etc.), a *complete physical examination, blood and spinal fluid Wassermann* and the *clinical manifestations.* The Wassermann test may be negative in known syphilitic bone and joint lesions; on the other hand, the finding of a positive Wassermann reaction does not necessarily incriminate syphilis as the cause of the joint discomfort. *Too much credence must not be placed upon a negative Wassermann reaction if bone or joint syphilis is suspected;* in all of these cases, one should *x*-ray the tibia and fibula for additional information.

No pathognomonic roentgen signs of syphilitic arthritis have been reported; suggestive features include erosion of bone and cartilage with bony proliferation and periostitis in a relatively symptomless joint.

Clinical details favoring the diagnosis of syphilitic joint disease include absence of pain, even with marked hydrops, free range of motion, good general health despite a long story of joint pathology, precipitation of the arthritis by a blow (occurring often in syphilitics), other evidence of syphilis, lack of response to salicylates and favorable response to antisyphilitic therapy.

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CHAPTER XLVIII

HEMOPHILIC ARTHRITIS

Hemophilic arthritis is uncommon. Reviews upon this subject have been presented by several authors (DuPan, Freund, Key, Konig, Reinecke, Zesas). Cases have been described by others (Bernstein, Firor, Rutherfurd, Sutton, Suzuki, Timperley, Wilson, Youmans and others). In one series of 98 patients with hemophilia, arthritis occurred in 79 per cent; more

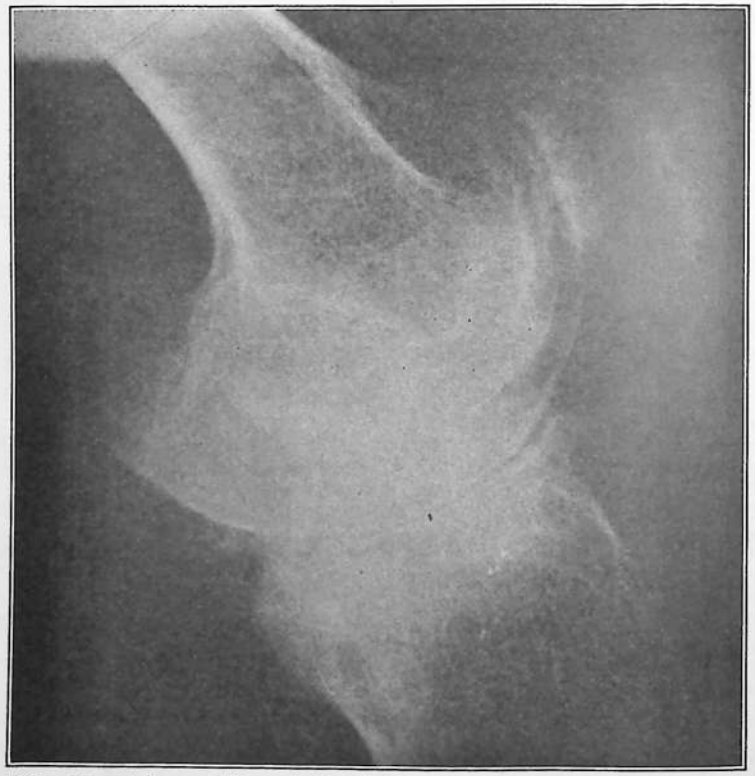

FIG. 193.—Chronic hemophilic arthritis of the knee following intermittent attacks of hemarthrosis for twenty years in this hemophiliac aged thirty-four.

than one-half of these developed permanent joint deformity.[34] The *joints involved in order of frequency were: knee* (68 per cent); *ankle* (56 per cent); *elbow* (53 per cent); hip (16 per cent); fingers (15 per cent); wrist (5 per cent); spine (3 per cent) and toes (2 per cent). Involvement of the shoulders was rare. These patients ranged in age from newborn to sixty-five years. Hemophilia is rare in negroes.

PATHOLOGIC CHANGES IN HEMOPHILIC ARTHRITIS:[18]

1. Distention of a joint with blood under pressure.
2. Hyperplasia of the synovial membrane.
3. Deposition of blood pigment in subsynovial tissues, followed by formation of dense fibrous connective tissue in this region.
4. Erosion of cartilage at the margins by the hyperplastic synovial membrane, and irregular maplike areas of cartilaginous destruction over the articular surface.

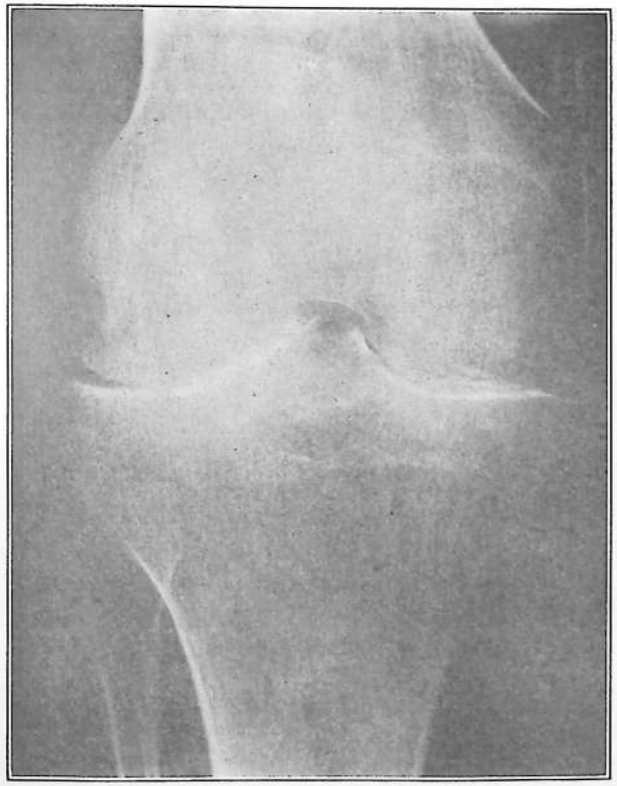

FIG. 194.—Same patient as preceding figure. Note marked narrowing of joint space (indicating destruction and wearing down of articular cartilage).

5. Cavitation of intra-articular bone (as mere depressions or deep cavities), probably from intra-osseous hemorrhage.
6. Marginal osteophytes (rare).
7. Marked deformity in some instances; bony ankylosis is practically unknown.

The arthritic manifestations of hemophilia have been divided into two groups: (1) Acute hemarthrosis; and (2) chronic hemophilic arthritis.

SYMPTOMS OF ACUTE HEMARTHROSIS:

1. *Rapidly appearing joint effusion* (blood), usually following slight trauma.
2. History of *previous similar attacks.*
3. Pain and disability varying with the degree of distention of the joint.
4. *Absence of local heat and redness.*
5. Slight febrile reaction and moderate leukocytosis; no general *prostration.*
6. *Roentgenogram:* Distention of the joint cavity—casting a dense shadow (blood).

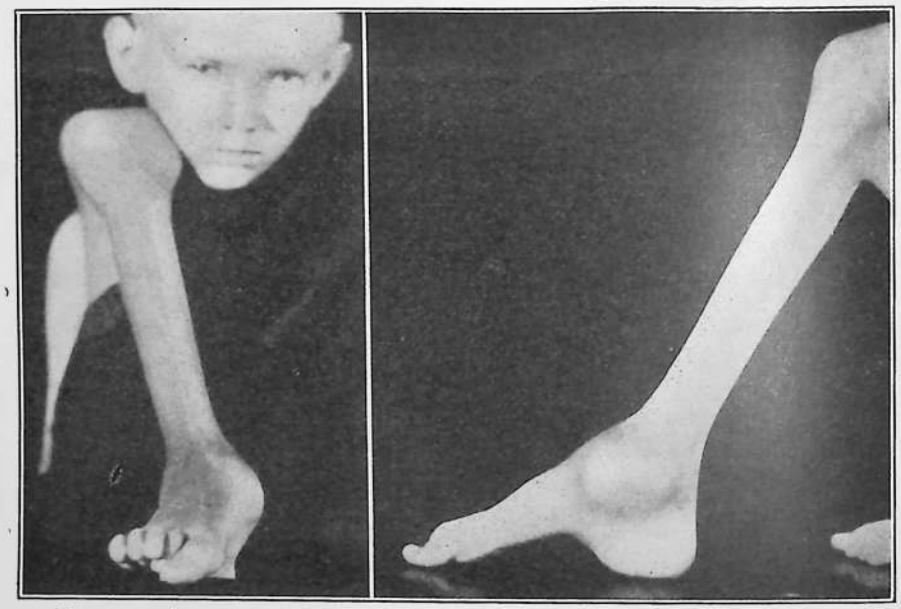

FIG. 195.—Acute hemarthrosis of an ankle in a hemophiliac. (Key, courtesy of Ann. Surg.)

The patient usually describes previous attacks of joint swelling, beginning early in childhood, subsiding following several days of rest, or resulting in chronic arthritis. There is joint swelling, following slight trauma (although acute hemophilic hemarthrosis has occurred during sleep). *In most instances, one sees a rapid development of the effusion,* which may become larger as the patient watches the joint; in others, the onset may be gradual. The amount of joint fluid varies considerably. Pain and limitation of motion may be severe or minimal, depending upon the degree of joint distention.

Patients who develop hemophilic arthritis are usually slender and pale. *The joint distention stands out in marked contrast to the emaciation of the extremities.* Absence of local heat and redness is an important sign of this disorder. In some cases, extra-synovial hemorrhage results in subcutaneous discoloration.

SYMPTOMS OF CHRONIC HEMOPHILIC ARTHRITIS:[19]

1. Tenderness and swelling of the joint with limitation of motion.
2. Muscular atrophy.
3. Tendency for remissions and recurrences.

FIG. 196.—Marked contractures of both knees in a severe hemophiliac seven years of age. (Key, courtesy of Ann. Surg.)

Hemophilic arthritis may follow any attack of hemophilic hemarthrosis; however, it does not occur commonly after the first bout of bleeding into the joint. In one instance, normal function was restored forty-five times with reabsorption of blood following hemarthrosis into a knee.[15]

Hemophilic arthritis is suspected if absorption of a hemarthrosis does not occur in a reasonable period of time (several weeks). Additional findings include periarticular thickening, deformities, contractures and muscular atrophy. Fibrous ankylosis occurs in some cases. *Hemorrhages into the joints tend to diminish in frequency with age. Hemophilic joints do not suppurate.*

Roentgen Findings in Hemophilic Arthritis.[7,16,28]—**Early Findings:**[5,30]—Distended capsule with a widened joint space.

Shadow of blood (which is denser than ordinary synovial fluid).

Later Findings:[12,22,26]—1. Maplike destruction of cartilage.

2. Cavities or cysts in intra-articular bone.
3. Increased density of synovial tissues often simulating calcified areas.
4. Narrowing of the joint space with irregular indented articular surfaces of bones, and deformity of the ends of the bones.
5. Atrophy of bones (in some cases).

Caffey and Schlesinger studied roentgenographically the joints of 5 children with recurrent hemophilic hemarthrosis. *Articular changes develop before adolescence in 89 per cent of cases of hemophilia and hemarthrosis;* however, little attention has been devoted to the *acceleration of epiphyseal maturation associated with juvenile hemophilic arthritis.* The accelerated epiphyseal maturation was explained on the basis of chronic irritation and hyperemia resulting from repeated hemorrhage. Injections of heparinized blood into joints of growing rabbits did not accelerate epiphyseal development experimentally.

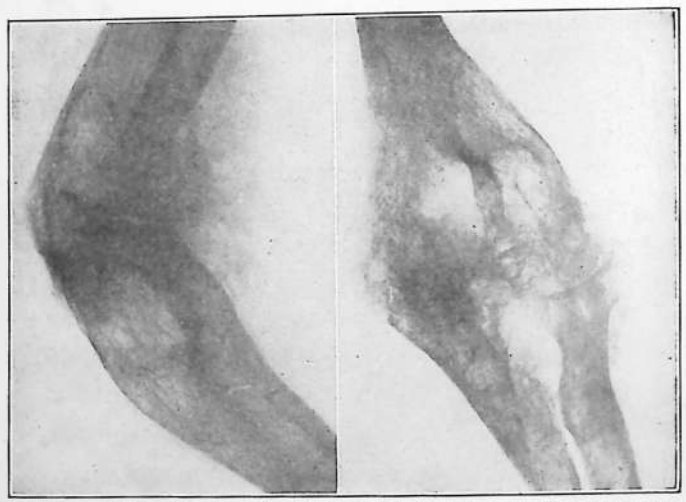

FIG. 197.—Hemophilia with organized blood-clot in the capsule of the elbow joint. (Holmes and Ruggles.)

McDonald and Lozner reported on the roentgen findings of the joints of 15 adults with hemophilia.[21] *Pathologic changes were seen by x-ray in one or both knees in every patient and changes were found in one or both elbows in all but one patient.* The degree of change, in general, was proportional to the age of the patient.

Not all hemarthroses are followed by roentgen changes. The roentgen picture of acute hemophilic hemarthrosis is similar to that of any traumatic, serous or purulent synovitis with distention of the joint space by fluid. One sees a generalized periarticular clouding of the soft tissues which is most marked along the lines of the distended capsule.

Chronic hemophilic arthritis may be *distinguished from degenerative joint disease* inasmuch as roentgen changes about the joint are usually seen before the age of twenty-one in hemophilic arthritis. Although cystic changes may be seen in the bones in both conditions, they are more common

in hemophilic arthritis; generalized bony decalcification is usually more severe and more frequently seen in hemophilic arthritis than in degenerative joint disease.

Rheumatoid arthritis is not usually confused with hemophilic arthritis. Hemophilic arthritis does not commonly affect the phalanges and wrists, while these joints are involved very frequently in rheumatoid arthritis. Although bony ankylosis is not uncommon in rheumatoid arthritis, it is very rare in hemophilia. The cystic changes in subchondral bone are seen more often in hemophilic arthritis than in rheumatoid arthritis.

Tuberculous arthritis is sometimes confused with chronic hemophilic arthritis. However, in tuberculous arthritis, the articular surface shows a ragged and irregular scalloping.

Treatment: In *cases of external bleeding*, in hemophilia, the use of sterile solutions (1:10,000) of *Russell viper venom* has been recommended.[1,2,15,20,24] Some have preferred Fer-de-lance venom because of its availability, effectiveness and low cost. Temporary reduction of the clotting time has been obtained in a majority of patients with hemophilia by oral or intramuscular administration of a human placental extract.[9–11] Intravenous injection of placental extract may prove fatal. *Ovarian therapy*[4] has been *disappointing.* Theelin injections (0.5 cc. intramuscularly) prevented further hemarthrosis in one patient.[36] Others have obtained best results by intramuscular injections of the patient's blood. Intramuscular injections (10–15 cc.) of whole blood taken from the mother during the menses have been used in several cases.[3] *Beef thrombin*, applied locally, may be of value in stopping bleeding from small wounds, such as tooth sockets.

Treatment of Hemophilic Arthritis[21]

Acute hemarthrosis:
- *Rest in bed with splinting of the affected joint* until pain and distention lessen markedly.
- Ice bags applied to the affected joints to lessen pain and decrease the period of invalidism.
- Trial of *intravenous oxalic acid* (5 mg. in 5 cc. of sterile water 2 or 3 times a day for several days).
- *If intense pain and distention remain, aspiration* (a somewhat dangerous procedure) with a small bore needle. (Fatality may follow this procedure.[25])

Chronic hemophilic arthritis:
- *Correction of deformities* (if necessary) *by conservative rather than by operative procedures;* use of traction, mechanical appliances, etc.; *no manipulative procedures.*[17]
- *Elastic bandages* (such as Ace bandages) often of benefit in providing added support to the joint.
- In severe hemorrhages, *transfusions* and trial of oxalic acid intravenously.

Steinberg and Brown reported that *oxalic acid* reduced coagulation time after intravenous injection into rabbits.[31] Schumann used an oxalic-acid

containing plant extract (Koagamin) with gratifying results in various forms of internal hemorrhage.[29] Oxalic acid is a strong organic acid. Normal human beings have from 5.5 to 7.5 mg. per 100 cc. of blood.

The *present concept of hemophilia* is that of an inherited, sex-linked defect in the clotting mechanism resulting in a prolonged coagulation time, due to abnormal resistance of the platelets to disintegration; this latter leads to a deficient supply of thromboplastin.[23]

Page and his associates caused a temporary fall of the blood coagulation time to normal in 3 patients with hemophilia by the intravenous administration of oxalic acid.

This drug is not a cure for hemophilia but may be of assistance in some cases of hemorrhage in hemophiliacs. One may give 5 mg. of oxalic acid in 5 cc. of sterile water intravenously two or three times a day for several days, if necessary.

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CHAPTER XLIX

INTERMITTENT HYDRARTHROSIS

This is a chronic disorder characterized by periodic recurrences of joint effusion persisting for several days, most often affecting the knees. Attacks usually occur at regular intervals,[35] are refractory to salicylates[23] and are not accompanied by evidence of local inflammation. Many of these patients eventually acquire definite rheumatoid arthritis.

Since the first report[31] in 1845, various reviews of the literature (Benda, Berger, Bierring, Linberger, Schlesinger) have appeared. The disease is relatively uncommon; not more than 120 cases have been reported, but doubtless many cases have not been recorded in the literature.

Synonyms include: hydrops articulorum intermittens; hydrops genu intermittens; hydrarthrose intermittens des genoux; intermittienende Gelenkswassersucht; Typisch recidivierender Hydrops genu; Periodische Gelenkschwellung; Hydrops hypostrophos.

The two varieties of intermittent hydrarthrosis which have been encountered are: (1) symptomatic intermittent hydrarthrosis, and (2) idiopathic intermittent hydrarthrosis. In the former, the recurring joint swellings are early symptoms of a rheumatoid type of arthritis which makes its appearance at a later date in these joints affected by the recurring edema or in other joints. While the cycles are usually regular for many months, the periods of freedom between attacks later become shorter and there is less complete recovery from the attacks.

Idiopathic intermittent hydrarthrosis indicates those individuals whose attacks seem to recur without the later development of rheumatoid arthritis. It appears, however, that most patients with either form of intermittent hydrarthrosis eventually develop a progressive rheumatoid arthritis.[33]

Etiology.—This is unknown. Cases have occurred in individuals between the ages of seven and sixty, most often between the ages of twenty and forty-five; sex appears to play no rôle. No cases have been reported in the colored race. The disease is rarely present in more than one member of a family, although the condition may occasionally be familial. Other etiologic factors which have been commented upon include: trauma,[16,28] infection with Brucella abortus,[1,29,40] malaria, rheumatic fever, lymphogranuloma, psychic factors,[12] sensitization to food,[24,34,39] bacteria or other agents,[7,27] association with menses,[38] childbirth[8,13,30] and endocrine disturbance.[19] The analogy of intermittent hydrarthrosis to giant urticaria was pointed out by Schlesinger. In several questionable instances of intermittent hydrarthrosis there was an associated rheumatoid arthritis.[6,14,17]

Symptoms: 1. Periodically recurrent joint effusion (usually involving the knee).

2. Usually unilateral although often bilateral.

3. Average interval between attacks—seven to eleven days (limits three to twenty-one days, only rarely beyond these).
4. Duration of effusion—three to five days.
5. Absence of pronounced local heat, muscular spasm, notable tenderness or severe constitutional reaction.
6. Local disability, decreased motion and occasionally, marked pain.
7. Total duration of cycles of disease: up to twenty-two years.

FIG. 198.—Intermittent hydrarthrosis of the knees. (Sharpe, courtesy of Ann. Int. Med.)

Most cases of intermittent hydrarthrosis begin in the third or fourth decades. Although the disturbance may be bilateral, in a majority of cases only one knee is involved. Other joints (as the ankle, hip and rarely a joint of the upper extremity) are occasionally attacked.

There is usually an abrupt onset of an increasing effusion in a knee with moderate pain and limitation of motion. There are no marked signs of local inflammation. A mild febrile reaction is often present; in some cases, there is no fever. Within several days, the fluid slowly disappears and the knee assumes a normal appearance. After a variable period of time, which is often fixed for each individual, joint effusion reappears; this is usually from seven to twenty-one days. The leukocyte count remains normal in most instances.

Laboratory findings are extremely variable. Secondary anemia and an elevated sedimentation rate are present in some cases.

Recurrences of the disease may continue for years, and even after long periods of remission. A well-known story is that of the athletic schedule of a prominent university which was especially arranged (supposedly) to permit matches to fall upon days when a stellar athlete, who suffered from intermittent hydrarthrosis, knew he was to have a remission.

Typical Case Résumé.—A typical history as we elicited it in one of our patients, a physician, was as follows:

"I am forty-eight years old and in good health. I was never sick except for an attack of tonsillitis at the age of fourteen; tonsillitis recurred almost every two or three years until the tonsils were removed in 1923.

"My intermittent hydrarthrosis began when I was fourteen years old (shortly following my tonsillitis); this began with recurrent swellings (every twelve days) of my right knee. The swellings lasted three days each time.

"At the age of twenty-two, I went into the Army and after a few months of physical exercise, the swelling disappeared for five years.

"In 1926, I had a synovectomy performed on my right knee. Three months later the swelling returned, first in my right elbow and three days later in my left elbow; these recurred every twelve days. Two years later, the swellings moved to my right hip and later to my left hip, and again recurred every twelve days.

"In 1930, I took *Fowler's solution* for a week and the *disease disappeared for five years.* Since 1930, I have tried: histaminase and histamine desensitization; roentgen therapy; injections of calcium and parathyroid extract; citrocarbonate for weeks; Fowler's solution (again); ergotamine tartrate by injections and orally; *intravenous typhoid vaccine* which *brought relief for six months* but which caused such severe reactions that I will never try this again; and injections of adrenalin.

"All of these were, of course, useless. I could never have the will power to remain on my elimination diet for more than two days; this produced no benefit. All skin tests for foods were essentially negative, as were repeated blood cultures, Wassermann reactions, sedimentation rates, blood counts and urinalyses.

"My mother's family had a history of bronchial asthma. There has been no rheumatism, gout or joint affection in the family."

It was of interest that when this physician again submitted to intravenous typhoid vaccine (fever) therapy, another (at least temporary) remission was produced.

Differential Diagnosis.—This is usually not difficult. *Other causes of hydrarthrosis of the knee* include the infections (tuberculosis, gonorrhea, syphilis, heredo-syphilis, undulant fever), trauma, and reaction due to local or general causes.[22] These do not usually assume a periodic, intermittent nature.

One should, however, perform a complete work-up of the patient including careful historical data in regard to possible exposure to undulant fever, syphilis or gonorrhea; roentgen examination of involved joints; blood and joint culture; Wassermann and gonococcus complement fixation tests; blood uric acid, agglutination test for Brucellosis and basal metabolism.

Pathology.—Pathologic changes found in the synovial membranes consist of synovial edema with villus formation, infiltration of the subsynovial tissues with lymphocytes and endothelial cells, areas of hyaline degeneration of fibrous tissue and enlargement of the endothelial cells leading to thickening of the endothelial layer with reduplication of the lining endothelium. There is in reality a permanent and progressive underlying synovitis.[33]

Findings have varied in the few cases studied to date. In one, the synovial surface was covered with long slender branching villi with edematous tissue and profuse infiltration with lymphocytes in both villi and subsynovial surface. Dense connective tissue, fat and sclerotic vessels were also found. In another instance, the villi were flattened and the round cell infiltration was confined almost solely to the villi. Others noted slight thickening of the lining membrane without fibrosis.[15,20] Some workers observed pathologic changes in the synovia resembling those seen in rheumatoid arthritis.[32] The synovial fluid may contain several thousand cells per c.mm. with more than 50 per cent of polys.[10]

Prognosis.—The prognosis has been rather poor until recently. A number of apparent cures have been reported.

The attacks have recurred regularly for years in many instances without change in the nature of the symptoms. Many of these individuals, however, eventually acquire definite rheumatoid arthritis.

Treatment.—Little success has been obtained with older forms of therapy (quinine, arsenic, desensitization with peptone, physical therapy, rest and splinting of the part, salicylates, aspiration and lavage of the joints with introduction of antiseptic solutions). More recently, cures have apparently been attained by various procedures such as removal from the diet of foods to which the patient was sensitive, synovectomy, intravenous typhoid vaccine, ergotamine tartrate[11,41,42] and local roentgen therapy.[26] Radical synovectomy is the operation of choice in intermittent hydrarthrosis although it does not always affect a cure.[21] Pregnancy has also had an ameliorating effect upon several cases.[18]

The periodicity of intermittent hydrarthrosis has suggested a possible allergic basis. Service has reported 4 cases with allergic histories in which the joint symptoms were relieved by correcting the existing allergy. He feels that *food allergy* is an important factor in hydrarthrosis. Others reported cases due to food sensitization. One of these was due to ingestion of English walnuts.

In the two instances of cure following *synovectomy* reported by Krida, one patient had attacks for nine months coming on five weeks after trauma and persisting despite conservative therapy; no positive skin tests were found although careful examination for sensitivity was performed. In the second case, the etiology seemed to be related to the menses.

In a case cured by *roentgen therapy*, no recurrence was noted for ten months following 8 treatments of 225 r each, at 25 cm. distance, 2 m.a. intensity and using a filter of 6 mm. Al. Treatments were given alternately to the internal and external surfaces of the affected knee.

Weismann-Netter used *ergotamine tartrate* in intermittent hydrarthrosis because of its favorable action in some cases of urticaria, and because of the analogy of periodic hydrarthrosis to local urticaria. Ergotamine tartrate inhibits the sympathetic nervous system. Seven injections each of ½ mg. of the drug were used at intervals of two days; there was an immediate marked improvement. Ergotamine tartrate has also been of value when given by mouth; 1 mg. per day has been used over periods of weeks or months. *Extreme caution* must be used if ergotamine is given over a period of weeks or more.

Histaminase has not been of value in reported cases; in two of the author's patients with intermittent hydrarthrosis, this drug produced no beneficial effects.

Penicillin has not been tried in these patients.

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CHAPTER L

GOUT

GOUT is considered to be a hereditary constitutional disease, occurring in males in over 95 per cent of the cases and usually characterized by recurring attacks of acute painful joint swellings.[8] *The clinical picture is extremely variable* from an asymptomatic increase in blood uric acid to a severe, crippling joint affection.

Although gout is classed as a disease of inborn metabolism, its exact etiology is unknown; the high blood uric acid level is an evidence of the inability of the individual to dispose of uric acid. This hyperuricemia may be noted for years before joint symptoms appear.

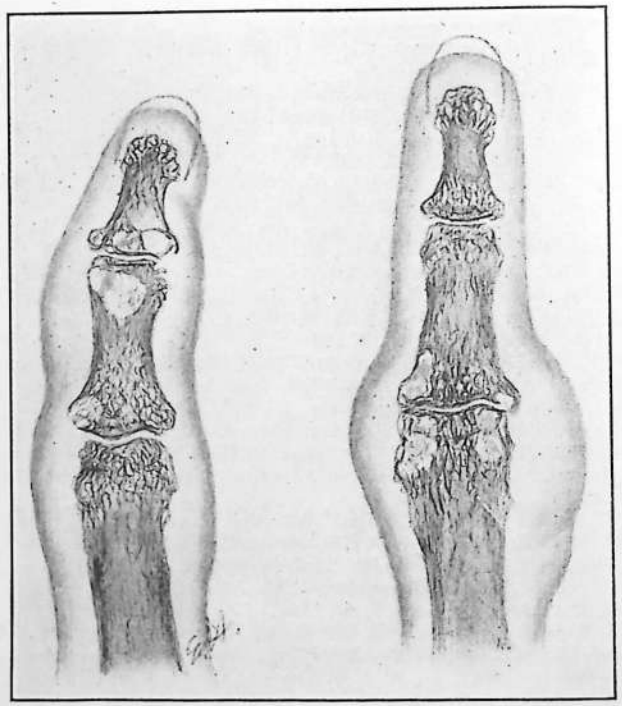

FIG. 199.—Gout. Drawing shows asymmetrical swellings and punched-out areas in the phalanges.

It is interesting that in the past ten years, 2400 deaths were attributed to gout in England and Wales; about 85 per cent of these were in men. During this same period, there were only 25 deaths allocated to this cause in the United States. Allowing for the difference in population, the deaths

in England from gout were two hundred times as frequent as in this country.[14]

Gout is usually seen as a monarticular arthritis in a male, coming on much more rapidly than any other form of arthritis, with the attack subsiding completely within a few days to a week in the usual case.

Main Symptoms and Signs of Gout

Tophi (present in only about half the cases at any given time).[52]
Elevation of *blood uric acid* at some time in about 75 per cent of patients.
Acute *recurrent attacks with complete remissions* (before the development of secondary changes in joints).[54]
Predominance in males (98 per cent of cases).
Involvement of big toe in initial attack in 70 per cent of cases, and sooner or later in 90 per cent.
Roentgen-rays of *little diagnostic value early* in the disease; bony erosions seen in only about one-third of cases even in the later stages.

Suspect Gout If:[48]

Acute joint symptoms follow an *operative procedure.*
There is a *family history* of gout.
There is a history of *olecranon bursitis* or Achilles tendinitis.
Acute joint symptoms follow slight trauma, or excessive holiday food or drink.
Sudden joint symptoms appear during early morning hours.
Acute arthritis occurs in men over the age of forty, in the absence of acute trauma or a history of gonorrhea.
Acute joint symptoms develop within minutes or a few hours.
Joint symptoms are *associated with a chronic nephritis or renal colic.*
Joint symptoms occur *in* patients with *polycythemia, leukemia* or after certain therapeutic procedures such as injection of liver extract, salyrgan, insulin, ergotamine tartrate (gynergen), thiamin chloride, decholin (dehydrocholic acid) or the use of a ketogenic diet.
A long automobile ride or strenuous exercise precedes the first attack.
Joint symptoms occur after hunting and fishing expeditions.
Joint symptoms recur in the spring or fall.
Joint pain is exceptionally severe.
Acute *involvement of the great toe occurs.*
The patient appears wearing a shoe cut out over the metatarsophalangeal joint of the great toe.
Complete recovery of an acute arthritis occurs within several weeks.
Symptoms are *relieved* within twenty-four to seventy-two hours by adequate doses of *colchicine.*
The patient is absolutely *free from joint symptoms between attacks.*
Periarticular fistulas or ulcers are present.
Joint symptoms develop or are made worse following the ingestion of a high fat and low carbohydrate and protein diet for several days (P.F.C. 50–300–30).[67]
The symptoms in a monarticular arthritis are entirely disproportionate to the findings.
The general reaction, especially the fever in any polyarthritis, is less than the findings might indicate.[125,126]

An understanding of gout would enable the general practitioner to diagnose this disease in from 1 to 5 per cent of patients who consult him because of joint symptoms.[9,35,47,55,119] Unfortunately, this disease is often overlooked until years after its onset, chiefly because one of the classical signs (tophi) does not usually become evident until then.[115]

Gout is one of the oldest of known diseases. It was described by Hippocrates as podagra, cheiragra or gonagra depending on whether the big toe, wrist or knee joint was involved. Colchicine was used probably as early as the fifth century.[45,89,106] Later a regimen was suggested which included a low meat intake, temperance, bleeding and colchicum for purging. The name gout was given in the thirteenth century (from the French "goutte," a drop); this was to signify that the poison entered the affected joint drop by drop.[60,61]

The Greek word-formation rheumatismós meant a flowing down—probably originally designating a flowing to and fro of the streams of humours in the abdominal region—and finally designating a humour flowing down into a joint cavity.[4,88]

The description presented by Sydenham in the seventeenth century is still one of the best clinical pictures of the disease. In 1797 Wollaston discovered that the "chalk stones" in gouty joints were composed chiefly of uric acid.[129] Garrod showed (in 1847) that patients with gout possessed increased amounts of uric acid in their blood.[38]

The treatment for gout differs radically from that of other forms of joint involvement; relief of joint pains and swelling in gout can be procured much more readily and promptly than in rheumatoid arthritis. If neglected, complications of gout such as nephritis, uric acid renal stones, possibly arteriosclerosis and vascular disturbances have been feared; however, there is no proof that these do not develop if the patient is treated.[116]

Acute gouty arthritis (also called "regular" or "acute gout") usually begins in men over thirty years of age (although cases have been reported in children; these are very rare). Most of these patients are engaged in sedentary occupations. Physicians and lawyers are affected frequently but laborers much less often. Lockie found 10 per cent of males over the age of twenty-five who had joint symptoms—had gout. Kinell and Haden noted that obese, bald men of ruddy complexion in the fourth or fifth decade were particularly prone to have gout. The attack is usually of sudden onset, lasts from three to eleven or more days and the symptoms then disappear completely. Although the great toe may be affected in over half of the cases, any joint may be involved.

The acute attack usually subsides spontaneously in several weeks or less with restoration of normal joint function. Another attack may occur in from twelve to eighteen months; this varies in severity and may or may not affect joints previously involved. Usually, further attacks come closer together and the disease becomes more severe. Although the first few attacks are usually afebrile and most often involve only one joint, subsequent attacks may be accompanied by a temperature of 101°, 102° or more and usually affect several joints.[56] *Premonitory symptoms* are often

lacking prior to acute attacks of gout. Some individuals may experience indigestion, nausea, anorexia, aching, stiffness, polyuria and nocturia.

The first attack of gout usually appears with dramatic suddenness. The sudden onset of severe or at times excruciating pain in an apparently healthy middle aged man, frequently beginning at night, often in a metatarsophalangeal joint, is almost diagnostic of gout. The affected joint may swell markedly within a few hours and become hot, red and extremely tender. In some cases, the swelling has appeared so rapidly that the patient could not remove the shoe without cutting it. *There is a much more marked inflammatory edema than in other forms of joint disease;* this swelling may extend some distance beyond the margins of the joint. Not uncommonly, there may be effusion into the large joints. The acutely involved toe or foot resembles an extensive cellulitis with its tense, red, shiny skin and distended superficial veins.

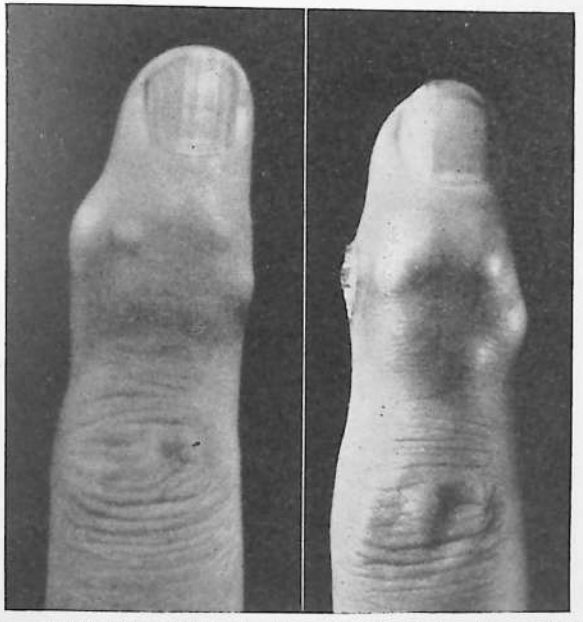

FIG. 200.—Tophi of the right index finger finally discharging sodium urates. (Ludwig *et al.*, courtesy of Ann. Int. Med.)

With subsidence of the redness and tenderness, *pitting edema* can be observed. The *skin* overlying the lesion *becomes thin and loose* and *may* itch and *desquamate.* This is very uncommon with other forms of joint disease.

Fever is usually mild, rarely persisting more than a few days. Fever is found more often in younger patients. The entire attack may pass with no fever.

One of the most annoying symptoms of gout is the *sudden occurrence* of joint symptoms persisting for a few days or weeks and then completely

disappearing, leaving the patient free of joint involvement until his next attack, which may occur months or years later. In some patients, attacks occur each spring and fall for unexplained reasons. In exceptional instances, attacks may be more than thirty years apart. *This sudden onset of attacks of pain with absolute disappearance of all joint phenomena between attacks is almost pathognomonic of gout.*

FIG. 201.—Gouty tophi in an ear.

Such a picture *may be seen occasionally in other diseases,* as acute rheumatic fever, gonorrheal arthritis, atypical rheumatoid arthritis, acute traumatic arthritis, cellulitis, suppurative arthritis, intermittent hydrarthrosis, periarticular fibrositis, psoriatic arthritis, arthritis with ulcerative colitis, hemophilic arthritis and subdeltoid bursitis. However, the patient with *acute rheumatic fever* is usually young, whereas first attacks of gout occur more often after the age of forty. Polyarticular involvement is much more common in rheumatic fever than in gout.[57] In younger persons, if multiple joints are involved, the fever, leukocytosis and increased sedimentation rate may cause confusion with rheumatic fever; however, the absence of cardiac involvement, epistaxis and tachycardia favor the diagnosis of gout. Following the subsidence of the gouty inflammation, the skin often peels over the affected joints; this does not occur in rheumatic fever.

Gonorrheal arthritis occasionally exhibits recurrent attacks. However, these patients are readily distinguished from those with gout. In gonorrheal arthritis, a genito-urinary involvement (primary or recurrent) can often be demonstrated. Gonorrheal arthritis is usually polyarticular at the onset but rarely remains so. In gonorrheal arthritis, the complement fixation test performed upon the blood is positive in more than 85 per cent of the cases, the blood uric acid is usually normal and tophi do not occur. Patients with attacks of gout move the involved joint frequently in an effort to find a more comfortable position on the bed or pillow. This is in contrast to the usual finding in acute specific arthritis (as gonorrheal arthritis) in which the extremity is held in one position.

FIG. 202.—Chalk gout, calcinosis, in a man, aged sixty-two years. There is no demineralization of the bones. There are many deposits of calcium of varying size occurring along the shafts of many of the long bones; in most cases, these deposits are near joints.

The main characteristics of gouty arthritis include the abrupt onset of severe joint pain and swelling (without known cause) usually in an apparently healthy male past the age of thirty, with development of heat and

redness in the joint with extension beyond the joint margins, a family history of gout, the finding of tophi and desquamation of skin as the edema subsides, the dramatic response to colchicine, the elevated blood uric acid and the interval of freedom between attacks.

Recovery from attacks of *atypical rheumatoid arthritis* may be so complete and so rapid as to make one consider the possibility of gout. However, the typical patient with rheumatoid arthritis exhibits a slender build, undernourishment, symmetrical involvement of joints, fusiform joint swellings, roentgen findings, insidious onset, etc. The foot in gout is usually warm and red in contrast to the cold, clammy bluish white foot in rheumatoid arthritis. If the diagnosis is in doubt, one must consider the blood uric acid value, the results of colchicine therapy, a family history of gout and the later course of the disease. The occurrence of gout and rheumatoid arthritis in the same patient has been reported but evidence of this is not convincing.

Joints Involved.—Podagra, although now used as a synonym for gout, designates an acute involvement of the metatarsophalangeal joint of the great toe. Involvement of the big toe by an acute arthritis is usually diagnostic of gout. However, not all patients with gout manifest this symptom. Although no joint is exempt from gout, the joints of the lower extremities are involved more frequently than the upper.[17,85,108] The big toe is affected in about 70 per cent of gouty individuals in the initial attack of the disease and is involved sooner or later in 90 per cent of these patients. It becomes very tender, swollen, hot and bluish or reddish. Maximum tenderness occurs at the medial side. *Any joint in the body may be involved;* unfortunately, this fact is not generally appreciated.

Joints rarely involved by gout include the sacro-iliac, temporomandibular, sternoclavicular articulations, spine, shoulders or hips.[89] The first symptoms of gout may appear in a knee, ankle or other joint. If the knee is involved, marked swelling and outpouring of fluid into the joint may occur; a similar picture may occur at the wrist. The hip joint becomes involved in about 2 per cent of cases of gout. Rotation of the head of the femur is impossible and the whole thigh may become tender. Hench has never seen involvement of the joints of the torso by gout and states that when acute or chronic arthritic involvement of the spinal column, hips and shoulders are present, the diagnosis of gout is very unlikely; the hips and shoulders have been thought to be relatively immune to uratic deposits. Recent autopsy material tends to question this statement. Gouty erosion of the sacro-iliac joints and uratic deposits in the vertebræ may occur, but are rare.

The **first attack of gout** is usually *monarticular* involving most commonly the big toe, instep, heel, ankle or knee.[52] The peripheral joints appear to be more vulnerable to attack than the knees, elbows, hips and shoulders. Although attacks may begin in the early hours of the morning, the symptoms are usually first detected when the individual puts his foot on the floor after awakening.

The medial or lateral aspects of the ankles may be involved with diffuse swelling and tenderness at the malleolus or extending backward into the tendo Achilles. The tissues between the latter and the ankle joint become tender, although the tendon itself may become tender occasionally. The area of maximum tenderness may shift slightly each day, moving slightly in any direction.

Any portion of the foot, especially the dorsum, may be involved, resulting in swelling of the foot with redness and tenderness (the latter usually confined to an area the size of a silver dollar). This may resemble a cellulitis and may be incised by mistake. This differs from the cold, moist, pale perspiring foot of the rheumatoid arthritic.

Later, multiple joint involvement may be noticed, one region being involved as another improves.

As the disease advances, feet, ankles, wrists, knees and elbows may become affected. The disease may be polyarticular (about 5 per cent of cases) at its onset; this is more likely to occur in younger patients, rarely involves the great toe, is accompanied by a marked fever which may persist for weeks, or longer, and is often misdiagnosed acute rheumatic fever [80]

Desquamation of the cuticle and itching often follow disappearance of the joint swelling.

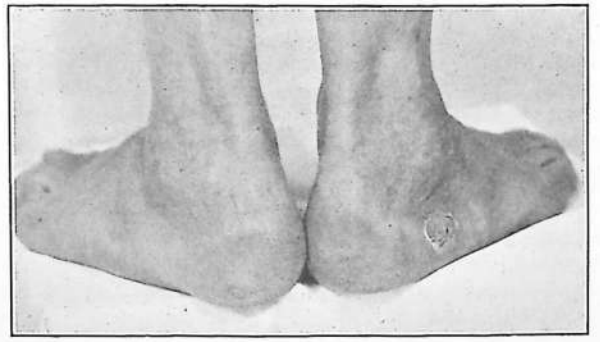

FIG. 203.—Ulcerated tophi of heels. (Christopher, courtesy of Jour. Am. Med. Assn.)

Tophi.—Tophi are found in only 2 per cent of cases of gout in the initial attack. The *tophi* of gout are often misinterpreted. They are deposits of monosodium urate in various tissues of the body. *Any lesion suspected of being a tophus should be opened, some of its contents examined mioroscopically and the characteristic appearance of the urate crystals observed.* Tophi are seen most frequently upon the helix or antehelix of the ear. They occur less often in the olecranon and prepatellar bursæ, and tendons of the fingers and wrists, toes, ankles and heels,[8] and in many other unusual locations.

Bunim and McEwen reported the finding of a urate tophus of the mitral valve.[20] Hench and Darnall had previously reported an intracardiac tophus.

They may vary in size from a pinpoint to a pea. Tophi the size of a baseball have been observed.[25] With ulceration of the skin over a tophus,

a white chalky material may discharge. These ulcerating lesions rarely become infected.

Tophi are sometimes mistaken for Heberden's nodes, subcutaneous nodules of rheumatic fever or rheumatoid arthritis, deposits of calcium, sebaceous cysts and for ganglions.

Periarticular calcium deposits of creamy, tooth-paste-like-material may occur in the neighborhood of joints.[27] These may be associated with a generalized calcinosis or may appear chiefly adjacent to joints. These should not be confused with tophi.

Chalk gout (more commonly called calcinosis circumscripta) may present swellings over joints which occasionally simulate gouty tophi. In circumscribed calcinosis, these concretions of calcium phosphate and calcium carbonate are confined almost exclusively to the toes, fingers, elbows and knees.[27,103] The disease develops insidiously and runs a mild course. The subcutaneous calcium deposits may or may not be painful; aching and stiffness may be noted in nearby joints. The pathogenesis of such calcium deposits is unknown.

Tophi

Occur in about *50 per cent* of all cases *of gout.*
Are the only pathognomonic sign of gout.
Are found in the helices or antehelices of the ear, knuckles, hands, toes, feet, other joints, cartilages of the eyelids and nose, heels,[22] subcutaneous tissues or muscles, tendons of the fingers, Achilles tendon, patella, patellar and olecranon bursæ, tongue, heart muscle, skin of the palms and soles, cornea and sclerotic coat of the eye;[128] on rare occasions, they have been found in the aorta, aortic valves, epiglottis and vocal cords, the prepuce of the penis and the corpus cavernosum.[8]
Are painless (except when they are the site of an acute attack, or interfere with motion).
Reveal, when opened, crystals of monosodium urate (sodium biurate), giving a positive murexid test.
Are easily overlooked unless gout is suspected.
May appear white, cream colored or yellow.
Rarely appear before the tenth year of the disease except in very severe cases; *do not wait until tophi are present to diagnose gout as the disease will usually have existed five or ten years before they are seen.*
May discharge a chalky mixture if the overlying skin breaks down.

Blood Uric Acid.—Uric acid does not exist as such in the blood; it is present there in the form of sodium biurate. We do not know what causes the elevated blood uric acid in gout or the cause of the precipitation of sodium urate into the tissues.

Lockie reported that the blood uric acid was frequently *not* increased during the first attack of gout. Many of these patients may take aspirin, which may lower the blood uric acid 50 per cent within twenty-four hours.

Elevation of the blood uric acid may occur in a variety of pathologic states including gout, acute and chronic glomerulonephritis, following liver therapy in pernicious anemia, cardiac decompensation, leukemia, polycythemia, lead poisoning, lobar pneumonia, acute febrile diseases (including pneumonia, typhoid fever, erysipelas, scarlet fever and tuberculosis); after poisoning by ammonia, veronal, carbon monoxide and methyl alcohol; during periods of regeneration of blood after hemorrhage or hemolytic icterus and occasionally in patients with diseases of the liver and biliary system; severe diabetes mellitus (especially with acidosis) and sometimes in chronic eczema, psoriasis and urticaria.[16] These diseases present no differential diagnostic problem in relation to gout. *The blood uric acid is elevated in about 75 per cent* of patients with gout.

Garrod found abnormal quantities of uric acid as sodium urate in the blood of gouty patients.[37] Wollaston analyzed the material from tophi and found that it contained lithic or uric acid;[129] before this, it was believed that tophi were composed of chalk (and they were then often called chalk stones).

Garrod showed that *when a thread was placed in the blood serum of a gouty patient, the sodium urate crystallized and adhered to the thread;* in the presence of normal serum urates, crystals did not form. Increased blood uric acid has been found at an early age in gouty subjects.

All mammals except man are able to destroy uric acid rapidly by means of uricase, a ferment found in the liver, muscles and kidneys except in man and Dalmatian dogs. In birds, ammonia and lactic acid are synthesized in the liver to form uric acid, but this does not take place in mammals.[21]

It has been suggested recently that the **serum uric acid** level be determined when gout is suspected. The fasting uric acid when determined by the Folin 1933 method (performed on serum derived from blood allowed to clot under oil) varied from 1.9 to 6.7 mg. per cent in 100 non-gouty individuals.[34,63] Ninety-seven of these showed a serum uric acid less than 6 mg. per cent. *A large number of serum uric acid determinations made in patients with gout revealed that in 98 per cent the value exceeded 6 mg. per cent.* The use of a purine free diet for less than three months did not significantly influence the serum uric acid level in these patients; however, *this figure showed an apparent temporary fall in gouty patients following the administration of aspirin, colchicine or salyrgan.* Serum uric acid exhibited marked fluctuation during attacks of acute gouty arthritis and during asymptomatic intervals. In several of the cases there was an apparent direct correlation between the height of the serum uric acid and the severity of the disease; in 1 patient studied over the course of a year, the mean serum uric acid showed a fall during a three day period preceding attacks of acute gouty arthritis but remained unchanged during the attacks. The elevation of serum uric acid may be transient at first and may become fixed only at a later date. *In early acute attacks of gout, the uric acid may be normal or elevated. This elevation becomes more pronounced and more constant later in the disease. In chronic gouty arthritis which is not under treatment, serum uric acid is usually elevated.*

For exact results by the Folin method, certain precautions should be observed:[80] blood should be drawn from the fasting patient, collected,

and allowed to clot in a tube under oil, and the required amount of serum transferred to an Erlenmeyer flask. If these precautions are observed, Talbot believes a serum uric acid level above 6 mg. per cent is practically always present in untreated cases of gout.[118] Not all observers agree with this as serum uric acid levels under 6 mg. per cent were found in 25 per cent of cases of probable gout in one series.[50]

Oppenheimer reported that intravenous or intramuscular injection of a solution containing *uricase* into hens with a high plasma uric acid concentration will lower the uric acid level; the higher the plasma uric acid level, the greater the uricolytic activity of the enzyme. The uricolytic activity of a single uricase injection seems to persist for at least twenty-four hours.[93]

FIG. 204.—Punched-out areas in a sixty-two year old brewmaster with gout. This great toe had suffered repeated attacks of acute swelling.

The uric acid partition in gout and hepatic disease has been studied.[2] Normal blood serum has a comparatively constant uric acid partition. Gout and hepatic damage result in a diminution of the free and elevation of the bound uric acid fraction.

Blauch found that about a third of the color produced by older methods of uric acid determinations was due to substances other than uric acid.[11] She devised a new method for estimating the true uric acid concentration of blood by means of uricase. Normal blood contains only 2 mg. per cent of uric acid by the uricase method.

Roentgen Examination.—Huber in 1896 first described the specific punched out areas of bone seen roentgenologically in gout.[62] These punched out areas may be seen in numerous conditions, including rheumatoid arthritis, degenerative joint disease, sarcoidosis, hyperparathyroidism, multiple myeloma, Hand-Schüller-Christian syndrome, syphilis, tuberculosis, yaws, metastatic malignancy, etc.

FIG. 205.—Bony changes in **gout** in a man aged sixty-four years. There are multiple small punched-out areas and in some regions, there are small areas of fragmentation.

Too much reliance has been placed upon roentgen examinations in the diagnosis of gout, often with very misleading results. **Early cases of gout may show no roentgen findings.** *Patients with rheumatoid arthritis and degenerative joint disease may show small punched out areas in bone which roentgenologists sometimes interpret as indications of gout.* Roentgen examinations may be of assistance but a negative roentgen-ray examination does not rule out gout in the acute case. *In one series of gouty individuals, 19 patients had their disease more than twenty-five years without* roentgen evidence of gout.[57]

The *most suggestive roentgen findings in gout* are punched-out areas which are usually 5 mm. or more in diameter, located in the subchondral bone of the base or head of the phalanges in the hands or feet. These can be seen in only 32 per cent of all cases, even in the late stages of the disease. These punched-out areas represent deposits of urate crystals. *In suspected cases, the feet (and particularly the great toes) should always be examined for such lesions.* Marginal bony hypertrophy is also common in gout. Occasionally, decalcification occurs in patients with prolonged, severe attacks. Findings similar to those of gout may be seen occasionally in syphilitic gummata, tuberculosis, leprosy, sarcoid[65] and yaws.

Postoperative Gout.—*Acute arthritis developing shortly following surgical operations is often gout.* The onset of postoperative gout may be from hours to several days after a surgical procedure.[48] *Whereas rheumatoid arthritis is often temporarily benefited by any operative procedure, the reverse is true in gout.* Reactivation of rheumatic fever may also occur a week or two following surgery.

Family History.—A history of gout in other members of the family or antecedents can be obtained in 50 to 60 per cent of cases if the examiner persists in his questioning.[113] In some patients with gout, a family history of the disease cannot be obtained. In one series of 55 gouty individuals, a hereditary factor was found in only 6 per cent.[84]

Talbott conducted a five-year study of 136 blood relatives of 27 persons with gout. None of the 136 relatives appeared to be suffering from gout. In 102 of these relatives, the serum urate determination was normal; the remaining 34 had some elevation of blood uric acid.[113]

Smyth and Freyberg studied the families of 2 gouty individuals and found that 7 of the 8 adult male members had hyperuricemia and 5 had clinical gout. None of the females in these 2 families had any evidence of gout. Blood uric acid values were not elevated in the feminine members of these families. This and other studies confirm the opinion long held that there is an important hereditary factor in the etiology of gout.[109]

When Dalmatian hounds (high uric acid excretors) were mated with collies (low uric acid excretors) it was found that high uric acid excretion was inherited as an almost completely recessive, non-sex-linked character.[122]

Olecranon Bursitis.—Another useful point in the diagnosis of gout is its *association with olecranon bursitis* and Achilles tendinitis. If there is no definite history of trauma, the development of olecranon bursitis should arouse a suspicion of gout; this complication is reported to be several times as common in gout as in rheumatoid arthritis. The attack may also affect the bursa over the patella.

Other Diagnostic Pointers.—Trivial trauma or dietary excesses often serve as provocative agents for acute attacks of gout; gout is not uncommon following large feasts or alcoholic overindulgences. It has also followed shortly after hunting or fishing trips.

Joint symptoms frequently begin in the early morning hours and may waken the individual from sleep. There is often sudden severe pain, swelling, warmth and redness of a great toe. The symptoms are often not noted until the patient bears weight on an affected foot. *The rapidity with which severe joint symptoms may develop in gout is a feature of the disease. This pain may be so excruciating as to require morphine.* Slight fever (usually 101° F.) frequently accompanies the attack.

Persons of all nationalities are subject to the disease. Cohen has discussed gout in the negro.[26] It may affect individuals of any age, cases having been reported in patients at the age of nine; however, this is extremely uncommon and the usual age of onset is past thirty. *In general, the younger the patient, the worse and more progressive is the gout.* Most of these persons are large, overweight and extremely healthy appearing individuals. *An acute arthritis occurring in a male over forty years of age should lead one to suspect gout unless a history of trauma or a possibility of gonorrheal arthritis is present.*

The most common factor precipitating an attack is minor or major trauma.

Latent gout has been activated by marked blood loss and transfusions, chronic glomerular nephritis, leukemia and polycythemia, as well as by emotional upsets, foreign protein therapy, exposure to cold and dampness and severe purgation.

The occurrence of gout in association with other diseases (polycythemia and leukemia) and following the use of liver extract,[31,94] gold,[99] thiamin chloride,[127] sulfathiazole, dehydrocholic acid,[15] insulin,[51] salyrgan,[98] gynergen or a ketogenic diet has been commented upon. Acute attacks of gout may follow the use of liver or liver extract in the treatment of pernicious anemia; these are rather infrequent.[30,33,112] The possible usefulness of injections of liver extract as a provocative test for gout was considered; however, these often failed to provoke an attack. Some individuals have employed injections of crude liver extract in the treatment of gout.[29]

Gout has been reported in several cases of congenital hemolytic icterus; in one of these patients, there was no recurrence of a gouty attack for seven years postsplenectomy.[31]

Acute attacks of gout have been reported immediately following an injection of liver in a patient with congenital hemolytic icterus.[31] Acute attacks of gout have also been reported following transfusion in a patient with severe anemia from melena.[94]

Gout has been reported *following hematemesis and melena.* Following such hemorrhage there is usually an increased regeneration of the blood with increased liberation and disintegration of the normoblast nuclei; the nucleo-proteins of the latter constitute increased material for the production of uric acid.[32] In 7 patients who were studied, increased uric acid excretion of the urine occurred following hematemesis and melena.

Morlock and Rosenberg reported a case with coëxisting tophaceous gout and non-tropical sprue.[86]

Since the time of Hippocrates it has been known that gout is very uncommon in women. It has also been stated that men deprived of their gonads do not become gouty.

Rosenberg reported a case with bisexual genitalia, male configuration of

the body but with a predominantly female personality who had characteristic attacks of acute gouty arthritis (and marked hypertension).[104]

Claiborne reported a seventeen year old girl with pretophaceous gout of three years' duration.[23] Typical tophaceous gout has been reported in women.[117,127]

Lambie reported tophaceous gout in a boy of fourteen years with "chronic erythronoclastic anemia;" this boy was frequently jaundiced but no hemolytic anemia could be found in any of his relatives.[72]

Thomson cautioned that gout may assume atypical forms in women, especially about the menopause, and because of its infrequency in females, is thus overlooked.[121] In his cases, gout occurred in a more chronic form in women, often superficially resembling rheumatoid arthritis.

When a normal subject ingests a high fat diet, there is an increase in the blood uric acid and a decrease in the amount of uric acid in the urine. On this basis, a high fat and low protein and carbohydrate diet was given to patients with gout;[53] the uric acid concentration in the blood increased, its excretion in the urine decreased and an attack of gout occurred within several days. Attacks of gout were noted in some instances before any rise in blood uric acid; in others the symptoms disappeared while the blood uric acid was still high. In chronic arthritis, the administration of a similar diet did not cause an exacerbation of symptoms. Lockie has suggested that in questionable cases, a diet high in fat (300 gm.) and low in protein (50 gm.) and carbohydrate (25 gm.) be administered; if this is followed within a few days by an exacerbation of joint symptoms, gout should be suspected. This diet should be continued for a period of from five to seven days as a therapeutic test.[77,83]

Laboratory Findings

Increased blood *uric acid* (seen in about 75 per cent of cases).
Sedimentation rate usually normal between attacks; frequently elevated during attacks.[59,67]
Roentgenograms generally negative early; of little use in diagnosis until late in the disease; roentgen-rays of the feet usually show signs of gout earlier than the hands. *Roentgen-rays show areas of erosion in only 32 per cent of cases.*[56]
Mild or moderate *leukocytosis* is the rule during attacks.
Anemia is seldom present in patients with gout except perhaps in the terminal stages.[44]
Decrease in *renal function* may occur.[97,107]

Renal Impairment.—Patients with gout may eventually die of uremia. Uric acid stones or gravel may be present in 10 per cent or more of patients with gout. Nephrosclerosis with deposits of urates in the parenchyma of the kidney occurs with about the same frequency. The *association of renal impairment or renal stone with joint symptoms should arouse a suspicion of gout.* Hypertension and arteriosclerosis are found more often in gouty subjects than in controls of the same age.[81]

No difference has been noted in urate clearance in gouty and in non-gouty persons.[28]

Frequency of Attacks of Gout.—Attacks have been found to be somewhat more common in the spring and fall. In a large series of patients, *the frequency of attacks averages about one each year and a half* although attacks may occur often during a single year in a given patient, and only at intervals of several years in others. *The average duration of an attack* has been estimated at slightly less than *two weeks;* in only 2 per cent of patients with gout is the condition chronic from the onset.

FIG. 206.—Gout. Note punched-out areas in several bones.

Occurrence of Widespread Ankylosis in Gout.—Bauer and his co-workers have described a severe case of gout in which there was a rapidly spreading ankylosis; many of the clinical features of this patient resembled those of advanced rheumatoid arthritis. Ankylosis in gout is a result of the deposition of monosodium urate crystals in the joint tissues. Such ankylosis is extremely rare and is usually limited to one or two joints. In this patient, widespread ankylosing deformities occurred, although the patient was only twenty-eight years of age.

Visceral Gout.—The terms irregular gout, visceral gout and abarticular gout are rapidly disappearing from common usage. Visceral or abarticular gout are names which have been applied in former years to gouty involve-

ment of organs other than joints. The renal impairment and formation of uric acid stones or gravel has already been commented upon. Degenerative vascular disease has been considered by some to be a complication of late gout, with death resulting from cerebral or coronary vascular accidents. Disturbances of hepatic function and intractable gastrointestinal symptoms have been described in patients with gout;[7,75] however, these are indefinite and there is no proof that they are associated with the gouty condition.

Alkaptonuria and Hyperuricemia.—Leslie reported a case of *alkaptonuria* with hyperuricemia.[74] This is the first reported case in which these two conditions of distinct metabolic disturbances have coëxisted. *Alkaptonuria is considered to be a deficiency disease* in which there is (probably) absent from the body some catalyst for a stage in the breakdown of tyrosine and phenylalanine.

Alkaptonuria is usually detected easily; most often the *history is that of a darkening or blackening of a urine moistened diaper when exposed to the air.* In some cases the urine is very dark when voided. In some instances, the disease may not be noted until later in life when a routine urinalysis discloses a reducing substance which proves not to be sugar. Other symptoms are not usually observed until later when **ochronosis** may occur.

Virchow first described ochronosis.[123] The latter is characterized by a *bluish discoloration of the cartilages,* most easily seen in the alæ nasi and ears; this is due to the deposition of oxidation products of homogentisic acid. It was thought that this pigment resulted from the combination of homogentisic acid or one of its derivatives with chondromucin or chondroitin-sulfuric acid in the cartilages.[3] In some instances, this abnormal pigmentation may be seen in the skin, nails and pingueculæ. The articular cartilages may undergo degeneration leading to a condition resembling degenerative joint disease. The intervertebral disks may become eroded in some instances.[110] Ochronosis does not occur in all cases of alkaptonuria.

Garrod reviewed the subject of alkaptonuria, tracing this back to 1584.[39] The reducing substance in the urine in these individuals was found in 1859,[12,13] and Brune reported the first case in this country in 1886.[18]

Alkaptonuria is considered to be a rare mendelian recessive character; the condition has been shown to be hereditary in most of the cases described. Homogentisic acid is apparently an intermediate product of protein metabolism. It can be produced experimentally by feeding excessive amounts of phenylalanine.

Urinary Uric Acid.—In some clinics a diagnosis of gout is not made unless the patient is unable to concentrate his urinary uric acid above 50 mg. per 100 cc. in an eight hour specimen, following several days of a purine free diet.[120] This test has been thought to be of value in differentiating true gout from the increased blood uric acid encountered in leukemia, cardiac decompensation, polycythemia, etc.; individuals with these latter conditions were said to be able to concentrate uric acid in the urine normally even though an elevated blood uric acid was present. Recent work, however, does not confirm this.[117]

Stages of Gout.—Hench has divided gout into several stages. The first stage is that of *acute* recurrent attacks of *gouty arthritis;* the second stage is chronic gouty arthritis. Talbott has called acute attacks of gout "critical" gout and has spoken of the period between acute attacks as "intercritical" gout.

In *stage one,* one sees the less severe, early attacks usually involving only one joint. The blood uric acid may or may not be elevated during the attacks. Tophi and roentgen evidence of bony erosion have not yet made their appearance. Later in stage one, the attacks become more severe, persist longer, often involve multiple joints, are at times febrile and usually show an elevated blood uric acid. In the latter part of stage one, bony or subcutaneous tophi are present in about 30 per cent of the cases. Complete symptomatic remissions still occur with complete disappearance of joint symptoms between attacks.

In the *second stage,* remissions become incomplete and some pain, deformity or stiffness persist between acute attacks. At this time, the blood uric acid is usually elevated (in untreated persons), subcutaneous tophi are present in almost all of the patients and bony tophi are common in the hands and feet. The end stage is one of relatively painless chronic gouty arthritis.[56]

Treatment of the Acute Attack of Gout

Rest in bed with elevation of the affected part, if possible; no weight bearing until acute pain and tenderness have disappeared if the lower extremities are affected.

Temporary immobilization of the involved part by light *splinting* and protection from bedclothes by the use of a cradle.

Colchicine, 0.5 mg. ($\frac{1}{120}$ gr.) by mouth every hour until pain is relieved unless toxic symptoms appear (nausea, vomiting and diarrhea); it is usually not necessary to exceed 8 to 10 doses of this drug during the first twenty-four hours. If diarrhea occurs, and pain persists, colchicine may be continued (0.5 mg. [$\frac{1}{120}$ gr.] four times a day) and the diarrhea controlled by paregoric and bismuth (4 cc. of the former every hour for 5 doses in addition to 2 gm. [30 gr.] of bismuth subcarbonate after every liquid stool). After the first twenty-four hours, if pain is relieved, colchicine is continued in a dose of 0.5 mg. ($\frac{1}{120}$ gr.) three or four times a day for three days of each week (unless diarrhea appears).

A diet of milk, fruit juices, eggs and cereals with a high fluid intake (for at least several days).

If necessary (very rarely), morphine may be given.

Therapy.—The affected part should be immobilized, elevated if possible, and enveloped in cotton wool; hot wet magnesium sulfate dressings may occasionally be helpful. In some instances, cold gives more relief than heat, but the former is usually not well tolerated.

Talbott recommends that a few tablets of colchicine ($\frac{1}{2}$ mg.—1/120 gr.) be taken at hourly intervals at the first appearance of premonitory symptoms of a gouty attack.[114] Further ingestion of colchicine is unnecessary if the attack does not materialize in a few hours. If acute gouty pain occurs,

the colchicine should be continued until an adequate amount has been taken. *Delay of a few hours in beginning colchicine medication may increase the severity and prolong the duration of acute gouty episodes.*

The average attack of gout requires approximately 10 tablets of colchicine (½ mg. each hour for 10 doses), although this varies from 8 to 16. Many of these patients learn by experience the optimum number of tablets to take with each attack of gout so that alleviation of joint pain may be attained without undesirable gastrointestinal symptoms. Joint pain begins to subside approximately twelve hours after the onset of colchicine therapy in most instances and pain is usually completely relieved in from one to two days. *Over 95 per cent of acute attacks are materially benefited by a single course of colchicine.* Should no benefit occur, a second course of colchicine should be given after an interval of three days; repetition of a course sooner might precipitate gastrointestinal symptoms.

In addition to colchicine, the patient with an acute attack of gout should be *at rest in bed*, and should have a *large fluid intake, soft diet (high in carbohydrate, low in fat and low in purines)*, should *avoid alcoholic beverages* and should receive some sedation. If the urinary output falls too low, the high concentration of urates in the renal tubules leads to a precipitation of some of the urates as crystals, resulting in serious renal damage. During the early hours of an acute gouty attack, aspirin, codeine or morphine may be required to relieve severe pain; they are seldom required longer than twelve to twenty-four hours.

Talbott has not observed any alcoholic beverage to be an offender in a large number of gouty patients.

If the patient notices a close association between the ingestion of any article of diet and an acute attack, this food should be avoided.

The drugs used most often in the treatment of gout are:

1. Colchicine.
2. Salicylates.
3. Alkalis and
4. Cinchophen.

Salicylates increase the urinary excretion of uric acid. Although *cinchophen* has a similar effect (due probably to inhibition of reabsorption of urates in the renal tubules), this drug is not used routinely in some large institutions because it *is not indispensable in the treatment of gout;* cinchophen intoxication and death may occur and the analgesic action of cinchophen or neo-cinchophen is less reliable in the acute attack of gout than colchicine.

Patients with gout should never be without a vial of colchicine, as it is almost specific for the relief of pain in this condition (although it is ineffective in non-gouty acute arthritis).

Talbott believes that *most gouty patients should take some colchicine from time to time between attacks of gout.* In those individuals who have less than one attack of gout per year, a few colchicine tablets (½ mg.) may be taken at times when mild pains and aches appear in the joints. Other patients may take from one or two tablets per week up to one or more per day.

In a few patients with severe gouty arthritis, *two or three tablets of colchicine have been given daily for five years* (with no untoward effects from the prolonged ingestion of the drug). The individual does not develop tolerance to the drug and the full course of colchicine is still just as effective in these individuals should an acute attack occur.

Talbott believes that alkaline waters and powders have some merit in the treatment of gout between acute attacks (*i. e.*, intercritical gout).

Practically every case of acute gout will respond promptly to colchicine therapy.[114] *Freedom from pain and decrease in swelling occur in from one to three days.* During the acute attack, a diet of milk, eggs, cereals, fruits and vegetables with a low purine content is advised; such vegetables include white or sweet potatoes, corn, lettuce, tomatoes, string beans, celery, cabbage, carrots, beets, Brussels sprouts, broccoli, turnips and eggplant. Hominy, noodles, spaghetti, macaroni and tapioca are also low purine foods and are permitted. When the symptoms have abated, the patient is placed upon a diet high in carbohydrate and low in protein, purine and fat (P F C—60—50—300). Fats inhibit the excretion of urates while carbohydrates tend to increase their excretion.

A high purine intake increases the blood uric acid in gouty patients; however, a high blood or tissue level of uric acid does not produce attacks of gout with regularity, even in gouty subjects.[8]

Colchicine may be continued in dosage of ½ mg. (1/120 gr.) three times a day for two or three days each week for several weeks, unless diarrhea occurs.

Colchicum was known to Byzantine physicians as early as the fifth century A.D. under the name of hermodactyl (finger of Hermes). It has also been known as amina articulorum (soul of the joints). A drug probably identical with colchicum was described in the Ebers Papyrus (1500 B.C.). Baron Anton von Storck[111] introduced colchicum autumnale (or meadow saffron) to most of Europe in 1763. He was physician to the Empress Maria Theresa. He stressed the analgesic action of the drug for articular pains and its diuretic properties. It is reported that Benjamin Franklin introduced colchicum into this country.[89] The term colchicum probably originates from an ancient district of Asia Minor named Colchis.

There is as yet no explanation of the action of colchicine in gout. It does not act as a diuretic; it does not increase excretion of urates or decrease the urate content of the blood. It is not an analgesic as it does not influence pains of non-gouty nature.

Cinchophen and salicylates produce an increase in the excretion of uric acid and a decrease in the blood uric acid in gouty or in normal subjects.

Alternate forms of treatment which have been used include the following:

Cinchophen: 0.5 gm. three or four times a day for three days each week; a large fluid intake; low purine and high carbohydrate diet; and sufficient sodium bicarbonate or potassium citrate to alkalinize the urine to prevent the precipitation of urates in the kidneys.

Glycine and salicylates: the excretion of urates is not materially altered by small doses of salicylates; this is usually accomplished only by the use of salicylates in amounts likely to cause gastrointestinal irritation and sweating. The synergistic action between salicylates and glycine (which increases the excretion of urates in normal persons) may be utilized in the treatment of gout.[101,102] Glycine may be given in doses of 3 gm. three times a day.

Fever therapy has not been used widely in the treatment of gout, but in several patients, good results have been obtained.[58]

Bartels has used *20 per cent glucose intravenously* in the treatment of acute attacks of gout.

Cinchophen (which was introduced under the proprietary name of atophan) was shown to increase the urinary output of urates and lower the blood uric acid.[90] In addition to this, cinchophen also has analgesic and antipyretic properties.

Because of an occasional unpredictable fatality following the use of cinchophen or neo-cinchophen, we do not advise the use of these drugs in the treatment of gout or arthritis. Complications such as acute liver necrosis are rare, but one who has seen such an occurrence prefers to employ other preparations, such as colchicine.

Prevention of Postoperative Gout

In known cases of gout, postoperative flare-ups may be avoided by a regimen suggested by Hench, carried out for five days prior to, and at least five days following operation. This includes:

- A purine-free diet, if possible (although there is no proof that this is indicated).
- Cinchophen, 0.5 gm. (7½ gr.) three times a day (unless the blood uric acid is elevated to high normal or above, in which case the dose is given four times a day). (We do not employ cinchophen because of its occasional toxic effect.)
- At least 2000 cc. of fluid daily.
- A high carbohydrate intake (at least 300 gm. daily).
- Sufficient sodium bicarbonate to keep the urine alkaline.

The manner in which colchicine produces relief in gout is unknown; it does not affect the level of uric acid in blood or urine. Recently, it has been found that dilutions of colchicine arrest mitosis of cells in metaphase and double the number of chromosomes; botanists have also used these effects to produce new plant types. The recent studies of the effect of colchicine on the growth of normal and malignant tissue have not explained its action in gout.

Colchicine must be used with caution in treating patients with liver disease.[105] Serious and fatal reactions have occurred when colchicine was given to rats with acute hepatic damage or within five days of partial hepatectomy.

The virtue of cinchophen in gout lies in its power to increase the excretion of uric acid. Some physicians employ cinchophen routinely and report no ill effects if the patients are under daily observation.

THERAPY BETWEEN ATTACKS OF GOUT

Prevention of Recurrences of Gouty Arthritis.—Bartels employed a *low fat, low purine, high carbohydrate diet* together with *cinchophen* (in decreasing doses) in an attempt to reduce the number and severity of further attacks of gout.[6] On such therapy, 31 patients had a total of 7 minor attacks of gout as compared with 84 major attacks during a comparable period before treatment. The blood uric acid was reduced in most instances and both the number and severity of attacks were greatly diminished. Patients even in the stage of chronic gouty arthritis responded to such therapy.

In a study of 14 gouty patients, Bartels found that for an average of eleven years they lost an average of twenty months away from work, with an average salary loss of $3640 and with medical expenses of $489.

Lennox showed that a high fat diet caused a retention of uric acid;[73] Adlersberg and Ellenberg confirmed this.[1]

The rationale of the treatment of gout with a low purine, low fat, high carbohydrate diet and cinchophen was as follows:

1. Cinchophen results in uric acid diuresis.
2. High carbohydrate intake also produces uric acid diuresis.
3. Low purine intake relieves the overtaxed purine metabolizing mechanism.
4. A high fat intake increases tubular resorption of urates, and is therefore to be avoided.

Cinchophen was given as suggested by Graham, beginning with ½ gram (7½ gr.) three times a day for three days a week.[41] These patients were advised as to the symptoms of cinchophen toxicity and the drug was discontinued if signs of toxicity appeared. As the blood level of uric acid fell, the dose of cinchophen was decreased. The use of a high carbohydrate diet was an added precaution against liver damage. If liver function tests showed a decreased function, cinchophen was stopped. The use of alcohol was discouraged because of the possible toxicity from a synergistic or intensified action of alcohol and cinchophen on the liver.

The blood uric acid level was followed every month or two; when this was lowered, the dose of cinchophen was reduced to two or one day each week, or omitted entirely when the reading was approximately normal.

The high carbohydrate, low purine, low fat diet was continued indefinitely but was liberalized if the blood uric acid level returned to normal and continued there. *Vitamins A and B were added* because the diet was low in both of these.

In addition to the *diet* and *cinchophen, any acute attacks of gout were treated by intravenous injections of glucose* and large tophi were removed if these were troublesome; physical therapy was used when indicated.

It was noted that when the diet and cinchophen were adhered to, the blood level of uric acid fell appreciably and no major attacks of gout occurred; minor attacks were very few in number. Eighteen patients who had suffered from attacks of gout for an average of seventeen years had

61 major attacks of gout in an observation period prior to treatment but noted only 3 minor episodes in the entire group during an equal time after treatment. In another group of 13 patients who had attacks of gout over an average period of four years, following the program of *special diet, cinchophen and abstinence from alcohol*, their total number of attacks fell from 23 major attacks prior to treatment to 4 minor attacks in an equal time subsequent to the institution of therapy. Seven patients did not follow the diet carefully and indulged in alcohol. This group had 23 major attacks in a test period prior to treatment as compared with 12 major and 4 minor attacks in a similar period while under diet and cinchophen (which were not followed too carefully).

Such a plan of therapy has definitely resulted in a marked decrease in the incidence of gouty attacks. It is hoped that a similar reduction in attacks can be produced if salicylates are substituted for the potentially toxic drug, cinchophen; salicylates, like cinchophen, aid in uric acid elimination in the urine.

Hench orders a diet very low in purines on two days each week and avoidance of all foods containing large amounts of purines at all times. Talbott and Coombs believed that there was no particular merit to a prolonged use of a low purine and a low protein diet in asymptomatic periods. They prescribe a diet adequate in protein, carbohydrate, fat, vitamins, minerals and fluids for most patients with gout; others use a high carbohydrate, low fat and low purine diet.[5]

Colchicine may be continued after subsidence of the acute attack in the following manner: ½ mg. (1/120 gr.) twice a day for three successive days each week, for weeks or months.

For mild aches and pains, one may use small doses of aspirin. *There is no safe method of administering cinchophen* even though the risk of fatal toxicity may be as small as one in 500,000.[49,95] It has been suggested that the risk of cinchophen therapy is much less than the danger of development of gouty nephritis, uric acid renal stones, or possibly coronary disease, if the drug is not used; it has been claimed that with the intermittent use of cinchophen, the cardiac and renal effects of the disease may be lessened. *If cinchophen (or one of its derivatives) is used, the patient should consume large amounts of carbohydrates, at least 2000 cc. of fluid per day, sufficient alkali to maintain an alkaline urine (such as 2 to 4 grams of sodium bicarbonate per day) and should report at once any pruritus, jaundice, nausea or other untoward symptoms.*

Alcohol is forbidden (except occasionally and in small amounts) in patients with gout. Although there is no good experimental evidence that alcohol causes recurrences of the disease, it has long been known that many of these patients have indulged heavily in spirits and alcoholic excesses frequently precipitate acute gouty attacks.

Although teetotalers may have gouty arthritis, the greatest number of attacks begin twenty-four hours following the drinking of wine, ale, beer, vermouth or champagne. Some of these patients tolerate small amounts of alcohol without flare-ups of the joint symptoms; we prefer to have the patient assume the responsibility for indulgence in liquors (if he insists upon this privilege).

Regimen Between Attacks of Gout

Simplicity and moderation in eating. Foods containing large amounts of purine are forbidden.
A *low fat, low purine, high carbohydrate diet* is employed.[56,79]
Colchicine, 0.5 mg. ($\frac{1}{120}$ gr.) twice a day for three successive days each week for six months after the acute attack unless diarrhea occurs.
Sodium salicylate (4 gm.) and glycine (10 gm.) daily for three consecutive days each week; these are employed as a urate eliminant.
Ferrous sulfate (gr. iii b.i.d.) and Brewer's yeast tablets (2 t.i.d.) (because of possible deficiency of iron and vitamin B in the diet).[56]
Avoidance of alcohol (except occasionally).
Avoidance of sudden chilling of the body.
Moderate amount of exercise (walking, golf, tennis, horseback riding) or massage if feasible.
Avoidance of excessive work, trauma, mental and physical fatigue.
Yearly vacations if possible.
If necessary, orthopedic measures such as physical therapy, immobilization in splints, correction of deformities, *et cetera*.[100]
In actual or threatened ulceration of tophi, excise the latter early.
If repeated major attacks occur frequently, despite this program, Bartels has suggested the use of small amounts of cinchophen, 0.5 gm. three times a day for three days each week. As the blood uric acid level falls, the dose of cinchophen is decreased. If liver function is impaired or if signs of cinchophen toxicity appear, this drug is stopped.

Low Purine Diet: Eat nothing that runs, crawls, swims or flies (*i. e.*, no meat, fish, fowl, etc.).

Eat no gravy.

Eat no peas, beans (kidney, lima or navy), spinach, asparagus, onions, kohlrabi.

Eat especially milk, eggs, cheese and nuts.

Other foods permitted include: bread and butter (except whole grain bread), all fruits, sugar, sweets, cereals (except whole grain), all vegetables, except those listed previously, gelatin, moderate amounts of tea, coffee or cocoa.

Foods Containing Moderate Amounts of Purines: Chicken, mutton, oysters, herring, ham.

Shad, tuna fish, salmon, lobster, crab, whitefish.

Asparagus, beans (lima, navy or kidney), peas, onions, spinach, mushrooms, kohlrabi, cauliflower.

Oatmeal, whole grain cereals, whole grain bread.

Foods Containing Large Amounts of Purines: Sweetbreads, liver, kidney, calf's tongue, squab, brains, sardine, anchovies.

Turkey, pork, venison, veal, beef, sausage, duck, goose, partridge, trout, carp, pike, perch, codfish, bacon, rabbit, sheep.

Lentils.

Gravies. meat soups, meat extracts.

Some clinicians have performed skin tests with purine and non-purine containing foods and have forbidden the use of those foods to which the patient gave positive reactions.[64] Others feel that small amounts of purine containing foods do no harm.

Treatment of Tophi.—If tophi on the hands or feet become large or ulcerate, excision with thorough débridement results in prompt healing and obviates the later dangers of infection, and possibly amputation.[56]

Linton and Talbott reported on the surgical treatment of tophaceous gout based on such therapy in 11 patients.[76] *Surgical treatment of gouty tophi is indicated when large deposits have developed and must be removed for cosmetic reasons or because of pain, interference with movements, discharging sinus and extensive phalangeal involvement of the toes or fingers.* Follow-up studies showed that only 3 of 93 tophi removed showed recurrences of any size. In all patients in whom tophi were removed because they were causing pain, pain was relieved following the surgical removal. Surgery should be done early in the course of tophi if these are causing symptoms.

ETIOLOGY, PATHOLOGY AND PROGNOSIS OF GOUT

Three enigmas of gout are the hyperuricemia, the altered excretion of urates and the formation of urate deposits in the tissues.[8]

Although Garrod stated that acute attacks of gout were caused by precipitation of sodium urate crystals in and around joints, the increased blood uric acid in acute or chronic nephritis or in leukemia does not often produce attacks of arthritis. In addition, injections of urates into veins or body tissues are painless.[89] Colchicine, which relieves the pain in acute attacks of gout, has no known effect on urates in the blood or urine.

Although former writers have believed that there was a precipitation of uric acid crystals into a joint in the acute attack of gout, many authorities have abandoned this concept of the attacks.

We still do not know what factors are concerned with the deposition of urates in the tissues. Deposition of sodium urate rarely occurs in conditions with hyperuricemia other than gout.

The cause of gout is not known. Much of the evidence indicates that this disease is probably a metabolic disturbance which involves uric acid metabolism or some abnormality in the renal excretion of uric acid. The increased concentration of blood uric acid in acute attacks of gout is considered by some to be a result of increased formation rather than of diminished excretion or destruction.

Thannhauser believes that the uric acid retention is primarily the result of renal dysfunction, possibly concerned with nervous connections of the kidney. This renal theory is not generally accepted because of the number of patients with gout who have no evidence of renal lesions. Grabfield suggests from experimental studies that the etiology of gout may be sought in functional disturbances of the vegetative nervous system, especially with involvement of the renal innervation.[40] Other workers feel

that the disease may be produced by any of the changes causing an alteration in the chemistry of the tissues favoring precipitation and fixation of uric acid salts.[91]

Krafka believes that the endogenous uric acid arises from the destruction of the extruded nuclei of normoblasts during the maturation of the erythrocytes and that any condition which stimulates the erythropoietic system is an important factor in the overproduction of uric acid and likewise a potential agent in gout.[69–71] Among the conditions which may stimulate the erythropoietic system and thus possibly cause attacks of gout are leukemia, polycythemia, lead poisoning (as lead is an active hemolytic agent), bleeding, hemorrhage, liver extract, etc. It has been suggested that all hemolytic or hematopoietic agents be used with care in individuals with gout.

FIG. 207.—**Gout:** large punched-out bony lesions of gout in big toe of a man aged fifty-nine years.

One of the theories concerning gout is that the onset is marked by an increase of joint fluid with a deposition of monosodium urate crystals in the fluid. These crystals adhere to and erode cartilage and replace the destroyed portion. The capsule and periarticular tissues also become involved and in the later stages, the urate crystals may replace portions of the ends of the bones and of the marrow. A similar pathologic picture may occur in the subcutaneous tissues, muscles, bursæ, cartilage or in the helices of the ear. Deposits of uric acid crystals may also occur in the kidneys and lead to uric acid stones.

Technics have been described *for the preservation and microscopic demonstration of deposits of urates in gouty tissues.*[36,96] With deGalantha's method, absolute alcohol is used as a fixative. Formalin dissolves urates; gouty tissues fixed with formalin will show no crystals but merely the clefts from which the urates have been dissolved.

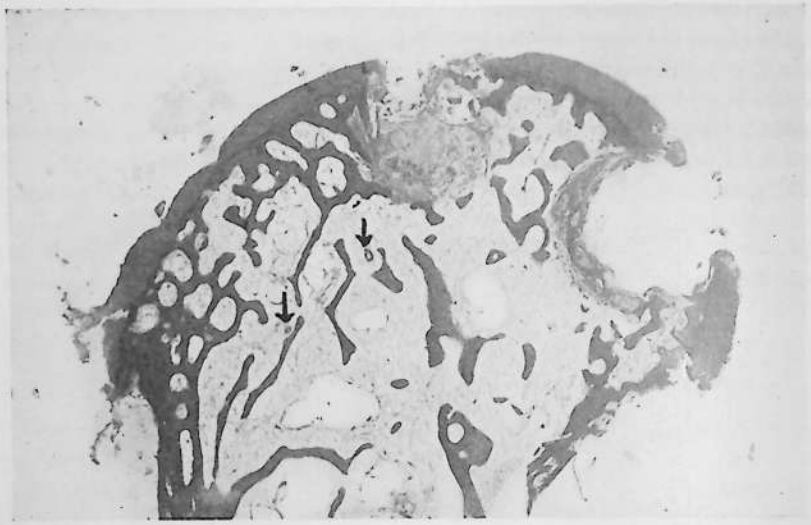

FIG. 208.—**Gout.** Photomicrograph (× 10): articular end of the proximal phalanx of the third left finger. Free surface of articular cartilage is frayed and uneven. Cartilaginous and bony overgrowth is present at the articular margins. Two *large punched-out areas* are seen *in the articular cartilage* and subchondral bone lined with fibrous tissue which is infiltrated with large numbers of inflammatory cells and studded with numerous masses of sodium urate crystals. Note the isolated urate deposits in the bone marrow (arrows). (Ludwig, Bauer *et al.*, courtesy of Ann. Int. Med.)

Most individuals believe that the excess uric acid in the blood in gout does not produce the disease but is merely an index of the metabolic disturbance causing it. In a study of gouty individuals, it was shown that diuresis began before clinical or subjective evidence of gout was seen. In these individuals there was a negative sodium and chloride balance accompanying the diuresis, and an increased excretion of calcium, potassium, ammonium, titrable acid, phosphate and urate. These observers did not find a decrease in the urinary uric acid prior to an attack of gout.

Excesses of alcoholic beverages and of food are not the cause of gout, but may be precipitating factors. The disease may affect vegetarians and total abstainers from alcohol.

Some individuals have emphasized the rôle of allergy in gout.[24,42,92] They believe that gout is an allergic reaction to an allergen in food, bacteria or tissue substance (which develops after trauma or operation).

The **pathologic changes appearing in gout** are probably secondary to the deposits of monosodium urate. Those factors which govern the diffusion of urates into various tissues are unknown; once extravascular, urates

may act as irritants leading to proliferation. The joint changes seen in gout depend upon the amount of urate deposited, its position and the reaction following these deposits. Contractures and limitation of motion from extensive fibrosis of periarticular tissues may occasionally follow uratic deposition. If the deposition of urates occurs in synovial tissues, pannus formation may be so great as to cover the articular cartilages producing a true fibrous ankylosis as in rheumatoid arthritis.[43,82,124] Actual bony ankylosis may occur in such instances.[87] Bony trabeculæ may be destroyed following invasion of the subchondral bone spaces by the pannus; this leads to the punched out areas noted on the roentgenograms. If this pannus is placed in the proper fixative, urate crystals can be demonstrated, thus differentiating the material from that seen in rheumatoid arthritis. Deposition of urates in articular cartilage may be responsible for extensive degenerative joint changes.

Most workers feel that just prior to an attack of acute gouty arthritis, urates are deposited in and around the joints producing an irritation which is manifest as an acute arthritis;[46] however, injections of urates into tissues of gouty or normal persons are painless. *Deposits of urates may occur* in bones, synovial membranes, joint capsules, ligaments, periosteum, tendons, bursæ, and in subcutaneous and intramuscular connective tissue. Necrosis may be a secondary change at the site of these deposits. In advanced gouty arthritis, fibrillation and overgrowth of cartilage may occur as well as fibrous or bony ankylosis. Tophi have been found rarely in heart muscle,[10] tongue, bronchi, pericardium, pleura, meninges and cornea. A case of a painful stump has been reported in which the discomfort was attributed to the pressure of minute uric acid crystals against nerve endings; blood uric acid was 7.2 mg. per cent.[66] Treatment of the gout resulted in relief of symptoms.

Prognosis.—This is poorest in those whose symptoms appear early in life and *best in those whose symptoms come on late in life.* Gout is a chronic disorder which is not cured when the acute attack has subsided. Treatment must be continued for life in order to postpone or prevent recurrence of symptoms. Faithful adherence to treatment (especially between acute attacks) will modify the course, reduce the severity and number of attacks, reduce the likelihood or prevent the occurrence of chronic gouty arthritis, and possibly postpone or avoid fatal renal and cardiovascular complications.

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CHAPTER LI

TRAUMATIC ARTHRITIS AND ALLIED CONDITIONS

It has been estimated that *75 per cent of industrial workers over the age of forty suffer from some type of joint affection.*[52] Outdoor work has been found to be associated with more arthritis than indoor occupations. Occupations in which joint involvement is particularly prevalent include bakers and metal workers (exposure to excess heat), dyeing and textile workers, miners, refrigerator workers, bath attendants, window and bottle washers (dampness), carpenters, farmers, laborers, postmen, locomotive engineers, policemen, chauffeurs, conductors, forestry workers and ship builders (extremes of weather).

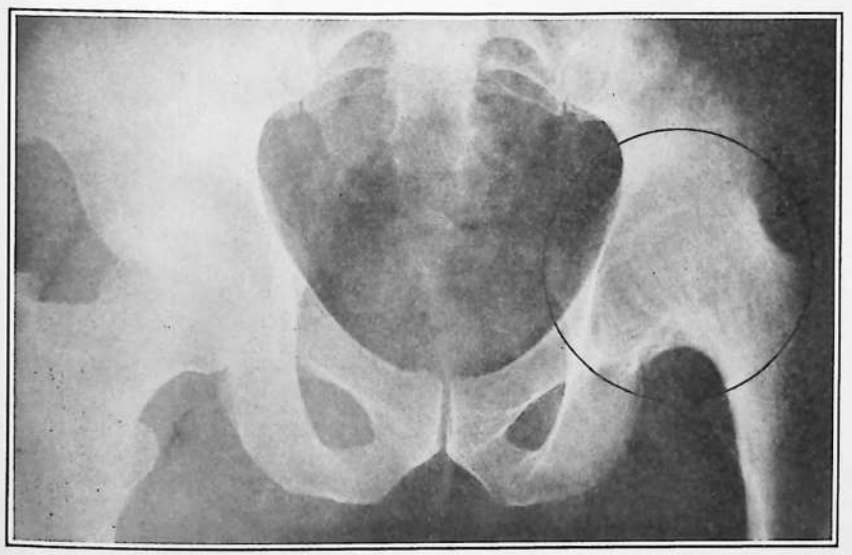

Fig. 209.—Effusion into the hip joint. Picture was taken within an hour after the patient, aged forty-five years, was struck in the lower abdomen by a heavy board.

Traumatic joint disease may result from severe blows or falls, or from trauma incident to poor posture, athletic activities or occupation.[9] Traumatic arthritis is a frequent cause of prolonged disability following injuries.[61] *In any form of joint disease, the possibility of repeated minor trauma or a sudden accident must be searched for in the history.* Such slight injury as repeated pressing of the knee against a door may result in pain in that joint. Arthritis may develop following the introduction of a foreign body (such as a needle, thorn or bullet) into a joint.[62]

In addition to traumatic arthritis, trauma to a joint may precipitate other forms of arthritis (rheumatoid, tuberculous, syphilitic, pyogenic, gouty).[16,54,56,69] Furthermore, any form of arthritis (but especially degenerative joint disease) may be made worse following trauma.

Key found that *traumatic arthritis may supervene after:* (1) a severe single injury to joint cartilage (such as a blow or fall); (2) repeated mild injuries to joint cartilage (as old injuries of the semilunar cartilage or crucial ligaments of the knee with injury to the articular cartilage); (3) disorganization of the mechanics of the joint; (4) abnormal function in a joint because of bony deformity; and (5) gradual deformity due to abnormal pressure.[63]

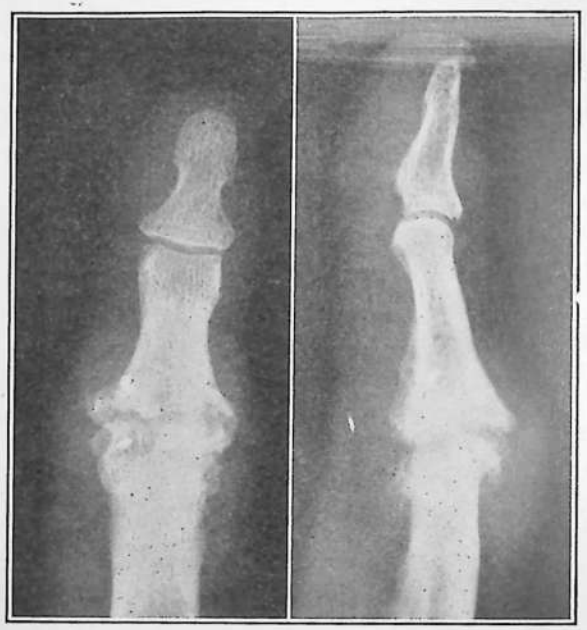

FIG. 210.—Arthritis due to foreign body. A piece of steel lodged in this index finger of the patient (now aged forty-eight) four years ago. Swelling of the finger began about two years ago and has continued to date. Roentgen-ray shows marked deformity of the proximal interphalangeal joint of the right index finger. This consists of marked hypertrophic changes with possible ankylosis of the joint. There are two small opaque foreign bodies in relation to the palmar and radial aspects of the joint.

Injuries from occupations or sports may result in specific types of disease of joints or bursæ. Some of the more common of these are:[52] weaver's bottom, nurse's feet, policemen's heel, glass arm (of baseball players), housemaid's (or nun's) knee, game knee, soldier's shoulder, tennis elbow, golfer's arm, etc. Our present concept of the pathologic process underlying some of these is as follows: weaver's bottom—bursitis at the tuberosity of the ischium; housemaid's knee—prepatellar bursitis; glass arm—subdeltoid bursitis. Other forms of traumatic joint disease include tennis elbow (from involvement of the conjoined extensor tendon at the lateral epicondyle of the humerus or the radio-humeral bursa,[17] or periostitis of the epicondyle, etc.); chauffeur's shoulder (degenerative joint disease of the shoulder joint from trauma due to the shifting of stiff gears);[93] driver's thigh (sciatic neuralgia because of the holding of the foot in one position

on the accelerator for long periods of time); shoulder affections in workers using compressed air tools,[99] the repeated vibration of which may eventually cause splitting and destruction of the joint cartilage.

Traumatic arthritis may follow prolonged poor posture, asymmetry of the skeleton, obesity, flat feet, etc., and involves especially the knees, ankles, spine and metatarsal joints (*i. e.*, weight-bearing joints). *The most vulnerable tissue in the joint is the articular cartilage.* Traumatic damage to hyaline cartilage of a joint may be serious because such cartilage has practically no power of regeneration; synovial tissue, on the other hand, may regenerate markedly.

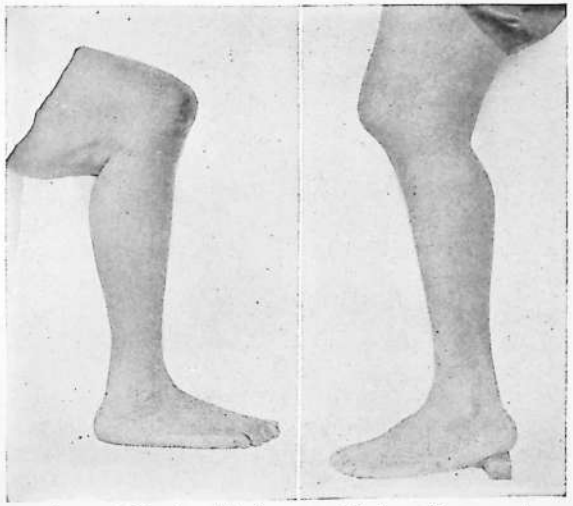

FIG. 211.—Bilateral prepatellar bursitis (housemaid's knee) in a scrub woman. (Lewin, Orthopedic Surgery for Nurses, courtesy of W. B. Saunders Company.)

Criteria proposed *for the diagnosis of traumatic arthritis:*[11]

1. Articular function must have been normal before the injury, or if previous disability was present, the extent of this must be known.

2. *Normal joint function (full painless motion) usually rules out the possibility of a traumatic arthritis.*

3. A diagnosis of intra-articular disease cannot be made upon pain alone; this may be caused by periarticular disease.

4. Traumatic arthritis usually produces muscle spasm and limitation of motion.

The effect of trauma upon articular structures depends upon the type, severity and duration of the injury as well as upon the reaction of the individual. *The period between the injury and the production of a traumatic arthritis may vary from days to several months. Pain and limitation of motion may persist for years following a single strain or contusion,* even without obvious anatomic change.[36]

Traumatic joint effusions usually show a lymphocytosis for several days after the accident.[10] The change from a bloody effusion to a serous,

amber colored, limpid effusion occurs within fourteen to eighteen days at the earliest. Cytologic examination of traumatic effusions reveals little or no polymorphonuclear leukocytosis but synovial cells and lymphocytes may be seen.

Characteristics of Traumatic Arthritis

Joint phenomena are usually monarticular; bilateral or polyarticular involvement is uncommon.

Symptoms may result from reactions in synovial membrane, capsule or articular tissues.

Symptoms include: pain, swelling, crepitus on motion and possibly muscular spasm and limitation of motion.

Skin temperature is usually normal.

Joint effusion may or may not be present.

In most instances, a traumatic arthritis begins within a week following injury although symptoms may not be noted for periods varying from several weeks to six months or more.[35,49]

Pathologic findings include degeneration of articular cartilage, capsular fibrosis, hypertrophy of the synovial membrane and production of new cartilage and bone around the articular margins.[99]

Roentgen examination may be negative in the early stages and may reveal osteophytes or hypertrophic changes later. Traumatic lesions (such as tears in the cartilage, hypertrophied synovial membrane, narrowed or obliterated suprapatellar or infrapatellar spaces, etc.) may be revealed by roentgenogram following the intra-articular injection of air, carbon dioxide or contrast medium.

Synovial fluid in traumatic arthritis contains large numbers of erythrocytes, a variable total white count (50 to 7300 cells per c.mm.) and differential leukocyte count, increased amount of bilirubin from the breakdown of erythrocytes, variable amounts of fat (torn from intra-articular fat pads or from bone marrow if intra-articular fractures are also present),[96] and the presence of mucin (even in effusions aspirated within twenty-four hours after injury).[68]

The icterus index of synovial fluid in traumatic arthritis is usually above 5; this is almost invariably under 5 in inflammatory effusions. The bilirubin content of synovial fluid is raised only rarely (except from trauma); uncommon causes include hemophilia, sarcoma, xanthoma and tabetic arthropathy.[50]

Acute traumatic arthritis, synovitis or periarthritis are usually recognized without difficulty. However, injury may reactivate a previous form of arthritis, or an acute arthritis may develop on a basis independent of the injury.

Differentiation of a sprain (tearing of a ligament) from traumatic arthritis or synovitis may be difficult; in synovitis, however, considerable fluid is usually observed in the joint.[19] *Years of pain and limitation of motion may follow a single strain or contusion even in the absence of anatomic changes.* Severe single injury or repeated mild trauma produces the joint picture of a degenerative joint disease.[28] Injury may also aggravate an existing arthritis.

Treatment: Physiologic rest of the affected joint. Temporary immobilization may be procured by plaster splints or adhesive strapping. Hot compresses or an electric pad may relieve discomfort. *Early aspiration of traumatic effusions* (twenty-four hours after injury) *with the application of a pressure bandage decreases the danger of chronic inflammatory changes in the joint.*[70,88]

Pathology.—The pathologic reaction resulting from such trauma resembles that of degenerative joint disease. Fischer believed that the latter was the only form of arthritis for which mechanical trauma was solely or mainly responsible and that occupational arthritis generally involved only the shoulders, hands and elbows.[33] In his opinion, deforming arthropathies of the feet, knee and hip were caused more often by static influences than by the trauma of occupation.[30]

FIG. 212.—**Traumatic arthritis of the right hip** with bony defect and numerous foreign bodies due to a gun shot. This man, aged thirty-seven years, was shot in the right groin one year ago. He was hospitalized for three days; no roentgen-rays were taken and the bullet was not removed. The left hip is negative. There is a mass of opaque material scattered in heterogenous fashion throughout the region of the right femoral head and neck. The joint space is considerably decreased in size and there are hypertrophic changes in the bone in the region of the weight-bearing surface on either side of the joint. In addition, there is a small groove on the superior surface of the neck of the right femur which may be the area through which the bullet pierced and was scattered. The bullet was probably of very soft material to allow this type of distribution.

Injury produces a two-fold effect upon joints: (1) mechanical damage such as capsular tear, joint effusion, detachment or laceration of cartilage, articular fractures, etc., and (2) joint reaction to such trauma. The latter includes inflammation and secretion of synovial fluid and the production of normal and abnormal bone due to a resumption of the osteogenetic functions of bone. Degeneration of the central portion of the articular cartilage may occur; this portion has no known blood supply. Recent traumatic effusions are usually hemorrhagic in nature.

The primary *pathologic reaction* in traumatic arthritis is a synovitis. Secondary hypertrophic changes may occur and may resemble those of degenerative joint disease. Doub and Jones studied 30 patients who

suffered fracture of one of the long bones of the leg and were not able to prove that a single severe trauma produced degenerative joint disease.[29] In 28 of these cases, no evidence of arthritis was noted in the adjacent hips or knees during a period of eight months following such injury. They believed that chronic trauma was more likely to result in degenerative joint disease than single acute trauma. The more common sites of bursitis are the subdeltoid, olecranon, prepatellar and trochanteric (hip) bursæ.[103]

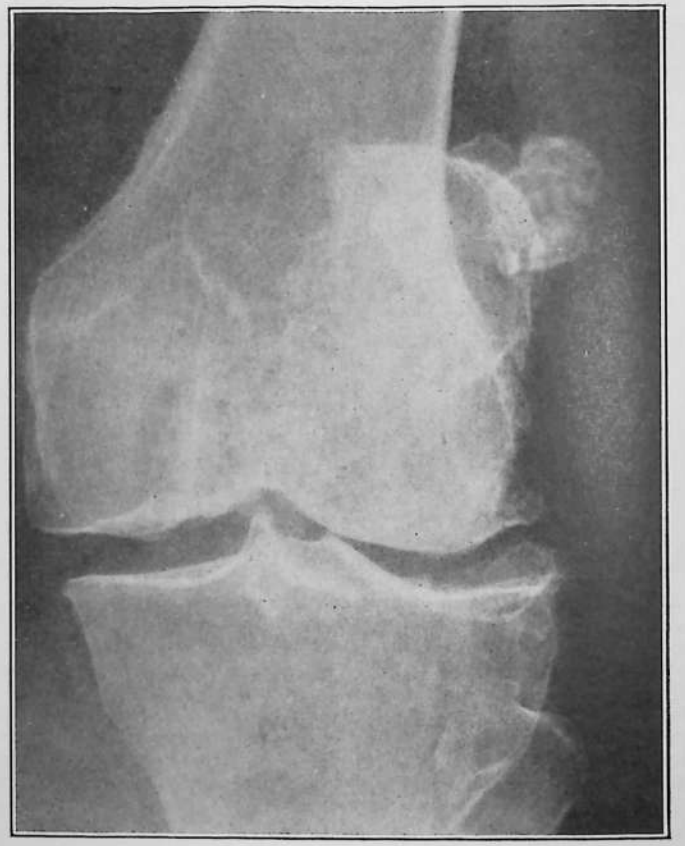

FIG. 213.—Calcification in the suprapatellar bursa; slight hypertrophic changes in the knee joint.

Trauma may also lead to *bursitis*.[27] One such bursa, which was removed from over the ischial tuberosities (misdiagnosed as a lipoma), weighed 2 pounds 10 ounces and measured 6 inches in diameter.[38]

Proliferation of periarticular fibrous connective tissue may cause stiffness in the joints of the hands or feet following injury. Limitation of motion and pain are evident; muscular spasm and contractures occur later. In traumatic tears of a joint capsule, a serofibrinous exudation occurs which may lead to the formation of adhesions. Such inflammatory

or traumatic edema is "the glue from which adhesions are made." *Joint adhesions*, following injury, usually form in the periarticular tissues.[60] Periarticular adhesions in plications of the joint capsule may produce stiffness of the joint.

Treatment.—In acute traumatic arthritis, *rest, heat and the removal of any large fluid collection* form the basis of therapy. In any hemarthrosis (except in hemophilia), aspiration of the bloody fluid should be performed as soon as possible to prevent the formation of dense adhesions. Hall and Hansson stress the necessity of vigorous early treatment to the involved joints to prevent chronic disability.[47,48] Carruthers advised early aspiration of synovial exudates in knees with avoidance of weight bearing for a week.[18] Others do not favor such routine aspiration in traumatic synovitis unless the fluid persists.

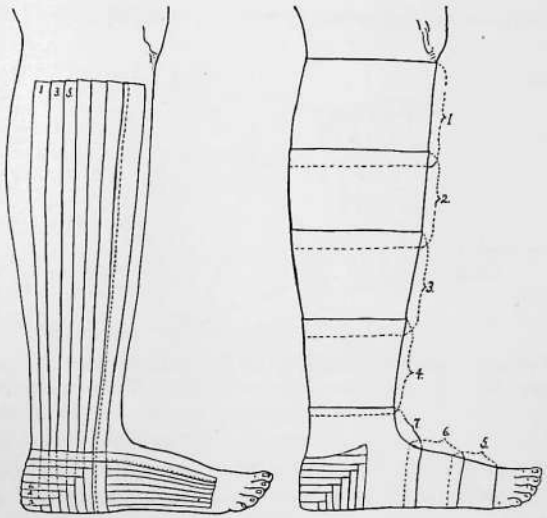

FIG. 214.—Technic of strapping the sprained ankle. (Ochsner, courtesy of Medical Record.)

A *sponge compression dressing* has been used in patients with traumatic synovitis of the knees. Forrester applies a protective cotton pad in the popliteal space, held in position by a circular dressing of sheet wadding.[34] A porous rubber bath sponge cut longitudinally to fit the contour of the synovial cavity is then held in place by a gauze bandage. The rubber sponge expands as the exudate resorbs, thus exerting a constant compression which hastens the absorption of the exudate. With this dressing, one may avoid the use of aspiration, casts or splints; the bandage is reapplied every four or five days. During this form of treatment, the patient may continue his work. Edema of the leg does not occur.

Additional Treatment of Traumatic Arthritis.—This includes: (1) Prevention of the disease by restoring normal alignment and obtaining accurate anatomic reduction in fractures; in the case of joint fractures, open opera-

tion may be necessary to obtain anatomic reduction; (2) medical treatment (by weight reduction in obese individuals with traumatic arthritis affecting weight-bearing joints); (3) physical therapy (rest, heat and massage) and (4) surgery (including removal of foreign bodies in joints).[63]

It is not good practice to leave *foreign bodies in or near a joint.* These may result in prolonged, mild, low-grade infection and destruction of the joint or progressive degenerative joint disease. Key cited cases in which foreign bodies in or around joints had resulted in marked joint changes at a later date. In one man of sixty-five, a bullet had been removed from the lateral condyle of the femur forty-five years ago, followed by drainage from the knee for several months. A degenerative joint disease gradually developed in this knee with narrowing of the joint space and marked hypertrophic changes. Foreign bodies left in an intervertebral disk might result in degeneration of the disk and fusion of the bodies of the vertebræ. The fusion produces no untoward symptoms in most instances.[64]

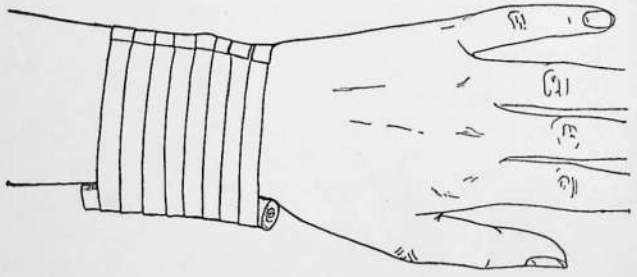

FIG. 215.—Technic of **adhesive strapping for the sprained wrist.** Note the thin roll of gauze bandage under the adhesive on the radial side. (Ochsner, courtesy of Medical Record.)

Other forms of therapy in traumatic arthritis include histamine iontophoresis,[70,71] diathermy,[13] and occasionally synovectomy[111] and arthroplasty.[82,83]

Graduated exercises have been recommended by Tucker to prevent marked wasting of the quadriceps muscle following injuries to knees.[115] These consist of:

1. Contraction of the quadriceps for two minutes every hour while lying on a couch.
2. Alternate flexion and extension of each knee (twenty times twice a day) with the knees bent and the legs hanging over the edge of the couch.
3. Lying flat in bed; raise the extended leg by flexing the hip. Abduct and then adduct the thigh at the hip.
4. As improvement occurs, bicycling of the legs.
5. Later, use weight-bearing exercises, such as raising the heels, bending the knees, and straightening upright while holding the back of a chair.
6. Skipping.

The joint which is the seat of a traumatic arthritis may be splinted by Castex or molded plaster of Paris; such splints should be used continuously

during the acute phase of the disease, being removed once daily to permit several full painless motions of the joint (in an effort to prevent the formation of dense adhesions). Temporary relief of pain may be obtained by the use of local heat and very light massage to the affected joint.

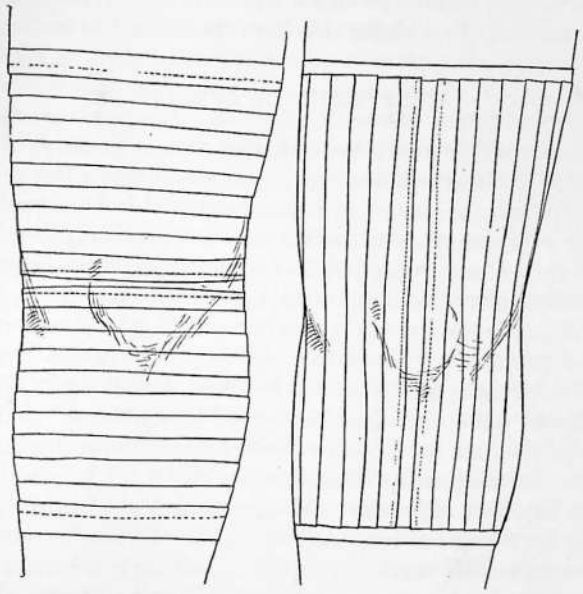

FIG. 216.—Technic of adhesive strapping for the sprained knee. (Ochsner, courtesy of Medical Record.)

In the **treatment of chronic traumatic arthritis,** Hench advises *rest* and *avoidance of any operative trauma; physical therapy; correction of posture;* use of *proper shoes,* supports and exercises for the feet; weight reduction

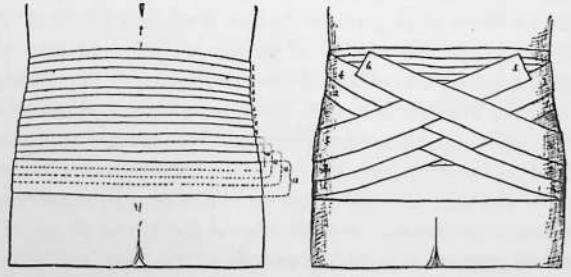

FIG. 217.—Ochsner **technic of strapping the low back.** (Ochsner, courtesy of Medical Record.)

(if indicated); and, in some cases, the use of splints or roller bandages, and special corsets or braces. He has stressed prophylaxis against traumatic arthritis by periods of rest in industrial workers and the insulation of traumatized joints by the use of leather pads and other forms of apparatus.

Proper adhesive strapping following procaine injection is the best treatment for *simple sprains.*

The most commonly affected sites are the ankle, wrist, knee and sacroiliac joints. Ochsner has diagrammed the technic of strapping these areas.[94] He has also advised rubbing about a teaspoonful of cold cream into adhesive as an easy method of removing this from the skin.

TREATMENT OF SPRAINS

A sprain may be defined as a stretching or tearing of ligaments or the capsule about a joint. Miltner and his co-workers found that after experimental sprains in rabbits, the tissues were grossly normal in four weeks but that microscopic evidence of inflammation persisted for several weeks longer.[91] In view of this, Hench suggested that sprained joints be protected for at least two weeks after the disappearance of clinical signs.[53]

In the average sprained ankle, rotation of the calcaneum occurs in the direction of pronation or supination, causing the thrust of the weight of the body to fall upon the lateral ligaments of the ankle joint; in the case of pronation of the heel, the calcaneo-tibial ligament is strained, while in supination of the heel, the calcaneo-fibular ligament is involved. Stretching or tearing of these ligaments results in a sprain.

Procaine Injection of Sprains.—Manpower shortage has been minimized in wartime by the use of local anesthesia injected directly into the most tender area soon after sprains or injuries when there is no accompanying open wound. Such injections greatly reduce the pain and permit the injured individual to remain active.[4,75]

In the usual sprain there is an injury characterized by partial tear of ligaments and small blood-vessels with the formation of a hematoma; the latter is composed of more tissue fluid than actual blood. Most of the pain and loss of function which result are probably due to the tension which develops from the confining of this fluid to a small space. *The fluid accumulation and the disuse resulting from pain on motion lead to stiffness of the part.*

The effect from injection of a local anesthetic solution into this tender area persists long enough to permit removal of some of this fluid, sufficient in amount to relieve the tension in the injured area. The relief of pain then permits sufficient muscular exercise to cause an increase in local circulation and to exert enough mechanical pressure to force the accumulated fluid into channels for its removal. The gentle massage produced by motion promotes drainage and decreases the effusion.

The **technic of procaine injection** is as follows:

1. The area to be injected is washed with soap and water, followed by the application of iodine and alcohol.

2. One or 2 per cent procaine is used; this is injected directly into the most tender subcutaneous tissues, withdrawing the barrel of the syringe before injection to prevent against injection into the general circulation (inasmuch as there is usually a large vascular network in the region of most sprains).

3. If possible, the tender spot or spots should be injected without withdrawing the needle to minimize the danger of infection.

4. No more than 2 separate punctures (with sterile needles) should be made at any one time (to reduce the possible avenues of entrance of infection).

Contraindications to procaine injection in sprains:

1. An *infected or potentially infected area* at or near the site of sprain.
2. An *open wound.*
3. *Idiosyncrasy to procaine;* another local anesthetic may then be chosen.
4. *Rupture of a medium sized or large vessel* at or near the area of sprain.

McMaster compared the results of the treatment of ankle sprain by three methods:[86]

1. Injection of procaine hydrochloride.
2. Adhesive strapping.
3. No treatment or in some, cold and hot packs or an elastic bandage for support.

These patients were *men* in the late teens and twenties (with some in the fourth, fifth and sixth decades) in *active military service;* eight women were included in the group. Sprains were of all grades, but were sufficient enough to cause the individual to seek relief.

Ninety per cent of the sprains were of the anterior talofibular ligament. The order of frequency of sprains of other ligaments was: the calcaneofibular, anterior inferior tibiofibular, medial or deltoid, posterior talofibular and posterior inferior tibiofibular. Fractures or sprain fractures were not included in the group.

Leriche and others have described the technic of local procaine injection.[76–78] From 10 to 20 cc. of 1 or 2 per cent procaine solution may be used and **all tender areas in the injured ligament, as well as tender areas in other ligaments are injected** until no tender or painful areas remain on palpation or on ankle or foot motion. McMaster did not hesitate to use 30 cc. or more of the anesthetic solution if necessary.

An *elastic bandage was then wrapped snugly around the ankle.* If pain was noted on walking, further injection was performed. *The patient then returned to duty* and was instructed to use the foot and ankle normally except that no running or jumping was done. The individual was requested to move the foot, even while sitting, and not to keep it immobile in one position even for short periods.

The elastic bandage was removed by the patient and the part was rewrapped in an hour or two to prevent possible circulatory constriction.

Best results were obtained in those injected by procaine and returned to duty immediately. Many had no disability; in the others, this rarely lasted more than twenty-four or forty-eight hours. Reinjection was never necessary; pain rarely recurred after the injection. Motion of the ankle was often normal or nearly so on the day following injection. For several days, forced passive motions as inversion and eversion were apt to be painful. Swelling decreased steadily and disappeared even in the severe cases within five to ten days. The injured ligaments remained tender to palpa-

tion for three to five days although there was no pain on active motion. Local or diffuse ecchymosis over the foot or toes often persisted for a week or two.

McMaster felt that the **success of the injection procedure depended upon** two factors:

1. *Complete adequate initial intraligamentous injection to produce total elimination of pain and tenderness; and*

2. *Continued use and motion of the foot and ankle;* this second point was the more important.

Stiffness, swelling and pain result and persist if the injection fails to relieve pain so that the patient limps and does not use the part normally or if he is apprehensive and does not move the part as instructed. Active use and motion seem to improve the local circulation, thus enhancing tissue metabolism and repair; the motion and use help to mechanically express the extravasated serum and blood from the region, materially reducing the swelling and stiffness with pain consequent to stasis.

Normal use is not contraindicated after injection as normal flexion and extension of the ankle does not aggravate the injured lateral or medial ligaments. The ankle may be turned again by strenuous running and jumping so that these should be prohibited for a few days.

Numerous workers have reported good results following local injection treatment for sprains.[1,5,37,67,107] Others reported satisfactory results following the use of ethyl chloride spray.[25,72]

McMaster concluded that irrespective of the type of treatment used, *the patients who returned to normal activity immediately and moved and used the foot and ankle improved much more rapidly than those who did not.*

If sprained joints are subsequently used for vigorous exercise, they should be protected by supportive bandage.[39,114]

In the knee the medial collateral ligament is injured five times as often as any other ligament; sprains of the shoulders, wrists and elbows are much less common. In men in service, the possibility of march fractures of the foot and tibia must be borne in mind.[14]

Principles of Adhesive Strapping.—1. Strapping must not interfere with the circulation of blood.

2. Straps must be of proper width and length varying with the part strapped.

Adhesive Strapping of the Sprained Ankle.—As a preliminary measure, the hair of the lower leg is *shaved* and the whole lower leg is *painted with compound tincture of benzoin* to lessen the tendency to irritation of the skin. A thin felt or flannel pad or several layers of *gauze* (about 3 or 4 inches long) and slightly wider than the tendo Achilles are attached to the skin *over the tendo Achilles* (by skin varnish or adhesive). This will tend to prevent any irritation to the heel from the adhesive.

Before applying the adhesive the patient holds tightly both ends of a short piece of muslin bandage which passes under his forefoot and maintains flexion of the foot. The pull on the forefoot is not relaxed until the adhesive bandage is completed.

1. Hold the foot relaxed at slightly less than a right angle.

2. For an average sized adult, prepare 7 strips of adhesive 20 inches long and $\frac{3}{4}$ to 1 inch wide, and 7 strips varying from 8 to 15 inches long and 1 inch wide.

3. Begin with a long strip about an inch above and posterior to the internal malleolus and carry this under the sole of the foot and up on the outer side terminating 5 or 6 inches above the external malleolus. Next apply a short strip with its midportion low over the tendo Achilles (which has been covered with gauze) and extend each end anteriorly on the dorsum of the foot, running parallel to the sole.

4. Apply alternately the additional 6 long and 6 short strips, overlapping each other by about one-third.

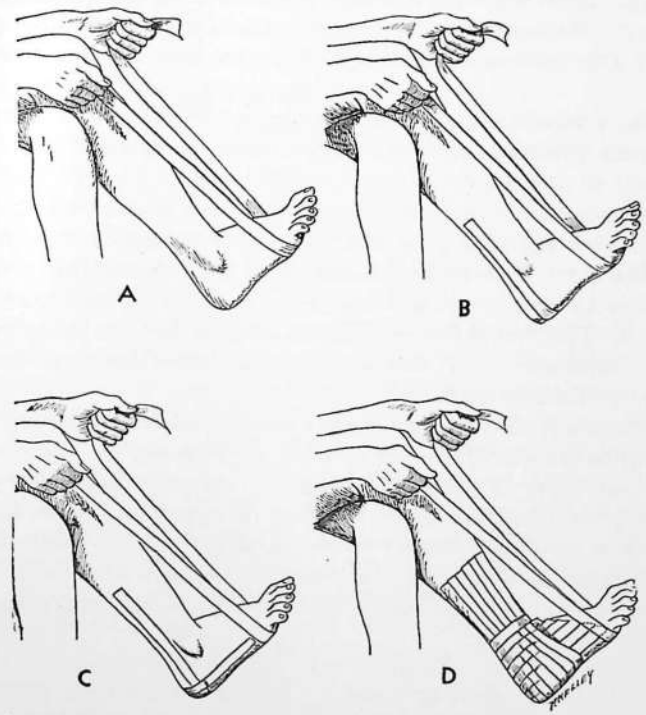

FIG. 218.—Adhesive strapping of the ankle (especially useful for sprains, and acute pain of foot strain). Adhesive strips are 1 inch wide. Place a pad of thin felt, flannel or gauze over the tendo Achilles before strapping. (Dickson and Diveley, Functional Disorders of the Foot, courtesy of J. B. Lippincott Company.)

A roller bandage is applied over the strapping and may be removed after the adhesive has become firmly attached to the skin.

Following recovery from severe sprains one may wear a leather or canvas anklet with whalebone or thin steel springs as reinforcement; areas are cut out at the malleoli and heel to prevent undue pressure. This usually extends for 3 or 4 inches above the ankle, and laces anteriorly. In some

individuals, it may be advisable to wear such a support for eight to twelve months after a severe sprain. One may also temporarily raise the heel on the affected side about ¼ inch if required.

Adhesive Strapping of a Sprained Wrist.—Place a hard roller bandage 3 inches wide and ½ inch thick on the more painful side of the wrist.

Fasten this in place by ½ inch strips of adhesive; several layers of adhesive may be applied over this (see diagram).

Strapping of the Sprained Knee.[94]—The knee is a relatively weak structure, exposed to trauma and is often injured. Prolonged disability may result from strains which produce rotation and lateral motion.

A sprain or tear of the lateral ligaments (usually the internal) is the commonest injury to the knee. Displacement of the semilunar cartilage is frequently associated with tear of a lateral ligament (in some cases producing no locking). The cruciate ligaments (especially the anterior) are often injured; other tendons and ligaments surrounding the joint are rarely injured.

A double or bipartite patella may appear as a joint body. Such a patella rarely causes difficulty unless the knee is injured. The extra patella may be removed if necessary.

A twisting strain in older individuals may produce loose bodies in the knee joint with resulting pain and swelling of the joint. Most of these loose bodies come from fragmentation of an osteochondritis. Loose fragments (with an osseous center) may grow in the joint and cause locking of the joint. They should be removed as soon as they are discovered.

Strips of adhesive 1 inch wide and about 3 inches less than the circumference of the limb are used.

The first strip is placed transversely over the middle of the patella.

Other strips are placed above and below, overlapping the preceding ones.

The second layer of strapping is applied vertically, beginning over the patella and coming within about ½ inch of the upper and lower borders of the preceding strapping; further strips are placed on either side of this.

A third layer of strapping is applied horizontally exactly as the first layer, except that strips are somewhat longer and come within an inch or two of encircling the leg.

A roller bandage may be applied over this for several hours.

In a study of 290 cases of **traumatic synovitis of the knee with effusion,** Titus obtained good results by means of *static current.*[112,113] He attributed the diminution in the extravasated fluid to the decongesting action of this high voltage current. In his cases, the fluid could be removed entirely in three or four treatments. In the period between such treatments, a figure of eight elastic bandage to the knee produced adequate support. In his experience, the application of radiant heat (such as infra-red radiation) was as effective as diathermy; massage was useful when applied above the joint to aid the lymphatics in removing the serous effusion. Favorable results were obtained with static current in sprains and in acute injuries of joints or muscles.

O'Donnell outlined the following *program to restore an injured knee to functional activity:*[95]

1. Application of *cold compresses* to the joint *for a day or two* if much effusion is present.
2. *Heat to the upper portion of the thigh,* followed about the second or third day by *gentle massage* of the upper third of the thigh, stroking upward.
3. *Muscle setting exercises of the quadriceps* to prevent atrophy of this muscle.
4. *Application of heat to the knee as the swelling subsides;* heat to the knee and thigh must be gentle and not too prolonged at first.
5. Begin *assisted active motions* (but do not cause pain); lateral and rotatory movements must be avoided. Between these treatments, the knee is immobilized by a *posterior molded plaster splint* or by adhesive strapping with additional protection by felt pads placed laterally.
6. Atrophy of the thigh muscles may be prevented by *early restoration of motion* with the aid of *strapping and crutches.*
7. As pain lessens, *resisted exercises* are begun and *deeper massage* is used. Resisted exercise may be performed by the patient by sitting on the edge of the table and resisting extension of the affected leg by crossing the other foot in front of it.
8. The physician must be sure that the patient is wearing shoes which fit his feet and will not throw the leg out of balance when unrestricted walking is permitted.

Moschowitz has described an **inflammation of the sartorius bursa** which has not been noted previously.[92] In his 25 cases, the disease occurred almost exclusively in women. The *characteristic history* was as follows: there was pain in both knees only on ascending or descending stairs; walking on the level caused no pain. Flexion or extension of the knee joint by the examiner was not painful and the joint was not tender. An area of tenderness was found at the insertion of the conjoined tendon of the sartorius, semitendinosus and gracilis on the inner aspect of the tibia. A slight swelling occurred occasionally in this area. Roentgen examinations were negative. Most of these patients had large lower limbs which were out of proportion to the size of the body; some were overweight. He explained the syndrome as due to a bursitis resulting from strain in using the sartorius and gracilis muscles, which lift the body upward and downward in stepping. No specific treatment was recommended; best results were obtained by a reduction in weight.

Strapping for Sacro-iliac and Lumbosacral Sprains.[94]—Strips of adhesive 3 inches wide are placed transversely around the body extending for at least 2 inches beyond the crests of the ilia; the lower strip is applied first across the lower portion of the sacrum.

These strips overlap each other by about one-half of their width; the highest strip comes just below the level of the lower rib margins.

A second layer of strips is applied obliquely posteriorly overlapping each other by about one-half.

A third layer is applied as the first, extending somewhat beyond the

ends of the first layer. Vertical strips are then used to cover the free ends of the adhesive anteriorly and to reinforce the strapping every 6 or 8 inches posteriorly.

Industrial Backache.—Industrial backache is a very important problem. The commonest types of industrial backache are:[65] (*a*) *injury to the erector spinæ muscle* as may occur following the carrying of a heavy weight; (*b*) *lumbosacral or sacro-iliac strain;* (*c*) *crushed or impacted fracture of a vertebral body* (this may occur after slight trauma, and is seen most frequently at the junction of the thoracic and lumbar spine; a lateral view of the spine may be necessary to demonstrate this lesion); (*d*) *injury of the intervertebral disks;* (*e*) spondylolisthesis (which is possibly a congenital lesion and may not be the cause of low back symptoms following injury). Kidner has stated that **undoubted evidences of organic changes include:** *muscular spasm, sensitivity to pressure* at the site of the lesion, *constancy of symptoms and signs, limitation of motion* appropriate for the lesion, and roentgen and neurologic findings.

Jepson described four groups of traumatic backache:[59] Trauma in a patient who has had arthritis of the spine or pelvis previously.

Trauma to the muscles, tendons and ligaments.

Predisposition to injury because of faulty posture.

Damage produced by occupational strain.

In medico-legal cases, when the question of traumatic arthritis is raised, it must be remembered that *in individuals of fifty years or more, degenerative joint disease is almost always present independent of any occupation, especially in the lumbar, cervical and sacro-iliac regions.*

TEMPOROMANDIBULAR JOINT SYNDROME

Costen described a group of symptoms associated with disturbed function of the temporomandibular joint; these included headache (the most common); impaired hearing; stuffy sensation in the ears; tinnitus; pain in the ears; dizziness; nystagmus; burning in the throat, tongue and side of the nose; dry mouth; and occasional herpes of the external auditory canal.[23,24] The symptoms were attributed to overaction of the joint at first, in addition to the later development of a loose, pathologic joint produced by absorption of the meniscus, condyles and surrounding bone.

Anatomically, the pain may be due to: (1) erosion of the glenoid or mandibular fossa with impaction of the condyles against the thin bone separating them from the dura; (2) irritation of the auriculo-temporal nerve by the uncontrolled movement of the condyles backward or mesially; and (3) irritation of the chorda tympani nerve by the condyle. Malocclusion and destruction of the mandibular joint are important in the etiology of glossopharyngeal neuralgia.

This pain may be partially or completely relieved by moving the condyle downward and away from the joint by interposing test disks between the jaws; the thickness of the disk may be judged grossly by the looseness of the lower jaw. The success of permanent relief depends on the chance

of perfect repositioning the jaw by dentures to increase its vertical dimension.

Wounds of Joints.—Cope has discussed wounds of joints from a civilian and military viewpoint. It is usually easy to ascertain the type of wound.[21] The *high velocity sharp-pointed bullet* may leave only one small puncture where it has entered; in other cases, one may see the wounds of entry and exit. *The exit wound may be far from the joint in another part of the limb in the case of a bullet with an oblique line of trajectory.* An antero-posterior and lateral *x*-ray should be taken before operative treatment is used in order to tell what damage has been done to the bones.

First aid treatment in any joint wound includes protection from further external infection and immobilization of the joint. Antitetanic serum should be given in most cases and shock and pain must be treated. Local pressure by bandaging will usually control moderate hemorrhage, but it may be necessary to temporarily apply a tourniquet if the main vessel is bleeding (such as the popliteal artery in a knee injury). Immobilization is usually accomplished by splinting; if necessary, the upper arm may be bandaged to the body or the lower limb splinted by the good limb.

In the civilian type of penetrating wound (as by a blunt or pointed instrument piercing a joint), some infection is practically always carried in. If the tissues are not disturbed, they can usually deal with this. Unless one is sure that the instrument was perfectly clean, it is usually advisable to excise the edges of the wound down to the synovial sac, put sulfanilamide or microcrystals of sulfathiazole into the wound and place a narrow drain down to but not into the joint. An adequate splint (or plaster cast) should then be applied. If no fever ensues, the splint may be removed in several days and gentle movements of the joint may be begun. If it is thought that the puncture wound may have been caused by an infected instrument, sulfonamides should be given orally for at least several days.

Foreign materials may be driven into the knee joint especially during automobile accidents. One must thoroughly explore all puncture wounds around the knee joint before sewing these.

Bick and Pheasant applied *sulfonamides topically to the deep tissues in traumatic wounds* or surgical incisions and avoided the skin and immediate skin and subcutaneous areas.[12] This was just as effective in preventing infection as when spread or sprayed over the entire wound area. It was found that far smaller doses than are usually recommended have proved adequate and did not retard healing significantly.

Sulfonamides were applied safely to synovial tissues of joint cavities and to bursæ and tendon sheaths. There was no increase in adhesions or fibrosis when reasonably small quantities were used. Sulfanilamide and sulfathiazole were used interchangeably in joints and synovial surfaces. Sulfanilamide was recommended because of its smoother handling and greater local staying power.

If infection develops, as evidenced by pain, fever and swelling of the joint, the *joint should be aspirated and cultured. Sulfonamides* should be given by mouth (as sulfamerazine, 1 gram every eight hours day and night

in adults), and if available, 20,000 to 50,000 units of penicillin may be injected into the joint in 5 or 10 cc. of sterile saline after withdrawal of the cloudy joint fluid; this may be repeated daily for several days, if it is found that the organism in the joint is amenable to penicillin therapy.

In *severe infections* with organisms amenable to penicillin therapy (particularly in hemolytic streptococcal and staphylococcal infections), **penicillin** *may be given intramuscularly every three hours day and night in a total dose of 100,000 units per twenty-four hours. Repeated aspirations of infected joints may be necessary;* in some cases it will be necessary to make a free incision for drainage; the drainage tube should not be placed into the joint.

Modern bullets traveling at a high velocity may go through a joint causing little disturbance and no appreciable infection; rapid healing and return of function with good movement may occur if the bony articular surfaces have not been damaged. If pain, fever and swelling do not occur after such a joint has been kept at rest, surgical interference is not indicated. When these cases are seen several hours after the injury, it is best not to interfere surgically if no untoward symptoms have developed. One may excise the wounds of entrance and exit down to the synovial sac if these are seen very soon after the infliction of the wound.

Considerable bloody effusion may occur in a joint cavity with loose bony fragments in a joint if a bullet traverses the joint and seriously injures the bony articular surfaces. If there is no retained foreign body in such joints, it is usually better not to open the joint, but to put it at rest by a molded plaster splint. *If it is believed that a portion of a bullet, bomb or shrapnel is within the joint, operative treatment is almost always indicated.* After the wound of entry has been excised, another clean scalpel should be used to open the joint and search for foreign bodies. Sulfanilamide may be implanted in the joint cavity. After resuturing, the limb is immobilized in plaster. Sulfonamides may be given by mouth to these patients for five to seven days. If infection occurs in the joint, the cavity should be tapped and 20,000 to 50,000 units of penicillin should be injected into the joint cavity. If a culture of the joint fluid reveals organisms amenable to penicillin therapy, daily injection of penicillin may be given into the joint cavity, and penicillin may be given intramuscularly in doses of 100,000 units per day in divided doses every three hours day and night. If one uses 2 parts of physiologic saline and 1 part of 1 per cent procaine as diluting fluid for the penicillin, no pain occurs after such intramuscular injections. Procaine does not inhibit the action of penicillin (contrary to its inhibition of sulfonamides).

In severe lacerated wounds with badly injured articular surfaces which may be broken into fragments and contaminated by various kinds of débris, amputation may be required if the main vessels of the limb are destroyed. *One should always give the joint a chance to recover if the main vessel is intact.* Antitetanus and anti-gas-grangrene serum should be administered and shock must be treated; when the patient is in condition, the wound is cleaned and infected tissues are removed. Sulfanilamide is implanted in the wound. A portion of the synovial membrane is then left open for drainage and the limb is encased in plaster. If infection occurs, sulfonamides and/or penicillin should be administered in adequate dose. Penicillin therapy should be pushed if culture reveals that the offending organism is amenable to such therapy. One hundred thousand units may be given daily in divided doses every three hours intramuscularly day and night, and 20,000 to 50,000 units of penicillin may be injected into the joint cavity daily.

The use of sulfonamides, penicillin, tetanus toxoid and other military measures have greatly decreased the necessity for amputation in these cases. In World War I, many lives were lost because surgeons tried to save limbs when the patients were rapidly losing ground from the general effects of infection. At present, if a suppurative process continues despite adequate drainage, and sulfonamide and/or penicillin therapy, and if the patient's general condition (as evidenced by fever, wasting and general appearance) makes it obvious that the patient's general strength is being definitely but steadily undermined, *amputation* may then be necessary. It is the consensus of present opinion that with newer methods of chemotherapy, *the surgeon must now really prove that amputation is a necessity and that further sulfonamides or penicillin will not be adequate assistive treatment.*

The most serious of these joint wounds are those of the hip and knee. At the end of World War I, over 80 per cent of all kinds of joint injuries healed without suppuration. The less complicated synovial sacs and more easily procured drainage in the upper extremity give a better outlook for wounds of these joints.[21]

Pneumatic Tools.—Pneumatic tools do not cause much occupational trouble. *Following prolonged use, a Raynaud-like syndrome develops* in the hands with numbness, tingling and coldness in the fingers, and occasionally in the whole hand; this has been called "dead hand."[7,102] Only 1 case of *Dupuytren's contracture* was seen following the use of the pneumatic drill.[22]

Small areas of decalcification of the bones of the carpus may appear as a late phenomenon in these workers, as may periosteal overgrowth on the volar surfaces of the shafts of the phalanges.

Actual injuries of joints may occur in these workers, with pain and limitation of motion, especially in the right elbow; these occur in men who have been working at this occupation for from three to ten years.

Caisson Disease of Bone.—Rendich and Harrington reviewed the literature on caisson disease of bone and described 4 cases from their service. *The primary lesion is an accumulation of nitrogen in the bone, resulting in massive aseptic necrosis.* Involvement of the articular cortex leads to the slow development of a deforming arthritis, with or without osteocartilaginous loose bodies. Characteristic findings include medullary calcification and disturbance of the lamellar structure of the ends of the larger long bones.[100]

Caisson disease may be a possible cause of bone and joint pains, particularly in the lower extremities (mainly in the knees). The joint affection is *usually*

monarticular in caisson disease. The aseptic necrosis results from interference with nutrition from embolism in a main nutrient vessel or from pressure on the vessel wall due to bubbles. There results absorption, collapse and new bone formation in or near the epiphysis. *Roentgen examination in cases of caisson disease* will show a multiple distribution of infarcts in the medullary bone while cortical changes are rare.

ACUTE BONE ATROPHY

Acute bone atrophy may develop within several weeks at the site of an injury or inflammation or at a distance from it; this is shown in the roentgen-ray as a characteristic patchy atrophy of bone.[31,110] Rapid muscular weakness and wasting accompanies the process. Shrinkage of the joint capsule with resulting ankylosis may occur. Accompanying vasomotor and trophic disturbances include cold, clammy skin, cyanosis, desquamation, atrophy of the skin and subcutaneous tissues and delayed healing of wounds.

Acute bone atrophy has been observed following various acute and chronic diseases of bones, joints and tendons; occasionally this atrophy may persist following healing of the original lesion.

Rieder found histologic signs of beginning bony absorption in clinical cases and animal experiments before these were revealed by roentgenogram.[101] Pronounced hyperemia of the blood-vessels of the bone was observed at this stage so that diminished blood supply does not play a rôle in the causation of acute bone atrophy. The bony structure was not affected by ligation of the nutritive arteries of the bone and of the arterial blood supply to the extremity. The dominant rôle of the vasomotor system in massive atrophy of an extremity (bones, skin, subcutaneous tissues, tendons, synovial membranes) is suggested by the abundant sympathetic nerve fiber supply of the bony framework.

Sudeck's atrophy, also called Leriche's disease is a not uncommon condition in which after acute trauma to a small joint (as an ankle or a wrist), the bones in the adjacent foot or hand may become rarefied and marked regressive changes may appear in the overlying soft tissues—such as swelling of the soft tissues, vasomotor and trophic changes in the skin and muscular atrophy. The underlying pathology is unknown.[51]

Jaffee reported a *similar series of events following trauma without fracture to large joints.* In his cases, a blow to the knee or a violent wrench led to severe disabling atrophy of the local muscles, occasionally with the formation of one or more small areas of rarefaction in the spongiosa near the joint. In a few instances, more extensive rarefaction of the bone occurred extending far along the bone or bones which helped form the joint. The rarefaction involved both the cortex and the spongiosa. On biopsy, this rarefaction was found to be due to hypervascularization of the affected tissues, particularly of the cortical bone. Recovery was aided by physical therapy and *use* of the affected parts.

Injury may also interrupt the circulation to joints. Fractures in the neck of the femur, condyles of the humerus or carpal navicular bone may cause extensive severance of connections resulting in aseptic necrosis of the bone of the articular fragment. Because of displacement and interference with callus formation from the articular fragment, there is a predisposition to non-union.

Treatment of acute bone atrophy includes removal of the original lesion and improvement in the circulation (as by contrast douches, massage, heat or elevation and lowering of the limb). If conservative measures prove of no avail, ramisectomy or sympathectomy must be considered.

STENOSING TENDOVAGINITIS AT THE RADIAL STYLOID

About 200 cases of this condition have been reported since its description by deQuervain in 1895. The *etiology* is thought to be *excessive use of the thumb* (as in writing, chopping wood and washing). Pathologically there is found a fibrosis of the common tendon sheath of the abductor longus pollicis and the extensor pollicis brevis at the lower end of the lateral aspect of the radius. Roentgen examinations are usually negative. *Symptoms* include pain in the wrist, especially at the styloid process, pain on motion of the wrist or thumb, and local swelling and tenderness.[114] *Treatment* usually consists of incision of the tendon sheath under local anesthesia.

Osteochondritis Dissecans.—Osteochondritis dissecans was recognized as early as 1738; the term was coined in 1905 by Konig, to denote a disease of obscure origin characterized by the demarcation and detachment of one or more fragments from articular cartilage and underlying bone of a joint surface. Pathologically, one sees an aseptic necrosis of bone, frequently preceded by trauma.[87]

The knee joint is affected in more than 85 per cent of cases, with the elbow joint next in frequency.[66] The hip joint is involved occasionally as are the metatarsophalangeal or ankle joints and rarely a terminal phalangeal epiphysis.[108]

The possibility of osteochondritis dissecans tali should be considered in a patient with chronic pain or discomfort in the ankle, following a sprain, dislocation or fracture—persisting despite the usual conservative therapy. A loose fragment or joint mouse may be suggested by a history of recurrent instability of the ankle.

Only 20 cases of osteochondritis dissecans of the ankle have been reported. Arthrotomy and removal of the loose fragments is the treatment of choice, followed by proper physical therapy (diathermy, massage and graded exercises).

Osteochondritis juvenilis of the acetabulum may affect the primary or secondary centers of ossification. It is most likely to produce change at the superior surface or rim of the acetabulum.[80]

Over 700 articles have been written on osteochondritis of the centers of ossification, although involvement of the acetabulum alone is reported infrequently.

Treatment is conservative and is directed toward relief of the strain resulting from weight bearing on the involved areas.[42,79]

Epiphysitis of the femoral head was first described as a clinical entity in 1909 by Legg. Levy and Girard studied 102 cases of this syndrome, of which 91 per cent were in males. In 10 per cent, the disease was bilateral. The age of the patients when first seen varied from three to thirteen years, averaging 8.6 years. Most of these individuals were seen early after the onset of the disease.

With proper treatment, the average case of Legg-Perthes disease ran a course of about two-and-a-half years; this might be prolonged to as much as four years with improper therapy.

The most effective method of treatment in these cases was elevation of the shoe on the well leg and having the patient walk with crutches without weight bearing on the involved leg. The patient must not walk or kneel on the involved leg. If an adduction contracture or considerable muscle spasm is present, preliminary traction is applied for several weeks until muscle relaxation and abduction are obtained. The hip may then be immobilized in abduction in a spica for three or four months, following which the patient may be allowed up on crutches with an elevation under the well leg and no weight bearing on the involved hip. Preliminary traction and cast treatment is necessary in only a few of these individuals. The results obtained by this form of therapy were as good as those following prolonged rest in bed and traction.

Complete rest in bed is necessary if there is bilateral hip involvement.[87]

Semimembranosus Bursitis (Baker's Cyst).—Significant *popliteal cysts* consist of enlargement of the semimembranous bursa; this has been called **Baker's cyst.** This enlargement may occur at any age. It may result in part from trauma in association with a torn internal meniscus of the knee joint or may be associated with rheumatoid arthritis or degenerative joint disease.[15]

This bursa is located under the deep fascia of the popliteal space between the medial head of the gastrocnemius and the semimembranosus muscle; it is attached to the posterior capsule of the knee joint and its bordering muscles and may communicate with the knee joint by a small opening.

The condition is not common. The swelling may fluctuate in size due to the passage of bursal fluid into the joint and *vice versa*, or possibly from variations in the secretory or absorptive powers of the wall of the bursa. The bursa may become obliterated by fibrosis.

Meyerding and VanDemark reported 15 cases of posterior hernia of the knee (semimembranosus bursitis) which produced firm fixed masses in the popliteal space, sometimes limiting flexion of the knee.[90] In each case, these popliteal cysts were found to be connected directly with the knee joint through an opening in the posterior part of the capsule. They believed that these cysts resulted from herniation of the synovial membrane through the posterior part of the capsule or from the escape of fluid from the normal anatomic connections of the knee joint into the semimembranosus or medial gastrocnemius bursæ.

Baker's name has been associated with these cysts since his article[2,3] in 1877 on synovial cysts of the knee. Posterior hernia of the knee joint was described in 1845 by Gruber.[44] Cravener pointed out the value of the injection of air into the knee joint in making the preoperative diagnosis.[26] Haggart (1938)[45] reported several cases of posterior hernia of the knee and Wilson and his associates described popliteal bursæ connected with the joint.[117]

The capsule of the knee joint is weak along the lateral side of the medial femoral condyle. All of the important bursæ of the popliteal space (semimembranosus, semitendinosus, gastrocnemius and popliteus) may communicate with the joint as well as with one another under certain circumstances. These communications with the knee joint are often open when the knee is extended and the posterior part of the capsule is tense but may close with flexion of the knee. *These cysts enlarge with motion* as the herniation and escape of synovial fluid increases with motion.

These hernial sacs may be from 8 to 10 cm. long and 5 to 7 cm. wide.[90] With enlargement, they may extend downward as far as the Achilles tendon. The hernial sac is usually attached to the joint by a pedicle which may become thick and firm and may be adherent to tendons, especially the gastrocnemius. The sacs may be multilocular and contain mucilaginous yellowish fluid. Occasionally the cyst is ruptured by injury.

Pronounced disability is uncommon from these posterior herniæ. The patient may complain of aching and stiffness in the knee and the presence of the swelling may be noted. *The cyst is usually not painful* or adherent to the overlying skin. *It may appear soft on flexion and firm on extension of the knee.* The patient can use the leg and walk although flexion may be somewhat limited. The majority of these cysts are unilateral although a few patients have had bilateral swellings.[58]

Transillumination of the area reveals the cystic character of the lesion; the fluid contents can be aspirated. If an *x*-ray is made after the injection of from 100 to 150 cc. of air into the knee joint after aspiration of synovial fluid, an air bubble will usually be seen in the hernial sac.[97] Occasionally, the reverse may be noted, *i. e.*, aspiration of fluid from the cyst, followed by injection of air into the sac may lead to the finding by *x*-ray of air in the knee joint. In most cases it will be unnecessary to inject air as a diagnostic measure; the possible dangers of infection and embolism should discourage this procedure unless absolutely essential.

Other swellings which may occur in the popliteal space include aneurysms, varicosities, benign and malignant tumors, enlarged lymph nodes, infected swellings (pyogenic or tuberculous) and Charcot's joint. Benign tumors which may occur in this area include lipomas and xanthomata; the former are usually soft, do not transilluminate light and yield no fluid on attempted aspiration. Xanthomata are firm; they also will not transilluminate light and cannot be aspirated.

The fibrosarcoma is the commonest malignant tumor in this region. This is firm and is more fixed to the deep structures; it will not trans-

illuminate light. In some instances, frozen sections may be necessary for diagnosis.

Aneurysms are rare in this region and are expansile and pulsating; arteriovenous fistulas are equally rare. It is usually easy to distinguish varicose veins in this region from popliteal cysts. Hemangiomata are not common in this area.

Popliteal lymph nodes subside when the nearby infection improves. Pyogenic abscesses are tender, hot and fluctuate and are associated with fever and inguinal adenopathy; aspiration reveals the pus. Tuberculous abscess may require aspiration with culture, or biopsy for diagnosis.

In most instances, if untreated, popliteal cysts progressively enlarge. *The treatment of choice is careful dissection and ligation of the pedicle;* recurrences are rare. Some patients improve temporarily with repeated aspiration, rest and bandaging.

PELLAGRINI-STIEDA SYNDROME

Pellagrini-Stieda's disease is the occurrence of an effusion of blood beneath the femoral periosteum at the insertion of the medial lateral ligament, when this has been torn. This hematoma may form quite rapidly and may calcify early (resembling at times an osteogenic sarcoma). Symptoms include stiffness and pain above the knee on the mesial aspect. The clot will be absorbed promptly with adequate rest and heat.

There may appear to be a formation of bone in the medial collateral tibial ligament of the knee following trauma.[55] Diagnosis is made by a history of trauma, swelling of the knee, pain on motion and roentgen-ray findings.

GANGLION

Trauma is a questionable factor in the formation of a *ganglion.*[98] *A ganglion is a cystic swelling containing thick mucoid material occurring usually along tendon sheaths or joint capsules.* It is most common in the hands or feet, and particularly on the dorsal surface of the wrist. The walls of such lesions are usually thin. They are more common in women than in men. Although the ganglion is attached to the joint capsule or tendon sheath, it does not communicate with the interior.[116] Symptoms are usually lacking although slight discomfort may be noted on motion.

Ganglia about joints and those involving tendon sheaths may develop after sprains or insidiously without any known trauma.[81]

Such lesions *may disappear spontaneously* in some instances; rupture of the ganglion from a blow (as from a heavy book), followed by pressure, has long been a household remedy for these. This does not usually produce a permanent cure.

Many ganglia which have been aspirated or injected with sclerosing solutions such as sodium morrhuate, tend to recur. The least number of recurrences in the past have followed surgical excision.

If resistant, or if connected with tendon sheaths, their careful *operative removal* has been advised. A ganglion may persist for ten years or more before spontaneous rupture occurs.

Lyle employed *roentgen therapy* in these patients with good results, using 1.5 erythema of radiation each month until results were obtained. If more than five treatments were required, the interval between treatments was then increased. In 81 per cent of 21 patients, the swelling disappeared; 78 per cent of those complaining of pain were relieved. Eighty-two per cent of those with weakness noticed return of normal function in the joint.

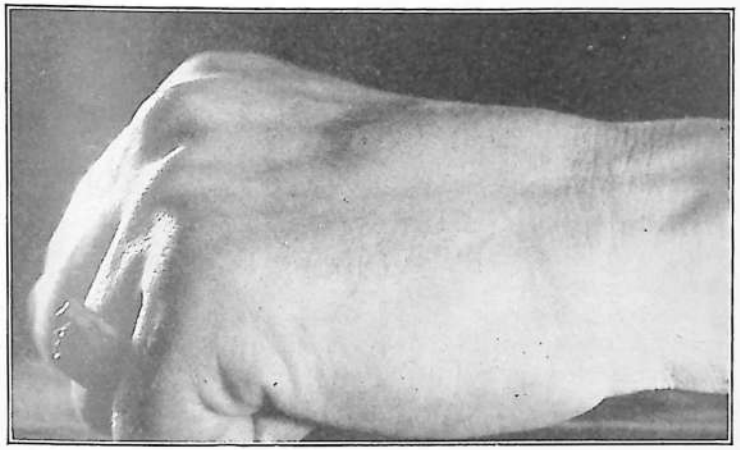

FIG. 219.—Ganglion arising from extensor tendon sheaths—oval and flattened cystic mass on dorsum of hand. (Brunschwig's Surgery, courtesy of C. V. Mosby Company.)

Bearse has advised against their excision.[6] His therapy consists in *puncture and evacuation* of the thick gelatinous contents *by a large bore needle;* in his hands, this is always effective. A tight bandage is applied for twenty-four hours following the puncture. The treatment may be repeated if the ganglion recurs. This obviates the necessity of an operative scar which otherwise might be present.

The etiology of ganglia is not yet clear. They may result from a low grade inflammatory process developing over the periosteum of the small bones about the wrist or within the joint capsule. Their only relationship to the sheaths of tendons is their proximity. They have been incorrectly termed cystic tumors of (or in) tendon sheaths. This misconception concerning their etiologic relationship to tendon sheaths is responsible for the fact that incomplete removal is common and is usually followed by recurrence.

Theories concerning their etiology include: herniation of the synovial membrane through a tear in the tendon sheath; cystic degeneration of connective tissue adjacent to a joint or tendon or colloid degeneration of a synovial fringe within a tendon sheath. They are found most frequently

in individuals who use the wrists and fingers to a marked degree such as in typists, pianists and washerwomen.

PERITENDINITIS CREPITANS

There is a palpable and often an audible crackling crepitus in some cases of acute or chronic traumatic tenosynovitis.[57] This may produce heat, redness, swelling and pain in the affected region of the arm or leg. Rest and immobilization for a few days should be prescribed.

INJURIES TO MUSCLES AND TENDONS

Haldeman and Soto-Hall discussed certain features of injuries to muscles and tendons. *Features* which they found *very suggestive of rupture of muscles* included:

1. A history of a *sudden sharp pain* or snapping sensation occurring *during violent muscular effort.*
2. *Inability to perform certain definite movements* following this.
3. The diagnosis is more certain if one sees the appearance of a *defect in the belly of the muscle* or in the tendon with subsequent ecchymosis.
4. *Roentgenogram of soft tissues* often reveals a *defect* in the shadow cast by the muscle; in some cases, a small chip of bone is seen attached to a tendon which has been torn from its insertion.
5. Electrical stimulation with the faradic current if applied to the muscle will cause it to contract and produce pain at the site of a tear in the muscle or tendon.
6. Local injection of procaine will eliminate pain and will permit one to test muscular function in those cases in which the diagnosis is difficult.

The supraspinatus muscle or tendon is the most likely to be torn in the upper extremity and the quadriceps is the most vulnerable in the lower extremity.[46] The average age of patients with tears of muscles or tendons was forty-two years, corresponding to the period in which the greatest activity is associated with degenerative changes.

The most *common etiologic factor* is acute *trauma.* Sudden application of a stretching force to a muscle that is contracting strongly is the most common mechanism producing injury to a muscle or tendon. Spontaneous rupture is relatively rare.

In 1922, Grassheim reported that in 500 cases of injury to muscles and tendons, the rupture occurred most frequently in the muscles of the calf, extensors of the leg, biceps, Achilles tendon and extensor of the thumb.[43] In recent years, other frequent sites of rupture of muscles have been the supraspinatus, triceps, rectus abdominis, adductors of the thigh and extensors of the fingers.[46]

McMaster's experiments on the breaking weight and site of rupture of a preparation of muscle and tendon showed that normal tendon does not rupture but that *the rupture occurs at the attachment of tendon to bone, the musculotendinous junction or through the muscle substance.*[85]

Codman has emphasized the importance and frequency of partial or

complete **tears of the supraspinatus tendon.**[20] Some individuals believe that Codman confused certain anatomic changes in the shoulder that are the result of senile wear and tear with purely traumatic lesions. Meyer described attritional changes in the shoulder with longitudinal fibrillations or fraying of the supraspinatus tendon.[89] Supraspinatus tears are rather infrequent clinically.[105]

Injury to the supraspinatus tendon is suggested by:[46]

1. A history of *fall on the shoulder or* on the *outstretched arm.*
2. Immediate and *complete loss of power to initiate abduction* of the arm as contrasted with the gradual onset of limitation of abduction which may occur in subdeltoid bursitis or an arthritis.
3. *Atrophy of the supraspinatus muscle,* varying in extent with the time which has elapsed since the injury.
4. When the patient attempts to raise the arm, he may lean to the affected side to permit the initiation of abduction by the weight of the arm; once the arm has been partially abducted, the deltoid muscle can complete this movement.
5. In some cases there is a *point of tenderness* just beyond the tip of the acromion over the roof of the shoulder if the supraspinatus tendon is torn.
6. *X*-rays of the shoulder may show calcification in the subdeltoid bursa, fracture or arthritis—which may be useful in the differential diagnosis.
7. Injection of from 10 to 15 cc. of 1 per cent procaine hydrochloride into the subdeltoid bursa will permit abduction if this is limited by reflex inhibition of function of the supraspinatus resulting from subdeltoid bursitis or adhesions; if active abduction is not improved following this procedure, a diagnosis of tear of the supraspinatus tendon is more likely.[46]

Partial tears of the supraspinatus tendon should be treated by the use of an abduction splint for four to six weeks, followed by physical therapy. *Complete tears should be repaired surgically* as soon as diagnosed.

Rupture of the long head of the biceps has been discussed by numerous authors.[40,41,46,74,104,109] The biceps tendon becomes frayed out (with advancing years) in the bicipital groove and over the head of the humerus where it is subjected to constant friction.

Rupture of the long head of the biceps is followed by *marked weakness of the flexor muscles of the elbow* and is often accompanied by *pain over the anterior aspect of the shoulder joint* or over the bicipital groove. The belly of the muscle assumes a spherical shape as a result of an abnormal depression at its upper lateral border (in marked contrast to the oval contour of the normal contracted biceps).[46] In recent tears, one should remember that the hematoma produced by a tear in the upper part of the tendon may gravitate downward through the sheath and present in the belly of the muscle. The treatment of a ruptured tendon is immediate repair.

At times, the long head of the biceps may tend to slip medially out of the bicipital groove on external rotation of the humerus; this produces a painful snap which can be felt and heard (over the occipital groove).

Weakness of the arm and localized tenderness result from permanent dislocation of the tendon out of its groove. The crepitations in some cases of subdeltoid bursitis may be confused with a *slipping (dislocation) of the biceps tendon;* in the former, the snap will disappear following procaine injection into the subdeltoid bursa. Surgical intervention is required for recurrent or permanent slipping of the biceps tendon.

The *rhomboideus major or minor muscles* may be strained by a sudden incoördinated movement of the shoulder; this occurs frequently in industrial practice. These muscles arise from the ligamentum nuchæ and the spinous process of the seventh cervical to the fifth dorsal vertebræ and insert into the vertebral border of the scapula. Contraction of these muscles draws this border of the scapula upward.

Following injury to the rhomboid muscles there is a localized tenderness between the mid-dorsal spine and the scapula; pain occurs at this point if the shoulder is passively flexed forward markedly or if it is extended against resistance. Treatment consists of partial immobilization of the scapula by drawing it backward and upward with adhesive. Physical therapy (baking and light massage) should be begun when the signs of injury have disappeared.

Other muscles arising from the spinous processes and inserting on the scapula or humerus include the *latissimus dorsi* and *trapezius muscles;* strains of these muscles give a clinical picture which is similar to injury to the rhomboids; therapy is the same.

Rupture of the rectus abdominis muscle may at times be mistaken for intraperitoneal disease (such as appendicitis). Rupture of this muscle may occur following a severe bout of coughing or sneezing, during pregnancy or labor or following influenza or typhoid fever (with degeneration of the muscle fibers). A large hematoma may form due to tears of branches of the epigastric vessels. The hematoma may require evacuation surgically.

Rupture of the quadriceps muscle or tendon is very common. This occurs at the point of attachment of the tendon to the patella or at the musculotendinous junction; occasionally, a tear may occur through the purely tendinous or muscular portions.

The *usual cause* of this condition is a *direct blow,* such as falling on a flexed knee. The injury occurs in both young and aged. It is diagnosed by the history and by the inability of the individual to actively extend the knee, in addition to the hiatus which is seen in the tendon or muscle. The gap in the muscle may be seen by a lateral soft-tissue *x*-ray; the superior border of the patella usually tilts anteriorly. Partial tears of the muscle may leave only a depression in the muscle substance without permanent disability. These usually heal readily following splinting of the leg in full extension for three or four weeks, with gradual mobilization following this. Complete tears of the quadriceps muscle or tendon should be repaired surgically.

Strong contraction of the quadriceps muscle may pull the tubercle from the front of the tibia (*Osgood-Schlatter's disease* or *Schlatter's sprain*).

Roentgenogram is necessary for diagnosis as local effusion in this region interferes with palpation.[106]

When the *patellar tendon is ruptured,* the patella is higher than usual and joint effusion is present. A roentgenogram will confirm the diagnosis. The patella sags downward following rupture of the quadriceps tendon above the patella; there is loss of the power of leg extension and a dimple may be seen above or below the edge of the patella.

Partial *rupture of one of the calf muscles* has been termed *"tennis leg;"* this usually involves the plantaris muscle, but may affect a belly of the gastrocnemius. Symptoms include a *snap* (audible or palpable) *with sudden burning pain in the calf* during a strong muscular contraction. The pain may extend to the popliteal space; it is aggravated by passive dorsiflexion of the ankle. Treatment includes adhesive strapping to immobilize the ankle in plantar flexion for several weeks, following which baking, massage and exercises are used.

Violent exercise (as in boxers, sprinters, etc.) may result in *injury to the Achilles tendon* which is usually ruptured at the musculotendinous junction. In complete tears, there is a marked depression (from separation of the fibers of the tendon); the foot cannot be flexed in the plantar direction actively against resistance. Partial rupture may produce tenderness and ecchymosis over a depression in the tendon. In the latter, treatment consists of adhesive strapping or a cast to immobilize the foot in plantar flexion. Complete tears require surgical intervention.

McMaster reviewed the literature and found 27 instances of late *rupture of the extensor pollicis longus* following Colles' fracture.[84] From one to three months may elapse before its appearance. The lesion is caused by chronic trauma from the irregularity of the bone over which the tendon moves.

The so-called **"baseball finger"**—an extremely common and troublesome injury—is caused by rupture or detachment of the extensor tendon at its insertion. Immediate hyperextension of the terminal joint for at least six weeks will often avoid deformity; later operative repair is frequently unsuccessful.

Herniation of a muscle occurs occasionally through its sheath. This results most often from a direct blow. It may be repaired with fascial strips.

Injections of procaine may relieve the pain of muscular injury, fibrositis or acute tendinitis.[32,73]

Bennett discussed the **"sore arm" of the professional baseball pitcher,** dividing the shoulder lesions into an anterior and a posterior group.[8] The *anterior group* included traumatic irritation, fraying of the supraspinatus and biceps tendons associated with constant rotation of the humerus, similar traumatic irritation of the subdeltoid and subacromial bursæ, irritation of the coracoid bursa by the head of the humerus in the "follow-through" and inflammatory processes in the various bursæ in this region. Conservative treatment is usually effective in these; this includes rest, local heat, and removal of any evident focal infection and improvement of the general health.

The characteristic *posterior lesion* is an exostosis which may develop at the postero-inferior border of the glenoid fossa, probably due to repeated strong pull of the triceps and posterior capsule. This lesion may seriously limit the career of the pitcher. This deposit irritates the capsule and its synovial lining and produces local pain and tenderness and irritation of the circumflex nerve, with resulting referred pain in the deltoid region. Symptoms appear only under the strain of "hard pitching." One cannot assure these individuals of rehabilitation of the shoulder so that it can withstand this strain.

Elbow lesions which may occur *in the pitcher* include loose bodies in the olecranon and coronoid fossæ and other osteochondritic changes. These produce few symptoms other than interference with complete extension of the elbow. Surgery is usually successful so that the pitcher's career is not interfered with.

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CHAPTER LII

INTERNAL DERANGEMENT OF THE KNEE JOINT

Internal derangement of the knee joint may be defined as interference with the normal mechanical function of the joint due to some constituent being out of place, or to the presence of an abnormal or foreign product within the joint.

The knee joint is the largest articulation in the body. Its strength results from the powerful ligaments, muscles and fascia surrounding it. Inasmuch as the articular surfaces are merely placed in contact with each other, their shape gives no strength to the joint. In spite of this, dislocations of the knee are uncommon. However, mechanical derangements are commonly encountered in the knee joint.

Symptoms of Internal Derangement of the Knee Joint[1]

Symptoms of internal derangement of the knee joint are similar regardless of the cause.
Sudden mechanical interference with joint function in an otherwise normal joint (such as locking of the joint or a sense of giving way of the joint).
Sudden appearance of symptoms following twist of the knee or trauma or unusual position, followed by distention of the joint with fluid.
Local pain, swelling and tenderness due to a resulting synovitis.
Variation of the site of pain and swelling depending upon the causative lesion.
Recurrences with "trick knee" not infrequent.

Internal derangement of the knee joint results from lesions of the semilunar cartilages; injury to the cruciate and lateral ligaments; loose bodies; traumatic lesions of the synovial membrane and infrapatellar fat pad; injury of the tibial spine; and exostoses.[11,16,20] The **causes of internal derangement of the knee joint** have been classified as follows:[1,3]

Semilunar cartilages (injuries or displacement).
Injuries to lateral ligaments (internal or external).
Tears and dislocations (anterior, posterior, bucket handle dislocation).
Cysts (internal or external semilunar cartilages).
Congenital abnormalities (lateral meniscogenic snapping knee).
Calcification or ossification of semilunar cartilage.
Hypermobility of cartilages.
Cruciate ligaments.
Stretching or tearing of the anterior or posterior ligaments.
Loose bodies.
Synovial origin (chronic synovitis, rheumatoid arthritis, osteochondromatosis).

Articular origin (osteochondritis dissecans; osteochondral fractures; detached osteophytes—of degenerative joint disease).

Soft tissue.

Adhesions.

Semilunar fat pads (hypertrophic fringes).

Synovial membrane—traumatic lesions.

Recurrent dislocation of the patella (slipping patella).

Fractures of the spine of the tibia.

Exostoses.

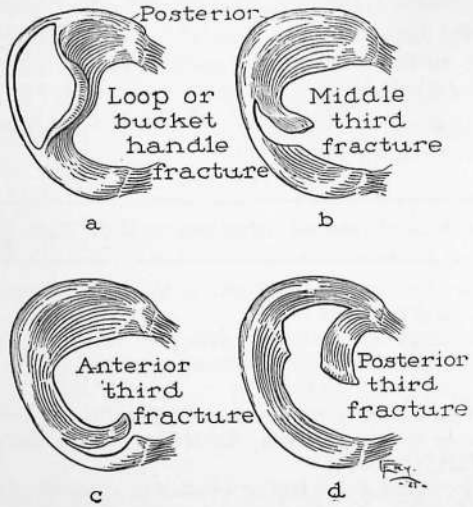

FIG. 22).—Types of injury to the semilunar cartilage. (Henderson, courtesy of Surg., Gynec. and Obst.)

SEMILUNAR CARTILAGES

The semilunar cartilages are attached to the tibia and to the internal and external lateral ligaments.[28] The external of these is smaller, more round and more movable than the internal, and is less likely to be "nipped" between the bones.[41] The fibrous semilunar cartilages are readily torn.

In 300 instances of knee joint injury, Speed reported that the internal semilunar cartilage was damaged in 153 cases, while the external semilunar cartilage was involved only 25 times.[38]

Semilunar cartilage damage is extremely variable from simple loosening, or nicking to actual splitting or being completely torn off. It is very uncommon to have both cartilages affected at the same time. Pinched fat tags may produce symptoms (severe pain and locking of the joint) resembling a slipped semilunar cartilage. This bruised swollen mass of fat is seen at operation with blood-tinged fluid present in the joint. The latter is common in skiing accidents.[38]

Cracking off of the tibial spines (particularly the medial) may be confused with rupture of the crucial ligaments; the diagnosis will be evident

by roentgen examination. The loose pieces must be fixed to the tibia by absorbable sutures.

Injuries or displacements of the semilunar cartilages are the *commonest cause of internal derangement of the knee joint.*[33,37] These are most frequent in young adults especially active in athletics. In 587 operations in cases of derangement of the knee joint, Henderson performed 343 for lesions of the semilunar cartilages (90 per cent of these being of the internal semilunar cartilage), and 244 operations for removal of loose bodies (osteocartilaginous).[24]

Injuries to the semilunar cartilage occur most often between the twenty-fifth and thirty-fifth year. They usually result from intolerable torsion strains.

The usual cause of injury to the internal cartilage is sudden internal rotation of the femur upon a fixed tibia with the knee flexed and abducted; in damage to the external cartilage, the femur is rotated outward on the tibia with the knee flexed and adducted, and the foot firm on the ground. The type of injury causing damage to the internal cartilage is much more common than that affecting the external. The latter is not attached to the lateral ligament and is more secure as it fits the anatomic contour of the joint more closely.

Symptoms of Tears or Dislocations of Semilunar Cartilage

Locking of the knee joint, in from 30 to 75 per cent of the cases, producing immediate disability, disappearing with a snap on manipulation; this is pathognomonic (in the presence of negative roentgen findings).[14] History of an injury (as in football, in miners, etc.) preceding locking of the joint.

Sudden pain in the knee and leg, with *local tenderness* and *swelling.*

Local site of tenderness: if anterior and mesial between the joint surfaces, anterior horn tears of the internal cartilage are suspected; if posterior and mesial, posterior horn tears of the internal cartilage; if externally, the external semilunar cartilage.

Development of synovitis subsequent to the injury.

Recurrences are common with progressive atrophy of the muscles of the leg and thigh in chronic cases.

Roentgen-ray findings are usually negative in acute cases; later one may see narrowing of the joint space on the affected side, and, in some cases, the development of degenerative joint disease. One must always rule out fracture, arthritis, loose bodies, exostoses and osteochondritis dissecans. *Air arthrography* has been used diagnostically in lesions of semilunar cartilages.[15]

Treatment of Lesions of the Semilunar Cartilage.—In acute cases, manipulation with or without anesthesia is advisable; some of these patients may not experience recurrences. Attempt to reduce the lesion immediately, before much swelling has occurred.

If recurrences follow, the cartilage must be removed following subsidence of the acute attack. Derangement of the knee joint requiring operation is rare in young children.[18]

Ninety per cent of cases show marked improvement following operation; 55 per cent of "cures" can be expected with non-operative methods. In general, the younger the patient, the quicker is the recovery.

Reduction of tear of a semilunar cartilage: With the patient lying on a low firm table with his knees hanging over the end, the physician stands between the patient's legs and flexes the involved knee as far as possible.[18] One hand grasps the ankle firmly while the thumb of the other exerts pressure upon the anterior end of the involved cartilage. With the patient's knowledge, forcible extension of the knee and inward rotation of the tibia is performed in internal tears, and extension of the knee and outward rotation in external tears. If the displaced cartilage is released by this maneuver, a snap may be felt, the leg may be extended fully and the knee feels normal to the patient. A posterior molded splint is then worn until pain and swelling have subsided. Shands advises immobilization of the joint for at least three weeks by rolls of cotton, thin board splints and an elastic bandage.[36] In order to minimize strain in walking, he builds up the shoe $\frac{1}{4}$ inch on the inner side in internal cartilaginous displacements, and on the outer side for external lesions. In addition, exercises of the quadriceps muscle and massage are used to maintain muscle tone. Absorption of joint fluid may be hastened by the use of infra-red heat or diathermy.

If repeated manipulation does not reduce the lesion, the patient is kept in bed and is made comfortable with the leg as straight as possible. Heat is applied and the relaxation following this may result in spontaneous replacement of the cartilage.

Physical examination does not reveal the exact location of the rent in the cartilage. If the lesion is situated near the attachment of the lateral ligaments in the periphery, healing may occur because of the presence of blood-vessels and fibrous tissue. Tears in the body of the semilunar cartilage will not heal. If one is not dealing with a permanent locking or an irreducible cartilage, immobilization in plaster splints should be carried out for four weeks to permit healing. If further attacks occur, if symptoms persist after immobilization, or if the displaced cartilage is palpable, operation is advisable.[32,40] If any portion of the cartilage is found to be loosened or torn, the cartilage should be removed as completely as possible. In most cases, individuals under twenty-five years of age can return to active athletics two months following operation. It has been stated recently that "There is no more danger in opening a knee joint than in opening an abdomen."[24] The older concept that opening a knee joint resulted in ankylosis or stiffness is no longer valid.

Menisci of the knee can be demonstrated in 70 per cent of normal individuals by *x*-rays made with the knee in forced adduction; this method is not applicable to patients with joint effusions.[17]

SEMILUNAR CARTILAGE CYSTS

These are found in approximately 3 per cent of cases of internal derangement of the knee joint which come to operation.[9,46] The commonest site of such cysts is anterior to the external lateral ligament of the knee joint in the middle of the external semilunar cartilage, on the outer aspect of the knee. These cysts are often multiple and contain a gelatinous material.

Such cysts produce pain on walking, and especially in climbing or descending stairs. A hard, slightly sensitive, cystic mass, moving with the tibia, may be felt anterior to the external lateral ligament at the joint line. It is felt most readily with the knee in extension. Most surgeons prefer to excise the cysts with the cartilage in these cases.

Ghormley and Dockerty have described cystic myxomatous tumors about the knee and have discussed their relation to cysts of the menisci.[22]

Other Lesions of the Semilunar Cartilages.—These include calcification, ossification (rare) and congenital variations in their size, shape and attachments.

The literature contains only 78 reported cases of calcification of the menisci of the knee joint. The calcium is usually laid down in parallel layers beginning at the periphery of the cartilage and involving the entire length. The condition often produces no symptoms and may be discovered by accident or in others, symptoms may be severe enough to necessitate removal of the cartilage. One may see pain, swelling and limitation of motion.

The calcification may be primary (non-traumatic) or secondary (following trauma). In the primary cases, degenerative joint disease of the knees is usually present.[43]

INJURIES TO THE LATERAL LIGAMENTS

The *lateral ligaments*, which are relatively feeble, are relaxed in flexion and tense in extension. *The internal ligament is more frequently strained than the external.*

Complete tears are uncommon and result from severe trauma; partial tears are frequent (more often affecting the femoral attachment than the tibial).

Symptoms are those of internal derangement of the knee joint, with pain, swelling and tenderness in the neighborhood of the affected ligament. There is also an increased lateral mobility of the knee.

Treatment.—Immobilization in plaster in complete extension for six weeks if complete tear occurs; in partial tears, immobilization for at least three weeks.[10] The splints may be removed daily for physical therapy; in severe injuries, motion of the joint is forbidden.

When the patient becomes ambulatory, apply a knee brace to prevent hyperextension and excessive lateral motion and raise the heel on the affected side $\frac{1}{4}$ inch to prevent ligamentous strain.[17]

Badgeley[1] advises surgical repair in the case of complete tears; reconstruction may be accomplished with strips of fascia lata.

CRUCIATE LIGAMENT INJURY

The very powerful *crucial ligaments* are more or less tense in any extreme position of the joint.[25] One or both cruciate ligaments may be torn; rupture occurs only following severe trauma.

Severe injuries of the knee joint often produce tear of a semilunar cartilage and of a lateral ligament with rupture of the anterior cruciate ligament.

Symptoms.—1. Instability and marked swelling of the knee joint.

2. Hyperextension and increased anterior displacement of the tibia (in the case of a torn anterior cruciate ligament).[30]

3. Increased displacement posteriorly (if the posterior cruciate ligament is torn).

Treatment.—Tears of both ligaments: plaster cast immobilization for two or three months (knee kept at about 15 degrees flexion).

If a stable knee is not produced, one may attempt to reconstruct the cruciate ligaments with fascia lata.[6]

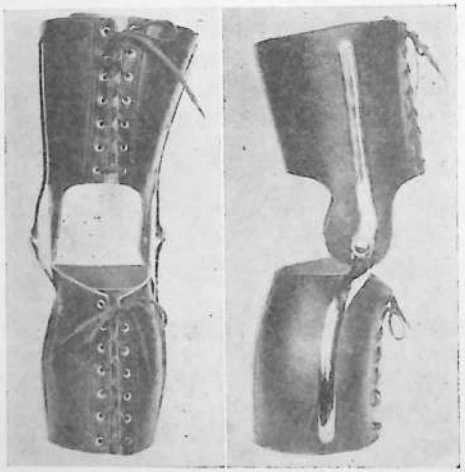

FIG. 221.—Jones knee cage for after-care in knee injuries. (Bennett, courtesy of Am. Jour. Surg.)

The *Jones knee cage* has been used with success in the treatment of chronic relaxations of the crucial and lateral ligaments, with pseudo-locking of the knee and excessive rotation and slipping of the tibia upon the femur.

LOOSE BODIES WITHIN THE KNEE JOINT

In a series of major injuries to the knee joint seen in hospital practice, the following incidence of pathologic conditions was found:[1] Semilunar cartilage injury, 33 per cent; loose intra-articular bodies, 27 per cent; tears of lateral ligaments, 18 per cent; recurrent patellar dislocations, 15 per cent; cruciate ligament tears, 4 per cent; semilunar cartilage cysts, 3 per cent; and ossified semilunar cartilage, 0.3 per cent.

Loose bodies are derivatives of synovial membrane or portions of articular surfaces or menisci.[23] Cartilaginous nodules may develop from synovial fringes and may become detached from these. They are a frequent cause of internal derangement of the knee joint.

Loose bodies may be found in chronic synovial reaction (as in degenerative joint disease, tabes dorsalis, tuberculosis or purulent arthritis); detached articular ecchondrosis; detached intra-articular epiphyses; loose bodies of osteochondritis dissecans, osteochondromatosis and synovial chondroma. They are most common in degenerative joint disease. They may be of fibrinous or cartilaginous origin.

FIG. 222.—**Loose bodies** in a knee joint.

Osteocartilaginous bodies in joints should always be removed in young individuals unless contraindicated. They may cause any degree of joint symptoms and disability. Loose bodies are differentiated from semilunar cartilage derangements chiefly by: (1) absence of history of recent injury; (2) short duration of the locking, which is mild in most cases; (3) very little or no joint effusion; (4) roentgen findings. Auscultation of joints may aid in localizing loose bodies and injury to semilunar cartilages.

The diagnosis may be suggested by:

1. A feeling of something slipping in the knee joint.

2. Palpation of the loose body.
3. Roentgen findings (including variability of location of the loose body).

OSTEOCHONDRITIS DISSECANS

This is a *partial or complete splitting off of articular cartilage with or without some of the subjacent bone* from the joint surface resulting in the formation of loose bodies (usually not more than two);[2,19,27,31] it is seen most often in adolescence or early adult life[5,21,42] although it has been reported in aged patients.[4] It occurs most frequently from the inferior surface of the internal condyle, proximal to the insertion of the posterior crucial ligament.[45] While it is most common in the knee or elbow, it has been described in the hip,[29] ankle[8] and metatarsal joints.[12] It is not a rare condition.

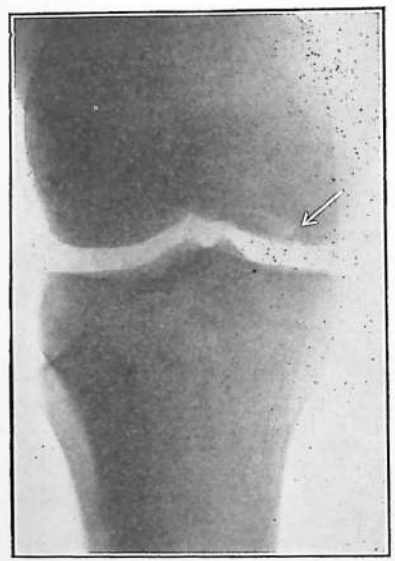

FIG. 223.—Osteochondritis dissecans. (Holmes and Ruggles.)

In this condition, there is an osteochondral separation of the intercondyloid part of the internal condyle of the femur. One may or may not obtain a history of injury.[31]

Symptoms, due to the formation of loose bodies, are usually of several years' duration and include local discomfort in the knee, slight locking or a sensation of catching of the knee. *Roentgen findings* are typical and consist of a hiatus on the mesial border of the internal condyle of the femur. Chips of cartilage and bone are broken off, resulting in loose bodies in the joint. This lesion is seen best in the antero-posterior film and may be overlooked in the lateral view. Roentgenologically this must be distinguished from the loose bodies seen after trauma or in synovial osteochondromatosis and from tuberculosis of the knee, torn medial meniscus, and from the punched-out areas of gout and chronic hemophilic arthritis. In the presence of

symptoms, *treatment* consists of surgical removal of the detached fragment with excision of the frayed edge of cartilage to prevent the formation of further loose cartilaginous bodies.

FIG. 224.—Multiple calcified bodies in the knee joint (osteochondromatosis). (Holmes and Ruggles.)

OSTEOCHONDROMATOSIS

This is the formation of large numbers (from 1 to 150) of osteocartilaginous bodies from synovial villi.[13,26] It is rare but occurs most often in young adults or early middle age. The knee joint may feel like a bag full of marbles.

Osteochondromatosis is relatively uncommon; multiple intra-articular cartilaginous bodies form and lie free or attached within the joint cavity. The etiology is unknown. *There is often an absence of symptoms*, which is in marked contrast to the findings in internal derangement of the joint. Joint fluid is seldom increased and no local heat or redness of joint tissues is noted.

Symptoms are usually those complained of in arthritic joints, with occasional sharp pains and some limp when the knee joint is involved. In some, the cartilaginous bodies may be palpated in the suprapatellar space or popliteal area.[44]

Surgical removal of the osteocartilaginous bodies is recommended to prevent further joint damage. If the synovial membrane is markedly involved, partial synovectomy is advised.

OSTEOCHONDRAL FRACTURES

These may occur in the patella, femoral condyles, etc., and require surgical removal if symptoms are produced.

RECURRENT DISLOCATION OF THE PATELLA

This may be complete or incomplete, the dislocation occurring laterally on active extension of the knee against force. When the lesion has been reduced, a tight compression bandage should be applied and the joint rested. Symptoms are as follows:

1. Most common in females.
2. Dislocation is usually outward.
3. May produce extreme pain and disability.
4. Patella returns to position in extension of the knee.

If recurrences occur despite the use of braces, surgical treatment is indicated, with transference of the patellar insertion inward.

FRACTURES OF THE TIBIAL SPINE

Fractures of the tibial spine are rare.[35] Avulsion of either tubercle or the whole spine may occur. The outstanding symptom is the presence of a bony block, causing locking. There is also pain, tenderness and marked swelling of the joint; the patient cannot extend the knee. *In most cases, the fragment must be excised.* In some cases with very little displacement of the fragment, one may immobilize the knee for four weeks, and provide support for the knee when the patient becomes ambulatory. The fat pad behind the patellar tendon is occasionally pinched on extension of the knee.[39] If this trauma is repeated, hemorrhage, organization and calcification may occur in the fatty tissue.

Injury to the infrapatellar fat pad results in tenderness, pain on extension of the knee, pain on climbing stairs, stiffness, weakness and occasional locking of the knee. Ossification has been reported occasionally in the infrapatellar[34] bursa and fat pad. Treatment consists of elevating the heel of the affected side from ½ to 1 inch, support to the knee to prevent full extension, massage and exercise of the surrounding muscles, and if necessary, surgical removal of the fat pad. Procaine injections may give marked symptomatic relief.

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CHAPTER LIII

THE PAINFUL SHOULDER

EXCLUDING acute traumatic lesions such as fractures, dislocations, contusions, sprains and ruptured tendons, 85 to 90 per cent of painful shoulders arise from calcareous tendinitis, acute and chronic bursitis (with adhesions), the scalenus syndrome and fibrositis.

It is not generally recognized that *a painful shoulder,* alone, *without evidence of joint phenomena elsewhere in the body, is seldom caused by arthritis. Probably 80 per cent of all cases of painful shoulder are due to* the lesion commonly known as *subdeltoid or subacromial bursitis.* This is often secondary to calcification in the short rotator tendons, particularly the supraspinatus.

Arthritis involving the shoulder joint alone causes not more than 5 per cent of all cases of painful shoulders. Of the 140 or more bursæ in the body (33 in each upper and 37 in each lower extremity), the subdeltoid is the one which most often gives symptoms.

Steindler classified the lesions about the shoulder joint into bursal lesions, primary injuries to tendons or the musculotendinous cuff overlying the bursa, sprains and tears of the shoulder joint capsule, acromioclavicular arthritis and brachial neuritis.[109]

The painful shoulder is common among baseball pitchers, orchestra conductors, violinists and those who use their arms vigorously; the terms "glass arm" and "musician's shoulder" are well-known in the baseball and musical professions.

In any pain about the shoulder joint, the physician should determine if this is traumatic, acute or chronic, and whether the main offender is bursa, muscle, tendon, nerve or joint.

Any patient with a painful shoulder should be seated on a stool, in a good light and stripped to the waist. A comparison is made anteriorly and posteriorly of both shoulders, noting any swelling, muscular atrophy or tenderness (over the head of the humerus, greater tuberosity or over the trapezius, pectoral or scapular muscles). One should palpate the scalenus muscles behind the sternomastoid at the base of the neck. The patient is then asked to slowly raise both arms laterally and to fully extend them above the head. Another useful test is to have the individual place his involved forearm horizontally behind his back (without assistance from the non-involved arm).

Full elevation of the arm includes rotation of the humerus upward on the scapula, and of the scapula on the chest wall; when the arm is fully elevated, the glenoid faces upward rather than outward.[65] The greater tuberosity of the humerus slides under the acromion during elevation of the arm, requiring a normal subdeltoid bursa for smooth, painless performance. If dense bursal adhesions or fusion of the shoulder joint are

present, the arm can be elevated almost horizontally due entirely to rotation of the scapula on the chest. If the physician stands on the side opposite the affected joint, he can control the motion of the scapula by clasping the hands below the axilla,[9] and can thus determine how much of the limitation of motion is due to lost movement of the humerus.

Approximate Frequency of Causes of a Painful Shoulder

Subdeltoid or subacromial bursitis (associated most often with *calcareous deposits,* particularly *in the supraspinatus tendon*)—at least 80 per cent of all cases.
Myofibrositis—8 per cent.
Arthritis of the shoulder joint—5 per cent.
Rupture of the supraspinatus tendon (common following industrial accidents to the shoulder).
Neuritis—a very infrequent cause of pain in the shoulder.
Other causes: *Cervical rib and scalenus anticus syndrome, arthritis of the cervical spine,* with narrowing of the intervertebral foramina (discogenetic disease), calcified cervical nodes, acromioclavicular separation, arthritis of the acromioclavicular joint, peripheral vascular disorders, cardiac disease (particularly following coronary occlusion), congenital deformities, apical pulmonary tumors, tuberculosis, syphilis (Charcot shoulder), bone tumors and metastatic carcinoma of bone (especially from carcinoma of the breast).

SUBDELTOID (SUBACROMIAL) BURSITIS

Numerous synonyms for this condition include: *periarthritis of the shoulder, subacromial bursitis,* painful shoulder, calcifications of the supraspinatus tendon, periarthritis humeroscapularis, calcified bursitis, bursitis calcarea subdeltoidea or subacromialis, Duplay's disease, etc.[35,74]

Codman, who has been the pioneer in this field, believes that bursitis like peritonitis, always has a cause and that many of the lesions producing painful symptoms in the shoulder are *due primarily to involvement of the supraspinatous tendon* with secondary lesions of the bursa.[25] The term subdeltoid bursitis has been more or less accepted in general practice, and usually is used to include a periarthritis of the shoulder, lesions of the musculotendinous cuff and calcification in the supraspinatus tendon. There are, however, several different lesions which may cause this syndrome.[80]

Causes of Subdeltoid Bursitis: 1. *Trauma* (overuse; prolonged hyperabduction; rupture of the supraspinatus, infraspinatus or long head of the biceps; acromioclavicular dislocation; fracture of the greater tuberosity; direct injury to the top of the anterior part of the shoulder with the arm in dorsal flexion; irritation from osteophytes).

2. Subdeltoid bursitis with adhesions occurs not infrequently in older patients who are confined to bed for long periods (as with coronary artery occlusion), or in association with diseases of the nervous system. The roughened bursal surfaces probably become adherent during this time (because of lack of motion).

Calcifications may occur in the injured areas of the tendon, with secondary bursal involvement.

3. The supraspinatus tendon is so situated that it is subject to frequent mild trauma, being pinched between the acromion and the head of the humerus.[32]

Calcification in the supraspinatus tendon has not been reported among children.

4. Foci of infection (?); these may play a rôle in some cases.

One occasionally sees a periarthritis of the shoulder which is aggravated by definite focal infection or which is relieved following removal of an infected focus.

Inflammation of the subdeltoid or subacromial bursæ usually results from lesions in contiguous tissues, such as the supraspinatus tendon, the floor of the bursa or the musculotendinous cuff composed of the tendons of the supraspinatus and infraspinatus and teres minor muscles.

Bosworth found *2.7 per cent of 6,061 unselected persons to have calcium deposits about the shoulder.* The majority of these developed slowly over a period of months.

Amorphous calcium deposits have been demonstrated frequently in the deltoid, trochanteric and radio-humeral locations.[26]

Kaplan reviewed the recent literature on calcareous deposits in tendons.[66] Calcifying tendinitis about the shoulder joint has its counter part in lesions about other joints, such as the elbows and knees. Calcareous tendinitis of the greater trochanter of the femur has been described.

It is probable that *acute or chronic trauma is the most likely factor involved.* The subdeltoid bursa may be injured by any fall on the hand or shoulder. Trauma may result from overuse of the shoulder, irritation due to occupational maneuvers (such as typewriting where the arms are held slightly abducted for long periods), or falls on the outstretched hand.[116] In elderly individuals, especially those who have done laboring work for many years, there is found a degeneration of the tendons of the short rotator muscles of the shoulder; under such conditions, mild trauma may produce injury or rupture of these tendons. In a series of 52 autopsies in an institution for elderly individuals, shoulder specimens revealed that some extent of rupture of the fibers of the supraspinatus tendon had occurred during the lives of 39 per cent of these patients.[3] Complete ruptures cause far more serious symptoms than partial ruptures and often lead to permanent disability.

"Subdeltoid bursitis" has occurred not infrequently in shoulders which have been exposed to drafts. Likewise, many cases are seen in the left shoulder, especially in female drivers; it is usually discovered that the left arm is held in a position of abduction, either leaning on the door sill or on a high arm rest. In such individuals, the window of the car has often been wide open. This may be mere coincidence rather than cause.

This condition has also been described frequently in patients with an upper respiratory infection, such as an acute cold, paranasal sinusitis and following the development of a periapical dental abscess. Some investigators believe that focal infection and "glandular dysfunction" are more important etiologic factors than trauma.[33] Voight has reported a large series of women with painful stiffening of the shoulder at the menopause; these women were engaged actively as typists, pianists or teachers using the arm a great deal in their demonstrational teaching methods.[115]

In one series of 200 patients with subdeltoid bursitis, only 67 gave a history of trauma (and in 50 of these a focus of infection or toxic element was also found). Focal infection was demonstrated in 176 of the 200 patients; 32 presented definite metabolic disturbances (hyperthyroidism in 18, hypothyroidism in 7 and diabetes mellitus in 7). Subdeltoid bursitis is common among diabetic patients. Inasmuch as the majority of patients

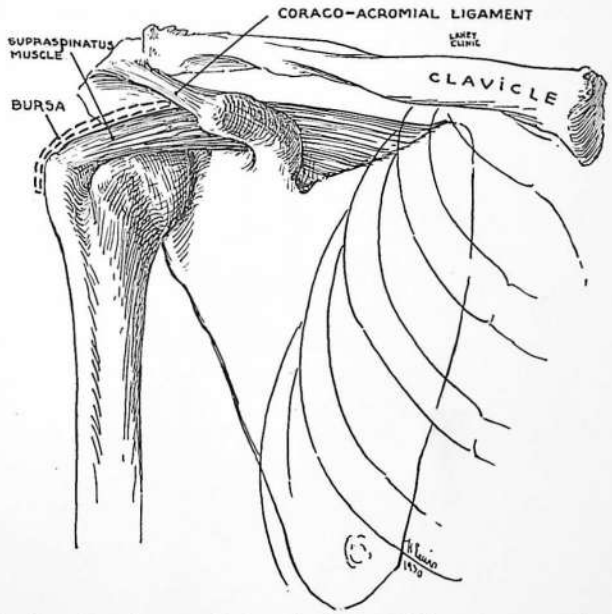

FIG. 225.—Drawing showing relation of subacromial bursa to supraspinatus tendon, coraco-acromial ligament and greater tuberosity of the humerus. (Haggart and Allen, courtesy of Surg. Clin. North America.)

with subdeltoid bursitis are over forty years of age, they fall into that group where focal infection is prevalent. *These lesions are probably produced by a combination of trauma* (acute and sudden, or oft-repeated and slight) *and the physiologic change of age in older patients.*

Injuries to the Shoulder and "Painful Shoulder."—Following trauma to the shoulder, as in fractures, the wall of the subdeltoid bursa may be torn and hemorrhage may occur into it; in injuries or dislocations in the acromioclavicular region, the bursa may be distended with fluid as a result of an exudative reaction.[58] This reaction may be mild with the production of only a slight exudate and congestion, but with the formation of adhesions between the layers of the bursa. Adhesions are produced within the bursa,

and one may find a villous synovitis (from hypertrophy of the synovial folds), or fatty degeneration of these villi. The result of such pathologic changes is decreased motion of the shoulder joint.

The origins and insertions of muscles around the shoulder joint may be damaged by straining or twisting. One occasionally sees a **traumatic tendinitis of the long head of the biceps** producing localized pain anteriorly over the shoulder, pain on raising the arm and tenderness over the bicipital

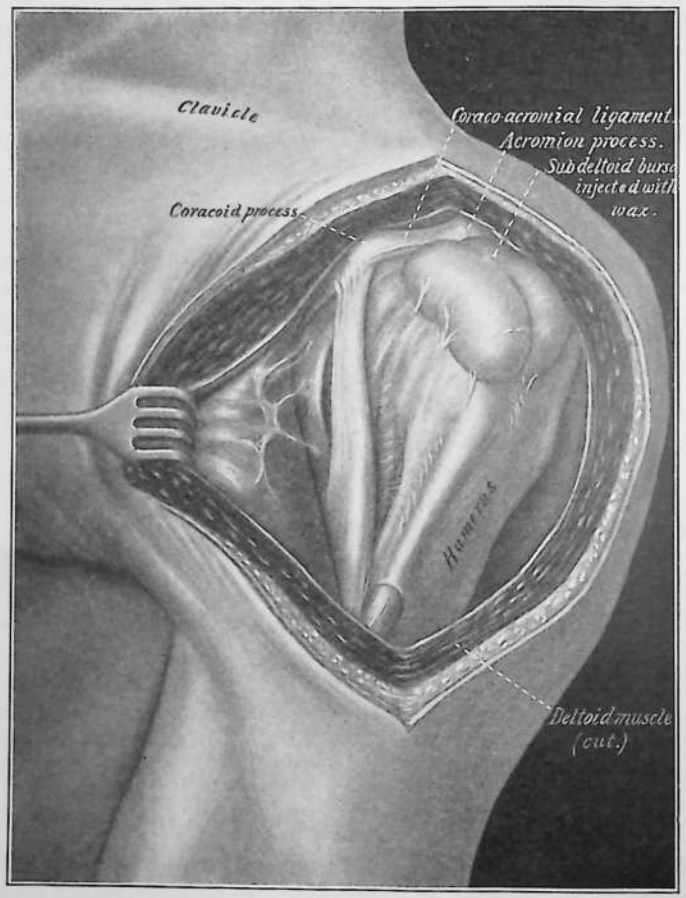

FIG. 226.—The subdeltoid bursa. (Baer.)

groove. Another uncommon lesion is *traumatic myositis of the scalenus anticus muscle,* causing pain at the shoulder radiating down the arm, tenderness over the scalenus anticus muscle mass (behind the lower portion of the sterno-mastoid) and some restriction of motion. Both of these conditions may be dramatically relieved by procaine infiltration.[65]

Other traumatic lesions about the shoulder include dislocation of the tendon of the long head of the biceps, rupture of the supraspinatus tendon and disability resulting from extensive lacerations.

Codman believes that the **most common cause of inflammatory changes within the subdeltoid bursa are tears in the supraspinatus tendon, which often result in calcified deposits within this tendon.** Complete ruptures do not cause calcified deposits.

Others state that the lesion begins as a tendinitis[19] which later undergoes local necrosis and calcification or that the constant movement of the soft parts on each other leads to fraying, roughening or obliteration of the smooth inner surface of the bursa.[78]

Location of the Subdeltoid Bursa.—*The subdeltoid, subacromial and subcoracoid bursæ are probably one structure, although these may be separated by thin films of tissue; this is the largest bursa in the body.* The bursa lies over the greater tuberosity of the humerus and extends downward under the upper portion of the deltoid muscle between its tendon and the tendon of insertion of the supraspinatus and the head of the humerus. It extends under the acromion and the coracoid and under the origin of the short head of the biceps. It may be as large as the palm of the hand. It is lined with a thin synovial membrane; the small amount of fluid secreted by the latter allows movement without friction between the two surfaces. *The subdeltoid bursa is intimately connected with freedom of motion in the shoulder joint.*[121]

Symptoms of Acute Subdeltoid Bursitis (Periarthritis of the Shoulder)

Agonizing *pain in the region of the shoulder joint,* aggravated by motion and *worse at night* (often preventing sleep).
Radiation of the pain at times into the neck, arm, insertion of the deltoid, forearm and even to the finger tips.
Limitation of motion (because of pain); *extreme pain on even the slightest abduction or rotation in acute cases.*
Tenderness in the region of the greater tuberosity of the humerus just below the acromial process (and at times over the entire shoulder); the coracoid process and bicipital groove may also be sensitive to pressure.
Roentgen findings (in about 50 per cent of cases).[101]
Muscular atrophy.

Symptoms of Subdeltoid Bursitis.—The *patient with acute subdeltoid bursitis* develops a pain usually several inches above the insertion of the deltoid muscle. This may be aching or severe and associated with sharp stabbing pain on certain motions, especially that of abduction; in acute cases, this is often agonizing. There may be a history of trauma. **The outstanding symptom is severe pain in the shoulder.**

Not infrequently, the onset is sudden. The patient holds the arm to the side of the body, supported by the good arm. *One of the misleading points concerning this pain is that it may be referred down the lateral aspect of the arm to the elbow, and even into the forearm and hand.* The pain is often so severe that the patient is willing to undertake any procedure for its relief.

In acute cases, exquisite *tenderness* is noted in the region of the greater tuberosity of the humerus. If the examiner can gain the patient's con-

fidence, assuring him that he will undertake no sudden painful motions of the arm, he will be able to move the shoulder joint to some extent antero-posteriorly without discomfort to the patient; however, any attempt at abduction may cause excruciating pain.

Two procedures are useful in diagnosis of this condition: (1) if the arm is abducted no pain is experienced through the first 15 degrees of motion; however, if the abduction is continued, the patient will experience slight or severe pain; (2) the patient is unable (because of pain, and later, of adhesions) to place the arm on the affected side horizontally behind the back in an attempt to grasp the opposite elbow. When this latter motion is attempted by the patient, he invariably tries to assist the affected arm by the hand of the sound arm. *In most acute instances, pain is so severe that sleep is impossible;* in many, large doses of morphine do not completely relieve the discomfort.

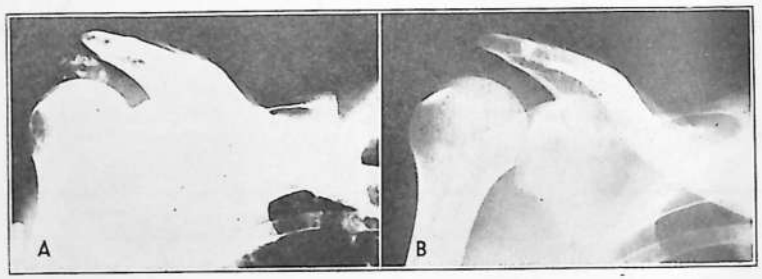

FIG. 227.—Calcification of tendon and bursa—preoperative (*A*); (*B*) postoperative: excellent result of operative treatment. (Rubert, courtesy of Arch. Surg.)

Kaplan has emphasized the frequent occurrence of muscle spasm (including the scalenus anticus) in many painful conditions about the shoulder. Such a spasm produces pressure on the brachial plexus, resulting in pain over the whole shoulder, radiating to the insertion of the deltoid or to the elbow, wrist or fingers. The distribution of the pain is not a reliable guide in locating the lesion.

During the acute stage of the process there may be a moderate leukocytosis and a slight increase in the sedimentation rate.

Roentgen Findings.—Fractures or dislocations must be ruled out by careful roentgen examination if there has been a history of trauma. A complete history, physical examination, and roentgen examination of this region (and sometimes of the cervical spine) will usually reveal the diagnosis.

Roentgen examination of the shoulder region *in* cases of *subdeltoid bursitis* reveals (in about 50 per cent of the patients) a calcified deposit over the greater tuberosity and an associated bony atrophy in this region (if the deposit has been present for some time).[51]

On roentgen examination, shadows of varying intensity are cast by the calcareous deposits; these may be thin and hazy or as dense as bone. The deposits may be single or multiple and may be unilateral or bilateral,

varying in size from a few millimeters to several centimeters in diameter. During the acute stage, the calcification on the affected side may appear homogeneous and putty-like; calcification may be found only on the non-affected side. The calcification appears more granular in chronic cases. During the acute stage, the bursa may be greatly distended with fluid.

If roentgenograms are taken without fluoroscopic inspection and control, they frequently fail to show calcium deposits in the shoulder, additional deposits or the exact relation of the calcium within the tendinous capsule of the joint.[15]

Sandstrom and others have shown that the calcium often present is in the tendinous and peritendinous tissues.[103]

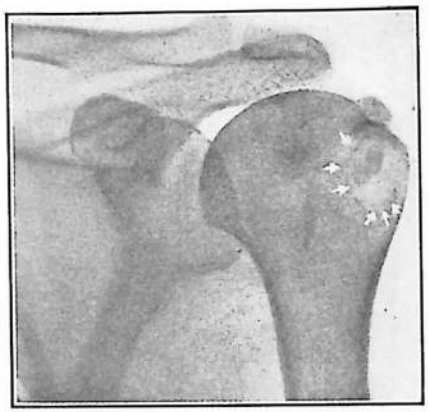

FIG. 228.—Acute subacromial bursitis. Note large mass of calcified deposit. Sharply localized pronounced bone atrophy is indicated by arrows. (Haggart and Allen, courtesy of Surg. Clin. North America.)

In taking such a roentgenogram in an effort to locate a calcified bursa, pictures should be taken in two positions, one with slight internal rotation of the humerus and the other with slight external rotation.[75] In some cases, the calcified deposits may not be visualized by roentgen-ray if there is complete internal rotation of the arm. Roentgenograms of both shoulders should be taken for comparison as bilateral calcified bursæ may be found; one of these may be absolutely painless. Although roentgen changes are often present, *negative roentgen-ray findings do not rule out subdeltoid bursitis.*[18,71,81] On the other hand, calcified deposits may be found in as many as 25 per cent of shoulders with no pain.[20,21]

Bosworth found calcium in routine fluoroscopic examination in one or both shoulders in 2.7 per cent of 6061 supposedly normal persons of the white collar class.[13] These were found most often in males in the period of greatest activity of adult life. The formation or regression of the deposits did not seem to be influenced by serious illness, arthritis or local or systemic infection. Occupations requiring constant prolonged abduction of the arms fostered the formation of calcium deposits in the shoulder. A single trauma *per se* did not cause calcium deposition. *Fluoroscopically visible*

calcium deposits formed within as short a period as two months, but most deposits required a longer time. Small and medium sized deposits disappeared in some cases without symptoms but *large deposits sooner or later resulted in a painful shoulder, although they remained quiescent and symptomless for months or years.*

Roentgenologically, the calcified area must be differentiated from the anterior tip of the acromion, chip fractures of the tuberosities, atrophy of the proximal end of the shaft with subchondral cysts (sometimes seen in arthritis), primary or metastatic malignancy, leukemia, Hodgkin's disease and free bodies in the joint.

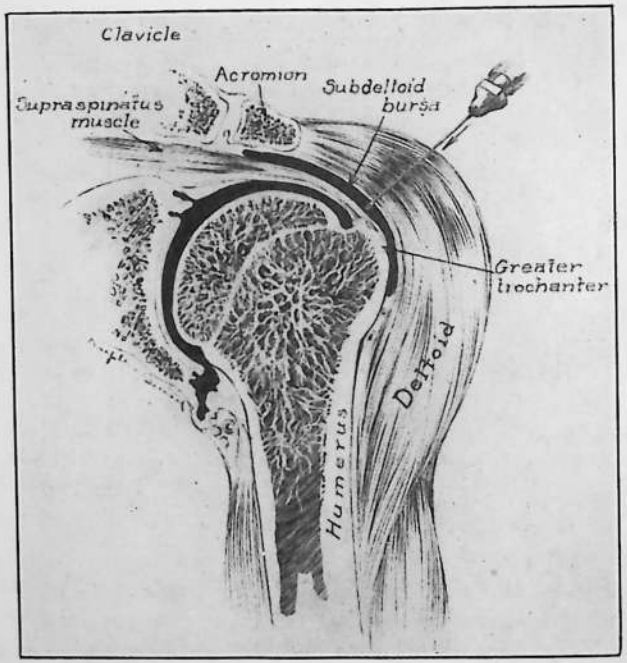

FIG. 229.—Injection of subdeltoid bursa. (Haldeman and Soto-Hall, courtesy of Jour. Am. Med. Assn.)

Causes of Calcification in Patients With Subdeltoid Bursitis.—*The calcified deposits actually occur in the tendons of the short rotator muscles,* most commonly in the supraspinatus tendon, but also sometimes in the tendons of the subscapularis, teres minor and infraspinatus.[41] Most observers feel that *deposition of calcium salts occurs in areas of local necrosis in the tendon fibers.*[6,93] Repeated minor trauma in these regions probably results in localized death of tissue. Because of the poor circulation, these necrotic areas become calcified.

The Nature of the Calcium Deposits.—The deposits may be unilateral or bilateral, single or multiple. Such deposits may vary in consistency from a milky fluid or chalky material or to discrete calcified nodules or

massive calcification. This material is usually the consistency and color of white tooth paste. The areas of calcification are composed of calcium phosphate and carbonate; traces of iron, magnesium and chlorides have also been found.

Treatment for Acute Traumatic Subdeltoid Bursitis.—Acute traumatic bursitis is often associated with injuries to other structures of the shoulder; those most commonly affected include the acromioclavicular joint, fractures of the humerus (head, neck or tuberosities), dislocation of the shoulder and damage to the supraspinatus and infraspinatus tendons. In these cases, attention must be given not only to the "bursitis" but also to the associated pathology. In other cases, the trauma may be slight, such as excessive use of the arm in abduction, twists and wrenches of the shoulder joint, blows upon the shoulder, or falls upon the hand or arm with the arm in partial abduction.

Most patients with acute traumatic subdeltoid bursitis respond well to conservative therapy including temporary use of a sling, procaine injection into the bursa and analgesics. If pain is not relieved promptly, roentgen therapy is advisable. Following the subsidence of pain, graduated exercises must be employed to prevent the formation of dense adhesions.

Kaplan and others[42] have reported good results in these patients by *aspiration of the bursa, injection of 5 to 10 cc. of 1 per cent procaine, followed by a thick axillary pad with a sling and swathe dressing in order to maintain a moderate amount of abduction.* An ice bag may be applied if this affords relief in the acute stage. Active exercises are begun in three to five days in order to avoid adhesions. If the patient refuses procaine injection, the most ideal immobilization results from putting the patient to bed and prescribing a sufficient amount of sedative to insure quiet.

Pendergrass and Hodes have reported rapid improvement[96] with roentgen therapy in 60 per cent of patients with acute or subacute subdeltoid bursitis. Others recommend an abduction splint with external rotation of the arm, maintaining the dressing day and night until the arm can be actively and normally abducted.[77] This is a very painful procedure and its value has been questioned. Cold applications and galvanism to the shoulder have been advocated during the acute stage.[37] Some prefer not to immobilize more than with a sling.

Papurt has devised a *shoulder abduction cap* consisting of an ordinary canvas aviator-type cap (fitting snugly and buckling under the chin) and a padded leather wristlet; the strap of the wristlet is attached to the cap through a reinforced loop on top of the latter. This is a convenient method for maintaining abduction of the shoulder in ambulatory patients.[94]

Treatment for Acute Subdeltoid Bursitis When Calcification is Present.—*The choice of therapy* in this extremely common condition *seemed to depend in the past in large part upon the field of specialization of the writers on this subject;* thus, the roentgenologist claimed excellent results with roentgen-ray therapy; the physical therapist stated that diathermy, galvanism or iontophoresis were the treatments of choice; the surgeon preferred surgical incision and removal of the calcified material; the ortho-

pedist suggested a regimen including splints and traction, while medical men (and some surgeons) found that good results followed procaine injection and washing out the bursa by needle irrigation, or multiple punctures in the wall of the bursa. Some surgeons have never been able to wash out calcareous material even with fluoroscopic guidance; these individuals prefer immediate operation in the male and in the female (unless the social position is such that the resulting scar is a factor).[9]

Dick, Hunt and Ferry reported *absorption of some of the calcified deposits* (if these were not too dense) by a program which included the use of *enteric coated tablets of ammonium chloride* in doses of 1 gram or more four times per day.[32] This produced a mild acidosis which they thought would tend to absorb calcified deposits. In addition to the ammonium chloride, they included in their program of therapy rest of the shoulder, physical therapy and elimination of focal infection. During the day, a sling was used for the affected arm and abduction of 40 to 60 degrees was maintained at night by resting the arm and forearm on a pillow, one end of which rested on the mattress and the other end over the chest and abdomen.

In these patients with acute subdeltoid bursitis with calcification, we attempt to *wash out the calcified material* after preliminary procaine injection. It is often possible, by the use of two large gage needles, to wash much of the soft calcified material out of these bursæ. In addition to this, we also employ *roentgen therapy* and temporary splinting of the area in abduction for several days until the acute pain has subsided. Analgesics are required (aspirin and codeine or morphine). After several days, *passive and then active motion* is encouraged to prevent the development of a frozen shoulder.

The presence or absence of calcification is not *per se* of any significance in determining the response to irradiation. We have seen excellent results in these cases following roentgen therapy. The acute symptoms may be relieved in many of these patients by narcotics and by tying the forearm to the head of the bed (elevating the arm above the head).

Treatment of Simple Non-calcifying Bursitis.—No operative procedures are usually required; immobilization of the shoulder and arm by adhesive strapping or by an axillary pad, body swathe and wrist sling are performed and the patient is given an analgesic. Rest in bed is one of the best methods of reducing motion at the shoulder joint. If the pain is severe, procaine injection and needling of the bursa may be performed. In some cases, aspirin, 0.6 gm. (10 grains) and codeine sulfate, 30 gm. (½ grain) by mouth every three or four hours are sufficient to relieve the discomfort; phenacetin may be substituted for the aspirin if marked sweating occurs. If the pain is excruciating, one may employ a mixture of aspirin, 0.6 gm. (10 grains) and dilaudid, 3 mg. (1/20 grain) by mouth every three hours (in adults). It may be necessary to give a sedative such as 0.1 gm. (1½ grains) of seconal or nembutal (by mouth) at bedtime if sleep is not forthcoming; this may be repeated once after three hours if necessary. *Do not use small doses of analgesics and do not withhold narcotics in this extremely painful condition.* Large doses of aspirin often give greater relief than morphine in this type of pain.

If the patient is not relieved of severe pain within thirty-six hours by splinting the shoulder and arm, or if the pain becomes increasingly severe, one may perform procaine injection and needling of the bursa; if relief is not obtained immediately, roentgen treatment is given to the involved area.

In acute or subacute subdeltoid bursitis, 50 r is given over the shoulders at a distance of 25 to 35 cm.[96] Treatments are given daily for four days. After a week, another single treatment is given, followed again in another week by another single treatment if necessary—giving a total of six treatments. If definite improvement does not follow no further therapy is given. Factors are 140 kv.; filtration ¼ mm. copper or less; 8 ma. Treatments are given anteriorly, posteriorly, laterally and on top of the shoulder; in some cases, the back of the neck is also treated.

In chronic subdeltoid bursitis, two treatments are given per week for six weeks, with a total dose of appoximately 600 r. In these patients, the dosage is modified to fit the case. When bilateral subdeltoid bursitis is present, one may desire to give 50 r to one shoulder and 75 to 100 r to the other.

The *pain may be somewhat aggravated for the first twenty-four hours following treatment*, but on the following day, there is usually a marked diminution of pain and increased motion of the arm. Following roentgen therapy, the calcified deposit may slowly disappear. It is not possible to say whether this calcium resorption is more rapid following irradiation than after other non-surgical therapy.

Calcified deposits may also disappear after diathermy treatments. Some authors have reported more rapid disappearance of the lesions following the production of a mild acidosis by ammonium chloride (1 gm. q.i.d.).

Roentgenologists have become enthused over the good results of roentgen therapy in acute and subacute subdeltoid bursitis.[5,7,17,23,24,31,49,55,75,89,96,97,104] At least 60 per cent of patients with acute or subacute subdeltoid bursitis are greatly benefited by roentgen therapy. Occasionally, patients who are not relieved by roentgen therapy may subsequently be improved by injection and washing out of the involved area. Conversely, roentgen therapy may result in improvement in patients where minor surgical procedures may have failed. *Roentgen therapy is of little or no value in chronic adhesive bursitis.* The success of surgical procedures usually varies with the experience and skill of the operator.

Treatment of Acute Subdeltoid Bursitis With Calcium Deposition Demonstrable by Roentgen-ray

Temporary splinting of the joint and sedation as described previously.
Procaine injection and needle irrigation of the bursa.
An immediate series of at least three roentgen treatments. Relief of pain and disappearance of calcium deposition have been noted by numerous authors following such therapy.[39,64]
If pain and calcification persist, one may employ *diathermy:* in some cases it may be necessary to *remove the calcified material surgically.*
Proper passive and active exercises and heat to the shoulder when the acute stage has passed.

Technic of Irrigation (Injection) in Acute Bursitis.—Darrach and Patterson have claimed that patients with subdeltoid bursitis are returned to work much earlier than with any other method employed (after less than five days of disability in acute cases).[29] The materials required are: (1) two 18 gage sterile needles (2½ inches long); (2) one 20 cc. syringe; (3) 60 cc. of a sterile 1 per cent solution of procaine; (4) one 2 cc. sterile hypodermic syringe and hypodermic needle; (5) 100 cc. of sterile physiologic saline; (6) one No. 10 Bard-Parker blade.

The procedure is as follows: With the patient in the prone position, the affected shoulder is cleansed with iodine and alcohol and the region draped with sterile towels. The points for insertion of the two large needles are now determined. The *first* of these is *at the point of maximum tenderness* (which is usually about 1 inch lateral to and on the same horizontal line as the coracoid process of the scapula). The *second* is about ½ inch behind the greater tuberosity of the humerus on a level with the superior facet. At each of these sites, a small wheal is made in the skin with procaine.

With the point of the scalpel, the skin is nicked at the first of these sites. Following the injection of procaine, one of the large needles is pushed through this skin incision. The point of the needle is directed posteriorly and upward toward the under surface of the acromial process of the scapula. The wall of the bursa can be felt (by its resistance) when the needle has reached a depth of from ½ to ¾ of an inch. A quick thrust then places the needle within the bursa. *The pressure within the bursa is at times so great that the cloudy fluid from the bursa may push back the plunger of the syringe.*

The second needle is injected at site number two (just posterior to the greater tuberosity) about one-finger's breadth below the acromioclavicular joint. This is also performed after preliminary injection of procaine into this area. The needle is pushed down gently to the superior facet of the greater tuberosity when bone can be felt at the tip of the needle. If the needle is slowly withdrawn for approximately ⅛ inch, and its tip pointed in the direction of the assumed position of the first needle, the bursa is entered when the second needle has not been inserted for more than ½ inch. Approximately 2 cc. of procaine is used in the insertion of each needle.

Both needles are now within the bursal sac. The 20 cc. syringe is connected to either of the needles and sterile saline is injected into the bursa; this is forced out of the other needle, washing out with it the calcium salts from within the bursa. The amount of saline required to wash the bursa clean may vary from 30 to 60 cc. *The acute bursal pain often disappears following the washing out with even the first syringe full of salt solution.*

When the bursa has been washed clean, the needles are removed and the area is covered with a sterile dressing. The patient may now move his arm freely in all directions. The period of disability is shortened if care is taken to avoid the injection of the salt solution into the tissues around the bursa. *This therapy is contraindicated in patients with old calcification and a hard bony-like appearance of the calcified area.*

Other Forms of Treatment in Acute Bursitis: 1. Multiple punctures of the bursa.

2. Simple procaine injection of the area involved without evacuation of the contents of the bursa.
3. Local physical therapy.
4. Surgical removal.

Multiple Punctures of the Bursa.—Flint first suggested needling the subdeltoid bursa in 1913 in cases of acute traumatic subdeltoid bursitis.[43] More than thirty years ago Weeks inserted a hypodermic needle over the swollen region of the subdeltoid bursa (in a patient with a painful shoulder) and removed several cc. of clear straw colored fluid with almost immediate relief of pain.[118] No additional treatment was given and the

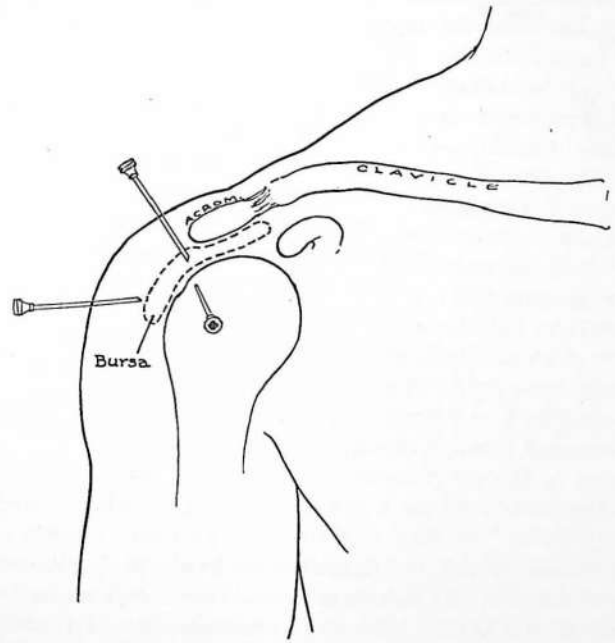

FIG. 230.—Technic of procaine injection of the subacromial bursa and adjacent capsule of the shoulder joint. (Haggart and Allen, courtesy of Surg. Clin. North America.)

patient was never again troubled with pain in the shoulder. Because of this experience, he and his colleagues attempted to aspirate the bursa in these cases and found that the mere passage of the needle through the bursa relieved the symptoms whether fluid could be withdrawn or not. *Simple multiple punctures of the bursal sac resulted in relief of symptoms in a large percentage of their patients.*[117] In their experience, the presence or absence of calcareous material made little difference in treatment as the calcified area subsequently disappeared.

This procedure is simple and can be performed without difficulty by anyone who will observe surgical asepsis and will acquaint himself with the anatomy of this region. All that is done is to infiltrate the skin and subcutaneous tissue over the bursa (usually the point of maximum tender-

ness) with 1 or 2 per cent procaine (using from 5 to 15 cc.). An intramuscular needle (gage 17) is introduced into this area through the line of the procaine injection and is passed into the bursa; if fluid is encountered, it is evacuated (from a few minims up to several cc. may be obtained); whether fluid is obtained or not, the bursa may be needled by a fan wise series of punctures, and a sterile dressing applied. The patient is given an analgesic such as aspirin 0.6 gm. (10 grains) and codeine 15 to 30 mg. ($\frac{1}{4}$ to $\frac{1}{2}$ grain). Pain may disappear within fifteen minutes after such treatment; however, one should order mild sedation for the ensuing twenty-four hours. Full use of the arm is usually possible within five to nine days.

Simple Procaine Injection.—Procaine injection of the bursa and supraspinatus tendon has been used widely in recent years in the treatment of subdeltoid bursitis.[113] The results obtained in this as well as the previous procedure may be due to an increased local circulation, which aids in the more rapid removal of the calcified deposit; in addition, by puncturing the supraspinatus tendon, some of the calcified material may rupture into the bursa. Rupture of the calcified material from the supraspinatus tendon into the bursa is the manner which Codman stresses as the probable means by which nature affords relief in this condition.

Because of the intense pain which is experienced in the shoulder region, some of these patients will not permit any operative procedure on the shoulder without anesthesia. Some workers have used intravenous sodium evipal or cyclopropane gas anesthesia, while others have found brachial plexus block to be an excellent anesthesia. These workers inject at least 20 cc. of a sterile 1 or 2 per cent procaine solution into the bursa, and in addition, attempt to inject the capsule of the shoulder joint and the tendon in the region of the calcified deposits. Such injections have proved very helpful in the acute cases, as well as in many of the more chronic adhesive types.

Kelly detected a number of patients with a painful shoulder who showed a circumscribed area of tenderness in the trapezius muscle.[70] Injections of procaine hydrochloride into the muscle (or possibly into the supraspinatus or levator anguli scapulæ which are directly beneath the trapezius) relieved the shoulder disability.

Travell, Rinzler and Herman reported a group of patients with pain in the shoulder region and arm who presented tender points in the muscles of the back and shoulder girdle.[113] Such trigger points were found most often in the serratus posterior superior, in the upper lateral portion of the muscle where it was partially covered by the scapula. Trigger points were also found in the infraspinatus and other areas. Treatment was carried out by injecting 1 per cent procaine (from 2 to 5 cc.) into the point of maximum tenderness (usually in the serratus posticus superior). The hand of the affected side was placed in the opposite axilla, the shoulder was depressed and the spine was flexed in order to expose the trigger point for injection. Two-thirds of the patients obtained full restoration of motion and complete remission of pain (after an average of 4 or 5 injections); most of the others showed some degree of improvement.

Local Physical Therapy.—Methods of local physical therapy which have been recommended in the treatment of acute subdeltoid bursitis include: (1) *local heat;* (2) *cold applications;* (3) *diathermy;* (4) *galvanization;* (5) *iontophoresis* (with mecholyl or magnesium sulfate).

Reports concerning the use of local forms of physical therapy vary markedly in both the methods employed and the results obtained.[72,106] Some recommend dry heat and diathermy. In the experience of others, these result in increased pain during the acute stages of a bursitis; local applications of moist heat, rest in bed, and morphine 15 mg. ($\frac{1}{4}$ grain) as necessary for pain, have produced better results for some workers (during the acute stages).

Coulter advocates daily treatments (twenty minutes each) for several weeks with **short wave diathermy** for acute subacromial bursitis; he also advises **infra-red radiation** for thirty minutes at least twice a day with the arm in abduction.[27] In some instances diathermy seems to aggravate the pain; it should be discontinued in such cases, and rest in bed with continuous moist heat to the shoulder may be tried. From ten to twenty or more of these treatments may be necessary for relief.[112] Troedsson found the anteroposterior technic to be superior to the lateral.[114] Diathermy resulted in dissolution of calcium deposits; larger and denser deposits required a greater number of hours of diathermy. His technic was as follows:

The active, small, pliable, black tin electrode (9 x 5 cm.) was molded over the anterior part of the shoulder, so that the long upper border was close to and parallel with the lateral margin of the acromial process; the patient was recumbent and the arm was supported on a pillow and rotated inward. The lateral margin of the electrode reached the middle of the deltoid and was held in position by sandbags.

The inactive electrode (13 x 12 cm.) was placed posterior to the shoulder so that the upper margin was barely visible and the lateral border was in line with the lateral margin of the thorax. By this arrangement, concentration of the current occurred directly over the region of the bursa.

Only 500 to 700 ma. of current was used (just enough to produce a comfortable, warm feeling under the active electrode). Treatments averaged forty minutes per day; these were given daily (or two times per day) averaging twenty or more treatments per patient.

Solomon and Morton found that *roentgen therapy and diathermy were equally effective in the treatment of periarthritis of the shoulder.*[107] Using the electromagnetic induction coil with a 22 or 25 meter wave length, the coil covered the entire shoulder anteriorly and posteriorly with a loop encircling the axilla if possible. In the acute conditions, the current was allowed to flow for three to five minutes, and was discontinued for a minute and was then repeated until the patient had received about fifteen minutes of treatment. In subsequent treatments, the total treatment was extended to a maximum of twenty minutes. In the more chronic cases, the current flowed continuously for twelve to fourteen minutes for the first treatment and subsequent treatments were increased to a maximum of twenty minutes. Treatments were given three times a week.

Echtman and others advocate the use of *cold wet compresses or an ice cap during the acute stage*. The technic consists in placing 2 or 3 wet towels over the shoulder and securing these by a spica bandage. The patient is instructed to keep the compress wet and cool by instilling cold water into it from time to time as necessary; an ice bag may be placed upon the compress. The compress is changed daily and this procedure is continued for a week. It is claimed that the patient is greatly relieved of his pain during this time, although limitation of motion may persist. Echtman also employs treatment by **galvanization** and **iontophoresis** with magnesium sulfate or mecholyl. Galvanization is the application of the galvanic current using only tap water (without the addition of any medication to the water). This is distinguished from iontophoresis (ionization or ionic

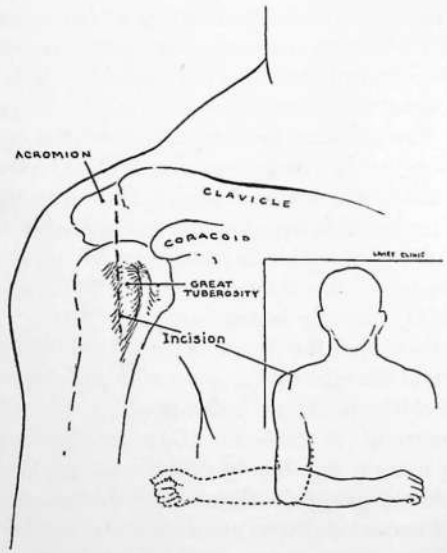

FIG. 231.—Incision for exploration of the subacromial bursa. Rotation of the forearm (see insert) permits visualization of the short rotator muscles. (Haggart and Allen, courtesy of Surg. Clin. North America.)

medication) which is the actual introduction of chemical ions into the tissues by means of a direct current. In galvanization of the shoulder, the positive pole of the galvanic apparatus should be attached to the metal electrode over the shoulder. The straight galvanic current only is employed; the duration of treatment is from twenty to forty minutes with the current being gradually increased to the patient's tolerance. For ionization, either a 1 per cent solution of magnesium sulfate or a 1 to 500 solution of mecholyl (beta methyl acetyl choline chloride) is used.

Exploration and Drainage in the Treatment of Subdeltoid Bursitis.—Some surgeons believe that the most rapid relief in the treatment of subdeltoid bursitis follows exploration of the bursa and removal of the calcified material.[8] This procedure is beyond the realm of the general

practitioner, and will not be described in detail. The operation is simple, being performed under anesthesia (local infiltration, brachial plexus block, or general anesthesia if desired). By means of a small vertical incision over the region of the anterior surface of the greater tuberosity (1 inch or more in length), the fibers of the deltoid are separated and the roof of the bursa may be pricked with the point of a knife and opened. The calcified substance can then be seen through the floor of the bursa; if this is pricked the toothpaste-like material is allowed to exude. This may be curetted gently if desired. Codman believed that *thorough curettage is unnecessary* as results in his hands were as favorable in cases where particles of the deposit were left among the tendon fibers. The muscle wound is gently sutured with catgut and the skin is closed.

Bosworth found that radical exploration of the shoulder joint through a division of deltoid fibers close to their attachment to the acromial margin permitted thorough inspection and produced no permanent defect so far as deltoid function was concerned.[14]

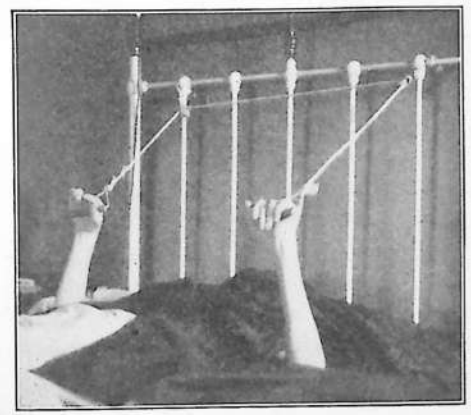

FIG. 232.—Simple traction apparatus for increasing shoulder motion. (Swaim and Kuhns, courtesy of Jour. Am. Med. Assn.)

The postoperative care of these patients includes either rest in bed for a period of one or two days, or carrying the affected arm in a sling. In either case, *one of the most important points in follow-up treatment is the use of active exercises usually begun within forty-eight hours after the operation. The patient is not forced, but is encouraged to move his arm as much as possible without producing discomfort.*

Special exercises to develop shoulder muscle power have been used to advantage by Haggart and Allen. These include swinging the arms anteroposteriorly and also mediolaterally with the patient in a stooped position; placing the hand of the affected arm on the opposite shoulder posteriorly; touching the floor without bending the knees; and holding the arm out horizontally so that the fingers just touch the wall and then attempting to "climb the wall" with the fingers of the outstretched abducted arm.

During this period of **after care**, additional therapy includes: A *sling to the affected arm*, or *rest in bed and exercises* as mentioned previously.

Hot applications, infra-red radiation, or both to the shoulder.

Massage of the muscles above and below the point of the shoulder.

These principles of after treatment should be instituted in all cases of subdeltoid bursitis as soon as is permissible (when the acute pain is relieved), in order to develop motion at the shoulder joint and to prevent muscular atrophy. **Regardless of the method of treatment of the bursitis, one should always follow this with exercises, local heat and massage**; the physician may be amazed by the rapid return of mobility and power in the shoulder joint. and by the absence of local muscular atrophy in this region.

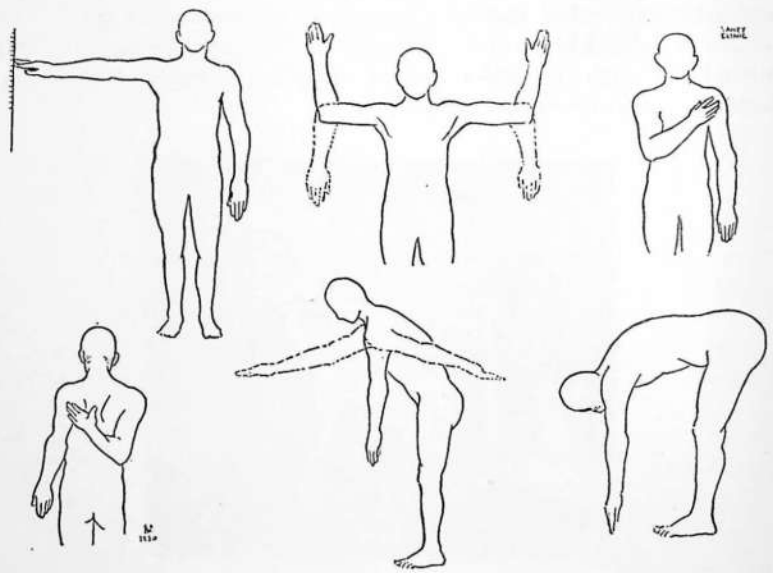

FIG. 233.—Exercises useful in redeveloping shoulder muscle power; of special value are climbing the wall with the fingers of the outstretched abducted arm, and swinging the arms anteroposteriorly and also mediolaterally (not shown) with the trunk flexed. (Haggart and Allen, courtesy of Surg. Clin. North America.)

Treatment of Chronic Bursitis.—Patients with chronic subdeltoid bursitis without calcification are *not* definitely benefited by surgical intervention; (chronic cases with calcified deposits are usually relieved by operation)- These patients usually complain of chronic shoulder pain without much limitation of motion. The pain is frequently more noticeable at night and prevents the patient from lying on the affected side. It may also be present during movements requiring abduction of the arm, such as putting on a coat. Ferguson has observed in some of these patients a radiation of pain down the arm and into the fingers with some associated swelling and stiffness.

Physical signs in these patients include:

1. *Tenderness* on deep pressure over the greater tuberosity; this is usually diagnostic.

2. *Pain on elevation of the arm* or in lowering the arm from a position of elevation. This occurs simultaneously with the passage of the greater tuberosity under the edge of the acromion process.

3. Small *calcium deposits* (more common in younger than in older patients).

4. Occasionally, *decalcification of the greater tuberosity* of the humerus with bony excrescences and cortical eburnation (in older patients).

5. A click or crepitation on elevating or lowering the arm; the click is simultaneous with the occurrence of pain. This is present in some of the older patients only and is more characteristic of rupture of the supraspinatus tendon.

Good results have been obtained in the treatment of chronic bursitis by means of roentgen-ray therapy in about 50 per cent of patients.[22,96] Pendergrass, Hodes and others advise one or two treatments a week, extending over a period of from four to six weeks. Symptoms may be exaggerated for a period of twenty-four hours following roentgen therapy. If patients with *chronic bursitis with calcification* do not respond to roentgen therapy or diathermy, one may advise *incision and surgical curettage of the calcified area.* If this is done properly, practically all of these patients will be relieved.

If roentgen therapy does not produce relief, and if the patient refuses surgery, one may prescribe diathermy and rest of the shoulder.[56,100] Rest of the shoulder is best carried out in these cases in a position of abduction.

Many physicians have seen no marked beneficial response of shoulder symptoms following removal of focal infection; occasionally, the latter has immediately preceded the stage of comfort. If definite focal infection is present, this should be attended to as a general health measure, but the patient should not be told that his bursitis will be cured by removal of his infected teeth or tonsils. Removal of an area of focal infection may, however, improve the general health and resistance of the patient.

CHRONIC ADHESIVE BURSITIS

In this group are placed those patients in whom adhesions have formed between the bursal surfaces so that the gliding mechanism of the bursa has been lost. This form of bursitis shows the poorest response to treatment. The main aim in therapy in these cases is to separate the adhesions in the bursa so that its gliding function can be restored.

This condition has been known as periarthritis, "frozen shoulder," tendinitis, etc.

In chronic adhesive subacromial bursitis, there is proliferation of fibrous tissue in the periarticular structures, hypertrophied synovial folds and dense adhesions within the adjacent bursæ.[11] The surrounding muscles become contracted following prolonged spasm; the muscles later atrophy and degenerative changes and scar tissue formation is noted in the tendons and capsule of the shoulder joint. *Adhesions between the roof and floor of the subacromial bursa which result in loss of the normal gliding function are probably the basic factor causing disability in chronic adhesive subacromial bursitis.*

Symptoms of Chronic Adhesive Subacromial Bursitis

Marked *atrophy* of *the deltoid and spinatus muscles*, often associated with coldness and stiffness of the hand and atrophy of the skin.

Inability to move the arm through its normal motions at the shoulder (particularly abduction and external rotation).

An aching *pain in the shoulder*, sometimes radiating to the insertion of the deltoid or to the hand.

Tenderness over the greater tuberosity of the humerus and usually along the deltoid muscle as far as its insertion.

Roentgen evidence of atrophy of the greater tuberosity and sometimes the presence of a calcified area in those cases which have followed an acute bursitis.

Treatment of Chronic Adhesive Subacromial Bursitis (Frozen Shoulder)[52]

1. Inform the patient at the onset that the *program of therapy will require at least several months*, and in some cases more than a year.
2. Patients with considerable pain should be *hospitalized* at the onset if possible (in order to effect more rapid progress).
3. In the presence of considerable limitation of motion and marked discomfort, put the patient to *bed* and suspend the arm in a Balkan frame apparatus with the *affected arm abducted* as far as possible without causing added discomfort. *Adhesive traction* is then applied to the upper arm (an average of 6 to 8 pounds of weight), utilizing the Blake board.
4. *Encourage the patient to move the arm* in the apparatus as much as he can comfortably.
5. Apply *intensive local heat* to the shoulder several times each day, together with daily massage.
6. After three to seven days (when the patient has become adjusted to his apparatus), institute intensive *active exercises* of the shoulder. Abduction may be increased 30° or 40° within a week on this program.
7. When maximum improvement (abduction) has been obtained, *manipulate the shoulder joint* in the patient's room, *using pentothal sodium anesthesia*, while the arm is in the apparatus. The operator must immobilize the scapulæ with one hand, while the other hand grasps the upper end of the humerus. The arm is abducted laterally in progressive stages; adhesions will be felt and heard to rupture during these manipulations. *By grasping the upper end of the humerus, there is less likelihood of fracture as the leverage action on that bone is reduced.*
8. *Do not attempt to obtain a normal range of motion in the first manipulation* because of the resulting increase in pain which would prevent further motion. Gradually increase the range of lateral abduction and rotation in progressive stages.
9. It is safer to manipulate too little rather than too much.
10. *Following each manipulation, inject 1 per cent procaine into the subacromial bursa and the periarticular structures* of the shoulder joint to reduce the degrees of discomfort following the manipulation.
11. Continue intensive heat and sedatives as required in the twenty-four hours following manipulation but do not insist that the patient move his arm in this period unless he wishes to do so; encourage movement, but do not insist upon it during this period.
12. Institute active arm exercises again on the second postoperative day (attempting to increase the range of lateral abduction and rotation).

Treatment of Chronic Adhesive Subacromial Bursitis (Frozen Shoulder)[52] —*(Continued)*

13. When maximum improvement has been obtained, again manipulate the arm under pentothal sodium anesthesia.
14. Progressively decrease and finally omit traction on the arm as the pain in the shoulder becomes less severe; this permits more active motion of the extremity.
15. When the patient becomes ambulatory, he may be discharged from the hospital. He should then begin *pendulum exercises*, swinging the arms forward and backward, and then raising the arms laterally, with the body bent forward. The pendulum type of exercise permits shoulder joint motion with a minimum of effort or discomfort as the effect of gravity is eliminated.
16. Following discharge from the hospital, *progressive active muscular exercises* must be insisted upon, together with daily heat at home, supplemented, if possible, by diathermy or infra-red radiation and massage to the shoulder muscles several times each week.
17. *If calcification is present* in the short rotator muscles or in the bursa, *attempts are made* at the beginning of treatment to *wash out as much of this as possible* by passing large bore needles into the area of calcification and washing salt solution or procain from a syringe through its needle and out of the second needle; in many instances, the calcified material will be sufficiently soft to permit this to be washed out in large part through the second needle.
18. *In individuals whose adhesions are extremely resistant to manipulation*, it may be unwise to attempt to rupture them. In such cases, *open operation is advised*, exploring the bursa, and dividing many of the adhesions with a knife; following this, one may perform the usual manipulative procedures. If there is extensive bony atrophy and dense firm adhesions, manipulations could easily fracture the shaft of the humerus and surgery is preferable.
19. In a few individuals with a dense calcified mass in the tendons or subacromial bursa, if this cannot be washed out and does not diminish in size following either roentgen, diathermy or ammonium chloride therapy and if dense adhesions are present in addition, it may be necessary to explore this type of individual and incise and curette the calcified deposit in the tendon.

The *symptoms* of chronic adhesive subacromial bursitis include *persistent pain* in the shoulder which may extend into the neck or down the arm to the hand. There is *marked loss of shoulder joint function*. In some instances, the only complaint is the latter. If the process has been present for some time, there is some degree of *atrophy of the shoulder muscles* and in some instances one notes *loss of finger joint function* with swelling of the hand and fingers. *Lateral abduction and external rotation are the particular shoulder motions that are lost.* There is usually an almost normal range of antero-posterior motion as well as internal rotation. *The points of tenderness over the shoulder are less acute and less poorly localized than in patients with an acute bursitis.* Tenderness is defined more accurately in the chronic adhesive cases if calcification is present. Roentgen examination reveals generalized atrophy of the bones of the shoulder girdle in chronic adhesive bursitis.

Chronic adhesive subacromial bursitis may result from an illness which forbids function of the shoulder joints, such as prolonged rest in bed following hemiplegia,[36] cataract operation, coronary occlusion, etc.

If the symptoms are mild and there is only moderate restriction of motion, exercises (as outlined previously), heat and massage may be the only therapy required; these patients should be encouraged to use the arm as much as possible and to place the arm above the head while sleeping.

Important Pointers in the Subdeltoid Bursitis "Syndrome"

Synonyms: Subacromial bursitis, periarthritis of the shoulder, painful shoulder, supraspinatus tendon calcifications, etc. By common usage, the term "subdeltoid bursitis" includes in addition to bursitis the frequently accompanying supraspinatus tendon calcifications.

Usual Pathology: Inflammation in the general region of the subdeltoid bursa frequently with calcification (not in the bursa as a rule, but in the tendons of the supraspinatus or of the other short rotators).

Etiology: Most commonly from acute or chronic trauma to tendons of the short rotators.

Outstanding Symptoms: Severe pain in the shoulder, tenderness over the greater tuberosity of the humerus, inability to elevate the arm because of severe pain, and often the presence of calcified material seen by roentgen-ray.

Types: Acute traumatic, acute non-traumatic (probably from calcium deposits), chronic (with and without calcium deposits).

Treatment: In general, best results are obtained *in the acute and subacute forms* by rest, roentgen-ray therapy, procaine injection, diathermy and special exercises and heat during the recovery phase. *In chronic adhesive cases,* adhesions must be broken up by heat, traction and manipulation (usually under pentothal sodium anesthesia). Exercise for the shoulder joint must be instituted as soon as possible in all cases after cessation of the acute pain. Surgical curettage of a calcified area may be necessary occasionally.

Results of Therapy: Improvement occurs in most patients, often even without treatment; in cases without treatment, symptoms usually persist for long periods. Relief of acute pain (usually within twenty-four hours) follows either roentgen therapy, procaine injection (properly done) or incision, drainage and curettage. Results are not as dramatic with diathermy. Chronic cases with calcification are practically always improved by surgical curettage of the calcified deposit; roentgen therapy and diathermy should be tried first in such cases.

Shoulder disability may be due to *rupture of the supraspinatus tendon,* a much more serious and disabling condition than that caused by calcium deposition.

Manipulation of the shoulder under pentothal sodium anesthesia may be necessary when the condition is advanced; these manipulations should include abduction, internal and external rotation. Most individuals with chronic adhesive bursitis cannot be manipulated under procaine anesthesia. Great caution must be employed because of the danger of rupture of tendons or bones. When the first snap is felt or heard, manipulations are discontinued; the results are usually fairly good. Several manipulations

are usually necessary in advanced cases. It is better to do too little, rather than too much, at one manipulation. This procedure produces an increase in pain in the shoulder for a period of twenty-four hours following the manipulation. After breaking up these adhesions, exercises for the shoulder (as described previously) form an important follow-up treatment. Recovery practically always occurs, but is usually measured in months, rather than days.

Remember That:

Injury is the most common cause of subdeltoid bursitis; involvement of the bursa is common in injuries to the acromioclavicular joint, dislocations of the shoulder, fractures of the head, neck and tuberosities of the humerus; injuries to the supraspinatus and infraspinatus tendons and injuries from overuse of the arm (as twists and wrenches of the shoulder joint); blows in the shoulder region; and falls upon the arm or hand.

A history of injury or infection may be absent.

In the acute stage, dry heat, massage and diathermy may increase the pain; after subsidence of the acute symptoms (seven to ten days) these measures, in addition to proper shoulder exercises, are of benefit.

Unsatisfactory results often follow treatment of painful shoulders because of incorrect diagnosis.

The calcification seen in these patients is actually present in one or more tendons of the short rotator muscles rather than in the bursa itself; this is most common in the supraspinatus and infraspinatus or both.

Such calcification may have been present for years without producing symptoms; recent trauma may precipitate symptoms.

Most of these patients are over forty years of age.

Pain may occur not only in the region of the greater tuberosity, but in addition for 6 to 10 inches below this region in the middle of the arm laterally; this is the rule and has led many examiners to an incorrect diagnosis. This pain is probably due to spasm of the lower fibers of the deltoid.

Pain of subdeltoid bursitis may be so great as to prevent sleep; sedatives must not be spared. The patient cannot lie on the affected side.

Patients with subacromial or subdeltoid bursitis tend to recover without treatment, although symptoms may persist for five years in some of these cases.

In some cases, the clinical picture is not influenced by the presence or absence of calcium deposits seen by roentgen-ray. *Calcification is not always an indication of the severity or duration of the symptoms;* removal of the deposit soon relieves the symptoms.

Ice caps and cold applications to the shoulder are usually much more effective than heat during the acute stages; heat sometimes increases the pain.

Exercises for the shoulder must be begun as soon as possible after relief of the acute pain to prevent the occurrence of dense adhesions.

One of the best exercises to develop shoulder power, following relief of the acute symptoms, is the swinging of the affected arm anteroposteriorly and also mediolaterally, while in the erect posture with the trunk flexed to about a 30° angle with the perpendicular.

Codman did not favor rupture of these adhesions under anesthesia, or open operation for the release of adhesions. He obtained *best results* in

this condition *if the patient was put to bed and gradual external rotation and abduction of the arm was produced* by applying a splint to the forearm, each end being tied to the head of the bed. He also used morphine, as necessary, and found that within a day the adhesions usually yielded, the tuberosity moved under the acromion and the arm became comfortable. The patient was kept in bed for one to two weeks with the arm abducted until the shoulder motions were free in all directions. During this period he was allowed out of bed each day for swinging exercises (as previously described) while in the stooped position.

RUPTURE OF THE SUPRASPINATUS TENDON

Rupture of the supraspinatus tendon is a frequent cause of disability of the shoulder. Codman believes that complete rupture of the supraspinatus is the commonest cause of prolonged disability from industrial accidents to the shoulder, and that incomplete rupture causes the majority of minor shoulder disabilities. Partial ruptures are more frequent than complete disabling ruptures.[92] Such a complete rupture includes a transverse tear through all of the fibers of the supraspinatus tendon, in addition to the floor of the bursa lying over it, and the roof of the joint capsule on its under surface. This condition is very infrequent in women.

Rupture of the supraspinatus tendon may be produced by muscular strain when the shoulder is raised in abduction or by a direct blow on the shoulder, as from a fall.

Its *symptoms and signs* include:

1. History of *injury* (usually a fall).
2. Sudden *sharp, brief pain in the shoulder*, followed by a relatively pain free interval of hours, and then recurrence of the severe pain at night.
3. *Inability to elevate the arm* (due to lack of supraspinatus function).
4. *Tenderness over the greater tuberosity* of the humerus.
5. Negative roentgen-ray findings.

The sudden pain at the onset of the injury is probably due to the tear of the supraspinatus tendon close to its insertion on the greater tuberosity. Recurrence of pain after a free interval is thought to be caused by distention of the tissues (joint, bursa, etc.) in this region by blood. In some patients, the pain may not be severe and only loss of power of abduction of the shoulder will call the patient's attention to his illness; however, pain is the predominating symptom in most of these cases. Codman has called attention to the presence of a sulcus, an eminence and tenderness at the insertion of the supraspinatus, with a velvety, soft crepitus as the tuberosity disappears under the acromion when the arm is elevated. The sulcus is usually large enough to admit the tip of the finger.

Treatment.—*In every case of complete rupture of the supraspinatus tendon, the treatment is operative,* consisting of suture of the divided tendon.[30,45] Excellent results have been reported.[120] Postoperative treatment includes an axillary pad with the arm in slight abduction; muscular exercise and massage may be begun a few days postoperatively.[119]

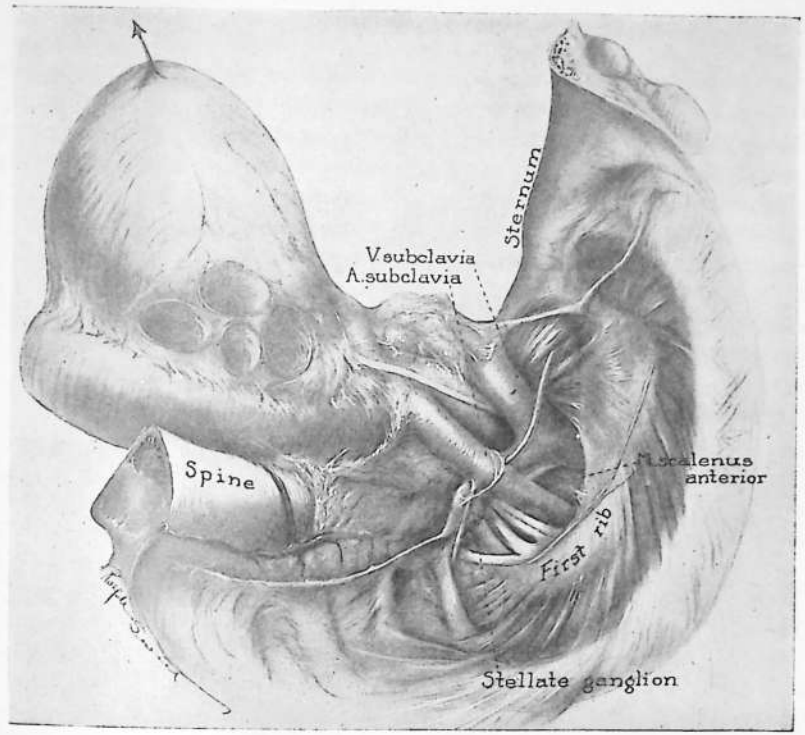

FIG. 234

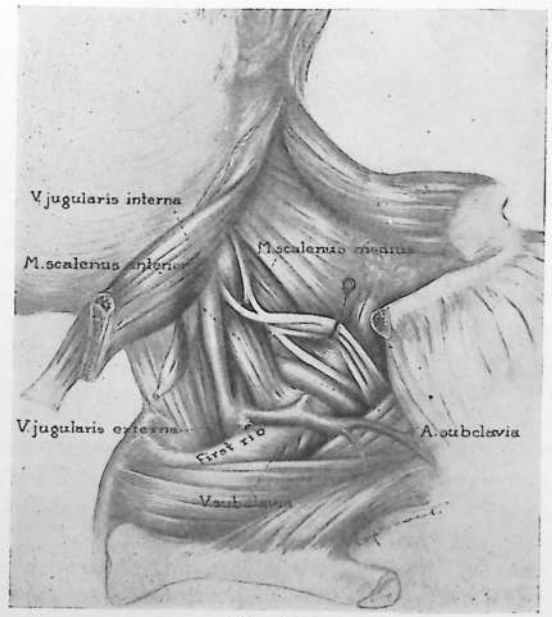

FIG. 235

FIGS. 234 and 235.—Dissection of the neck to show the arrangement of the brachia plexus, subclavian artery and the scalenus anticus muscle. (Naffziger and Grant, courtesy of Surg., Gynec. and Obst.)

Partial ruptures of the supraspinatus tendon may heal without operative intervention. The most common form of *treatment for partial rupture* is fixation of the arm in abduction for five to six weeks by splints or plaster dressing to relax the tendon and approximate the torn ends, in addition to massage and stooping exercises. The patient should not use the arm when standing until he can swing his arms freely (and without pain) in the stooping position. Lateral or anteroposterior pendulum-like motions can be performed without much muscular effort when the patient is in the stooping position; in addition, the weight of the arm assists in stretching any contracted tissue about the joint.

CERVICAL RIB AND SCALENUS ANTICUS SYNDROME

Symptoms caused by a cervical rib or pressure of the scalenus anticus muscle on the brachial plexus have led to confusion in diagnosis of patients with shoulder pain.[16,84,110]

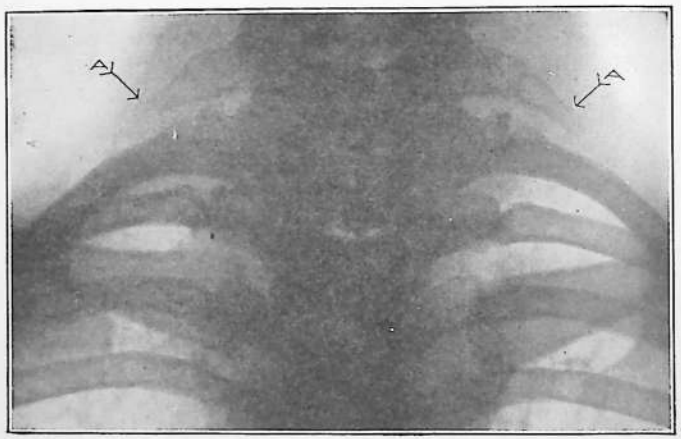

FIG. 236.—Double cervical ribs at *A*. (Holmes and Ruggles.)

Cervical Rib.—This is a fibrous, cartilaginous or bony rib extending outward from any of the cervical vertebra from the second to the seventh. *Cervical ribs* are present in about 1 per cent of all individuals.[50] From 10 to 45 per cent of patients with cervical ribs exhibit symptoms from these.[2]

Palpation of cervical ribs is not an easy procedure in many cases; roentgen examination should always be made if the symptoms of cervical rib syndrome are apparent. Even after the roentgen-ray has demonstrated a cervical rib, palpation is at times difficult.

The Scalenus Anticus Muscle runs from the transverse processes of the third, fourth, fifth and sixth cervical vertebræ downward and outward (at an angle of about 30 degrees with the spine) over the cords of the brachial plexus and the subclavian artery to its insertion on the scalene tubercle of the first rib.

Symptoms Produced by Cervical Ribs or by Spasm of the Scalenus Anticus Muscle.—The cords of the brachial plexus may be irritated by being stretched over the sharp edge of the cervical rib, resulting in a friction neuritis;[62] in the presence of a cervical rib, the scalenus anticus muscle may compress the subclavian artery or brachial plexus against this rib. The lower nerves of the brachial plexus are more commonly involved.[95] As the scalenus anticus muscle is innervated by the brachial plexus, irritation of the brachial plexus may result in a spasm of this muscle, which in turn produces further compression of the plexus, forming a vicious circle. In patients in whom no cervical rib is present, the scalenus anticus muscle may elevate the first rib to an abnormal extent and thus interfere with the brachial plexus and subclavian artery; the symptoms resulting from such an action of the scalenus anticus muscle are identical with those of a cervical rib. Symptoms may also be the result of an abnormally low position of the shoulder,[28] high fixation of the sternum and ribs, low origin of the brachial plexus, lesions of the cervical spine and traumatic or infectious lesions of the shoulder girdle.[47]

Age at Which the Symptoms of Cervical Rib Usually Appear.—The average age of appearance of symptoms is about thirty years.[99] When the shoulder girdle is high (as in childhood), symptoms are less prone to appear. There is a progressive descent of the shoulder girdle which is not complete until adult life. In some individuals, faulty posture may result in a further droop of the shoulder.

Symptoms of Cervical Rib and Scalenus Anticus Syndrome

- Neuralgic pain extending from the neck to the hand (usually on the ulnar side).
- Tiredness or weakness of the extremity.
- Heavy dragging sensation in one or both shoulders or pain over the deltoid extending down the arm and often centering about the elbow.
- Tenderness over the lower third of the scalenus anticus muscle immediately above the clavicle.
- Paresthesis or anesthesia in a similar area; occasionally areas of hyperesthesia.
- Symptoms usually made worse by work and exercise.
- Cramps in the fingers; coldness, numbness or tingling in the hand.
- Occasionally tremor and discoloration of the fingers.
- Decreased amplitude of the radial pulse (or absence of pulse) on the affected side.
- Presence of a bruit over the clavicle.
- Increase in pain by carrying objects with the arm extended, or by downward pressure on the shoulder of the affected side, or by rotation of the head toward the affected side; alleviation of pain on elevating the shoulder by supporting the elbow.
- Increase in the pain when the patient is prone.[31]
- Slight atrophy of the small muscles of the hand in some cases.[122]
- Sensation of a lump at the base of the neck in some patients.
- Little or no impairment of shoulder function.

The pain usually begins in the neck and spreads down the arm as far as the hand; in some cases, the pain may be noticed first in the hand and then radiates upward into the neck and chest. In some patients, the pain radiates up the neck and over the scapula.

In about 4 per cent of the patients with cervical ribs, a dilatation of the subclavian artery occurs just beyond the point of constriction.[53,83] *Pressure on the brachial plexus by cervical ribs or the scalenus anticus muscle may cause pain simulating angina pectoris or coronary artery occlusion.* The pain of brachial plexus pressure may be experienced in the anterior aspect of the chest or precordium. This pain may also be induced by exertion (as in some cases of angina pectoris); however, only certain forms of exertion (such as pulling the shoulder downward or turning the head to one side) will precipitate the brachial plexus pain. The continued carrying of heavy weights on the shoulder (soldiers, hod carriers) may be the exciting cause of symptoms.[85] This pain may not cease as soon as the exertion has been discontinued; however, it may readily be differentiated from coronary occlusion by a careful history, the absence of shock, fever, and the presence of a normal sedimentation rate, heart sounds and electrocardiogram.

Murphy described compression of the brachial plexus in 1910.[82] Ochsner, Gage and DeBakey popularized the scalenus anticus syndrome by their report in 1935; their patients were cured by division of the anterior scalenus muscle.[89] Adson and Coffey had previously (1927) suggested division of the anterior scalenus muscle.[2] In 1938, Naffziger pointed out that a tightened scalenus anticus muscle could cause compression of the subclavian artery and that the latter could cause pain in the shoulder and hand. Freiberg suggested (1938) that spasm of the scalenus anticus muscle might be a common complication of many common lesions about the shoulder girdle.[46] Kaplan believes that *any painful lesion about the shoulder may excite spasm of the scalenus anticus muscle with its characteristic train of symptoms.*[67]

The *etiology of the scalenus anticus syndrome* is compression of the brachial plexus which develops in some individuals because of abnormal development of the shoulder girdle or of the brachial plexus permitting friction or pressure on the brachial plexus and subclavian artery. This pressure or friction irritates some of the fibers supplying the scalenus anticus and scalenus medius muscles, causing abnormal elevation of the first rib. Such elevation results in squeezing the lower trunks of the brachial plexus and subclavian artery. The exciting cause of the symptoms may be trauma to the shoulder region or excessive strain on the shoulder and supraclavicular region, as in hod carriers and soldiers.[85,98]

Compression of the brachial plexus as in the scalenus anticus syndrome results in *pain, numbness and tingling which may extend from the cervical, scapular or pectoral regions to the hand and fingers. The pain is usually dull, gnawing or burning* and is affected by work and posture. Coldness and cyanosis of the fingers are infrequent.

The medial cord of the brachial plexus is usually affected, resulting in pain primarily in the arm between the shoulder joint and elbow following

the sensory distribution of the ulnar nerve.[108] *Scalenus tenderness* is a constant finding. *Symptoms may vary from an excruciating pain to a mild, burning sensation in the shoulder, arm and finger.*[63] Most of these patients complain that their symptoms are *worse* or are severe *at night* while in bed.

Pain in the shoulder may produce spasm and contractures of the muscles of the shoulder girdle; the scalenus anticus muscle often takes part in this spasm. Spasm or marked hypertrophy of the scalenus anticus may cause direct pressure on the brachial plexus or indirect pressure by unduly elevating the first rib (which latter then presses into the brachial plexus and subclavian artery as they arch over it to go down the arm.)

When the shoulder droops, the first rib becomes relatively higher than normal, leading to symptoms. The shoulders are relatively high in children and infants and a gradual descent occurs to the adult position.

Relief is usually obtained by shrugging the affected shoulder or bending the head and neck to the affected side.

Conservative therapeutic measures include keeping the arm above the shoulder with the hand behind the head. Such elevation of the arm and shoulder girdle displaces the brachial plexus upward out of the scalenus triangle and relieves the tension upon the nervous and vascular structures. *Heat* should be applied over the region of the scalenus muscle.

Theis reported that 17 of 20 patients were improved merely by correcting their posture, preventing fatigue and in some instances, immobilizing the arm and shoulder.[111] Russel had reported (1907) a patient whose pain was controlled by merely putting the involved hand on the opposite shoulder (*i. e.*, simple elevation of the shoulder).[102]

Reichert reported relief in 60 patients with scalenus anticus syndrome by medical management alone.[98] This consisted of: (1) elevation of the shoulder by a sling or by holding the hand across the chest in a buttoned shirt or coat; (2) eliminating work which aggravated the symptoms, and (3) *special instructions concerning sleeping habits.*

Three pillows were employed. Number one was placed with one long edge at an angle of 45 degrees to the top of the bed; the second was placed at right angles to the first with one-half of this pillow overlapping the top half of the first. A third pillow was placed horizontally (parallel to the top of the bed) directly on top of the upper ends of both number one and number two pillows. The patient's head then rested upon pillow number three (so that there was a thickness of three pillows under the head); with the patient lying on his back, one shoulder rested on pillow number one and the second on pillow number two. When the patient rested on his side, the shoulder rested upon the mattress between the pillows. These pillows were so arranged as to force the shoulders, head and neck forward when the patient was on his back, and to prevent sinking of the head toward the under shoulder when the patient was on his side.

Many patients with scalenus anticus syndrome have learned that sleeping with the arms over the head relaxes the squeezing mechanism which compresses the brachial plexus between the scalenus anticus and medius muscles and the

first rib; although this position usually affords relief, it is rarely maintained while the patient is asleep.

Gage produced *temporary relief of scalenus spasm by procaine injection into the muscle* and suggested that this might be of importance in identifying the syndrome.[48]

Relief of pain after procaine injection of the scalenus anticus usually appears in two to five minutes, the maximum effect being observed in ten to twenty minutes.[65] If one happens to also inject the cervical sympathetic fibers, redness of the eye and lacrimation may be noticed on the side injected, together with enophthalmos, drooping of the eyelid and constriction of the pupil; this usually disappears within forty-five to ninety minutes.

Technic of Scalenus Anticus Injection[63]

1. Turn the patient's head and chin to the non-affected side.
2. Place the tip of the left middle finger against the outer border of the sternocleidomastoid muscle just above its clavicular attachment.
3. Place the forefinger about ¾ inch away, pushing the clavicular head of the sternomastoid medially at the same time.
4. Make pressure downward behind the clavicle so that these 2 fingers straddle the first rib near the insertion of the scalenus muscle. It may be necessary to alternately flex and extend the head with the free hand to aid in following the course of the scalenus muscles.
5. The two fingers crawl upward along the belly of the scalenus anticus to about 1½ inches above the clavicle.
6. The muscle is held between the two fingers, as before and 1 per cent procaine is injected through a syringe and ½ inch hypodermic needle which may be inserted full length. From 2 to 10 cc. may be used.
7. Do not attempt to infiltrate too deeply. Care must be taken not to infiltrate the phrenic nerve or brachial plexus.
8. The plunger of the syringe must be withdrawn before injecting to be sure that the point of the needle is not in a blood-vessel.
9. The needle is usually pointed almost directly posterior, toward the transverse processes; if contact with the processes is made, the needle is withdrawn slightly before injection is begun.

If necessary, operative section of the scalenus anticus may be performed under local anesthesia through a small supraclavicular incision.

Occasionally, a cervical rib may produce advanced vascular impairment; early surgical intervention will result in anatomical and functional recovery.[105]

Treatment.—Cervical ribs may vary in size from merely an elongation of a transverse process of one of the lower cervical vertebræ to a separate rib several inches long.[115]

The methods of treatment which are available include: Conservative orthopedic measures.

Section of the scalenus anticus muscle.

Removal of the cervical rib.

In many of the patients, symptoms are mild. These individuals may not submit to operative procedures. These patients are often subject to remissions and exacerbations. If the symptoms are not severe, attempts should be made to elevate the shoulder upward and backward by means of a sling, and to develop the trapezius and levator anguli muscles by means of physical therapy. *Corrective exercises and good posture* may be helpful in some patients. Repeated injections of procaine may give prolonged or even lasting relief; these should not be performed too often as they may lead to scar tissue formation or possibly damage to the sympathetic nerves from too deep an injection.

If more radical treatment is required, the choice is *section of the scalenus anticus muscle;* this has produced favorable results.[44] Tenotomy of the scalenus anticus muscle has replaced the older operation of resection of the cervical ribs in many instances.[1,57,68] The operation of tenotomy is much safer, gives better results than rib resection and is not accompanied by unfavorable postoperative sequelæ. In some cases, however, in which there is evident pressure from the cervical rib or its tendinous attachment, resection of this rib and its attachment may be necessary. When large accessory ribs (5 cm. or more in length) spring from the seventh cervical vertebra, section of the scalenus anticus and resection of the rib are usually necessary. If cases are carefully selected, operative results are usually good. Care must be taken in the selection of cases that this syndrome be differentiated from other conditions (such as subdeltoid bursitis, arthritis of the cervical spine or shoulder, neuritis, peripheral vascular disorders, cardiac disease, congenital deformities, apical pulmonary tumors, etc.).

SHOULDER LESIONS IN BASEBALL PITCHERS

Bennett described the lesions which may occur in the shoulder and elbow in professional baseball pitchers. He emphasized that the pitcher is subject to all the disabilities of the shoulder to which the average person is, such as subacromial and subdeltoid bursitis, irritation of the supraspinatus, irritation of the biceps tendon, traumatic synovitis and the inflammatory diseases.[10]

In the pitcher, the changes are most often seen in the *anterior group of disorders, i. e.,* inflammatory and traumatic lesions of the supraspinatus tendon, the subacrominal bursa, the subdeltoid bursa, the biceps tendon and the coracoid bursa. With rotation of the head of the humerus, the supraspinatus tendon may be rubbed against the acromion producing traumatic irritation or fraying of the tendon. The biceps tendon may be injured by the same motion. Trauma between the head of the humerus and the coracoid process may produce lesions of the coracoid region. The soft tissues between the head of the humerus and the coracoid process are traumatized at the finish of the follow through when the head of the humerus is pressed against the coracoid process. The supraspinatus is irritated by the overhand pitcher while the coracoid bursa is irritated by the sidearm pitcher.

The anterior group of lesions produces sensitiveness about the point of

insertion of the supraspinatus and discomfort on abduction of the arm. If there is involvement of the biceps tendon, one notes sensitiveness in the region of the bicipital groove and discomfort when the forearm is flexed against resistance. Crepitus may be elicited on active motion if these tendons are frayed by trauma.

In the simple traumatic group, rest and local heat form the basis of treatment; most of these traumatic injuries respond to conservative care. If the tendons become frayed, the biceps may respond to surgical treatment but one could not expect a reconstructed supraspinatus tendon to perform the accurate and strenuous function required in a professional pitcher.

Bennett believes that infected teeth or tonsils may cause or aggravate a sore shoulder.

Lesions in the posterior part of the shoulder shorten the career of the professional pitcher. Due to excessive use of the arm and marked pull on the posterior capsule and triceps tendon, *a deposit of bone may develop on the posterior inferior border of the glenoid fossa.* This deposit may irritate the capsule and synovial membrane as well as the circumflex nerve producing referred pain to the deltoid region. Symptoms of these lesions include local discomfort and sensitiveness in the posterior portion of the shoulder, in addition to pain referred to the deltoid.[10] With such a lesion, the pitcher cannot throw hard after an inning or two and must leave the game. This lesion does not cause discomfort during normal motion of the shoulder or even on throwing at moderate speed. Although it is possible to remove this exostosis, it is questionable whether this should be done as it does not cause symptoms except on hard throwing, and it is doubtful whether the professional pitcher can resume his occupation after such an operation.

LESS COMMON CAUSES OF PAINFUL SHOULDERS

These include malingering, hysteria, calcified lymph nodes in the neck, tumors in the shoulder region, including superior pulmonary sulcus tumor,[86,87] arthritis (rheumatoid, osteo-, tuberculous, syphilitic), fractures and dislocations about the shoulder, referred pain (from angina pectoris, aortitis, aneurysm, coronary occlusion, pneumonia, pleurisy, pneumothorax, pulmonary neoplasm, gall bladder disease, cervical Pott's disease, herpes zoster, etc.), tumors of the spinal cord and nerves, peripheral neuritis, discogenetic disease, etc. Shoulder pain arising from *arthritis of the cervical spine* is not uncommon and has been discussed in the section dealing with arthritis of the spine. *Calcified lymph nodes* in the neck may produce shoulder pain by irritation of the brachial plexus (mechanically or by actual inflammatory spread).

One commonly sees persistent *pain in the shoulder region as an aftermath of myocardial infarction* or in patients with severe and frequent attacks of angina pectoris.[4,12,38,40,60,76,79]

Askey reviewed the syndrome of painful disability of the shoulder and hand following coronary occlusion, and described 7 cases.[4] Kehl added 6 cases of Dupuytren's contracture as a sequel of coronary occlusion.[69]

This pain is usually referred to the left shoulder, may persist for weeks or months and increases with motion of the arm. It is possible that the symptoms may develop from relative disuse of the shoulder and increased muscular tension in this region.

The changes which occur in the hands and shoulders following myocardial infarction in some patients may be mistaken for rheumatoid arthritis, periarthritis of the shoulder, etc.

Chronic arthritis of the shoulder joint is uncommon.[73] There is usually an associated arthritis of other joints. *Symptoms* include: Dull pain in the shoulder increased by motion.

Limitation of motion in all directions (especially abduction and external rotation).

Early and progressive muscular atrophy.

Crepitus over the shoulder (in many cases).

Absence of palpable swelling or tenderness (except in severe acromioclavicular involvement).

Roentgen changes in later stages.

Therapeutic aids in chronic arthritis of the shoulder: Immobilize for one to three weeks in a metal splint, Castex or plaster cast with the arm in 90 degrees of *abduction*. If the patient remains in bed, sandbags and pillows may be used to maintain the arm in abduction and external rotation.

Remove the splints several times each day for application of *heat* (such as baking, infra-red radiation, etc.); this decreases muscular spasm and pain.

With the subsidence of muscular spasm and painful motion (in from one to three weeks), use *gentle massage* and active *exercises* to prevent contractures (especially in the adductor muscles) and to lessen muscular atrophy. It may be advisable to re-apply the splint during the night for several months even when acute symptoms have disappeared.

If contractures have formed, attempt to increase abduction of the arm by the use of pillows between the arm and body or by splints. Correct any faulty body mechanics.

Manipulation under anesthesia may be necessary in severe contractures, when the process is inactive. Never use great force. Attempt to correct abduction and external rotation. Splinting is required following this until the acute pain has subsided. Active exercises are then instituted.

When **acromioclavicular separation** is suspected, roentgenograms should be taken with the arm in a position of abduction and another in adduction; pictures should be taken while the patient is standing as this procedure may reveal separation when other methods fail. Acromioclavicular separation is not uncommon. A true arthritis of this joint may also occur producing a roentgen picture resembling rheumatoid arthritis or degenerative joint disease with narrowing of the joint space.[90]

Coracoclavicular articulations are not rare. In 1000 roentgenograms of unselected shoulders, 12 showed these joints, being bilateral in 6. There seemed to be no anatomic or physiologic significance of these joints. The joint is formed by a large clavicular bony process, with a facet conforming

to the adjacent end of the coracoid. The clavicular bony outgrowth is present at the site of the tuberosity for the attachment of the coracoid ligament.[88]

Thinning of the intervertebral disks (*discogenetic disease*) is common in the cervical spine in older patients. This causes narrowing of the corresponding intervertebral foramina with compression of nerve roots.[54] Symptoms resulting from *discogenetic disease of the cervical spine* include pain in the shoulder or over the precordium and muscular weakness of the upper extremities.[91] This condition is discussed in the chapter on Spondylitis. For the physician who desires more detailed information concerning the painful shoulder, one may consult the very interesting book of Codman upon this subject.

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CHAPTER LIV

DUPUYTREN'S CONTRACTURE

In 1832 Dupuytren, a French surgeon, described a pathologic condition consisting of contracture of the palmar fascia, associated flexion contractures of the fingers and marked loss of function.[8] Dupuytren attempted to straighten the fingers by extension, application of ointments and by massage, but without avail. Reviews of the literature are available.[4,6,12,15,30]

Etiology.—The *etiology* of this condition is still *unknown*. Factors which have been incriminated include heredity,[19] senility, acute or chronic trauma,[26] fibroblastic diathesis or predisposition, and many others of extremely dubious significance such as toxins from focal infection, circulatory stasis, chronic inflammatory processes, diabetes mellitus, arthritis, gout, embryologic malformations, peripheral or central neurologic disturbances, endocrinopathies, etc.[11,17,18]

Dupuytren's contracture has been noted in several generations in the same family and a high familial incidence has been noted in large series of cases.[27,30,31]

More recently, Dupuytren's contracture has been reported *following myocardial infarction*.[3,13,16,20]

Nippert suggested that Dupuytren's contracture developed because of an increase in sympathetic tone.[2,7] Hale Powers placed Dupuytren's contracture in the same category as scleroderma, hypertrophic pulmonary osteo-arthropathy and other trophic disturbances and explained the palmar phenomenon on the basis of irritation and hyperexcitability of the sympathetics due to visceral disease, which may often be intrathoracic.[30] Visceral disease may precipitate peripheral dystrophies through irritation of the sympathetic ganglia. Some cases of Dupuytren's contracture are associated with Raynaud's disease.

The greatest incidence of the disorder is in middle life.[10,14] *Males* are affected from *five to ten times as commonly as females*.

Although many of these patients give a history of hard labor, it does not seem to be related to occupation or hard labor in most instances.

Pathology.—Normally, the palmar fascia (or aponeurosis) is extremely important anatomically as it forms (together with the transverse carpal ligament and the fibrous digital sheaths which are attached to it) a continuous aponeurosis which aids the efficiency of the flexor tendon, counteracts local pressure and protects the tendons, nerves and vessels over which it stretches. It is a thick triangular membrane with its apex at the transverse carpal ligament; it separates distally into four thickened bundles, one of which goes to each digit. There are many transverse fasciculi connecting these bundles with similar fasciculi communicating with the overlying skin and beneath with the volar interosseous membrane, the digital

fascial sheath, the metacarpal bones, phalanges and the interphalangeal joints.

Because of such wide distribution of its fibers, contracture of this fascia will lead to flexion contractures of the fingers with marked functional impairment of the whole hand.[2]

On the basis of studies of microscopic sections of the palmar fascia in 57 cases of Dupuytren's contracture, Meyerding and his associates believed that the pathologic picture was explained best on a basis of a chronic inflammatory process. They pointed out the tragic danger of confusing Dupuytren's contracture, which is a benign process, with fibrosarcoma.

Horwitz studied the histology of the tissue removed in 35 cases of Dupuytren's contracture. Many writers have extensively reviewed this subject.[6,9,12,25,28] Most of these writers believe the essential process to be a proliferation or hyperplasia of connective tissue with transitions from the early features of a cellular tissue to the final stage of scarring. Some individuals have interpreted the process as a focal hypertrophy of connective tissue arising in the walls of the blood-vessels while others, without convincing evidence, have described it as a chronic inflammatory process. Meyerding and his associates stressed the importance of chronic inflammatory changes in the skin, subcutaneous tissue and interstitial connective tissue together with the fibroblastic process involving the palmar fascia.[21,22,25] Horwitz concluded that the essential process was benign *fibroplasia of the palmar connective tissues.*[11] In his experience, inflammatory features were not constant and there was no evidence that the proliferative process arose from the walls of blood-vessels. The clinical behavior and histologic features of the fibroblastic process resembled those of other localized fibroplasias, as keloid and fascial desmoid. Keloid is localized overgrowth of hyaline connective tissue developing in the skin of the predisposed person; this usually occurs in a scar although it may develop spontaneously. Desmoids are fibromas of the abdominal wall; some of these are congenital while many others arise in the scars of uncomplicated laparotomy. Dupuytren's contracture, keloid and fascial desmoids are prone to recur in some cases despite extensive surgical excision.

Symptoms.—Males are affected much more frequently than females. One or both hands may be affected.[10] The *order of frequency* of involvement of the fingers is ring finger, little finger, middle finger, index finger and thumb—the *ring finger being affected most often.* When several fingers are involved, the involvement may not occur at the same time and may not progress to the same extent.[32]

Very few of these patients have pain or local sensitivity so that the symptoms are often subjective. In early cases, the condition is diagnosed on routine physical examination, without the patient's knowledge of such involvement.

In Horwitz's 35 patients, 3 cases appeared from thirty to thirty-nine years of age; 12 cases from forty to forty-nine years; 14 cases from fifty to fifty-nine years, and 6 cases from sixty to sixty-nine years. The youngest of these patients was thirty years of age; the deformity in this individual had been present for four years. Thirty-three of the 35 patients were males. Eighteen of the 35 were engaged in occupations where repeated trauma to the involved hand could occur. Nineteen of the patients had bilateral involvement. Associated diseases included arthritis in 2 cases, diabetes mellitus in 1 and gout in 1.

Signs of Dupuytren's Contracture

Development of a *nodule on the palm of the hand* or fingers.
Most commonly affected region: palmar aspect of the metacarpophalangeal joint of the ring finger.
Puckering of the palmar skin, most frequently at the base of the fourth and fifth fingers.
Further nodules and funnel-like depressions of the palm occur with more induration and creasing of the skin and atrophy of the subcutaneous tissues.
Appearance of a *thickened band* from the concavity of the palm to the proximal interphalangeal joint of the involved finger.
In some cases, there may be definite tenderness, swelling and local redness, but this is very uncommon.
Progressive involvement of the skin and fingers.
Gradual and varying degrees of *contractures of the fingers.*
Limitation of motion of the fingers to varying degrees by the contracted fascial bands.
Months or years later, *flexion contracture* of the affected finger appears.
The progress of the lesion may be arrested in some cases.
The condition may progress to involvement of the greater portion of the palmar fascia and all of the fingers, leading to marked flexion contracture of the fingers.

Treatment.—To date, the *only relief of advanced cases of Dupuytren's contracture is by adequate surgery.*[7,33] Uniformly *good results will be obtained by complete and precise excision of the palmar fascia.* Unfortunately some physicians do not realize that careful surgery will restore the function of the hands in these individuals.

Adams employs a transverse incision across the hand in the distal flexion crease, following which the skin flaps are carefully raised from the adherent contracted fascia beneath this.[1,2] This *raising of the skin flap is one of the most difficult stages of the operation* because the fingers are often flexed into the palm and one must take great care to avoid trauma to the delicate skin flap. The skin is freed distally to the base of the fingers and proximally to the base of the palm. One may observe that the contracted fascia becomes involved with the vascular bundles and digital nerves at the base of the fingers. The fascia should be dissected free at its more proximal normal end, where it is cut across and turned up so that the digital nerves and vessels can be identified, permitting careful elevation of the fascia. The digital and palmar ramifications of the fascia can be excised without damage to vessels or nerves.

All vessels must be clamped and tied as they are encountered as healing of the wound will not occur if a hematoma forms under the skin flaps. *Strict*

asepsis and a bloodless field are extremely important. Care is taken to sterilize the skin for twenty-four to forty-eight hours preoperatively and the operation is performed under general anesthesia with a blood pressure cuff on the upper arm as a tourniquet.

In extensively involved hands, if the skin over the palm and the base of the fingers is hopelessly involved, this should be excised and the defect covered by a primary full thickness graft which may be obtained from the inner aspect of the upper arm.

The *postoperative care* of these patients is very important. Limited motion of the fingers should be encouraged from the first day of operation. As soon as wound healing permits, physical therapy and active use of the hands are instituted. One must not remove the skin sutures too early to avoid separation and delayed healing; these are usually removed after two or three weeks.

During the operation, one should use fine hemostats and forceps, retractors with blunt edges and moist sponges.[23] Meyerding advises the use of a posterior aluminum splint (well padded with gauze) to hold the involved fingers in extension. Dressings are changed at the end of the first week postoperatively. During this first week, he employs radiant heat to the palmar surface of the hand and fingers for thirty minutes each day, following which passive movements of the fingers are carried out for five minutes, the movements being sufficiently gentle to avoid opening the skin wound. When the skin incision is well healed (by the end of the first week), the hand and forearm are placed in a whirlpool bath (with water at 110° F.) for thirty minutes daily—an ideal preparation for massage and manipulation. Active assisted exercise is then instituted. The patient is then given flexion and extension exercises for the fingers and is warned against repeated deep trauma to the hand.

Conservative care, such as stretching, heat and massage, ointments and irradiation has not prevented or cured advanced Dupuytren's contracture. *Surgical results are satisfactory in about 90 per cent of cases* (with excision of the fascia, temporary splinting and physical therapy).[5,24,29] In some early cases, it is possible to perform a subcutaneous section of the constricting bands of palmar fascia.

Trophic changes with deformities of the fingers and hands as a result of local ischemia are well known sequelæ of vasoconstriction from splinting of injured hands and probably from overactivity of the sympathetic nervous system. Johnson noted the appearance of disabling changes in the hands resembling sclerodactylia *following myocardial infarction* in 39 of 178 (21.8 per cent) consecutive cases.[13]

Pain and stiffness of the fingers were the first symptoms which attracted the attention of the patient; these appeared from three to sixteen weeks after the acute myocardial infarction. There then appeared a firm, bilateral, uniform, symmetrical swelling of the entire hands including the fingers; this swelling did not pit on pressure. The skin became tight and smooth and the normal wrinkles in the skin were less deep or entirely obliterated;

this was especially noticeable in the transverse wrinkles over the dorsum of the fingers. The color of the hands varied from an erythema to a cyanosis. The fingers and hands became cold but no sensory changes were noted. The volume of the pulse in the radial and brachial arteries was not significantly altered although these vessels were thickened and sclerotic to varying degrees.

Manipulation of the fingers produced pain; the fingers could not be fully flexed or extended. There was no joint effusion or crepitation.

The swelling of the hands and fingers subsided in time but the stiffness and pain in the fingers persisted. The thin, glossy skin later became dull and thickened and was bronzed in some cases. The soft tissues over the shafts of the phalanges became shrunken so that the bony prominences and joints of the hands and fingers became more apparent. Movements of the fingers were painful and limited.

Some of the patients developed marked contractures of the fingers, but others had few residual deformities and little decrease in motion. In the severe cases, the hands felt stiff, wooden and withered with a loss of flexibility and softness, justifying the term "hard fingers" or sclerodactylia. No ulcers or gangrenous areas developed. The elbows and wrists were not involved in the process and there appeared no changes in other joints, except the shoulders. These changes in the fingers and hands following myocardial infarction resembled those of sclerodactylia (of scleroderma) and Raynaud's disease.

In 38 of the 39 cases, pain occurred in both hands; 34 patients had pain in one or both shoulders. All but 4 patients showed atrophy of the hands and all but 2 had limitation of motion of the fingers. *Twenty-two of the patients developed contracture of the palmar fascia.* The same number had residual stiffness of the shoulders.

There is no obliteration of the joint spaces and no bony proliferation. The interphalangeal surfaces remain smooth. Marked decalcification of bone may occur in the phalanges.

Johnson believes that the pain of coronary occlusion may produce vasoconstriction of the peripheral arteries either directly through the cord (reflex arc) or indirectly by adrenalin stimulation of the higher centers.

Johnson explained the disabling changes in the hands after myocardial infarction as due to nutritional disturbances (especially anoxia) which may result from local vasoconstriction and anoxemia in the hands and fingers. The vasoconstriction, which results from the cardiac pain, is relatively temporary and intermittent so that the condition does not go on to trophic ulcers or gangrene. The vasoconstriction involves only the peripheral arteries of the hands, with preëxisting arteriosclerotic narrowing of these vessels.

Wright has discussed the nerve paths between the hand and the heart. Pain impulses originating in the heart pass by sensory nerve fibers to the eighth cervical and upper four thoracic nerve roots. The pain is then referred down the inside of the arms (first thoracic) and to the sides of the

chest (first, second and third thoracic). Rami from these segments of the cord connect with the upper thoracic and lower cervical sympathetic ganglia which furnish vasoconstrictor fibers to the upper extremity.[34]

Kehl reported 6 cases of *Dupuytren's contracture occurring after myocardial infarction;* he suggested that this was due to irritation of the sympathetics, and thought that the changes were in the same category as scleroderma, pulmonary hypertrophic osteo-arthropathy and other trophic disturbances.

Johnson suggested the name "post-infarction sclerodactylia" and concluded that it was produced mainly by ischemia resulting from reflex vasoconstriction of the arteries of the hand induced by cardiac pain, plus the lesser ischemic effects of arteriosclerosis of these arteries.

Changes in the palms were not noted in any instance before the onset of the cardiac difficulty. The *palmar changes appeared typical of Dupuytren's contracture,* beginning usually as a firm nodule in the palmar aponeurosis, most frequently in the ulnar area near the metacarpophalangeal joint, and progressing slowly. It extended gradually to involve the whole palmar fascia, excluding the tendons. The overlying skin appeared hard, folded, thickened and closely adherent because of the fibrosis and contracture of the underlying fascia and the loss of the subcutaneous fat. The contracture often became bilateral although it did not appear simultaneously in both hands. In the advanced stage, permanent flexion of one or more fingers resulted.

In 5 of Kehl's 6 cases, other symptoms in the hands included pain, stiffness, numbness, tingling, coldness and livid discoloration.[16] Palmar changes were noted from three to eleven months following the coronary accident. Four of these patients also had shoulder disability, two of these complaining of severe bilateral shoulder pain.

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CHAPTER LV

PAINFUL FEET

It is unfortunate that many physicians have had little training in the management of disabilities of the feet. For this reason, patients frequently wander from physician to physician, and even from orthopedist to orthopedist as well as from one shoe concern to another while seeking relief for their foot problems. *The treatment of foot ills should be represented by at least as many specialists in the medical profession as the care of the heart, or of the gastrointestinal tract.* Although most foot problems could be dealt with adequately by properly trained general practitioners, a small percentage of these will require specialized care. *It is hoped that in the very near future physicians will acquire special knowledge of the foot and its disabilities, and not relegate this phase of medical care to the shoe salesman.* The latter would willingly relinquish responsibility which should properly belong to the physician.

Medical schools, in general, do not devote proper time or clinical facilities for the teaching of the common ailments of the feet. Orthopedic specialists will occasionally prescribe orthopedic oxfords and arch supports without removing the patient's shoes. Such a situation is deplorable, but certainly does exist.

It is extremely enlightening to spend several hours at an orthopedic shoe shop listening to the patients' complaints, and to the clerks' advice. Frequently, these individuals confide to the shoe salesman that they have seen as many as five or six orthopedists within the past six months in an effort to relieve their painful feet. Some of these individuals had had the benefit of careful examinations; others had hurriedly been given a prescription for shoes and supports. In some instances, the orthopedist had accurately measured the foot and had definitely designated the position of the support, and its height. Prescriptions differed markedly from various orthopedists for the same individual. From the wording of many prescriptions, it was apparent that some physicians had no intimate knowledge of lasts, shoes or shoe construction.

The practicing physician can properly care for 90 per cent of patients with painful feet.[15] Most physicians, however, have little knowledge of the cause and correction of pain in the foot. Patients wander from physicians because of the latter's lack of understanding of a few symptoms and simple methods of correction. **In some acute cases, rest in bed, felt pads, adhesive strapping, simple exercises for the feet, attention to proper posture and gait, correct shoeing, and hot and cold foot baths may work wonders.** The use of a simple arch prop, as a temporary splint, during the period of rest in bed for any illness, may lessen post-illness disability of the feet. Patients who do not respond to procedures available to the general practitioner should be referred for further detailed management.

Predisposing Causes of Foot Strain[27,43,80]
Excessive standing, walking or running especially in individuals unaccustomed to these; change from a sedentary occupation to one involving additional hours of standing or walking.
Prolonged rest in bed (producing atrophy and decreased tone of muscles).
Obesity (or marked increase in weight, as in pregnancy).
Improper shoes and stockings (cramping the toes and preventing normal muscular function).
Poor posture, especially in standing and walking.
Knock knee (genu valgum), coxa vara, congenital dislocation of the hip, scoliosis, etc.

The etiology of pain in the foot may vary with the age of the individual: in the *young child*, weak, pronated feet may result from faulty use or from an associated deformity such as genu valgum, disturbing the line of weight upon the knee.[21] Foot strain in *older children* may be caused by excessive exercise or occupations; in *middle age*, obesity (especially if there is a rapid increase in weight), vocational disorders, arthritis or faulty shoes play an important rôle. **Vascular disease is a common cause of pain in the foot in the aged.** Prolonged *standing, carrying heavy objects, and walking over hard pavements or irregular ground for many hours may all lead to pain in the foot.*

Systemic diseases which must be considered in the etiology of pain in the foot include diabetes mellitus, arteriosclerosis, intermittent claudication, deficiency of vitamin B_1, arthritis (tuberculous, gonorrheal, rheumatoid, etc.), syphilis, rheumatic fever, gout, Raynaud's disease, Buerger's disease, poliomyelitis, spastic paralysis, erythromelalgia, cardiac and nephritic disease. *Pain in the sole of the foot may be caused*[64] also by: deformities; injuries; neoplasms; contractions; scar tissue; absorption of fat from pressure or after prolonged illness; and plantar neuralgia.

Circulatory disorders of the foot which may present cutaneous manifestations include:[63] obliterating arteriosclerosis, thrombo-angiitis obliterans, acrocyanosis, Raynaud's syndrome, erythromelalgia, immersion foot and vascular anomalies (such as port wine or capillary angioma, spider nevus, small cavernous angiomas, lymphangiomas and diffuse lymphangiomatosis of the leg and foot).

NORMAL PHYSIOLOGY OF THE FEET[62,75,77]

The center of gravity of the body is normally balanced over the triangular weight-bearing area of the foot, with its apex posteriorly. This weight is divided equally between the heads of the metatarsal bones and the tuberosity of the os calcis. The anterior shift of the center of gravity which accompanies poor posture disturbs the muscle balance in the foot, and causes a strain on one group of these, leading to what is known as the weak foot. Physicians desiring detailed anatomic or physiologic data on this subject are referred to the works of Dickson and Diveley,[16] Morton, Lake, Lewin,[46] Houser[32] or Schuster.

The foot is in part like a spring, composed of a bony framework with arches that are maintained by the physiologic tone of muscles, ligaments and fascia. The whole weight of the body in walking and standing comes down on an elastic arch composed of small bones joined by ligaments and connected by muscles;[48] unfortunately this complicated structure is not left free to perform its function, but is encased from childhood in a dis-

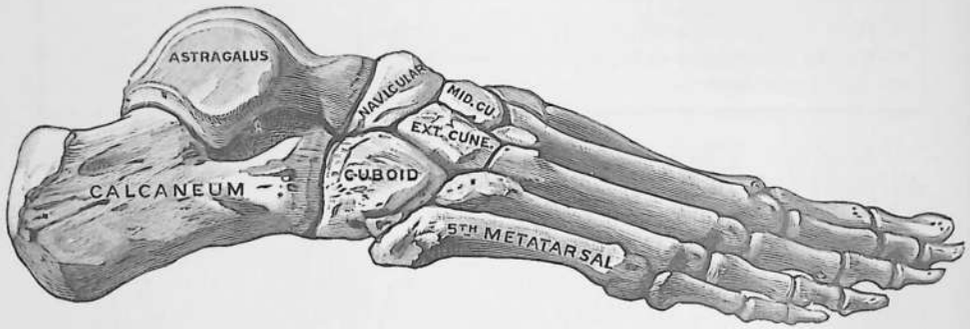

FIG. 237.—The bones of the right foot, viewed from the outer side. (Gerrish's Anatomy.)

torting leather covering which may compress and deform the forefoot and weaken its muscles. *Inasmuch as the shape of the shoe very seldom corresponds to the shape of the adult foot, the shoe is one of the most important factors contributing to foot strain.*

Physicians commonly speak of a *longitudinal* and a *transverse arch* of each foot. The **longitudinal arch** runs anteroposteriorly along the inner border of the foot from the tuberosity of the os calcis to the heads of the metatarsals, centering about the inner three metatarsals, cuneiforms,

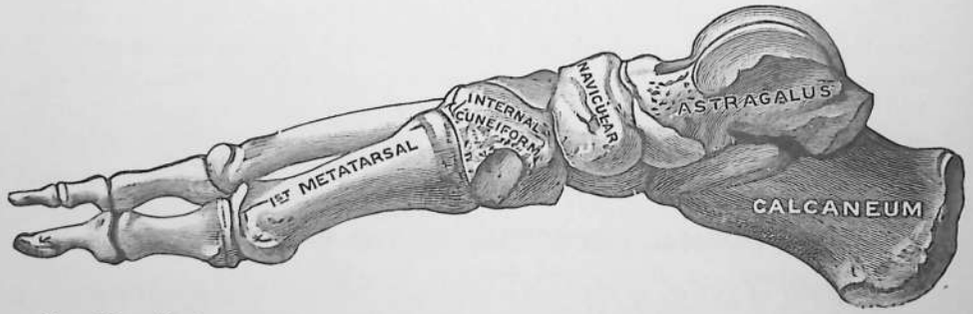

FIG. 238.—The bones of the right foot, viewed from the inner side. (Gerrish's Anatomy.)

scaphoid, astragalus and the calcaneus. The medial portion of the longitudinal arch rests upon the os calcis and the head of the first metatarsal. The **anterior transverse metatarsal arch** passes through the necks of the second, third, and fourth, and the heads of the first and fifth metatarsals. This becomes flattened on weight bearing, but returns to its arched position when the weight is removed. All of the heads of the metatarsals rest

upon the ground in weight bearing so that no transverse arch is present at that time.

Bruce and Walmsley examined a series of sections and dissections in fetal and adult feet.[10] In all of the fetuses, there was a longitudinal arch, and also a transverse arch in the region of the bases of the metacarpals; there was no evidence to suggest the presence of a transverse arch in the region of the metatarsal heads in either fetal or adult feet. Morton believes there is no anterior transverse metatarsal arch formed by the anterior ends of the metatarsal bones, and that even if one were present, there are no muscles or ligaments in that portion of the foot which are able to sustain such an arch.[61] The crown or keystone of the longitudinal arch is formed by the head of the astragalus; the inner edge of the second metatarsal is said to form the crown of the disputed anterior arch.

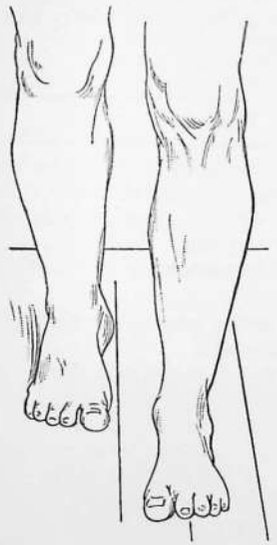

FIG. 239.—Illustrating the automatic adduction of the forefoot on the right side, due to the obliquity of the bearing surface of the metatarsus, in the proper attitude for walking. (Whitman.)

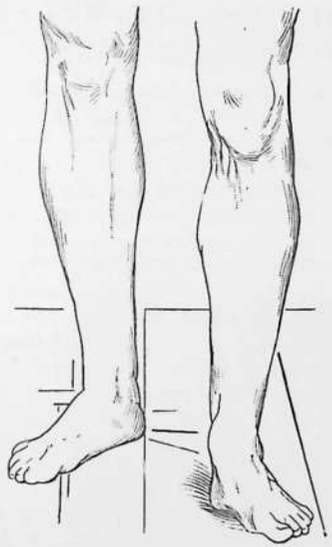

FIG. 240.—The improper attitude of outward rotation of the limbs usually accompanied by eversion of the feet, in which there is disuse of the leverage function. (Whitman.)

The *ligaments which aid in maintaining the integrity of the longitudinal arch include the "spring" ligament* (or inferior calcaneoscaphoid), which is the main ligamentous support on the inner side, and the long and short calcaneocuboid or plantar ligaments (supporting the middle and outer part).

The *plantar fascia* is more superficial than these ligaments and acts as an important anteroposterior support of the longitudinal arch. This is a strong band of fibrous tissue.

The *main muscular support on the inner side of the foot* consists of the *tibialis anticus and posticus* and the flexor longus hallucis. The first of

these runs from the outer side of the lower leg to its attachment to the internal cuneiform and base of the first metatarsal bone. These muscles aid in supporting the inner side of the arch and adduct the forefoot in addition to their other actions.[73]

Lake has emphasized that the longitudinal arch is an architectural feature of the foot which does not depend upon muscular tonus;[43] *the arch is not obliterated even when all muscular activity is abolished.* He has shown that the *form of the arch is normal even during weight bearing with the patient under spinal anesthesia,* during which tonus is abolished. Division of all of the tendons to the foot in a freshly amputated leg does not reduce the arch, even when loads equal to one-half of the patient's weight are placed on the articular surface of the tibia. If, however, the inferior calcaneoscaphoid (spring) ligament is cut, there is an immediate drop of about half an inch in the arch. Another similar drop occurs when the plantar fascia is divided, and there is a complete drop of the arch when the long and short plantar ligaments are divided.

Notes on Physiology of the Feet

Main functions of the feet: (*a*) A base for erect standing; (*b*) levers for walking, running and jumping. Heel: primarily receives and secondarily supports weight; mid-foot: supports weight; forefoot: propels body forward primarily; supports weight secondarily.

Body weight in standing is transmitted equally backward to the heel and forward to the metatarsal bones.

The forefoot propels the body forward free from strain most efficiently when the heel has left the ground before the forefoot touches it.

There is no standard height for a longitudinal arch; this varies with different individuals and races.

The longitudinal arch serves somewhat as a shock absorbing agency.

The height of the longitudinal arch does not necessarily indicate its usefulness.

The skin is thick over the heel, outer border of the sole, pads under the heads of the metatarsals and over the pulp of the toes, suggesting that these portions are the weight-bearing parts of the foot.[52] This is true except that the toes themselves do not support any of the weight of the body.

Stress is borne by metatarsal bones in ratio of 2, 1, 1, 1, 1 (beginning with the great toe); in normal standing, the inner two and the outer three metatarsals support an equal stress.

The heads of the metatarsals act as the fulcrum upon which the body weight is lifted in walking.

The distal extremities (heads) of the first and second metatarsals are normally equally distant from the heel.

In walking, weight is borne first on the heel, then on the fifth metatarsal and finally on the first metatarsal; **the line of weight bearing normally passes through the center of the knee and ankle joints and over the inner border of the second toe.**

In running, stress is borne chiefly by the first and second metatarsals.

Any defect of the first metatarsal segment may impair the mechanism of the foot.

The normal range of motion of the ankle is as follows: Dorsal flexion—10° to 20° less than a right angle; plantar flexion—50° to 60° more than a right angle.

Abduction and *adduction* are the principal movements permitted at the mid-tarsal joints while *inversion* and *eversion* are allowed at the subastragaloid joint. Eversion and abduction usually occur together as do inversion and adduction. Other movements in the foot are normally prevented by the ligaments, *i. e.*, the *spring ligament* (inferior calcaneoscaphoid); *interosseous ligament* (calcaneoastragaloid); and *deltoid* (internal lateral ligament).

Definitions

Adduction: Inward position of the forefoot in relation to the mid-line of the limb.

Abduction: Outward position of the forefoot in relation to the mid-line of the limb.

Inversion: Turning in of the sole of the foot.

Eversion: Turning out of the sole of the foot. Practically, inversion and adduction are combined and eversion and abduction are combined.

Valgus: Deformity of the foot in a position of abduction and eversion.

Varus: Deformity of the foot in a position of adduction and inversion.

Supination: Same as inversion.

Pronation: Same as eversion.

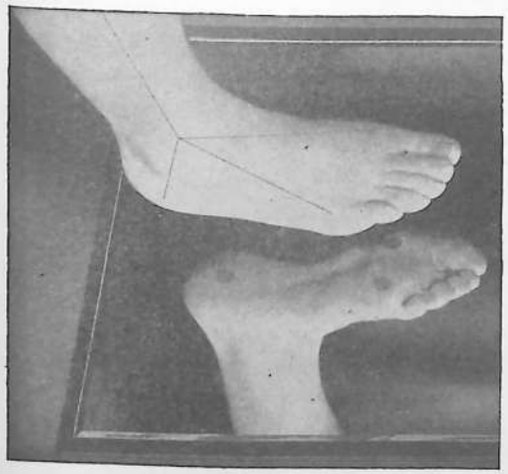

FIG. 241.—Distribution of weight to the foot. (Geo. E. Keith Company.)

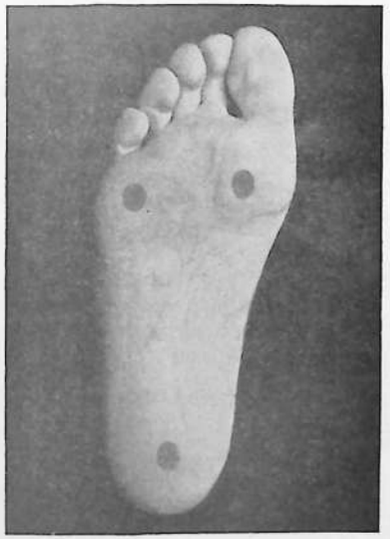

FIG. 242.—Weight-bearing points of the foot. (Geo. E. Keith Company.)

The muscles are important structures in the foot. The plantar flexors are approximately five times as strong as the dorsal flexors and the gastrocnemius and soleus are three times as powerful as the remainder of the foot

muscles combined.[97] *The main movements of the foot are carried out by the tibialis anticus and posticus and the peronei.* The tibialis anticus elevates and the peroneus longus depresses the first metatarsal. The tibialis posticus produces inversion and adduction; the peronei permit eversion and abduction. *The peronei are the main evertors of the foot,* aided by the peroneus tertius and the extensor longus digitorum. *Inversion is due chiefly* to the effects of the tibialis anticus and posticus, and the flexor longus digitorum and flexor longus hallucis.

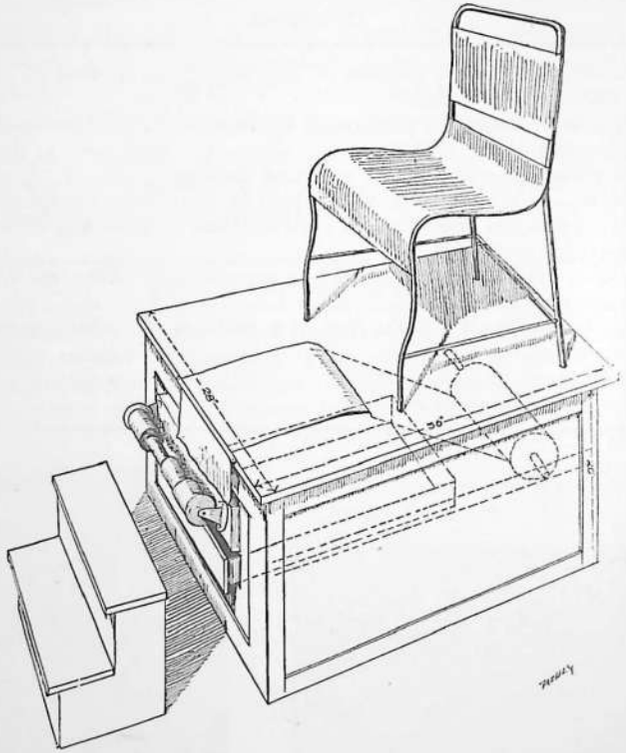

FIG. 243.—Foot examining stand. (Dickson and Diveley, Functional Disorders of the Foot, courtesy of J. B. Lippincott Company, also Jour. Am. Med. Assn.)

Diveley has devised a small foot examining stand which may be prepared by any carpenter.[17] This is 20 inches high and measures on its top surface 28 by 36 inches. It is built of 1 inch wood. A chair for the patient is mounted solidly on the rear of the platform. Threaded from below is a roll of white wrapping paper which rolls through a slit below and beneath the chair and on to the front of the platform. The loose end is held in a slot on the front end of the stand formed by a thin steel bar, which holds the paper in place and acts as a tearing edge. On the front face of the stand there is a roll of adhesive, and just beneath, a drawer. A combination of 2 steps and seat may be used as a seat for the physician during the examination of the feet and as a stairway to the platform for the patient. When

the doctor is seated upon this, the patient's feet are level with the physician's eye.

Examination of the Foot Should Include:

History of foot pain or preëxisting arthritis; relation of pain to standing and walking; occurrence of pain while at rest in bed or upon arising; occupation.

In any case of foot strain, the **history** is extremely important. One should inquire concerning: (1) *Pregnancy* (as this often leads to foot strain due chiefly to the gain in weight); (2) any recent gain in weight; (3) *backaches* (which are often due to foot strain); (4) any preceding *accident* or fall; (5) *change of occupation* from a sedentary one to one requiring the patient to be on his feet much of the time; (6) any *recent change of shoes* (especially from high to low heels); (7) previous treatment and the results of this; (8) the time of onset of the foot discomfort, its duration, suddenness of onset, etc.

Examination of the patient's shoe; one should see if this is too long, short, narrow or wide. *Note the points of greatest wear on the shoe.* See what has happened to the heels, soles, uppers and counters. Individuals with short heel cords scuff off their soles at the tip. In those with pronated feet, the heels become worn on the inner side. This also occurs in weak feet that abduct; these patients, in addition, wear away the sole under the great toe joint. In those with low second, third and fourth metatarsals, a hole is worn in the bottom of the sole.[66]

See that the stocking is not too tight for the foot.

Posture of the body; look for scoliosis, deformity or decreased motion in the ankles, knees or hips; *gait.*

Measurement of leg length; determination of comparative levels of gluteal folds. **Shortening of one leg may be an important cause of persisting low back pain.**

Measure weight distribution on the feet by a pair of bath room scales.

General contour of the foot; spreading of the forefoot.

Palpation of all joints of the foot; search for *areas of tenderness.*

Range of motion of all joints of the foot; tension of the heel tendon and tone of the plantar fascia.

Shape and position of the toes.

Presence of corns, calluses, depression of the second, third or fourth metatarsal heads, exostoses on the dorsum of the foot, any swelling at the ankle joint or on the sole or dorsum of the foot, swellings around the malleoli, thickening of the Achilles tendon, plantar warts, hammer or cock-up toes or other deformities of the toes, bunions, hallux valgus or rigidus, varicose veins, leg ulcers.

Examination of the longitudinal and transverse arches, including their height and the rigidity of any of the joints.

Examination for pulsation in the dorsalis pedis and posterior tibial arteries; color and temperature of the feet.

Examination of the foot at rest and on weight bearing.

Roentgen examination in some cases if symptoms persist after proper support is given.

Bony prominences of the foot: 1. Internal malleolus.
2. Tubercle of the scaphoid (below and in front of No. 1).
3. Head of the astragalus (between 1 and 2, situated deeply).

4. Sustentaculum tali (below 1).
5. Sesamoids of the first metatarsophalangeal joint (felt on deep palpation from the plantar surface).
6. External malleolus.
7. Outer aspect of the os calcis and its peroneal tubercle (below 6).
8. Tuberosity at the base of the fifth metatarsal.

FIG. 244.—Imprint of a flat foot. (Drew.)

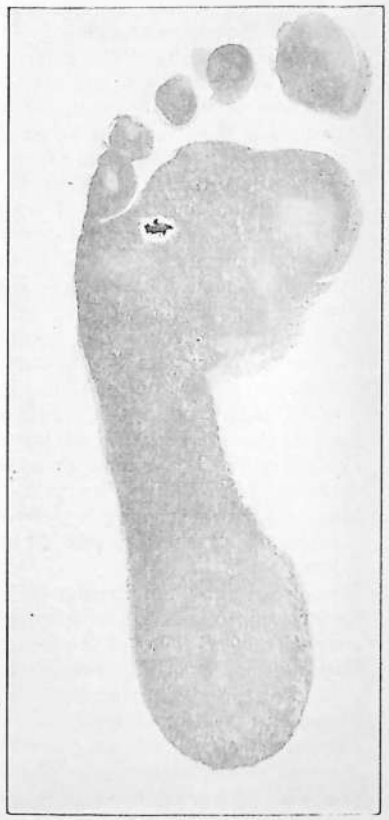

FIG. 245.—Imprint of a normal foot. (Drew.)

Pain in the foot may be a manifestation of general or local disturbance. The character, location, duration, mode of onset and time of occurrence of the pain must be studied; other factors which must be taken into consideration include age, sex, occupation, weight, systemic disease, poor posture, local conditions of the feet and wearing apparel.

The most common condition of the feet encountered by the medical officer is *pes valgoplanus*—relaxation of the longitudinal arch.[86] Normally this arch is maintained by tension in the long plantar ligament, the ligamentous attachments of individual bones in the arch to one another and

by the muscular sling formed by the tendons of the tibialis anticus muscle on the inside and the peroneus longus muscle laterally.

Chronic foot strain is probably the commonest cause of painful feet. Ligaments of the feet are strained by prolonged standing; stretching of these ligaments may result in abnormal separation of the bones of the foot.

All flat feet are not necessarily painful: *In races who do not usually wear shoes* (and in small children), *the feet are often flat in weight bearing; however, they are mobile. Most painful feet lack mobility because of shortening of the ligaments by fibrous adhesions,* as may occur in chronic foot strain or less often from arthritic changes in the tarsal joints.

Common Signs of Disorder of the Foot:[83]—1. *Pronated (flat) feet.*

2. *Callus* on the sole, behind the second and third toes; *corns* on the tops of the toes, hyperextension at the metatarsophalangeal joints and flexion in the proximal phalangeal joints.

3. *Convexity of the inner border of the foot* as the head of the astragalus approaches the supporting surface and finally crosses the inner border of the foot print.

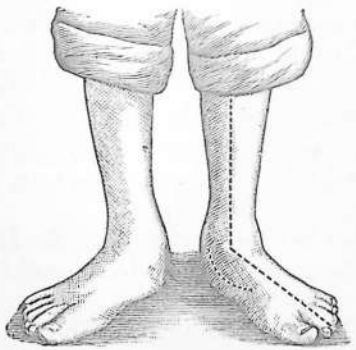

FIG. 246

FIG. 247

FIG. 246.—Typical "flat-foot" of moderate degree, illustrating the component elements of abduction and depression of the arch. (Whitman.)

FIG. 247.—Fixed depression of the anterior metatarsal arches, showing the prominence of the depressed joints in the sole, also extreme hallux valgus. (Whitman.)

4. Abduction, dorsiflexion and supination of the forepart of the foot as compensatory to the changes in the posterior portion of the foot.

Shortened Calf Muscles may be an important cause of disorder of the feet. Such shortening is due *most commonly to long use of high heels in women,* although occasionally it may be congenital or may follow prolonged illness in bed. In women who have worn high heels for long periods, marked shortening of these muscles may take place. *An abrupt onset of foot trouble may occur from the strain produced in walking when these women finally revert to low heels.* When shortening of the gastrocnemius and soleus muscles has occurred, the normal range of dorsiflexion of the foot is decreased.

FLAT (WEAK) FEET

Flat feet may be congenital or acquired.[55] The latter may be classified as flaccid (or static), spastic and rigid. In the *flaccid type* of flat foot, the main or longitudinal arch is depressed only with weight bearing and assumes a normal contour when the weight is removed. It has been suggested that a better name for this type of flat foot might be abducted or weak foot because some of these patients may have high arches (and may suffer greatly). During activity these individuals turn their feet outward so that the weight-bearing line prolonged downward through the center of the patella along the crest of the tibia no longer falls in the normal position between the first and second toes, but may be to the inner side of the first metatarsal.

If the flaccid flat foot is untreated, peroneal muscular spasm may occur, producing the spastic flat foot resulting in tenderness over the peroneal tendons and limitation of inversion (which is very painful).

The *rigid flat foot* is an end stage, with the foot firmly fixed in abduction and in which simple measures will not restore the normla balance of the foot. Many of these patients have severe pain.

Shuster divides the etiology of flat feet into the vocational, overweight, disease, traumatic and paralytic groups.[73] In the disease group he includes tuberculosis of the tarsal bones, rheumatoid arthritis, typhoidal infection of the bones of the foot, rickets involving the feet and gonorrheal arthritis. Trauma may include a fracture of the os calcis, sprained ankle, Pott's fracture, short leg, favoring one foot after injury or because of a wart on the sole, Morton's toe, corns, etc.

Vocational flat feet may develop from standing on hard surfaces for long periods, as in policemen, mailmen, physicians, dentists, waiters, teachers, barbers and bakers.

Failure of the tibialis anticus and posticus (which maintain the longitudinal arch of the foot) throws strain upon and stretches the ligaments with weight bearing; this leads to the pain in early flat foot. Foot strain is common in adolescents and early adult life, particularly in those standing for long periods (such as internes and nurses). *If these individuals are treated as soon as pain or tiredness of the feet is noted, one may prevent the development of a deformity.*

Flat feet are caused mainly by improper shoes, overstrain and debilitating illness.[84] Foot strain may be alleviated by proper shoes and snugly fitting supports for weak feet or strained arches, but proper exercises and massage are required to develop the foot muscles and effect a permanent effect.

Before flat foot can occur, a severe strain must have been placed on the ligaments, which together with the muscles, act as safeguards to keep the arches from flattening. Ligamentous strain is preceded by muscular strain and weakness. It is only after both the muscles and ligaments give way that flat feet occur.

Strain of the anterior arches is due mainly to cramping of the forefoot; this will usually not occur if one wears shoes which are big enough and properly fitting stockings; treatment includes raising the arch by pads, or by a metatarsal bar, and by exercises.

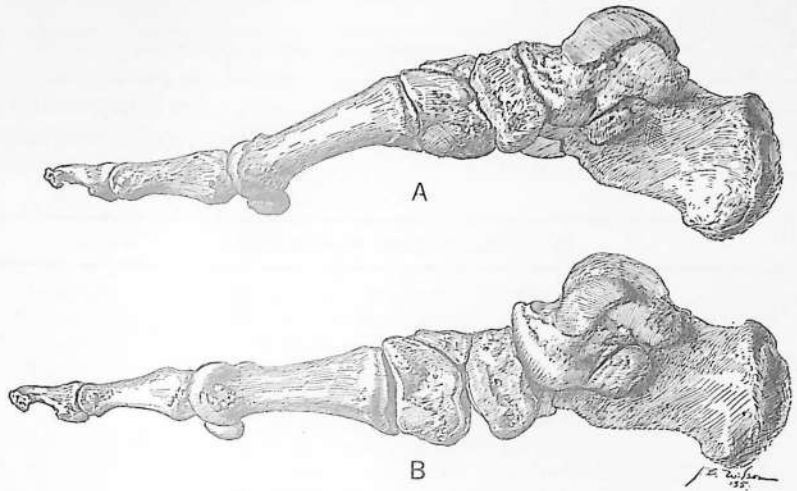

FIG. 248.—Medial aspect of the foot: *A*, Normal longitudinal arch. *B*, Arch in flat foot. (Shands, Orthopedic Surgery, courtesy of the C. V. Mosby Company.)

FIG. 249.—The ordinary type of weak foot in a child. The degree of abduction is indicated by the relation of the patellæ to the feet. (Whitman.)

The human foot was probably at one time an organ for gripping.[7] Painful feet may result in part from the use of shoes which cramp the feet and from our habit of walking on hard pavements. Others do not believe that

most foot disorders are due to faulty shoes. Some feel that the high, narrow, wobbly heel with the short, low pointed vamp is the greatest single cause of discomfort in the feet.

Morton believes that the commonest pathologic causes of pronated feet are: Abnormal laxity of the plantar ligaments which permits excessive dorsal mobility so that the first metatarsal does not come into firm contact with the ground at the same time as other metatarsals; and a congenitally shortened first metatarsal bone.

Symptoms of Weak Painful Feet

Weakness in the feet; pain, dull or sharp, burning or stabbing, especially on the medial side of the feet; *ready tiring of the feet* or ankles varying widely in character; coldness, numbness or severe cramps, generalized over the ball of the foot or localized in one joint; *aching or cramps in the calf at night or occasionally during the day*, or pain in the ankle, knee (especially on the inner side from strain of the internal lateral ligament), thigh, hip and lumbar region following weight bearing or activity; pain along the longitudinal arch and inner border of the foot (due to ligamentous strain); pain along the course of the tibialis anticus in the front of the lower leg along the tibia; spasm and exhaustion of pronator muscles; pain on the inferior and medial aspect of the head of the first metatarsal bone, occasionally with numbness of the toe and pain near the origin of the plantar fascia in the heel and at or under the external malleolus, in weak feet.

One may have pain in the feet, legs and low back for hours on retiring; this usually disappears by morning.

Burning or callus formation on the soles of the feet under the heads (distal ends) of the second and third metatarsals.

Tendency to sit and rise more, and to walk and stand less than previously.

With the establishment of local inflammatory conditions in the feet: localized areas of sharp pain; numbness or tingling in the feet; pain in the legs, thighs and back; profuse sweating of the feet; edema of the foot and ankle; discoloration or mottling of the feet.

In both of these conditions, weight which is borne normally by the first metatarsal segment is shifted at least in part to the second. This latter bone is not built to support such added strain and its ligaments become stretched, permitting the foot to pronate or to roll toward the inner border. Most of the patients with symptoms of foot disorder have developed a moderate degree of such pronation. Normal feet may be strained by excessive loads or long hours of standing, because one naturally assumes a neutral position in standing (with the toes turned outward about 15 degrees), so that strain falls upon the ligaments. Increased strain falls upon the inner side of the foot when the feet are turned out in walking.

In the strained foot *there is overstretching of the inferior calcaneoscaphoid ligament, plantar fascia and plantar ligaments and straining of the muscles controlling the inner side of the foot.* Relaxation of the calcaneoscaphoid ligament permits forward pressure of the head of the astragalus. The latter approaches and finally crosses the inner border of the foot print.

As a compensatory mechanism, *abduction, dorsiflexion and supination occur in the forepart of the foot.*

Signs of Painful, Flat (Strained) Feet

Abduction, dorsiflexion and supination of the forefoot. The weight-bearing line (dropped from the middle of the patella) should come between the first and second toes in the normal foot; in flat feet this line often falls mesial to the great toe.

Prominence of the internal malleoli with a prominent convex curve along the inner and inferior aspects of the arch.

Pain and tenderness beneath the scaphoid, head of the astragalus, sustentaculum tali and internal malleolus and along the plantar surface of the inner border of the foot and over the central portion of the plantar fascia.

Lengthening of the inner borders of the feet (because of separation of the os calcis and scaphoid, stretching of the spring ligament, and dropping of the head of the astragalus between the two bones).

A downward and outward position of the tendo Achillis (instead of the normal directly downward position).

Swelling of the feet.

List of the foot to the inner side; leg seems displaced medially.

Marked wear of the soles of the shoes along the inner side. Weight is borne on the inner side of the foot.

Knock knee may be an early sign of development of flat foot.

In weak feet, with peroneal spasm, there is slight or marked limitation to adduction or turning in of the foot when the leg is extended. The flat foot is actually a valgus foot, *i. e.*, abducted and everted.

Secondary symptoms include: *tenderness and callus formation under the first metatarsophalangeal joint* and the inner side of the heel; callus formation beneath the prominent scaphoid bone (if marked relaxation of the longitudinal arch occurs); and an awkward gait.

If not relieved, eversion and abduction lead to osseous, irreducible flat foot. When rigid flat foot has occurred, there is no spring in the gait and patients use their lower extremities like pedestals.

Deformities secondary to flat feet include hallux valgus, hallux rigidus and hammer second toe.

Roentgen examinations of the feet may aid in establishing an accurate diagnosis in some cases; this includes a dorsoplantar roentgen-ray film centering upon the middle cuneiform bone. A lateral view is of value in lesions of the posterior part of the foot, and an oblique view may be needed for the mid-tarsal region. Findings of importance in such films include:

- Enlargement of the second metatarsal bone (in comparison to the three outer metatarsals).
- *Shortness of the first metatarsal*, noting the position of its head in comparison to the head of the second metatarsal.
- A broad line of lesser density extending from the first intermetatarsal space backward between the medial and middle cuneiform bones; this is an *indication of dorsal hypermobility of the first metatarsal segment.*
- Loss of cartilage and development of bony ridges in the intertarsal and metatarsophalangeal joints in most cases.

With this **there follows an internal rotation of the knees and of the hips** with a resulting forward tilting of the upper portion of the pelvis. *This produces a lumbar lordosis* when the shoulders are brought backward in order to maintain a state of equilibrium. Thus, **foot strain is a very common cause of backache.**[65]

Standing with the foot rotated outward produces muscular relaxation and if persisted in for long periods stretches the ligaments on the inner side of the foot and produces weak foot. If the forefeet are rotated outward in walking, the ligaments and muscles on the inner side of the foot are stretched and weakened as most of the weight is thrown on this area, again leading to flat foot.

Outline of Treatment of Most Cases of Early, Flaccid Flat (Weak) Foot[26,99]

No weight bearing if pain is present at rest.
Proper shoes, with heel and sole wedges, if indicated; measure and correct weight distribution by a pair of bathroom scales.
Temporary use of arch supports in some cases (especially if markedly overweight or if required to stand or walk for long periods).
Correct posture in standing and walking.
Physical therapy (including *heat* and *massage*) and *exercises* for the muscles of the foot and leg.
Reduction of weight if indicated.
Stretching of shortened calf muscles, if necessary.

Treatment of the Weak (Flat) Foot.—Pain and impaired function are indications for treatment. **If pain is present at rest or if there is an associated arthritis, prohibit weight bearing until pain subsides** if this is economically possible; use a Castex bandage (or light removable plaster cast) to maintain the foot in inversion, the forefoot in adduction and to support the longitudinal arch. This may be removed each day to permit exercise of the invertor muscles of the foot; exercises must not cause muscular fatigue or pain.

Many individuals with weak, pronated, painful feet may be markedly benefited by a **main arch pad** of felt, cork or rubber, averaging $\frac{3}{16}$ inch (beveled to $\frac{1}{32}$ inch on the lateral side) and a **heel wedge at the rand** (just beneath the sole). *The proper placing of the rand wedge* is extremely important, lest one turn the heel of the shoe and not the foot. The physician must insist that the entire heel be taken off, the wedge placed next to the sole (heel rand) and that the heel then be replaced. This wedge averages $\frac{1}{4}$ inch in thickness with its highest point at the breast of the heel (anteriorly on the mesial aspect), running to $\frac{1}{32}$ inch posteriorly and laterally.

In mild cases or those associated with knock knee, throw the weight toward the outer side of the foot, by using a **crooked and elongated heel,** or by **raising the inner border of the sole and heel of a flexible shoe approximately $\frac{1}{4}$ inch.** Shaping of the heels and soles to produce inversion of the foot in weight bearing may prove useful.

In most cases of ordinary static flat foot, Steindler prefers to use a

pronatory inner wedge under the heel and a *supinatory outer wedge under the little toe.* These are not used in rigid or in juvenile flat foot. If the flat foot is supple and pliable he applies a wedge to raise the inner border of the heel sufficiently so that the os calcis is placed squarely on the ground. In addition, in order to equalize any tendency to excessive supination in the forefoot, a short cleat is applied to the outer side of the forepart of the shoe. If the varus position of the heel can be maintained until the weight of the body has been transmitted to the head of the fifth metatarsal through the mid-foot, this provides security from strain provoking pronation.

In patients with short Achilles tendons, the whole heel must be raised to relieve this strain.

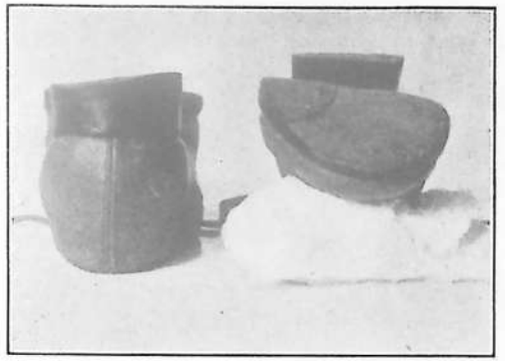

FIG. 250.—Shoes corrected with cleats; inner wedge heel and outer patch over toes. (Steindler, Jour. Bone and Joint Surg.)

If necessary, an *arch support* is used temporarily when the patient becomes ambulatory; *properly made supports are comfortable, support the arch, favor walking on the outer rather than the inner border of the foot,* and toeing straight ahead or slightly inward. The altered heel or arch supports are used for several months after the patient can perform a day's work or exercise without fatigue or pain in the feet.

Measurement of Weight Distribution.—Various methods have been devised to *measure weight distribution as received by the feet.* Apparatus may vary from simple bathroom scales to elaborate electrical machines which measure the weight borne by various portions of the foot (such as heel, outer part of the foot and great toe). *The average practicing physician,* when confronted with a patient with painful feet (with or without low back strain) *should use two small bathroom scales, placed close together,* having the patient stand with one foot on each. The patient should not look at the recordings on the scales as it is simple to shift the weight consciously in an attempt to balance these. *In the normal individual, an equal amount of weight will be borne on each foot.* Apparent or actual unequal leg length of moderate amount may be one of the basic factors which leads to increased weight bearing on one foot, producing not only foot strain, but in addition, low back pain. If the practicing physician would adopt

the simple two scale method, he would be amazed at the *improvement which would follow in many cases of low back pain and foot strain by the simple expedient of fitting an adequate lift to one heel in an effort to balance the weight equally on the two legs.* We have observed many instances of chronic low back pain, in individuals who had wandered from specialist to specialist over a period of months or years, which decreased and completely disappeared when the weight distribution was properly balanced and a proper lift was applied to one heel. The practicing physician should not at once get the impression that correction of improper weight distribution will cure all low back pain; however, this important addition to our therapeutic armamentarium should be well known to all practitioners.

Arch Supports.—Many physicians ask: *"What type of arch supports should I use for my patient?"* "Must I send the patient to an orthopedic supply house or to an orthopedist for these?" "Is there any possibility that I may have these made for the patient locally at little expense?" "Should one of the popular advertised supports be used?" "Should an arch support be worn indefinitely by the patient?" "May such a support harm the patient?"

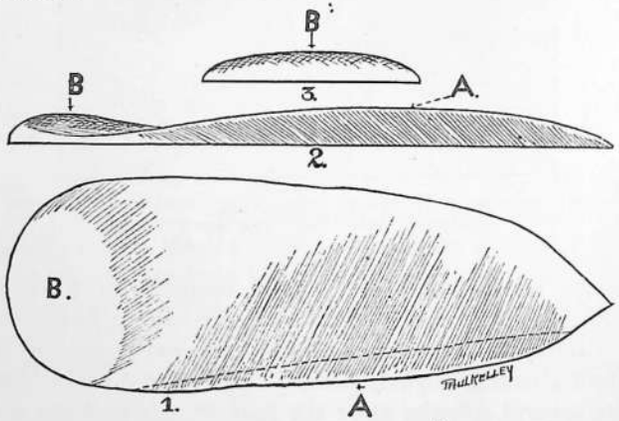

FIG. 251.—Stock sponge rubber support advocated by Dickson and Diveley. *A*, Longitudinal arch portion; *B*, transverse arch portion. *1*, shows shape of inlay; *2*, contour (lateral view); *3*, contour of transverse arch section. (Dickson and Diveley, Functional Disorders of the Foot, courtesy of J. B. Lippincott Company.)

In former days, feet required no support because man walked on soft earth which permitted the heel, outer arch and first and fifth metatarsal heads to become embedded, thus creating a natural support. City walking is unnatural so that firm shoe construction has become a necessary evil.

Muscular and ligamentous strain and not lowering of the arch *per se* causes pain and disability in the foot. Due to a failure of overstrained and fatigued muscles, ligaments become irritated and may serve as a source of tenderness and pain. The foot with a high arch is even more likely to give trouble than a low one. The physician must not dismiss the thought of foot strain merely because he can detect no lowering of the arch.

In a large number of individuals, the arch of the sole of the shoe is not

as high as the arch of the foot. If the patient is vigorous enough to overcome this handicap, no harm results; in others, this leads to foot strain. **In many cases of foot strain there are overstrained and tired muscles, irritated ligaments, some disturbance of circulation in the foot and some rolling of the foot on the inner side.**

It seems reasonable that *the overstrained muscles on the inner side of the foot should be supported.* This may be done by increasing the height

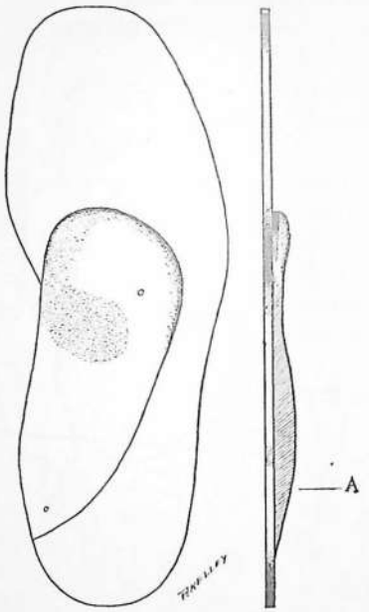

FIG. 252

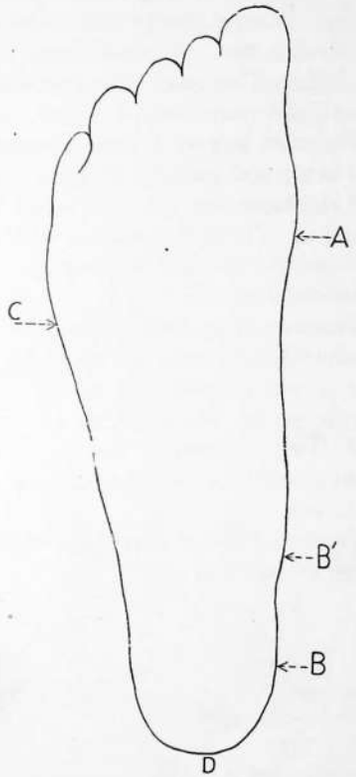

FIG. 253

FIG. 252.—Shows position and high point of support (*A*) for flat foot advocated by Diveley and Dickson. (Functional Disorders of the Foot, courtesy of J. B. Lippincott Company.)

FIG. 253.—Points for determination of position of arch support of Diveley and Dickson. Support (for pes planus) extends from *A* (head of first metatarsal) to *B*. Other points: *D*, back of heel; *B'*, sustentaculum tali; *C*, head of fifth metatarsal. Highest point of support lies under sustentaculum tali. (Dickson and Diveley, Functional Disorders of the Foot, courtesy of J. B. Lippincott Company.)

and length of the curve of the steel shank of the shoe, *by raising the inner anterior border of the heel,* by temporary strapping with adhesive, or by felt, rubber or metal support to fit the arch of the foot.

The conservative treatment of flat foot and pes cavus provokes more controversy than any other phase of orthopedic surgery.[20] *Arch supports are usually only palliative treatment.* However, if arthritic changes in the tarsal bones prevent restoration of mobility of the foot, a well fitting arch support may be needed indefinitely.

Some surgeons are opposed to the use of any type of support for the arches.[3] Hench and Meyerding feel that rigid metal supports are not advisable in general in arthritic patients as they are not well tolerated and may favor pressure atrophy of the plantar muscles.[30] They have used metatarsal bars, heel pads or wedges, and soft felt pads placed under the instep. Most surgeons feel, however, that support is usually necessary. *Arch supports are of minor importance in the treament of the uncomplicated weak foot.* They play an important rôle, however, in the treatment of the *complicated* weak foot.

The main purpose of such a support is to mechanically shift and hold the foot in a proper position, until the threshold of fatigue of the plantar flexors and the supinators has been raised by means of exercises. *The arch support in itself will not cure the condition but will temporarily assist readily exhausted muscles.*

Functions of an Arch Support: To make it uncomfortable to walk or stand in any except the proper manner and to relieve the inner border of the foot of strain; by so doing, it will tend to exercise the foot in a normal fashion and may permit tightening of stretched ligaments.

Common Types of Arch Supports Used for Pronated (Weak) Feet:

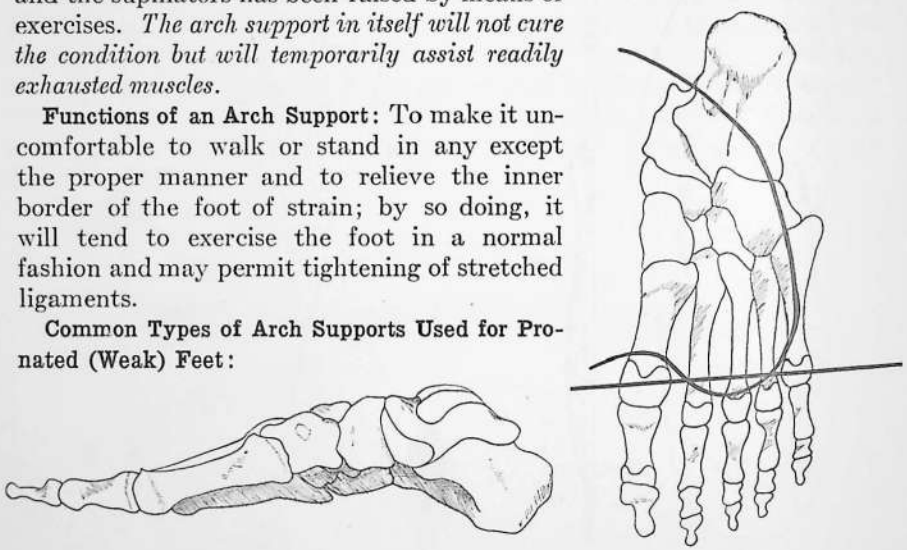

FIG. 254.—Showing position of arch support recommended by Graham. (Courtesy of Am. Jour. Surg.)

1. Longitudinal. 2. Anterior. 3. Combined longitudinal and anterior.

Material in Arch Supports: Felt is usually preferred.

Sponge rubber is used in patients with very sensitive feet or where there is very little fat on the plantar surface of the foot.

Leather is used where there is a desire to incorporate an additional piece of steel, as in congenital deformities.

Steel is used especially in obese individuals. Plates are usually heavy. The **Whitman plate** is one of the best metal supports and may be used for heavy patients with flexible feet. Steel plates may produce some weakness of the small muscles of the foot.

The Whitman plate attempts to hold the foot in a saddle and prevent lateral spreading and bulging on the inner side of the foot. It must be constructed very carefully from a plaster model of the foot. This attempts to force the individual to hold the foot in a normal position during weight bearing.[1]

Very few physicians are well trained in the proper fitting of Whitman plates. Unless one is expert at such fitting, he should not attempt to use such metal supports.

Lovett obtained relief most quickly in moderate and severe cases of foot strain by the use of properly fitting steel supports to support the strained and irritated arch. He fashioned plates of soft steel of various sizes into a neutral shape over casts of normal feet; these were then easily shaped to any foot by means of a monkey wrench and round-headed hammer. There were no flanges on the plates; the latter extended from

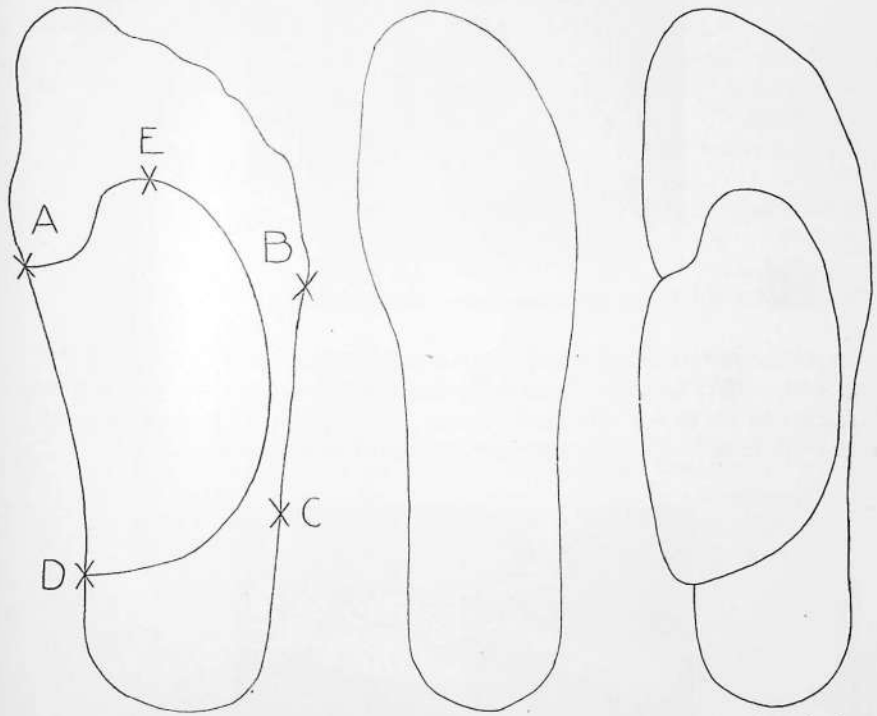

FIG. 255.—Arch supports recommended by Graham (Am. Jour. Surg.): *A*, Just proximal to the head of the first metatarsal; *B*, proximal to the head of the fifth metatarsal; *C*, calcaneocuboid joint; *D*, just distal to the tuberosity of the calcaneus; *E*, neck of the third metatarsal.

the back of the heel to just behind (proximal to) the heads of the metatarsals anteriorly. They were slightly narrower than the foot and fit the foot as smoothly as a wet cloth. If necessary, they were lowered until they were comfortable when worn in the shoes. It was occasionally necessary to raise the plates slightly in a few days after the swelling of the feet subsided. The plates were then tempered and covered with leather or nickel plated and worn as long as necessary. Plates which do not fit properly may further strain the foot by keeping it in a poor position or by contusing the soft parts. Plates sold in shoe stores often have too short a curve, are soft and bend under use.

Accurately Fitting Arch Supports.—These may be prepared from a plaster mold of the foot while the latter is held in a corrected position.[68] The support made from this will carry all of the correction the wearer can stand, at least in the beginning. Plaster impressions may be made by powdering the foot and applying about 4 thicknesses of wet hard coated plaster bandage (from 4 to 6 inches wide) against it. If the plaster is rubbed against the foot, the mold can be removed in five or six minutes and can be used either as a positive or a negative because of its thinness.

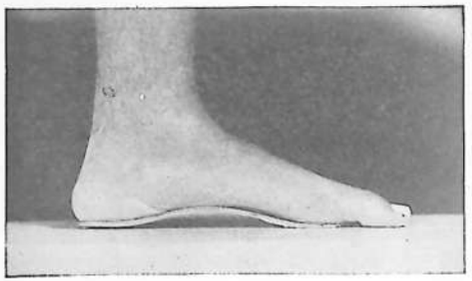

FIG. 256.—Metal plate beneath the foot. (Kuhns, courtesy of Rheumatism.)

In many instances, shoe men do not accurately measure the foot for supports. Unfortunately, the orthopedist often gives the patient a prescription which merely (routinely) reads: "Anterior and main arch supports—$\frac{3}{16}$ inch." We have seen such prescriptions many times.

FIG. 257.—Type of metal foot plate sometimes used to support the arches. (Kuhns, courtesy of Rheumatism.)

At present *we are experimenting with a pliable material which can be molded* (when warm) *exactly to the patient's foot,* when held in the desired position; *in this manner, much of the guesswork in the manufacture of arch supports should be eliminated.* When cool, the material hardens and maintains its position on weight bearing. It can be adjusted or remolded easily at any time by reheating.

Indications for Longitudinal Arch Supports: Individuals (especially if obese) who must stand for long periods, who develop weak, pronated (flat) feet (in which the internal longitudinal arch of the foot has become partially or completely obliterated on standing), and in whom pain or impaired function of the feet is noted. Such supports are often required at least temporarily during pregnancy; after periods of prolonged rest in bed from illness; in changes from a sedentary to an active occupation requiring prolonged standing; or after marked gains in weight.

Indication for Anterior Arch Supports: *Pain, tenderness and often callus formation under the heads of the second or third metatarsal bones.*

Combined Longitudinal and Anterior Arch Supports: A combination of anterior and longitudinal arch supports may be advisable temporarily until the patient can strengthen these arches by proper exercises and rest; heel wedges and sole cleats will often be of equal value. Combined longitudinal and anterior arch supports are frequently required.

Arch Supports Should Feel Comfortable.—Arch supports may feel peculiar to the patient during the first ten days they are worn. *If they are uncomfortable for more than ten days, they should be re-shaped.* Proper arch supports should not only offer support, but should make it uncomfortable for the patient to walk in any other than the proper position.

Approximate Cost of Arch Supports.—This varies with the type of support used. In most instances, a pair of either anterior or longitudinal felt arch supports costs the patient approximately $1.50; the combined anterior and longitudinal arch support costs about $2.50. Removable supports sell for $4 or $5.

Average Height (Correction) Incorporated in Anterior Arch Supports.—This may vary from $\frac{1}{8}$ to $\frac{1}{2}$ inch, but averages about $\frac{3}{16}$ of an inch.

Use of Arch Supports in the Uncomplicated Weak Foot

These are merely a temporary measure to produce proper posture of the foot until exercises strengthen the plantar flexors and adductors.[98]

Arch supports alone will not cure the condition.

Progress is more rapid without the use of arch supports in coöperative, intelligent patients who must not stand for long periods and whose muscles are not too easily fatigued.

Such support assumes in part the hammock function of the supinator muscles and the calcaneoscaphoid ligament, and must conform in shape to the pedal arch system and in size to the individual foot, if correct position of the foot is to be maintained.

The arches represent the major stress areas of the foot; *they must be properly supported to correct improper stress throughout the entire foot.*

Ready made factory supports do not adequately conform to the variations in size and shape of the pedal arch system; **supports should be made to fit the individual foot. The foot should not be fitted to the support.**

In general, if an arch support is needed, it should be constructed for the patient's foot and should extend from the heel to a point just proximal to the heads of the metatarsals, and from the inner border of the foot across the sole toward the outer border of the foot.

Use of Arch Supports in the Uncomplicated Weak Foot—
(Continued)

In many cases, $\frac{3}{16}$ inch will be found to be the approximate height for both the anterior and longitudinal supports. This will vary considerably depending upon the size and configuration of the foot. A system of trial and error must be used, eliminating some of the support if this causes pressure and pain, and adding additional support where the patient feels that this may be necessary.

In acutely painful flat feet of short duration, Shands recommends *support to the longitudinal arch by means of a felt pad and adhesive strapping.*[79] This pad extends forward from the anterior portion of the heel to a position about 1 inch proximal to the first metatarsophalangeal joint. The pad is semicircular in outline with the curved edge extending laterally as far as the proximal end of the fourth metatarsal bone. Seen in longitudinal section, the felt pad is arched with the maximum height occurring at the juncture of the scaphoid and internal cuneiform (which is approximately on a line with the proximal end of the fifth metatarsal). If seen in cross-section, the highest portion of the support is at the scapho-internal cuneiform junction on the medial border of the foot, tapering off from here in all directions (outward, forward and backward). *These felt pads may be made by any physician and do not require any unusual skill.*

Felt may be purchased in sheets and cut to proper shape or stamped out by dies. A large 7 or 10 inch blade will bevel this to eliminate any ridges or irregularities. The pad may be attached to the shoe by means of rubber cement applied to the felt: it becomes secure in a few minutes.

Average Height (Correction) of the Support in Longitudinal Arch Pads.—This averages about $\frac{3}{16}$ inch, but varies with each individual and must be determined by trial.

How Can a Physician Prepare Arch Supports for His Patients?—Dickson and Diveley[16] advocate the use of a firm sponge rubber arch support. This is made in three sizes so that practically any foot can be fitted. The highest point of their longitudinal portion is $\frac{1}{4}$ inch and that of the anterior portion, $\frac{3}{16}$ inch. This support extends from a point just posterior to the second metatarsal head to a position under the heel with the highest point under the sustentaculum tali. The anterior portion of their support lies between the first and fifth metatarsal heads. The height and shape of the support may be altered by a sharp knife or by grinding on a small coarse emery wheel. The support may be fastened in the shoe by several shoe tacks and should then be covered by thin leather. *Do not make the support too high at the beginning of treatment,* or unnecessary discomfort may result. The height of either portion of the support may be increased by placing pieces of sponge rubber or piano felt between the support and the insole of the shoe.

Deficiency of the First Metatarsal Segment.—Morton believes that the main disturbance in disorders of the longitudinal arch and of the anterior portion of the foot is a "functional deficiency of the first metatarsal segment," so that *an excessive load and strain is placed upon the second metatarsal segment* and its tarsal joints (part of which is normally borne by the first metatarsal segment). Whether this is caused by laxity of the plantar ligaments or by a short first metatarsal bone, the point of strain is focused upon the basal joints of the second metatarsal. To relieve this disability an arrangement must be worked out which will permit the first metatarsal segment to again carry its normal portion of the body load.

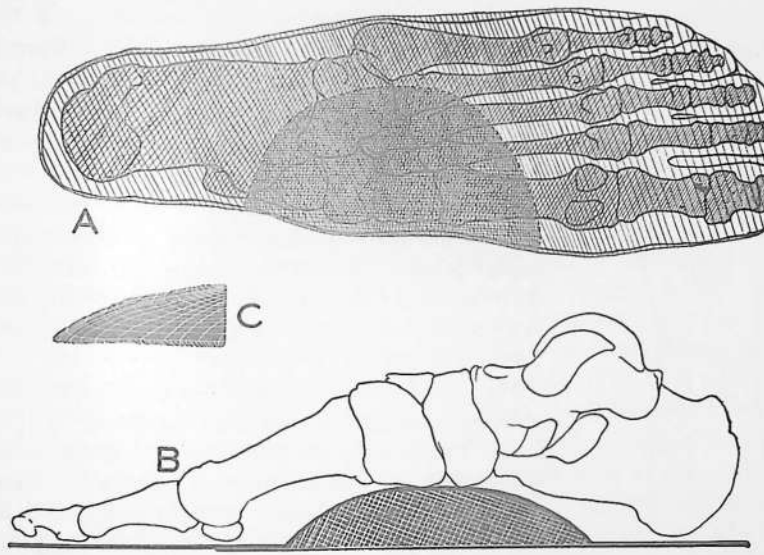

FIG. 258.—Longitudinal arch support suggested by Shands: *A*, Plantar view; *B*, medial aspect of the foot with the support in place; *C*, cross-section of the support showing the thickest portion of the medial aspect, tapering laterally. (Shands, Orthopedic Surgery, courtesy of the C. V. Mosby Company.)

If the disturbance is caused by a laxity of the plantar ligaments, a dorsal hypermobility results which prevents the first metatarsal from coming into firm contact with the ground at the same time as the other metatarsals, thus increasing the load borne upon the second metatarsal. Morton compensates for this condition by placing a support of sufficient height under the head of the first metatarsal so that contact is made by this metatarsal at the same time as this occurs with the other metatarsals; thus the first metatarsal segment again carries its portion of the burden, and allows the second metatarsal segment to support only its normal share of the body weight. For this purpose, there has been devised a **compensating insole** which fits into the shoe with a "platform-like projection" just under the head of the first metatarsal bone. This may also be useful in cases of foot strain due to a congenitally short first metatarsal bone. The height of the small platform thus placed can be determined only by trial, as the

"compensating insole" can be removed easily for adjustment of the height of this portion. In some cases, however, any support under the first metatarsal head causes mid-tarsal pain and tends to throw weight to the outer aspect of the foot causing irritation of the fifth metatarsal.

Foot complaints continue to be a major military problem. As many as 40 per cent of the complaints in the orthopedic clinic at Army camps may be referable to the foot. A large number of soldiers recently inducted into the Army develop painful feet during their basic training.[33] *Most of these respond satisfactorily to rest and conservative management.* The more severe cases can usually be divided into the traumatic lesions of the feet, severe strains, sprains and fractures, and postural foot disorders and skeletal abnormalities.

Bingham[33] found that about *10 per cent of all patients at an Army Hospital with the more severe foot disorders had a congenital insufficiency of the first metatarsal segment.* The condition was usually bilateral. Symptoms included an *aching and burning pain under the callus over the second and third metatarsal heads,* together with pain and stiffness in the second and third metatarsophalangeal joints. Most of these patients stated that their feet had "always been bad;" however, their symptoms were usually aggravated by strenuous military duties.

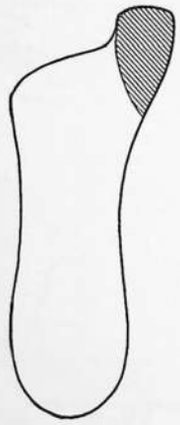

FIG. 259.—Compensating insole with platform to lie under head of short first metatarsal bone. (Morton, The Foot, courtesy of Columbia University Press.)

Physical findings are very few in these individuals and an *x*-ray examination is advisable in every case. Palpation of the first metatarsal shaft may reveal that the first metatarsal segment is shortened or displaced slightly upward and medially. *The most accurate means of confirming the diagnosis is by an anteroposterior x-ray film of the foot in the standing position,* centered over the first cuneiform (dorso-plantar view). It will be found that *the first metatarsal is shortened, as compared with the second,* and may be in varus. The cortex is thinner than normally and may appear atrophic. The sesamoids may appear displaced posteriorly or adducted. A lateral view may reveal a relative dorsiflexion of the first metatarsal shaft and relaxation may be further evidenced by widening of the joint between the first and second metatarsals and the first and second cuneiforms. The second metatarsal shaft may appear hypertrophied and enlarged, with its cortex thickened.

The compensating insole is fitted by the shoe size. The insoles which may be made from a suitable thickness of sole leather from a tracing of the patient's foot, should be worn for an hour or more daily, increasing the time gradually until they are comfortable and the symptoms have disappeared. They may be transferred from one pair of shoes to another. Bingham found that *over 75 per cent of soldiers requiring compensating insoles could return to full military duty while wearing the insoles.*

PROPER SHOES

We cannot hope for improvement in the foot and shoe situation as long as we have untrained shoe clerks and disinterested physicians. If the physician is to supervise the fitting and adjustment of shoes for his patients, he must become perfectly familiar with the science of fitting the foot and the changes necessary in the base planes of the shoe.

Many people buy shoes for their eyes rather than their feet. Unfortunately, some physicians know very little concerning the type of shoe best suited to an individual's foot, and for this reason the patient is swayed entirely by the shoe clerk's advice.[28] Short shoe fitting is responsible for most ills that feet and shoes are heir to. Most women have the false impression that a proper "orthopedic shoe" is necessarily an ugly and non-stylish affair; this is far from the truth.

Moffett has analyzed shoe sizing and fitting. **The proper shoe should fit.**[57] *If shoes are uncomfortable or painful to wear, they should not be used regardless of their quality or beauty.* The making of footwear has now become a major mass production and distribution industry within the last fifty years in this country.

Unfortunately, few orthopedists have made studies of lasts, shoes, their construction and fitting; however, the orthopedist continues to be the recognized authority on the foot. Many general practitioners dislike to refer foot complaints to an orthopedist because in many instances the treatment prescribed is limited to a prescription for orthopedic shoes and arch supports which he himself can prescribe. General practitioners, unfortunately, show little interest in the foot. Chiropodists do not often order orthopedic shoes for patients but in general, confine most of their activity to strapping, massage, heat and the use of pads. The situation resolves itself into the definite fact that **shoe men have been charged with taking over the care of the foot even though they lack the fundamental knowledge of that intricate mechanism.**

Foot disorders may be caused by weak shoe construction, insufficient cuboid space in the shoe to permit adequate seating of that bone and by compression and distortion of the first metatarsal head.

The main objection to correct shoes is their appearance, because of the bulky lasts which are necessary to full extension for the toes and relaxation for the weight-bearing part of the foot.

The responsibility for correct fit of a shoe must be relegated to the shoe technician. He should be held responsible for a misfit, and not the manufacturer who lives miles from the scene. *The consumer must be taught that the size and width and correct fit of a shoe is usually much more important than the trade name.*

Standard lasts are now available from some manufacturers so that there is nothing to prevent an intelligent shoe man from calling in insole patterns of the various lasts, in the same size and width, and by lining up these patterns at the heel and great toe, he can determine the flares or foot shapes on the insole pattern.

Men interested in fitting the foot should know that the bulk of the wood on most good lasts is on the bottom and not in the circumference. *These men must be taught the value of wood distribution to permit them to select lasts which conform to the various types of feet:* short thick, long bony, inflare, outflare or straight types, high, low and medium arches.

Shoe men should demand that manufacturers use lasts that have bottoms comparable to the human foot: with first and fifth toe convexities, and second, third and fourth metatarsal head concavities, with concavities for the longitudinal arches in accordance with the general foot type.

Although a pair of feet may be placed in as many as six different sizes and widths of shoes, it is never fitted by six different sizes. There is but one true last for that foot.

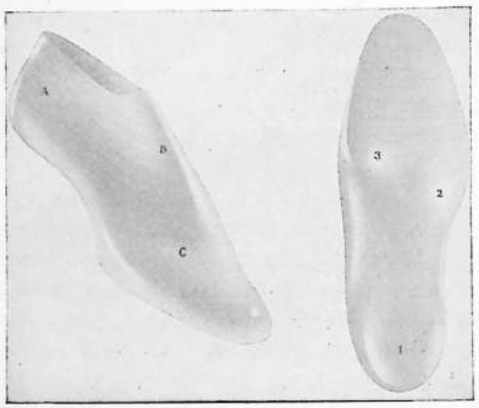

FIG. 260.—Basic last of a shoe. (Geo. E. Keith Company.)

Very few individuals know how to make correct alterations in shoes with the result that the consumer often pays for a sales talk. Very frequently, one foot is slightly larger than the other, but not sufficient to split sizes. A well-placed insole can make up this difference. The forepart of the shoe is seldom wide enough in a weak, relaxed foot. In these cases, it is sensible to fit the forefoot and remodel the back. This may be done by rebinding the sides and backquarter, or opening the back and cutting the insole to conform with the desired heel fit. Other shoe remodeling consists of relasting for bunions, hammer toes, wedging, flares and extensions (both lateral and plantar). Many other alterations may be made in shoes to improve their fitting quality.[91]

By means of an ingenious apparatus known as an *electrobasograph,* Schwartz and his co-workers were able to study the effect of various shoes upon the gait.[74,76] Their records indicated *the increasing instability of the feet associated with increased height of the heel of the shoe.* They also demonstrated that stability of the foot in the shoe was not related to various criteria of shoe fit (such as length, width and comfort in the shoe). Their evidence disclosed *that instability and abnormality in weight bearing may result from improper design of the last in relation to the feet in question, even though the size of the shoe may be correct and the foot be free from pain.*

The correct shoe should contain sufficient space for independent movement of all of the toes and should conform unerringly to the sole of the foot. The innersole saddle should fit against the spring arch so that the foot and shoe move together.

A correct shoe cannot be obtained unless one has a working knowledge of lasts. A last is a wooden form representing the foot and over which the shoe is made. For practical purposes, the foot may be divided into an *inflare* and an *outflare group,* and their variations. Except for the acquired or congenital clubfoot, the inflare is the so-called normal foot. A proper last for such a foot should have a wide outer swing and ample cuboid space to allow the foot to be normally seated. The last must have a high wall to include the fifth toe area and thus prevent lateral and dorsal pressure. The outflare must have a pronounced outer sweep to prevent lateral compression to the already painful and distorted toes.

Fitting of a foot requires careful measurement **from heel to ball** *and* **from heel to toe.** *One must stress the importance of the heel to ball fitting. Good fitting requires that the center of the first metatarsal head be lodged at the turn of the sole.* A shoe that is too long will crease at the toes and cause "shoe bite." A short shoe is a factor in first and Taylor bunions, hammer toes, etc.

A hard and fast **rule of correct fitting** *is to select the bottom of the shoe that is as wide as the bottom of the foot in weight bearing* (including the carrying of a heavy pack in the case of soldiers or others who are accustomed to prolonged carrying of heavy objects).

The heel and cuboid area are extremely important in correct shoe construction. The heel should be cupped, as is the human heel. The cuboid area, which includes the outer weight-bearing assembly, requires reinforcement with a shank of proper strength in accordance with the size range. The inner arch should usually be met by a supporting saddle raised approximately $\frac{3}{16}$ inch for the normal adult foot, thus directing the weight toward the outer side where ample support has been provided. A slight groove should be provided in the insole at the first and fifth heads to provide a well to permit unrestricted action of the sesamoids, the great toe, the head of the fifth metatarsal and a convexity upwards should be present for the second, third and fourth metatarsal heads.

Modifications often required in shoes include *main arch pads, inner heel wedges* for the ordinary type of weight disturbance and *lateral flare extensions* by means of wide welts and broadening of the heel or sole base in severe cases. In most cases, one heel raise, one arch support, or one heel or sole flare may be sufficient to balance the entire body. *If, however, bilateral modifications are indicated, one side invariably requires more support than the other.* Accurate modifications are not possible unless an attempt is made to determine and properly balance the distribution of body weight (as for example, by a set of bath-room scales).

Questions and Answers Concerning Shoes.—What is the last of a shoe? This is a wooden model, approximately the shape of the foot, over which the shoe is built.

What is meant by *straight, inflare or outflare lasts?* These designate the amount of area within the shoe falling on either the inside (inflare) or outside (outflare) or neither side (straight) of a perpendicular plane passed through the shoe, splitting the heel.

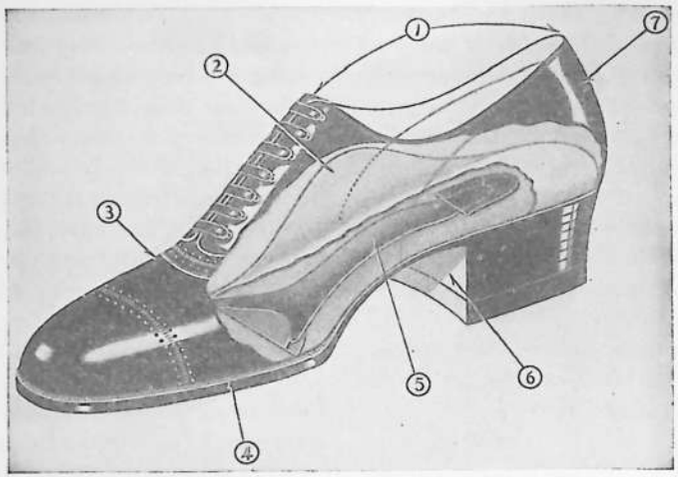

FIG. 261.—Shoe showing **shank** (5) of a shoe extending from the heel to the forepart of the foot. (Geo. E. Keith Company.)

What is a **combination last?** The combination last means that the shoe is made with several different widths, *i. e.*, at the heel, waist, ball measurement and tread measurement. The heel is always narrower than the ball of the foot; this secures a snug fit for the shoe.

What is the *vamp* of a shoe? This is the upper part of the shoe which is anterior to the laces (extending from the upper toward the toes).

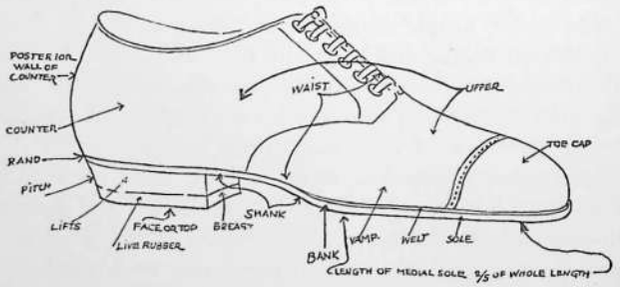

FIG. 262.—External parts of a shoe. (Ashley, courtesy of Medical Record.)

What is the *quarter* of a shoe? This is the remainder of the upper behind the vamp.

What is the *waist* of a shoe? This is the portion immediately in front of the heel. The waist must not sag or break, or the cuboid bone will drop. Leather, wooden or steel shank pieces are used to prevent this.

What is the **shank?** The shank is a thin supportive bar (usually steel) placed above the sole and running from the anterior third of the region of the heel, forward for a distance of from 2 to 5 inches. In most low priced shoes, the shank of a man's shoe averages $\frac{1}{2}$ inch in width and $\frac{1}{16}$ inch in thickness; in women's shoes it varies from $\frac{1}{8}$ to $\frac{1}{4}$ inch in width. In most low price shoes, the shank is usually not more than $2\frac{1}{2}$ inches long, and is found in irregular places.

There is often considerable argument concerning the **advantages of the flexible and the rigid shank shoe. A flexible shank is permitted** for healthy individuals of normal weight, with good musculature and normal feet who must not stand or walk for long periods on cement pavements or hard tiled floors and for those who walk mostly on soft, elastic ground (as farmers).

The **rigid shank** is advised for all overweight individuals or those walking or standing for long periods on hard, inelastic surfaces, for those with weakened muscles and for most cases of weak or deformed feet or foot strain.

What is the **counter** of a shoe? This is the heavy leather support around the back of the shoe; in most ordinary shoes this extends only to the anterior margin of the heel.

What is the **Thomas heel?** This is a heel, the anterior border of which is S shaped so that the heel extends forward for $\frac{1}{2}$ to $\frac{3}{4}$ inch more on the medial side than on the outer side.

What is a **metatarsal bar?** This is a wedge of leather attached to the under surface of the sole, running diagonally across the sole of the shoe behind (proximal to) and parallel with the heads of the metatarsal bones.

When may a metatarsal bar be used? This is indicated in patients with severe pain or tenderness over the heads of the second, third or fourth metatarsals (or stiff painful toes), if an anterior pad does not provide relief from pain on walking; the elevation may vary from $\frac{1}{8}$ to $\frac{1}{4}$ inch. This permits a rocker action on the front part of the foot and prevents marked weight bearing on the heads of the metatarsal bones.

What is a **heel wedge?** This is a small pad of leather which may be placed on the inner or outer side of the heel in order to raise one aspect of the heel (usually the inner) somewhat higher than the other.

If it is desired to elevate the inner margin of the heel by wedges, what is the average height of such elevation? This averages from $\frac{3}{16}$ to $\frac{1}{4}$ inch. In slightly pronated feet, no heel wedge may be necessary; in second degree pronation, one may use a wedge $\frac{1}{8}$ inch high; in third degree pronation, a wedge from $\frac{3}{16}$ to $\frac{1}{4}$ inch may be used in some cases.

When may heel wedges or Thomas heels be of value? These may be useful in early flat foot if used in a flexible shoe. They may be of special value in rural practice as any shoemaker can build them; if additional support is desired, one may attach a longitudinal rubber strip ($\frac{1}{2}$ to $\frac{3}{4}$ inch wide and 1 inch thick) on the inner portion of the sole running from the heel anteriorly for 2 or 3 inches. Such a strip of rubber may be cut from a rubber heel.

What is the cost of orthopedic corrective shoes? These average from $8.50 to $13.00 per pair depending upon the type of shoe employed.

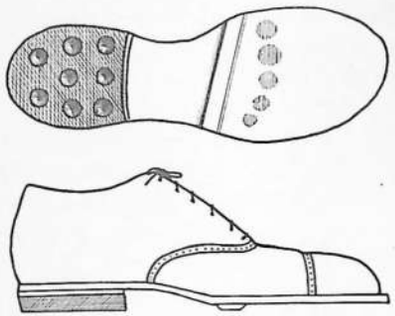

FIG. 263.—**Metatarsal bar.** (Shands, Orthopedic Surgery, courtesy of the C. V. Mosby Company.)

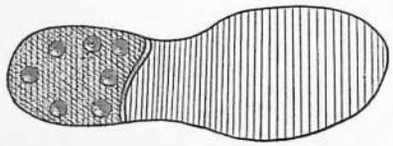

FIG. 264.—**Thomas heel;** note the forward extension of the heel along the inner margin. (Shands, Orthopedic Surgery, courtesy of the C. V. Mosby Company.)

FIG. 265.—Shoe corrections as applied (**heel and sole wedge,** and **metatarsal bar**). (Steindler, courtesy of Jour. Bone and Joint Surg.)

If an individual last is made for the patient, the cost will approximate $30 for the first pair and from $15 to $18 for subsequent pairs. In many

patients with abnormalities of the feet, it is advisable to make a last for the patient from a plaster model of his foot.

Are *orthopedic shoes made in nice looking models?* Yes. An orthopedic shoe may be as attractive as others.

Advantages of Orthopedic (Corrective) Oxfords

Orthopedic shoes are built *heavier* than ordinary shoes.

The *shank* is usually long and broad (to allow enough base), is often triple fluted, and is placed in a definite position, usually on the outer central side of the sole, running from the heel to the beginning of the metatarsal bones.

Most orthopedic shoes have *long counters* which act as a base for the correction which is placed in the shoe, and give further support against pronation.

The *heel* of an orthopedic shoe is not higher than 1½ inches, and usually has a broad base. A high-heeled shoe pitches the body forward and would cause the patient to fall except for a compensatory lordosis as an attempt to regain equilibrium; there occurs an associated flexion of the knees and extension of the leg on the foot. This leads to back strain and displacement of the viscera, with shortening of the gastrocnemius. These individuals then experience difficulty on attempting to change from high to low heels. High heels lead to frequent sprains of the ankle because of the unstable base of a high, narrow heel.

The orthopedic shoe has *greater depth in its throat to allow for the insertion of corrections* (such as arch supports) without binding the foot in the vamp.

Orthopedic shoes are made with a *combination last* to insure a snug fit, and a well fitting heel which does not permit extra motion.

How long must corrective shoes be worn by patients with weak, pronated feet? In the younger age group (from twenty to thirty-five years), orthopedic oxfords and arch supports may be required for from six months to two years; if proper exercises and rest are performed during this period, these appliances may usually be discontinued. In older patients (especially those over fifty years) it is frequently necessary to continue the use of orthopedic oxfords and arch supports for an indefinite period.

What is the **advantage of a straight inner border of the big toe** *found in most orthopedic shoes?* This permits the great toe to lie in its natural position without being pushed toward the others and causing a tendency to hallux valgus. The front part of the foot may also assume its normal position. In many orthopedic oxfords with $\frac{8}{8}$ (1 inch) or $\frac{10}{8}$ ($1\frac{1}{4}$ inch) heels, a straight inner border is used. When the heel is elevated further, the so-called three-quarter inner border of the great toe is used, *i. e.*, a slight deviation from the straight line.

Are orthopedic shoes made to order individually for each person? No. Most good orthopedic houses carry in stock a full line of all sizes and types of corrective shoes, in many combinations, so that the proper shoes will be on hand in 99 per cent of cases. If necessary, shoes may be made to order from individual lasts made from plaster models of the feet. If shoes

must be made up for the individual to a special last, it may require four weeks for such a procedure, if artificial drying processes are not used.

Manufacture of a Shoe: A piece of leather (previously soaked in water until mellow) is hammered out, cut to the desired shape, and fastened with nails to the sole of a *last* (wooden model of a foot). When dried, this retains the shape of the last and forms the *insole* of the shoe. The insole is the foundation of the shoe and can cause crippling of the feet if improperly made or if it develops defects after some wear.

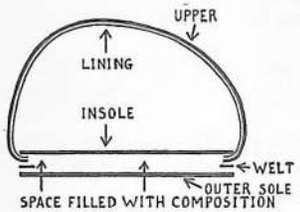

FIG. 266.—Cross-section of a shoe. (Marlin, courtesy of British Med. Jour.)

The *upper* (*with lining*) is fitted over the last and is sewn to the insole. A strip of leather (known as the *welt*) about ½ to ¾ inch broad and ⅛ inch thick, is sewn to the edge of the upper, running around the sole as far as the heel. The *outer sole* is now sewn to the welt (without taking stitches through the upper). The space between the outer and inner soles is often filled with various compositions, such as sawdust and glue. Softening of this material from heat and moisture unfortunately causes bumps and permits the insole to sag in the center.

A well tanned piece of leather will stand a remarkable amount of flexing and punishment because it has been impregnated with fats in its fibers in the tanning process so that internal friction is reduced to the vanishing point. These lubricating fats may be destroyed by frequent wetting and drying out, perspiration, etc.

Leathers used in shoes are divided into three classes: *sole leather, upper leather* and *linings*. Sole leather provides the outsole, insole, welt, shank, rand, toe box and counter of most shoes. Upper leather is cut to form the vamp, quarters, tip and tongue of the shoe. Linings may be cut from leathers especially tanned for this purpose or from stock for uppers.

Fine quality high grade shoes are made of skins. **Kidskin** applies to all leathers made from goatskin. Kidskin is comfortable as it is extremely pliable; in addition, it has multitudes of pores which aid in ventilation so that kidskin is one of the coolest leathers to wear.[13]

Calfskin is used widely in the making of men's dress shoes and it is also used extensively in lighter weights for women's shoes. Calfskin is durable and has a soft, fine-grained lustrous finish. Calf suede is the most important suede leather used in shoes. Reversed calf is obtained by finishing the flesh side or by buffing the grain side; this provides a sueded leather of similar appearance to buckskin.

Suggestions in Selection of Proper Shoes Include:

An almost straight inner border along the first metatarsal; the inner side of the great toe should form a straight line with the inner border of the heel. In some cases, variations (abduction or adduction) are necessary.

A wide outward and backward sweep from the tip of the great toe around and behind the fifth toe, depending upon the type of foot; the highest part of the toe of the shoe should be over the great toe as the head of the first metatarsal is the thickest.

Sufficient room in the ball of the shoe to permit separation and straightening of the toes, and flexion on standing or walking.

Soles wide enough to prevent the margins of the foot from overhanging the edges, and to permit normal spread of the metatarsals with weight bearing; the sole of the shoe must conform to the outline of the weight bearing foot.

Snug grasp of the heel by the counter and snug fit at the waist.

Firm upward pull over the instep by the vamp.

The vamp must be sufficiently wide that there is no undue compression of the toes at the side or on top; in addition, it must fit snugly to prevent wrinkling of the leather.

Rigid shank (extending from the bend of the sole at the ball to the heel) in most people unless feet are flexible; flexible shank may be used in children and young adults. A steel spring in the shank of the shoe may be unnecessary if the patient walks properly and does not wear too high a heel. A steel shank may discourage flexion and adduction of the forefoot on walking.

Many shoes do not furnish sufficient room over the instep especially for individuals with moderate or highly arched feet. Tight lacing in these individuals with improper shoes may push the instep down resulting in mechanical elongation of the foot, stretching of the arch and leading eventually to foot strain.

Soles of the shoes thick enough to prevent trauma from hard pavements, but not too thick so as to act as a splint.

Heel: broad and low (¾ to 1½ inches in height). Very pointed heels cause difficulty in balancing. If high heels come too far forward, there is a constant tendency to overbalance backwards. *Rubber heels* should be used by those living in cities; these act as shock absorbers.

In patients with shortened tendo Achilles and calf muscles, low heels must not be used because of the resulting strain which they cause. Exercises to stretch the tendon (*q. v.*) may be used so that they can be made to accommodate to lower heels.

Combination last often advisable: *i. e.*, ball, one or more sizes wider than the heel.

Test of proper length of shoe: first metatarsal head must push downward at inner flare of sole; tip of the shoe approximately ½ to ¾ inch in front of the tip of the great toe. *More foot troubles are caused by short shoes than by narrow ones.*[72]

One must order shoes slightly longer than expected in those who must carry heavy loads; the feet of these individuals should be measured, if possible, while carrying buckets of water or other weights equal to the usual loads. One must be careful that the shoe is not too long permitting the foot to slide forward from the upper portion of the heel.

Suggestions in Selection of Proper Shoes Include—*(Continued)*

Corrective shoes should be used in housework, shopping, walking, etc.; occasional use of "stylish high heeled dance slippers" is not harmful. High heeled shoes worn only for dress occasions may be sanctioned. *High heeled misshapen shoes throw a tremendous strain upon the muscles supporting the arch.*

The shoe must be tested by standing in it, as the distance across the metatarsals increases on standing.

Shoes enlarge sidewise with wear, but never lengthwise; shoes tend to be shortened when repaired.

Proper shoes must be worn in pregnancy to give adequate support to the foot; leg muscles must be prepared by proper exercises. Most foot discomfort may be avoided by the use of proper posture and gradual return to the feet following delivery. During pregnancy, it may be advantageous to fix small pieces of felt under the region of the longitudinal arch for temporary support.

If the patient has arthritis, Krusen advises a stiff shanked well fitting orthopedic shoe, in which there is a soft pad under the instep to aid in supporting the longitudinal arch, a metatarsal bar to aid in supporting the anterior arch, and a heel pad or wedge.[37] When a longitudinal pad is used, a heel wedge is not necessary.

Patent leather includes patent kidskin, calfskin, colt and side leather. A varnish-like surface is applied to the skins which are then subjected to a careful baking process thus hardening the surface, producing patent leather.

Genuine buckskin is derived from the skins of deer. Although this is high in price, its softness, fine appearance and coolness makes it ideal for high grade sport shoes.

Pigskin may be chrome tanned to produce a leather that is soft, mellow and pliable; it is very durable, porous and is used mainly for sport type shoes.

Suede leathers (usually calf, but in some instances, kid, sheep or side leather) are made to resemble genuine buckskin in texture by finishing the flesh side of the hide rather than the grain side in order to produce a rich, velvety nap.

Cordovan is a fascia which is found as a central layer in horsehide, between the hair and the flesh sides.

Sole leathers are tanned by several methods (oak, chrome and union), each of which produces leather of different quality, color and texture. *"Oak" sole leather* is the most familiar; this is produced by tanning hides with a combination of vegetable tanning materials. Prior to tanning, hides are soft, slimy and readily subject to decay. Proper tanning of hides leads to a firm, flexible material which resists decay.

Perspiration leads to deterioration of leather. The constant wetting of the inside of an individual's shoes dissolves lubricating fats from the leather, so that the latter loses its pliability and hardness, and cracks and

rots. Perspiration, together with pressure, causes destruction of linings leading to bulging or wearing away of counters, brittle, curling and cracked edges of insoles and eventually, rotting of fibers of the leather of even the outsoles.

Wet shoes should not be dried by placing them on a radiator. although they may be placed near this.

FIG. 267.—**Incorrect walking position** (toes pointing outward). (Geo. E. Keith Company.)

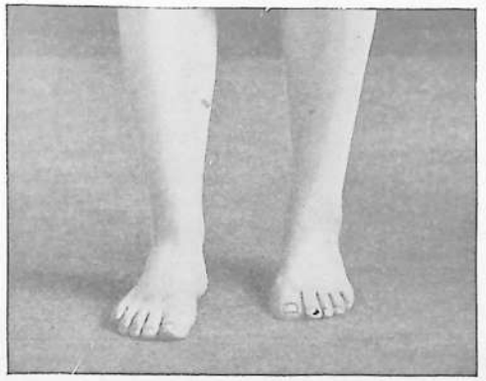

FIG. 268.—**Correct walking position** (toes pointing straight ahead). (Geo. E. Keith Company.)

Shoe men express the *thickness of sole leather* in *"irons"* ($\frac{1}{48}$ inch); a 10 iron sole is thus $\frac{10}{48}$ inch thick. Most women's soles average from 4 to 7 irons, while heavier types of men's shoes are from 9 to 12 irons.

The greatest factor in the **cost of a shoe** is *labor* which represents about 20 per cent of the wholesale cost. The *cost of the sole* is the next largest item. *A shoe which retails for $10 or $12 usually has a factory cost of between $6.50 and $7.50.*

Proper Shoes for Children.—Adams has emphasized the need for room in infant's shoes to permit development during early years. He has noted

the following points concerning children's shoes: 1. The correct shoe for *infants* is one with a soft sole and upper without any conformation to the foot. Infants not yet standing or walking do not need shoes.

2. There should be ample breadth, depth and fullness in the toe.

3. The shoe should be ½ inch longer than the full impression of the child's foot obtained while standing.

4. The heel must be close fitting and well shaped.

5. There should be adequate depth on the dorsum at the vamp from the ankle to the metatarsophalangeal joints.

6. A broad flexible sole with an almost straight outside line is advised.

The heel varies with age: in the baby, the heel and sole are both soft, the "paper bag shoe;" from one to three and one-half years, the heel and sole are of equal height; from three and one-half to five years, the heel is twice the thickness of the sole.

High shoes are preferable in young children especially if they are overweight; this aids in preventing lateral twists of the feet.

Patent leather shoes are not advised for children as these are not porous and may lead to maceration between the toes due to collection of perspiration and resulting irritation of the dorsum of the toes, and the production of corns (as patent leather is not as flexible as ordinary leather).

The child's foot must be accurately measured each time he buys new shoes; do not merely order a size larger shoe. The shoe should be at least ½ to ¾ inch longer than the foot, on weight bearing. *Don't hand the older brother's shoes down to other children unless these fit accurately; this is a bad practice.*

The lack of children's shoes during World War II has unfortunately led to a great increase in foot ailments in children.

Proper Standing and Walking Positions

Feet 4 inches apart.
Toes pointing straight ahead.
Weight on the outer margins of the feet.
Arches cupped as the palm of the hand.
Head up, shoulders back moderately, abdomen in, pelvis up; attempt to flatten the lumbar curve.
Normal convexities of the lumbar and cervical curves and concavities of the sacral and thoracic curves are forward (these do not appear until after the fourth or fifth month of life).
To demonstrate good posture: Stand with the back against a wall, with the head, shoulders, buttocks, calves of legs and heels touching the wall; place the hand between the small of the back and the wall. Abnormal lumbar lordosis produces a large space here. The pelvis may be made to rise by pushing the small of the back against the hand (as occurs when the buttocks are squeezed together).
It is important to move the patient about constantly and to prevent prolonged standing; otherwise there is a natural tendency to stand in the neutral position whereby all strain is on the ligamentous structures.

Stockings.—Schuster has written that our present stockings and socks —made for either foot—act in a detrimental manner and cause deformities of the toes and nails, especially in children. This may be the starting point of a hallux valgus and contraction of the extensors of the smaller toes. A correctly shaped sock should conform to the shape of the foot; it is almost impossible to buy such socks. The sock should be from ¾ to 1 inch longer than the foot (or larger if necessary to allow for shrinkage). Light woolen socks keep the feet warm and absorb perspiration; cotton socks come next in preference, while silk is not to be recommended for one on his feet a great deal.

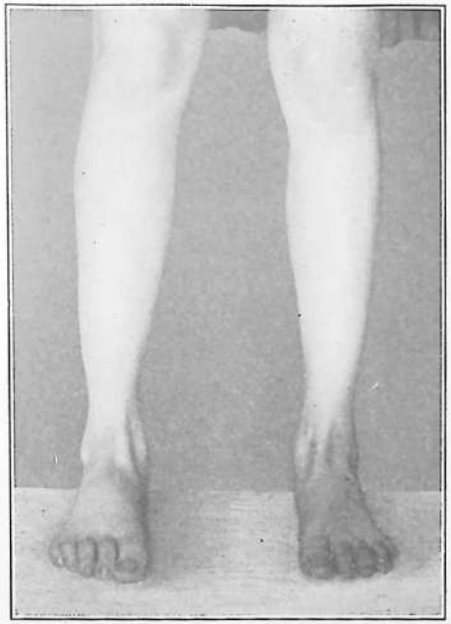

FIG. 269.—Voluntary correction of flat foot, illustrating particularly the restoration of the arch and the normal line of strain. (Whitman.)

EXERCISES AND POSTURAL AIDS FOR WEAK, PAINFUL FEET

Exercise is probably more important than mechanical supports in the treatment of weak feet. Exercises strengthen the lifting muscles on the medial border and transverse portion of the arch (*i. e.*, the tibialis anticus and posticus, flexor longus hallucis, flexor longus digitorum, interossei and peroneus longus). Proper function of the foot muscles prevents painful lowering of the arch and renders mechanical support unnecessary. *Exercises must not be instituted in the acute stages of foot strain as they may aggravate the condition.*

Foot exercises aim to strengthen plantar flexors and adductors and to stretch the dorsiflexors and pronators. **Plantar flexors are strengthened by rising on the toes, while rolling onto the outer sides of the feet strengthens the supinators.**

Foot exercises should be performed barefooted. The following exercises may be used in patients with weak, pronated feet. *Exercises are instituted gradually, are done slowly and regularly, and must never cause fatigue or pain.* At the onset, one or two of the exercises may be performed five times once or twice a day. The physician may select those exercises which are best suited to the individual. These may be changed from time to time to avoid monotony.

The following list of exercises is presented so that the physician may select those best suited to his patient. Usually, 2 or 3 exercises performed well are distinctly superior to a great number of exercises performed poorly, if at all. We employ exercises

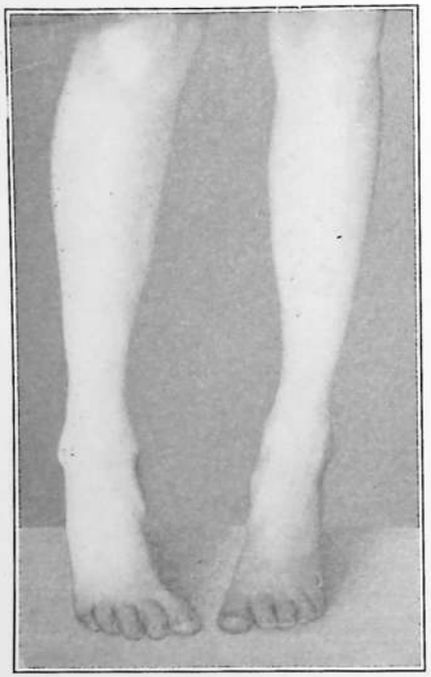

FIG. 270

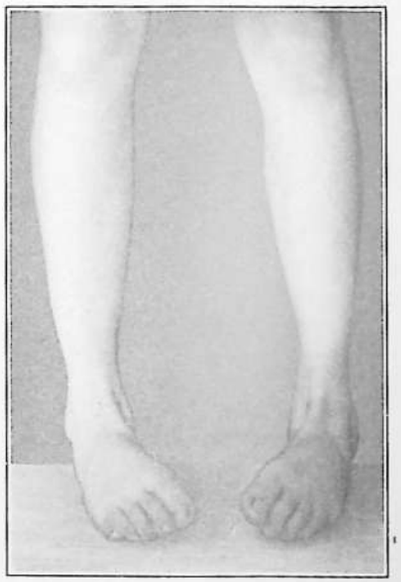

FIG. 271

FIG. 270.—The **tip-toe exercise,** raising the body on the adducted feet. (Whitman.)
FIG. 271.—The tip-toe exercise resting on the outer borders of the feet. (See Fig. 270.) (Whitman.)

number 2, 5, 10 and 12 in most of these patients. *Never begin exercises for painful, uncomfortable feet until symptoms have been relieved.*

1. Ober feels that the best exercise for weak feet is to flex all of the toes strongly at the same time and to dorsiflex, adduct and invert the foot; this position is maintained momentarily. The contracted muscles are then relaxed slowly. The exercise is repeated several times each morning and evening.

2. **Turn the toes inward as far as possible, with the heels out; rise on the toes and come down on outer border of the toes and heel.** Attempt to walk for several minutes each day with the toes turned slightly inward, coming

down first on the heel, then on the outer margin of the foot, and then on the ball of the great toe.

3. Spread and flex the toes sharply downward; this may be done with the shoes off or on.

4. *Stand on the toes with these turned inward, roll out on the sides and return to a flat position.*

5. Place about 10 small marbles on the floor. **While sitting with the bare feet, pick up one marble at a time with the foot and place it in the opposite hand** (*i. e.*, right foot to the left hand and *vice versa*). Repeat this several times a day using a dozen marbles each time with each foot.

FIG. 272

FIG. 273

FIGS. 272 and 273.—**Heel tendon stretching exercise.** (Lewin, courtesy of J. A. M. A.)

6. Walk on the outer borders of the bare feet.

7. With the feet parallel and about 8 to 10 inches apart, slowly rise on the toes and return to the heels.

8. Stand with the toes pointing inward and attempt to externally rotate the knees without lifting the big toe from the floor.

9. While seated on the floor, bring one sole into contact with the other as much as possible.

10. *If it is necessary to stand for long periods, rise on the toes for five to ten seconds every ten minutes.*

11. Attempt to separate the toes widely.

12. *Cross the legs when sitting, with the feet resting upon the external borders;* cup the arches.

13. Walk across the room on the outer side of the feet and return on the toes.

14. Walk in bare feet on coarse uneven ground (pebbles, grass).[100]

15. Grip and throw pine cones or chestnuts with the feet.

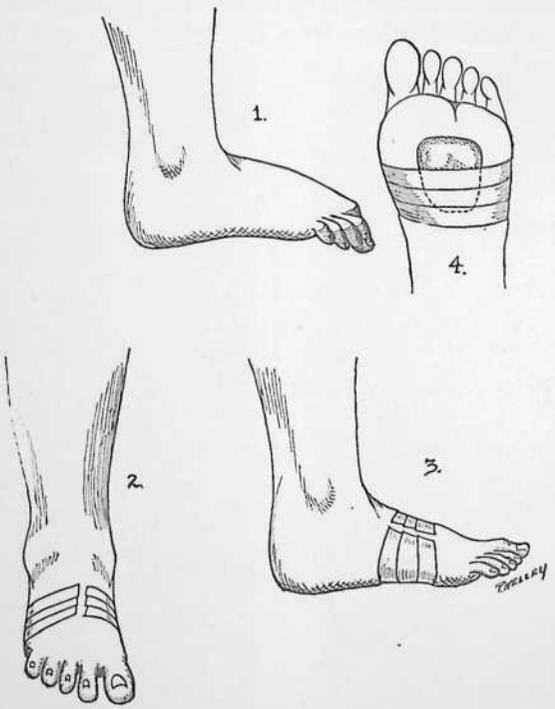

FIG. 274.—**Anterior (metatarsal arch) strapping.** Soft foot pad (as in 4) may be incorporated into strapping. Distal border of the pad must be proximal to the metatarsophalangeal joints. The pad lies over the second, third and fourth (but not the first or fifth) metatarsals. (Dickson and Diveley, Functional Disorders of the Foot, courtesy of J. B. Lippincott Company.)

16. Jump rope.

17. Any exercise requiring moving about on the toes, including tennis, running, baseball, dancing, etc.

18. Daily walk of 1 or 2 miles, preferably at mid-day, gripping ground with the toes to cup the arches.

19. Wall maneuver; chair maneuver (described later).

20. Stand upon one foot (bare) directed straight forward, flex the opposite thigh to a right angle and allow the lower leg to hang straight downward relaxed at the knee; repeat, standing upon the opposite foot.

21. In women with short tendo Achillis, lean forward against a wall with both hands against the wall; push the heels down.

22. *In young children, exercises may be performed if made into a game.*

During recovery from any illness, foot exercises should be performed to prevent painful feet when a return to walking is made; the use of bedroom slippers is inadvisable if there is any tendency to foot strain. Baking and massage are also useful aids in stimulating the nutrition of weakened muscle groups in the legs.

Details of the Chair Maneuver.—Purpose: to adduct the foot, externally rotate the astragalus and push the first and fifth metatarsals into the ground simultaneously.

The patient sits on a chair, with the leg below the knee vertical, foot on the floor.

The physician prevents leg movement by holding the leg above the ankle.

The patient moves only the front part of the foot to a mark placed on the floor $1\frac{1}{2}$ inches internal to the head of the first metatarsal.

The big toe is now depressed until the skin beneath the metatarsal head touches the floor.

This is the proper position for the foot.

Details of the Wall Maneuver.—This exercise consists of walking across the room on the *heel, outer side of the foot and big toe,* while steadying the body with one arm outstretched laterally along the wall.

In this exercise, while walking the heel touches the floor with the foot adducted.

The body moves forward with the weight on the outer side of the foot.

The first metatarsal is depressed while the body weight is still on the outer side of the foot and the body is made to rise on the toes with a spring.

The patient continues to walk across the room in this manner.

ADHESIVE STRAPPING TO THE WEAK FOOT

Pain may often be relieved by the proper application of several strips of adhesive to the weak foot. Such strapping should not be continued for long periods because of the danger of irritation to the skin.

As a temporary measure **for relief of pain in the so-called transverse arch,** place a small felt or sponge rubber pad on the sole just proximal to the heads of the second and third metatarsal bones; hold this in place by adhesive. This pad must be skived at all margins. In most adults, it would average 2 inches in length, $1\frac{1}{2}$ to $1\frac{3}{4}$ inches in width and $\frac{3}{16}$ inch in height (at its highest point). Circular strapping of the bare feet may also relax the overstretched transverse ligaments.

Adhesive strapping may be used temporarily in patients with weak feet. In addition, *the inner border of the heel may be raised about $\frac{1}{4}$ inch to relieve strain* on the dressing and to get the patient used to carrying weight on the outside of the foot.

A *simple form of adhesive dressing* may be applied as follows: (1) Paint the skin with Tr. Benzoin Comp. (2) Have the patient hold the foot in

FIG. 275.—Method of applying adhesive strapping to hold the foot in the adducted attitude. (Whitman.)

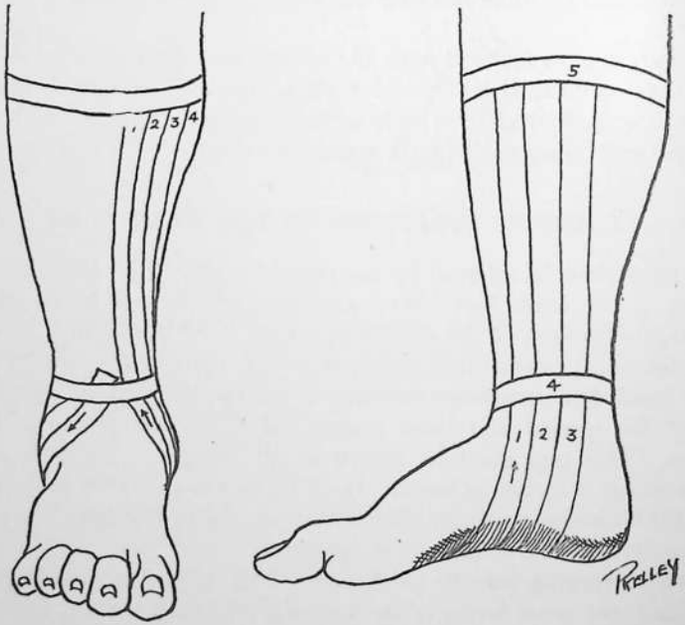

FIG. 276.—**Strapping of the longitudinal arch useful in acute foot strain.** Straps *1*, *2* and *3* are applied firmly. Transverse straps (*4* and *5*) are applied lightly so that no circulatory constriction results. The transverse strips need not encircle the leg but may be of only sufficient length to cover the free ends of *1*, *2* and *3*, and prevent these from slipping. (Dickson and Diveley, Functional Disorders of the Foot, courtesy of J. B. Lippincott Company.)

a mid-position by a bandage sling. (3) Apply a strip of adhesive about 18 inches long and 2 inches wide, beginning on the outer aspect of the foot, passing under the foot and running up the inner side of the leg. (4) A second longitudinal strip may be applied slightly anterior to the first strip. (5) These two longitudinal strips are held in place by transverse strips from 6 to 8 inches long. (6) Gauze is applied around the adhesive to hold this firmly to the skin and to prevent damage to the stocking. The gauze may be removed in twenty-four hours. Don't apply adhesive if there is marked eczema, many thin varicose veins or skin sensitivity.

Additional Aids in the Treatment of Painful (Weak) Feet include: (1) Elastic metatarsal straps with a metatarsal pad for patients with metatarsalgia; (2) the elastic web band where there is effusion in the mid-tarsal joints causing pain along the branches of the median plantar nerve; and (3) sponge rubber heel inserts in patients with tenderness of the tuberosity of the os calcis.[88]

Physical therapy in the weak foot aids in breaking up adhesions and reëstablishing a normal circulation. Corbusier advises the use of a *hot leg whirlpool bath* at a temperature of 100° F. for twenty minutes daily, in addition to massage of the muscles of the leg and foot, manipulation of the foot and ankle joints, stretching of tendons, voluntary inversion, supination and dorsal flexion to over-correct the lesion, and later exercises in bare feet to develop the muscles.

Relief of Painful Feet by Contrast Baths

The acute symptoms of painful, flat feet may be relieved in some cases by contrast plunges or contrast baths. These are combined with periods of rest for the feet during the acute stages of pain, and absence of weight bearing exercises. They may be given as follows:

Two buckets of water are obtained, one warm and the other cool; temperatures should be such that the patient may submerge the feet into these without marked discomfort.

Place the affected foot or feet into the bucket of warm water for one and one-half minutes, then into the cool water for one-half minute.

Repeat this procedure for approximately twenty minutes twice a day always beginning and ending with warm water.

If the patient cannot use these buckets, hot and cool Turkish towels or hot and cool sprays from a bath nozzle may be applied for similar time intervals.

The patient should recline for thirty minutes following the contrast baths. Mild exercise of the toes and ankles during this period of rest will aid in maintaining this circulatory stimulation. Such contrast baths may be used once or twice a day if necessary.

Faradic treatment of the short muscles of the sole of the foot daily for two to four weeks may afford relief from foot discomfort. The foot may be placed in a foot bath and the current passed through the water.

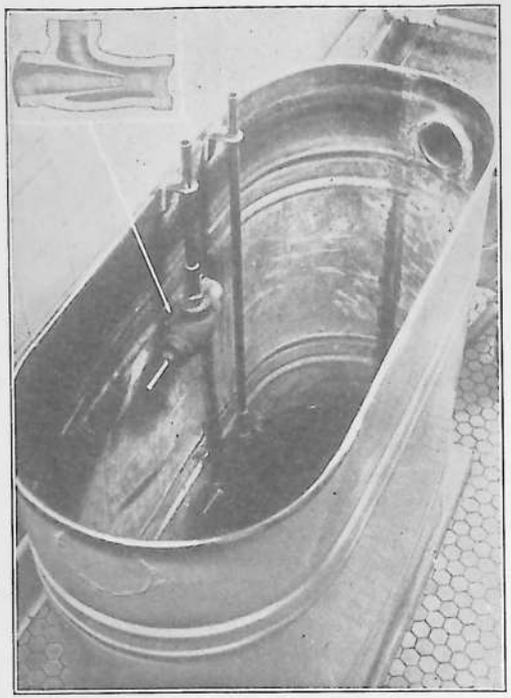

FIG. 277.—**Whirlpool bath.** (Boynton, Arch. Phys. Ther.)

FIG. 278.—**Whirlpool bath.** (Boynton, Arch. Phys. Ther.)

Krusen and Cherry have described a simple mechanical apparatus and a foot exercise block to be used as adjuncts in the treatment of pronated feet.[38] The object of the device is stretching of the gastrocnemius and peroneal muscles simultaneously. The foot block is a simplified substitute for the mechanical stretcher which can be made easily by a good carpenter. Stretching of the peroneal muscles and heel tendon is of value in those cases of weak feet where shortening can be demonstrated.

Flat Feet in Children.—*The feet of most infants are flat at birth and remain so for several months.* Flat foot in young children is often due to a congenital defect; here, one seldom sees pain, but *the child tires easily, walks peculiarly or doesn't care to walk far.* In the later months of infancy, some may require treatment, such as well fitting leather shoes with wedges on the inner side of the sole and heel. *It is very rarely necessary to use arch supports in children with weak feet as there is no marked pain or sensitivity; in these children, one may raise the heel or the inner border of the shoes from* $\frac{1}{8}$ *to* $\frac{1}{4}$ *inch as necessary. Exercises for flat feet are seldom necessary or possible in small children; these may sometimes be instituted as games.*

In all cases of knock knee in children, an examination must be made for weak foot as the latter is a common cause of strain of the internal lateral ligament of the knee.

Spasmodic Flat Foot in Children.—In spasmodic flat foot there occurs a spasm of the peroneus tertius and of the extensor longus digitorum. The main action of the latter muscles is abduction and eversion of the foot; this occurs chiefly at the subastragaloid and midtarsal joints. In most of these cases, spasm of the peroneal muscles is a protective reflex mechanism secondary to some lesion (such as strain or injury) of the midtarsal or subastragaloid joints. *In this condition, the child cannot stand on the outer border of the foot because of spasm of the peroneal muscles.*[50]

Treatment of Spasmodic Flat Foot.—Some individuals feel that peroneal spasm requires no special treatment and that the disease is self-limiting.

Others have reported good results from such procedures as baking and massage to overcome spasm of the peroneal muscles, repeated adhesive plaster strapping to force the foot into slight varus as relaxation of the peronei occurs, application of a plaster splint or cast if necessary, crushing or anesthetizing the peroneal nerve, or injecting procaine into the extensor and peronei muscles or into the midtarsal joints to relieve the spasm.[47,101]

If spasm can be corrected voluntarily, use exercises for the feet, especially for the invertors, reëducation of general posture, inside wedges for the shoes, and splints for the feet during sleep to maintain inversion of the feet.

If spasm disappears only under anesthesia, an orthopedic surgeon should properly manipulate the foot and fix this in a plaster splint in full adduction and inversion for six weeks. Exercises are then used.

If spasm is associated with definite contracture, the peronei must be divided.

Treatment of Advanced Flat Foot.—If the arch is flattened only on weight bearing, the same treatment is used, as outlined previously for foot strain, but arch supports are used in all cases.

In later stages, when the arch has passed beyond control by the patient, even in recumbency, treatment is necessary except in two types of cases: (*a*) congenital flat foot; and (*b*) absence of symptoms.

In advanced painful flat foot, relief may be obtained by manipulation to forcibly remold the arch of the foot and break up adhesions. This type of case should be referred to the orthopedic specialist who, after breaking up adhesions, will usually apply a cast with the foot in the corrected position. After approximately one or two weeks, removable casts and non-weight-bearing exercises are used until the patient has good control of the arch; in severe cases, weight bearing is not permitted for at least a month. Later, arch supports are fitted and weight-bearing exercises are performed. *In these patients, arch supports are usually worn indefinitely. In rigid flat foot due to arthritis, manipulation is usually contraindicated.*

Surgical procedures may be required.[85] Zadek has reviewed a considerable number of operations which have been advised for the treatment of flat foot. He believes that strain and stability in the rigid flat foot take place at the joint between the astragalus and the os calcis. He advises a transverse wedge arthrodesis for painful rigid flat foot, obliterating motion between the astragalus and the os calcis. A wedge of bone is removed transversely at the astragalocalcaneal joint, thereby diminishing the eversion of the os calcis.

If patients with advanced flat foot continue to bear weight, a proliferative arthritis may develop from constant strain. In young patients with such a disturbance, an arthrodesis of the affected joints (astragaloscaphoid and astragalocalcaneal) and correction of the deformity may be necessary; this is not usually done in elderly patients.

DEPRESSION OF THE METATARSAL ARCH

When the foot is not bearing weight, the under surface of the heads of the second, third and fourth metatarsals are on a higher level than the head of the first; the second is the highest of the three, and the fourth is the lowest.

The heads of the first and fifth metatarsals bear weight first followed in order by the fourth, third and the second; when weight is relieved, the fourth, third and second metatarsal heads rise to an elevated position.

The anterior metatarsal arch may act as a shock absorber for the anterior part of the foot; it has no strong ligamentous support and depends upon the function of the flexor and extensor muscles of the toes.

Impairment of the metatarsal arch is very uncommon in children, but is commonly seen in adults, especially women. *Its commonest cause is the high heeled, narrow, short, improperly fitting shoe.*

Depression of the anterior metatarsal arch results in *tenderness and callus over the metatarsal heads* and often leads to *cramps in the calves* and pain in the anterior part of the lower leg; the depression of these heads removes their shock absorbing action. Repeated shocks or trauma to the heads of these bones irritate the soft tissues under them (especially under the second, third and fourth heads) leading to callus formation. This may cause a burning sensation or numbness or tingling in the ball of the foot.

Treatment includes: (1) *Removal of the painful callus* under the metatarsal heads; (2) *baking or diathermy* if the tissues are very tender; (3) *warm foot soaks* in physiologic saline or in water to which epsom salts have been added (if there is marked burning or itching in the soles); (4) felt or sponge rubber supports from $\frac{1}{4}$ to $\frac{1}{2}$ inch thick as a temporary measure or (5) a metatarsal bar and (6) properly fitting shoes. The support should fit between the first and fifth metatarsal heads, curving with the second, third and fourth heads, being thickest under the second and thinnest under the fourth metatarsal heads, and extending back to the tarso-metatarsal junction; anteriorly, it comes just proximal to (but not directly under) the second, third and fourth metatarsal heads. *The convex surface of the pad is placed against the sole of the foot.* On all sides, skive the pad very thin at the margins to prevent annoyance on weight bearing. *This pad may be fastened to the foot with adhesive or may be placed directly into the shoe in the following manner:*

Draw a line over the sole of the foot with an indelible pencil at the proximal margins of the second, third and fourth metatarsal heads; moisten the inside of the shoe at this area and have the patient walk for several minutes with the foot in the shoe. This will result in transference of the line from the foot to the shoe so that the pad can now be inserted correctly into the shoe. Unfortunately, shoe repair men frequently place this anterior pad in position by guesswork, and not by actual measurement.

Many individuals with tender second, third and fourth metatarsal heads are greatly relieved by a **metatarsal bar,** *i. e.,* a strip of leather approximately $\frac{3}{4}$ inch wide and $\frac{3}{16}$ inch thick running across the outer sole from just proximal to the first metatarsal head to a point proximal to the fifth metatarsal head.

Additional treatment includes heat to the lower leg and foot for thirty minutes, followed by massage of the leg and foot for five or ten minutes each day.

COMMON ABNORMALITIES OF THE FEET

Hammer-toe: This usually begins in childhood and is *most common in the second toe* (although not uncommonly it is a congenital defect). It consists of extension at the metatarsophalangeal joint with flexion at the proximal interphalangeal joint; *this is usually caused by pressure of a short narrow shoe or tight sock upon a long toe.* Pain results from the development of a callus or corn over the proximal joint. Therapy consists of manipulation, splinting and the use of felt pads to depress or raise the affected joints in the early mild cases, and surgical resection in the more advanced cases.[87] Medical treatment usually requires months. If the little toe is affected, amputation of the toe may be the best form of therapy.

Hallux Valgus: *The great toe is abducted so that it lies on top of, or under the other toes.* Associated with this is marked prominence of the first metatarsophalangeal joint, with bony enlargement of the inner side of the

first metatarsal head, over which a bursa may form (due to pressure from the shoe).

Hallux valgus may be present without the formation of a bunion and without marked discomfort even in the presence of marked deformity. Short, narrow, pointed shoes or snugly fitting socks are the main cause of hallux valgus.

In mild cases, with slight discomfort, relief may be obtained by the use of proper shoes, insertion of a pad between the first and second toes during sleep, stretching of the great toe, and proper arch supports and exercises. A metatarsal strap will often correct the alignment of the toe in mild cases; the spreading (splaying) of the anterior metatarsal region must be properly cared for and is an essential feature in the treatment of these patients.

Batts has cut out part of an ordinary surgical rubber glove to serve as an efficient, inexpensive and comfortable device. The only portion of the glove retained is the thumb, attached by a ½ inch width of the edge of the glove to the wrist portion; the four fingers and much of the palmar portion of the glove is cut away. The tip of the rubber thumb is then cut off. The thumb compartment is now drawn over the great toe and the ring of rubber usually fitting the wrist, is pulled up over the ankle. The action of this cut glove is similar to that of the stretched and non-functioning abductor hallucis.[6]

Severe deformity of hallux valgus and hallux rigidus can be corrected usually by removal of the exostoses from the metatarsal head and excision of the proximal half of the proximal phalanx.[67]

Hallux Rigidus: This is a limitation of plantar or dorsi-flexion of the great toe usually resulting from bony proliferation at the margins of the metatarsophalangeal joints.

In *hallux rigidus*, permanent subjective relief may be obtained in early cases by a properly made shoe with pads, supports and a metatarsal bar; this condition, however, is difficult to treat. Surgical procedures include removal of the osteophytic ridges with remodeling of the metatarsal head.

Metatarsus Varus: In this condition, the metatarsal bones and toes are bent at the tarso-metatarsal junction so that they are turned toward the opposite foot. Although this is often a congenital anomaly, it may be acquired in patients with flat feet. These patients experience very little inconvenience if they are supplied with shoes which conform to the shape of their feet (*i. e.*, inflare shoes).

Humped Foot: This is a peculiarity of the shape of the dorsum of the foot resulting from a more acute angulation than normal between the internal cuneiform and the base of the first metatarsal bone. Unless this hump is irritated by undue pressure from a tightly laced shoe, few symptoms appear.

Anterior Metatarsalgia may occur in the foot with a high longitudinal arch, the foot with a short Achilles tendon, the abducted foot, or without evident pathologic change; this latter has been called *Morton's metatarsalgia*.

It is characterized by the *sudden onset (while walking) of a severe crampy pain in the anterior portion of the foot, forcing the patient to remove the shoe at once*, and to move and rub the toes until the pain is eased.

This is usually unilateral, affecting females more frequently than males. Schuster attributes it mainly to lateral compression of the forefoot forcing the head of the fifth metatarsal inward and behind the head of the fourth, thus causing pressure on the large superficial branch of the external plantar nerve which lies between the heads of these two bones. This results in a neuralgic pain.

Symptoms of this disorder include: 1. Burning, numbness, tingling or cramplike pain in the forepart of the foot, often under the fourth metatarsal head occurring when wearing shoes (in most instances).

2. Pain may be so intense as to require *sudden removal of the shoe and rubbing of the foot* to obtain relief; this is often relieved by squeezing the metatarsals together. A sense of soreness persists at night in some cases, keeping the patient awake. In long standing cases, a traumatic neuritis is set up causing pain whether a shoe is worn or not.

3. *Tenderness* and numbness may persist for several days in the forepart of the foot or toes especially about the head of the second, third or fourth metatarsals.

4. Pain may radiate up the foot or leg, or to the end of the toe.

5. In some cases, the only discomfort may be an aching across the metatarsal arch.

6. Painful *callus* is frequently seen under the metatarsal head.

A roentgenogram should be made in all cases to rule out arthritis, marked enlargement of the metatarsal head or fracture of the second toe.

The prognosis is often good if seen early, but poor if symptoms have persisted for several years.

Treatment consists of the use of felt pads placed just proximal to the head of the fourth metatarsal bone and a metatarsal elastic strap to decrease splaying of the foot; if the pain persists, a metatarsal bar may be tried. Roentgen therapy has also been used.[36]

In the treatment of some of these patients it may be necessary to inject from 2 to 3 cc. of 1 per cent procaine hydrochloride into the dorsum of the foot between the fourth and fifth toes; this is done behind the fourth and fifth metatarsal heads and at an angle of 45 degrees toward the bases of the metatarsal bones. A 26-gage needle may be pushed in until resistance is no longer noted (about half the length of the needle) and the procaine injected slowly; the injection may be repeated in a week if necessary. Schuster obtained benefit in over 80 per cent of patients by this method.

Morton has advised excision of the metatarsal head if relief is not obtained by conservative therapy; this is seldom required.

McElvenny believed that Morton's toe was caused by a tumor involving the most lateral branch of the medial plantar nerve; these tumors are neurofibromas or angioneurofibromas.[54] If symptoms are severe, he advised that the tumor be excised.

Overlapping Toes: These usually result from narrow shoes (or may be congenital). Such toes may be held in normal position by adhesive strips. If the fifth toe overrides the others markedly, it may be amputated.

Achillodynia: This is an inflammation of the bursa which lies between the os calcis and the tendo Achilles at the point of attachment of the tendon. Symptoms include pain over the bursa with swelling, especially during walking. Treatment consists mainly of rest, removal of pressure from shoes and heat to the bursa; in some cases, it may be necessary to inject this with procaine for relief.

Painful Heel: This may be caused by traumatic rupture of the Achilles tendon, inflammation of the retrocalcaneal bursa, exostoses, degenerative joint disease, strain on the attachment of the plantar fascia or inflammation of a bursa (occasionally present between the tendo Achillis and the skin).[2]

Pain on the sole at the center of the heel on walking may be due to the presence of an exostosis, an inflamed bursa or undue trauma to the tissues in this area. If no exostosis is present (as seen by *x*-ray), relief is usually obtained *by procaine injections* (1 to 3 cc.) into the painful region and insertion into the heel of the shoe of a *soft rubber doughnut, i. e.*, a piece of rubber approximately $\frac{1}{4}$ inch thick with an area about $1\frac{1}{2}$ inches in diameter cut out in its center portion.

Painful heels which have developed during the first few weeks of military training are usually relieved by rest and contrast baths and injection of procaine into the painful area.

Shaffer's Foot: This is a type of claw foot, with contracted plantar fascia and shortening of the gastrocnemius and soleus muscles, which results in limitation of dorsal flexion of the foot. It is characterized by pain in the calves, around the knees and occasionally up the thighs (on walking), due to strain of the gastrocnemius muscle.

Bony Spurs: Bony spurs *on the anterior plantar surface of the os calcis* or on the dorsal surface of the astragalus and tarsal scaphoid *occur more commonly in the absence of gonorrheal infection than in its presence.* Kuhns believes these result from chronic strain at points of ligamentous attachments.[34]

Corns and Calluses: These deserve proper therapy. These can often be removed by several applications of salicylic acid, followed by soaking the foot in warm water. Preparations of salicylic acid which may prove useful are:

(*a*) A 10 to 25 per cent solution of salicylic acid in collodion painted on the callused area nightly for three nights,[58,71] or

(*b*) 10 to 25 per cent salicylic acid in equal parts of lanolin and petrolatum rubbed into the involved area for three nights.

Corns and calluses can usually be lifted out following either of the above, in addition to several soaks in warm water. In some cases which do not respond to the usual routine, roentgen therapy may be necessary.

Corns are always caused by pressure: the latter must be removed or the corn will reform. Felt rings (available in drug stores), placed over the area of the corn, remove the immediate source of pressure, but proper shoes must be worn, which are not too tight or too short. After the removal of a corn, callus or wart, the skin area should be covered for at least several weeks by adhesive or moleskin.

In the presence of ingrowing toe nail with slight inflammatory changes, all pressure must be removed. This includes tight fitting shoes or stockings. A small piece of cotton may be pressed (with a probe) under the edge of the nail, between it and the swollen portions of the toe. Additional relief may be obtained pulling the soft tissues away from the nail further by means of a narrow adhesive strip which circles the toe. In all such cases, the toe nails must be cut straight across and scissors must not be dug down into the corners. Local application of 2 or 3 per cent salicylic acid in ammoniated mercury ointment will aid in decreasing the inflammatory changes. If necessary, surgery may be required to remove a portion of the sharp nail.

Plantar Warts: These may vary in size from pin head to the size of a quarter. They are seen most often in individuals of high school or college age and are most common in front of the heel and on the metatarsal pads but may appear on any part of the foot. The irregularly shaped papilloma lies under the surface and does not project from the sole; it is covered by a horny plate of skin. By means of transmitted light from a nearby area, the punctate appearance is seen through the keratinized layer.

Plantar warts may cause slight discomfort or acute pain. *Most warts are infectious and inoculable,* and are probably caused by a filterable virus. These are rather painful to pressure and may bleed easily. They may be removed surgically or by roentgen therapy. If no treatment is used, a marked local spread of the lesion may occur.

If a single massive treatment with radium or roentgen-ray fails to cure these, radiation should be discontinued; *if recurrent treatment is given, radiation ulcers may be caused.* A long interval of time between roentgen treatments seems to increase rather than decrease the danger.

If radium is decided upon, screen the wart closely. Electrodesiccation, properly performed, has given excellent results.[51]

Pes Cavus: In this condition, there is an abnormally high arch and a tendency for the foot to roll outward. This may be due to unbalanced muscular action (possibly from a short heel tendon) or to a congenital abnormality (with a short, contracted plantar fascia). Most of the weight is borne on the heel and ball of the foot instead of the three usual points; this strains the plantar ligaments and plantar fascia, especially when a low heel shoe is worn.

Symptoms of pes cavus include pain in the ball of the foot, a sense of tiredness in the longitudinal arch, cramping in the calf and occasionally, backache. In addition to the abnormally high longitudinal arch, one notes a tendency of the foot to roll outward, a shortened heel tendon, marked prominence of the ball of the foot, contraction of the plantar fascia, tenderness over the heads of the metatarsal bones and over the plantar fascia, decreased mobility of the foot and the formation of callus over the metatarsal heads.

Therapy of this disorder includes the use of shoes in which the heel is not too low and in which a metatarsal wedge (average $\frac{1}{8}$ inch) has been placed under the fifth metatarsal head on the outer side of the shoe; some orthopedists prefer to employ a metatarsal bar across the sole just proximal to the metatarsal heads. The arch support advised for pes cavus has its high point of the longitudinal section between the astragalar head and the scaphoid (*i. e.*, at the highest part of the arch). Anteriorly the sponge rubber support is high enough and far enough forward to elevate the distal portion of the metatarsal bones and thus relieve pressure on the metatarsal heads. Exercises and physical therapy are also of value.

IMMERSION FOOT

Immersion foot (and its counterpart on land—*trench foot*) are due to prolonged exposure of the dependent lower extremities to cold and moisture. Our medical officers with the troops at Attu and Kiska found that wet cold is far more difficult to deal with than more severe degrees of dry frost.

White outlined his experiences with survivors of torpedoed vessels who developed immersion foot during a prolonged period adrift in the cold water of the North Atlantic.[94,95]

Ungley studied 80 cases of immersion foot seen in Scotland. Exposure lasted for periods up to fourteen days. The immersed limbs became numb, the hands became clumsy and some patients described walking as if on cotton wool. Cramps occurred occasionally in the calves but pain, tingling and itching were unusual. After some days or even hours, swelling of the bare feet was noticed. Webster, Woolhouse and Johnston, three Royal Canadian medical officers, made a similar study of 142 cases of immersion foot resulting from enemy action in the North Atlantic.[92]

The crew in a crowded lifeboat or Carley craft is forced to sit with their wet feet dependent and immobile, thus leading to edema, and if there is an added factor of starvation with lowered serum proteins, the edema will become more pronounced even in the absence of cold. Lifeboats and rafts are not dry and boots are not issued at present to keep the feet both warm and dry. The same condition occasionally involves the knees or buttocks if the man has been forced to kneel or sit on the bottom of a wet boat or rubber raft for prolonged periods.

With exposure to cold sea water for hours or days, the feet may become chilled, numb and swollen, and blisters may develop. The blisters are not usually ruptured (and infection does not usually occur) as these men in the boats have no opportunity to walk on their anesthetic feet (and thus rupture the blisters). White reported[94] that *in 100 cases of immersion foot, amputation was required only twice.*

Individuals with immersion foot show the member to be *swollen, cadaveric in color and blistered.* **Recovery is usually complete even though massive gangrene from the ankle down may appear imminent.** With recovery, there is usually an early *hyperemic stage* and a *later period of fibrosis.* With severe and prolonged exposure, pain may be severe in both of these and may cause prolonged incapacity.

As soon as the legs become warm and the circulation recovers, the *hyperemic phase* develops. This is a *vasodilator response*—a sterile inflammatory reaction due to absorption of damaged tissue. The skin is extremely hot; the deep vessels show bounding pulsations and heat radiates to the surface. There may be profound impairment of the circulation of the superficial tissues as evidenced by *petechial hemorrhages* due to ruptured capillaries and marked congestion of the feet when dependent. When the nerves recover, a *burning pain* develops because of cutaneous anoxia which is due to the combination of heat which increases the metabolic demands of the cells for oxygen and the inadequate superficial circulation. *The toes may remain anesthetic for prolonged periods* although recovery of sensation may take place rapidly in the proximal parts of the feet.

The circulation returns to normal and the hyperemia subsides in from one to four weeks. The *blebs absorb* and the *skin peels off* with some scarring. Some of the mild cases may then return to duty but those with more severe damage have a slow recovery of cutaneous sensation, with limited movement of the toes and pain which makes walking difficult.

Persistent disability may be due to the fibrosis which follows chilling short of frostbite. White and Warren made biopsies of skin and superficial muscle from the feet of men who were still incapacitated several months after their exposure. They found atrophy and thinning of the epidermis and marked fibrosis and deposits of collagen around the nerve endings and subcutaneous blood-vessels. Scar tissue was also found infiltrating the muscle fibrils. The amount of scar tissue reaction depended upon the initial exudation of fibrin and tissue anoxia. This could explain the late pain, rigidity and weakness of the feet which seems to improve spontaneously after six to eight months at which time the collagen ceases to contract. Ungley suggested that prolonged discomfort was due to incomplete recovery of the sensory nerves.[89]

Malnutrition and avitaminosis doubtless *play a rôle* in the pathogenesis of these painful, swollen feet after prolonged periods adrift. *Marked pain, numbness and swelling may occur from several weeks of dehydration and starvation with exposure in the warm waters of the Gulf Stream* due not to cold, but to hypoproteinemia and vitamin B depletion.[96]

Men with immersion foot rescued at sea—**must be kept off their feet.** The *skin should be protected against rupture of the blebs* and pressure sore formation in the weight-bearing portions of the anesthetic skin. If streptococcal infection should begin in these chilled, numb, swollen and blistered lower legs, moist gangrene and possible loss of the leg follow.

These individuals should be carried below and be kept in *bed.* The *bodies should be warmed* with blankets and hot water bottles and warm tea, coffee or soup should be given. A high protein, high vitamin, nutritious easily digested diet should be prescribed as soon as tolerated. *Treatment for shock* may be necessary in some individuals so that *transfusions of plasma* will help restore depleted fluid volume and at the same time, combat hypoproteinemia.

The injured feet must be kept cool (while the body is being warmed).

Cooling of the feet can be accomplished by exposing the legs below the knees to a cool room temperature, and by allowing cool air from an electric fan or blower pass over them. **Do not rub these feet or paint with a strong disinfectant!** *These regions should be treated as a second degree burn with aseptic precautions*, including sulfonamide therapy, a booster dose of tetanus toxoid and masking of the dresser's nose and mouth. Cooling and elevation of the legs aids in draining the edema fluid and shrinking the blisters. This treatment is continued on ship or in a hospital (for a week or more) until the hyperemia has subsided and circulation through the thrombosed subcutaneous vascular bed has recovered.[94] Webster, Woolhouse and Johnston have stressed the need for cooling the extremities in this acute hyperemic stage; the ideal cutaneous temperature is between 70° and 75°, at which point, pain is relieved.[92] Such cooling lowers the metabolic need for oxygen sufficiently so that adequate quantities can be supplied through the injured capillary bed. With cooling of the feet, there is improvement in the appearance of the skin, shrinking of the blisters and relief of pain.

In severe cases, sterile pledgets of cotton are placed between the toes, the foot is covered with a sterile towel and thoroughly dried ice bags are then placed around each foot and over the towel, the whole being incased in an oiled silk bag. Thick layers of insulating material such as cotton waste are then wrapped around this. An outer covering consists of a rubber pillow-case, tied loosely below the knee. From 2 to 4 ice bags may be necessary at first to cool the foot to 70° to 75°. They may be changed about every four hours at which time the toes are palpated to be sure that they are not too severely chilled. Such treatment may be required for several days or a week or two; with subsidence of the hyperemia, less ice is required and cool air alone may be adequate.

The skin desquamates as the circulation returns to normal and blebs absorb. Disability may last for months in severe cases although mild cases may return to duty in two to four weeks. Prolonged disability is due to fibrosis of the skin and underlying tissues which causes intermittent shooting pain at rest and increasing discomfort on walking. The toes are rigid and the feet are weak in these more severe cases. Although physical therapy and exercises may result in some improvement, *in some cases no treatment is particularly effective until six to eight months later when the infiltrating scar tissues cease to contract.* These individuals then become free from pain and may return to light duty. In an occasional severe case, pain has been present for three years or more. *Sympathectomy has been advised for this late stage of fibrosis* by improving the impaired superficial circulation.[95]

Ungley described a condition similar to immersion foot affecting the ungloved hands of an airman who had lain exposed for twenty-four hours on a snow-covered mountain.[90] He disliked the term immersion foot and suggested "peripheral vasoneuropathy."

INFLAMMATORY LESIONS OF THE FOOT

Madden has discussed the common *inflammatory disorders of the skin of the feet:* contact dermatitis, pyoderma, paronychia, psoriasis, lichen simplex chronicus, disturbances of the sweat glands, persistent erythema of the soles, radiodermatitis, urticaria, granuloma annulare, acrodermatitis perstans, pustular bacterid and ainhum.[49]

Fungous infections of the foot include the superficial mycoses (dermatophytosis and moniliasis) and the deep mycoses (mycetoma—or Madura foot, sporotrichosis, blastomycosis and chromoblastomycosis).[14]

The great number of drugs recommended indicates that *there is no specific cure for dermatophytosis.* In mild intertriginous infection, 2 per cent salicylic acid in borated talc may be effective. In acute vesicular cases, relief may be obtained by removing the tops of the vesicles with scissors and applying cold wet dressings of 1 to 6000 aqueous solution of potassium permanganate. In severe infections, the patient should stay off of his feet. With subsidence of the acute process, *potassium permanganate foot baths* may be continued and a diluted *ointment of benzoic and salicylic acid* may be applied, as well as fungicidal agents, such as *Castellani's paint* or 2 per cent *gentian violet.* In chronic infections, *roentgen therapy* may be of value. Infected toenails improve slowly; treatment includes use of *Whitfield's ointment* and Castellani's paint, scraping the surface off the nails before applying medication. Roentgen therapy should be given cautiously over the nails. *It is not advisable to remove the infected nails inasmuch as the new nails tend to be infected.*

Dermatophytosis pedis is difficult to control in the Army. Treatment includes the use of 1 to 6000 potassium permanganate soaks several times a week, exposure of the feet to sunlight and air when possible, use of lightweight hose and 10 per cent boric acid foot powder. Whitfield's ointment or 8 per cent salicylic acid in collodion has been used in the Army for years. Castellani's paint has proved very useful in these cases, being applied three times per week. *Camphor and phenol* seem to be no more effective than Whitfield's ointment, although the former may produce immediate relief from burning and itching. It must be used with caution or burns will result.

Blastomycosis occasionally involves the foot. The lesion usually begins as a small papulopustule which becomes crusted and gradually enlarges by peripheral extension producing sharply outlined patches limited by a rounded or polycyclic border. Microscopic examination of the tissue will disclose the blastomyces within the giant cells in the granuloma; a potassium hydroxide preparation of the pus from a small abscess in the border of the lesion will show the fungi under the microscope. Small lesions may be excised successfully. Large doses of potassium iodide by mouth and roentgen therapy locally are usually effective.

Mycetoma or Madura foot is the only fungous infection involving the deeper layers showing a predilection for the foot. It is *rare in the United States* with less than 30 patients having been reported, mainly from the southwestern states; the disease is endemic in India and parts of Africa.

In most instances the disease has occurred in men who walk barefooted on a farm. The incubation period is very variable, from a month to several years.

A small red nodule appears at the site of injury, usually on the plantar surface of the foot. This enlarges, softens, and breaks down, discharging a yellowish, blood-streaked viscid fluid which contains granules. A sinus penetrates deeply into the foot from each open lesion; these sinuses do not heal. New nodules may appear, enlarge and open and after many years, the foot may become bulbous and its entire surface may be covered by bullæ and pustules. From each of these a sinus passes deeply to the bones, resulting eventually in osteomyelitis. The foot may become markedly deformed and useless and the leg becomes atrophic. Pain is usually absent and the general health seems unaffected. The process is usually extremely chronic.

The *diagnosis* can usually be made early in the disease *by* the presence of *discharging granules* resembling fish roe and by the demonstration of the fungus in these granules (microscopically). Although large doses of potassium iodide may be helpful in the early stages of the process, cure has been obtained in most of the reported cases by amputating the infected foot. There are no reports available on the effect of sulfonamides or penicillin upon this disease.[14]

Most of the recorded cases of **sporotrichosis** in the United States have occurred in the Mississippi Valley region. The offending fungus is widely distributed in the excreta of human and animal carriers and on many plants. The diagnosis is usually made by the appearance of an ulcerating nodule at the site of an injury to the skin, followed by the appearance of a chain of indolent subcutaneous nodules. Other deep fungous infections which may simulate this include tularemia, tuberculosis, syphilis and pyogenic infections. The sporothrix can be demonstrated on culture or by animal inoculation, complement fixation or agglutination tests. Large doses of *potassium iodide* are specific in sporotrichosis; local roentgen therapy hastens involution of the lesions. *Farmers and gardeners* are particularly prone to develop the disease.

Ainhum is a syndrome characterized by a *constriction at the base of a toe,* usually the fourth or fifth toe. A deep groove is formed in the plantar fold. The etiology of the condition is unknown and the disease usually progresses to ulceration or gangrene. *Spontaneous amputation* may occur; in other cases, surgical amputation of the affected toe may be done.

Tumors of the foot are frequently multiple.[42] These include foreign body granuloma; pyogenic granuloma; angioma; angioblastoma; angioneuromyoma; Kaposi's sarcoma (idiopathic multiple hemorrhagic sarcoma); synovial cysts; ganglion; fibroma; xanthoma; melanoma; carcinoma and sarcoma (which is relatively uncommon on the foot).

ARTHRITIS INVOLVING THE FEET

The joints of the hands and feet are often involved in chronic arthritis. The common mild disabilities of the feet may progress to arthritis, if proper treatment is not instituted. It has been stated that adequate early treatment will prevent practically all disability of the feet in arthritis.[39] *In the arthritic patient, foot pain may be due to:* localized pressure, strain of ligaments or muscles, or acute or chronic local inflammatory changes. The effect of contractures of the hip or knee, or faulty posture upon the function of the foot must be considered. Actual arthritis of the small joints of the feet may produce local pain. Stiffness of various joints may result with atrophy of the small muscles of the foot and eventual production of claw shaped toes, dislocations, corns, calluses, etc. Kuhns has studied over 1200 patients with chronic arthritis and has noted that rheumatoid arthritis and degenerative joint disease may cause limitation of motion in the tarsal and phalangeal joints, with muscular weakness; this latter, with weight bearing is the main factor causing progressive deformity.

Treatment of Rheumatoid Arthritis of the Feet[40]

Rest in bed; avoid all weight bearing until pain and swelling have subsided; this may require from several days to weeks.

While in bed; apply *plaster or Castex cast to the foot* molded in such a manner as to support the pedal arches. Keep the foot at right angle to the leg, and the forefoot and os calcis in a neutral position (neither varus nor valgus) in line with the axis of the lower leg. Cut into the anterior and posterior halves as soon as hardened. If muscular spasm prevents full correction, casts may be reapplied at intervals (five to ten days) as the spasm subsides. *When the deformity is fully corrected, wear casts at night only for comfort and support.* Discard the casts when pain and stiffness in the feet are no longer present on awakening and when all tendency to deformity has disappeared.

Remove the anterior half of the cast and apply radiant *heat or hot compresses for twenty minutes several times a day.*

Exercise the feet when muscular spasm and pain subside. Design exercises chiefly for invertors and evertors of ankle, and flexors and extensors of toes (such as inversion of ankle, rotatory movements of forefoot and acute flexion of toes). *Exercise must not cause pain or fatigue* and may be repeated several times a day for periods of ten minutes or less. Passive exercise is forbidden in acute arthritis of the feet.

With subsidence of pain and swelling, permit weight bearing with *well fitting shoes* with firm soles and fairly stiff counters; in the presence of a tendency to valgus deformity in the os calcis, *elevate the anterior medial corner of heel* from ⅛ to ¼ inch. In more severe deformities, use a properly constructed *arch support.* In arthritic feet with midtarsal distortion and associated spasm or fibrosis, foot plates made of malleable metal conforming to plaster of Paris impressions of the distorted feet may produce good results. Such plates are used at first without attempt at correction, but these may be adjusted slowly, at intervals, to obtain correction.

Treatment of Rheumatoid Arthritis of the Feet—(*Continued*)

Additional supports if necessary: leather strap around the forefoot proximal to the metatarsal heads, in patients with spread of the anterior part of the foot and early dorsal flexion of the proximal phalanges; strap under the scaphoid bone and about the malleoli to lessen strain and prevent lateral spreading of the small bones of the midfoot, if there is marked weakness at the ankle joint and proximal tarsal joints.

Heat may now be used in the form of *hot foot baths* for ten minutes one or two times a day.

Gentle massage, especially to the periarticular tissues, may be helpful in weak or aged individuals after the acute stage of the arthritis has passed. *Weight bearing and postural exercises* may be used at this stage.

Continue supervision of the patient for at least a year.

If more resistant deformities occur, Kuhns has advised the use of one of the following:

Repeated adhesive strapping of the foot every four or five days with as much correction as possible.

Series of plaster casts to obtain correction.

Manipulation under general anesthesia.

Operative procedures (to decrease pain, lessen deformity, and to produce a painless member for weight bearing and walking)

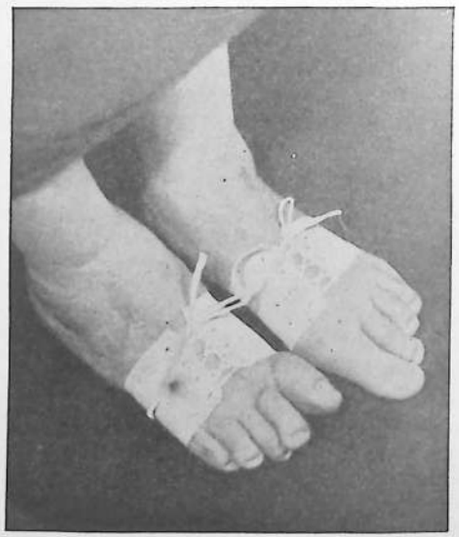

FIG. 279.—**Leather cuff worn about the forefoot** for spreading of the anterior arch and flexion deformity of the toes. (Kuhns, courtesy of Rheumatism.)

Diagnosis of chronic arthritis of the feet in the early stages is suggested by the same symptoms as chronic strain: swelling at the ankle joint and on the dorsum of the foot, and frequently, tenderness and swelling along the

tendon sheaths of the peroneal and tibial muscles, tenderness and thickening over the affected joints and pain on motion. Later, muscular spasm leads to stiffness and deformity about the toes and tarsal joints. In the terminal stages, adhesions, ankylosis and bony deformity may occur.

In chronologic order, Kuhns has noted (in cases with rheumatoid arthritis) eversion of the forefoot, valgus position of the os calcis, flexion deformity of the toes, weakness and atrophy of the muscles of the lower leg, acute flexion of the toes at the proximal interphalangeal joints, extension at the metacarpophalangeal joints, lateral deviation of the proximal phalanges and hallux valgus.

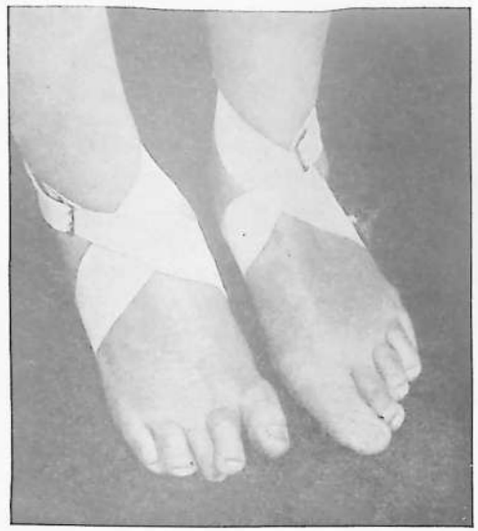

FIG. 280

FIG. 281

FIG. 280.—**Ankle strap to support the midfoot** and relieve strain about the ankle. (Kuhns, courtesy of Rheumatism.)

FIG. 281.—Cuff of plaster which can be worn about the forepart of the foot to prevent spread of the forefoot and to aid flexion of the toes. (Kuhns, courtesy of Rheumatism.)

In most *arthritic feet, rigid shoes are indicated*, with a heel raise applied to one shoe if indicated by the weight distribution test. Felt arch supports are usually tolerated best. *If the forefoot is extremely painful, interior anterior pads cannot usually be worn.* In such individuals, a **metatarsal bar** is extremely helpful in relieving pain in the forefoot. This is placed on the outer side of the sole, running from behind the first metatarsal head to behind (proximal to) the fifth metatarsal head. This is made of leather, approximately $\frac{3}{4}$ inch wide and $\frac{1}{4}$ inch thick.

OPERATIVE TREATMENT

This is not often required in the arthritic foot unless treatment has been neglected. *Operations must not be performed during the active stage*

of the arthritis. Gradual, gentle stretching or manipulation under anesthesia, followed by support with Castex or plaster splints is often effective in correcting arthritic deformity. *Manipulation of the foot to rupture adhesions may be necessary;* this is performed best under general or spinal anesthesia to permit muscular relaxation. Kuhns uses no special technic for manipulation of the foot, but first brings the deformed portion of the foot further into deformity and then slowly and forcibly brings this to the corrected position. If several manipulations do not produce full correction of the deformity, he advises open operation.

The **manipulations of the feet** recommended by Bankart are as follows:[4]

Movements must be forcible. *Damage to the foot with the hands alone is practically impossible. No wrenches or apparatus must be used!*

During manipulative movements, one usually hears adhesions give way.

The 3 movements described below apply to the right foot; for the opposite foot, position of hands is reversed. The surgeon stands at the end of the table facing the patient, who lies on his back with his feet about 6 inches from the end of the table; anesthesia is given.

(*a*) The right hand holds the heel; the hand rests on the table. The left hand is placed under the outer side of the foot and the dorsum; perform plantar flexion, forcible adduction and inversion of the foot, using a pushing and twisting movement.

(*b*) The left hand holds the heel; the right hand grasps the medial aspect of the foot along the inner side of the first metatarsal. Keep the foot extended, forcibly adduct the forepart of the foot and evert the sole with a powerful twist.

(*c*) Hold the back of the heel in the right hand. The left hand grasps the forepart of the foot across the heads of the metatarsal bones. The thumb is on the medial border, the fingers are on the outer border and palm lies against the ball of the foot. Forcibly dorsiflex the foot and twist the sole outward, putting the shoulder behind the hand. An assistant grasps the lower leg firmly above the malleoli to prevent strain to the knee and to protect the bones above the ankle joint from fracture.

Timbrell Fisher is an ardent advocate of manipulation.[23] In **manipulation of the ankle** (as for adhesions) he advises:

General anesthesia with patient lying on his back.

Firmly grasp the forepart of the foot with one hand and the lower leg (above the ankle) with the other.

For dorsiflexion, flex the knee to relax the gastrocnemius and soleus; for plantar flexion, extend the knee.

Exert traction during movements.

If adhesions are present in extensor sheaths, exert firm pressure with the thumb at any especially tender spot and rapidly perform plantar flexion; for adhesions of flexor tendons behind the internal malleolus, exert similar pressure and evert and dorsiflex the foot; for adhesions of the peroneal tendons, invert and dorsiflex the foot.

The midtarsal is the most important of the tarsal joints. It is formed on the outer side by the cuboid (anteriorly) and the os calcis (posteriorly),

and on the inner side by the scaphoid anteriorly and the head of the astragalus posteriorly. Inversion, eversion, abduction and adduction take place at this joint (especially in its inner segment).

Degenerative Joint Disease of the Foot[40]

Severe deformities are uncommon and disability is much less frequent than in rheumatoid arthritis.

Bony proliferation about the articular margins may cause decreased motion in the tarsal joints, leading to pain and swelling after prolonged standing or walking.

Treatment includes correct posture, proper shoes, temporary or permanent use of arch supports, correction of obesity (if present), radiant heat or hot foot soaks, massage several times a day, exercises (later) for correct alignment of the feet and good posture. Rest in bed or plaster casts are usually not necessary, and operative removal of bony proliferations at joint margins is of no value.

Bony spurs may form where there is great strain upon attachments of muscles or ligaments (*i. e.*, under the os calcis, on the dorsum of the foot, and near the base of the fifth metatarsal). Although most of these cause no symptoms, if pain occurs, it is usually relieved by conservative therapy. A foot plate with a hole at the site of the spur or a sponge rubber pad may be used temporarily to relief painful spurs under the os calcis, in addition to injections of procaine. In some cases, simple operative removal of the spur has been advised.

For *painful calcaneal spurs* which did not respond to the use of pads, proper supports, shoe correction, etc., Regan attempted to obliterate the bursa at the tip of the spur by injecting 0.5 cc. of sodium morrhuate in 5 per cent benzyl alcohol after a preliminary injection of a few drops of 2 per cent procaine hydrochloride.[69] Thirty-one of 33 patients obtained relief after the first injection. The solution is injected into the peribursal tissues, producing an inflammatory process which results in the formation of fibrous tissue and obliteration of the bursal sac. No bad effects were noticed from the injection. The solution may cause severe pain if this is injected into the subcutaneous tissues in large amounts. Two or three years of relief was usually obtained, after which subsequent injections were made.

If episodes of acute arthritis of the foot occur (often associated with an acute infection such as a cold or sore throat), treat as in rheumatoid arthritis of the feet. Deformities do not usually occur following these.

Operative procedures are not often necessary.

The deformity in flat foot may become fixed by scar tissue and secondary shortening of periarticular tendons. Slight or moderate degrees of contracture may be overcome by manipulation and proper after-care. **Manipulation of the midtarsal joint** may be done as follows:

The patient lies upon the table with the knee flexed.

Grasp the heel with one hand and the front part of the foot with the other.

Forcibly invert the foot.

In obstinate cases, use a wedge-shaped block covered with leather as a fulcrum upon which is placed the middle of the medial border of the foot.

Follow-up care includes exercises for the muscles controlling the arch, with deflection of weight bearing to the outer side of the foot by raising the inner side of the sole and heel or stiffening the inner side of the uppers.

Immobilization in plaster following manipulation often fails because of muscular wasting. Temporary fixation in plaster may be necessary in infants and children because proper control and after-care is difficult.

In some cases of metatarsalgia, manipulation is of benefit by breaking up adhesions. This is done by grasping the heads of adjacent metatarsal bones by the thumb and forefinger and by moving these up and down in opposite directions. This should be followed by circular adhesive strapping for twenty-four hours, special exercises for the intrinsic muscles of the sole, and an anterior arch support or a metatarsal bar.

After-care of manipulated feet: Maintain the foot in a corrected position by adhesive strapping or plaster cast and apply heat to lessen pain and edema. With subsidence of the reaction, use massage and exercises, arch support (if required) and proper shoes. If extensive adhesions have been torn, the patient should remain in bed for several days.

Surgical procedures may be required in the treatment of severe claw toe, hammer toe, unduly prominent metatarsal head, painful deformity of a distal phalanx, ankylosis of phalanges, tarsal, or tarsal-metatarsal joints, hallux valgus, hallux rigidus, etc. If marked claw toe or metatarsalgia is present, a transverse cleat or anterior heel is used. In patients with persistent pain and old infiltrated tissues, local diathermy is used. Operative details are beyond the scope of this text and should be performed only by capable orthopedic surgeons.

CONGENITAL FLAT FOOT

One should prevent the child from walking barefoot or in slippers.

More than 50 per cent of all small children have weakness and other disturbances of the feet.[70] *Many of these disappear spontaneously* when the child develops better balance and skill in walking.[41] *About 0.5 per cent show persisting ligamentous relaxation of the feet* with displacement of the medial tarsal bones downward and inward on weight bearing. Patients with milder deformity may show generalized relaxation of the subcutaneous tissue and ligamentous structures (the Ehlers-Danlos syndrome).[5] *Children with congenital flat foot all show unusual ligamentous relaxation at birth* which is an aid in diagnosis.[81] In some instances, the dorsum of the foot can be brought to the anterior surface of the tibia.[9] *Depression of the arch on weight bearing is of no importance in examinations before the age of two years.*[8]

The *simplest exercises for children with congenital flat foot* are those given to secure a habitual position of standing and walking with the weight carried on the outer side of the foot; they are instructed to walk along a line with the foot in slight inversion and to stand with the feet parallel with the knee cap rolled outward.[7]

Operative procedures for the foot should not be undertaken before the age of twelve even if there is serious disability. Although most symptoms can be relieved even in adults with proper shoes and supports under the longitudinal arch, operative correction may be needed after the age of twelve (and preferably when full bony development has occurred) in the presence of serious disability. Tendoneses or transplantations of tendons alone are usually not effective.[60] In the less extensive relaxations with little osseous deformity, arthrodesis of the astragaloscaphoid joint or scaphocuneiform joint may be sufficient. Further arthrodeses may be performed especially along the medial side of the foot if a more extensive procedure is required.

Congenital flat foot is relatively common, appearing in varying severity in 0.5 per cent of all small children. In infancy, it may be recognized by the extreme ligamentous relaxation about the foot and the tendency of the foot to go into calcaneovalgus. Serious deformity and disability may appear with growth and changes may be found in the posterior tarsal bones, especially the scaphoid and astragalus.[41] Kuhns has suggested that this is an inherited condition.

Treatment of congenital flat foot should be begun in early childhood, holding the foot while at rest and in weight bearing in inversion and with a slightly elevated longitudinal arch. Proper shoes and supports under the longitudinal arch and the inner side of the heel may be required.

Use firm shoes with the inner side of the heel $\frac{1}{4}$ to $\frac{1}{3}$ inch higher and $\frac{3}{4}$ inch longer than outer edge (*i. e.*, the crooked and elongated heel). In severe cases, use a small leather strip on the medial side of the sole just behind the ball of great toe.

In older children, one may employ exercises for the adductor and invertor muscles.

If conservative measures fail, the Michael Hoke operation may be done (*i. e.*, arthrodesis of the scaphocuneiform joint with the foot in a corrected position); this is followed by the use of arch supports and foot exercises.[31] White has recently described a new surgical approach in these cases.[93]

PLANTAR FASCITIS

Inflammation of the plantar fascia may cause pain in the feet. In about one-half of these patients, there is a preceding influenza or sore throat; in other cases, a focus of infection has been incriminated. This produces *tenderness of the entire plantar fascia coming on several weeks after an acute infection.* The pain in later stages is localized to the region of the heel. Swelling may also be noted.

In those cases following an acute infection, spontaneous cure usually results within a month or two. In other instances, the process may become chronic and persist for years. In one instance a cure resulted within two months following the removal of several infected teeth in a patient whose symptoms had been present for five years.[11]

The main therapeutic attack upon plantar fascitis includes *radiant heat* locally, use of a rubber pad to protect the heel, *procaine injections into the plantar fascia* for temporary relief of discomfort and removal of focal infection.

MARCH FOOT

March foot (also called march fracture) is found most often in young soldiers and nurses, probably as the result of acute foot strain,[18] although this may occur in seasoned soldiers in excellent physical condition (during long marches or athletics).[59]

March foot is a progressive lesion unless it is treated early. Patients with march foot have marched for several miles with pain beginning during the march. Symptoms include dorsal swelling, ecchymosis (in most cases), pain and a palpable mass along the shaft of the involved metatarsal. Continued walking on such a foot may result in an impacted or complete fracture with displacement of the fragments.[12,19]

Bush found that 85 per cent of soldiers with march fractures were wearing shoes one to three sizes too short for their feet, resulting in pressure over the instep or bases of the metatarsals.[12] These may not appear until several weeks after the fracture. *Roentgen examination* in the early stage discloses slight fuzziness due to the formation of subperiosteal new bone on one aspect of the neck of these bones; later, definite new bone may be seen by roentgen examination and the mass may be felt. In some of these patients, a fracture may be found at this site. All of these heal within one to two months. There is no specific therapy.

In patients with complete or displaced fractures, a plaster cast is applied for approximately three weeks; the individual is disabled for three to four weeks. Walking casts may prove useful. An anterior arch pad with adhesive strapping and properly fitting shoes must be worn; the patient must not remain on the feet for long periods until the lesion has healed.

EPIPHYSITIS

Syndromes characterized by definite symptoms and similar epiphyseal changes seen by roentgen-ray include:[56] avulsion of the tibial tubercle **(Osgood-Schlatter)**; tarsal scaphoiditis **(Köhler)**; osteochondritis deformans of the hip **(Legg-Perthes)**;[29] infraction of metatarsal heads **(Freiberg)**; kyphosis dorsalis juvenilis **(Buchmann)** and traumatic malacia of the carpal semilunar bone **(Kienböck)**. It has been noted that *all of these follow trauma, usually occur in growing children, involve epiphyses which are ununited, and are frequently accompanied by rarefaction, fragmentation or abnormal calcification in epiphyses.*

Epiphysitis of the Heel (Calcaneal Apophysitis).[45]—The epiphysis of the heel is subject to direct trauma and strong lateral pull from muscles.

Inflammation occurs most frequently *in boys ten to seventeen years of age.* Involvement is *usually bilateral;* history of trauma may or may not be found.

Symptoms include pain at the back of the heel, aching along the Achilles tendon or calf muscles and pain on climbing stairs.

Examination shows a *tendency to walk upon the toes* to relax the pull of the Achilles tendon, with marked localized tenderness over the attachment of the tendo Achillis, tenderness of the heels posteriorly, and limitation of dorsiflexion with pain on forced motion.

Roentgen findings include: a fluffy, moth-eaten, flattened or fragmented epiphysis of the heel seen in the lateral view; marked irregularity of the adjacent posterior surface of the calcaneum with a punched-out appearance; and a cloudy irregular epiphyseal line.

Symptoms disappear when the epiphysis unites with the calcaneus (at about seventeen years); the epiphysis of the heel appears at about the age of ten.

Epiphysitis of the heel must be differentiated from other painful conditions of the heel which include localized bursitis (under the Achilles tendon or over the heel), tenosynovitis of the tendo Achillis, arthritis, periostitis, tuberculosis of the os calcis and localized osteomyelitis.

The best treatment includes elevation of the heels in order to relieve tension upon the tendo Achillis; local heat and massage to the feet may relieve pain during the acute stage. No strenuous exertion is permitted until the acute stage of the disease has passed. In the occasional resistant case, the foot must be immobilized in slight plantar flexion for several weeks with plaster of Paris casts or Castex.

FREIBERG'S DISEASE

This is a juvenile form of osteochondritis usually affecting the head of the second metatarsal bone.[22,25] *Symptoms* include pain, tenderness, and later, bony swelling in the forefoot. Typical *roentgen findings* are flattening of the head of the second metatarsal bone, followed by areas of sclerosis in the subchondral bone and expansion of the head by osteophytes. *Conservative therapy* includes the use of an anterior arch pad or a thick metatarsal bar on the shoe to prevent weight bearing on the affected area; occasionally, it is necessary to surgically remove the enlarged head of the metatarsal bone.

KÖHLER'S DISEASE

This is an uncommon juvenile type of osteochondritis, beginning insidiously most often in males from two and one-half to seven and one-half years of age.[35] *The essential pathologic finding* is a distortion of the nucleus of ossification of the tarsal scaphoid. *Symptoms* include localized pain, swelling and tenderness on the dorsum of the foot (over the affected scaphoid) and limp.

Roentgen examination reveals marked flattening and irregular outline of the calcified area of the scaphoid, narrowing in the anteroposterior diameter and increased density as though it had been crushed. Roentgen appearances become normal within a year or less. The condition is most frequently unilateral. A normal foot is usually obtained as complete regeneration of the involved bone occurs in an average of less than three years.

No treatment is required except protection against excessive trauma as normal bone formation always occurs; symptoms may be relieved by local strapping or arch support. The disease may be bilateral.

BUNIONS AND THEIR TREATMENT

A bunion is a painful and inflamed bursa overlying an enlargement of the metatarsophalangeal joint of the great toe. Rubbing of the foot against the shoe causes the development of the bursa over the head of the first metatarsal; this bursa becomes thickened, enlarged and inflamed, resulting in a bunion.

Dorsal bunion is usually due to dorsal protrusion of the head of the first metatarsal as a result of the dorsiflexed position of this bone. There is usually a plantar flexion contracture at the metatarsophalangeal joint with a more or less pronounced dorsiflexion contracture of the first metatarsal at the cuneiform joint.[44]

The wearing of shoes which produce no pressure on the first metatarsal head will often relieve mild enlargements and prevent the formation of bunions. Rest and hot compresses or infra-red radiation should be used if the bursa becomes acutely inflamed. *Operation,* when required, usually consists in division of the contracted structures on the lateral aspect, with

FIG. 282.—Hallux valgus. (Shands, Orthopedic Surgery, courtesy of C. V. Mosby Co.)

a bursectomy and a capsuloplasty on the medial aspect; any obstructing bone is removed.[82] For the severe type of bunion, the Mayo operation may be performed; this consists essentially in excision of the head of the first metatarsal and turning in the bursal sac as a flap to make a new joint.[53] Lake does not advocate this operation inasmuch as the bursa may hold a latent infection. Keller's operation consists in removal of the base of the proximal phalanx instead of the metatarsal head with removal of the large exostosis and bony lips on the metatarsal head. In less severe cases the Lapidus operation may be advisable although this is not so satisfactory; this is an excision of the projecting portion of the enlarged head of the first metatarsal, together with fusion of the base of the metatarsal to the cuneiform and second metatarsal.

Tailor's bunion is a bony protuberance at the distal end of the fifth metatarsal from pressure on the small tuberosity at the lateral aspect of the fifth metatarsal head with production of an exostosis.[78]

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CHAPTER LVI

BACKACHE

Ligamentous and muscular strain cause the majority of backaches. *Herniated intervertebral disks*, although a popular diagnosis at present, account for only a small percentage of all cases of backache. Pain in the low back is the most frequent type of backache. Backache is one of the commonest symptoms encountered in the practice of medicine. By the time middle age is reached, almost everyone has at one time or another experienced low back pain.[67]

No panacea has been presented for the treatment of low back pain.[26] Backache may vary from a mild soreness or a dull ache and dragging sensation to a sharp stabbing, intermittent or constant pain which may be increased or decreased by positions or occupation. Many physicians dread to see a patient with backache walk into the office. If an effort were made to enumerate the causes of backache, almost every known disease would be incriminated. The causes of backache are almost without end.[124]

The more important known causes of low back pain include:[124]

1. Bad posture and static abnormalities; poor occupational attitudes.
2. Lumbago (fibrositis of the low back).
3. Trauma resulting in lumbosacral, sacro-iliac or muscular strain.
4. Diseases or abnormalities of the vertebræ (arthritis, tuberculosis, osteomyelitis, congenital malformations, malignant disease—often metastatic).
5. Abnormal conditions or diseases in the tissues in the spinal canal (as tumors).
6. Visceral diseases (pelvic, prostatic).
7. Neuroses, malingering.

ETIOLOGY OF BACKACHE

1. **Orthopedic.**—(*a*) **Faulty posture** (scoliosis, lordosis) and muscular insufficiency; (*b*) *ligamentous or muscular strain* (including acute or chronic lumbar, **lumbosacral or sacro-iliac strain**); (*c*) **weakness of the back** (due to prolonged illness, increasing obesity, sedentary occupation, insufficient exercise); (*d*) **injuries** (including contusions to the lower part of the back: muscles, ligaments, joints and bones); (*e*) contraction of the fascia lata; (*f*) **herniation of the intervertebral disk,** narrowing of the disk; (*g*) deformity of the spine as a result of osteochondritis and epiphysitis; (*h*) **spondylolisthesis**; (*i*) **fractures of the vertebræ** (body, pedicles, transverse processes, lamina, facets), pelvis or coccyx; (*j*) sacro-iliac subluxation; (*k*) post-

operative; (*l*) injury to the ligamentum flavum with subsequent thickening and fibrosis; (*m*) senile osteoporosis; (*n*) **shortening of one leg.**

2. **Congenital Anomalies of the Spine** (which are predisposing factors only).—(*a*) Additional lumbar vertebræ; (*b*) spina bifida; (*c*) changes in size and shape of the transverse process of the fifth lumbar vertebræ; (*d*) difference in the shape and the directional slant of the articular facets at the lumbosacral articulation; anomalous facets, or lumbarization of first sacral vertebra; (*e*) sacralization of the fifth lumbar vertebra.

3. **Rheumatic and Arthritic Disturbances.**—(*a*) **Rheumatoid arthritis of the spine** (*Marie-Strümpell arthritis*); (*b*) **hypertrophic spondylitis, degenerative joint disease** (osteo-arthritis of the spine); (*c*) osteoporosis; (*d*) **fibrositis,** *including* **lumbago** (really a fascitis—rheumatic involvement of the white fibrous fascial planes in the muscles of the back); (*e*) **constriction of the intervertebral foramina.**[117,127,128,156]

4. **Infections, New Growths and Endocrine Pathology.**—(*a*) *Associated with most general infections with prostration and fever* (influenza, pneumonia, the acute exanthemata, undulant fever, tularemia, typhoid fever, etc.); (*b*) general disease with actual muscular invasion: trichinosis, typhoid fever, influenza; (*c*) infectious diseases of the spine: **tuberculosis of the spine,** osteomyelitis of the vertebræ, including typhoid osteomyelitis or periostitis; (*d*) **tumors** *of the spine:* especially Ewing's tumor, metastatic tumors from the breast, cervix and prostate; primary osteogenic sarcoma; myeloma; benign tumors: osteoma, osteochondroma, giant cell tumors, hemangioma; (*e*) focal infection (?); (*f*) *syphilis* and Charcot's disease of the spine; (*g*) osteomalacia, hyperparathyroidism, *Paget's disease* and senile and postmenopausal *osteoporosis* with or without pathologic fractures; (*h*) retroperitoneal infections and new growths; (*i*) rickets;[106] (*j*) diabetes mellitus.

5. **Neurological.**—(*a*) *Herpes zoster;* (*b*) *meningitis;* (*c*) *poliomyelitis;* (*d*) *tumors of the lower cord,* cauda equina or lumbar or sacral nerve roots; (*e*) *tabes dorsalis;* (*f*) syringomyelia; (*g*) radiculitis and nerve root lesions; (*h*) hysteria; (*i*) **malingering;** (*j*) **psychoneurosis;** (*k*) *neuritis* and neuralgia; (*l*) degenerative processes of the spinal cord tracts; (*m*) epidemic encephalitis; (*n*) multiple sclerosis.[134,135]

6. **Referred Pain From:**—(*a*) *Genital organs:* (1) retroversion or retroflexion of the uterus; (2) carcinoma of the cervix of the uterus, especially in the late stages; (3) inflammations of the uterus and adnexa; (4) disturbances of menstruation (including changes during menses and pregnancy); (5) prostatic diseases (acute or chronic prostatitis, carcinoma of the prostate).

(*b*) *Gastrointestinal tract:* (1) chronic constipation; (2) visceroptosis; (3) ulcer or neoplasm of the large intestine; (4) retrocecal appendix; (5) hemorrhoids; (6) spasm of the sigmoid or rectum; (7) colitis; (8) proctitis.

(*c*) *Urinary tract:* (1) renal stone; (2) lesions of the seminal vesicles and prostate.

The Commonest Cause of Backache.—Pain may occur anywhere in the back but its most common location is in the lower portion.

Acute or chronic strain of the low back (occupational or recreational), frequently due to faulty posture and muscular insufficiency, is probably the commonest cause of backache.[8,108]

This may be lumbosacral or sacro-iliac strain or muscular strain in the low back.

The actual lesion is usually a sprain, or slight or extensive tear of a ligament, associated in some cases with muscular injury.[142] Chronic relaxation of the ligaments may follow a loss of resiliency after the hemorrhage which may accompany such an injury.[29] **Most cases of low back pain are due to lumbosacral or sacro-iliac strain.** Exact statistics as to the cause of backache are difficult to find. Patients rarely die of backache so that they are out of the realm of study by pathologists.[48]

In one series of 2050 cases whose chief complaint was backache, the etiology was found to be:[112] chronic arthritis (1050 cases), rheumatoid arthritis (300), sacro-iliac sprain (326), lumbosacral sprain (114), combined sacro-iliac and lumbosacral sprain (85), lumbar muscle strain (100), lumbar myositis (25), spondylolisthesis (22), fractured transverse process (14), fractured articular facet (7), fractured lumbar vertebra (5), Kummell's kyphosis (2). In another carefully analyzed series of 100 patients with a presenting symptom of backache, the following causes were found:[50] **sacro-iliac affections, 32 per cent; lumbosacral affections, 23 per cent** (9 of lumbosacral strain; 4 of anomalies of the fifth lumbar vertebra; 4 of narrowed fifth lumbar vertebra; 4 of calcification of the ilio-lumbar ligaments; 2 of spondylolisthesis); degenerative joint disease (hypertrophic arthritis) 24 per cent; postural strain, 5 per cent; old fractures, 4 per cent; old Pott's disease, 3 per cent; scoliosis, 2 per cent; and 1 per cent each of Paget's disease, congenital anomalies of the first few lumbar vertebræ, metastatic malignancy, congenital anomalies of the sacrum and tumor of the cauda equina.

Judovich and Bates studied more than 1000 cases of low back pain.[82] In their experience, *the most frequently encountered predisposing factors were* lordosis, scoliosis, trauma and spinal arthritis. In some cases, chronic backache was caused by a shortening of one extremity by $\frac{3}{8}$ inch or more; this was sufficient to tilt the lumbar spine and produce a compensatory scoliosis.

The etiology of backache apparently varies with one's specialty. Some gynecologists claim that gynecologic lesions are frequent causes of pain in the low back, while others have stated that backache due to purely gynecologic conditions is relatively rare.[54] Similar statements have been made regarding urologic lesions. In the experience of some industrial physicians injuries to the low back (as its ligaments, muscles, joints and bones) are the most frequent causes of pain in this region;[93] the commonest of these is strain of the back (either acute or chronic).[92] In the opinion of others, myofascitis is the commonest cause. Still others feel that a combination of factors (postural, arthritic, traumatic and congenital) causes most backaches.[68] In introspective patients even minor backache becomes of great importance.

Site of Origin of Low Back Pain.—*Anatomic variations in the lower spine in most cases do not cause pain by themselves*, although they may predispose to weakness of the part. The pathogenic significance of a long impinging transverse process and of sacralization has been doubted.[70,130] Posterior displacement of the fifth lumbar vertebra undoubtedly causes severe pain in the back, as do deficiencies resulting in instability of the lumbosacral joint with pressure on the lumbosacral articular facets.[118,143] **Fifty per cent of patients with architectural weakness of the spine have no symptoms resulting therefrom**; *35 per cent of all spines probably possess some such weakness* (including sacralization, spinal cleft, defective lumbosacral articulation or increased lumbosacral angle).[36]

Steindler abandoned the former classification of sacro-iliac and lumbosacral groups, and followed the routes of sensory supply to the tissues of the lumbosacral region.[151,152] He showed that in a large group of patients presenting what was called the "*posterior division syndrome*" (involvement of the posterior divisions of the spinal nerves), radiation of the pain is a reflex phenomenon, as both it and the local pain could be suppressed by procaine hydrochloride injections.

Most low back pain results from minor injuries to the soft tissues in that region. Steindler has attempted to interpret the syndrome of low back pain and sciatic radiation. *One of the major obstacles in the diagnosis of low back pain is the presence of sciatica.* There is no anatomic proof that sciatic radiation of pain is caused by direct pressure on nerves except in cord tumors and herniations of the disks. Steindler found no proof of direct root compression in congenital anomalies which might cause narrowing of the intervertebral foramina; in his opinion, spondylolisthesis, sacralization, laxity of the neural arch, etc., cause sciatic radiation indirectly by favoring muscular or ligamentous strain.

In many instances of low back pain, sciatic radiation of pain is not due to direct irritation of nerve trunks or roots, but is probably a reflex phenomenon. *The sensory fibers to the injured ligaments, muscles or aponeurotic structures furnish the afferent branch of this reflex arc, connecting with the sciatic nerve possibly in the cord, and not lower than the spinal ganglia.* Steindler has stressed the presence of sharply defined pressure or **trigger points in the back**, varying with the location of the primary injury. *Procaine injection of these points results in disappearance of the low back pain and the sciatica;* irritation of such points before local anesthesia results in exaggeration of the sciatic radiation. Among the *trigger points* enumerated by Steindler are the following: (1) Lumbosacral syndrome: at the *lumbosacral junction;* (2) tensor fasciæ latæ syndrome: *the lateral border of the fascia and the iliotibial band;* (3) sacrospinalis syndrome: at the *posterior superior or inferior iliac spine;* (4) gluteal syndrome: at the *insertion of the gluteus maximus;* (5) transversosacral syndrome: at the *transversosacral articulation* (in cases of sacralization of the fifth lumbar vertebra); (6) myofascial syndrome: *vague points of tenderness along the sacrospinal muscle.*

Judovich and Bates have shown that **irritation of the twelfth dorsal or first lumbar nerves may simulate sacro-iliac and lumbosacral pain or other low back pathology.** The posterior distribution of these nerves includes the upper half of the buttocks, extending medially to slightly within the sacro-iliac joint and laterally to the posterior margin of the great trochanter.

Bates stressed the segmental neuralgias which may simulate the symptoms of visceral disease.[10] He employs regional infiltration to relieve somatic pain and for diagnostic purposes. He has used the pitcher-plant extract and inorganic ammonium salts instead of procaine injections. Judovich obtained an immediate intensification of pain. followed in the subsequent

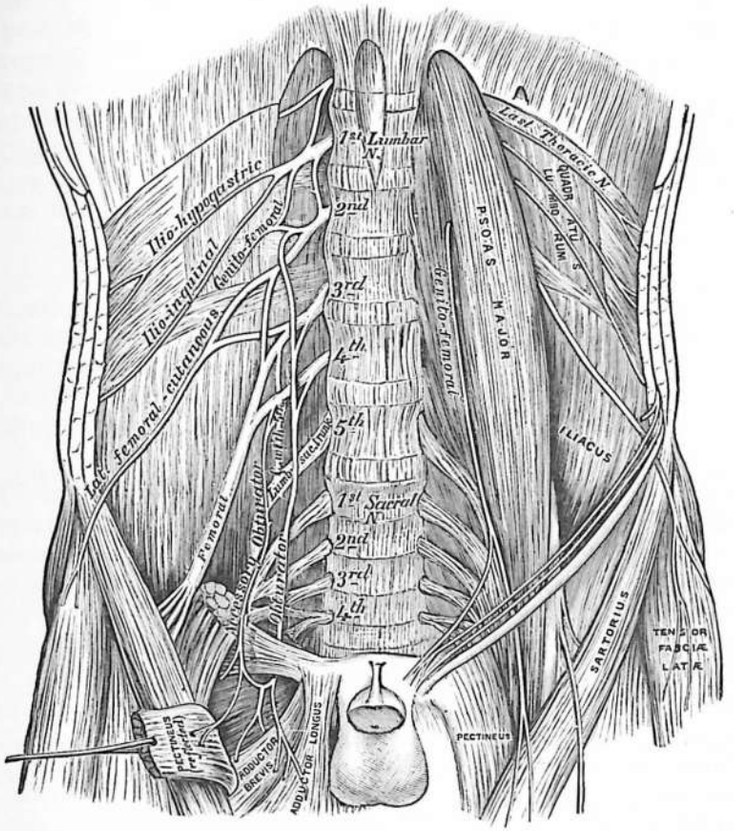


FIG. 283.—The lumbar plexus and its branches. (Gray's Anatomy.)

thirty minutes by a gradual relief of discomfort by *paravertebral injection of the involved root in intercostal neuralgia* by an aqueous extract from the pitcher plant. It was found that the active principle in the pitcher plant extract was an ammonium salt.[154] Perineural injection of 0.5 to 1 per cent solutions of *ammonium chloride* produced the same effect.[11]

The Important Ligaments Surrounding the Spinal Column.—In all lame backs, one must have a good working knowledge of the anatomy of the back. The spine should be considered as a *flexible jointed rod* composed

of 33 bones with its base in the pelvis. The vertebræ are secured to each other by powerful ligaments so that motion between individual vertebræ is slight. Powerful muscles control movements.

The dense ligaments surrounding the spinal column are numerous. These include the *anterior and posterior longitudinal ligaments* (which run the entire length of the vertebral column), the ligaments connecting the arches and those connecting the processes (the *ligamenta flava*, the *ligamenta transversa* and the *interspinous ligaments*). Together with some lesser ligaments, all of these blend together to form a dense sheath for the vertebral column.[167]

The anterior longitudinal ligament is wide and well defined, and shows definite expansions at the levels of the intervertebral disks. The posterior longitudinal ligament is not well defined. The ligamenta flava have been described in detail by Naffziger and his co-workers.[119] These consist chiefly of yellow elastic tissue and join adjacent laminæ and articular processes.

Muscles Situated in the Lumbosacral Region.—It has been estimated that there are 13 pairs of spinal muscles with 67 million possible combinations of muscular imbalance. *One hundred and forty-four muscles attach directly to the movable spine.*

The *psoas muscles* arise from the twelfth thoracic and all of the lumbar vertebræ. The *erector spinæ* arise from the spines of the sacrum, lumbar and 3 lower thoracic vertebræ and the sacro-iliac groove. The *latissimus dorsi* derives part of its origin from the lumbar and sacral vertebræ. The *serratus posticus inferior* also arises in part from the upper 3 lumbar vertebræ.

Why Should Pain Occur in the Lower Back More Commonly Than in the Upper?—This portion of the spine acts as a support for the long vertebral column above, as well as being most active in spinal motion. It is also an area of marked anterior curvature. In addition, bony anomalies are very frequent in this region.

Except for the lower cervical spine, the lumbar spine possesses more direct or indirect visceral attachments than any other portion of the vertebral column. The nerve supply of many organs is derived from this portion of the cord. Powerful groups of muscles attach the legs and the trunk above the pelvis to the lower back. *The lumbar spine, which is freely movable, is itself attached to a relatively fixed pelvis.*

Backache is Infrequent in Children.—*Ligamentous and muscular strain account for the majority of cases of backache.* In children, these tissues are more resilient than in later life and are less easily damaged by acute or chronic strain. The musculature is adequate for their activities and little strain is placed upon the ligaments. Low back pain occurring in children is usually the result of disease of the abdominal or pelvic viscera, acute infectious disease (especially the contagious exanthemata), or neoplasm.

History Taking in Patients With Low Back Pain.—In any patient with low back pain, in addition to a general history, one must obtain a **history of the local condition,** including *possible injury or trauma*, the character of the pain, how long it lasts, what brought on the attack, and what seems to give relief.[124]

Low back pain is most common between twenty and fifty years.[88] *Women often date its onset from pregnancy. In most patients, initial symptoms date from twenty-five to thirty-five years of age when the muscles begin to lose tone. Traumatic accident may initiate symptoms,* but often the onset is insidious with exacerbations after severe or sudden back strain.[15]

The first symptoms are often *muscular fatigue* and ache in the low back radiating out over the sacro-iliac joints into the buttocks. *Stiffness of the back* is often a morning complaint and low back pain may be experienced on sudden, unguarded movements. *Lifting, coughing and straining may be painful. Backache may be accentuated by dampness* and possibly by a focus of infection. *Menstruation* and pelvic inflammatory disease may increase the pain.

Significance of Low Back Pain

Pain in the early morning on awakening, rapidly improving after arising and resuming the day's activities, is characteristic of mild chronic **arthritis** of the spine; the articulations become stiffened during rest and limber up readily when motion is begun.

Pain disappearing during the night and increasing during the following afternoon or evening, with the onset of fatigue following daily activity, is often seen in **postural backaches** resulting from poor body balance.

Pain in the spine due to neoplasm may be *aggravated by immobilization.* Severe pain may accompany **metastatic malignancy** in the spine. This is not relieved by rest on a rigid bed, or by braces, casts or local heat. This is usually relieved only by opiates.

Traumatic *or inflammatory spinal lesions* are usually benefited by rest.

Definite localization of pain without radiation may occur with **fractures** or abscesses of the vertebræ.

Low back pain is the main symptom of lumbosacral and sacro-iliac strains; in these affections the pain may be localized or generalized across the back.

A history of something snapping or slipping in the back suggests a periosteal or **ligamentous tear.**

Recurring attacks of low lumbar backache with sciatica in the back of the leg are usually due to **defective intervertebral disks;** in most instances, the sciatica and backache are intensified by sneezing or coughing during the periods of acute pain.

Traumatic ligamentous or periosteal **lesions** can frequently be demonstrated by localized tenderness with at least temporary *relief of pain following* the local injection of several cc. of 1 or 2 per cent *procaine.*

The patient usually feels best when lying upon a firm, unsagging surface. *Sciatica* may not be present at the onset but occasionally may be the only symptom.

It is extremely important to know whether the low back pain is *continuous or recurrent;* **pain due to metastases is usually continuous,** and is not relieved by rest in bed.

The presence of unilateral or bilateral sciatic radiation with recurring attacks of low back pain, aggravated by coughing or sneezing—is often indicative of a herniated intervertebral disk; such a combination of symptoms—with *recurring attacks*—is seldom seen in other conditions except spondylolisthesis, defective fifth lumbar vertebra with destruction of the articular processes, and tumors of the cauda equina.

Questionnaire for Use With Patients With Low Back Pain.—*When did the pain first occur?*

Did it follow injury (such as a blow to the low back or pelvis) **or any unusual effort or motion** on your part? (such as lifting heavy objects, sudden twisting, pushing heavy objects, unusual athletic activities, prolonged activities in a stooping position, efforts at pulling on a tight shoe or sock, unusual exertion during sexual intercourse, etc.).

Is the pain **continuous or recurrent?**

Did it follow an operation (including delivery)? Have you ever had an abdominal operation?

What is your **occupation?** Have your *changed* this recently?

What is your exact *position during work?*

Does the backache appear after exposure to dampness or drafts?

What is the exact **location of the pain?**

Have you had *similar attacks previously?* How were they induced?

Was there any *sensation of something slipping or snapping in the low back?*

Does any position relieve the pain?

Is the pain accompanied by unilateral or bilateral **sciatica?**

Is the sciatica aggravated by coughing or sneezing?

Is the discomfort in the back associated with *pain or burning radiating around the body?*

What is the *severity* of the pain? Is it sharp, shooting or aching? What is the effect of change of position?

Does this sharp pain persist or is the pain at present really a dull ache with occasional exacerbations of sharp pain on exertion?

Is the discomfort *becoming worse or better,* or is there no change?

What produces an increase or a return of the discomfort?

What has produced relief of the discomfort (position, strapping, rest, heat, medication, etc.)? Is the backache *worse in the morning* on arising or later in the day?

Is the pain *relieved by lying down?*

Is the pain *worse at* the time of the *menstrual periods?*

Have you recently developed a *vaginal (or urethral) discharge?*

Have you ever had *gonorrhea? Syphilis?*

Have you recently **lost** *or gained* a large amount of **weight?**

Are you *constipated?* Are your *bowel movements* of normal size, color and consistency?

Do you have piles (hemorrhoids)? Do they cause symptoms? Do they bleed?

Have you ever had a *renal stone?* Is your *urine bloody or smoky* at any time?

Have you ever had *tuberculosis?* Has anyone in your family had tuberculosis?

Have you recently had a *sore throat, tonsillitis,* sinusitis or any other illness?

Have you noticed small *blisters (vesicles) on the lower back* at any time since the onset of this pain?

Did you ever *previously* have "*lumbago*"?

Do you have a **law suit** considered or pending concerning this condition?

Have you recently worn **new shoes**? Do you have pain in the feet and legs?

Do you *tire easily?*

When was your last *vacation?*

Are you easily *excited?*

Do you sleep well? How many hours do you sleep?

Backache

1. Backache is merely a symptom—not a disease.
2. *All patients with backache must have a general physical examination,* as well as a careful examination of the back.
3. Examination must be done in a good light.
4. One must search carefully for *discrepancies of posture* in patients with backache, including *shortening of one leg.*
5. Any patient with persistent backache should have a *complete roentgen examination of the low back* to check the diagnosis, forestall any serious conditions and aid in making an early diagnosis; *a negative x-ray examination is of definite value.*[124]
6. Examination must include back, front and side views of the patient while standing, as well as side bending, hyperextension of the spine, forward flexion and examination of the back while the patient is sitting, noting in addition flexion of the back and lateral motion.
7. With the patient lying on his back, test for restricted flexion by flexing the knees and hips.
8. Always test *straight leg raising* with the patient on his back.
9. A *neurologic examination* must be performed in any patient with persisting low back pain; this includes examination of the reflexes, sensation and muscle power.
10. Routine *x*-ray examination must include anteroposterior, lateral and stereoscopic views. Special views may be indicated (particularly oblique) for the lumbar facets and sacro-iliac joints. *Roentgen examination may disclose* defective intervertebral disks, congenital abnormalities, malignancy, tuberculosis, syphilis, arthritis, osteomyelitis, osteoporosis, subluxations, spondylolisthesis, dislocations, ankylosis, fractures, abnormal changes in curves, etc.

Radiation of Pain.—The patient's attention may be directed away from the original causative site because of radiation of pain. The physician must be alert lest he ignore the primary cause of discomfort. The sensory roots of the spinal nerves lie in close proximity to the intervertebral articulation so that *even mild affections of the intervertebral areas may produce radicular irritation with referred pain.*[23,47] Inasmuch as *radiating pains may* also *result from intraspinal lesions* (of the cord, etc.), neurologic exam

ination (including tendon reflexes, sensations of heat, cold and pain in the buttocks and lower extremities, etc.) must be performed; the presence of bladder and rectal symptoms may give a clue to a neurologic lesion.

Patients with *recurrent attacks of intractable low back pain and sciatica in the back of the leg* probably have herniation of one or more intervertebral disks (or less often, cauda equina tumors, spondylolisthesis or congenitally defective fifth lumbar vertebra with destruction of the articular processes).

Pain radiating into the legs is common in low back affections, as low grade inflammatory changes occur readily in the ligaments around the joints; the lumbosacral nerve roots lie in proximity to the sacro-iliac and lumbosacral joints. In many cases, **the sciatic pain may be reflex in character.**

Reflex sciatic pain can usually be abolished by local procaine infiltration into a "trigger point" in the low back. (Such procaine infiltration will not lessen the sciatica associated with a herniated disk.)

Narrowed, irregular lower intervertebral foramina or prolonged contraction of the pyriformis muscle (which lies beneath the nerve) may also cause sciatic pain.

Many patients with low back pain note a *marked aggravation of their symptoms on coughing or sneezing.* It was formerly thought that such a condition was met with only in intraspinal lesions producing a spinal block; however, *aggravation of low back pain by a cough or sneeze has been seen frequently in traumatic and non-traumatic types of low back pain,* inasmuch as such acts are associated with spasmodic contractions of the muscles of the abdomen and back.

Relation of Degree of Backache to Trauma.—The amount of disability resulting from a given trauma is not necessarily directly proportional to the physical force of the injury. *Mild injury may result in very painful conditions;* the more important factor seems to be the manner of application of the force. Slight muscle strain or impact may produce fracture of the transverse processes or muscular and fascial tears.

Examination of the Painful Back.—The back is a flexible, mobile structure. The joints of the spine are similar to other joints such as the knee and wrist, except that the former have but a small range of motion. They possess articular cartilage, ligaments and synovial membrane, and are supported by muscles.[87] If their surrounding muscles are overstretched and rendered painful, the joint is said to be **strained.** Overstretching of their ligaments is known as a **sprain;** following this, a traumatic synovitis with effusion and adhesions may occur. The intervertebral spinous ligaments are strong but require the protection of healthy surrounding muscles; injury to the latter permits the ligaments to become overstretched, resulting in relaxation of the joints, chronic strain and faulty posture.

Normally, the *cervical and lumbar curves* are anterior while the *dorsal and sacrococcygeal* are posterior. The apex and juncture of each curve are the *areas of greatest stress* with the most vulnerable points being the cervicodorsal, lumbosacral and sacro-iliac junctions.

A complete physical examination should be performed, including the head, neck, heart, lungs, blood-pressure, abdomen, extremities, genitalia,

etc. The patient's *posture* and *feet* must be examined carefully in all cases of low back pain.

Examination includes location of the pain; search for "*trigger points*" in the low back; localized vertebral tenderness; inspection and palpation of the entire spine and adjacent muscles; measurement of the legs and of the *amount of weight borne on each foot* (using two scales); examination of the feet, reflexes, motions of the spine (lateral flexion to the right and left, and to the right and left oblique; extension to the right and left oblique; straight forward flexion and straight backward extension noting in each exact one finger location of pain; increase or relief by any one motion; any segments of limitation of motion and increase or decrease in referred pain on each motion; rotation); examination of other joints in the body; special examination of the sacro-iliac joint, articulations of the spine and laboratory examinations.[120] A good test of spasm is to watch the patient lie down. In patients with acute low back pain due to tears of muscles, ligaments or periosteum, examination should be limited in extent to prevent further damage. Forceful testing of ranges of mobility and painful manipulations are of course contraindicated in such cases.

FIG. 284.—Example of a simple examining gown giving adequate exposure of the back. Suitable for male or female patients. (Barker and Trescher, Backache, courtesy of J. B. Lippincott Company.)

Additional Examinations in Patients With Low Back Pain.—Is the **posture** good?[63]

Note the general muscular development of the individual and his state of nutrition.

With the patient stripped, the spine should be examined for *kyphosis, scoliosis* or *lordosis.* There is no single factor causing scoliosis. The motor system of the back is maintained in normal balance by the functional integrity of its muscles, tendons, bones, joints, blood-vessels and nerves. Palpate the spinous processes from the upper cervical region to the coccyx, noting any *tender points on the tips of the spinous processes or in the ligaments between them.* **Note especially tenderness at the lumbosacral and sacro-iliac junctions.**

One must determine carefully the location of the pain: lumbar, sacro-iliac, mid-dorsal, over the spinous processes, over the gluteus maximus

muscles, particularly in the upper third or over the erector spinæ muscles, especially at their insertion.

In the patient with backache, the physicians must note any stiffness of the spine, muscle spasm of the erector spinæ muscles, limitation of motion of the spine, sciatic scoliosis, the patient's attitude in standing and walking as well as his method of getting out of a chair or out of a bed.

Palpate on each side of the spinous processes over the **erector spinæ muscles** and the articular processes between the vertebræ noting muscle spasm, inequality of tone, tenseness of muscles or pain. Palpation should also be practiced farther laterally and should include all of the muscles of the back not previously examined.

Other frequent points of tenderness include: the origins of the gluteus maximus and medius along the iliac tuberosities and crests; the pyriformis muscle (mid-way between the posterior superior iliac spine and the greater trochanter) and the fascia lata (over the greater trochanter and between this and the crest of the ilium).[45]

Attempt to reproduce or alleviate the pain by flexion straight forward, laterally, and in the right and left oblique, hyperextension and extension in the right and left oblique. Note the location of the pain if such is produced. Is there *restriction of motion in any segment* of the spine?

Can the patient rest equally well on either buttock, or on both, or does resting on one buttock produce pain in the back?

Does **rectal examination** produce tenderness?

Can the patient sit down and get up easily?

If there is radiating nerve pain, one must learn if this is sciatic, external femoral cutaneous, ilio-inguinal, iliohypogastric or femoral.

Is there tenderness or muscle spasm along the superior part of the gluteus maximus or about the posterior aspect of the greater trochanter?

Is the back relaxed normally when the patient walks, or is it held stiff?

With the patient on his side, does lateral compression over the iliac crests produce pain?

Is the discomfort increased by **raising the leg** (with the knee straight)?

Have the patient lie on his face and bend the knee in order to see if this produces pain or if the pelvis rises or if the tensor fascia lata becomes tight. Then raise both legs together without bending the knees; if there is no pain with this maneuver there is no lumbosacral lesion, but there may be a tight fascia lata.

Elevate the straight leg with the knee extended; attempt to obtain one finger spot of pain if possible. Look for a tear of the gluteus muscle and anesthetize this; if this relieves the pain, the diagnosis of gluteus muscle tear is substantiated. If there is no relief, this is probably a referred pain.

Is there *tenderness along* the course of the *sciatic nerve?*

Is a protuberant abdomen present? Is visceroptosis evident?

Are all of the joints of the lower extremities freely movable and painless? Are other joints in the body involved in an arthritic process?

Is the pelvis level?

Are the lower extremities of equal length and circumference?

Are the *tendon reflexes* normal in the lower extremities?

Is there any **loss in power** in the lower extremities?

Is there any **impairment in the sensation** of heat, cold or pain in the lower extremities or in the buttocks?

Is the sense of position and vibration normal in the lower extremities?

Careful **examination of the feet** (flat feet, points of tenderness, callus formation, deformities, etc.).

Is there any tenderness over the ligaments supporting the tarsal scaphoid or over the plantar ligaments?

What is the site of greatest wear in the patient's shoes?

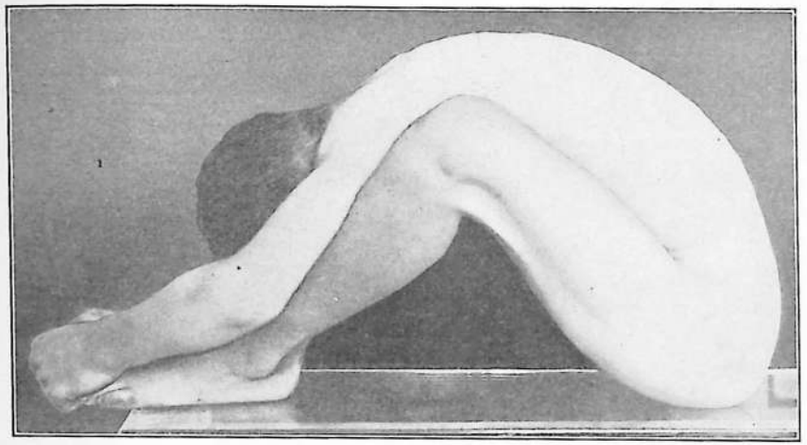


FIG. 285.—Normal contour and flexibility of the spine. (Whitman.)

The Ober Test.—Ober believes that the *ilio-tibial band* may be an important factor in the occurrence of lame backs, with or without an associated sciatica. In his experience, contracture of the ilio-tibial band is not an uncommon cause of low back pain, either in the region of the lumbar vertebræ or sacrum or in the sacro-iliac joints. Sciatica is often associated with this condition.

The test used by Ober to demonstrate contracture of the ilio-tibial band is performed as follows:[122] The patient is placed on his side upon a table so that the shoulder and pelvis are perpendicular to the table.

The patient lies on either side first, and at the conclusion of the examination of this side, the opposite leg is tested with the patient lying on the other side.

The leg on which the patient is lying is flexed at the knee while the hip is also flexed.

The examiner then stands behind the patient.

If the patient is lying on his left side, the left hand of the examiner is placed on the hip of the patient (in the region of the trochanter) to steady him.

The right hand then grasps the right leg (which is flexed to a right angle at the knee) just below the knee.

The right thigh is abducted widely and then hyperextended while abducted; the lower portion of the right leg is kept level.

The right hand of the examiner is passed backward along the leg until it grasps the right ankle, maintaining enough tension to prevent the hip from flexing.

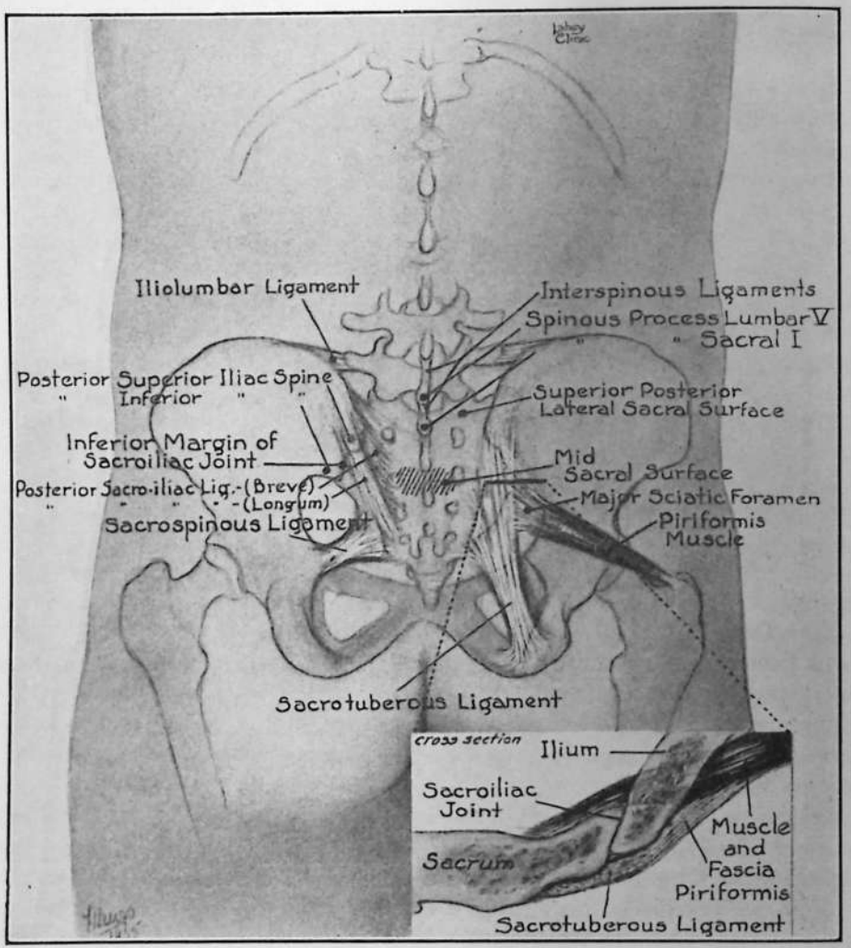


FIG. 286.—Landmarks in the low back. (Haggart, courtesy of Jour. Bone and Joint Surg.)

The examiner allows the thigh to drop toward the table (without bearing down on the leg). *In the presence of a tight fascia lata and ilio-tibial band, the leg will remain abducted,* the degree of abduction depending upon the amount of contracture present.

A caution which must be noted is that if the hip is allowed to flex or to rotate internally, relaxation of the ilio-tibial band occurs and the leg will fall from its own weight, thus rendering the test useless.

This sign has been demonstrated both in the conscious and in the anesthetized individual.

It has also been known to disappear without any reasonable explanation.

Ely's Sign.—Although this sign was described as pathognomonic (it really is not) of sacro-iliac irritation or disease,[42] it has been suggested by Ober that a positive Ely's sign is merely an indication of contracture of the fascia lata.[121] The test is performed in the following manner: The patient lies face downward with the knee flexed.

The leg is then hyperextended at the thigh.

If the test is positive, the pelvis will rise from the table before complete extension of the thigh.

Goldthwait's Sign.—The patient lies upon his back.

Throughout the test, the knee is maintained in extension and the foot is held at 90 degree dorsiflexion to maintain the gastrocnemius (as well as the hamstrings) taut.

One hand is placed under the lower portion of the patient's spine.

The leg is then raised slowly with the other hand.

If pain occurs before the lumbar spine begins to move, the lesion is probably either an arthritis or a sprain of the ligaments of the sacro-iliac joint.

If pain occurs only after the lumbar spine has moved, the pathology is probably in the lumbosacral region (although it may be sacro-iliac).

The test is now repeated on the side opposite to that on which the pain was noted. In this instance, if the lesion is lumbosacral, raising the leg to the same height that produced pain in the other leg should also produce pain in this leg; if, however, the lesion is primarily sacro-iliac, the leg on the less involved side may be raised to a much higher level without pain than on the involved side.

Definite limitation of flexion of the hip with the knee extended (*straight leg raising* or Lasègue test) is of importance. If limitation to flexion occurs during the first third of this act, there is probably spasm of the gluteal muscles; further flexion pulls upon the hamstring muscles and tends to rotate the ilium on the sacrum. Limitation of flexion only in the final third of this maneuver is probably indicative of lumbosacral strain. If pain occurs on only one side, this is suggestive of a sacro-iliac lesion on that side. Lesions of the sciatic nerve or protruded disks also cause limitation of straight leg raising.

Gaenslen's Sign for Sacro-iliac Lesions.—This consists of hyperextension of one hip with the opposite knee and hip acutely flexed to fix the lumbar spine, including the lumbosacral joint. Pain is said to occur only in sacro-iliac cases.

Pointers in Normal Surface Anatomy of the Back.—A median furrow or groove runs from the skull to the sacrum.

The first cervical spinous process which is readily palpable is that of the second cervical vertebra.

The *spinous process of the seventh cervical vertebra* is usually *prominent.*

The *spinous process of the third thoracic vertebra is on a line between the spines of the scapulæ.*

The *spine of the seventh thoracic vertebra is level with the lower angles of the scapula.*

The *line joining the crests of the ilia crosses the spinous process of the fourth lumbar vertebra.*

Determination of Shortening of a Leg.—*A shortened lower extremity may be an important contributing cause of low back pain.* One method of determining shortening of a leg is by means of the *fluoroscope.* Differences of

Interpretation of Physical Findings in Patients With Low Back Pain

If one leg is shorter than another, the pelvis will be tilted downward on the short side.

Normal flexion of the spine forward (with the knees straight) is about 90°; normal backward flexion is about half of this and normal lateral flexion is approximately 40°.

Tenderness in the middle of the spine is usually indicative of *vertebral pathology;* tenderness lateral to the spine is probably *muscular* or indicative of sacro-iliac joint pathology. *Persistent localized tenderness is of utmost importance in localizing the lesion.*

Persistence of the lumbar curve on lying down indicates spasm of the lumbosacral region.

A normal spine arches in the lumbar region on bending forward.

Preservation of the physiologic lumbar curve in as pronounced a degree in the sitting position, as in the standing, usually indicates increased muscular tension or prevention of motion; a congenital spinal abnormality may also be present.

Symptoms from congenital defects of the lumbosacral joint appear usually in the third decade. The most frequent distribution of pain is in the 5th lumbar and 1st and 2d sacral segments.

Patients with *lordosis* (commonly from poor posture) may complain of pain only after being in bed.

A *painful coccyx* may be palpated by rectal examination; the sacro-iliac joints should also be examined per rectum.

A painful coccyx may be suspected if the patient can sit on either buttock but not on both.

Patients with low back pain associated with contracted fascia lata lie on the side with the knee or knees flexed; lying on the back or abdomen usually produces more discomfort.

In low back pain associated with a contracted fascia lata, tenderness about the posterior aspect of the greater trochanter is common.

Lateral compression over the iliac crests may produce pain in patients with sacro-iliac sprain.

Sacro-iliac strains are probably much less common than is generally believed.

Interpretation of Physical Findings in Patients With Low Back Pain—*(Continued)*

Signs of sacro-iliac strain include point tenderness just below and lateral to the posterior inferior spine, and pain on marked compression of the anterior superior spines of the ilia.

Patients with lumbosacral strain are usually most comfortable when lying on the unaffected side, with moderate flexion of the body, hips and knees, and with a pillow between the knees.[84]

In patients with lumbosacral strain, there is practically no movement in the low back in the erect posture, and acute tenderness is present in the lumbosacral region of the affected side.

The pain in the lower extremities accompanying lumbosacral anomalies is segmental in distribution and is localized to the distribution of the 3, 4 and 5 lumbar and 1, 2 and 3 sacral segments.[165]

Patients are usually uncomfortable in bending toward the lesion in lumbosacral and sacro-iliac strains, and in bending away from the lesion in injuries of muscles or fascia.

Patients with low back pain commonly exhibit tenderness and muscle spasm along the superior part of the gluteus maximus muscle.

List is more commonly away from the side which is affected.[144]

Limitation of straight leg raising is present in sciatica, short hamstrings, contracture of the gluteal fascia, spasm of the gluteal muscles, lesions of the lumbosacral or sacro-iliac joints, or herniated intervertebral disks.

Localization of the pain may be aided by asking the patient to lift a heavy weight, or to stand on his toes and drop suddenly on his heels.[91]

Pronation of the foot leads to foot strain and often to backache;[102] a low pedal arch does not necessarily indicate a weak foot.

In overstrain in the feet, tenderness is often present in the plantar ligaments, or in the ligaments supporting the tarsal scaphoid.

Constantly pronated feet often wear down the posterior lateral edge of the heels but throw weight on the medial edge of the soles, so that the shoe is more worn over the ball of the great toe than over that of the small toe.

leg length of 1 mm. may be detected accurately, by using a *specially prepared fluoroscopic screen* through which run a vertical and a horizontal line at right angles to and bisecting each other. The method is simple. Observations are usually made of the levels of the iliac crests (or of the roofs of the acetabula or of the lateral or medial condyles). The patient stands with the weight-bearing points of the feet the same distance apart as the femoral heads. The levels of the iliac crests are examined fluoroscopically using the special screen. To determine the amount of shortening of the leg whose iliac crest appears lower than the other, an ordinary telephone book is used, pages being placed under the foot of the short leg until the iliac crests are on the same level; the height to which the foot must be raised indicates the amount of shortening of the affected leg.

Simplified Office Procedure to Determine Shortening of a Leg.—A simple modification of the fluoroscopic method permits its use by general practitioners. The necessary equipment includes a piece of black thread several feet long, a small line level (which may be procured in a hardware store for less than fifty cents), and two thumb tacks. The patient stands barefooted in any doorway in the office. A thumb tack is placed in the wooden support of one side of the doorway at approximately the height of the

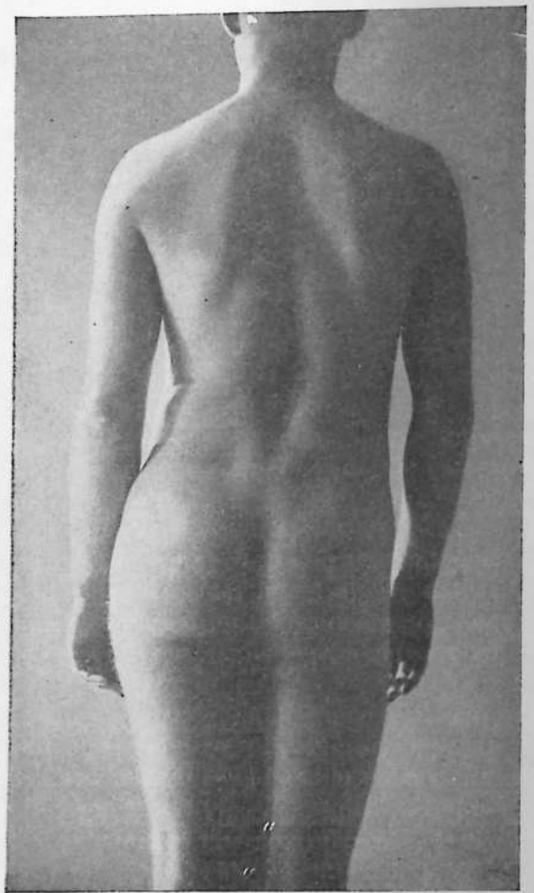


FIG. 287.—Showing postural scoliosis with lateral tilting of the pelvis due to a short right leg. (Barker and Trescher, Backache, courtesy of J. B. Lippincott Company.)

anterior superior spines of the ilium. The black thread is attached to this, the line level is threaded on the line, and the free end of the black thread is attached to the opposite side of the doorway at such a point that the line is level, as shown by the instrument. A small dot is made by a pen on the skin over each anterior superior spine. The examiner now sits on a small stool in front of the patient and observes the position of the black thread in relation to the anterior superior spines. If these are both on the

line, the legs are considered to be of equal length. If one is lower than the other, a sufficient number of pages of a telephone directory are placed under the short leg until both anterior superior spines appear on the same level. The height of these pages is approximately the amount of shortening of the affected leg. Throughout the procedure, the feet of the patient are placed squarely with the toes pointing straight forward and the feet approximately the same distance apart as the femoral heads.

Individuals who practice leg measurement usually do so in the following manner: The patient lies squarely on his back on a flat table or couch.

The anterior superior spines and the internal malleoli are marked.

Measurements are taken with a steel tape from the anterior superior spine of the ilia to the internal malleoli. This measurement should be checked several times as discrepancies are frequent.

Correction for Shortening of a Leg.—Differences in leg length of $\frac{1}{4}$ or $\frac{3}{8}$ inch are common and are generally considered as normal variations; such an amount of shortening rarely produces symptoms but may be a possible factor in the causation of backaches, and should be at least partly corrected if backache is present. A difference of $\frac{1}{2}$ inch or more is apt to produce trouble and in general should be relieved; otherwise, this may lead to compensatory changes in the upper part of the skeleton.

Tilting of the pelvis due to a short leg may produce strain at the sacroiliac joint and lead to sciatic pain.

In some cases, a short leg may result from poliomyelitis, tuberculosis of the hip or knee joint, osteomyelitis of the bones of the leg with surgical intervention, fractures, epiphyseal injuries in young individuals, etc.

If there is only moderate shortening, this may be corrected by a heel lift plus a small heel pad within the shoe. If marked shortening is present, a cork elevation may be used inside of the shoe; in some patients, this must be carried forward to the toes. If a very great difference in leg length exists, a cork extension may be placed on the outside of the shoe, supported by steel double uprights extending almost to the knee. If the shortening is more than 6 inches, a steel extension with a leather sole is less expensive and lighter than cork.[141]

Significance of Changes in the Tendon Reflexes in Patients With Low Back Pain.—Patients with back pain complicated by **sciatic neuritis** usually show a diminished or *absent Achilles reflex.*

Primary, or more frequently, metastatic *tumors of the spinal cord* or vertebræ with compression of the cord may produce reflex changes in the lower extremities.

Reflex changes may also be present in patients with low back pain due to tabes dorsalis, infections of the spinal cord or meninges, etc.

Diminution or absence of ankle jerks may occur following *herniation of a lumbar intervertebral disk.*

Herniation of the fourth or fifth lumbar disk may show a diminished or absent Achilles reflex on the side of the sciatica.

Roentgen Examinations in Low Back Pain.—A roentgen-ray examination of the lame back may forestall serious conditions and will often aid

in checking the diagnosis. If there is any question as to the correct diagnosis, **don't guess and don't wait for someone else to order the roentgen examination.**

Laboratory Aids in the Diagnosis of Low Back Pain

In some patients with low back pain, one is not certain of the diagnosis after a detailed history and careful physical examination. In these, recourse may be had, if necessary, to the following examinations:

Complete *blood count*, *urinalysis* and blood *Wassermann* reaction (which should be performed *routinely* on all new patients).

Blood *sedimentation rate* (to indicate the presence of an inflammatory or destructive process).

Roentgen-ray the lame back for rheumatoid conditions, degenerative lesions, trophic diseases and various tumors. A negative roentgen-ray of the lame back may be very important.

Roentgen examinations:

Of the affected portion of the spine: anteroposterior, lateral and oblique (three-quarter view with the patient turned 30° to either side). The oblique view assists in visualization of the articular facets.

Of the lumbosacral angle.

Stereoscopic examination of the pelvis and sacro-iliac joints.

If necessary (rarely), anteroposterior views of the entire spine.

Of the pelvis with the patient standing on each leg separately to determine sacro-iliac relaxation.

Of other organs (kidneys, gastrointestinal tract, etc.) if indicated to rule out renal calculus, gastrointestinal lesions, etc.; these are made only if directly indicated (by the history or results of the physical examination).

Lumbar puncture (if necessary); if indicated, insertion of air, pantopaque or lipiodol followed by roentgen examination.

Special blood chemical studies: Blood uric acid, calcium, etc.

Eighty to 90 per cent of all backache is probably caused by postural defects with acute or chronic ligamentous or muscular strain; roentgen examination is of little assistance in diagnosis in these patients. Roentgen examinations may mislead the physician by revealing an abnormal condition (such as a congenital anomaly), which may not be the actual cause of the patient's low back pain. However, *such an examination is always indicated in patients with low back pain not responding to the usual regimen of therapy*. In this manner, one may find an arthritis involving the articular facets, congenital anomalies which may predispose to a weak back, spondylolisthesis, Paget's disease, metastatic malignancy (in the later stages), herniation of the nucleus pulposus (if lipiodol or pantopaque is instilled), etc. Roentgen examinations may disclose sacro-iliac relaxation. Poor quality of roentgenograms taken in the wrong position or at wrong angles, may lead to error. In some cases, a compression fracture or spondylolisthesis may be noted only by a lateral view. Roentgenograms made with the patient not lying straight on the table may lead to a false impression of spinal curvature.

The **minimal roentgen examination in the patient with undiagnosed low back pain** includes anteroposterior and lateral views of the lumbosacral regions (including the lower lumbar vertebræ, sacro-iliac joints and sacrum). Slight changes in the sacro-iliac joints cannot be determined accurately by flat plates; stereoscopic anteroposterior views may be required. In many cases, oblique views are required to demonstrate the lower lumbar facets. By means of carefully prepared charts the interpedicular space may be measured at each vertebral level in an effort to discover the cause of back pain.

FIG. 288.—Showing slumped posture with head forward, chest drooping and the abdomen protruding, with increased lumbar lordosis. (Barker and Trescher, courtesy of J. B. Lippincott Company.)

Special Roentgen Technic for Sacro-iliac Relaxation.—By means of a special technic with the patient standing on each foot separately, roentgenograms are made which may show evidence of undue sacro-iliac relaxation as demonstrated by an abnormal range of motion at the pubis. If roentgenograms of the pelvis are made in a normal patient standing on each leg separately, not more than $\frac{1}{2}$ mm. of motion in males or 2 mm. in females occurs at the pubes. This can be shown if the two films are compared. Patients with sacro-iliac sprain may experience pain when standing on one leg although the sacro-iliac joints themselves may appear normal in the roentgenogram.

The usual horizontal films are made first. The table is then tilted to a vertical position and the patient stands, facing the table, on a box several inches high. He bears his entire weight on one foot, the other leg hanging over the edge of the box. A film is made, the hips being supported by the usual table belt. Another film is made of the other hip using the same technic, the weight being borne on the opposite foot. In this examination, not more than 2 mm. difference is noted normally in the variation of the level of the symphysis. If this is greater than 2 mm., sacro-iliac relaxation is present.

Sacro-iliac relaxation with pregnancy comes on early, in some cases having been noted five weeks after conception. Pre-menstrual low back pain may be due to sacro-iliac relaxation in some patients, and changes may be demonstrated at the symphysis pubis by roentgenograms made with the patient standing on each leg separately.

Lumbar Puncture.—*Lumbar puncture should be performed in patients with suspected cord lesions, tabes dorsalis, meningeal infections* and in some cases of possible *herniation* of an *intervertebral disk.* The pressure of the cerebrospinal fluid is noted and a test is made by jugular compression for the presence of a block in the spinal canal. If indicated, radiopaque solutions such as pantopaque, campiodol or lipiodol, or air may be injected into the spinal canal (Caution!) following removal of an equal amount of spinal fluid to determine the site of a cord lesion (especially tumor).

Concealed disks may show no signs with contrast myelography. Negative roentgen findings do not rule out herniated disks. The symptoms of recurring attacks of severe low back and sciatic pain in the back of the leg are more diagnostic than the *x*-ray in most instances of herniated disks.

The spinal fluid should be examined for *cell content, protein* and *Wassermann test;* other tests are made if indicated.

If a herniated disk is suspected, the first few cc. of fluid obtained should be examined for protein content.

Blood Chemistry.—Blood calcium should be determined in patients with suspected hyperparathyroidism. Gout is rarely a cause of low back pain; however, if other evidence of gout is seen (tophi, intermittent attacks with free intervals, involvement of a great toe), a blood uric acid should be performed.

FAULTY POSTURE

Faulty body mechanics may hasten the appearance of symptoms by causing malalignment of the spine and inefficient functioning of the supporting structures. Congenital anomalies may permit a slight trauma, such as sudden unguarded movements to produce a severe sprain of the low back.

One of the commonest factors tending to make a back less strong is faulty posture. This is particularly important in middle and later life. Seventy per cent of young men entering college do not exhibit good posture.[69]

Most postural defects result from an exaggeration of the normal lumbar lordosis with which there is an associated forward tilt of the pelvis.[69] Backache has been called the price which man must pay because of his departure from the position of the quadruped. *Ninety per cent of all backache results from faulty body mechanics or postural disturbances due to strain of ligaments, muscles or joints of the back with chronic weakness of the part.*[34,56] Because of the great increase in the anteroposterior curves of the spine in these patients (especially in slender individuals), the spine is being used near its extreme point of possible movement, so that there is no factor of safety for motion. *Strain of the supporting ligaments* then results from the continued use of such faulty posture; any sudden force may result in acute strain. Scoliosis will also lead to back pain in later life from strain, muscular fatigue or pressure upon nerve roots.[53] For a more detailed discussion of the study of body mechanics, the reader is referred to the excellent text of Goldthwait and his associates.

Evidence of Good Posture
Head straight above the chest, hips and feet so that a plumb line from the mastoid process bisects the tip of the shoulder and hip joint and scaphoid bones of the feet and is slightly in front of the center of the knee joint.
Chest up and forward and the abdomen in or flat.
Normal curves of the back not exaggerated.
Knees slightly hyperextended.
No evidence of weak feet.
Evidences of Poor Posture
Forward posture of the head.
Flattening of the chest.
Protuberant abdomen.
Exaggeration of the dorsal kyphosis.
Exaggeration of the lumbar lordosis.
Downward tilt of the pelvis.
Marked hyperextension of the knees.

Other conditions which tend to result in a "weak back" include prolonged illness in bed, increasing obesity, sedentary occupation, insufficient exercise, poor sitting posture in a chair or automobile, etc. These may lead to decreased muscular tone. Pronation of the feet may produce strain upon the knees and back.

LUMBOSACRAL AND SACRO-ILIAC STRAINS

These lesions probably cause a large percentage of all backache. *Of the patients with low back strain, about four-fifths involve the lumbosacral and about one-fifth the sacro-iliac region.* The lumbosacral joint is much more unstable than the sacro-iliac joint; the latter is strongly constructed. **Strain or slipping in the low back usually results in injury to the more vulnerable lumbosacral joint.**[64]

The lumbosacral junction is the most vulnerable portion of the spine and is subjected to the greatest strain. Its joints bear the body weight and any additional weight lifted by the upper extremities or carried on the shoulders. *The foramina between the sacrum and the fifth lumbar vertebra are the smallest of the intervertebral foramina, but contain the largest spinal nerves;* these nerves and their surrounding plexus of veins almost fill the canals. The fifth lumbar nerve may be affected by a slight inflammatory change in the posterior articulations, by extrusions of the nucleus pulposus, by changes in the shape or size of the canal secondary to displacement of the fifth lumbar vertebra or sacrum, or to atrophy of the intervertebral disk.

The chief etiologic factors of sacro-iliac and lumbosacral sprain are trauma, posture and occupation; these are responsible for 80 per cent of such cases. *Other causes include* abnormal weight distribution secondary to hip joint disease or short leg; mechanical instability of the foot; toxic states with

acutely weakened musculature; relaxation of the pelvic floor with retroversion; pregnancy; pelvic inflammatory disease; obesity with a pendulous abdomen; psychoneurosis, etc.

The lumbosacral and sacro-iliac joints are under excessive strain whenever the body is erect, because of the obliquity of the sacrum, and of the angle between the sacrum and the fifth lumbar vertebra made necessary by the change from quadruped to biped walking. They are frequently asymmetrical in shape and size, predisposing to instability. They may be damaged separately and injury to one is reflected in extra strain on the other.

All four of these joints should be considered together and all structures of the low back may be considered as one large joint. Under such circumstances *sprain of the back* is much the same as of the ankle. *When a sprain occurs, there is tearing of ligaments of one or more of the four joints, producing hemorrhage in the periarticular structures and possibly, effusion into the joint.* Rupture of the ligaments is painful because of damage to their sensory nerve endings and pressure on nerve roots in the immediate neighborhood of the joint. Symptoms and signs are somewhat similar whichever joint is affected.

Lumbosacral Strain.—The lumbosacral junction is the great fulcrum of the body. Any change of the sacrum toward the horizontal position or increase in lumbar lordosis predisposes to lumbosacral and lower lumbar strains. With an increase in the horizontality of the sacrum, the lumbar lordosis is exaggerated; this increases the shearing stress between the articulations of the lower 3 lumbar vertebræ as well as the lumbosacral angle.

If gradual strain occurs from postural causes (most often in women), the pain is insidious in onset and the disability becomes chronic (with intermittent symptoms for months or years before a physician is consulted).

The backache of postural lumbosacral strain is usually greater at night and may awaken a patient from sleep; in these patients, relief is obtained by sitting up in bed, by using a lumbar pillow or by getting out of bed.

Postural lumbosacral strain may produce pain on getting up in the morning; this pain may soon wear off. It is localized in the low back in the lumbosacral region and may not radiate down the legs. The discomfort is more in the nature of a dull ache.

Sacro-iliac Strains.—These are *approximately one-fifth as frequent as lumbosacral strains. The* **sacro-iliac joints** *allow but a few degrees of rotatory and sliding motion and* **have the strongest ligamentous support of any joints in the body.** The sacro-iliac joint is subject to marked shearing rotation forces. Tears of the ligaments and capsules of the sacro-iliac joints are always possible due to rotation sprains. Acute sprains of this joint may occur following some unusual exercise or slipping when lifting.

Acute strains will often show four important signs.[4] *These are: Localized tenderness over the strained joint.*

Spontaneous pain in the direction in which the strain was produced (*i. e.*, if forced flexion produced the original injury, then flexion performed at the time of the examination will also cause pain).

Symptoms of Acute Lumbosacral Strain

Occurs **predominantly in manual laborers.**

History of trauma is usually present (may be major such as twist or wrench of the back, as in lifting a heavy weight; or minor such as pulling on a tight fitting sock or shoe).

Symptoms may be entirely out of proportion to the trauma; *individual cases vary greatly in the extent of their symptoms and signs.*

Onset of pain may be mild, increasing in severity as the patient continues at his work until it may become very severe within several days; the pain may remain of moderate or mild degree.

Pain is usually sharp and severe, is made much worse by movement and may decrease markedly or completely with rest.

The patient **cannot stand erect because of pain,** *but bends the body forward, leaning toward the sound side.*

Marked spasm of the low back muscles *occurs, and motion of the low back is almost negligible.*

The normal lumbar curve is obliterated.

There is **acute tenderness over the fifth lumbar spinous process** *and in the lumbosacral region on the affected side.*

Pain is also caused by straight leg raising, full flexion of the hip and internal or external rotation of the flexed hips.

Tenderness may be present in the unaffected lumbosacral region, or in both erector spinæ muscles or buttock.

The **position of greatest comfort** *is usually in recumbency, lying on the sound side with a moderate degree of flexion of the body, hips and knees and with a pillow between the knees; rest in bed for one day may result in marked improvement.*

At the onset, the pain is usually one sided and most severe just above or below and medial to the posterior superior spine of the ilium.

If the strain is more severe, pain is also noted on the opposite side and in the lumbar muscles; *pain may appear* within several days *in the leg of the affected side.* The pains in the leg may be burning in character and areas of anesthesia or hyperesthesia may be found. Radiating pains may occur in the distribution of the sciatic nerve, lumbar plexus or gluteal nerves in both sacro-iliac and lumbosacral sprains.

Relief by rest—usually by recumbency.

Relief also by immobilization (as adhesive or plaster).

Symptoms of Sacro-iliac Strain.—Spasm of the hamstring muscles produces *limitation of forward bending from the erect posture* and of straight leg raising on the involved side in the recumbent posture.

Forward bending from the sitting position is not limited as sitting relieves spasm of the hamstring muscles.

Flexion of the thigh upon the abdomen (with the knee flexed) causes slight or no pain.

Straight leg raising (Lasègue's sign) (*i. e.*, flexion of the thigh with the knee straight) produces pain in the sacro-iliac region or along the posterior aspect of the thigh; tension of the hamstring muscles on the tuberosity

of the ischium with the knee straight produces more motion at the sacro-iliac joint.

Point tenderness is greatest just lateral to and below the posterior inferior spine. (It must be remembered that tenderness over the sacro-iliac joints may occasionally be due to the presence of subcutaneous fatty nodules.[158])

Differential Diagnosis Between Sacro-iliac and Lumbosacral Strains

There are *no pathognomonic diagnostic tests to establish lumbosacral or sacro-iliac lesions;* the following points should suggest, but not absolutely determine this differentiation.

Test	Sacro-iliac strain	Lumbosacral strain
Strong compression of anterior superior spines of ilia	Causes recurrence of pain	No pain
Downward and outward pressure of the anterosuperior spines with the patient supine	Causes recurrence of pain	No pain
Point of maximum, persistent, localized tenderness	Just below and lateral to posterior inferior spine	In lumbosacral region, or over spinous process of 5th lumbar vertebra or just lateral to it.
Flexion of the thigh with the knee bent	Only slight or no pain	Very painful.
Rectal examination	Acute tenderness may be found; lower portion of joint can be palpated	Essentially negative.
Rotation of the lower spine	Restricted, especially toward the affected side	Not limited.
Bilateral pain along sciatic nerve	Uncommon	Much more frequent.
Hyperextension of one hip with opposite knee and hip acutely flexed	Pain	No pain.
Position in bed	Cannot lie for long periods on affected side	Can sometimes be made comfortable on either side or on back.
Flexion of spine while in sitting position	Forward flexion is normal	Forward flexion is as limited as when standing.
Lateral deviation of the lumbar spine	Usually present	Seldom found.

Pain may be produced by strong compression of the anterior superior spines of the ilia or on pressure of these downward and outward. The first of these maneuvers strains the posterior-inferior portion of the joints, and the latter the anterosuperior portion of the sacro-iliac joints.

The lower portion of the sacro-iliac joint is very tender on rectal examination.

Conservative Treatment of Low Back Strain (usually *caused by ligamentous or muscular tears*):

1. Secure immobilization and ligamentous relaxation of the low back by keeping the patient constantly on a *firm mattress* with *fracture boards* under this. Further relaxation is obtained by placing a *pillow under the knees* if the patient is lying on his back, or under the abdomen, if the patient is prone. There is no good mechanical basis for leg traction in these patients.

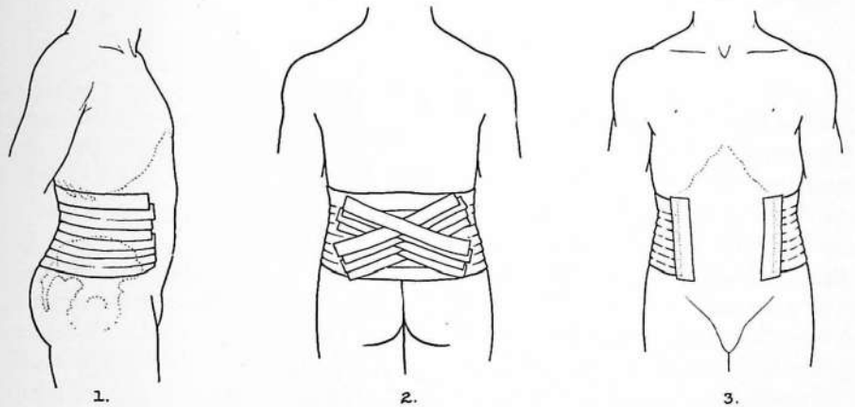


FIG. 289.—**Strapping of the low back.** *1*, shows overlapping horizontal adhesive strips; *2*, shows diagonal layer of overlapping strips; *3*, shows final layer of horizontal adhesive strips and vertical strips anteriorly to prevent the ends from slipping.

2. *In severe ligamentous strains, apply a plaster spica or a plaster back shell to maintain this position.*

3. The patient **should not be ambulatory** except in the mildest cases; unfortunately, the severity of the injury is often underestimated.

4. *Procaine injection* at the site of tenderness may relieve pain temporarily.

5. **Heat** (such as infra-red or hot fomentations) should be given for twenty to thirty minutes three times a day to the low back to further relax muscle spasm, relieve pain and hasten the absorption of any exudation.

6. With subsidence of pain and muscle spasm—when the patient can move in bed without marked discomfort (averaging about ten days after the onset), *postural exercises* are begun.

7. When the patient assumes an upright position, some **support** should be given **to the low back,** extending at least several vertebræ beyond the attachments of the sprained ligaments. In mild cases, a low back corset

is sufficient support; in severe strains, a higher corset support may be required with paraspinal steel uprights and circular steel bands to adequately protect the low back.

8. **Analgesics,** such as aspirin and phenacetin, 0.3 gm. of each may be required every three or four hours during the first day or two in bed; in some instances, it is necessary to add codeine phosphate, 15 mg. every four hours for several days to obtain freedom from pain.

9. As pain and limitation of motion disappear, the patient may gradually, after several months, discard his support for longer and longer periods.

10. *If symptoms continue,* consider the possibility of arthritis, a congenital anomaly of the low back leading to a potentially weakened back, improper positions at work or working too many hours with the back bent, etc.

11. Kuhns believed that it probably *requires at least two months for a ligamentous tear in the low back to heal completely.*

COCCYGODYNIA

This condition is usually accompanied by a *history of direct trauma to the coccyx,* such as a fall or a kick producing a sprain of the sacrococcygeal ligaments. *Males are seldom afflicted* as their narrow sacro-iliac notches permit the coccyx to be tucked in between the ischial tuberosities and to be protected from falls in a sitting position.[85,89,157]

Pain is located *at the sacrococcygeal junction* and is always in the **midline**; it *may radiate* to either buttock.[103,172] There is usually only an aching, but shooting pains may occur in the buttocks or down the legs. *Pains are aggravated by sitting on a hard surface or on rising from a sitting posture,* and lessened by sitting on an air cushion or contracting the glutei while sitting erect. Occasionally, excruciating pain may almost prevent sitting. *Severe pain on defecation* may be present. *External and rectal palpation reveal tenderness on pressure over the coccyx,* and there may be increased mobility and angulation of the coccyx on the sacrum. Roentgen examination may reveal a deformity in some cases.

The pathology of coccygodynia is not known. Key examined sections of 6 specimens microscopically and found normal bone and connective tissue with no inflammatory or arthritic changes.

Treatment of the acute cases includes: (1) Reduction of a fracture (if seen by roentgen-ray) by grasping the coccyx between the thumb on the posterior surface and the finger in the rectum; local or general anesthesia may be used.[85] (2) Hot sitz baths and analgesics to relieve pain. (3) Use of a rubber ring, when sitting. (4) Massage of the pelvic muscles by the finger in the rectum. (5) Some patients are relieved by strapping of the low back. (6) In severe cases, rest in bed is required. (7) Procaine injection of the tender areas in the soft tissues over the coccyx may relieve pain. (8) Some neurologists treat this disorder as a functional neurosis, using psychotherapy.

Thiele reported *spasm of the levator ani and/or coccygeus muscles* in 64

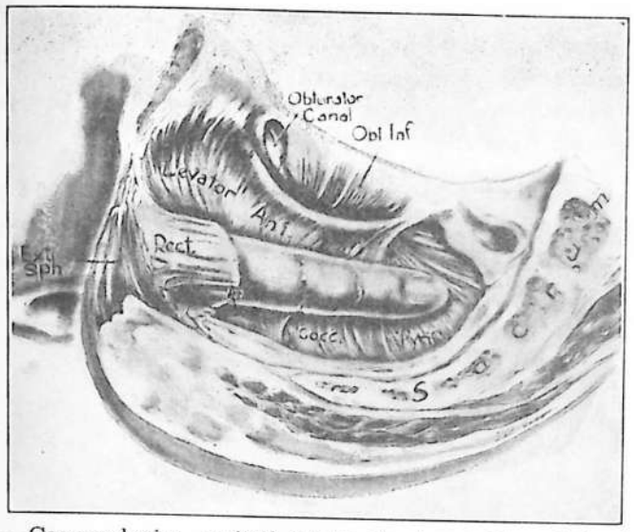


FIG. 290.—Coccygodynia: sagittal section showing position of the finger during massage of the levator ani, coccygeus and piriformis muscles. The finger sweeps from side to side, massaging lengthwise of the muscle fibers. (Thiele, courtesy of Jour. Am. Med. Assn.)

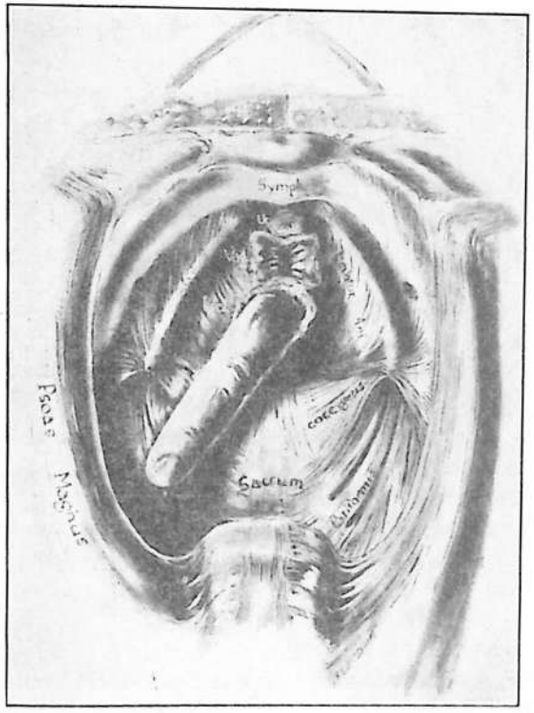


FIG. 291.—Anteroposterior view showing position of finger during massage of levator ani, coccygeus and piriformis muscles. Note that only the finger tip reaches the piriformis muscle. (Thiele, courtesy of Jour. Am. Med. Assn.)

of 69 patients with coccygodynia.[161] *Spasm of the piriformis muscle* occurred in 31 of 33 patients with pain in the supragluteal area or down the posterior aspect of the thigh. He believed that coccygodynia was often due to spasm of the levator ani and coccygeus muscles and that pain down the back of the thigh resulted from spasm of the piriformis muscle. *Massage of the pelvic muscles by the finger in the rectum* was followed by improvement in over 90 per cent of these cases. Each massage did not exceed one or two minutes and an average of 11 massages were given in eleven weeks.

In chronic cases, persisting more than several months, one may use, in addition, *roentgen therapy, epidural procaine injection, injection of alcohol into the painful area* or, if necessary, *removal of the coccyx*. This operation may prove difficult. Steinbrocker treated coccygodynia effectively by injection of from 5 to 10 cc. of 1 per cent procaine into the tender areas in the soft tissues over the coccyx.[150]

THE FACET SYNDROME

In a group of patients with low back pain, symptoms have been ascribed to pathologic conditions in the inferior articulations of the lumbosacral joint.[49,60] These are due most often to a *minute subluxation of one or both of the inferior articular processes of the fifth lumbar vertebra*. The facet syndrome may be due to causes other than subluxation (such as fracture of the facets, hypertrophic changes, etc.). A characteristic of *fracture* is sudden lightning pain on certain types of motion, relieved by changing this position.

The *symptoms and signs* of this condition (which are also present in acute low back strain) include: 1. Sudden onset of low back pain following exertion in a stooping position.

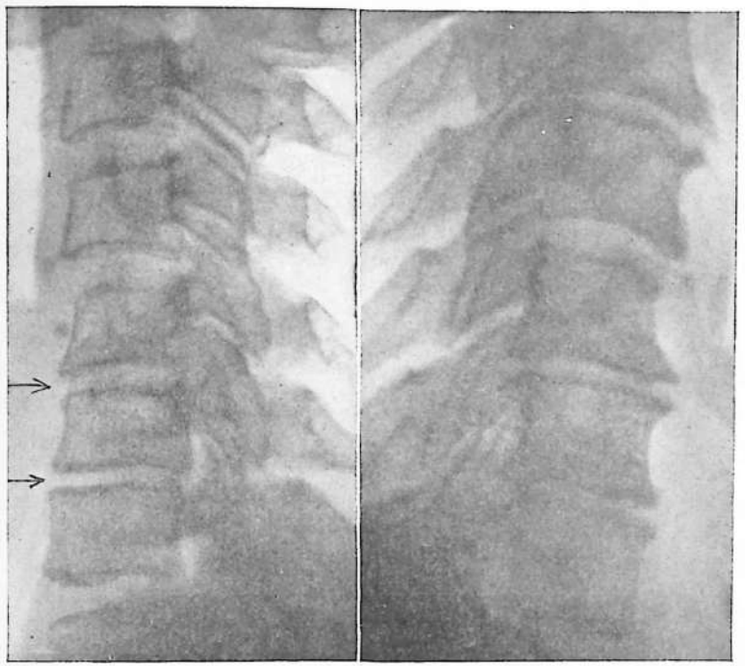
2. Inability to bend over because of pain.
3. Tenderness over the lumbosacral region.
4. Limitation of flexion of the spine.

A sudden sharp pain may be noticed in the low back on lifting a weight, or on bending over, or on performing any motions in a stooping position. The patient may complain of a snap or sudden sting in the low back or a sensation as though something had given way. The attacks may be of varying severity, forcing the patient to bed or permitting a continuation of work, but in a reduced amount. The lumbosacral joint is tender and forward bending is very painful and limited. This can be demonstrated well by having the patient lie flat on the back and raise the trunk (holding on to the examiner's hand) while the patient's lower extremities are maintained extended.[163] There is no pain on iliac compression and roentgen-ray examinations are usually negative.

These respond well to manipulative measures. **It is this type of patient who may be "miraculously cured" by certain mechanical maneuvers** *of osteopaths or chiropractors, which are frowned upon and looked askance at by the medical profession.* Physicians must realize that manipulative procedures (*properly performed*) may produce good results in certain conditions.

The **manipulative procedures recommended for these patients** are as follows:

The patient lies flat on his back on a firm table and braces himself by holding the sides of the table. The operator, standing at the foot of the table, flexes the patient's hip and knee joints while grasping one ankle. The patient is then asked to give a violent kick at which time the physician pulls strongly on the ankle in the line of the body and at a slight angle with the horizontal. Best results are obtained when the kick of the patient and pull of the physician are made simultaneously so that maximum force is obtained when the leg is in full extension. Following several such kicks,



A *B*

FIG. 292.—Discogenetic disease, cervical spine. (*A*) Marked thinning of two disks, no bony reaction. (*B*) Thinning of four disks, generalized exostotic deformities. Note absence of any pathologic changes in the apophyseal joints. (Oppenheimer, courtesy of Radiology.)

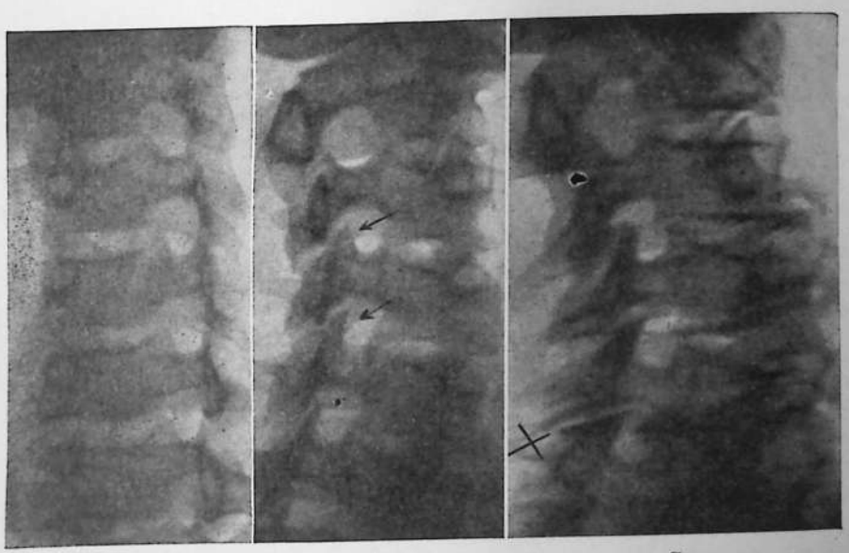
the patient is able to flex his trunk on the lower extremities much more than prior to the manipulation. *Pain decreases following such a manipulation*. The average treatment requires from 5 to 10 kicks, being continued until flexion at the hip joint is free and until only slight soreness remains in the lumbosacral region. The kick and pull produces a forward thrust of the sacrum on the fifth lumbar vertebra in the opposite direction to which the slipping has occurred.

Following such a manipulation, a regimen of *rest, support* for the low back, *heat and massage* are continued for at least two weeks to prevent

recurrence. The patient is advised against the performance of any motion which may have caused his acute condition. Faulty posture is corrected. If indicated, a low back corset support is worn for some months.

THE DISCOGENETIC SYNDROME

The discogenetic syndrome is the name applied to *narrowing of one or more intervertebral disks with shifting of the strain of weight bearing posteriorly to the small articular facets.*[7] *Subluxation of the facets may occur.* If the process occurs at the lumbosacral disk, the lumbosacral angle increases in acuteness as the fifth lumbar vertebra settles on the sacrum. This may lead to mechanical irritation or compression of the fifth lumbar nerve roots as their foramina become smaller; this causes pain in the low back or in a sciatic distribution. If a large amount of the disk substance is destroyed, the resulting increased irritation between the fifth lumbar and first sacral vertebra may lead to degenerative joint changes.



A B C

FIG. 293.—Collapse of foramina in discogenetic disease. (*A*) Normal. (*B*) Two articular processes displaced. (*C*) Pronounced displacement; foramina 8 shaped. Note exostoses of the bodies in *C*, and tiny spurs at the tips of the processes involved in *B* and *C*, in the absence of changes of the joint space. (Oppenheimer, courtesy of Radiology.)

The lumbosacral intervertebral disk is destroyed more often than other disks; that between the fourth and fifth lumbar segments is next in frequency.[168]

Narrowing of the intervertebral disks between the fourth and fifth lumbar, or between the fifth lumbar vertebra and the sacrum, may be the result of acute injury, gradual traumatic wear and tear, or chronic lordosis (thus shifting the weight-bearing stress to the back of the disk). Lateral roentgen-ray examination may reveal the narrowed intervertebral disk space and increase in the lumbosacral angle. Other roentgen findings include decrease in diameter of the foramina of exit of the fourth or fifth lumbar nerve roots and subluxation of the articular facets.

Conservative treatment includes rest, heat and massage to the low back, exercises to strengthen the gluteus maximus and abdominal muscles and to stretch the iliotibial band and the tight hamstrings, a back brace, and manipulation under general anesthesia, if required. If improvement does not occur, an arthrodesis of the fourth and fifth lumbar vertebræ to the sacrum must be done. If sciatic pain is present, one must always consider herniation of an intervertebral disk.

CONTRACTION OF THE FASCIA LATA

It has been suggested that the *iliotibial band* may be a factor in low back pain. This band extends from the crest of the ilium downward over the lateral aspect of the leg to its insertion into the outer tuberosity of the tibia. In its lower portion, it forms the tendon of the tensor fasciæ latæ. There is, in addition, a widespread aponeurotic connection between the tendon of the gluteus maximus and the posterior part of the iliotibial band.

Contracture of the fascia lata on one side produces a lateral deviation of the spine on that side and a tilt of the pelvis; such a unilateral contracture may account for irritation in the sacro-iliac joint because of the marked leverage produced by the contracture.

Contracture of the fascia lata may result from: 1. Prolonged lying in bed during a severe illness.

2. Faulty posture, especially while in bed.

3. Wedging effect of voluminous diapers in children (keeping the child's legs abducted and externally rotated) over long periods. This is open to some question.

Symptoms: 1. Sensation of a dull ache or strain in the low back either in the sacro-iliac or lumbosacral regions.

2. Severe sciatica may accompany this.

Physical Signs:[121] 1. Limitation of straight leg raising.

2. Inability to touch the floor when stooping (with the knees straight).

3. Limitation of flexion of the lumbar spine; bending the body forward while in the sitting position with the legs extended rarely produces flexion of the lumbar spine and pelvis even to a right angle.

4. Positive Ober's sign (described previously).

Ober does not believe that all lame backs result from contraction of the fascia lata; other conditions such as ligamentous or muscular strain, malignancy, spinal tumor, poor posture, arthritis, etc., are often the cause of such low back pain. *Contraction of the fascia lata* results in poor posture which will not respond to exercises or special apparatus, but *is benefited markedly by a single operative procedure: dividing the fascia lata above the trochanter.*

Seventy-five per cent of 415 patients have been completely relieved by this procedure (with an average period of three months before complete relief was noted). In some patients, symptoms were relieved immediately following operation. In most of the patients in whom relief did not follow operation, there was found a neoplasm, severe arthritis or congenital anomaly of the spine. Recurrence has been observed in patients in whom a tight fascia lata on the opposite side was at fault, or where an incomplete operation had been performed.

Good results of operative intervention in this manner have been explained in two ways: The mechanical strain of the lumbosacral or sacro-iliac joint due to the pull of the contracted iliotibial band has been relieved.

A tight iliotibial band may conceivably produce a squeezing effect on the sciatic nerve where it passes over the acetabulum.

At present, contraction of the fascia lata and its operative relief by dividing the fascia have "gone out of style" with the current wave of enthusiasm concerning herniated intervertebral disks as a cause of recurring attacks of severe low back pain and sciatica.

INJURY TO FIBERS OF THE ERECTOR SPINÆ MUSCLE

Symptoms.—Moderate, diffuse, dull muscular pain across the whole lower lumbar region, increased by motion; stiffness rather than a shooting pain; no reference of pain; relief by hyperextension; sharply localized tenderness in the muscle at the point of rupture; in some cases, a tilt of the spine to the affected side; motions in all directions possible but guarded.

TRAUMATIC BACKACHE

This may result from:[81] trauma added to a preëxisting arthritis of the spine and pelvis; trauma to the soft parts of the back (muscles, tendons, ligaments and periosteal tissue); predisposition to injury from faulty posture and occupational strain such as heavy lifting. Combinations of these are frequent. *Traumatic back injuries are most common in the lumbar region,* next in the cervical, then in the dorsal (which is the least flexible and least liable to injury).

Means has discussed low back pain in industrial workers. Backache is often a troublesome entity in these individuals, while the *compensation factor may lead to confusion.*[107] Too frequently, these patients have been diagnosed as a sacro-iliac sprain following a hasty inspection, and an adhesive support has been the only therapy.

Low back pain in industrial workers may be due to:

1. Lumbosacral sprain.
2. Muscular strain.
3. Sacro-iliac subluxation.
4. Subluxation of the articular facets.
5. Contusions.
6. Fractures of vertebral bodies and transverse processes.
7. Protrusion of the intervertebral disk.

8. Non-traumatic causes as fibrositis associated with acute infections such as pharyngitis; prostatitis and diseases of the spine as arthritis, malignancy, tuberculosis.
9. Orthopedic causes: spondylolisthesis, spina bifida, scoliosis, malalignment of articular facets and poor posture.

Sprains indicate tearing the fibers of a ligament or separating them from their attachments. Lumbosacral sprain is usually associated with severe pain, muscle rigidity and deep-seated localized tenderness; there is often swelling over the affected area and aggravation of the symptoms by motion.

Strains result from stretching of muscle fibers so that fibers of the muscles rupture and capillary hemorrhages may occur. *In most of these milder muscular strains, healing occurs quickly following the application of heat and adequate rest,* resulting in a short period of disability. Localized tenderness and muscular spasm are present and pain may be referred to the back of the hips or to the inguinal region.

Sacro-iliac subluxation, or painful slipping of the sacro-iliac joint may follow sudden lifting with one leg straight and the other slipping, or sudden rising from a stooped position with quick twisting of the trunk. The patient develops a sudden painful catch in the sacro-iliac region and may not be able to straighten up. The lumbar spine is flattened and the muscles appear rigid; lateral curvature of the spine usually appears away from the affected side. The patient can bend forward better when sitting than when standing because of relaxation of the hamstrings while sitting.

The articular facets may have a tendency to slide particularly when there is an upward pull on the sacrum as in lifting. Symptoms are in general similar to sacro-iliac sprains except the difference in the position of the tenderness.

Contusions such as a sudden blow on the back may result in superficial or deep bruises with hemorrhages into the muscles. *Fractures of the transverse processes* occur frequently, often in association with contusions or muscular strains. There is usually localized tenderness, together with roentgen evidence of the lesion. Fractures of the body of the vertebræ must be treated with the patient in a hyperextended position to prevent subsequent strain and root pains from faulty alignment.

The symptoms of protruded intervertebral disk may not appear for months or years after an accident; in some cases, one cannot elicit a history of trauma. In most instances, these individuals are young or middle aged adults.

Patients with fibrositis or myositis of the low back associated with an acute illness such as a pharyngitis not infrequently blame a lift or injury several days or weeks before for their low back pain.

Acute low backache may be associated with prostatitis, which may be relieved by prostatic massage.

Numerous factors are at work in industrial workers which produce chronic strain, manifesting itself most often in the lumbar area; these include advancing age, overweight, pronated feet, hard work, long hours of standing, etc.

A number of morning backaches may be due to modern designs in beds with box springs and soft mattresses.

Much doubt and controversy clouds the relationship between trauma and disease. Unfortunately, most individuals believe that pain in the back must be due to injury. In some instances, an injury can flare up a latent tuberculosis of the spine or cause more rapid growth in a malignancy of the spine. Trauma may also instigate an osteomyelitis.

The effect of trauma upon arthritis of the spine is at times difficult to determine. Trauma may aggravate mild arthritic symptoms. However, the physician must remember that *degenerative changes with spur formation and lipping are present in practically all spines after the age of fifty.*

The physician must take an *accurate history* in all industrial cases, including the position of the patient at the time of injury, how soon the pain developed, its location, character and duration, as well as any other factors which may be of importance. The examination of patients with low back pain must be done extremely thoroughly.[162]

Prophylactic measures *may be employed in industry in an effort to avoid industrial disability. An effort should be made to see that the individual is suited for the work in which he is engaged.* Although a worker may have ability for a position, he may not be fitted physically for such occupation. Plant physicians should attempt to prevent disability by careful check on the individual workers in an effort to see they are not employed in positions to which they are not physically capable of handling.

Chronic Industrial Backaches.—Industrial backaches cost the employer large sums of money. *They are common excuses for malingering and may lead to loss of morale.* They start from single or repeated trauma, inefficiently treated. This includes the use of the back in a strained position, such as in the ditch digger, jeweler, miner, etc., in which there is strain upon the muscles and ligaments of the lower spine. Many have real pain while some simulate it.

Involuntary protective muscle spasm is always present in acute back pathology, and is most marked in the immediate region of the lesion; this may involve the whole back. It is constant and relaxes only when the patient is recumbent. Tenderness over the site of an organic lesion is constant and does not vary from place to place.

LESIONS OF THE INTERVERTEBRAL DISKS AND LIGAMENTA FLAVA

Lesions of the intervertebral disks and ligamenta flava may cause radiation of severe sciatic pain. Many cases diagnosed as lumbosacral or sacro-iliac strain are examples of herniation of portion of an intervertebral disk. Such herniation occurs most often in the lower lumbar region. Goldthwait suggested that lumbago and sciatica may be caused by injuries to the intervertebral disks.[55] Herniations or ruptures of normal intervertebral disk tissue into the spinal canal have been removed surgically because of pressure on the spinal cord or nerve roots.[2,30,41] These were considered to be chondromas or loose cartilage until their true nature was discovered.[115]

Although lesions of the disks are common, clinical manifestations do not occur in all cases. In many instances, these manifestations, if present, are almost indistinguishable from those due to diseases of muscles, joints, viscera and other parts.[129]

The common disk lesions are those due to rupture and those due to degeneration. From strain or trauma, a piece of cartilage may be detached from a normal disk or the nucleus pulposus may be squeezed out of the center of the disk. This displaced material may be pressed beyond the margin of the disk through the fibers of the longitudinal ligaments causing encroachment on the cord or vertically into an adjacent vertebral body through traumatic fissures of the cartilaginous and osseous surfaces or through congenital defects.

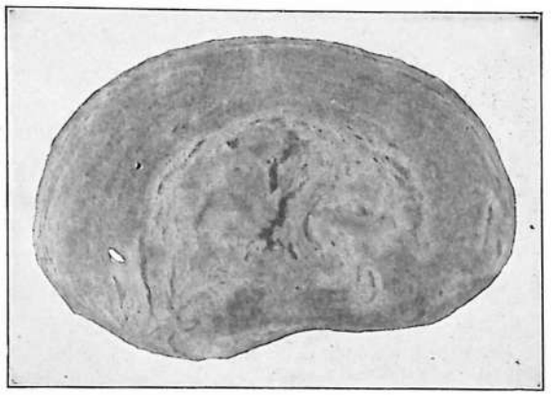


FIG. 294.—Transverse section of an intervertebral disk showing ruptured nucleus pulposus. (Batts, courtesy of Jour. Bone and Joint Surg.)

The disks grow flat and less elastic with advancing years from dehydration and increase in fibers of the cartilage of the disk. Although the disk is resistant to most infections, once infected, it deteriorates rapidly.

Oppenheimer concluded that *disk lesions are about twice as common as duodenal ulcers.* The cartilages of the disks of adults are usually the weakest parts of the spine. Following wear and tear, numerous minimal injuries are produced from which the disk rarely recovers completely. Regardless of the cause of injury and degeneration, *flattening of the disks leads to narrowing of the corresponding intervertebral spaces with displacement of the articular processes,* narrowing of the neural foramina and abnormal contact between vertebral bodies. Narrowing of the neural foramina may cause radicular neuritis if the nerve root is compressed.

The disk has no nerves and is not painful when diseased. The most sensitive parts in its neighborhood are the nerve roots and the apophyseal joints. Arthritis develops in these joints in about 20 per cent of cases of disk lesions, producing pain in the back and limitation of vertebral motion. The apophyseal joints usually remain intact and symptoms of nerve root compression develop without pain and rigidity of the spinal region involved.

The Intervertebral Disks.—These disks originate from the fetal notochord as a highly specialized structure. Their embryology, anatomy, physiology and pathology have been excellently described by Professor Schmorl of Dresden, Germany.[138–140] In the adult, the disks consist of *fibrocartilaginous plates* or pads (between the bodies of adjacent vertebræ); in the center of each disk there is a semi-gelatinous material known as the *nucleus pulposus*.[12] This latter is almost 90 per cent water at birth. *The construction of the nucleus pulposus produces the resilient quality of the spinal column.*[170] This elastic semifluid mass receives (from the bony surfaces) the force of the blows and acts like a spring distributing the shock of various jars and lessening their effect upon the nervous system. It also acts as a medium for transmitting pressure from one vertebra to the next. There are 23 intervertebral disks in the human spine, one being placed at each intervertebral level above the sacrum.

Fig. 295.—Schmorl's nodules, or rupture of the nucleus pulposus (seen extending into adjacent vertebral bodies). (Batts, courtesy of Jour. Bone and Joint Surg.)

The cartilaginous plates of the intervertebral disks are thin layers of hyaline cartilage forming the top and bottom of the disk, and fusing anteriorly and laterally with the bony edges of the vertebræ. These plates of cartilage enclose the "*annulus fibrosus*" which is a dense fibrocartilaginous envelope consisting of concentric folds of fibrous tissue. *The nucleus pulposus is enclosed within this strong elastic container.* The fibers of the annulus are fewer and thinner toward the posterior portion of the disk (toward the spinal canal).[72] The annulus fibrosus is reinforced anteriorly by the strong anterior longitudinal ligament and to a small extent posteriorly by the posterior longitudinal ligament. The latter is rather small in the dorsal and lumbar areas. There are no reinforcing ligaments or muscles on the posterolateral portions of the disks, where these border on the spinal canal. The normal function of the nucleus pulposus thus depends upon the integrity of the annulus fibrosus.

Intervertebral disks are thinnest from the third to the seventh thoracic vertebra and thickest in the lumbar spine. Intervertebral disks have no blood supply in adult life, but obtain their nutrition by diffusion of lymph through tiny perforations in the bony surface of the spongy bodies of the

vertebræ. *Disease of the disks alone is painless as these possess no nerve supply; pain occurs when secondary changes result in the bodies of the vertebræ and in their facets* or when the protruded disk substance presses upon an adjacent spinal nerve.

Changes in the intervertebral disks occur with increasing age; after the age of fifty-five, there is usually some abnormality in these structures in all individuals.

Diseases Affecting the Intervertebral Disks: Developmental changes.
Pathology of the nucleus pulposus.
Pathology of the cartilage.
Pathology in the body of the vertebra.

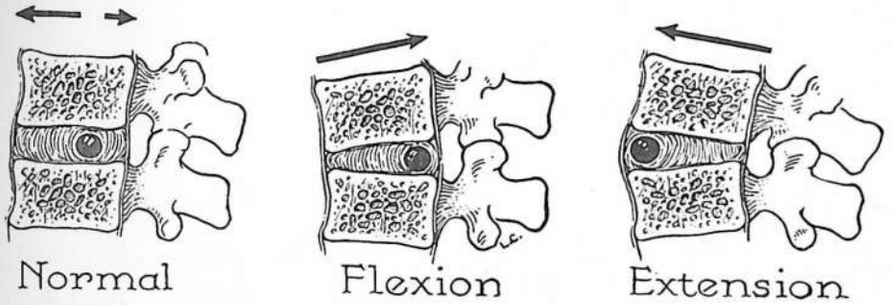


Fig. 296.—Intervertebral disk with nucleus pulposus. Normal excursion of nucleus during flexion and extension of spine. (Lewin, Backache and Sciatic Neuritis.)

Developmental Changes of the Disks: These consist chiefly of congenital absence of a disk and nuclear expansions into the body of the adjacent vertebræ.

Disturbances of the Nucleus Pulposus: Retropulsion or antepulsion of the nucleus within the disk.
Prolapse of the nucleus into the adjacent vertebral body.
Retropulsion of the nucleus into the spinal canal.
Lateral shift of a nucleus.
Calcification of the nucleus.
Dehydration of the nucleus.
Infiltration of the nucleus by other tissue, especially fibrous tissue.
Expansion of the nucleus.

Some degree of prolapse of nuclei has been found in more than one-third of 5000 routine autopsies.[138,139] *With extrusion of only a small portion of the nucleus, the function of the disk may not be markedly impaired immediately,* but dehydration and decreased function of the disk will probably appear at an earlier age than otherwise. Injuries, resulting in tears of the annulus fibrosus, or in the cartilaginous plates, or both, usually precede prolapse of a nucleus.[136] Following frequent trauma from every day use or after severe injury, the annulus fibrosus occasionally becomes weakened and permits bulging of the disk or herniation of the nucleus. *This occurs most commonly on either side of the posterior longitudinal ligament where the*

wall of the disk is weakest. Such bulging or herniation of the disk may cause pressure upon a single nerve root or upon the entire cord. Retropulsion of a nucleus into the spinal canal may produce pain and neurologic findings (even with the production of paraplegia[21]). Prolapse of the nucleus pulposus may also occur from a congenital defect in the cartilaginous plate; with such a defect there may result a gradual leakage of the nuclear fluid with thinning of the intervertebral disk. Because of the close adherence of the cartilaginous plates to the adjacent surfaces of the vertebræ (above and below), the nucleus pulposus may prolapse into the spongy bone of the vertebræ. This may be an etiologic factor in vertebral epiphysitis due to irritation and disturbed function.[24] Such a prolapse of the nucleus pulposus into the vertebræ may be revealed later as a punched-out area in the vertebral body or as a calcified nodule.

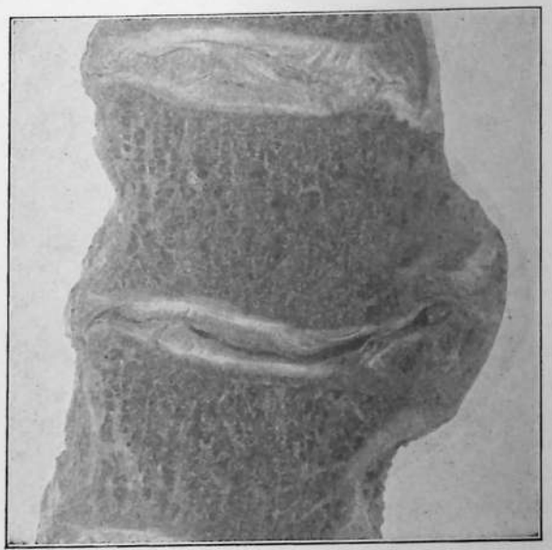


FIG. 297.—Mid-sagittal section through two adjacent vertebral bodies and intervertebral disks. The upper disk is normal. The lower disk is thinned out, and the nucleus has been extruded anteriorly where it is covered by osteophytic proliferation and bony bridging between the anterior portions of the vertebral bodies. (Batts, courtesy of Jour. Bone and Joint Surg.)

In patients with scoliosis, a lateral shift of the nucleus pulposus has been observed; this may be responsible in part for the difficulty sometimes encountered in attempting to correct a scoliosis.

Although protrusion of an intervertebral disk may occur at any level in the spinal canal, more than 95 per cent of these occur in the two lower disks (*i. e.*, between the fourth and fifth lumbar vertebræ and between the fifth lumbar vertebra and the sacrum).[116]

Calcification of the nucleus pulposus may be demonstrated by roentgen-ray; its causes are unknown but such deposits increase with age. Calcified deposits are found much more commonly in the annulus than in the nucleus.

These may appear in the roentgenograms as branched shadows with several processes.

Dehydration of the nucleus increases with age.[104] It has been estimated that at birth the water content of the nucleus is 88 per cent, while in late life this may decrease to less than 70 per cent. Such a dehydration may be hastened by stress and strain.

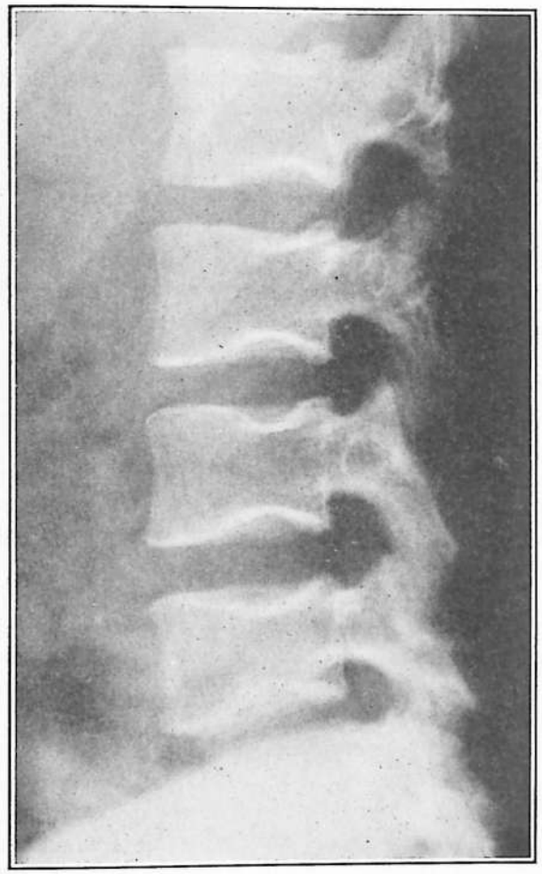


FIG. 298.—**Herniation of the nucleus pulposus** into the body of practically every vertebra from the first thoracic to the sacrum. The density of the bones appears normal. This is too diffuse for a traumatic type of herniation. The patient is a white male, aged twenty-one years. His chief complaints were pain and heaviness in both legs from the knees down (of six months duration). Neurologic examination was negative. There was no block in the subarachnoid space. Following air injection, there was demonstrable no encroachment upon the lumen of the canal in the lumbar region.

Infiltration of the nucleus by blood-vessels, bone or fibrous tissue may occur; the latter is especially frequent following trauma or in old age.

When the vertebral bodies have become softened from senile osteoporosis or other causes (as osteochondritis, neoplastic invasion, etc.) expansion of the nucleus pulposus may occur due to the release of the

surrounding pressure. In such instances, the disks assume a spherical appearance by roentgen-ray, while the bodies of the vertebræ appear biconcave.

Pathology of the Cartilage of the Intervertebral Disks: Such lesions include: congenital defects of the cartilage; injury following acute or chronic trauma; fissuring or cracking of the cartilage and other pathology of the cartilage plate (such as drying of the disk, increased moisture in the disk, etc.).

A defect in the cartilaginous plate may result in prolapse of the nucleus pulposus. Congenital absence of a disk has also been observed.

Injury of the disk as a result of chronic trauma is most frequent in patients from twenty to forty years of age. As the disk has no nerve supply, its involvement does not thus produce pain until the vertebræ have become involved. In experimental work in dogs, the annulus fibrosus was punctured and the nuclear material allowed to escape with resultant prolapse of the nucleus into the bodies of adjacent vertebræ.[86] Following this procedure, destruction of the disk followed with lipping of the vertebræ and degenerative joint disease. It has been suggested that a similar condition occurs in man, *i. e.*, following thinning and destruction of the disks, rubbing together of the bodies of the vertebra produces sclerosis, lipping and degenerative joint disease.[59] Acute injury of the cartilaginous plate due to trauma may be an etiologic factor in the production of degenerative joint disease at a later date.

The nucleus pulposus may be injured by an improperly performed lumbar puncture. Lewin has termed this a *non-stop lumbar puncture* in which the needle is pushed through the subarachnoid space and enters the intervertebral disk, where it may injure the nucleus pulposus.[95]

It has been suggested that **Kümmel's disease** may originate from injury to the cartilaginous plate on either side of the vertebræ resulting in protrusions of the nucleus pulposus into the vertebræ. Kümmel's disease is a progressive rarefying osteitis of the spine, or a crumbling collapse of the body of a vertebra, noted several months following a severe injury to the back (such as an unrecognized compression fracture). It produces marked disability and few early signs. Because of the latter, the patient may be labeled neurotic, malingering or hysterical. If successive roentgenograms are made, one will note a progressive decalcification of the bodies of the vertebræ going on later to wedging and collapse, if untreated.

Changes in the disks are so common in elderly individuals that it is almost impossible to find all disks completely normal in patients over the age of fifty-five. With the onset of degeneration within a disk, cavities may be produced in its interior although the periphery of the annulus fibrosus remains intact. With advancing age, dehydration or decreased resiliency of the disks appears.[137]

Herniation of a Lumbar Intervertebral Disk.—Herniation of one of the lower lumbar intervertebral disks is an important cause of severe intractable sciatic pain.[96,148] This may produce pressure upon a portion of the cauda equina with symptoms not unlike those of lumbosacral or sacro-iliac strain with sciatica, so that clinical differentiation may be impossible. Protrusion

Symptoms of Herniated Lumbar Intervertebral Disk

Unilateral or bilateral **pain** (slight or intractable) **in the low back,** posterior thigh and calf; **sciatic pain is the commonest and most troublesome complaint.**

Incapacitating low back pain usually precedes sciatic pain by weeks, or even years.

The pain is usually made worse on sneezing, coughing or straining.

There may be pain in the buttock, sacro-iliac or lumbosacral region.

Paresthesias (numbness, tingling, etc.) may occur in the lateral aspect of the leg below the knee or in the foot.

With severe damage to the cauda, one may note bilateral pain, incontinence of urine and feces, and weakness of muscles or actual paralysis.

History of trauma to the low back in at least half of the cases (such as lifting a heavy weight with a feeling that something gave way in the lower back).

History of **remissions and exacerbations** of symptoms in most cases.

Recurring bouts of low back pain with unilateral or bilateral sciatica in the back of the leg, usually aggravated by coughing or sneezing are most often due to herniation of an intervertebral disk.[80]

Physical and Laboratory Findings

Unilateral (and sometimes bilateral) *limitation of straight leg raising* in almost all cases.

Spasm of the lumbar muscles with limitation of motion in this region (stiff lumbar spine).

Diminution or obliteration of the normal lumbar lordosis in about half of the patients.

Frequent occurrence of local tenderness over the lumbar spinous processes and the interspinous ligament at the side of the lesion.

Sciatic scoliosis (fixed list of the lumbar spine in either direction) in about one-third of the cases.

In some cases, tenderness over the buttock, sciatic nerve and sacro-iliac ligaments; *sciatic tenderness in 64 per cent of patients.*

Neurologic changes in half of the patients (*diminution or absence of ankle jerk on the affected side in 60 to 70 per cent of patients,* hypesthesia of the foot and lateral aspect of the leg, partial or complete paraplegia, etc.)[146]

Spinal fluid protein usually increased.

Roentgen Findings

Narrowing of an intervertebral space is objective evidence of a defective disk. This is seen in more than 50 per cent of cases.

Abnormalities include narrowed intervertebral joint spaces, formation of new bone about the margins of the joint, sciatic scoliosis, partial obliteration of the normal anterior lumbar lordosis and the occasional finding of a "Schmorl's nodule."

The lesion is often demonstrated following introduction of pantopaque, lipiodol or thorotrast into the spinal canal.

In most instances, contrast myelography is unnecessary; this usually will not reveal small or "concealed herniations."

of the lumbar intervertebral disk usually occurs at the side of the canal producing compression of the fifth lumbar or first sacral nerve root. An accurate diagnosis can usually be made in such patients and the extruded fragment may be removed surgically.[9] Love has stated that herniated intervertebral disks are responsible for less than 2 per cent of low back and/or sciatic pain.[97] However, it is probably the most common cause of severe *recurrent* low back pain and sciatica.

Oppenheim and Krause published the first case report of an operation for removal of a protruded intervertebral disk into the spinal canal.[126] In 300 cases of protruded intervertebral disks studied by Walsh and Love, 96 per cent of the protrusions occurred at the third, fourth or fifth lumbar interspace.[166] The lesion was confined to a single disk in 88 per cent of the cases. In most cases, there was an associated thickening and fibrosis of the ligamentum flavum.

SYMPTOMS OF HERNIATED LUMBAR DISK

Dandy believes that recurring attacks of low lumbar backache plus sciatica in the back of the leg are almost pathognomonic of defective intervertebral disks. Similar symptoms may be produced by *spondylolisthesis, congenitally defective vertebræ and spinal tumors.* He believes that defective disks can be diagnosed in 95 per cent of cases from the signs and symptoms alone. Roentgen examination will separate from this group spondylolisthesis and congenitally defective vertebræ and in more than half of the cases will disclose narrowing of the intervertebral space if a defective disk is present. In his experience, *multiple disks were involved in about 80 per cent of the cases.* He has found that *complete removal of the affected disks reduces recurrence of symptoms to a minimum* and results in fusion between the affected vertebræ, obviating the necessity for fusion by spinal grafts. In his cases, *if backache or sciatica returned or persisted after operation,* either an affected disk had been missed or had been incompletely removed, or a tumor was present.

The commonest type of pain in the lumbar lesions in the series reported by Walsh and Love was a **low back pain with sciatic extension.** The *frequency of symptoms* was as follows: unilateral sciatic pain (70 per cent); bilateral sciatic pain (16 per cent); accentuation of pain on coughing or sneezing (48 per cent); pain at night (27 per cent); paresthesias (50 per cent); sphincteric incontinence (5 per cent); positive Lasègue sign (84 per cent); sciatic tenderness (64 per cent); diminished or absent Achilles reflexes (60 per cent); decreased or absent hamstring reflex (21 per cent); muscular weakness (21 per cent); sensory loss (23 per cent).

Mixter and Barr obtained a history of trauma in 49 per cent of their patients (22 per cent had done heavy lifting). *Leg pain was present in every patient in their series.* In 13 per cent, this was confined to the thigh alone; in 66 per cent, the thigh and calf were involved, while in 21 per cent there was pain in the thigh, calf and foot. In 48 per cent, pain in the leg preceded the back pain. Other findings included a stiff lumbar spine in 70 per cent, loss of the lumbar curve in 47 per cent, list or scoliosis in 37 per cent, diminished or absent ankle jerk in 70 per cent and subjective or objective sensory changes in 33 per cent. **Every patient showed diminution of straight leg raising.**

The spinal cord and nerve roots may also be compressed by herniation of the nucleus pulposus in the cervical region.[155]

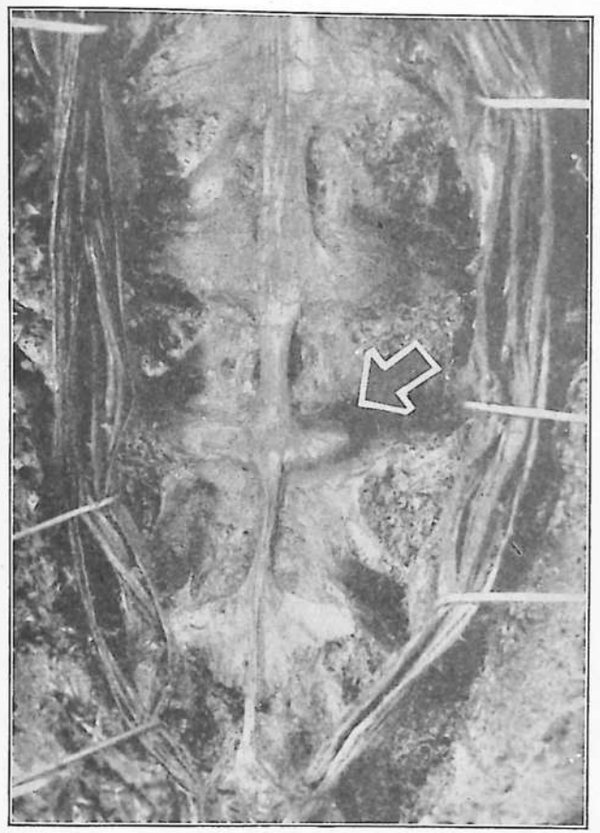
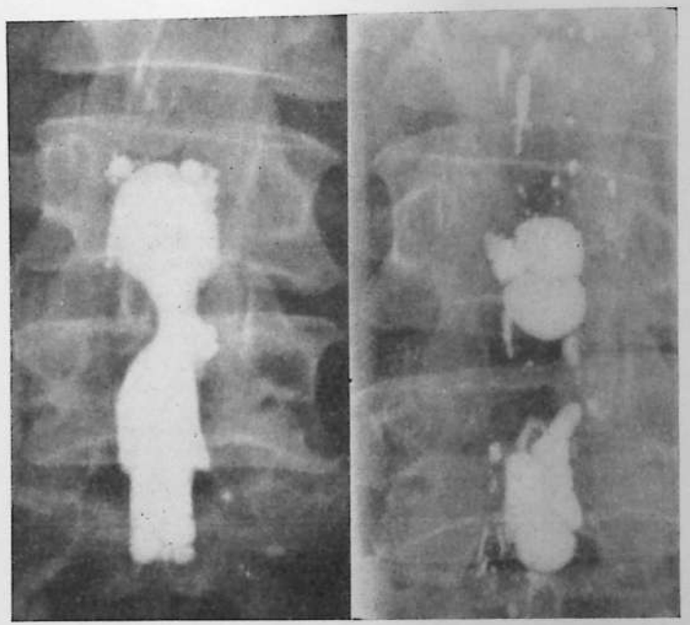


FIG. 299.—Posterior aspect of vertebral disks and posterior longitudinal ligament with the cauda equina retracted laterally. Arrow indicates the ruptured disk. Pedicles have been cut and the entire neural arch has been removed. (Batts, courtesy of Jour. Bone and Joint Surg.)

Diagnosis.—Severe, intractable, disabling pain in the low back and sciatic distribution in the back of the leg frequently having long remissions is one of the outstanding symptoms of this disorder.[171] This usually runs down the back of the thigh and along the outer side of the calf. Pains in the back may precede the leg pain. **There is an interference with straight leg raising in practically every patient.** A *history of injury* may be obtained in about 50 per cent. Motor weakness may occur in severe cases but sphincteric paralysis is rare. Although a thickened ligamentum flavum or arachnoiditis

may give the same history and physical findings as a protruded disk, this is unimportant as two or all three of these may be present in the same patient and require surgical intervention. Protrusion of a disk occurs at least ten times as often as the other two together.[116]

This condition can usually be diagnosed by the history and physical examination alone. Spurling and Grantham reported that *herniation at the third lumbar interspace* resulted in tenderness over the third lumbar spine, decreased lumbar lordosis, positive Lasègue test, decreased or *absent knee jerk*, normal Achilles reflex and hypesthesia and paresthesia in the fourth and fifth lumbar dermatomes.[101,147] In involvement of the



A *B*

FIG. 300.—*A*, Defect in column of radiopaque oil opposite the 4th lumbar intervertebral space due to protruded disk. *B*, Large defect opposite 4th lumbar intervertebral space. This defect was proved at operation to be a recurrent protrusion of the 4th lumbar disk. (Love, courtesy of Proc. Mayo Clin.)

fourth lumbar interspace, the patellar and Achilles reflexes were normal and the hypesthesia and paresthesias were in the fifth lumbar and first sacral dermatomes. With herniation at the fifth interspace, the Achilles reflex was diminished or absent and the hypesthesia involved the first and second sacral dermatomes.

If signs and symptoms of a protruded disk causing severe sciatica are questionable, *lumbar puncture* is indicated. It has been advised that this be performed as low as possible (at the lumbosacral articulation) and that the *first several cc. of fluid withdrawn be examined for protein content.* This is *usually elevated.* In such patients, the lesion (if large enough) can often

be localized by roentgenograms after the introduction of iodized oil, pantopaque, thorotrast or air into the spinal subarachnoid space.[65,66]

The *examination with iodized oil as suggested by Barr* and his colleagues *is as follows:* Following the withdrawal of 6 to 7 cc. of spinal fluid, from 2 to 5 cc. of iodized poppy seed oil (lipiodol) is injected slowly into the lumbar canal (usually between the fourth and fifth lumbar spinous processes). If the ampoule is warmed to 105° F. prior to injection, the injection will be facilitated as the oil will be more fluid.[22]

The patient sits up for a few minutes to collect the oil as one mass in the sacral cul-de-sac. He is placed on a tilting roentgenoscopic table in the upright position.

An attempt is made to keep the oil in a single mass. By tilting the table the oil flows slowly up or down the anterior aspect of the subarachnoid space (the patient lying face down on the table).

The lesion to be demonstrated is a small anterior extradural nodule at the disk levels.

Any constant filling defect is recorded by serial roentgenograms taken in various angles of rotation of the patient.

Inasmuch as 95 per cent of protruded disks occur in the lower lumbar region, *the upper dorsal region is not examined unless the patient has symptoms in that area.* Care is taken not to allow the iodized oil to enter the skull.

Lipiodol has some disadvantages: (1) it is slowly absorbed and may act as a permanent foreign body occasionally leading to considerable pain from irritation; (2) if the patient is tipped head down, lipiodol may enter the cranium and remain there, producing headache.[116]

Pantopaque is less viscid, tends to remain in one globule and is much more easily removed following roentgenograms. This may supplant lipiodol in those few cases which require contrast myelography.

Iodized oil in the spinal canal may produce further arachnoiditis if meningeal irritation is present. Lipiodol should be instilled only on the day of operation. It may aggravate the patient's symptoms. Such an oil is a foreign body irritant and should be used only in selected cases; it is contraindicated in the presence of an intraspinal inflammatory lesion.

Some individuals prefer air or oxygen injection (40 to 50 cc.) for diagnosis of intervertebral disk protrusion.[25] When this is used, it is allowed to ascend into the cerebral subarachnoid spaces where it is absorbed within a short period. In the hands of roentgenologists well trained in this work, air may be satisfactory in many instances.

Thorotrast has been used with excellent visualization, but the possibility of late toxic effects from this substance have prevented its widespread acceptance.

Treatment of Protruded Intervertebral Disks.—No therapy is required if there is no compression of nerve roots or spinal cord. The protruded fragment usually appears as a hard crumpled mass of fibrocartilage; at times, this is attached to the disk margin but usually is seen lying free. It may vary in size from 2 mm. in diameter up to a mass large enough to block

the spinal canal. Occasionally, one finds a soft bit of tissue, surrounded by gelatinous material.

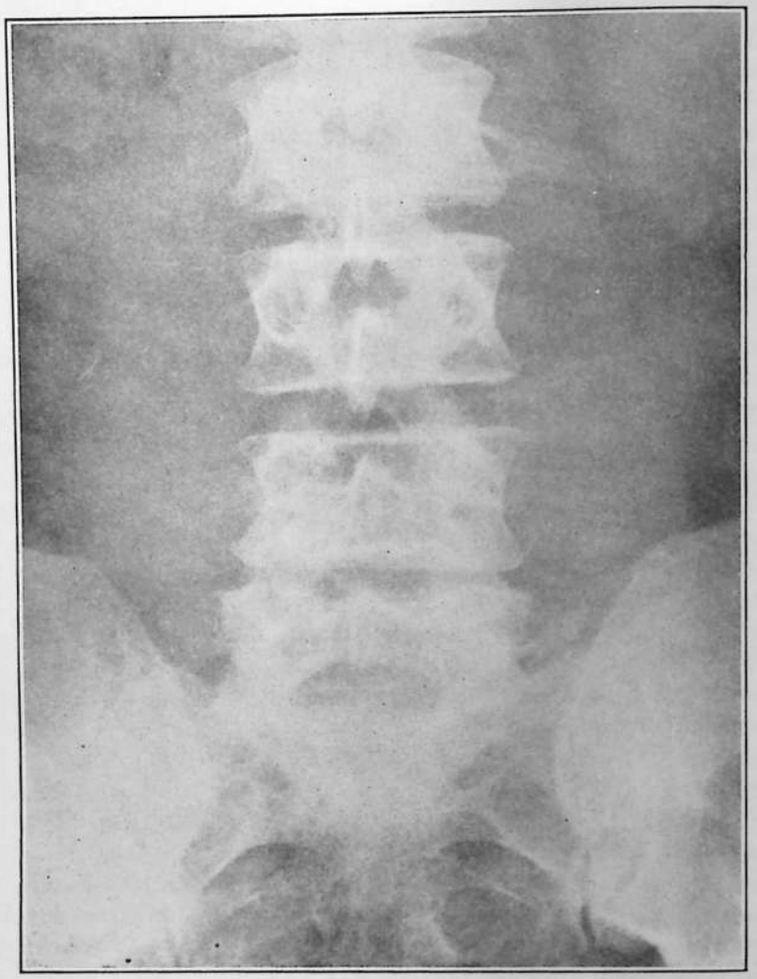


FIG. 301.—Lumbar spine showing **narrowing of the intervertebral space** between the 4th and 5th lumbar vertebra and between the 5th lumbar vertebra and the sacrum; the intervertebral disks are narrowed. This patient, now thirty-nine years of age, fell astride a log fifteen years ago. Thirteen years ago he developed pain in the back which has persisted chronically. In June, 1939, he developed sudden severe pain radiating down both legs in the sciatic distribution. A filling defect was seen following lipiodol instillation; a **herniation of the intervertebral disk** was found at operation impinging upon the fifth lumbar nerve on the left side.

If producing repeated symptoms (as checked by careful neurological study), despite conservative care, remove the protruded portion of the disk.[125]

Fusion is not done unless there is an accompanying spondylolisthesis or other disease process leading to an unstable low back.[52]

The usual exposure is unilateral between the laminæ without removal of bone (Love's operation) *or with the removal of a small bit of lamina if necessary* when the interlaminal opening is too small. Dandy advises *removal of the entire necrotic content* of the interior of the disk with curets. In "concealed disks," Dandy breaks up the interior of the disks with forceps and removes enough of the lining cartilage to bare the bone and permit granulations to fill the cavity.[31–33]

This must be followed by approximately two or three weeks of rest in bed, after which the patient is allowed up with a brace or supportive belt. Exercises are then employed to strengthen the back muscles. Mixter and Barr advise against driving an auto, any lifting or sudden strain for a period of three months; muscle building exercises must be continued during this period. Light work may be performed at the end of three months and moderately heavy work may be resumed at six months. All very heavy work or heavy lifting should be forbidden, although in some instances, patients have done this without apparent harm.

Results Following Operative Removal of Protruded Disks.—*Almost 100 per cent of 58 patients were immediately relieved of pain in the leg following operative removal of the ruptured disk. Conservative measures are suggested in all cases of suspected herniation of an intervertebral disk. If the recurring sciatic and low back pain are not relieved by ordinary conservative measures operation is advised. This should be performed only by individuals with considerable experience in spinal surgery.* Love and Walsh reported only 2 hospital deaths and 5 recurrences in a series of 500 patients operated upon.[98] Barr and Mixter reported good results in 80 per cent of patients, fair results in 10 per cent, and poor results in the remaining 10 per cent.

Radiologic Diagnosis of Disease of the Disks.—Roentgen-rays taken immediately following acute trauma may not reveal all of the damage to the disks, and a delay of several months following injury is sometimes necessary before radiologic examination shows secondary changes. Herniation of the nucleus pulposus does not produce roentgen findings at once. *Important points to be observed include the width of the intervertebral space and the condition of the margins of the bodies of the adjacent vertebræ. Smooth excavations into the bodies of the vertebræ may be noted radiologically.* Following herniation of the nucleus pulposus, a fibrous reaction occurs which is followed in some weeks by a rim of cartilage and later by a surrounding wall of sclerosed bone. Several months following the injury roentgenograms may show deposition of calcium in the bodies of the vertebræ. These depositions were formerly thought to be enchondromas and are now known as *Schmorl's knots or nodules.*

Pathology of the Bodies of the Vertebræ.—This is usually present as osteoporosis, spherical disks or biconcave bodies of the vertebræ. It is probably caused by softening of the bodies of the vertebræ, possibly as a result of chronic systemic disease and expansion of the nucleus pulposus. Generalized spinal osteoporosis may be seen, especially in women over the age of fifty and has been called a post-menopausal osteoporosis.

Significance of Changes in the Intervertebral Disks.—Many changes in the intervertebral disks are of little significance.[57] The mere presence of such changes in a patient with backache does not necessarily indicate an etiologic relationship. *Herniation of the nucleus pulposus occurs most commonly into the body of the vertebræ and is not often of clinical importance.*

Posterolateral herniations with pressure on lumbar nerves are those for which operative intervention may be required.

Narrowing of an intervertebral disk is not always an indication of protrusion of the disk; this may result from degenerative or fibrotic changes in the disk without protrusion. Protrusion posteriorly may be present in the absence of narrowing. Inasmuch as narrowing of the intervertebral space at the site of a protruded disk occurs in less than 50 per cent of cases, *ordinary roentgenograms have a limited value in the diagnosis of herniation of a disk.*

Escape of the nucleus pulposus with subsequent development of a localized arthritis may occur from trauma to the annulus fibrosus by a lumbar puncture needle.[113]

THICKENING AND FIBROSIS OF THE LIGAMENTUM FLAVUM

This ligament arises from the upper and dorsal margins of each lamina and passes upward between the lamina above.[17] These bands fuse in the mid-line. They possess lateral expansions which extend downward toward the anterolateral surface of the neural canal, *forming the posterior margins of the intervertebral foramina.* Each ligament lies directly above the intervertebral disk.

A narrow space is formed by the ligament above, and the intervertebral disk below. The nerve roots, after emerging from the dural canal, *pass through these spaces and may thus be compressed either by posterior protrusion of the disk, enlargement of the ligament, or both.*

The ligamentum flavum is normally composed of yellow elastic fibers, and is approximately 2 or 3 mm. thick in the lumbar region. Enlargements of the ligament result mainly from injury or scar tissue; in such cases, the ligament may become 1 cm. thick. Such enlargement is most frequent between the fourth and fifth lumbar vertebræ.

Thickening of the ligamentum flavum has been reported in almost 90 per cent of cases of herniated intervertebral disks. This thickening rarely occurs (4 per cent of cases operated upon) without an associated protrusion of the disk. The finding of such a hypertrophied ligament should lead to careful search for a protruded underlying disk.

Symptoms are identical with those of rupture or dislocation of portions of the disk. If severe pain results, the hypertrophied ligament is removed.

SENILE OSTEOPOROSIS OF THE SPINE

General osteoporosis is commonly present in elderly persons.[51] Generalized osteoporosis in adults has been explained on the basis of chronic or intermittent negative mineral balance over a period of many years.

Albright and his associates have defined osteoporosis as a condition of adult bone in which there is a failure of osteoblasts to lay down an organic matrix as opposed to osteomalacia in which there is a failure of deposition in this matrix of the calcium-phosphate-carbonate complex.[3] The etiologic factor in osteomalacia is an acute calcium-vitamin D starvation in the adult.[105]

Senile osteoporosis of the spine *is a common finding* characterized pathologically by marked decalcification of the bone. In elderly persons, it is often a crippling disorder in which there is *severe pain in the back* together with *progressive spinal deformity.* Black has recently studied over 200 cases of senile osteoporosis; about 80 per cent of these occurred in females.[14] The patients ranged in age from forty-five to eighty-seven years, averaging sixty-two years.

In some of the patients, marked osteoporosis was present without symptoms. *Most of the patients presented the following picture:* about the age of fifty-nine, there develops a gradual weakness, dull ache or fatigue in the low back, gradually becoming worse for a period of several years. After a fall, twist, or during lifting or bending, the patient may notice an acute pain or sudden snap in the back; this may be of such marked severity that the patient is confined to bed for days or weeks. Intermittent attacks of sharp pain follow; a dull persistent backache may be present in the intervals. Relief is usually secured by rest, especially in bed, support to the back and physical therapy. Lifting, twisting or activity usually aggravate the discomfort. The pain is most often noticed in the lumbar and lower dorsal regions. In some cases, the pain may radiate along nerve roots to the legs, or to the front of the chest or abdomen.

Mental depression is common. The patient walks with small steps; the back feels insecure, as though it may give way. Muscular spasm is present if pain is severe. There is often a kyphosis in the thoracic region, obliteration of lumbar lordosis and increase in cervical lordosis.

In *differential diagnosis,* one must rule out hyperparathyroidism, metastatic malignancy, multiple myeloma, spondylitis, fracture, Cushing syndrome, osteoporosis associated with hyperthyroidism, etc. In patients with senile osteoporosis, normal values are usually obtained for blood calcium, phosphorus, phosphatase, sedimentation rate, B. M. R., urinalysis and blood count. Roentgen examination reveals the osteoporosis of the vertebræ, collapse of the vertebræ and expansion of the intervertebral disks in long standing cases, and in some cases, pathologic fractures.

The etiology of this disorder is unknown. Factors which may play a rôle include: inadequate intake of calcium, poor absorption of calcium from the intestinal tract, inadequate intake of vitamin D and possibly an endocrine factor. The *frequency of the occurrence of osteoporosis of the spine and pelvis in women after the menopause* has been commented upon. Albright found that these patients could be put into a positive calcium and phosphorus balance by the *intramuscular injection of estrogenic substance.* It is too early to state whether this substance will be of practical value in the treatment of these patients.

Black has suggested the following *regimen of treatment* for these patients: a diet adequate in carbohydrate, fat, protein and vitamins and high in calcium and phosphorus; tribasic calcium phosphate, 4 grams and 4 cc. of cod liver oil three times a day before meals; support for the spine; use of mild sedatives, heat and massage in the acute stages if necessary for relief of discomfort. It is usually advisable to supply these patients with high back corsets reinforced with steel stays or a Taylor brace. In women, estrogenic substance should be used in addition to the above therapy. Although therapy relieves pain and tends to stop progression of the deformity, recalcification of the affected vertebræ has not been demonstrated roentgenologically. Treatment of these patients should be continued for at least several years.

CONGENITAL ANOMALIES OF THE SPINE LEADING TO LOW BACK PAIN

The normal human vertebral column is composed of 7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 coccygeal segments. In about 20 per cent of individuals variations from this pattern occur.[114] An abnormal number of cervical vertebræ is uncommon while the thoracic and lumbar total varies between 16 and 18, with the greater tendency toward the lower figure. Congenital abnormalities are very common in the low back.

More than one-third of all spines roentgen-rayed for any purpose have shown congenital abnormalities.[73] *Most of these do not cause symptoms,* but congenital defects are probably an important factor in producing weakness of the architecture of the spine, leading to points of lowered resistance to strain. *In these patients, the spine is probably more vulnerable to injury than in normal persons. Symptoms are usually not incited by the congenital abnormalities themselves, but by trauma or poor posture.* Congenital defects of the spine may decrease the motion or mechanical stability of the spine, and render strain more likely. Among the **congenital abnormalities** which may directly or indirectly lead to low back pain are:

1. Abnormality of shape and directional slant of the articular facets at the lumbosacral joint.
2. Spina bifida.
3. Increase or decrease in number of the lumbar vertebræ.
4. Abnormalities in size and shape of the transverse processes of the fifth lumbar vertebra.
5. *Spondylolisthesis.*
6. Failure of fusion of the first sacral segment.

Wagner has listed 12 types of congenital defects of the lumbosacral joints which produce referred pains in the extremities. One of the commonest is a difference in shape and directional slant of the articular facets at the lumbosacral joint. Instead of the crescentic and moderately long outline normally presented by the lumbar articular facets, these may be short and flat on one or both sides, resulting in a decrease of stability and predisposing to strain on the less secure side. Such changes may be associated with an occult spina bifida.

Trotter has studied a large number of cadavers and has found accessory articular facets at the sacro-iliac articulation in 50 per cent of white and in 20 per cent of colored patients.[164]

Lumbosacral anomalies usually cause symptoms only from faulty mechanics. The low back remains compensated so long as the ligaments and muscles are of proper tone and strength to allow the individual to continue his activities without fatigue. When the muscles and ligaments fail to support the lumbosacral area, decompensation and back pain occur. Muscles lose tone and power with age; thus, although a back may be well compensated at twenty, at the age of forty muscular power may be insufficient to adequately protect the low back from strain.[62]

Present evidence suggests that the form and number of segments of the human spine are not constant. There seems to be an evolutionary trend toward a decreased number of thoracolumbar segments and an increased stability at the lumbosacral junction.

Sacralization of the lowest lumbar segment means that the lowest lumbar segment is so altered in form that it must be classed as sacral, leaving only 4 lumbar elements. A *transitional sacral vertebra* is one which has assumed sacral characteristics such as enlargement of the transverse processes and variation in the plane of articular processes from a sagittal to a coronal direction.[130]

In view of man's upright posture and forwardly inclined sacrum, structural variations at the lumbosacral joint are very significant, as they may result in a change of weight transmission at the important junction of the mobile spinal segment and the immobile portion.

In quadrupeds, the lumbosacral facets lie most often in a sagittal plane; in man, these facets tend to assume a coronal plane. Great stability and considerable agility would be attained if the lumbosacral facets were placed in a symmetrical oblique plane.

An attempt was made to correlate the roentgen and anatomical findings at the lumbosacral junction by comparison with dissected specimens. It was found that the degree of degenerative changes (osteo-arthritis) present in a given case was a great deal more advanced than the *x*-rays indicated, especially in the posterior elements of the vertebral segments. They also noted that although the *x*-ray picture at times suggested failure of fusion, anatomical specimens showed these segments intact.

It was also found in dissected specimens that no facet plane of the lumbosacral region was purely oblique, sagittal or coronal, but that there was some curved component, corresponding to a segment of a cylinder, either completely or in part. Although the lumbosacral facets appear in a roentgenogram to be oblique, they are actually curved. Variations in the plane of the posterior lumbosacral articulations are often difficult to detect even with stereoscopic and oblique projection roentgenograms.

One of the best criteria for judging the probability of a relationship between symptoms of pain and articular asymmetry is early degenerative changes at these joints. The first sign of this is narrowing and irregularity of the interarticular space and increased density of the subarticular bone.

Osteo-arthritic lipping may be seen later as evidenced by a sharpening of the normally round, smooth contour of the articular facet margins.

Approximately 25 per cent of all human spines show asymmetry of lumbosacral articulations.

Fusion of enlarged transverse processes to the sacrum is not a mechanical disability; bilateral articulation of enlarged processes with the sacrum or ilia usually results in no asymmetry of weight transmission. Spinal mechanics are altered if there is unilateral impingement without fusion or bilateral and asymmetrical impingement and fusion; this produces a lateral fulcrum so that the articulations on the side opposite to the impinging process move through a larger arc of motion and thus bear a major share of the transmitted stresses.

The most common forms of *failure of fusion* in the spine include: failure of fusion of the lamina; of the laminæ and pedicles; and of the various apophyses which may then remain as separate ossicles. The stability of the spine is affected if these failures of fusion include the last lumbar and first sacral segments.

Occasionally they are misinterpreted as fractures.

Most of the opposing surfaces of the vertebral bodies in man approximate a horizontal plane. At the lumbosacral junction, the plane of this articulation averages 45 degrees from the horizontal (measuring the degree of tilt of the upper surface of the sacrum to the horizontal). An increase of this inclination will increase shearing forces at this joint and a decrease in the inclination will reduce these.

The posterior articulations resist the preponderance of the shear stresses at the lumbosacral joint. The lowest incidence of degenerative changes (osteo-arthritis) (as evidenced by overgrowth of bone along the joint margins) occurred when the lumbosacral articulations were in a bilateral, symmetrical, coronal plane; this position occurred in 22 per cent of all spines examined. Oblique (curved) articulations were found in 44 per cent and showed the second lowest incidence of arthritis. The highest incidence of arthritic involvement was found in those with asymmetrical articulations —seen in 25 per cent of spines which were examined. Sagittal articulations were seen in only 8 per cent of the spines; these also showed a high incidence of arthritic change.

SPINA BIFIDA

Spina bifida refers to a group of congenital malformations of the vertebral canal in which a protrusion of some of its contents occurs. *It occurs in one out of every thousand children and is fatal in about 90 per cent of these.* The defect is usually visible at birth; however, an *occult type of spina bifida* may occur, which may never lead to symptoms and may be discovered only by accident. This defect occurs most often in the lumbosacral and cervical region; in these two positions, the neural arches unite (posteriorly) at a later date than in other portions of the spine. Because of the presence of a bony defect posteriorly, protrusions of membranes or even of the spinal cord may occur. Rarely, such a protrusion has occurred anteriorly, leading to additional symptoms of an abdominal mass.[83]

The *posterior protrusion* from such a defect may vary from 1 to 5 inches. It is usually soft and elastic and may be pedunculated. In the presence of an *occult spina bifida*, the diagnosis is at times suggested by the finding of *hypertrichosis of the overlying skin, a dimple of the skin* or *an overlying lipoma* in the subcutaneous tissue.

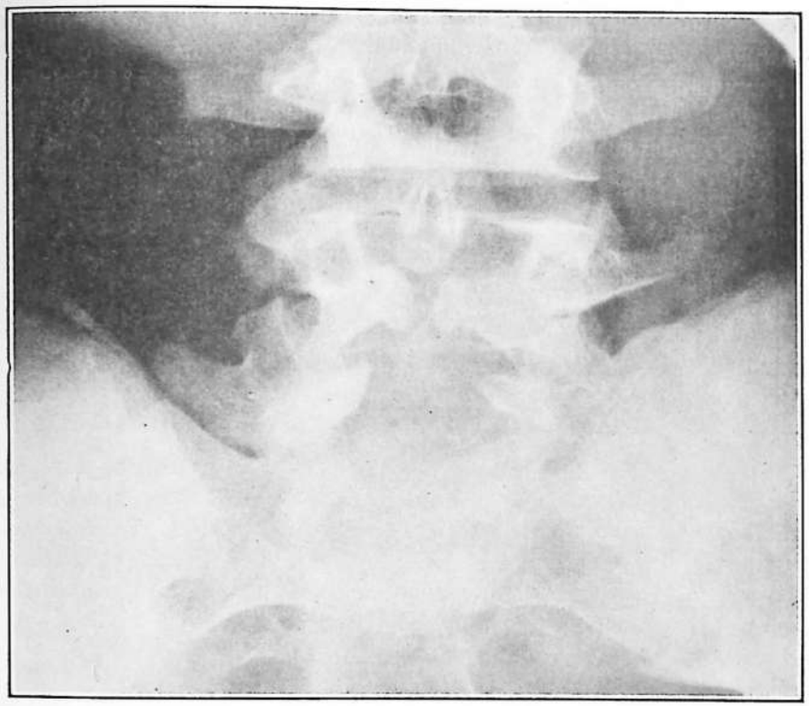


FIG. 302.—**Spina bifida occulta** in a boy aged seventeen years. This involved two segments. In addition there is an anomaly, either a sacralization of the fifth lumbar vertebra on the left or a failure of sacralization of the first sacral segment on the right.

Symptoms arising from a spina bifida may include: 1. Local swelling (cyst formation) over the affected region.

2. Low back pain.
3. Enuresis or incontinence (of bladder and rectum).
4. Areas of anesthesia and muscular atrophy (or complete paralysis of the lower limbs).
5. Association of club foot (in some cases).

The occult form may be found in 5 per cent of all spines examined roentgenologically.[37] These patients may manifest no symptoms; the lesion may not be known until a roentgen examination of the back is made for some cause. Changes in the articular facets of the spine are at times associated with a spina bifida occulta.

VARIATIONS IN NUMBER OF THE LUMBAR VERTEBRÆ

Four or six lumbar vertebræ may be found. Kuhns believes that an increase in the number of lumbar vertebræ tends to make strain somewhat easier, while fewer vertebræ may lead to increased stability of the spine.

VARIATIONS IN THE FIFTH LUMBAR VERTEBRA

Investigators could not determine what should be called a normal fifth lumbar vertebra after examining 10,000 specimens.[140] Very long transverse processes of the last lumbar vertebra have been found in 25 per cent of cases of low back pain, often with the formation of a true joint between their ends and the top of the sacrum. This has been called the "*lumbosacral transverse joint.*" If such an abnormality is present unilaterally, greater stability of the spine occurs on that side permitting peculiarities of motion and increasing the likelihood of a strain. If the formation is present bilaterally, the joint is made more secure than normal, but less flexible.

In some of these cases, a joint may be formed by the enlarged transverse process and the ilium leading to low back pain due to the development of a bursitis or an arthritis.[13] Taylor has stressed the importance of anomalies of the lumbosacral articulations.[160]

EXAGGERATION OF THE LUMBOSACRAL ANGLE

The normal weight-bearing axis of the body passes anterior to the lumbosacral junction, producing a shearing strain proportional to the obliquity of the superior surface of the first sacral vertebra with the horizontal. This angle is approximately 43 degrees but may reach nearly a right angle. Accentuation of this angle adds constant strain upon the ligaments and muscles.

SPONDYLOLISTHESIS

Spondylolisthesis was first described in 1782. Unfortunately, this condition is better known among obstetricians than among general practitioners. In a series of more than *1000 consecutive patients complaining of sciatica and low back pain, 4 per cent had spondylolisthesis.* This is no higher than the average incidence of this defect. Although a relatively uncommon condition, *it is an important cause of pain low in the back.*

This generally consists of a *forward displacement of the fifth lumbar vertebra and spinal column upon the sacrum,* or the fourth lumbar vertebra and spinal column upon the fifth lumbar vertebra, from congenital abnormalities or from trauma.[74]

The term is derived from the Greek, meaning a sliding of the vertebra. With the sliding of the last lumbar vertebra over the sacrum and into the pelvis, the spinal column is carried forward with the vertebra. There is no dislocation of the articular processes of the vertebra and the sacrum, but the slipping of the vertebra is permitted by a stretching or fracture of the interarticular processes. The fifth lumbar vertebra thus becomes

much elongated anteroposteriorly and may even appear to be bent over the sacrum like a clamp.

This condition results usually from a lack of fusion of the ossification centers of the lateral portions of the neural arch producing a weak spot made up of cartilage or held together by fibrous bands. Following injury or the carrying of heavy weights, these gradually give way to the pressure of the trunk, allowing the body of the vertebra to warp over the sacrum and into the pelvis. It has been suggested that trauma during delivery or shortly thereafter may cause some cases of spondylolisthesis.[78]

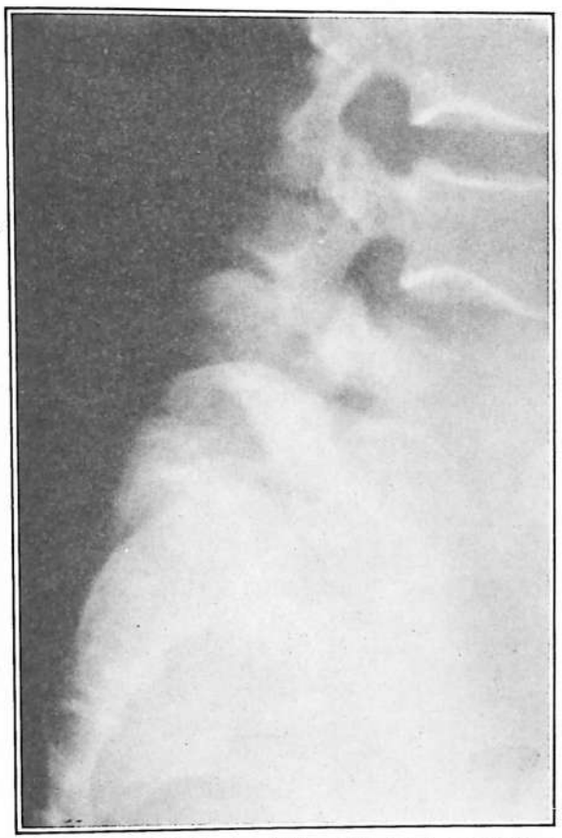


FIG. 303.—Marked **spondylolisthesis** of the fifth lumbar vertebra on the sacrum due to an anomaly in the development of the pedicles and lamina of the fifth lumbar vertebra. The patient, who is forty-eight years of age, was well until he fell backward, dropping 6 feet, striking his left side and back. He then developed pain on motion of the spine and deep muscular pain along the left side of the vertebræ in the lumbar region.

In more than 100 such patients operated upon at one clinic, every case showed a failure of fusion in the lamina between the superior and inferior articular processes, the defect usually being bilateral; the osseous portions were connected with fibrous tissue. The posterior portions of the

abnormal vertebræ were underdeveloped and hypermobile. Spina bifida occulta and an exaggerated lumbosacral angle were often found associated with the spondylolisthesis.

There is *spasm of the erector spinæ muscles* and *marked limitation of anteroposterior motion* in the involved region. These individuals present a *tender depression above the sacrum*, over the last lumbar vertebra.[20] A prominence can be felt above the eminence of the sacrum in some cases by rectal or vaginal examination. Another suggestive diagnostic feature is the *short step and narrow tread* (tight rope walker's gait). *Final diagnosis must be made by roentgenologic examination.*

Spondylolisthesis may cause no symptoms and may be detected only by a routine roentgen examination of the lower spine. *Backache* is the most common symptom.[110] *Mild cases with minor or no symptoms require no special treatment other than caution against overactivity or injury.* If acute pain occurs, this may be relieved by immobilization of the spine in flexion by a cast extending from the thighs to above the rib margins. If less acute symptoms are present, a Castex jacket or brace may be used by the ambulatory patient. Postural exercises are also prescribed to strengthen the muscles of the back.

In other individuals, low back pain may be severe; this may become progressively worse and require surgical interference. Pain may be localized at the lumbosacral joint or may radiate down along the course of one or both sciatic nerves. Symptoms are aggravated by strain and exercise. *A spondylolisthesis cannot be reduced or replaced and a* **spinal fusion** *may be necessary for permanent relief.*

Failure of Fusion of the First Sacral Segment.—In this condition, the sacrum is divided into two parts, the first sacral portion being of the lumbar type.

LOW BACK PAIN REFERRED FROM OTHER ORGANS

Gynecological.—*Backache due to gynecological conditions is not as common as was formerly believed.* In one series, it was concluded that 98 per cent of all backache in women was due to cervicitis. Uterine displacements are one of the first conditions mentioned by fourth year medical students as a cause of backache. However, Goldthwait states that *backache due to purely gynecological conditions is relatively rare.* This relationship between low back pain and pelvic disorders of women has been much disputed. Most individuals now concur that the rôle of gynecological disturbances in backache has been grossly exaggerated. *Gynecological pathology may occasionally be an important cause of low back pain, but most low back pain will develop from disorders of the spine, especially of its ligaments and muscles.*

Retroversion and genital relaxation are the main gynecological causes of backache. Others have stated that the retroflexed or retroverted uterus never causes backache. The discomfort in these cases is referred most often to the region of the sacrum. Less frequent causes include uterine fibroids, malignancy of the female genital tract and disease of the tubes and ovaries.[19,99] *Late stages of carcinoma of the cervix of the uterus may be attended by severe pain low in the back.* A common cause of backache of multiparous women is malposition of the pelvis, which produces strain of ligaments and muscles; although no bony changes are seen in the early stages, lateral films taken in the upright position may reveal an increased sacral angle or a horizontal sacrum.[94]

Genito-urinary.—The rôle of disease of the genito-urinary tract in the production of low back pain has also been exaggerated.[6] Twenty-five per cent of patients with *prostatitis* may complain of low back pain. Disease of the seminal vesicles is occasionally the cause of this discomfort. In a large series of patients with chronic prostatitis, pain was frequently referred over the sacro-iliac joint or along the sciatic nerve.[173] *Neoplasm of the prostate* is associated (in some instances) with low back pain (even without evident metastasis to the spine). The pain of renal stone is usually located in the costovertebral angle; as the stone passes down the ureter, pain may be felt lower in the back.

Gastrointestinal Lesions.—Disease of the abdominal viscera may lead to low back pain. Any organ which is supplied by nerves originating from the lumbar and sacral segments of the cord may produce pain low in the back. Painful stimuli from an organ are conducted to the segment of the cord from which its sensory fibers are given off.[71] There they come in close relation with fibers for pain from the body surface arising in the same segment. This pain is referred to the surface rather than to the viscera because of the excess of sensory and localizing power of the surface over that of the viscera.

Gastrointestinal lesions which have produced pain low in the back include ulcer or neoplasm (usually in the colon), perforation of the bowel along the lower posterior abdominal wall, retrocecal appendicitis, intra-abdominal adhesions, hernias, chronic constipation, visceroptosis, hemorrhoids, colitis, proctitis and spasm of the sigmoid or rectum. The strain produced in the back from faulty alignment of the body in visceroptotic subjects may lead to low back pain. Pain in the back associated with duodenal ulcers (posteriorly) is usually much higher than the sacro-iliac joint, and should not be confused with local lesions in the low back.

Infections and New Growths.—*Many acute generalized infections with fever are associated with low back pain.* Such pain may occur in influenza, grippe, severe colds, the acute exanthemata, malaria, typhoid fever, undulant fever, pneumonia, tularemia, etc. In some generalized diseases, actual invasion of the muscular tissue may occur locally (as in trichiniasis).

Pain may also be caused by pressure upon nerves by *tumors* of the vertebræ; these are frequently *metastases from the breast, cervix of the uterus and prostate.* **Such pain may occur months before the metastatic vertebral lesions are demonstrable by roentgen-ray.**

An individual with metastasis to the spine hesitates to move or turn when he has found a comfortable position in bed as motion aggravates the pain; the individual with degenerative joint disease with root pains turns frequently and changes his position often as motion usually gives some relief.

It is not necessary to have involvement of the nerve root itself by the

metastatic process to have radicular pain. Root pains may be present in involvement of the vertebral body, its projections or soft tissue coverings.

Spinal Metastasis vs. Degenerative Joint Disease of the Spine (Osteo-arthritis)		
Distribution of pain	Usually limited to 1 or 2 nerve roots distribution (in a narrow zone).	Usually has wide areas of distribution.
Influence of weather	None; pain is constant	May be influenced by weather changes with changes in barometric pressure, etc.
Influence of roentgen therapy	Usually relieves pain	May ameliorate pain to some degree.
Motion	Markedly aggravates pain; once comfortable, hesitates to change position	Helps to relieve pain; patient changes position often.
Tenderness on deep percussion	Pain in every instance, if percussion is performed over an involved vertebra	No percussion tenderness.
Serum phosphatase	Usually increased	Normal.
Sedimentation rate	Usually increased	Normal.

When roentgen evidence of metastasis to the spine is demonstrable, the lesion is advanced and diffuse.[169] Practically all of the spongiosa of a vertebral body can be replaced by metastatic tissue without evidence of the lesion demonstrable by *x*-ray.[145] In most instances, osseous lesions become massive and widespread before they become apparent roentgenologically.

Pain is one of the earliest and most outstanding symptoms in spinal metastases. This is a *root or radicular pain* which is almost always limited to dermatomes or segments, supplied by the nerve roots which exit through the foramina formed by the involved vertebræ. This pain is aggravated by coughing, sneezing, yawning, straining at stool, bending movements of the spine or sudden jarring of the body.

Arthritic Backaches.—Rheumatoid arthritis of the spine (rheumatoid spondylitis) is a frequent cause of pain in the back. This type of pain may be most severe in the morning. The radiation of pain produced by an arthritis of the spine is commonly overlooked. Many *abdominal operations have been performed upon patients because of pain due solely to spinal arthritis.* Arthritic pain often varies with weather conditions and is usually relieved by analgesics. A discussion of this subject is presented elsewhere. (See chapter on Spondylitis.)

Hershey noted the high incidence in anatomic specimens of hypertrophic arthritic changes with spur formation in the region of the anterior surface of the sacro-iliac joint, particularly in the area traversed by the lumbosacral trunk.[75] *The lumbosacral trunk of the sacral plexus passes over the lower portion of the sacro-iliac joint anteriorly,* crossing the joint and lying directly on the capsule. In one-third of 64 specimens studied, the lumbosacral trunk was in direct contact with the sacro-iliac joint at the point at which it traversed the joint in its lower third. Hypertrophic changes at the sacro-iliac joint in the form of severe spur formation on the anterior

line of the joint were found in 25 per cent of the anatomic specimens studied. These changes can be seen in anteroposterior *x*-rays of the pelvis.

Other infectious processes such as *tuberculosis of the spine, osteomyelitis* of the vertebræ, typhoid osteomyelitis or periostitis may produce low back pain. Psoas abscess, however, is rarely a cause of low back pain.

Macey believed that backache could result from **changes in the interspinal ligaments** connecting the adjoining spinous processes.[100] These interspinal ligaments are stretched in flexion and function constantly. Backache due to changes in these ligaments is characterized by a localized painful region between the spinous processes which is aggravated by activity demanding a flexed position and which pain is relieved by procaine injection into the interspinal ligament.

Charcot's Disease of the Spine.—This is a possible cause of a radiating type of low back pain. Marked spinal deformity may be present with little protective muscular spasm. On roentgen examination, bizarre destructive distortions of the bodies of the vertebræ may be seen with or without luxations.

NEUROLOGICAL CAUSES OF LOW BACK PAIN

An enumeration of these has been presented in the classification of the causes of low back pain. Of these, several deserve special mention.

Herpes Zoster.—This disease is an infection, probably due to a filterable virus, involving especially the posterior roots and horns of the spinal cord. Although it may occur at any age, it is most common in middle-aged or older individuals. Its symptoms include: 1. *An erythematous skin eruption* in the distribution of one or two posterior root zones. Any root may be affected, but the fifth dorsal segment is the one most commonly involved.

2. Slight *fever.*

3. The appearance of small *vesicles* (about three days later); severe burning or sticking pains, and marked hypersensitiveness of the same area.

4. Brown pigmentation and pain may remain for months following disappearance of the vesicles.

Factors Useful in Detecting Malingering[156]

Estimate the patient's sincerity of purpose from his general demeanor.

Does the patient's body build, posture or occupation make him liable to injury?

There should be a reasonable degree of consistency between the severity of the injury and the symptoms.

Symptoms should usually appear within a few hours following injury in most instances of organic disease.

Was backache present previously?

Muscle spasm and sometimes *scoliosis* are present in organic disease of the back; *it is almost impossible for one to feign these except momentarily.*

In *organic disease, points of tenderness remain rather well localized during the first and subsequent examinations;* this is not often true in malingerers.

Examination of the articular facets, as well as the vertebral bodies, should be performed.

Other Neurologic Causes of Low Back Pain.—Meningitis and poliomyelitis usually offer but little diagnostic difficulty. *Tumors of the lower cord or cauda equina* may cause severe pain in the low back. Tumors within the spinal canal are uncommon, and are only one-sixth as frequent as intracranial tumors; more than one-half of the intraspinal tumors are extramedullary.

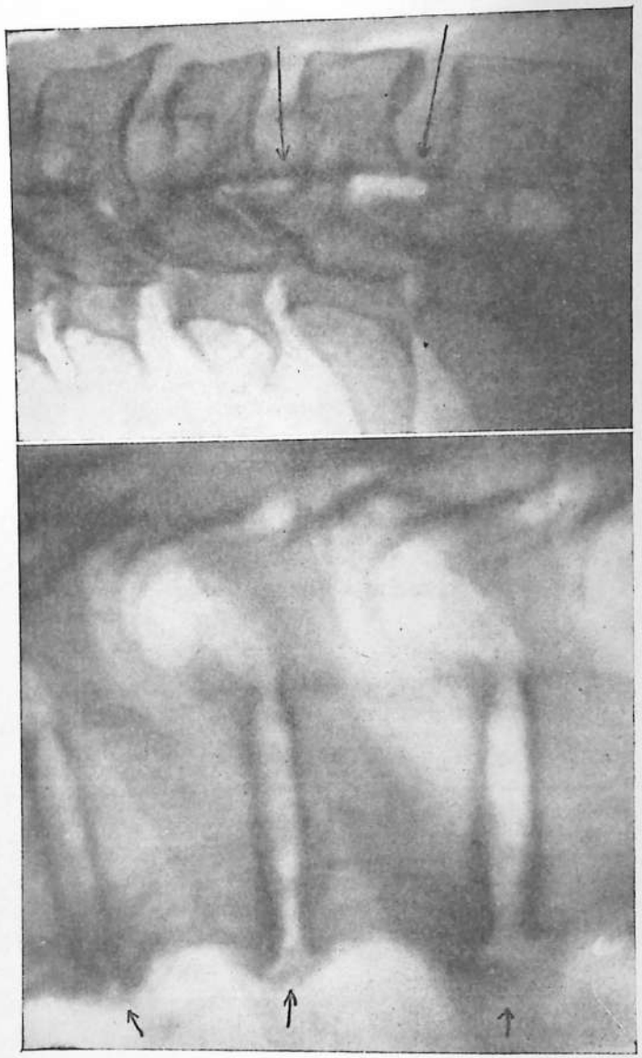


FIG. 304.—Spondylarthritis. *A*, *Calcification of anterior ligaments*. *B*, *Calcification of posterior ligaments*. Note ankylosis of lower apophyseal joints in *B*; disks are normal. (Oppenheimer, courtesy of Radiology.)

Tumors of the cauda equina may produce intense pain in the small of the back. **Symptoms of cauda equina tumors include**: 1. Severe pain in the low back and in the sciatic region or perineum.

2. Sphincteric difficulties.

3. Absent knee jerk and cremasteric reflexes.

4. Anesthesia and trophic ulcers in the perineal region.

Extradural tumors in this location are most often sarcomas; half of these arise from the vertebræ or are metastatic from distant points.

Eaton has discussed *pain caused by disease involving the sensory nerve roots*, secondary to extramedullary tumors of the spinal cord, protruded disks and other spinal lesions.[39] Such pain occurs in the distribution of the nerve root and is frequently localized in a small part of the total region supplied by that nerve root.

Such pain is produced or intensified by: (1) any sudden increase in intra-abdominal or intrathoracic pressure, as may occur in coughing, sneezing, lifting or straining at stool; or (2) by stretching of the involved nerve roots as on straight leg raising, spontaneous elongation of the spinal column during sleep, stooping forward with the knees straight or bending the head forward on the thorax.

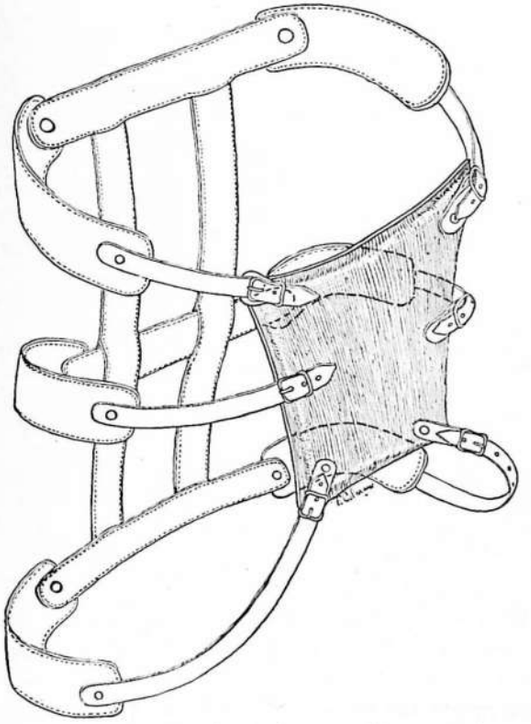


FIG. 305.—Goldthwait adjustable **back brace**. The posterior steel bars can be fashioned by the surgeon to fit the back snugly. (Barker and Trescher, Backache, courtesy of J. B. Lippincott Company.)

TREATMENT OF BACKACHE

A Correct Diagnosis is Essential to the Successful Treatment of Low Back Pain.—When the cause of the individual's backache is found, this condition must be treated. **In the majority of cases, treatment is that of acute or chronic**

strain of the low back. The pain of febrile conditions usually disappears with rest in bed and subsidence of the high fever. If abdominal visceral pathology is found, this should receive proper attention. Urologic, gynecologic, neurologic or psychiatric lesions may require the assistance of specialists in these fields. If several causes are found for the backache, the most serious lesions are treated first.

In the Acute Case of Back Strain Where a History of Injury or Strain is Present, a General Plan of Treatment Consists of:

Rest in bed, if possible, in recumbency, with a **firm mattress** or a fracture bed; pillow under the knees or low back in lumbosacral lesions. The patient *may* also lie on either side or face but should avoid sitting erect.

Fixation of the low back by adhesive strapping or binder.

Some patients respond well to periods (an hour or two at a time) of *hyperextension of the spine* by means of a small canvas hammock or sling 6 inches wide placed crosswise under the small of the back, acting as a *lumbar sling* to raise the lumbar area several inches from the bed; while the patient is in the sling, moleskin traction with 4 to 6 pound weights may be applied to each lower limb, as tolerated several hours a day.[111]

Heat (applied locally); constant warmth to low back (use baking or hot fomentations, heating pad, infra-red lamp or diathermy). If marked swelling or local bleeding are present, cold compresses or ice bags may be applied locally for the first several days.

Massage (which should not be begun too early).

Analgesics are usually necessary to relieve pain at least in the early stages. Nerve block may be used if necessary.

The back must be kept at rest until all signs of spasm and pain have disappeared.

In patients not responding to the above regimen, manipulation, with or without intravenous pentothal anesthesia, may be performed by one trained in such methods; following this, it may be advisable to employ a hyperextension plaster cast to maintain fixation for several weeks.

Corrective exercises (used when improvement occurs).

When ambulatory, supports for the low back (adhesive strapping, belts, or if necessary, carefully molded plaster or celluloid cast for several weeks) and if required, a heel lift if a short leg is found. Patient may need crutches temporarily.

The prognosis depends in a large measure upon whether a normal spine is involved. Injuries to abnormal spines require a longer period of healing; such spines may never return to a normal function. **No one method of treatment is appropriate for all patients with low back pain.**

General measures for the relief of low back pain include: *Rest,* relaxation *and support for the spine,* proper beds, strapping, heat, massage, belts, corsets and braces (not to be worn too long), proper shoes and exercises to improve body posture and to strengthen abdominal and spinal muscles. Other measures include roentgen therapy,[61] nerve block, fasciotomy, epidural injections, removal of a prolapsed disk, spinal fusion and facetectomy. Less than 10 per cent of cases of acute or chronic backache eventually require surgical treatment.

Pointers in the Treatment of Low Back Pain

Irritation by *adhesive plaster* is prevented by *painting the skin with compound tincture of benzoin before strapping.*

Strapping should be as tight as comfortable, should be done transversely and obliquely and should be maintained for a total of at least two weeks if possible. Adhesive strapping gives support for only a few days and must therefore be renewed frequently (every three or four days).

These patients should lie in the *horizontal position* with only one pillow under the head.

Relaxation of muscular spasm and support to the low back is obtained by a pillow under the knees and a small hard pillow under the lumbar spine (if lordosis persists); this is particularly helpful if sciatica is present.

Treatment is essentially the same for lumbosacral or sacro-iliac strains. Patients with sacro-iliac strains appreciate support of the pelvis with a belt; lumbosacral strains require support to the lower lumbar spine.

Heat is of benefit as it hastens the absorption of the exudate occurring about a strain, relieves muscle spasm and makes the patient more comfortable. Cold compresses or ice bags may be used for several days to lessen local swelling following acute lesions of muscles, ligaments or joint capsules.

Deep massage is not advocated in early stages of acute strains; although it decreases exudation and improves the local circulation; it may hinder healing of the torn ligaments.

The period of required rest in bed may vary from several days to several weeks with simple strains; support to the low back (as a corset or brace) should always be worn on resuming activities.

When low back pain is caused by static faults, postural misalignment must be corrected by a corset or brace, and corrective exercises, baking and massage should be instituted. Such exercises will restore the muscular balance of the thorax, abdomen and spine. Attention should also be given to other contributing factors such as weak feet, shortened lower extremity, disease of the hip joint or knee, etc. Supports for the low back (such as the Camp low sacro-iliac belt or high back support, Gossard corsets, or modified corset braces with paraspinal steel uprights and a metal pelvic band if required) can be obtained at most orthopedic appliance houses. The cost of these varies from $10 to $30, depending upon the type selected. A heel lift may be required for those patients with a shortened leg.

If pain continues despite recumbency and other supportive measures, manipulation under anesthesia or surgical procedures must be considered. In inveterate cases which do not respond at all to therapy, a fusion operation must be undertaken if a herniated disk is definitely ruled out.

Rest in Bed.—Rest of sufficient duration is essential in the acute stage of low back strains if one is to allow healing and attempt to prevent recurrences. **Rest and support of the affected joint are the primary requisites in the treatment of lumbosacral or sacro-iliac affections.** The preferred type of rest is on a thin, firm, non-sagging mattress; if there is any tendency for sagging, boards should be placed under the mattress. To make a *fracture bed at home:* Take 3 boards approximately 10 inches wide, $\frac{7}{8}$ inch

thick, and 5 feet 10 inches long, fastened together side by side by 2 cross cleats. These are placed lengthwise on the springs of the bed under the mattress. A small pillow under the knees and under the lumbar spine may prove helpful. Lying face down is also valuable as this permits the use of baking or hot fomentations to the back for the relief of pain; this position is more comfortable if one or two pillows are placed under the hips to provide sufficient flexion of the thighs.

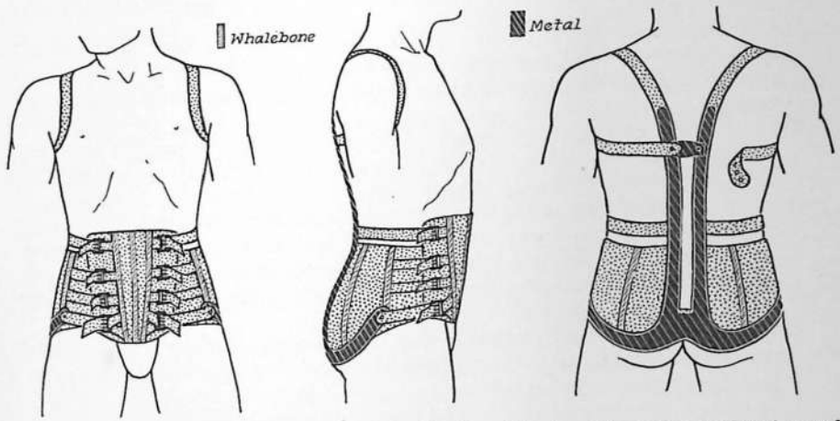


FIG. 306.—**Reinforced corset support** for the back, with shoulder straps; this is useful in the more severe types of low back pain when added support is required.

Many individuals are unable to spare the time necessary for rest in bed, and must be strapped so that the low back has a proper amount of support. For ambulatory patients, the wide lumbosacral belt or low back brace may be used as required. The most commonly employed form of support consists of strips of adhesive plaster 2 inches in width placed transversely across the back between the lower rib margins and the greater trochanter of the femur. For sacro-iliac strains, strapping need not extend above the iliac crests, but should exert lateral compression upon the ilia; in lumbosacral strains, it is continued above the rib margins. Occasionally, a light plaster cast may be required for stronger support. *The tendency is to use too little rather than too much adhesive.*

Heat.—This is a valuable form of treatment for back strain. The simplest form of heat therapy is an electric pad or hot water bottle placed under the painful area with the patient lying on his back; it is important to be sure that no metal comes in contact with the skin to avoid burns. These may be used for several hours at a time. Heat derived from an infra-red lamp or baker is also useful. Diathermy has been used also but is unnecessary, unless it is convenient and can be afforded.

In applying heat to the painful back, the patient should lie on the abdomen with a small pillow under the ankles and a pillow under the abdomen. Several folds of bath towel wrung out in hot water may be placed on the back, and may be covered with a blanket, while the patient lies relaxed. If desired, a hand lamp or baker may be applied while the patient lies relaxed in this position.

Massage.—Unfortunately, massage has not been used at the proper time or in the proper dose in some of these patients. *Massage should not be begun too early and once begun, should be continued well into the period of convalescence.*

One of the home remedy methods is a form of massage known as "ironing the back." This consists in a husky individual proceeding to iron a piece of flannel placed over the painful area on the back (with the patient lying on the abdomen) as though the patient were not there. Great care must be used to prevent the hot iron from touching the skin.

Exercises.—Abdominal and breathing exercises should be instituted as soon as possible. See Chapter XVII for details of exercises for the low back. These will stretch out the dorsal spine and ribs by using a hyper-extended position. When the patient becomes ambulatory, he is taught by exercises how to stand, sit and bend.[18] A support must be used for at least several weeks for the pelvis, lumbar spine, abdomen and lower half of the dorsal spine.

Exercises are usually necessary to restore atrophied muscles and to improve poor posture. In some instances, one must *stretch contracted muscles and fasciæ* (such as the erector spinæ, hamstring muscles, tensor fascia lata, contracted calf muscles, contracted gluteal muscles, etc.). The *erector spinæ muscles* may be *stretched* actively by having the patient lie on the back and markedly flex the thighs and knees, with the head bent forward, so that the chin almost touches the knees while the hands clasp below the knees holding these toward the chin. The *gastrocnemius* may be *stretched* by having the patient stand about 3 feet from a wall, with the soles firmly on the floor. The patient then leans forward as far as possible toward the wall, without bending the knees. The gastrocnemius may also be stretched by standing with the forepart of the foot raised on a book while the heel is on the floor.

The *anterior fascia of the thigh* may be *stretched* by having the patient stand with the chest against a wall, while the knee is forcibly flexed (in which position it may be held by the patient at the ankle). The *hamstrings are stretched* if the patient sits on the floor with the legs straight and feet upright against a baseboard. The patient then bends forward and touches the toes without bending the knees. Lateral portions of the fascia lata are stretched by having the patient stand for several minutes with the hip thrust out on the affected side while balancing the body by placing the hand on that side against the wall. Another maneuver which stretches the fascia lata is having the patient lean on a table with the hip thrust toward the table. *Stretch in the gluteal muscles* will be improved by contracting these at least ten times per hour. Abdominal muscles may be set by slow and rhythmic voluntary contractions of the muscles while lying on the back with the knees flexed. As improvement occurs, the abdominal muscles may be contracted with the knees flexed and the head and chest raised from the bed, with the hands placed on the knees.[124]

Analgesics.—One or more of the following drugs may be required for the temporary relief of pain in acute low back strain in adults:

Aspirin 0.3 gm. (gr. v) and *phenacetin* 0.3 gm. (gr. v) every three hours as necessary.

Codeine sulfate 15 to 30 mg. ($\frac{1}{4}$ or $\frac{1}{2}$ gr.) every three hours as necessary.

Morphine sulfate 15 mg. ($\frac{1}{4}$ gr.) may be required in occasional cases.

Judovich and Bates relieve *dorsolumbar pain* by nerve block with procaine and a substance ("Sarapin") obtained from the pitcher plant. In resistant cases, they employ from 20 to 30 per cent alcohol in procaine for these injections. In cases of dorsolumbar irritation they perform a *paravertebral block of the twelfth dorsal and first lumbar nerves;* when done correctly, there is a complete disappearance of both the anterior and posterior area of pain and tenderness within five or ten minutes.

Brav has compared the results of injection with procaine to those of merely inserting the needle (without injection) in patients with low back pain. *Dorsolumbar pain was relieved in 55 per cent of the patients by insertion of the needle alone.*[16] This is similar to the relief obtained by Sidel and Abrams in arthritic patients by injection of mere physiological saline solution.

Remember that malignancy must be suspected in continuous low back pain or bilateral sciatica which does not respond to proper therapy within a reasonable time.

In patients with **recurrent acute back strain,** if *ambulatory treatment* is necessary, this should consist of: 1. A supporting cloth corset or belt.

2. Baking and massage.

3. Exercises after the pain has subsided; prohibition of lifting, bending and strenuous work until proper exercises can be performed without pain.

4. Manipulative treatment, in some cases.

Most women will consent to the use of a thin cloth corset with laced back; this is applied and laced tightly (but comfortably) each time it is put on. It is as efficient for this condition as the more expensive types of surgical corsets. A well fitted canvas belt is usually prescribed in men with this disorder.

A small infra-red lamp may be bought or rented to heat the involved parts. Heat is used daily for several months, and *mild exercises are begun as soon as pain has subsided;* this tends to lessen the muscular atrophy which has occurred. Simple postural exercises are advised to develop the erector spinæ and the gluteal muscles. Swimming is one of the best forms of exercise for such patients after the stage of acute pain has passed.

When pain has disappeared, gradual restoration to activity is suggested with the use of a belt or brace, and work is resumed only when heavy setting up exercises can be done without pain. Three weeks is a minimum safe disability period for this type of case; six weeks is often better to avoid recurrence and ultimate chronic backache.

Corsets: Ober emphasized the fundamental observations on corsets made by Reynolds and Lovett in 1910. Bad corsets could produce static backache if they were too short in front and too long behind; if they were cut to exert the greatest pressure at the top and bottom behind, and at the waist line in front, particularly; and if the sacral curves were strongly marked but otherwise the corsets did not follow the rest of the anatomy of the back.

Good Corsets[123,133]

1. Should be short posteriorly and long enough in front (especially at the bottom) to support and lift the abdomen.
2. Should not be tight around the waist in front.
3. Should *follow the curves of the back snugly* with the back steels of the corset properly bent so that this occurs.
4. Should *fit snugly around the pelvis,* particularly between the trochanters and iliac crests, being long enough posteriorly to hold well over the buttocks.
5. Should *stop just below the inferior angles of the scapula above.*
6. The cloth of the corset must be cut so that the crest of the ilium fits into a sort of pocket to prevent the corset from sliding up or rolling backward over the crests.
7. Back laced corsets are adjusted more easily than solid back garments. Ober feels that if sacral pads are placed inside or extra belts are used outside, this reveals lack of knowledge of the corsetier in its correct construction.
8. The corset should not encircle the pelvis as if this were in a vise.
9. The patient's own corset muscles (the abdominal muscles and gluteus maximus) should be exercised properly so that they will eventually take over the function of the corset, which is then discarded.

Manipulation of the Low Back.—A few sprains are accompanied by subluxation of the affected joint, usually the sacro-iliac, or by an acute severe muscular spasm. They are the cases which respond so miraculously to manipulation. Unfortunately, the average surgeon considers any form of manipulation beneath his dignity. Manipulative methods of therapy in this disorder have been ignored by most of the medical profession.[44,109] *Phenomenal recoveries have occasionally followed such procedures, often producing immediate relief with a concomitant click or snap.* These good results have been attributed to the presence of replacement of subluxations or malalignments of contiguous articular surfaces, which had resulted from the laxity of the ligaments in the low back incident to a strain or sprain.[79] Manipulative treatment is of little value in the recurrent cases as only temporary relief is obtained. The essentials of these manipulations consist in stretching (more or less violently) muscles which are in acute painful spasm and in reducing any existing subluxation. *The most effective methods have as their basis strong flexion or extension of the thigh on the trunk with the knee straight and the patient flat on his back or face.* This transmits force to the low back muscles and shifts the pelvis on the spine by power carried through taut hamstrings or hip flexors. Manipulation is performed without anesthesia in some acute cases; chronic ones may require anesthesia, such as intravenous pentothal sodium. Preliminary use of heat, such as infra-red is useful in securing relaxation. Manipulation should be per-

formed only by individuals well trained in such therapy; it is inadvisable for the general practitioner to attempt such movements.

Taylor believes that manipulation is more efficient than heat in relieving muscle spasm in patients with low back strain.[159] He does not believe that any one maneuver is specific in these cases; all of these seem to produce good results including merely forcing the back through its normal ranges of motion, or torsion or a stiff jerk of the leg from a flexed position on the involved side. Any of these manipulations reach the spastic muscle fibers. Taylor also emphasizes that the patient must obtain *proper after-care following such manipulation;* this includes strapping of the lumbosacral area, rest in bed on a non-sag mattress, and later, physical therapy (when the acute stage has subsided).

In certain chronic strains, because of the presence of adhesions and muscle shortening (from protective spasm), proper mechanical alignment may be impossible without manipulation of the pelvic or spinal joints. These are usually performed under general or spinal anesthesia.

Lewin makes the following suggestions relative to manipulation of the low back:

Do not manipulate in the presence of tuberculosis, osteomyelitis, acute infection, acute arthritis or neoplasm.

Do not manipulate without a recent roentgen-ray.

To obtain complete relaxation, it is usually necessary to use ether.

Do not injure the patient—even though he may be asleep.

Manipulate by skill, rather than by force.

The bone and joint changes are usually worse than the roentgenogram reveals.

Do not manipulate unless you are prepared to supervise the after-care.

Procedures for Manipulation in Low Back Strain.—The patient lies flat on the back and relaxes. Relaxation is necessary as manipulation must be forceful. The right thigh is flexed slowly at the hip joint to a position 20 degrees less than a right angle (with the knee in full extension). The left thigh is then flexed likewise and following this both thighs are flexed together. Both thighs are then acutely flexed upon the abdomen with the knees flexed. The explanation of these maneuvers is as follows: The straight leg raising individually produces motion of each innominate on the sacrum at the sacro-iliac joints through the taut hamstring muscles; the motion with both legs together also produces flexion of the pelvis on the lumbar spine. Flexion of both knees and hips results in flexion in all of the lumbar segments.

The patient lies flat upon a table, while an assistant holds the patient's shoulders firmly to the table. The pelvis is rotated forcibly to the extreme right and left, thus activating especially the posterior articulation of the lumbar vertebræ and lumbosacral joint.

With the patient lying on his abdomen the thighs are slowly hyperextended separately, counterpressure being made over the corresponding sacro-iliac joint. Both thighs are then hyperextended with pressure over the lumbosacral region. The last maneuver consists in lifting the entire

pelvis from the table, thus hyperextending the lumbar spine. These maneuvers produce torsion of each innominate upon the sacrum at the sacro-iliac joint by traction through the taut iliofemoral ligament and rectus femoris, and then extension of the pelvis upon the spine and hyperextension of the lumbar vertebræ.

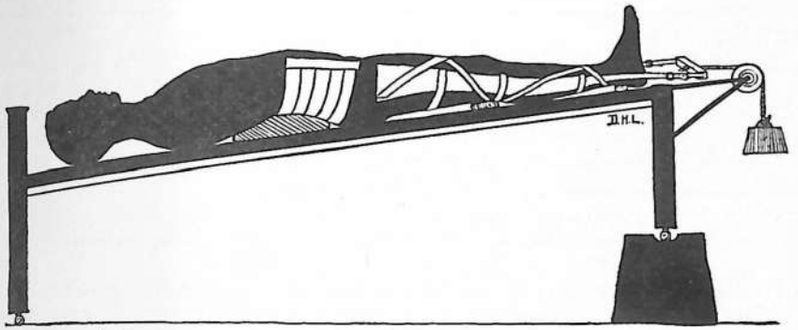


FIG. 307.—Adhesive strapping of the low back and adhesive leg traction in a patient with sciatic neuritis. Foot of bed is elevated 8 inches. Note support under the lumbar spine, knee and above heel. (Lewin, Orthopedic Surgery for Nurses, courtesy of W. B. Saunders Company.)

During one of these manipulations, Horwitz and Davidson have occasionally found an audible or palpable click or snap, often producing partial or complete relief immediately if the patient is conscious. Following these procedures, broad adhesive supports encircling the entire body or firm corsets are applied at once and a pillow is placed under the knees to obliterate lumbar lordosis; within a few days, massage and mild exercises (for the lumbar, abdominal and pelvic musculature) are instituted.

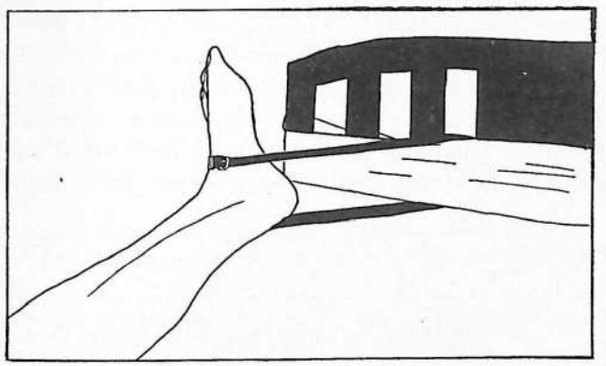


FIG. 308.—Simple method of maintaining traction on the leg. The foot of the bed is elevated. An old inner tube is drawn over the lower end of the bed; a small leather strap attached to this maintains traction on the leg. (Warren, courtesy of Jour. Bone and Joint Surg.)

If contracted ligaments or muscular elements are torn during the procedure, the pelvis, spine and one or both hips must be immobilized for approximately a month, following which, the patient is allowed out of

bed with support to the lower back. *Manipulation is not practiced in this country to the extent that it is used abroad.* Some of our orthopedic surgeons fear that manipulations such as these may cause avulsion of the disks.

Treatment of Severe or Chronic Low Back Strain[48]

Hospitalization.
Rest in bed on a non-sagging mattress (or with fracture boards on the springs and a firm hair mattress).
Traction to each leg (6 to 8 pounds), using moleskin adhesive tape or padded ankle cuffs; head and pelvic traction or a plaster cast may be necessary to relieve muscle spasm in some cases.
A small pillow under the lumbar spine for support.
Daily baking and massage alternating with diathermy; if one of these produces greater relief than the other, the less effective is discarded and the other is employed daily.
If sciatic pain is present and is not relieved by the above procedures, *herniation of an intervertebral disk* should be considered. If none are found, epidural injections may be performed.

Simple recumbency may be accomplished by placing the patient in bed on a Bradford frame without external immobilization, and using traction appliances on the head, lower extremities or pelvic girdle. Head traction is easily applied to the chin and occiput or to the occiput and forehead by suitable traction straps. Traction on the pelvis may be obtained by Buck's extension on both legs, or better, by traction on a well fitted pelvic girdle belt. Children between ten and sixteen years can easily endure traction of 20 to 25 pounds in each direction, weight being removed only during meals or bathing, etc. In advanced scoliosis, especially with rotation of vertebræ, lateral traction by weights and belts may be used.

Such a program may result in added pain during the first several days; within four to five days the patient should notice less pain if improvement is to be expected on this regimen. If relief is obtained (less pain and diminished muscle spasm), activity is gradually resumed after a period of ten to fourteen days. The patient then wears a well fitted corset and continues periods of rest in bed and massage for several months.

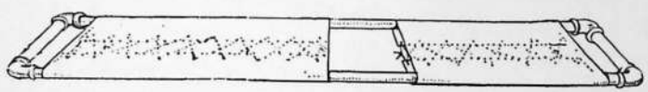


FIG. 309.—*Bradford frame:* Canvas strips are laced across the frame, which is made of ordinary gas pipe, in such a way as to leave an appropriate interval for the buttocks, under which the bed-pan may be passed. (Whitman.)

In the arthritic type of backache, the use of the convex *Bradford frame* is especially effective, although some difficulty may be experienced in accustoming the patient to lie upon this appliance. This is a rectangular structure built from iron pipe, and is covered with two sections of strong canvas, tightly drawn over the frame; the sections may be separated to

permit a bed-pan to be used. The frame must not be wide, especially in those patients with pains radiating down the lower extremities; additional benefit usually accrues if mild traction is applied to the lower extremities with counter-traction to the head and cervical spine. Sedation is required while the patient is accustoming himself to the frame.

Another useful appliance is the *Castex body shell,* carefully molded over the patient's back while he is in the prone position, with the spine adequately lordosed. The patient rests in this shell, thus splinting the back. Some individuals prefer this to the plaster of Paris shell because of the

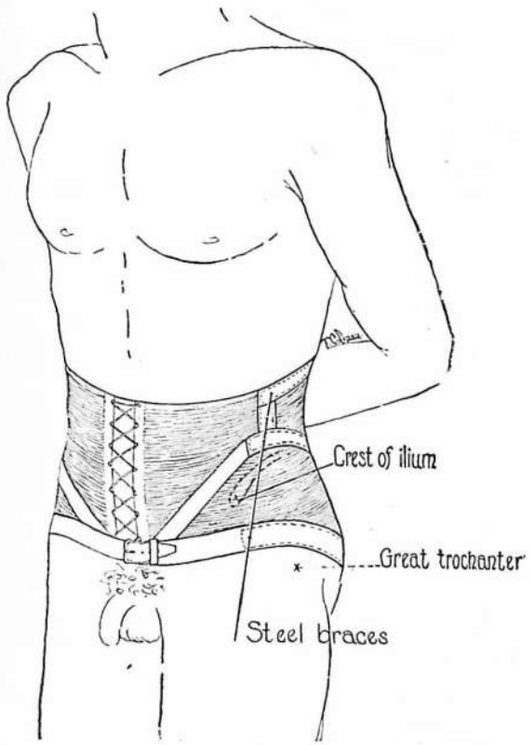


FIG. 310.—A *Bennett low back brace* designed to support the lumbar spine. (Barker and Trescher, Backache, courtesy of J. B. Lippincott Company.)

extreme lightness of the Castex and its ready applicability; the high cost is one of its drawbacks. The mode of application of "Castex" bandages and other details concerning them are described elsewhere.

Following subsidence of the acute symptoms, *low back support* should be continued for some time so that recurrences may be avoided. Of the many forms of low back braces, one of the lightest and most effective is the *Goldthwait low back brace.* The Bennett brace is also very satisfactory; however, in those patients requiring hyperextension (as in vertebral fractures), a Knight or Taylor type of brace may be used.

In backache due to *arthritis of the sacro-iliac joint, roentgen therapy* is

advisable if this is associated with or an early sign of rheumatoid spondylitis; if there is no associated spondylitis, a sacro-iliac support or polo belt should be worn and the individual should change to a less strenuous life. Rest, heat and massage will often prove very beneficial. Heavy lifting must be forbidden; we often tell these patients that they must *lift nothing heavier than a mortgage.*

In very robust individuals who must continue heavy work which subjects them to repeated strain, surgical fusion should be considered if conservative measures are of no avail,[111] although the results of many such fusions are disappointing. In all such cases, if sciatica is associated with low back pain, a herniated disk must be considered.

Congenital anomalies of the spine per se do not usually cause pain. Most of these patients who develop backache can usually be greatly relieved by the use of a chairback brace (extending from the lower portion of the sacrum to the twelfth dorsal or first lumbar vertebra, being reinforced with paraspinal steel supports), in addition to heat and mild massage. These measures will relieve strained muscles and ligaments and enable most of these individuals to carry on normal (but not excessive) activities.

Injuries to the Erector Spinæ Muscle.—Treatment includes a non-sag mattress; rest in bed, flat on the back with a firm pad, such as a folded sheet (not a soft pillow) under the lumbar region; and analgesics as required. If the patient tires of this position, he may lie on his face with pillows under the hips and chest. Both of these maneuvers hyperextend the spine and relax the damaged muscles. The *hard bed* is essential to the treatment of all back injuries. The principle of *hyperextension* is also used widely. *Heat* is applied to the painful area. *After forty-eight hours, massage* is begun and the patient may return to work in ten to twenty-one days.

Treatment of Chronic Industrial Backache

Gain the patient's confidence.

Insist upon six to ten weeks of complete rest, preferably in bed, but sometimes in plaster of Paris to *immobilize the lower spine* (the jacket is not sufficient and a spica must be used).[40] If placed in a large body cast, most patients who are malingering will admit this after several days in the cast; the latter is uncomfortable if the patient has no actual pathology of the back. *Complete rest alleviates pain when there is a real traumatic lesion because it removes all strain from damaged joints, muscles and ligaments.* If this does not stop the pain, the patient is usually malingering or has active bone disease such as metastatic carcinoma or a cord tumor.

When pain is overcome by complete rest, structures of the back may be reëducated with various forms of heat, massage, and active and passive motions. These must be continued long after the patient becomes ambulatory. During convalescence, an efficient back brace is worn. *The results of surgical fixation of various parts of the spine to relieve chronic pain, especially in laboring men, have been very disaupointing.*

Herniation of an intervertebral disc must always be considered.

Surgical Procedures for Low Back Pain.—Surgical intervention for the relief of low back pain does not appeal to many patients and may be frowned upon by some general practitioners. However, *about 10 per cent of patients with low back pain will not respond to the usual conservative non-operative procedures.*[27] This group is usually willing to undergo any necessary procedure for relief of long continued pain in the back.[153] The general practitioner must realize that the mortality from an operation such as spinal fusion is less than one-half of one per cent; this is as low as that of appendectomy in good surgical hands. It has been found that spinal fusion may "cure" 70 per cent of patients with severe low back pain who have not responded to non-operative means and will relieve an additional 16 per cent of from 50 to 90 per cent of their symptoms. Operative intervention is undertaken only if response to conservative treatment is without avail and if severe pain persists. Some lesions respond[88] poorly to non-operative treatment.

In patients with recurring severe low back and sciatic pain, in the absence of spondylolisthesis or congenital malformation of the fifth lumbar vertebra, a *herniated intervertebral disk* is probably present and should be removed.

Operative Procedures for Low Back Pain Include:

Manipulation under anesthesia, followed by plaster fixation.
Removal of a herniated intervertebral disk.
Transverse division of the fascia lata (Ober operation).
Procaine or saline injections along the course of the sciatic nerve (occasionally relieves persistent sciatic pain).
Epidural injections.
Removal of long or deformed transverse processes or thickened articular facets (Putti, Ghormley, Williams, Yglesias, Mitchell).
Fusion operations: for the lumbosacral joint and lower lumbar spine, the Hibbs type of operation;[76] for the sacro-iliac joints, the Smith-Petersen, Gaenslen[46] or Campbell; trisacral fusion (Chandler).
Miscellaneous: Subperiosteal stripping of the gluteus maximus muscle (of Heyman and Roberts); section of the pyriformis muscle, etc. (Freiberg).

Treatment of Contracted Iliotibial Band.—If sciatica is not present, the low back pain may be relieved by *special exercises.* These are done as follows: Standing with the affected side about 2½ feet from a table, the patient grasps the table with one hand and bends the affected hip toward the table as far as possible while maintaining the shoulder and pelvis in the same plane. This position is maintained for several seconds and is repeated five times. This is increased in number daily until the exercise is performed about twenty-five times twice a day.

With the recent work on herniated disks, the Ober operation is being performed much less often. Before performing an *Ober operation,* the surgeon must be sure that no pathologic condition exists in the spinal canal, especially in the cauda equina. Amelioration of low back discomfort and sciatica by the Ober operation may result from release of the squeezing

effect of the short iliotibial band and contracted fascia on the sciatic nerve where it passes over the acetabulum, lessened pressure on the sciatic nerve from a spastic gluteus maximus, but more likely, from relief of the pull of the contracted band which is producing a mechanical strain of the sacro-iliac or lumbosacral joint.

Tensor Fascia Lata Fasciotomy (Ober Operation).—This may be done in some patients where the predominating symptom is a sciatica of less than a year's duration without roentgen evidence of generalized spondylitis. The operative procedure consists in *cutting the fascia over and adjacent to the tensor fascia lata muscle.* The rationale behind its use is that some cases of sciatica result from a contracted iliotibial band and fascia lata.

FIG. 311.—Posterior view of the hip showing the *pyriformis muscle* as it emerges from within the pelvis through the great sacro-sciatic foramen. Note that the sciatic nerve passes under the pyriformis muscle. (Thiele, courtesy of Jour. Am. Med. Assn.)

Because of the simplicity of the operation and its minor character as well as the fact that it can be performed under local anesthesia, it is *particularly indicated in elderly patients or in patients whose general condition contra-indicates a major operation.* Another favorable feature is the short period of hospitalization required (about a week).

There has been no entirely satisfactory explanation why fasciotomy may relieve low back pain and sciatica. Although it is claimed that the Ober test indicates a tight fascia lata, reports are available to show that this test may be positive in individuals who have never had sciatica, in patients following spinal fusion which has resulted in complete cure of the patient's pain, and in some cases even after fasciotomy has produced

relief of the patient's symptoms. Likewise, the Ober test is negative in some patients with sciatica following a period of non-operative treatment.

Epidural Injections for Low Back Pain (See Sciatica).—Many technics have been outlined for injection in sciatica; some of these consist of injecting the fluid directly into the nerve trunk while others are concerned with epidural deposits. Fenz showed that the effect of these injections was due to the procaine and not to the large amount of fluid deposited as the same amount of physiologic saline had no effect whatever.[43]

Spinal Fusion.—If it is necessary to fuse a joint, some other joint must take up its work; this added strain may cause as much trouble in some cases as was present before the fusion. The main point referable to surgery of the spine or to fusions of joints is to be perfectly sure that the lumbar spine is flattened, or that the joints are in a position mid-way between full extension and full flexion. In this position, muscle balance is not disturbed and there is a better likelihood of good results.

The operation of spinal fusion has stood the test of time. The theory behind the use of spinal fusion is that a complete removal of all motion will result in a cessation of inflammation which may exist in and around the articulations and intervertebral foramina and, in addition, will relieve the supporting muscles and ligaments of strain which they could not bear previously. Fusion of the fifth lumbar and first sacral vertebra are usually advocated; in some cases, the fusion extends to include the fourth lumbar or other vertebræ.

In 147 cases of developmental abnormalities of the lumbosacral joint producing pain and disability, the operation of spinal fusion resulted in 73 per cent of cures, 14 per cent of patients improved, and no benefit in 13 per cent.[77]

The *Hibbs type of spinal fusion* is an exposure of the articulations and excision of the spinous processes close to the lamina. The articular facets are removed thoroughly. The lamina are curetted and, with a small chisel, chips of bone are placed across the articulations. The spinous processes are then cut up into small particles and replaced, thus reinforcing the affected area. Bone grafts unite very slowly so that the patient must wear some type of external immobilizing apparatus for eight to fourteen months.

The *Smith-Petersen fusion operation* consists in the removal of a rectangular piece of bone crossing the ilium and sacrum and wedging the sacro-iliac joint. The joint is destroyed by curettage or erosion and the rectangular piece of bone is wedged back in such a way as to cross the joint with cancellous bone.

Contraindications to Low Back Fusion Operations:[28] 1. Multiple or infectious arthritis.

2. Compensation cases before financial settlement.
3. Poor risks or elderly patients.
4. Girls before puberty.
5. Any patient without definite evidence of osseous deformity or disease until conservative measures have been tried thoroughly.

Indications for Fusion Operations on the Low Back and Pelvis[92]

Disease of a destructive character (as tuberculosis) which heals more readily with continued immobilization.
Patients with extreme muscular weakness without expectation of return of muscular power (as in poliomyelitis).
Continuance of severe disabling low backache (with or without congenital anomalies in the lumbosacral region) after a thorough trial of conservative therapy. Herniated disks should always be considered.
Spondylolisthesis producing symptoms.
Pain caused by an atrophic intervertebral disk associated with degenerative joint disease.
Chronic pain following fractures or fracture dislocations of the lumbosacral or sacro-iliac joints.
Marked asymmetry of the lumbosacral joints.
An abnormally acute lumbosacral angle.
Marked posterior displacement of the fifth lumbar vertebræ.
The presence of a transitional vertebræ.
Congenital abnormalities producing serious interference with movement or stability in the low back (rarely alone an indication).

Period of Hospitalization and Follow-up Care Following Fusion Operations.—These patients are maintained in recumbency in the hospital for approximately two months postoperatively, wearing a Taylor brace extending from the buttocks to about the tenth dorsal vertebra. At the end of this period, the patient is ambulatory and activities are gradually increased until the brace is discarded about five months postoperatively. An office worker may return to his occupation in three months and a manual laborer in about four or five months after the operation. The patient will usually complain of some backache and slight twinges of sciatic pain for four to ten months postoperatively; this may be due to the long period of rest in bed and immobilization of the back, or to the presence of fascial adhesions and remaining inflammatory changes.

Causes of Failure of Relief by Spinal Fusion: 1. Presence of generalized arthritis of the spine.

2. Incorrect diagnosis; the presence of tumors of the cord, protruded disk, early spondylitis, etc.

3. The occurrence of pseudarthroses.

4. Improper operative procedure.

Pseudarthrosis has occurred in as many as 20 per cent of patients following spinal fusion. However, with the institution of a better technic and with extension of the fusion from the fifth lumbar vertebra to the sacrum, only about 6 per cent develop this complication.

This operation should not be performed upon all patients with an unstable fifth lumbar vertebra, but only in those (about 10 per cent) in whom the symptoms from the strain on the local muscles, ligaments and joints have not been relieved by non-operative measures.

Unstable Lumbosacral Joints.—This term includes any bony arrangement at the lumbosacral junction which is inadequate for support without causing undue stress or strain upon the adjacent ligaments and muscles. Included under this heading are: transitional vertebræ (sacralization and lumbarization), spondylolisthesis, abnormally acute lumbosacral angle, unstable lumbosacral facets, bony defects in the posterior elements of the lumbosacral vertebræ and displacement of the fifth lumbar vertebra on the sacrum.[62] If these patients do not respond to conservative therapy including an adequate period of rest, proper support to the back, heat, massage, etc., Haggart has advised that a fusion of this joint be performed in the younger patients (thirty to forty-five years of age) who are not emotionally unstable. Arthrodesis of the spine for low back pain should not be done in those emotionally unstable or in elderly patients. A modified Hibbs technic was employed, securing additional bone from the posterior superior iliac spine. The lumbosacral joint was fused in most cases; in patients with spondylolisthesis, the fourth lumbar vertebra was included. The patients remained in bed for six to eight weeks postoperatively and wore a Taylor back brace for an average of three months after becoming ambulatory. A preliminary period of muscle training was carried out preoperatively on all patients; this was also performed postoperatively.

LUMBAGO

Lumbago has been defined as a rheumatic affection (probably a fibrositis) of the muscles of the lumbar region. It may be either acute or chronic in nature.

The factors involved in the etiology of lumbago include: (1) localized *exposure to cold;* (2) *strain;* (3) subnormal physical condition of the patient; and (4) possibly focal infection. An occasional patient with lumbago improves promptly following removal of an infected focus (such as an abscessed tooth); this is the exception rather than the rule.

Symptoms: 1. Acute sudden agonizing *pain in the back.*

2. *Immobility of the lower spine* because of *pain on motion.*

3. Increased pain following a period of prolonged rest.

The typical patient is an office worker who returns to his home in the evening and decides to perform some amateur gardening. He becomes sweaty and continues to work in his wet clothes after sundown and after the air has become rather chilly. This individual is not used to prolonged exertion in the stooping position. The next morning on awakening he notices severe pain in the low back which is diagnosed lumbago. Cases of lumbago have begun not infrequently following sleeping in drafts with the back exposed.

This often begins on one side of the mid-line. Its onset is frequently sudden. The pain may be agonizing in intensity and often radiates around the body. This may begin in one of the erector spinæ muscles and spread to other portions; in some instances, it goes to the lower dorsal region. The lower spine is held rigid because of the severe pain on motion.

The patient stands with a slight flexion of the trunk and with his head thrown back. The spinal curve is obliterated. Although these patients are sometimes unable to stand because of pain, they are *fairly comfortable while lying in bed.* After long periods (ten to twelve hours) of rest in bed, pain is present on awakening; it is lessened if the patient moves about. The pain of these individuals may be exaggerated by alcohol.

Chronic lumbago results from repeated acute attacks. If lumbago is chronic from the beginning, degenerative joint disease of the spine should be suspected.[38]

Causes of Lumbago.—Many individuals consider lumbago to be a *myofascitis* (*i. e.*, a fibrositis) involving any fibrous tissue in the low back. Douthwaite states that the most acceptable explanation is a change in the fluids of the muscles and connective tissues resulting in irritation of the nerve endings. Such a change may be due to failure of local capillary circulation with a decrease in the local metabolism. Under these conditions, oxidation is retarded permitting sarcolactic acid to accumulate and producing nodules by a change in the physical state of the tissue fluid. The nodules may be felt, but on microscopic examination appear no different from ordinary muscular tissue.

In the *differential diagnosis* of lumbago, one must consider: Low back strain, degenerative joint disease, herniated intervertebral disks, tumors of the lower cord and cauda equina, metastatic carcinoma, perinephric abscess, tuberculosis of the spine, Paget's disease, gout and subacute bacterial endocarditis.

Degenerative joint disease should be considered in cases of chronic lumbago in which history of acute attacks is not present. Roentgen-ray examination will disclose the arthritic lesions. Patients with cauda equina tumors present an extended lumbar spine and a rigid lordosis with the head and shoulders thrown well back. This is not the posture of lumbago. Other signs of cauda equina tumors include loss of ankle and anal reflex or loss of perineal sensation.

Metastatic Carcinoma: In such lesions of the spine, the motion of turning over in bed may produce severe pain; no marked pain occurs from the same maneuver in lumbago. Neoplasms often produce bilateral pain radiating down the legs.

Perinephric Abscess: Pain is usually higher and tenderness is present in the costovertebral angle. Local swelling may not be present for some weeks. Other symptoms include an irregular septic type of fever, chills, sweats, leukocytosis and unilateral pain.

Tuberculosis of the Spine: Sudden acute pain is unlikely. Tuberculosis of the spine occurs most frequently in young patients; lumbago is seen most often in individuals of thirty-five years or more. The spines of the vertebræ are tender in tuberculous or neoplastic conditions but not in lumbago; the adjoining muscles may be tender in lumbago.

Paget's Disease (osteitis deformans): Symptoms are not common. Mild changes of osteitis deformans are seen in the pelvis and lower vertebræ after the age of sixty.

Gout: Gout has been accused of being a very occasional cause of lumbago and should not be overlooked as a possibility. It has been stated that the deposition of the highly insoluble crystals of sodium biurate may set up an acute inflammatory process. The diagnosis of gout is suggested by the presence of tophi, elevated blood uric acid, occurrence in males (in 98 per cent of the cases), involvement of the great toe, and history of recurrent attacks of acute pain with intervals of complete freedom from discomfort, etc.

Subacute Bacterial Endocarditis: An embolus may occasionally occur in the back resulting in acute sudden pain with muscular spasm and tenderness. Other signs of the disease are usually present; these include a cardiac murmur, loss of weight, fever, petechiæ, hematuria (microscopic), positive blood culture, etc.

Patients with lumbago may develop sciatica; this is almost always unilateral and may be a referred pain. It is possible that the rheumatic infection may spread downward into the intervertebral foramina to involve the sciatic nerve. If sciatica occurs, the duration of the pain will be several weeks rather than several days. If the patient with chronic lumbago develops faulty posture placing abnormal stress on certain joints, degenerative joint disease may develop at these sites.

In recurrent severe low back pain and sciatica, one must always suspect the possibility of a *herniated intervertebral disk.*

Treatment.—This consists mainly of *heat, exercise and analgesics.* Because of the pain, many patients will not exercise in the acute stage; however, if motion is continued, following local procaine injection if necessary, for twenty minutes in the mild attacks, the pain may disappear permanently. Heat may be used in the form of a hot water bottle, electric pad, or if desired (but unnecessary), diathermy or infra-red rays. Following the application of heat for at least thirty minutes morning and evening, deep massage should be employed (even though this seems uncomfortable to the patient).

These patients should be given an analgesic, such as aspirin and phenacetin, āā 0.3 gm. (gr. v) with or without *codeine* sulfate 15 to 30 mg. ($\frac{1}{4}$ or $\frac{1}{2}$ gr.) every three or four hours as needed; in some cases, morphine or dilaudid may be needed. Inhalations of amyl nitrite occasionally give temporary relief of this pain.

In some of these individuals marked relief is obtained by tight *adhesive strapping* of the low back; others are forced to lie still in bed to obtain relief. We have obtained *prompt disappearance of pain in many of these patients by injection of from 20 to 30 cc. of 1 per cent procaine subcutaneously into the painful areas.*

Kraus described the use of *surface anesthesia with ethyl chloride spray,* combined with active motion in the treatment of acute muscular spasms such as lumbago and low back pain and sciatica recurring after old injuries.[90] Ethyl chloride is sprayed on the skin area and the patient begins careful active motion of the part in the direction in which the motion had been painful and limited. As new painful areas develop with increase in motion,

these are also sprayed and active motion is continued. The treatment may last from ten to thirty minutes and should be done within the limits of pain. *Immediately following the treatment, camphor liniment is applied to the skin to avoid frostbite.* Patients with severe involvement required daily treatments for a week, following by treatments every other day for an additional week; in mild cases, one treatment sufficed. It was emphasized that the *ethyl chloride, alone, without active motion, did not achieve good results.*

In those individuals who develop sciatica, diathermy is not employed as early *sciatica is often aggravated by diathermy.* In chronic cases of lumbago, manipulations under anesthesia may be required, because of the presence of adhesions resulting in stiffness of the spine; these are followed by massage and movements. Recovery has been prolonged over a period of six months in severe cases. Many physicians in this country have never been able to understand the enthusiasm of the British for the diagnosis of myofascitis, and especially for the manipulative treatment.

TREATMENT OF BACKACHE: GYNECOLOGICAL

Long standing mechanical changes are believed to be a common cause of backache in multiparous women. The relationship between gynecological disorders and backache has been commented upon frequently.[1,35] Gynecological conditions causing low backache include mechanical changes incident to repeated pregnancies, disease of the tubes or ovaries, myoma uteri, retroposition of the uterus, tumors of the uterus (especially late stages of carcinoma of the cervix), cervicitis, etc.[5,58] Factors which may act as contributory causes to the mechanical changes present in these patients include flat feet, poor posture, hemorrhoids, constipation, fatigue, anxiety neurosis following economic or marital difficulties, and possibly, focal infection.[149]

The force of gravity is usually transmitted in a line passing posterior to the lumbar vertebral bodies and bisecting the sacro-iliac joints. Normally, the vertebral column presents four curves, anterior in the cervical and lumbar region and posterior in the thoracic and sacral portions. *During pregnancy, there occurs an increase in the normal lumbar lordosis,* resulting in compensatory changes in the dorsal and sacral spines. This is aided by the relaxation of the soft tissues accompanying pregnancy. Following the change in the lumbar curve, the pelvis, because of its attachment to the spine at the sacro-iliac joints, is tilted and rotated anteriorly, increasing the sacral angle (produced by the superior surface of the first sacral vertebra and the horizontal).

Due to the effect of the gravid uterus, the increased lumbar lordosis, the dorsal kyphosis and the anterior tilt of the pelvis, stretching and thinning of the abdominal muscles with shortening of the long muscles of the back occurs. The postpartum practice of lying upon the abdomen to aid return of the uterus to a normal position may increase the deformity. *With future pregnancies, obesity and fatigue, the abdominal muscles become still further stretched and thinned with a resultant loss of tone, and the traction of the posterior muscles becomes unopposed.* With lordosis of the lumbar spine, and spasm of the erector spinæ and atonic abdominal muscles, backache usually results. It is not determined whether this type of backache is due to pathology of the small joints, muscular spasm or strain of the ligaments.

If treated early, good response may be obtained by simple procedures including rest, heat, massage, postural exercises, support to the low back and to the weakened anterior abdominal muscles, and reduction in weight if obese. If untreated, various complications may occur such as strain of ligaments, degenerative joint disease, horizontal sacrum and migration of the nucleus pulposus.

A simple program of treatment for early cases includes:[94]

Rest in bed for at least several hours in the middle of each day. A non-sagging mattress is used or if this is not possible, wide boards are placed under the mattress so that no sag is permitted.

To correct the anteverted pelvic position the patient flexes the knees and clasps these toward the chest, while lying on the back; a hassock of proper height may be placed under the flexed lower extremities for comfort. A somewhat less satisfactory procedure is that of standing against the wall and straightening the back against the wall.

Additional exercises include: straight leg raising and the pelvic roll.

If the patient has severe pain, Buck's extension may prove useful; this may be removed for several hours a day if marked pain is present.

Support to the back must be used. This may be reinforced by semi-rigid steels posteriorly spanning the lordotic curve and exerting pressure on the buttocks. An additional inner belt fitting above the pubis anteriorly is of aid.

If pain is not relieved by these measures, additional methods which may be employed include:

(*a*) Hot wet stupes to the back at intervals (two or three times a day).

(*b*) In patients at the menopause, small doses of phenobarbital (such as 15 mg.—¼ grain three times a day), or the following prescription may be of value:

℞—NaBr . . . . . 16|0
Elixir phenobarbital . . . . . 90|0
Sig.: One teaspoonful three times a day after meals.

In other gynecological conditions which cause backache, treatment is instituted in the usual manner. If an infiltrating irremediable carcinoma of the cervix causes back pain, it is usually necessary to employ codeine sulfate 30 mg. (½ gr.) every three hours, or morphine sulfate 15 mg. (¼ gr.) every three hours; dilaudid 3 mg. ($\frac{1}{20}$ gr.) every three hours has been effective in relieving such pain. A combination of 0.6 gm. (10 gr.) of aspirin with 30 mg. (½ gr.) of codeine sulfate is more efficacious than strong narcotics in some cases; this may be repeated every three hours if necessary.

Although retroposition of the uterus may be a cause of back pain, one

must not promise that relief of this condition will produce a prompt cessation of the pain. In the particular case at hand, although a retroposition may be present, the back pain may be due to other conditions, such as faulty posture.

Raddin reported relief of backache in about 95 per cent of women by conservative tampon treatment; in about 15 per cent of cases, cervical cauterization was performed in badly eroded and infected cervices.[131]

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CHAPTER LVII

SCIATICA

SCIATICA is merely an inclusive term which has been used to describe a painful discomfort along the posterior aspect of the hip, thigh and leg.[30] The diagnosis and treatment of sciatica has baffled physicians since ancient times.[8] Inasmuch as the sciatic is the longest nerve in the body, it is of course subject to trauma and infection throughout its entire course.

Cotugno originated the term sciatica in 1764, which was exactly one hundred years before Lasègue described the sign which bears his name.

Sciatica is not a specific disease but merely designates pain in the lower limb approximately along the course of the sciatic nerve. It is no more a clinical entity than is headache. The actual pathologic changes present in many cases of sciatica are unknown.[8]

Low back pain and sciatica have been ascribed to various causes from year to year, including gynecological factors, sacro-iliac subluxation, postural abnormalities, congenital anomalies, fascitis and contractures, spastic pyriformis, protruded intervertebral discs and thickened ligamenta flava.[152]

At various times, numerous conditions have been named the outstanding cause of sciatica; these include foci of infection, arthritis of the hip, spasm of the pyriformis muscle, postural deformities, and more recently, herniated disks, thickened ligamenta flava, etc.

In our experience, the **more common clinical causes of sciatic pain** are: myofascitis (fibrositis) of the low back, often with spasm of the hamstring muscles;[83,149] lumbosacral or sacro-iliac strain with ligamentous tears at the musculotendinous junction; post-traumatic; herniated intervertebral disk; spondylolisthesis; congenitally defective fifth lumbar vertebra; metastatic malignancy; sciatic neuritis; cauda equina tumor and spondylitis.[24,62,81]

Sciatica may be due to functional decompensation of the supporting structures of the low back, mainly muscular and ligamentous; aggravating factors include trauma (which often precipitates the attack), *increase in weight, faulty posture* with exaggeration of the normal lumbosacral angle, *flat feet, knock knee, unsuitable occupation, degenerative changes in the intervertebral disks and anatomical variations in the lumbosacral region.* Excessive bulging of an intact disk or actual rupture with extrusion of part of the disk into the spinal canal are important causes of sciatic pain. In *bilateral sciatica, suspect pathology of the spinal cord, cauda equina, vertebræ or pelvis, tabes dorsalis or polyneuritis. Sciatica is very uncommon in childhood and adolescence because of strong muscular reserve support for the weak lumbosacral region.*[27]

Kuhns believes that the *most frequent cause of low back disability with sciatic radiation* is **injury to the ligaments, muscles and fascia** which support or invest the low back or pelvis.[87] He states that the cutaneous radiation

is most often a reflex phenomenon with referral of pain from deep structures to the cutaneous distribution of the same nerve root.

Lumbosacral sprains, anomalies and diseases of the vertebræ, and faulty body mechanics are contributing factors.

Kuhns reported *relief of pain referred along the sciatic nerve by conservative measures in 79 per cent of patients.* Low back disability due to ligamentous and muscular strains responds most often to conservative treatment.

Among 1000 patients examined because of sciatic pain and low back disability over a period of ten years, 576 were diagnosed primarily as muscular or ligamentous strains in the region of the lower lumbar spine and sacrum. An additional 325 patients were diagnosed as having a ligamentous strain together with disease or deformity of the vertebræ.

In most cases of low back pain, Kuhns believed that the lesion causing the pain was a *ligamentous tear* or a separation of fibers at the musculotendinous junction. Local tenderness over ligamentous structures helps to support this thought, as does the occasional occurrence of superficial ecchymoses and temporary relief which follows procaine injections into the most tender areas as well as the disappearance of pain and disability following immobilization and support with relaxation of the injured part of the back.

Causes of Sciatica

1. *Most common: Myofascitis* of the low back; lumbosacral and sacro-iliac sprains with *ligamentous tears* at the musculotendinous junction; herniated intervertebral disk.
2. *Vertebral causes: spondylitis, spondylolisthesis,* primary or metastatic *tumors* especially from the prostate and breast, congenitally *defective fifth lumbar vertebra* with destruction of the articular processes, unstable vertebral facets and other congenital abnormalities in the lumbosacral region[1,14,114] and osteomyelitis.
3. *Spinal cord involvement: tumors of the cauda equina,* arachnoiditis, pachymeningitis (tuberculous or syphilitic), tabes dorsalis, herpes zoster, hemorrhage around the cauda equina (and as above—herniated intervertebral disks[4,9,11,96,107,145] and possibly thickened and fibrotic ligamentum flavum);[21,136] *sciatic neuritis*[66] (with diabetes, vitamin deficiency, syphilis, pernicious anemia, etc.).
4. *Lesions of muscles and fascia: myositis* of the gluteal muscles; tightness of the tensor fascia lata or iliotibial band;[111,112] ischiogluteal bursitis; spasm or irritation of the pyriformis muscle from arthritis or sacroiliac joint disease;[49,51] faulty muscle balance from *poor posture,*[102,151] pes planus and obesity (and *myofascitis* of the low back soft tissue structures with production of pain in the sciatic distribution reflexly—mentioned above).
5. *Pelvic lesions: sacro-iliac subluxation;* infection, arthritis or tumors of the hip; sacro-iliac arthritis; femoral or inguinal hernia; tumors of the lumbosacral plexus (as retroperitoneal sarcoma, carcinoma of the ovary, prostate or uterus, etc.); infections or tumors of the prostate or female genital tract, following forceps delivery[23] and occasionally in the late stages of pregnancy (from pressure); psoas abscess.
6. *Hysteria, malingering* and *psychogenic* causes.
7. *Miscellaneous:* psoas abscess, tuberculosis, actinomycosis, undulant fever[3,42] and gout.

Studies Which Should Be Considered in Any Case of Sciatica

Complete history and *physical examination* (including search for infectious foci, *diabetes mellitus*, perineuritis from metallic poisoning, *pelvic* or abdominal *pathology*, etc.). Inquire carefully concerning history of strain of the back, especially from lifting, sudden twisting, etc. *Always do a rectal examination.*[63]

Complete blood count, urinalysis and blood Wassermann reaction.

Orthopedic examination.

Neurologic examination; test the Achilles reflex and sensation carefully.

Injection of procaine hydrochloride at site of trigger points of pain in the low back; if sciatic pain is a reflex phenomenon, it should be temporarily relieved by this procedure.

If necessary, lumbar puncture with estimation of pressure and examination of spinal fluid (cells, protein, Wassermann, colloidal gold curve. Protein should be determined on the first few cc. of fluid withdrawn).

Roentgen examination of the lower spine and pelvis, including anteroposterior, lateral and 45° angle views, as well as lateral views of the lumbosacral spine in positions of flexion and extension; injection of lipiodol, pantopaque or air if protrusion of a lumbar intervertebral disk is suspected and cannot be definitely diagnosed without this.

In suspected metastatic malignancy, determination of the *sedimentation rate* and *alkaline phosphatase* may be of value. Both of these are often increased in metastatic malignancy.

In patients with *recurrent intractable low back and sciatic pain,* with pain radiating down the back of the thigh and lower leg, not relieved by conservative measures, always suspect *herniation of a disk.*

Course of the Sciatic Nerve.—This is the *largest nerve in the body,* and is about ¾ inch wide. It arises from the anterior divisions of the fourth and fifth lumbar and the first three sacral nerve roots. The lumbosacral plexus lies inferior and medial to the sacro-iliac joint against the posterior surface of the pelvic cavity. The branches of the nerve plexus are closely interwoven with the surrounding blood-vessels. *The fifth lumbar nerve is the largest of these but passes through the smallest foramen* and may be involved in inflammation of the lumbosacral disk or articular facets.

The sciatic nerve leaves the pelvis through the great sacrosciatic foramen, usually between the pyriformis muscle (above) and the superior gemellus below.[13] In about 10 per cent of cases, the peroneal division passes through the former muscle.[48] It courses mid-way between the tuberosity of the ischium and the great trochanter and then down the back of the thigh as a flat ribbon. The division of the sciatic nerve into the common peroneal and the tibial branches may occur shortly after its exit from the pelvis, but more commonly about the middle of the thigh. These two branches are bound together by fibrous tissue as far as the upper end of the popliteal space. The sciatic nerve supplies the hamstrings and all of the muscles below the knee.

Lasègue's Sign and Its Significance.—This is pain in the back of the thigh or near the sacrosciatic notch on extension of the knee after the hip joint has been fully flexed.[90] It was formerly thought that the sciatic

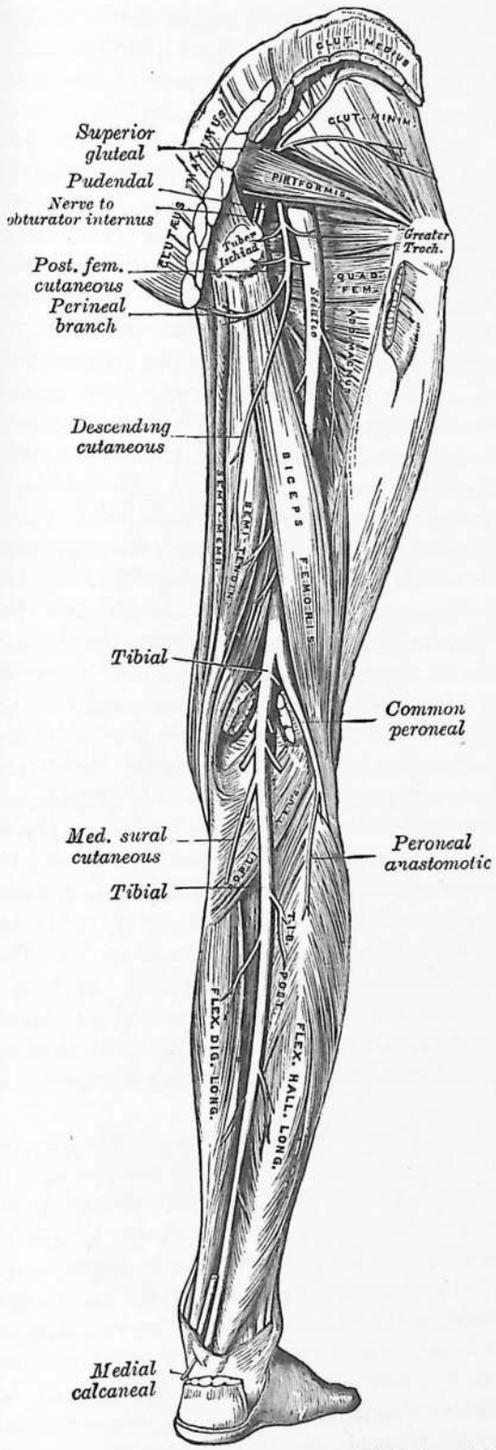

FIG. 312.—Nerves of the right lower extremity. Posterior view. (Gray.)

nerve was stretched during the performance of the Lasègue sign. Freiberg has shown that the nerve is not stretched in this maneuver until the hip has been flexed over 90 degrees; this amount of flexion is not obtained in the patient with a positive Lasègue sign.

Freiberg has demonstrated in cadavers that the Lasègue maneuver stretches the pyriformis muscle (through the biceps femoris muscle and its connection with the great sciatic ligament). The pyriformis muscle originates in part from this ligament and in part from the capsular ligament of the sacro-iliac joint. Spasm of the pyriformis muscle may cause direct involvement of the sciatic or peroneal nerves which often perforate this muscle. In other cases, the sciatic nerve lies against the bone of the great sciatic notch and therefore may be compressed by an inflamed or spastic pyriformis muscle. The gluteus maximus muscle lies directly over the pyriformis muscle. This latter is the one muscle which bridges the sacro-iliac joint. Freiberg believes that a positive Lasègue test indicates involvement of the pyriformis muscle.

Roentgen Findings on Patients With Sciatica vs. Controls.—Brav, Bruck and Fruchter compared the roentgenologic appearance in 70 cases of low back and sciatic pain with those in 35 normal controls with no symptoms.[18] In the two groups, *there was an equal number of normal roentgenograms, vertebral epiphysitis* and *osteochondritis, narrowing of the intervertebral disks* (except the fifth lumbar) and *vertical herniation of the nucleus pulposus. Lumbosacral anomalies were present in about half of both groups.* Lumbarization of the first sacral segment and sacralization of the fifth lumbar were seen almost entirely in females. Degenerative joint disease was twice as common in those with backaches as in the normal controls. Scoliosis and increased lumbar lordosis were twice as common and flattening of the lumbar spine six times as common in the backache cases. Lumbar abnormalities were four times as frequent and lumbosacral abnormalities were four and a half times as frequent in the group with backaches. Sacro-iliac changes were present in 6 of the backache cases and in none of the controls.

Willis has discussed the *anatomical variations of the low back in relation to sciatic pain.*[152] He found that the incidence of anomalies in painful backs was greater than in those free of symptoms or in the run of dissecting room cadavera.

Badgley discussed the *articular facets in relation to low back pain and sciatic radiation.*[5] He believed that 80 per cent of cases of low back pain with sciatic radiation are on the basis of referred pain, and not direct nerve irritation.

Spondylolisthesis.—*Sciatic pain occurs among only a small percentage of patients with spondylolisthesis.*[103] Meyerding has diagnosed a protruded disk in 15 of 25 cases in which spondylolisthesis was associated with sciatica. In these cases, he advocates surgical removal of the protruded disk and fusion of the last 3 lumbar vertebræ with the sacrum.

Sciatic Neuritis.—This is a neuritis with organic changes in the sciatic nerve.[99] It may be primary or secondary and may be due to deficiency of vitamin B_1, syphilis, involvement in a malignant growth, severe liver disease,[93] alcohol, lead, arsenic, exposure to cold and dampness, infection, diabetes mellitus, etc.

Alpers believes that true sciatic neuritis is not uncommon; others claim that it is rare.[71]

Alpers, Gaskill and Weiss analyzed 55 cases of sciatic neuritis. The patients were of middle age or early old age.[2] True sciatic neuritis was always characterized by *tenderness of the nerve trunks and usually also of the muscles of the calf and thigh.* The other characteristics may be found in any type of sciatica and include weakness or atrophy of leg muscles, decrease or absence of Achilles reflex, pain on stretching the sciatic nerve, sensory disturbances and inability to extend the leg.

Pain usually occurs along the course of the sciatic nerve or one of its branches. In most cases, this begins as a dull ache which becomes sharp and is often unbearable. The acute stage may persist for several weeks and may prevent sleep.

In the chronic stage, the pain is less sharp and may be localized in the buttock, behind the knee, on the lateral aspect of the leg or over the dorsum of the foot. *Motor symptoms are not common;* in the severe chronic cases, coldness and blueness of the foot, paresthesias or anesthesia, and muscular atrophy may occur below the knee.[65] The Achilles reflex may be decreased or absent. The whole course of the sciatic nerve may be tender. Pain may be increased by motion of the limb. *The loss of the Achilles reflex is an important sign of sciatic neuritis; the knee jerk is preserved.* (In anterior crural neuritis, the Achilles reflex is present but the knee jerk is lost.) The ankle jerk is mediated by the first two sacral radicles.

Scoliosis may occur either toward or away from the affected leg in chronic cases. Saline injections of the sciatic nerve often relieve cases with homolateral scoliosis; such injections are not of benefit if contralateral scoliosis is present. Some of the latter may be dramatically relieved by paravertebral alcoholic injection, especially at the fourth lumbar intervertebral foramen (about an inch lateral to the middle of the fourth lumbar spine); such injections should be made only if a spot of intense tenderness is present at the proposed site for injection. These injections should be made only by a trained orthopedist or neurosurgeon.

HERNIATION OF THE INTERVERTEBRAL DISKS

In the past ten years, interest in low back pain and sciatica has shifted from spondylolisthesis to herniation of the nucleus pulposus. The embryology and anatomy of the latter date back to the anatomic studies of von Luschka in 1858. The accurate and painstaking necropsy studies of the Dresden pathologist Schmorl have led to a correct clinical or pathologic approach to the subject of the intervertebral disk, a recent contribution.

The **intervertebral disk** consists of an outer dense fibrocartilage which is firmly attached to the adjacent vertebræ and forms a strong elastic ring, the annulus fibrosus; this acts as an envelope for the interior semi-

fluid mass of fibrogelatinous pulpy center, the nucleus pulposus. This firm fluid cushion or shock absorber is subject to the laws of fluids.[84] It is incompressible and confined to its normal position and shape by the strong elastic annulus fibrosus. The amount of compression of the intervertebral disk depends upon the strength and elasticity of the annulus fibrosus. Inasmuch as the nucleus pulposus is incompressible, its main function is to transmit the static and muscular forces from one vertebra to the next with the nucleus pulposus remaining as the axis of motion on which each vertebral body must work.

The *most common pathological change of the intervertebral disk is prolapse of the nucleus pulposus* into the spongiosa of the adjacent vertebra, leading to the formation of the so-called **Schmorl's nodules** in the vertebra. Schmorl concluded that small posterior prolapses of the nucleus pulposus beneath the posterior longitudinal ligament were common but that these seldom caused clinical symptoms. Schmorl believed that the rupture of the nucleus pulposus was the starting point for numerous changes in the spine and that the rupture was facilitated or caused by degenerative changes in the annulus fibrosus, mild trauma being an added factor.

In patients with intractable, recurrent sciatic pain, posterior protrusion of a lumbar intervertebral disk into the spinal canal is one of the more common mechanical derangements of the low back.

Kocher described clinically the first herniated disk in 1896.[52] Herniated intervertebral disks were seen by Virchow; Goldthwait described their pathology in 1911. *Mixter* and *Barr* correlated the pathology with the sciatic syndrome in 1934.

In 1911, *Goldthwait* reported a case of sciatica and paraplegia which was due to a posterior herniation of the intervertebral disk at the lumbosacral junction and suggested that these displacements might be the cause of many cases of "back strain, lumbago and sciatica."[39] Within the following fifteen years, neurosurgeons (*Elsberg, Stookey, Adson, Dandy* and *Bucy*) reported cases of "enchondroma" developing from the intervertebral disk, producing compression of the cord. Dandy noted that Goldthwait's remarkable anticipation of ruptured intervertebral disks never received the credit it deserves.[31,58] Mixter and Barr localized these lesions before a total block occurred. Reichert first substituted air for iodized oil as a contrast medium.[117,118] (Air shadows are not as striking as iodized oil shadows but in the hands of some skilled workers with excellent plates, they have proved satisfactory.) Semmes then reported 16 cases diagnosed without contrast medium, but solely by history and neurologic examinations.[126] Elsberg recorded the first example of "hypertrophy" of the ligamentum flavum in 1913.[35] Dockerty and Love found that degeneration and fibrosis are found in the thickened ligamentum flavum. Histologically, these authors found fraying, splitting and fibrillation of the normal elastic fibers, with varying degrees of fibrosis, hyaline changes in the blood-vessels and occasional calcification.

Mixter and Barr in 1934 established rupture of the disk as a clinical entity and a cause of intractable low back pain and sciatica; they reported a series

of 19 cases of rupture of the disk from the Massachusetts General Hospital.[106] Love and Walsh later (1938) reported larger series of cases in which the protruded disk was removed at operation. The "enchondromas" of the earlier reports were probably prolapsed nuclear material pressing on the cord, cauda equina, nerves or posterior longitudinal ligament.

Saunders and Inman and Oppenheimer have presented extensive reviews of the anatomy, physiology and pathology of the intervertebral disks.[116,125]

Structural changes in the intervertebral disks may be due to age and degeneration, developmental defects, trauma, infection and malignancy.[101]

There is some confusion in the use of the terms herniation and rupture of the disk; clinically, these are usually used interchangeably. However, it is preferable to speak of this lesion as a *herniated disk.* This may be ruptured or unruptured depending upon whether or not it has extruded itself entirely or in part through the posterior longitudinal spinal ligament.[76]

Hyndman, Steindler and Wolkin believed that *herniated disk was the most common cause of referred sciatic pain. The myofascial syndrome may be associated with or provoke a reflex sciatic radiation.*

The initial event in the development of a herniated disk is severe compression of the nucleus pulposus between the cartilaginous disk plates. The herniated disk with its root symptoms and signs is probably a by-product of a back injury and may require time to develop following the injury.

Variation in signs and symptoms may be related to the different positions of the extruded disk. When located centrally or slightly off center, the syndrome may simulate that of a cord tumor except that the spinal fluid pathway is not blocked. In the more laterally situated lesions, the cord escapes damage and the symptoms are those of motor and sensory root pressure.

Dandy has recently written that recurring attacks of low lumbar backache, with sciatica in the back of the leg (usually intensified by coughing and sneezing during the periods of acute pain) are almost pathognomonic of defective intervertebral disks.[34] He advised surgical intervention when the backaches were severe and frequent and reported that the operation was without risk and the results were permanent and excellent. He believed that one should diagnose herniated intervertebral disks in 95 per cent of the cases from the symptoms and signs alone and used roentgen examinations only to rule out spondylolisthesis and congenitally defective vertebræ (which produce similar symptoms). Narrowing of the intervertebral space is objective evidence of a defective disk. Spinal tumors, including tumors of the cauda equina may give similar symptoms but will show a xanthochromic spinal fluid in most instances.

Dandy believed that when symptoms recurred following surgery, there was either a recurrence of the original disk, another disk which was missed at operation or a tumor of the spinal cord or cauda equina. He advised removal of the affected disks as completely as possible with curets, baring

the bone, which permits narrowing of the joint; the cavity is later filled with connective tissue.

An inherent weakness in the lower lumbar spine may be the underlying cause of defective disks,[34] with the superimposed factor of trauma, such as a heavy lift, twist, bend of the back or a severe cough or sneeze. Dandy believed that the initial pain probably results from movement of the lateral facets and that the disk was affected secondarily.

In his experience, all spinal injections for diagnosis and localization are unnecessary as they are painful, do not localize the lesion in more than 25 per cent of cases and may cause serious after-effects. Dandy advises against fusion by spinal grafts on the grounds that it is not necessary.

Patients with protruded disks may range in age from sixteen to sixty-five years. Males are affected more than three times as often as females.

In 50 per cent of patients with protruded disks, *the disability occurs immediately following injury;* in an additional 30 per cent, it appears after a latent period. *The most frequent causes of injury include* lifting a heavy weight, falling from a height and a twisting strain.[68]

In 155 cases of protruded disks, 90 per cent occurred in the lumbar region, involving especially the fourth and fifth lumbar disks.[12] The lesion is usually lateral, directly beneath the articular facet and if large enough compresses the nerve root between the ruptured disk and the facet just before the exit of the nerve through the intervertebral foramen. Although the *lesion* may be so large as to occlude the dural sac, it *is usually a small, elastic, pea-sized nodule beneath the posterior longitudinal ligament;* when the ligament is incised, the nodule can usually be removed as a rolled-up completely detached bit of intervertebral disk tissue.

In a high percentage of cases of ruptured disks, there is an associated local thickening of the *ligamentum flavum.*

The *protrusions of the disk may occur either due to a sudden rupture of the annulus fibrosus* as a result of raising the internal disk pressure above the breaking point of the annulus fibrosus, *or as a result of a fissure in the annulus fibrosus from wear and tear* of ordinary use or degenerative change with a slow, gradual extrusion of disk tissue with resultant sciatica when the extruded mass becomes large enough to press upon a nerve root. *These latter patients may have recurring episodes of low back pain for years before the mass is large enough to precipitate an attack of sciatic pain.*

The *most common location of the lesion* is in the lower lumbar region; this area is subjected to the greatest mechanical stress in pushing or heavy lifting.

Barr reported in 1938 that more than 90 per cent of patients with sciatic pain recover spontaneously or after conservative treatment and do not require operative intervention.[10] It is extremely important that the general practitioner remember this and not be swept away by the current wave of hyperenthusiasm concerning herniated disks; only a small percentage of patients with low back and sciatica pain eventually require surgical intervention.

In *60 per cent* of Barr and Mixter's 155 cases of proved protruded disks,

the *pain was constant from onset until operation;* in 40 per cent there were relapses and remissions. Backache preceded the sciatica in 30 to 40 per cent. *In 80 per cent of cases, the referred pain was unilateral.* Almost all of the patients complained of pain in the posterior and lateral thigh. Ninety per cent had pain in the posterior and lateral calf; 20 per cent had pain in the lateral border, sole or dorsum of the foot or great toe.

The **physical signs** included:

1. *Pain on straight leg raising* in practically every case.
2. *Sciatic scoliosis* in 60 per cent.
3. Reversal of the usual lumbar curve and *limitation of motion of the lumbar spine* in 90 per cent.
4. *Local tenderness over the lumbar spinous processes* at the level of the lesion in 50 to 60 per cent.
5. *Radiating pain on coughing, sneezing, percussion of the lumbar spinous processes* or jugular compression in 40 per cent.
6. Atrophy of the buttock, thigh or calf in 50 per cent.
7. *Diminished or absent ankle jerk* in 70 per cent.
8. Sensory changes in the leg in 35 per cent (most often on the lateral calf, lateral border of the foot and fifth toe and posterior thigh).
9. Motor weakness of the dorsiflexors of the foot or gastrocnemius muscle in 15 per cent.
10. Sphincter disturbance in 5 per cent.

In one large series of cases, the protruded disk occurred at the third lumbar in 2 cases, at the fourth lumbar in 81 and at the fifth lumbar in 57 cases and at the first sacral in 1.

Herniated intervertebral disks probably account for a large proportion of the hitherto incurable low back complaints and sciaticas. The pain may appear spontaneously or after a sudden twist, heavy lift or an accident to the spine. The pain usually recurs in attacks but may be continuous and often becomes constant. The pain is usually intensified by movement of the spine.

Dandy believes that the diagnosis and localization of a protruded disk should be nearly absolute on the clinical history alone and that lumbar punctures and intraspinal injections of contrast media are unnecessary. He stresses the importance of the subjective history of **low backache plus sciatica, occurring in attacks,** *usually after a relatively trivial injury such as a lift, strain or bend.* In the acute stages the pain in the back and the sciatica are usually made *worse by sneezing or coughing.* Pressure on the nerve may produce sensory or motor loss; diminution or loss of the Achilles reflex is found in only half of the cases. In industrial work many of these patients are considered neurotic.

Dandy believes that the overwhelming majority of cases with recurring attacks of low back pain and sciatica in one or both legs are due to protruded disks. Patients with psychogenic low back pain have constant (not recurring) pain and fail to have exacerbations of pain on sneezing or coughing. In a small percentage of cases of protruded disks, the pain may

be constant and there may be no aggravation of the symptoms on sneezing or coughing.

Love and Walsh and Spurling and Grantham found that **96 per cent of all protruded disks were found at the fourth and fifth lumbar disks,** being fairly well divided between these two locations.[97,137]

Dandy believes that a **history of low midline lumbar backache with recurring** (not continuous) **pain down the back of one or both legs, and intensification of the pain on coughing or sneezing**—is the story of a protruded intervertebral disk. Of lesser importance are the diminished or absent Achilles reflexes in some cases and sensory or motor losses in the distribution of the fourth or fifth lumbar or first sacral nerve. It may be difficult to elicit a history of trauma as the injury is usually minor such as a sudden movement, wrench or a heavy lift. *Protruded disks are probably uncommon after severe back injuries.* The *recurring* pain is essential to the diagnosis and is of assistance in differentiating it from psychogenic backaches, which are usually persistent.

Severe, intractable, disabling sciatic pain is an outstanding symptom of herniated disk. There may be long remissions. **Interference with straight leg raising is an outstanding sign.**

Most neurosurgeons do not suggest operative intervention unless the severe sciatica has been present for months despite conservative care, including rest in bed or use of a plaster jacket or other apparatus to hold the lumbar portion of the spine in flexion.

The important statistical point is that *approximately 96 per cent of all protruded disks are in the lumbar region and that 98 per cent of lumbar disks are at the fourth and fifth disks.* Occasionally one encounters protruded disks at the second or third lumbar interspaces but in these patients there is usually pain in the front of the leg or diminished patellar reflex. Herniated disks may occur in the cervical region.[127] In the usual case with pain in the lower lumbar region and down the hip and back of one or both legs, the involved disk is nearly always the fourth or fifth lumbar. Dandy believes that a reduced or absent Achilles reflex points to involvement of the fifth disk but this may also be found with a lesion of the fourth disk. *With protrusion of the third lumbar disk, pain is noted in the front of the thigh instead of the back of the leg.*

Hyndman and his associates believe that paresthesia strongly indicates organic involvement of the nerve root in question as opposed to reflex pain and that this is a dependable symptom. They found some form of paresthesia in 85 per cent of their cases of protruded disks.

Narrowing of the fourth lumbar disk as seen by *x*-ray is of some diagnostic importance if the clinical picture is characteristic.

There is no positive method of differentiating between herniations of the fourth and fifth disks pre-operatively. Although a diminution in Achilles reflex suggests a lesion at the fourth disk, this may be present with lesions at the fifth disk; some workers (Spurling) believe that diminished Achilles reflex is evidence of involvement of the fifth rather than the fourth disk. The operative exposure, however, permits exploration of both disks after

removal of the ligamenta flava, if necessary. *Semmes has operated under local anesthesia; he reported that pressure with the forceps on the ligamentum flavum over the involved disk induced the original pain, whereas pressure on the other will not.*

Dandy has suggested a **test which may afford greater precision in localizing the disk** without exploring it. After a unilateral exposure of the spine has been made (with a cutaneous incision of about 4 inches), the fourth and fifth lamina are uncovered. A periosteal elevator is used to push the spines of the fourth and fifth spinous processes downward (caudally) in order to determine the mobility of each vertebra. *The protruded disk will usually be found where the greater movement is shown,* because the defective disk has weakened the spinal column locally, leading to mobility. This free play at the disk is responsible for the aggravation of the pain by sneezing or coughing. If the individual can stiffen his back before sneezing or coughing, the pain will be ameliorated. If the third disk is suspected, one can elicit this same free play of the spine there.

This spinal mobility test is not absolute as the degree of mobility is not always decisive. A disk may heal occasionally and solidify the vertebræ, leaving the protruded cartilage encapsulated attached to and under the nerve. In more than 85 per cent of cases, this spinal mobility test will correctly determine the site of the lesion.

Lesions which have been found in association with protruded disks in some cases include *thickening of the ligamentum flavum,* chronic adhesive arachnoiditis, hypermobility of the involved vertebræ and edema of the involved nerve roots.

Most workers no longer regard a thickened ligamentum flavum as an entity or responsible in itself for root pain. This ligament is an unyielding bulwark against which the herniated disk pinches an emerging root. It is very doubtful if it is responsible for root pain in the absence of a herniated disk.[154]

Ghormley, Bickel and Dickson discussed acute infectious lesions of the intervertebral disks, resulting in eventual thinning of the affected intervertebral space with later proliferation of new bone along the vertebral margins.[56]

One should remember that *sciatic pain of any origin may be accompanied by limitation of straight leg raising,* diminution or absence of an ankle jerk and limitation of motion of the lumbar spine.

The **differential diagnosis of protruded disk** must be made from such conditions as rheumatoid spondylitis, low back strain, fibrositis of the low back, primary or metastatic tumors, *tumors of the cauda equina, congenitally defective fifth lumbar vertebra* with destruction of the articular processes and *spondylolisthesis* and *metastatic carcinoma* to the spine.

Conditions simulating the clinical picture of protruded disk include intradural *neuromas,* infection about a root and thickening and fibrosis of the ligamentum flavum. This latter practically never causes sciatica in the presence of a normal intervertebral disk.

Roentgen examination will aid in differential diagnosis. Spinal cord

tumors produce sensory or motor disturbances. Carcinoma invading the low retroperitoneal spaces may simulate a protruded disk, but the pain in the former is not intermittent and is not aggravated by sneezing or coughing. Cauda equina tumor is now diagnosed rather seldom in comparison to the frequency of protruded disks.[122] The *sciatic nerve plays no rôle in the knee jerk or anal reflex.*[20] In **affections of the cauda equina,** one may obtain sphincteric disturbances, diminished or absent anal or patellar reflexes, anesthesia over the buttocks or above the knee, and atrophy of the gluteal and quadriceps extensor muscles.

Sciatic pain may continue for varying periods from a *tumor of the cauda equina;* the latter may exist without any sensory, motor or reflex changes. The early diagnosis of such neoplasms rests almost entirely with the laboratory, including lumbar puncture and protein determination of the spinal fluid and lipiodol, air or pantopaque myelography.[82]

Campbell stressed again the *importance of considering carcinomatous metastases in severe low back pain which is unrelieved by the ordinary analgesics,* and which is deep seated, gnawing and usually not related to movement; such pain becomes more intense and continuous in the later stages.[25]

Dandy has suggested that while the protruding portion of the disk causes sciatica, the necrotic interior of the disk causes backache.[34]

He has stressed the importance of what he terms **"concealed disks,"** which outnumber the protruding ones two to one. These former cannot be detected by injections of contrast media. In 80 per cent of his cases, two disks were involved, and occasionally a third.

At operation, this lesion is difficult to detect because it protrudes so slightly. However, there is always a tiny bulge of the intervertebral space with thickening of the spinal ligament.

The symptoms of these "concealed disks" are similar to those of protruding ones. Root pains result because they are adherent to the nerve (and not from mechanical pressure on the nerve).

Dandy believes that "concealed disks" explain many negative explorations, the disk bulging so slightly that it would not be disclosed by iodized oil or air injections into the spinal canal; it is found at operation only by a careful inspection beneath the dura. The spinal ligament over the involved disk is thicker than normal. The slight protrusion is softer to touch than a normal disk and gives a sense of fluctuation to the forceps. After the covering ligament has been incised, the forceps sink with almost no pressure into the intervertebral space; the forceps do not sink into a normal disk. When the forceps are withdrawn, a soft brownish mushy material clings to the blades; more of this material can be removed by a curet from the cavity in the depths of the disk.

Contrast Myelography.—Iodized oil may be required for the localization of tumors of the spinal canal from the third disk upward.[74]

The use of lipiodol for diagnostic and localization purposes should be reserved for those cases in which surgery is necessary because of the potential dangers associated with injection of lipiodol into the subarachnoid space.

Dandy discourages the use of spinal contrast media mainly because the results are often negative when an actual protruded disk is present. Dandy is opposed to the use of spinal injections of any kind in the diagnosis of protruded disks because these, in his opinion, are unnecessary, may be misleading, add to the patient's discomfort and lead to the permanent deposit of some of the iodized oil in the brain and spinal cord. *He believes that the diagnosis of a protruded disk can be made better by history and examination than by x-ray following injection of contrast media or air.* He does not even advocate the aid of a lumbar puncture to determine the presence of a block or to note the increase in spinal fluid protein which is usually present.

Intrathecal injection of iodized oil is followed in about 50 per cent of cases by mild fever (of short duration) and less often by headache. An increase in the spinal fluid cell count and protein may persist for several days. Fatal reactions are rare.

Permanent clinical ill effects from intrathecal injection of from 2 to 4 cc. of iodized oil are rare. The oil slowly becomes fixed in the sheaths of the spinal nerves and caudal sac, but usually produces only a mild arachnoiditis with fine adhesions between the dura and the arachnoid membrane. If the dura is opened at operation after the injection of iodized oil, the latter may become encysted by proliferation of the arachnoid.

In about one-third of individuals who have been given intrathecal injections of iodized oil, a varying number of small globules may be found within the intracranial cavity, but no symptoms have been noted due to this.

Nicols and Nosik have discussed myelography using thorium dioxide solution (*thorotrast*) as a contrast medium.[109,110] Apprehension regarding the use of thorotrast is based primarily on its radioactive properties. The thorotrast was removed by a subsequent forced cerebrospinal fluid drainage; the residual thorium dioxide in the bodies of these patients represents a radium equivalent of alpha and gamma radiation which is far below the minimal reported toxic level.

A new radiopaque substance, *pantopaque,* has been tried and seems more efficacious than iodized oil. Pantopaque has a greater tendency to remain intact as a single globule. Being less viscous than the older iodized oils, it can be withdrawn easily from the spinal subarachnoid space when *x*-ray studies are concluded. The oil is injected on the fluoroscopic table, and the needle is left in place; when the studies are completed, it is withdrawn under fluoroscopic control.

Friberg believed that herniations up to 8 by 8 by 8 mm. could lie lateral to the dura and nerve root without a demonstrable filling defect in the myelogram.

Small posterior protrusions of the disk have been found in 15 per cent of autopsies; these may be congenital and have no clinical significance.

Barr and Mixter believe that as yet we have no reliable method of clinically differentiating between lesions at the lumbosacral junction from

those one or two disks higher. They do not regard the presence or absence of an ankle jerk of differential diagnostic value.

Dandy believes that lumbar puncture is rarely necessary for diagnosis unless one suspects tumor of the cauda equina. If needed for diagnosis, *lumbar puncture should be done as low as possible,* preferably at the lumbosacral interspace. The first 2 cc. of fluid obtained should be examined separately for protein content. Careful manometric tests should be done to rule ou dynamic block and further fluid is withdrawn for cell count, Wassermann and colloidal gold tests. Although the protein content of the spinal fluid may be normal in a few proved cases of protruded disk, this averages more than 60 mg. per 100 cc. of fluid.

Surgery for herniated disks:

Complete relief from radiating pain has been obtained in 77 per cent of patients by operative intervention with removal of the protruded disk; an additional 18 per cent have only minor discomfort in the affected leg.

Operation is performed only in those who have had a trial of adequate conservative care without relief of symptoms *or in those with definite evidence of serious nerve-root pressure* (such as motor weakness, paralysis, loss of sphincter control, areas of anesthesia, etc).[134,144]

A partial laminectomy with removal of adjacent portions of two laminæ but without sacrificing the continuity of any whole lamina, is usually sufficient to permit exposure and removal of the lesion and of lipiodol if this has been left in the lumbar sac.

If an articular facet has been sacrificed, or if marked abnormal mobility is demonstrated at the site of the lesion, a fusion is performed in the involved area (usually the fourth lumbar to the first sacral vertebræ).

It is probable that *many patients with low back pain and sciatica with mild symptoms have a protruded disk and recover under conservative care.* In individuals whose symptoms are not relieved within a few weeks by rest in bed or by the application of a plaster jacket with the lumbar spine in slight flexion, the possibility of a protruded disk must be seriously considered and surgery may be required.

Manipulation of the spine should not be performed if a protruded disk is suspected, or paraplegia may result.

The **important surgical advances in herniated disks** include the substitution of a *unilateral approach* for the previously used bilateral removal of one or more laminas; further *reduction of the amount of bone removed* by cutting away only the edges of the laminas contiguous to the disk (thus maintaining the integrity of the laminal arch) and finally the *removal of many of the disks without sacrificing any bone,* as suggested by Love. After the ligamentum flavum is removed, there is usually ample room between the laminas. The disks can now usually be explored after removing only the ligamentum flavum with at most, only a bite of the lamina following Love's technic.

Dandy's *exposure is unilateral between the laminæ without removal of bone (Love's operation)* or *with the removal of a small bit of lamina if necessary when the interlaminal opening is too small.*

When Should Operative Treatment be Advised for Defective Disks?

1. Spontaneous cures of herniated disks are uncommon, as remissions may recur but repeated attacks follow slight strains as slipping, twisting, bending or lifting.
2. Protruded lumbar disks are probably much more common than is generally believed today; they are certainly *one of the most debilitating ailments* and one of the easiest to diagnose properly and cure permanently *with little or no risk in good operative hands.*
3. The great majority of patients with recurring, intractable low back ache and sciatica with aggravation of pain on coughing or sneezing probably have defective disks.
4. *Any patient whose intractable low back pain and sciatica* (not due to spondylolisthesis or congenitally defective fifth lumbar vertebra with destruction of the articular processes, or to tumors of the cauda equina) *does not respond to adequate conservative care including rest in bed on a firm mattress* with a pillow under the knees, heat to the low back several times a day, and analgesics, or whose pain (in the low back and sciatic distribution) recurs frequently when he is up and about despite the use of a support to the low back, *should be operated upon. The amount of pain which such a patient has will usually drive him to eagerly seek operative intervention.*
5. *Patients with persistent or intermittent severe low back pain and sciatic pain radiating down the back of the leg, aggravated by sneezing or coughing frequently have a defective lumbar disk.*[33]

The entire operation is now performed extradurally, removing only a small notch of bone from one side of the lamina and often removing the disk through the interlaminal space without removing any bone.[32]

Dandy advises removal of the entire necrotic content of the interior of the disk with curets; he considers this to be the best insurance against recurrences.

Dandy believes that the 5 per cent recurrences which have been reported following removal of protruded disks are due to failure to open the cavity in the body of the disk. He states that he has had no recurrences.

Barr and Mixter reported complete relief in 77 per cent of all patients who underwent operation for protruded disk, with or without spinal fusion.[12] Ninety-one per cent of those on whom the fusion operation was done in addition to the removal of the protruded disk obtained complete relief as contrasted to 69 per cent in those in which the disk was removed without the fusion operation.

Dandy believes that fusion operations are unnecessary and inadvisable in protruded disks if uncomplicated. He has found that firm union eventually results after the disk has healed so that fusion is unnecessary (as the latter requires immobilization in a cast for approximately two months—an unnecessary and severe hardship).

A congenitally defective fifth lumbar vertebra with or without spondylolisthesis may simulate or be associated with a protruded disk. In such patients, if no protruded or concealed disk is found, a fusion operation is performed. *Fusion operations must not be performed until a careful search*

has been made for an offending disk or the patient's symptoms may persist after grafting and it may be necessary to later remove the bone graft and subsequently remove the protruded disk. Roentgen examination will disclose both the spondylolisthesis and defective fifth lumbar vertebra so that the surgeon may be prepared for the necessity of a fusion operation.

Dandy insists that the protruding cartilage be removed and that the interior of the disk must be treated by breaking up the contents with the forceps and removing as much as can be pulled out of the necrotic interior of the disk; if this latter is not done, a considerable percentage of recurrences may follow.

In "concealed disks," Dandy advises the breaking up of the interior of the disks with forceps with the eventual cure depending upon the subsequent extrusion of the necrotic contents through the opening.[34] He has recently suggested that it was better to remove enough of the lining cartilage to bare the bone and permit granulations to fill the cavity. Since he introduced this procedure, uneventful recoveries have been the rule and it has not been necessary to reopen a wound. This applies to both concealed and protruding disks.

In all cases of protruded or concealed disks, the center and much of the periphery of the disks are destroyed. Dandy opens widely the ligament covering the disk, cuts the edges downward with a narrow sharp periosteal elevator and thoroughly curets as much as possible of the whole interior of the disk. After this débridement, many small and even large pieces of cartilage can be removed.

Patients should wear a back brace for three months after operative removal of a disk and should avoid heavy lifting and back strains during this period; thereafter, they may continue in any operation.

Ghormley, Love and Young reported on a *combined operation* in low back and sciatic pain in which a member of the neurosurgical staff explores the spinal canal for evidence of an intraspinal lesion and removes this if it is found and a member of the orthopedic surgical staff carries out bone grafting to bridge the affected parts.[55] They believe that *a majority of patients with protruded intervertebral disks are relieved by removal of the protrusion alone, but that bone graft or fusion operation should be done also in those with a well developed spondylolisthesis or in those with extensive lumbosacral arthritis and a static type of backache in addition to a protruded disk.*

Dangers of the operation for protruded disks include accidental cutting of the dura with annoying escape of fluid into the field; accidental pinching of the nerve root by the bone biting instrument; and tearing of the thin-walled venous plexus which lies anterior to the dural sac. The latter occurs very often when the dural sac and root are retracted in exposing the disk and profuse bleeding may occur.

Farrell and MacCracken believe that spinal fusion alone is as effective as laminectomy and fusion; they believe that the need for laminectomy in treating protruded intervertebral disks has never been established.[44]

Ecker believes, however, that questionable, slight or moderate protrusions of the intervertebral disks should be left alone and the overlying nerve root adequately decompressed.[40] Only a minority of the lesions found at operation are large. Ordinarily one finds a disk which bulges more or less. He believes that the pain produced by small and moderate protrusions of the disk can be adequately relieved by removing the structure against which the nerve root is being displaced (*i. e.*, the ligamentum flavum at its lateral extremity). In his hands, this procedure has apparently yielded results as satisfactory as removal of the protruded portions of such disks.

INJECTIONS IN THE TREATMENT OF LOW BACK PAIN AND SCIATICA

Various combinations of therapy have been preferred by those treating sciatica. In one series of 28 patients confined to bed, 85 per cent were completely relieved by double Buck's extension, diathermy, epidural injections, intravenous foreign protein and removal of foci of infection.[29] Epidural injections, diathermy and a low back belt relieved a similar number. Others have used roentgen therapy,[73,81] short wave diathermy,[119] bee venom,[98] heel lifts when a short leg was found,[70] injections of the sciatic nerve with quinine and urea hydrochloride,[142] fasciotomy,[72,113] section of the pyriformis muscle,[50] etc.

A simple *method of maintaining traction* of the lower extremities utilizes an inner tube of a tire and a canvas tape or trouser belt.[148] The tube is placed around the footboard of the bed with its lower border at the top of the mattress. The belt encircles the tube, perpendicular to its length. The feet of the patient are placed in the loop of belt, which comes above the ankles, calcaneal tubercles and the toes. With the foot of the bed elevated on blocks (or a chair) the patient pulls himself toward the head of the bed, thus applying tension on the inner tube.

Brav and Sigmond reviewed the **history of the local and regional injection treatment of low back and sciatic pain.**[17] This dates back to the earliest descriptions of *acupuncture* (about 300 A.D.). Huang Fu's classical treatise appeared describing the method of treating painful lesions by *insertion of long needles* into the subcutaneous or muscular structures. *Recently, it has been popular to inject the sciatic nerve trunk and its surrounding tissues with anesthetic solutions,* quinine and urea, chloroform, alcohol, osmic acid, procaine, eucupin, ammonium salts and pitcher plant extract. Following the original descriptions by Sicard and Cathelin in 1901, varying results have been reported following the epidural injections of procaine, saline, camphorated oil, magnesium sulfate, iodized oil, Ringer's solution, oxygen, antipyrine and liquid petrolatum. Warm water or 2 per cent phenol has been injected into the muscles of the back in cases of lumbago.

Steindler and Luck reported (in 1938) on the use of *procaine solution* as a diagnostic agent in low back pain and sciatica.[139]

Copeman[28] and Gorrell[59] note successful results in the treatment of lumbago (fibrositis of the low back) by injection of *nupercaine* into the tender areas. Walker, Teneff and Russetzki injected procaine periarticularly and intramuscularly.[123,141,146]

Kreuz reported relief of low back pain by injection of procaine directly into the pyriformis muscle while Gratz used insufflations of air into the fascial spaces with marked success. Forestier reported on injections of *iodized oil* into the local points of tenderness and Copeman then suggested that simple needling of tender areas of the lower back was sometimes sufficient to relieve local and radiating pain.[47]

Galland injected from 0.2 to 0.4 cc. of a 2 per cent solution of *iodine in alcohol* into the posterior aspect of the sacro-iliac joint to relieve discomfort in low back pain.[53] Harris and Verrall reported instant relief of low back pain and sciatica from injection of *alcohol* deeply into the tender area below and behind the fourth lumbar transverse process.[67]

Thomson[142] employed injections of ½ to 1 cc. of *thiosinamine* compound into the tender nodules of the back with good results in lumbago and Tolderlund advised the use of intramuscular injections of *formic acid.*[143] Others injected[100] a preparation containing formic acid, sulfur, iodine and camphor into the tender lumbar areas and still others claimed good results following the intramuscular injection of *distilled water* and *hypotonic saline* into the areas of referred pain.[135]

Haggart injected 150 cc. of 1 per cent procaine around the exit of the sciatic nerve from the notch (in sciatica of unknown origin).[63] Haldeman and Soto-Hall employed injections of 20 to 30 cc. of 1 per cent procaine into the posterior sacro-iliac ligaments in sacro-iliac lesions with immediate disappearance of pain.[64] Douthwaite remarked on the use of *acupuncture* in the treatment of sciatic pain[37] and Goulden[60] and Bring[19] used this method successfully along the course of the sciatic trunk.

Brav and Sigmond divided a group of 62 consecutive patients with low back pain and sciatica into three groups. The first group was treated by procaine injection, the second by saline injection and the third by insertion of the needle, with no injection. In all cases, the injections were given deep intramuscularly in the paravertebral region. Of those injected with procaine, 57 per cent obtained relief; *53 per cent of the group treated with saline were relieved as well as 59 per cent of those treated by the needle alone.*

Steindler advises treatment of the local low back lesions by immobilization by plaster cast.[138] In 100 consecutive cases in his experience in which low back pain was associated with sciatic radiation, the sciatic pain disappeared following procaine injection in 69 (in 84 per cent of which the end-results were good). *Steindler believes that the procaine injection will thus also predict with a fair degree of certainty which cases of sciatic pain will be relieved by proper orthopedic procedures.*

Sciatic radiation is a great obstacle in the accurate diagnosis of some cases of low back pain. Steindler believes that in many cases of low back pain, sciatic radiation is purely a reflex phenomenon without direct irritation of the sciatic nerve trunk or its branches.

In most instances, low back strain is due to minor injuries of the soft tissues. Steindler has noted several **trigger points** in the low back in which marked pain occurs on pressure. These include the: (1) lumbosacral syndrome, with a pressure point at the lumbosacral junction; (2) gluteal syndrome, with a pressure point at the insertion of the gluteus maximus; (3) the sacrospinalis syndrome (the most frequent type) with a pressure point at the posterior superior or inferior iliac spine; (4) the transversosacral syndrome, with the pressure point at the transversosacral articulation in cases of sacralization of the fifth lumbar vertebra; (5) the tensor fascia lata syndrome with a tender point at the lateral border of the fascia and iliotibial band, and (6) the myofascial syndrome with vague tenderness along the sacrospinalis muscle and its fascial sheath.

When one irritates the trigger points of these areas, there is an immediate exaggeration of the sciatic radiation. If the trigger point is injected with 3 cc. of 1 per cent procaine, the sciatic radiation disappears rapidly. Steindler believes that this demonstrates that sciatic pain is very often a secondary reflex phenomenon from traumatic or mechanical lesions arising in the muscles and ligaments.

In 1926, *Carnett advocated procaine injection of the posterior divisions of the intercostal and first lumbar nerves for relief of pain and tenderness along the peripheral distribution of these nerves.*[26] Labat and Greene reported relief of pain by injection of weak dilutions of 95 per cent alcohol in 1 per cent procaine into tender areas of the back (determined by means of a percussion hammer).[88] Judovich and Bates later observed that in many instances, low back pain was due to irritation of the posterior divisions of the twelfth dorsal and first lumbar nerves, with pain and tenderness along the cutaneous distribution of these nerves in the upper gluteal area and anteriorly, in the groin and upper inner thigh. They noted many patients who were thought to be suffering from lumbosacral and sacro-iliac strain but were actually instances of *twelfth dorsal and first lumbar neuralgias* with referred pain and tenderness along the peripheral distribution of these nerves.[79,80] Temporary relief was obtained first by injection of a procaine solution around the points of emergence of the nerves from their foramina; permanent relief was obtained later by a combination of an *injection of procaine and an aqueous extract of the pitcher plant.* Others later reported on the improvement following injections of 1 per cent eucupin in oil into an area in the low back midway between the fifth lumbar spinous process and the iliac crest for low back and sciatic pain.

SCIATIC PAIN OF REFLEX ORIGIN

Sciatic pain of reflex origin means that the *pain impulse travels from a strained soft tissue structure of the back to the spinal cord and after making synaptic contact with other sensory units at different levels and with different anatomic distribution, results in a painful sensation in the territory of the second unit.* Breaking of this reflex arc at the point of the primary local irritation (the soft tissue in the back) will immediately abolish the radiating sciatic pain.

Many of these patients with reflex sciatic pain show *trigger points, i. e.,* small localized spots which cause pain on pressure (most commonly at the lumbosacral junction, the posterior superior iliac spine, the gluteal

insertion at the outer posterior rim of the ilium and the posterior border of the tensor fasciæ). These structures, as well as all ligaments, tendons and aponeuroses of the lower back derive sensory innervation from the posterior primary division of the lower lumbar and sacral nerves. The sciatic trunk is composed of the last two lumbar and first three sacral roots from the anterior primary divisions of the spinal nerves.

Stimulation of the circumscribed painful area in the back will produce or intensify the sciatic radiation of pain in those of reflex character, while infiltration of the area with 5 to 10 cc. of 1 per cent procaine abolishes the sciatic radiation at once. Thus, it is evident that some patients with sciatic radiation have their pain not from mechanical irritation of the posterior roots, but from reflex routes. *In these patients with reflex sciatic pain, there are no paresthesias, anesthesias or deep reflex changes. The pain does not reach to the sole or heel in these reflex cases.* **The presence of areas of anesthesia, loss of reflexes in the leg, paresthesias or extension of the radiating pain to the outer side of the foot or heel would rule out the presence of a reflex radiating sciatic pain.**

Treatment of these patients with myofascitis and a radiating sciatic pain consists of rest in bed on a firm mattress under which fracture boards have been placed, hot packs or applications and massage of the contracted back muscles and analgesics (such as aspirin and phenacetin, 0.3 gm.—gr. v of each three times a day and at bedtime, together with small doses of codeine phosphate 15 mg.—¼ gr.—every three to five hours, if required). Some of these patients are improved by temporarily flexing the hip slightly with a pillow under the knee, and with traction in the direction corresponding to the position the patient assumed in standing. Procaine injections into the trigger spots should be done at once to abolish radiating pain.[140]

The patient must remain in bed until the spontaneous sciatic pain has disappeared and until he can roll over in bed without pain. Following this, he may be up on crutches after a proper support has been obtained for the back.

Low Back Pain With Sciatic Radiation:

Reflex *pain from myofascial trauma may be differentiated from a herniated disk* by the absence (in the former) of paresthesias, anesthesia, reflex changes and lack of extension of pain to the foot or toes. The sciatic radiation in reflex pain from myofascial trauma may be abolished by injecting procaine into the appropriate trigger points (an extremely useful test).

Other causes: tumors of the cauda equina, spondylolisthesis, defective fifth lumbar vertebra with destruction of the articular processes, metastatic malignancy, etc.

In patients with lumbosacral strain or in recurrent sacro-spinalis strain, a brace may be prescribed, reaching to just below the shoulder blades and having a pelvic and supra-iliac cross band and a snug apron in front. If there is a complicating arthritis of the spine, the brace should reach higher

and is usually provided with shoulder straps.[76] Hyndman and his associates prescribed a corset with a sacro-iliac pad which extends downward to cover the gluteal region and the upper part of the thigh in patients with more acute sacro-spinalis sprains, gluteal sprains or sprain of the fascia lata.

Johnstone deplored the tendency for industrial surgeons to lump cases of backache among working men under the term "**industrial backache.**"[77] He found that backache was not related to injury in 70 to 80 per cent of these cases.

Acute backache with acute onset of pain after mild or major trauma has been presumed to result from *subluxation of one of the intervertebral joints or to an incomplete tear of an adhesion in periarticular tissues* (if fractures have been ruled out).[95] However, there is little evidence that such adhesions exist and many individuals doubt the occurrence of such subluxations. However, *spinal manipulations* are occasionally of value in these cases;[86] such manipulations can only displace articular surfaces or break down adhesions. Little reported on the results of 200 such patients treated by manipulations with relief of disability in four or five days in the average case. Miltner used manipulation without anesthesia in individuals when backache was caused by subluxation or when there was impingement and overriding of articular facets; chronic myofibrositis; adhesions and mild stiffness of the spine from a chronic sprain or a burned out arthritis.[105] Jostes also recommended manipulation in such cases.[78]

Temporary Treatment of Sciatica Until Etiology is Determined[1]

1. *Rest in bed* on a *firm mattress* with fracture boards under this.
2. Partial flexion of the leg by placing a *pillow under the knee* and a small firm pillow under the lumbar hollow.[46]
3. *Heat* to the gluteal region and low back as hot fomentations, infrared or diathermy.[153] If heat increases the pain, it should be discontinued. Heat should be applied for thirty minutes three or four times a day, if possible. Avoid massage during the acute stage.
4. Large doses of *salicylates* (1 gm. every 4 hours) may be needed to relieve the discomfort. Intravenous injection of 1 gm. of sodium salicylate two or three times a day often produces surprising relief. In some cases, one must employ codeine phosphate 15 mg. (¼ gr.) every three or four hours in addition to the salicylates. If marked nervousness is present, phenobarbital may be given—15 to 30 mg. (¼ to ½ gr.) three or four times per day. Occasionally, it may be necessary to use morphine temporarily for relief. Liniments or analgesic ointments (methyl salicylate) may be applied locally.
5. *Most physicians no longer employ extension of the leg by weights* in an effort to relieve muscle spasm as this is unphysiologic—stretching the leg muscles and pulling on the sciatic nerve, instead of relaxing it.
6. Any evident definite *focus of infection* (particularly apical infection) should be *treated or removed* (prostate, teeth, sinuses, tonsils).
7. In *stubborn cases, epidural injections* of normal saline or procaine *or perineural injections* of procaine at the sciatic foramen may be used with success.

Temporary Treatment of Sciatica Until Etiology is Determined
(Continued)

8. If sciatica is caused by a myofascitis of the low back, *injection of procaine into the trigger points* should relieve the sciatic pain immediately. These patients should then be given adequate heat, massage and graded exercises.
9. In cases with severe ligamentous injury to the low back, one may apply a plaster back shell or plaster spica for immobilization.
10. Small doses (100 to 200 r) of *deep roentgen therapy* to the lumbosacral region and distribution of the sciatic nerve have proved of benefit in some of these cases. Treatments may be given every five days and definite improvement should be seen by the fourth session.
11. The *rapid sinusoidal current* may give prompt relief of sciatic pain. Weiss advises a sinusoidal current of 110 voltage with 7200 interruptions per minute.[150] One electrode is placed on the posterior aspect of the knee or ankle and the other on the sacrosciatic notch. The current is gradually increased until tonic spasm of the muscles occurs; this is maintained for one minute and is then gradually decreased.
12. In individuals not responding to conservative care, followed by a period of support to the back, heat, graded exercises, etc., if low back pain and sciatica are severe, recurrent and intermittent, one should *suspect a herniated disk.* Removal of the disk is necessary for relief of intractable pain unless conservative measures afford relief.
13. When the acute pain has subsided, *massage and postural exercises* are begun, in bed, and the patient is taught how to flatten his lumbar spine, to breathe—correctly, etc. Before allowing the patient up, he should be fitted with a *low back support* (such as a well fitted corset, a spinal brace of the Knight or Goldthwait design or a plaster support).[69] He must be taught how to maintain his *improved posture. Heel lifts may be required if a short leg is found.*

Technic of Epidural Injections.—Materials needed:[45,54] 100 cc. of warm sterile physiologic saline containing 1 per cent procaine; 2 lumbar puncture needles; a 20 or 50 cc. syringe; a 2 cc. syringe; a 10 cc. syringe for evipal (if this form of anesthesia is used); an ampoule of epinephrine for emergency use in case of shock (practically never necessary).[129–131]

Object of the injection: introduction of fluid into the epidural space through the sacral hiatus to stretch the nerve roots.[43,147]

Site of Injection:[7,104] The sacrococcygeal hiatus (foramen) which is at the lower extremity of the sacral canal, approximately 2 inches cephalad from the tip of the coccyx (which can be located with the finger). This foramen is in the mid-line and is bounded above by the concave lower border of the sacrum and on the sides by the two sacral cornua; injection is made mid-way between these palpable cornua. As a check, the sacral spines may be palpated with the index finger and the triangular depression felt at the lower end of the sacrum marks the position of the sacral hiatus.

Local anesthesia with procaine may be used; Slot prefers general anesthesia with evipal given intravenously (which produces unconsciousness within a few seconds). The patient lies on the affected side, with the thighs flexed and the buttocks perpendicular to the edge of the bed (as for lumbar puncture).

Surgical Procedures to Be Resorted To, If Necessary

In severe cases some orthopedists advise a well fitted plaster spica with traction of about 10 pounds to the affected leg. It may be advisable to use a plaster cast from the axilla to the trochanters so that the patient may be ambulatory.
Epidural injections.[94,128,129]
Injections into or around the sciatic nerve.[6]
Division of the fascia lata.[133]
Section of the pyriformis muscle.
Manipulation of the low back.[38,92]
Subarachnoid injection of absolute alcohol (use with great caution!).[57]
Operative removal of a herniated intervertebral disk.
Lumbosacral fusion.

The lumbar puncture needle is passed through the skin at an angle of 70 degrees to it. When the sacrococcygeal ligament has been penetrated completely (by sense of feel), the direction of the needle is changed to one almost upward toward the head of the patient and parallel to the general direction of the canal (and the spine). The needle then enters the epidural space.

Slight suction is made with the syringe; no cerebrospinal fluid should be obtained as the needle is outside of the spinal theca (the dura terminates at about the second or third piece of the sacrum).

Inject slowly from 50 to 70 cc. of physiologic saline; no great resistance should be encountered.[16] Withdraw the needle and seal the puncture site on the skin with collodion.

If it is advisable to use **manipulation,** this may be given now (by an experienced operator) as follows:

(*a*) With the patient on his abdomen, extend the spine with the operator's knee on the affected region, and pull backwards with the two hands placed in front of the body (pushing forward at the same time with the knee).

(*b*) With the patient on his back, flex the thighs alternately with the opposite thigh held flat by an assistant.

(*c*) Rotate the spine and mobilize the sacro-iliac joint by fixing the iliac bone and forcibly rotating the opposite side; this should be done on both sides.

(*d*) Knead the muscles firmly in the gluteal and lumbosacral regions.

After-care: (*a*) Remain in *bed* twenty-four hours; very little postoperative pain is usually noted and the pain of the sciatica has disappeared in most cases.

(*b*) Apply *radiant heat* and *massage* to the back the following day.

(*c*) In some, give a course of *exercises designed especially for the extensor muscles of the back* as follows:

With the patient lying on the back, contract the gluteal muscles and pull the buttocks together.

In the same position, lift alternately one leg and then the other at least 12 inches above the bed with the knee fully extended.

Lift the head and shoulders from the bed without using the hands or arms.

The above exercises may later be performed against resistance.

When the patient becomes ambulatory, he should frequently contract the gluteal muscles so that this becomes more or less automatic.

The epidural injection should be performed only if no cause is found for the sciatica, and if the case is refractory to the usual conservative measures.

Repetition of Injections: If necessary, these injections may be repeated at any time.

Subarachnoid (Intraspinal) Alcoholic Injections for Sciatica.[61,124]—*Purpose of the Injection:* Relief of intractable pain by subarachnoid injection of 95 per cent or absolute alcohol, by blocking a certain number of the sensory tracts while the motor roots are unaffected, being protected against the action of the alcohol because they are myelinated.[36,115]

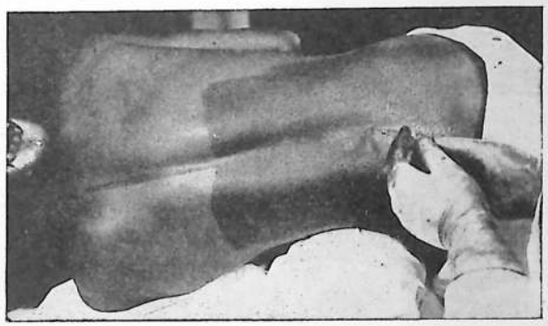

FIG. 313.—**Intraspinal alcoholic injection.** Position of patient at time of injection Needle in the fifth interspace is at the highest level of the cord; trunk flexed laterally over sand bag. (Goff, courtesy of Am. Jour. Surg.)

Indications: Intractable sciatic pain (as in root pressure from degenerative joint disease about the foramina involving the lumbar and sacral roots; postural changes with anomalies of the vertebra with similar pain, unrelieved by other methods; prolonged pain from severe chronic low back strain in an occasional case, etc.). This procedure may possibly replace chordotomy, rhizotomy and other operations which have been performed for intractable pain (including that due to malignancy in the pelvis and abdomen).

Technic: Place the patient on his non-painful side, with the thighs and knees flexed; introduce a lumbar puncture needle into the subarachnoid space at the level of the root most involved (usually the fifth interspace in the lumbar region). This is done as a routine lumbar puncture.

Place a sand bag under the loin and hip to raise this part higher than any other part of the spinal canal; the head is kept flat on the side, without a pillow.

During a period of at least three minutes, allow from 0.25 to 1 cc. (4 to 16 minims) of *sterile* 95 per cent alcohol to enter the canal *drop by drop*.

The specific gravity of the alcohol is about 0.816 and that of the spinal fluid −1.007 so that the alcohol rises at once and forms a puddle around the desired root, directly above the point of the needle. The patient is pronated slightly forward so that the posterior roots (usually not myelinated) are somewhat higher than the myelinated motor roots. Because the sensory and motor roots are so close together as they approach the foramina, the alcohol also bathes the motor fibers to some extent.

Withdraw the needle, but keep the patient in position for thirty minutes, being watched by an attendant. He is then kept flat on his abdomen for two hours with the foot of the bed elevated from 4 to 8 inches, and remains in bed at least twenty-four hours.

If necessary, a sedative may be given for headache and pain.

If the patient is not relieved of pain and has no headache, the procedure may be repeated after six days one or two segments above or below the previous site.

During the period of relief from pain (which may last eight months), institute corrective exercises, muscle building, etc., to produce a permanent cure.

Usual Results: Immediate relief of pain.

Sensation of warmth and numbness in the whole extremity, which may feel asleep for several days but which can always be moved.

Occasionally, pain returns for a few days, but disappears within two weeks after the operation.

Urinary retention may necessitate catheterization for several days.

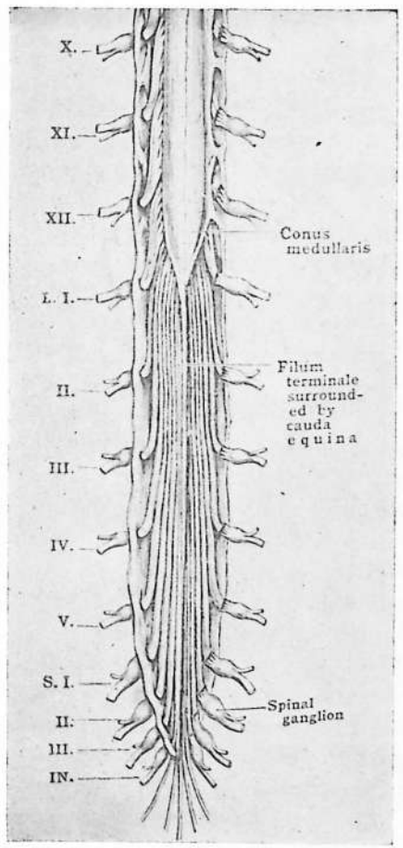

FIG. 314.—Position of nerve roots in cauda equina (with dura split open). (Goff, courtesy of Am. Jour. Surg.)

Caution: Use sterile alcohol.

Never insert the needle above the first thoracic vertebra.

Never inject more than ½ cc. of alcohol between the first and second thoracic vertebra.

This procedure should be done only by one experienced in this technic.

TECHNIC OF PERINEURAL PROCAINE INJECTION OF THE SCIATIC NERVE

Haggart has described a method of combined perineural injection of the sciatic nerve and manipulation of the low back which is applicable to

those cases of sciatica with unknown etiology or as a method of producing immediate cessation of pain until a clinical cause has been found.[68] In 75 of these patients, he obtained immediate relief. In 38 of the cases it was possible to make a definite diagnosis at a later date.

Object of the Injection: To infiltrate the perineural area of the sciatic nerve as it emerges from the pelvis through the greater sciatic foramen and to inject, in addition, the adjacent fascia and substance of the pyriformis muscle.

Site of the Injection is the intersection of two lines: (*a*) a line perpendicular to the spine beginning at the level of the apex of the intergluteal fold (this is to be 3 inches long in males and 3½ inches in females); (*b*) a perpendicular to this line running from the ischial tuberosity. The point thus obtained may be marked by a dot of gentian violet. As a further precaution and check, this point must not be lateral to (outside of) a line drawn between the spinous process of the fifth lumbar vertebra and the mid-lateral aspect of the greater trochanter.

Method of Injection: The buttock is painted with tincture of iodine or other antiseptic and the skin at the site of injection is anesthetized with 1 per cent procaine. A 20 gage 6 inch needle is then inserted anteromedially at an angle of 45 degrees with the surface of the skin. When the fascia beneath the gluteus maximus is met, additional procaine may be necessary. The needle is carried farther inward and shortly thereafter, the correct location of the needle will be shown by a momentary lightning pain in the course of the sciatic nerve (down the posterior mid-line of the leg). The needle is now withdrawn ½ cm. Two additional needles are passed similarly, with the first needle as the center of a small circle. These course downward and inward until the pyriformis muscle is encountered (recognized by its resistance in comparison to the gluteal muscle). All 3 needles enter the greater sciatic foramen.

One per cent procaine containing 2 drops of 1 to 1000 adrenalin per ounce is used. Fifty cc. is injected immediately around the sciatic nerve and an additional 50 cc. is injected through each of the remaining two needles. If the sciatic pain is not diminished or stopped by this procedure, the needles are withdrawn and reinserted.

Caution: An occasional patient is sensitive to procaine. Patients must be questioned concerning previous procaine sensitivity. If such sensitivity is suspected, 15 cc. of 1 per cent procaine is injected about the nerve and the remainder of the injection is performed with physiologic saline.

Postoperative Care: Rest in bed. If the sciatic pain is of long duration, apply skin traction to the leg by the Russell method. An initial pull of 25 to 50 pounds is used for the first thirty-six hours; this is then reduced to a maintenance traction of 10 to 15 pounds. Traction is applied for an average of two weeks.

Manipulation of the back under anesthesia (intravenous pentothal) may also prove of benefit in these cases.[75]

Additional therapy consists of: exercises to develop the low back muscles and those of the gluteal group, postural training when ambulatory, hot

fomentations and infra-red light to the low back, the buttocks and the upper thigh.

Saline Injections of the Sciatic Nerve.[89]—*Indications:* Persistence of sciatic pain for four weeks or more, despite conservative treatment, especially when there is: (*a*) tenderness to pressure over the sciatic notch or between the tuberosity of the ischium and the lesser trochanter; (*b*) aggravation of pain by flexion of the hip with the knee straight; (*c*) a history of recent injury such as a fall on the buttock.

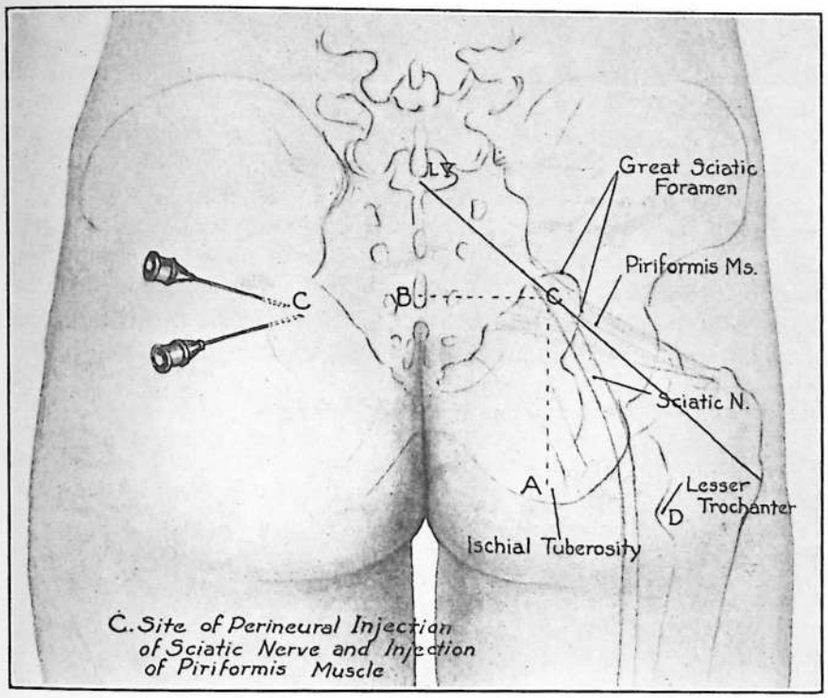

FIG. 315.—Technic of **perineural injection of sciatic nerve** as it emerges through the greater sciatic foramen. The needle is introduced at the intersection of the dotted lines (*BC* and *AC*) and the line from the femoral trochanter to the fifth lumbar spinous process. (Haggart, courtesy of Jour. Bone and Joint Surg.)

Technic: Outline on the skin the course of the sciatic nerve: from the sciatic notch downward and outward between the lesser trochanter and the tuberosity of the ischium.

Sciatic Notch: Approximately 4 inches lateral to the mid-line at the upper end of the intergluteal cleft and the same distance above the tuberosity of the ischium.

Infiltrate the skin with 2 per cent procaine at a point 1½ inches below the sciatic notch.

Insert a lumbar puncture needle attached to a 2 cc. syringe at this point and pass the needle to the notch. This is usually somewhat less than 3 inches from the surface.

When the nerve is found, an electric shock will be felt down the leg into the foot.

One cc. of sterile physiologic saline solution is injected; if the needle is correctly placed this causes pain in the foot. If no sensation is experienced in the foot the needle must be readjusted.

Slowly inject 2 cc. of 2 per cent procaine into the nerve. *Do not use alcohol* or paralysis of muscles below the knee and foot drop will result.

Following the procaine injection, pain ceases and the foot becomes numb.

Now use a 50 cc. syringe and slowly inject 50 to 80 cc. of sterile physiological saline into the nerve.

In most cases, the leg is without pain following the injection.

Postoperative Reaction and Care: Restlessness and slight fever may persist for twenty-four hours.

The patient should remain in bed for at least twelve hours after the injection and preferably for several days.

Repeat the injection (in most cases) after an interval of five days; the second injection is best performed at the level of the ischiac tuberosity.

Rationale of the Injection: Swelling of the nerve tends to rupture adhesions which may have formed.

These injections will be used much less frequently if the possibility of a herniated disk is borne in mind in all cases of recurring sciatica.

MERALGIA PARESTHETICA

This syndrome consists of *numbness, paresthesias and burning pain over the middle and lower portions of the anterior and lateral aspects of the thigh.*[91,121] It is caused by irritation of the lateral femoral cutaneous nerve within the spinal canal or intervertebral foramina, within the abdomen or pelvis, or in the thigh.[15,85] Irritation may be caused by:[41] multiple sclerosis, radiculitis, spinal anesthesia, lesions of the cecum or appendix,[108] hypertrophic spondylitis,[120] pregnancy, trusses, belts and corsets, pressure from the fascia lata, etc.

The syndrome *usually begins in middle age*, although it has been reported in children of fifteen. It is about three times as common in males as in females. Affected individuals are usually obese. Symptoms are most often unilateral. In some cases, symptoms may persist for more than thirty years, if untreated.

The condition is *usually self-limited* but in some cases it may be necessary to release pressure upon the nerve by slitting the fibrous tunnel in the fascia lata through which the nerve passes, or to resect or sever the affected nerve.

Additional therapeutic pointers include correction of pressure from a belt or truss, reduction of weight (if obese), warm baths and light massage.

ANTERIOR CRURAL NEURITIS

This is often misdiagnosed sciatic neuritis.

Anterior Crural Nerve: Origin: third and fourth lumbar nerves.

Distribution: to the muscles of the anterior aspect of the thigh (except the tensor fasciæ femoris), to the skin on the inner side of the leg as far as the foot, the front of the thigh and to the knee joint.

This may be injured by a wound, pressure from pelvic masses, or growths from the ilium or lumbar vertebræ. Neuritis may also follow influenza, vitamin B_1 deficiency, etc.

Symptoms: Pains down the front of the thigh and leg.

Absent knee jerk (with preservation of the Achilles reflex).

If the nerve is paralyzed, there is loss of extension of the knee (from loss of power of the quadriceps femoris). Motor or sensory branches of the nerve may be injured separately or together.

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CHAPTER LVIII

TUMORS OF JOINTS AND TENDON SHEATHS

METASTATIC involvement of joints, tendons or bursæ is very unusual. Tumors arising from joint structures (except bone) are very uncommon.[36,42] They may be grouped as follows:

Benign:

A. Osteochondroma or chondroma (the commonest tumor).
B. Xanthomatous giant cell tumors.[27,55]
C. Fibroma.[59]
D. Lipoma.[14,48,49]
E. Angioma.[2,39]

Malignant:

A. Synovioma (synovial sarcoma).
B. Chondromyxosarcoma.
C. Liposarcoma.
D. Fibrosarcoma.
E. Myeloma.[21]

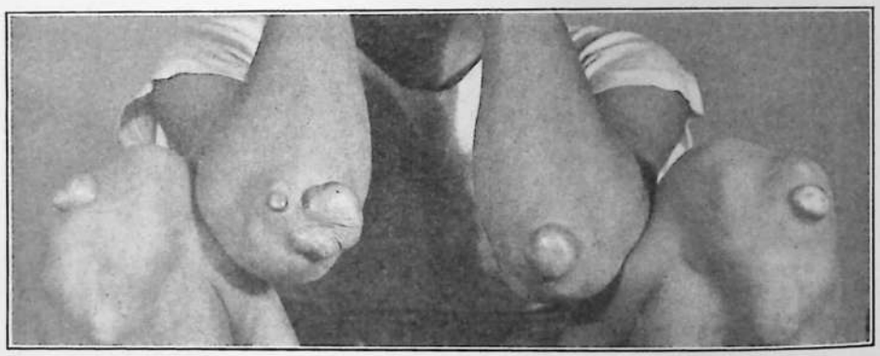

FIG. 316.—Multiple **xanthomata** near joints (knees and elbows) in young colored male.

Osteochondroma.—These growths resemble benign chondromata histologically; they show areas of calcification and ossification.[7,43] Only 126 instances of this disease could be found in the literature until 1934.[22,32,56] Chondromatous metaphasia of synovia of the knee joint has been reported.[10] The essential points concerning these lesions are as follows:

Most common between the ages of twenty and fifty, especially in males.

Frequency of joint involvement: knee, elbow, ankle, hip and shoulder, and occasionally in the hands and feet.

Usually a monarticular disease.

Pearly bodies in the joint may vary from several to over a hundred in number.

May produce a bag of marbles sensation in the knee.

Bodies may be partly calcified or ossified; usually readily detected by roentgen-ray.

Treatment: surgical removal with excision of synovial membrane containing any additional bodies.

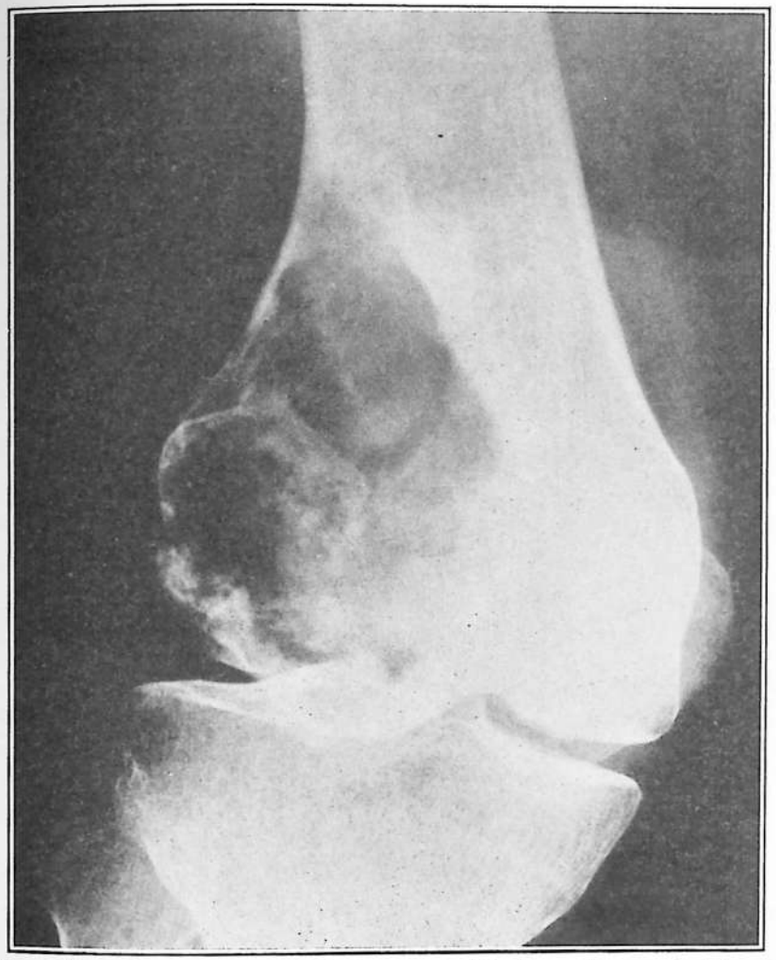

FIG. 317.—**Giant cell tumor** of the lower end of the femur in a young girl, aged twenty-two years.

Xanthomatous Giant Cell Tumors: Histologically are composed of foam cells, loose connective tissue and giant cells.[45]

Second commonest form of benign joint tumor; very rarely diagnosed preoperatively.

Usual location: knee joint.

May occur at any age; grow slowly.

DeSanto and Wilson reported 9 cases of xanthomata of the knee joint

and listed 32 from the literature. No case has been diagnosed correctly preoperatively.[15] O'Donoghue reported a xanthoma of the knee joint; this was solitary, pedunculated and well delineated. The symptoms were similar to those of rupture of the medial meniscus.[45]

Points suggesting a diagnosis of xanthoma of the knee joint:[15] obscure intermittent joint swelling, pain, free fluid in the joint, occasional locking of the joint and a movable tumor (most often medial to the patella). Dark or sanguinous fluid may be found on aspiration of the joint; this may contain a large amount of cholesterol.

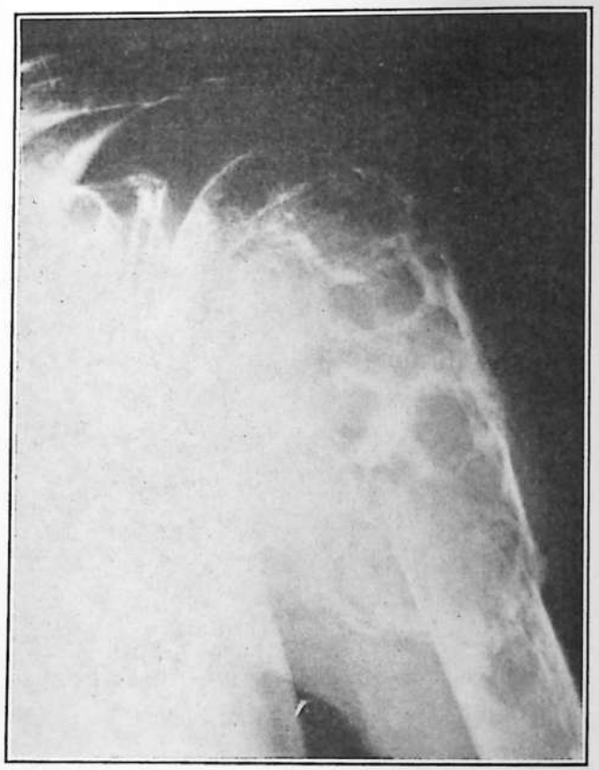

FIG. 318.—Giant cell tumor of the upper end of the left humerus in a man, aged thirty-four years.

Galloway. Broders and Ghormley reported a clinical and pathologic study of *xanthoma of tendon sheaths and synovial membranes*.[20] They showed that these were slowly growing painless tumors averaging approximately five years before their discovery. These vary in shape and size, but average 2.5 by 2 cm. when first seen by the physician. In most instances, these are *firm, round or oval lesions*, occurring most often on the fingers, being two to three times as common on the upper extremity as on the lower. The average age of their patients was forty-four years, with the majority of cases being in the third to sixth decades. These workers thought that a

mild, constant and unnoticeable trauma and possibly infection were important secondary factors in the production of the tumors. The primary factor was an alteration in lipoid metabolism.

When xanthomas involve joints, pain and heat may or may not be present; examination of the aspirated fluid reveals an elevated cholesterol content.[13]

A giant cell tumor of the synovial membrane of the knee joint was reported in a negro of fifty-two years. These are extremely rare.[28]

Lipoma: May arise from extracapsular fat or occasionally may be intracapsular (lipoma arborescens).

Large extracapsular lipomas may be diagnosed by roentgen-ray.[23,40,54]

Intra-articular lipomas (*lipoma arborescens*) represent hypertrophied synovial tissues distended with fat; they are very rare (approximately 10 cases reported in the literature).

Treatment when necessary: surgery.

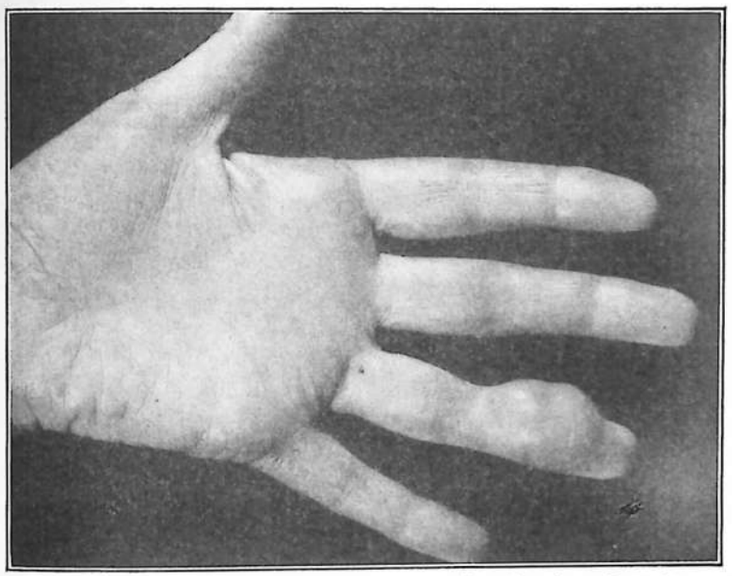

FIG. 319.—Coarsely nodular tumor of two years duration in a forty-four year old woman. Soft mass easily shelled out arose from flexor tendon sheath. Microscopic diagnosis: **xanthoma.** (Brunschwig, courtesy of Surgery.)

Fibroma and Angioma.—These are extremely rare.[3,47]

Trauma is not a factor in hemangioma of joints.

Roentgenograms may be negative.[11]

Symptoms suggesting tumor of the spinal cord should lead to an investigation for the possibility of **hemangioma of the vertebra.**[5] Hemangiomas of the vertebræ have been found in over 10 per cent of a large series of routine autopsies; these rarely result in compression of the cord, but if they occur in young males, they seem to produce symptoms rather often. Less than 75 cases of hemangioma of the vertebra with compression of the cord have

been reported.[17,24] Although the hemangioma may show no demonstrable roentgen signs, there is usually seen a reduction of bone density between parallel trabeculæ which are increased in density. If the diagnosis is made before the cord has been irreparably damaged, roentgen therapy may effect a cure. Laminectomy is advised when paraplegia has developed.

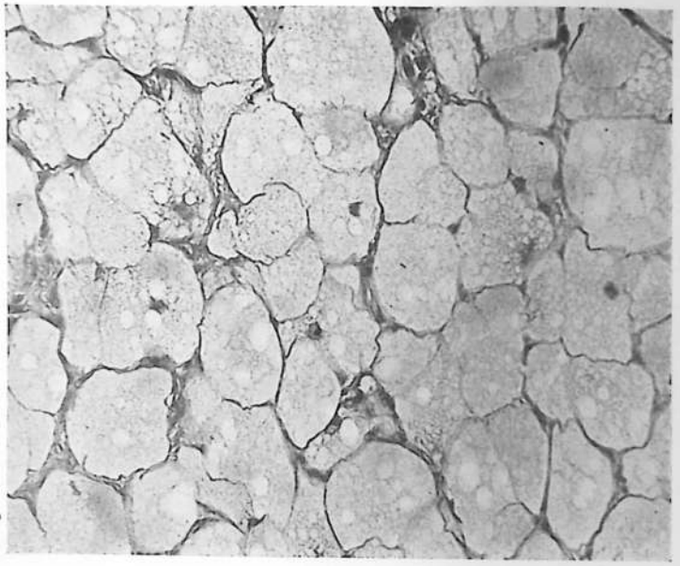

FIG. 320.—Photomicrograph of a **xantholipoma** of the knee joint. (Geschicter, courtesy of Am. Jour. Cancer.)

Primary hemangioma of the vertebra is comparatively common but rarely gives rise to symptoms.[35] The diagnosis can usually be made by the coarse meshed appearance of the vertebra from vertical striations alternating with large areas of bony rarefaction. If the lesion produces neurologic symptoms, surgery should be attempted if the patient is a good operative risk; if not, radiation therapy may be tried. Kaplan reported a hemangioma of the elbow successfully treated with radium at an early age.[34]

The possibility of *disseminated hemangiomas of bone* must be borne in mind when diffuse osteolytic lesions of the skeletal system are seen. In such cases, one must also consider the possibility of xanthomatosis, hyperparathyroidism, multiple myelomata, metastatic malignancy, etc.[8,46]

Malignant Synovioma:[12,50,53] (Fibrosarcoma or Synovial Sarcoma).

Synoviomata, or tumors of articular synovial membrane, are infrequent;[4,19] the diagnosis is seldom made clinically.[1] *The prognosis is usually unfavorable because of local recurrence and metastasis.*

Haggart reported removal of a synovioma of the knee joint, with apparent recovery (no evidence of recurrence eight and a half years following operation). The cystic swelling appeared on the lateral aspect of the knee and arose from the synovial membrane in the posterior compartment of

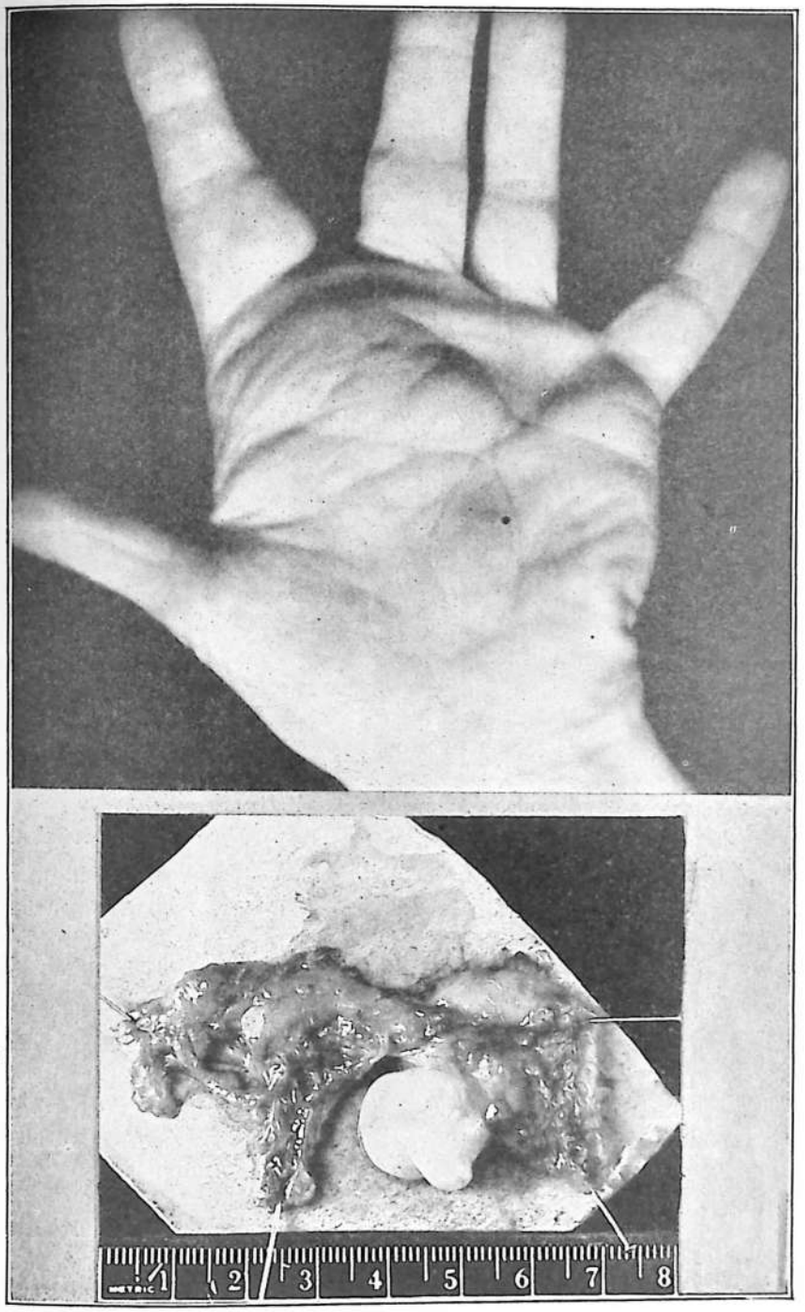

FIG. 321.—Fleshy tumor of the palm and base of the index finger present since childhood and a localized manifestation of a generalized von Recklinghausen's **neurofibromatosis;** no functional impairment. Photograph of the excised specimen shows plexiform neurofibroma with one myxomatous portion (the white mass attached to the lower margin). (Brunschwig, courtesy of Surgery.)

the knee, communicating with the knee joint. It was firmly adherent to the external semilunar cartilage, which was removed with the tumor mass.[26]

King described 7 cases of synovial sarcomata originally attached to a tendon sheath. In 6 of the 7, excision was followed by recurrences or metastases. Wide excision together with radium implantation gave the only one satisfactory result.[38]

The tumor may at times be cystic and may show a bluish discoloration over its surface. It is often very soft.[18] Microscopically it is composed of two types of cells—one, cuboidal (epithelium or endothelium) and a smaller spindle shaped cell.

The structure is identical with that of tumors from peripheral nerves (neuro-epithelioma).

Origin: synovial membrane.[30]

Age incidence: from nine months to sixty-four years, usually affecting young persons.

Order of incidence of joint involvement: knee, fingers, ankle, metatarsus.

May be of low or high grade malignancy; they are usually highly malignant.

Symptoms: if the knee is involved—dull aching pain, made worse on weight bearing; may exist without impairment of joint function. Spontaneous hemarthrosis may occur.[51]

Usually discovered at exploratory operation.

May be detected by roentgen-ray using soft tissue films.[57]

A provisional diagnosis of synovioma is justified if one sees by roentgen-ray a rounded, sometimes lobulated, sharply defined soft tissue mass occurring near or involving a joint and in which mass is found a scattered and irregular deposit of amorphous lime.[41]

Metastasis: may occur to the lungs.

Treatment: radical wide excision; tumors are not radiosensitive.

Prognosis: poor; 20 per cent five-year survivals—no survivals over ten years; tendency to recur following excision.

Tumors of Tendon Sheaths.—The following tumors have been reported:

Benign:

A. Chondroma and osteochondroma.
B. Ganglion.
C. Giant cell tumor.[16,58]
D. Uncommon: fibroma,[31] lipoma,[6,53,60] hemangioma,[9] lymphangioma.[29]

Malignant:

A. Fibrosarcoma (rare).[33]
B. Chondrosarcoma.

Primary tumors of tendons are very rare, the most common being a sarcoma.[25,44] Primary neoplasms of tendon sheaths are more frequent.[37] The basic cell of such tumors is the synovial cell. The latter is of mesenchymal origin and possesses multipotential qualities in that it may become cartilage or bone or may revert to fibroblasts.

Osteochondroma: Usually causes only mild pain on motion of the involved tendon. If these lesions increase in size rapidly, the possibility of malignant change must not be forgotten. If they are causing symptoms, they may be excised.

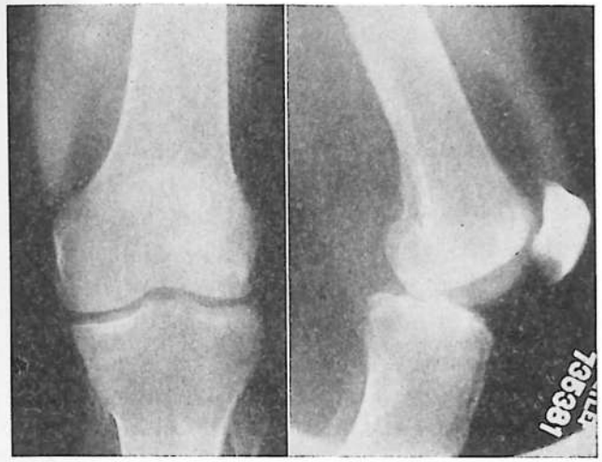

FIG. 322.—Anteroposterior and lateral views of knee joint; the shadow of a **lipoma** may be seen beneath and mesial to the quadriceps tendon. (Ghormley, courtesy of Minnesota Med.)

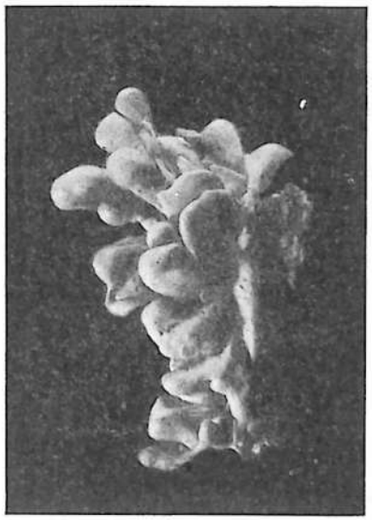

FIG. 323.—**Arborescent lipoma** from knee joint. (Geschicter, courtesy of Am. Jour. Cancer.)

Ganglion has been discussed in another section. It probably represents degenerated connective tissue.

The **giant cell tumor** is probably the commonest tumor of tendon sheaths. It has also been called an endothelioma, myeloma, giant myeloma, xan-

thoma, granulation tissue tumor and xanthomatous giant cell tumor. It is seen most often in the tendon sheaths of the hands and feet but has been

FIG. 324.—**Synovial sarcoma** of the elbow. (Knox, courtesy of Am. Jour. Cancer.)

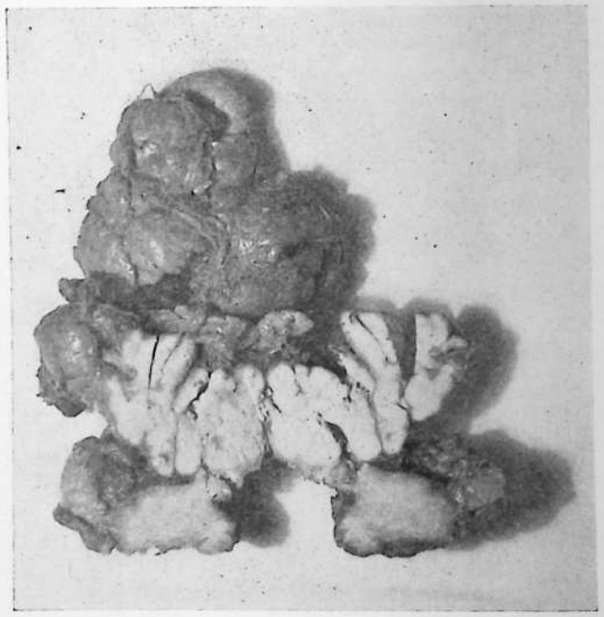

FIG. 325.—Cellular **synovioma** or low grade fibrosarcoma of a knee with invasion of the bursal and joint cavities, showing lobulated character of the growth. (Geschicter courtesy of Am. Jour. Cancer.)

reported at the wrists and ankles. The onset is insidious and growth is usually slow. Although such tumors may attain the size of a fist, they are rarely larger than a walnut. On examination, they appear as round or

lobulated smooth masses over which the skin is freely movable. They are encapsulated and are easily shelled out at operation; metastases do not occur. Microscopically they are composed chiefly of fibroblasts with a scattering of xanthoma and giant cells, blood pigment and cholesterin crystals.

Bursal Tumors.—Until 1934, only 30 tumors of bursæ could be collected from the literature. The prepatellar bursa was affected most often.[61] Such tumors include chondroma, osteochondroma, myxoma, and fibrosarcoma.

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CHAPTER LIX

THE SULFONAMIDES

Sulfonamides are now used successfully in the treatment of many diseases, particularly in **pneumonias** (except those of virus etiology), **hemolytic streptococcal** infections, **gonococcic** infections, most types of **meningitis**, infections due to the **colon bacillus** (particularly urinary tract infections) and others. The sulfonamide preparations most commonly used in large teaching hospitals at present are **sulfadiazine** and **sulfamerazine**; the latter has replaced sulfadiazine in many instances inasmuch as sulfamerazine is equally effective and may be given less often (every eight hours) to produce comparable results. The relative toxicity of these two drugs is approximately the same when prescribed in doses which produce comparable blood levels. Many newer sulfonamides are being tried clinically and it is probable that these may soon supplant sulfamerazine and other sulfonamides in use at present. Sulfanilamide is still used locally in wounds by many individuals because of its solubility and less irritating local effects. Sulfapyridine is seldom used and sulfathiazole has been supplanted by sulfadiazine or sulfamerazine, except possibly in the treatment of staphylococcic infections (when penicillin is not available).

Sulfonamides in Arthritis and Allied Conditions

1. *Contraindicated in active rheumatic fever* as they may aggravate the condition; sulfanilamide and sulfadiazine are extremely *useful in prophylaxis against* recurrent hemolytic streptococcal sore throats and recurrences of rheumatic fever.
2. Sulfonamides have *not* proved *of value in* the treatment of *rheumatoid arthritis;* they have been of value as a pre- and postoperative medication before removal of focal infection in these individuals, being given in an effort to prevent an untoward reaction.
3. Sulfonamides are *effective in* most cases of *acute specific infectious arthritis caused by* the *pneumococcus, hemolytic streptococcus, gonococcus* or *meningococcus* and in the arthritis associated with lymphopathia venereum, bacillary dysentery and scarlet fever.
4. Sulfonamides have been of value in infected wounds and in joint surgery.
5. There is no indication for any of the sulfonamides in the treatment of uncomplicated cases of gout or degenerative joint disease (osteoarthritis).

Penicillin is an extremely potent weapon with which to combat such devastating joint affections as gonococcic, pneumococcic, staphylococcic, streptococcic, meningococcic and syphilitic joint involvement. In patients with arthritis due to organisms susceptible to the action of sulfonamides, the latter must be given at once and in full dosage. If good results are not

evident within a very short period, or if sulfonamides are contraindicated in that particular individual, one should use penicillin at once (if the organisms causing the specific arthritis are susceptible to the action of this drug).

Sulfonamides Most Commonly Used at Present

1. **Sulfamerazine.**
2. **Sulfadiazine.**
3. Sulfathiazole (the sulfonamide of choice in staphylococcic infections).
4. Sulfanilamide.
5. Sulfasuxidine (used mainly in intestinal infections, bacillary dysentery and preoperatively in gastro-intestinal surgery). (This has supplanted sulfaguanidine to a large extent.) Sulfadiazine is now preferred in bacillary dysentery.
6. Others not yet thoroughly tried: sulfapyrazine, sulfamethazine, sulfamezathine.
7. *Sulfonamides no longer used extensively:* sulfapyridine, sulfaguanidine, sulfamethylthiazole, neoprontosil.

Average Dose of Sulfonamides

1. **Sulfamerazine:** in severe infections in adults (as in lobar pneumonia, hemolytic streptococcal infection, gonorrheal arthritis) *2 or 3 gm.* are given as the *initial dose*, followed by *1 gm. every eight hours* thereafter. Two gm. of sodium bicarbonate are given with each dose of sulfamerazine and fluids are forced so that the *urinary output* is *at least 1200 cc.* per twenty-four hours.
2. **Sulfadiazine:** the *initial dose* in adults in severe infections is *4 gm.*, followed by *1 gm. every six hours.*[26,34] One and one-half gm. of sodium bicarbonate are given with each dose and fluids are forced so that the urinary output is at least 1200 cc. per twenty-four hours.
3. If sulfonamides cannot be given or retained by mouth, either of these drugs may be given intravenously as the *sodium salt* dissolved *in sterile distilled water as a 5 per cent solution.*[22] Two and one-half gm. or more are usually given intravenously as the initial dose; it is usually possible to give subsequent doses by mouth. If necessary, intravenous injections may be given every eight to twelve hours in doses of from 1 to 2 gm. varying with the size of the individual, severity of the infection, etc.
4. If *sulfathiazole* is used, the *initial dose* is *4 gm.* for adults with severe infections, followed by *1 gm. every four hours.* Sodium sulfathiazole may also be given as a 5 per cent solution in sterile distilled water. Sodium sulfathiazole has been administered subcutaneously as 0.5 per cent aqueous solution at a rate of 100 cc. per hour without serious untoward local reactions.[63]
5. Sulfonamides are usually continued until the temperature has remained normal for forty-eight hours, unless the infection does not respond to sulfonamides.
6. In acute specific arthritis due to the gonococcus, pneumococcus, hemolytic streptococcus, meningococcus and other susceptible organisms, give full doses as in "1 or 2;" if prompt improvement does not occur, begin penicillin therapy.

Sulfamerazine.—Sulfamerazine is the monomethyl derivative of sulfadiazine.[41,75] It is two and a half times as soluble in water as sulfadiazine. The acetylated form has approximately the same solubility as free sulfamerazine and is almost twice as soluble in water as acetylated sulfadiazine.

Roblin and his associates found that sulfamerazine was more effective than sulfanilamide or sulfathiazole in white mice with infections due to streptococci, staphylococci and pneumococci.[30] It was thought that the superiority of sulfamerazine was due to the higher blood levels obtained with this drug.

Sulfamerazine is more rapidly and completely absorbed from the gastrointestinal tract and more slowly excreted than sulfadiazine.[69] Adequate blood levels of sulfamerazine can be maintained by doses administered at intervals of eight hours. Sulfamerazine diffuses readily into the pleural fluid in concentrations approximately the same as those of the blood and into the spinal fluid in concentrations about 50 per cent of that in the blood.[40]

Therapeutic results in meningococcic, pneumococcic, streptococcic and colon bacillus infections compared favorably with those following the use of sulfadiazine.

Keefer and his associates presented a **clinical evaluation of sulfamerazine.**[3] When equal doses of sulfamerazine and sulfadiazine were given by mouth to normal subjects, blood levels of sulfamerazine were on the average more than double those of sulfadiazine.

Keefer gave, as a general rule, 2 gm. of sulfamerazine initially followed by 1 gram every eight hours thereafter in 278 patients with bacterial infections. *Children weighing 60 pounds or more were given the same dosage;* in those under 60 pounds, the initial dose was 0.5 gm. and the daily maintenance dose was computed on a basis of 0.5 gm. for each 10 pounds of body weight and was given in 3 equally divided doses at eight hour intervals. It was found that *sulfamerazine showed a therapeutic efficiency similar to that of sulfadiazine in the treatment of pneumococcal pneumonia, meningococcal meningitis and erysipelas.*

It appears as though sulfadiazine and sulfamerazine are equally effective therapeutic agents in the treatment of meningococcic meningitis.

Sulfamerazine was clinically effective in doses which averaged one-half to one-third smaller than the amounts of sulfadiazine usually given in similar conditions. Toxic reactions to sulfamerazine were approximately the same as those caused by sulfadiazine although there was a greater tendency for sulfamerazine to produce leukopenia and occasionally drug rashes and drug fevers. Sulfamerazine is tolerated well by small infants and children.

Hall and Spink found sulfamerazine to be as effective as sulfadiazine in pneumococcic pneumonia, bronchitis or streptococcic infections, but to be less effective than sulfathiazole in the treatment of staphylococcic sepsis. Several cases of meningitis due to type B influenza bacilli and 3 patients with meningococcic meningitis recovered following sulfamerazine. Sulfamerazine did not appear to be any more toxic than sulfadiazine in human beings and did not produce any demonstrable neurologic complica-

tions. Sulfamerazine produced less nausea and vomiting than sulfapyridine and fewer skin eruptions and instances of drug fever than sulfathiazole.

Flippin and his associates found that sulfamerazine was as effective as sulfadiazine in pneumococcic pneumonia. They have advised the following dosage schedule in adults: 3 gm. of the drug initially followed by 1 gm. every eight hours until the temperature remains normal for forty-eight hours and the patient shows evidence of clinical improvement. If oral therapy is impractical or impossible, the sodium salt of sulfamerazine is given intravenously as a 5 per cent solution in distilled water. *The rapid absorption obtained with sulfamerazine makes the parenteral route unnecessary in most instances.*

Sulfamerazine is absorbed more rapidly and more completely than sulfadiazine from the gastrointestinal tract. Sulfamerazine is excreted more slowly in the urine than sulfadiazine. Blood levels rise more rapidly and higher levels are reached and sustained longer following oral administration of sulfamerazine than with an equal amount of sulfadiazine.[38]

The higher concentration of sulfamerazine in the blood compared with other sulfonamides is due in part to the more complete absorption from the gastrointestinal tract and in part due to the slower rate of excretion.[29]

The toxic effects of sulfamerazine are dependent upon precipitation of the drug in the renal tubules, pelves or ureters.[90] Both free and acetylated sulfamerazine are much more soluble when the pH of the urine is raised from 6.5 to 7.5.

NEWER SULFONAMIDES

Sulfapyrazine seemed to produce fewer severe renal complications than other sulfonamide drugs (except sulfanilamide).[7] This newer sulfonamide preparation was found to be effective clinically in lobar pneumonia, meningococcus meningitis, erysipelas, bacillary dysentery and non-specific respiratory infections.

Sulfamethazine is two-and-a-half times as soluble in water as sulfamerazine.[76]

Sulfamethazine is effective in human beings against infections due to pneumococci, meningococci and gonococci; no serious toxic reactions are encountered.[47,67]

Sulfamezathine and its acetyl derivative were found to be four times as soluble as corresponding compounds of sulfadiazine. Sulfamezathine was rapidly absorbed from the intestinal tract. The drug was effective against pneumococcic infections and did not show evidence of gastrointestinal irritation or urinary complications such as oliguria.[68]

Complications Following Sulfonamides.—Complications following sulfonamides vary with the sensitivity of the individual, the particular disease treated, the specific drug used and the supportive treatment adopted. In most instances, the signs and symptoms of sulfonamide intoxication are similar regardless of which drug causes the reaction.[14,28]

Toxic symptoms are not usually related to the strength of dosage as these may occur after one or two doses have been given. In these cases, it would appear that the person's sensitivity caused the reaction; this seems more common following the use of sulfanilamide and sulfapyridine. Symptoms which are common in such cases include nausea, vomiting, psychosis, ocular and auditory disturbances and dizziness.

Mild *toxic manifestations* occur commonly in the **blood**; these include mild *anemia, leukopenia,* leukocytosis and *thrombocytopenia.* These usually clear up rapidly within twenty-four hours after cessation of drug therapy. *Agranulocytosis* occurs rarely, but may appear suddenly after a prolonged course of the drug.

Toxic reactions involving the white blood cells are usually negligible following the use of sulfadiazine in uncomplicated cases of pneumonia. Leukopenia may occur occasionally but should not cause undue apprehension unless the drug is given for ten days or longer; however, one must respect a progressive lowering of the white blood count and granulocytes.

Acute *hemolytic anemia* occasionally follows the administration of sulfanilamide and sulfathiazole and is more common in children. Although it appears most often about the second day, it may occur as late as the twentieth day. Symptoms may appear rapidly and consist of nausea, vertigo, jaundice, hemoglobinuria, urobilinuria, reticulocytosis and a secondary fever.[10] Acute hemolytic anemia is rare following the use of sulfadiazine; if this occurs, the drug must be stopped and blood transfusions employed.

Renal complications are relatively *common* in all groups. *Hematuria* is the most common, but *oliguria, anuria,* renal or ureteral colic may occur. *Crystals are often deposited in the renal tubules leading to mechanical obstruction and suppression of urine. This complication is much less frequent if fluids are forced so that the urinary output is maintained at at least 1200 cc. per twenty-four hours and if sufficient alkali is given to maintain a pH of 7.5 or higher in the urine.*

Toxic reactions in the urinary tract following sulfadiazine are more common after the fifth day of treatment. These are due in part to the presence in the urinary tract of crystals composed of the drug. The presence of *crystalluria alone does not indicate renal involvement* unless this is associated with progressing oliguria, hematuria, azotemia or pain in the loin. Cautious treatment may be continued unless a considerable number of red blood cells are detected in the urine or other evidence of renal damage is apparent; hematuria is often a precursor of severe renal insufficiency. *In almost all instances in which severe renal reactions to sulfadiazine have been observed, sufficient fluids or alkalis had not been given and sulfadiazine had been given for four or more days.* **Treatment of renal complications following sulfonamide medication** includes prompt cessation of the drug, alkalinization of the urine, forcing of fluids, giving hypertonic dextrose solution to promote diuresis and ureteral catheterization if necessary. Mercurial diuretics or cathartics such as magnesium sulfate are contraindicated in such cases.

The frequent occurrence of crystalluria and hematuria following the use of sulfathiazole and sulfadiazine assumes special importance to our armed forces stationed in warm climates where it is extremely difficult to

maintain a satisfactory urinary output. Sulfamerazine has a greater solubility than other pyrimidine derivatives. Sulfamerazine and its acetyl derivative have been found to be 20 per cent more soluble than the respect tive forms of sulfadiazine in urine and water.

Urinary tract complications may occur following the administration of sulfamerazine and are due to the extrarenal precipitation of crystals resulting in mechanical obstruction of a free flow of urine. Such complications are prevented by an adequate fluid intake and alkalinization of the urine to maintain a pH of 7.5 or higher.[35,37,48,80]

Complete anuria may be due to calculi blocking the urinary tract at the ureterovesical orifices.[78] When complete anuria occurs cystoscopy should be done to determine the presence of such an obstruction. If none is present, ureteral catheterization should be performed and the kidney pelvis and ureter should be thoroughly lavaged with warm distilled water until urine is seen dripping from the catheter and the upper urinary tract is clearly outlined in a retrograde pyelogram. It is sometimes advisable to keep the ureteral catheters in place for twelve hours and irrigate these from time to time with sterile saline. Fluids should always be forced if there is any marked diminution of urinary output; renal concretions may be redissolved or washed out.

Skin rashes occur in various forms following the administration of sulfonamides. Following sulfanilamide, a diffuse erythematous or macular eruption is most common.

Cutaneous and episcleral reactions may occur at any time after sulfadiazine therapy but are seen usually after the third day of treatment.

Purpuric rashes have been noted, especially in children following the administration of sulfanilamide or sulfapyridine. Exfoliative dermatitis and urticaria may occur in instances of individual sensitivity. Pemphigoid eruptions have been noted in a few instances following the administration of sulfathiazole.

Dermatitis occasionally follows local application of sulfonamides.

One of the most alarming toxic complications of sulfadiazine therapy is generalized exfoliative dermatitis.[50]

Psychoses are usually transient. Other untoward symptoms following sulfonamides may include cyanosis, pruritus, abdominal pain, distention, diarrhea, conjunctivitis, disturbed visual acuity and *painful joints*.

Fever *due to sulfadiazine* may occur at any time but most commonly after the fourth day of therapy.[31] *Whenever possible, one should discontinue chemotherapy in the presence of drug fever, as this condition may be followed by more serious toxic reactions.*

Six cases of multiple **peripheral neuritis** have been noted in 600 patients treated with sulfonamides. In 5 instances, the total dose of the drug was 50 gm. or more.[9,51]

Following continuous sulfonamide therapy, as in the prophylaxis of rheumatic fever, sulfonamide **resistant strains of bacteria may develop.**

The **vomiting** which sometimes follows sulfadiazine is rarely severe enough to necessitate stopping the drug.

Minimize Toxic Complications From Sulfonamides by:

1. Questioning the patient concerning previous sensitivity to sulfonamides.
2. Maintain a *urinary output* of at least 1200 cc. daily.
3. Give sufficient *alkali* to keep the urine alkaline.
4. Watch for jaundice, rash or drug fever.
5. If the drug is to be administered for prolonged periods, examine the blood and urine frequently.

Toxic Reactions Following Administration of the Sulfonamides[13]

(SA = sulfanilamide; SD = sulfadiazine; SP = sulfapyridine; SM = sulfamerazine; ST = sulfathiazole.)

1. *Nausea and vomiting:* frequent after SP; common after SA; uncommon after ST, or SM.
2. *Marked leukopenia:* 0.3 per cent of SA; 0.6 per cent of SP; 1.5 per cent of ST (true agranulocytic angina—rare). *Mild leukopenia is common after sulfamerazine.*
3. *Fever:* 10 per cent of SA; 4 per cent of SP; 10 per cent of ST; 3 per cent of SD; 4 per cent of SM.
4. *Anuria with azotemia:* 0 with SA; 0.3 per cent with SP; 0.7 per cent with ST.
5. *Hematuria:* none after SA; microscopic hematuria in about 8 per cent of SP patients; 9 per cent of ST; 4 per cent of SD or SM; gross hematuria in 1 per cent or less of sulfapyridine or sulfathiazole treated patients.[33]
6. *Neuritis:* rare with SA; none in SP; uncommon but occurs with ST.[24]
7. *Rash:* in 2 per cent of SA or SP; 3 to 5 per cent with ST, SD or SM (has been as high as 9 per cent in one series).[82]
8. *Injection of sclera and conjunctiva:* none with SA or SP; occasionally with ST, SD or SM.
9. *Painful joints:* very rare after SA or ST.
10. *Hepatitis:* in 0.25 per cent of SA; rare in others.
11. *Renal calculi:* none with sulfanilamide; 0.3 per cent with ST and SP; as high as 1 per cent or more with SD and 3 per cent with SM.[23]
12. *Psychosis:* 0.5 per cent of SA patients; 0.3 per cent of SP; rare in ST; more common with SD.
13. *Stomatitis:* rare with SA; none with others.
14. *Hyperleukocytosis:* SA if acute hemolytic anemia occurs; same in SP; none in ST.
15. *Dizziness:* common with SA or SP; uncommon with ST, SD or SM.
16. *Purpura hemorrhagica:* rare with all.
17. *Acute hemolytic anemia:* about 2 per cent of SA; about 0.5 per cent of SP; very rare or absent in ST; about 0.5 per cent in SD and SM.
18. *Cyanosis:* very common with SA; not often seen with SP; not seen with others.
19. *Acidosis:* in about 2 per cent of cases after SA; none after SP, ST, SD or SM.

Patients receiving sulfadiazine or sulfamerazine should receive a fluid intake sufficient to maintain a urinary output above 1200 cc. per twenty-four hours, and should ingest enough sodium bicarbonate or potassium citrate to keep the pH of the urine at 7.5 or higher. In patients with cardiac failure or renal dysfunction, this procedure must be modified. One must remember

that it is possible that renal failure may be associated with a direct toxic effect of sulfonamides on renal parenchyma or a hypersensitivity phenomenon.

Precautionary Measures During Sulfonamide Therapy

1. Determine whether the patient has ever had *previous sensitivity* to any sulfonamide preparation; previous untoward reaction from any member of this group of drugs may be a distinct *contraindication* to its use. Such sensitivity includes drug fever, agranulocytosis, gross hematuria, dermatitis, hepatitis or acute hemolytic anemia.
2. *The patient taking any sulfonamide preparation should be seen by the physician at least every second day.*
3. The *physician must always look* for *jaundice*, evidence of *anemia, dermatitis* and *injected conjunctivæ and scleræ.*
4. Any untoward symptom may be a precursor of a toxic reaction; inquire as to the general health of the patient, nausea, malaise, headache, aching of the body, vomiting and gross hematuria.
5. The urinary output must be measured each day and the urine must be examined at least grossly for hematuria. *A urinary output of 1200 cc. per day or more should be maintained* (to decrease the possibility of formation of renal calculi[72,79]). Any marked decrease in urinary output is an indication to stop the drug and force fluids.
6. A mounting temperature on the fourth or fifth day after this has become normal previously is often a sign of *drug fever;* the drug should usually be stopped in these patients.
7. *Stop the drug and force fluids at once if there is* drug fever, oliguria, gross hematuria, jaundice, acute hemolytic anemia, rash, conjunctivitis (especially with sulfathiazole) or marked progressive leukopenia with a marked decrease in the polymorphonuclear leukocytes.
8. When large doses of sulfonamides are used, as in pneumonia, a white blood count and differential count should be done at least every several days. If therapy is required for more than seven days, a complete blood count should be done frequently even when one is employing small doses of these drugs.
9. The patient must receive an adequate diet in respect to calories and vitamins, and should receive any other medication definitely indicated.
10. Sufficient alkali should be given to keep the urine alkaline; this decreases the tendency to renal complications.
11. If the hemoglobin falls below 40 per cent during therapy, blood transfusions may be necessary. If acute hemolytic anemia does not occur, the drug may be continued.
12. Great caution must be used in treating patients with renal or hepatic damage with sulfonamide preparations.
13. Patients receiving sulfonamide preparations should not drive automobiles or fly planes.

Sulfonamides do not cure all infections. They will prove of great benefit and will at times be life-saving if used intelligently, used early and combined with other supportive measures. Unfortunately, some physicians have forgotten that even with the assistance of these drugs, the patient needs adequate rest, fluids, caloric and vitamin intake, specific serum in some instances, good nursing care and careful observation by the medical consultant.

The original Prontosil was the product reported on in 1933 by Foerster who is credited with the first recorded cure of staphylococcic septicemia. In 1935, Domagk, in an epoch making paper, introduced to the world the marked therapeutic effectiveness of this substance in experimental streptococcic and to some extent, staphylococcic infections.

Diseases in Which Sulfonamides Are of Value

1. Infections due to **beta hemolytic streptococci** (including erysipelas; puerperal sepsis; some cases of septicemia, meningitis, otitis media, lymphangitis, sinusitis, peritonitis, Ludwig's angina, osteomyelitis, mastoiditis, cellulitis, scarlet fever, etc.; it is not usually required in mild cases of tonsillitis, but will usually markedly hasten convalescence).[74]
2. Infections due to **meningococci.**
3. Infections due to **gonococci.**[16]
4. **Pneumococcal** infections.
5. **Urinary tract infections** (except those due to the Streptococcus fæcalis). *Sulfonamides* have been effective in such infections when due to Staphylococcus albus and aureus, E. coli, B. ærogenes, B. proteus, B. pyocyaneus and others.
6. Infections due to staphylococci.
7. Infections due to **Clostridium welchii.**
8. *Lymphopathia venereum.*
9. *Trachoma.*
10. *Chancroid.*
11. Actinomycosis (?).
12. There is much doubt concerning the efficacy of sulfonamides in chronic ulcerative colitis or Brucellosis even in the acute stage.[17]
13. Sulfonamides have *not* been of value in poliomyelitis, encephalitis, rabies, rickettsial infections, filariasis, trypanosomiasis, whooping cough and many other infections.

Sulfadiazine

This possesses certain advantages in that it is readily absorbed from the gastrointestinal tract, is excreted more slowly by the kidneys than sulfathiazole or sulfapyridine (and thus must not be given as frequently) permitting higher blood levels with the same dose, its acetylation is uniform and averages only 20 per cent, allowing more of the free or active form of the drug in the blood stream and less of the crystals in the kidneys, and the acetylated sulfadiazine is more soluble in the urine than sulfapyridine or sulfathiazole so that there is less crystal formation.[71,73] Only 1 patient in 10 receiving sulfadiazine shows crystals in the urine as compared with 6 in 10 receiving sulfapyridine or sulfathiazole.

It penetrates all body fluids, being found up to 55 per cent of the free drug in the spinal fluid. It causes practically no nausea or vomiting and produces a more abrupt drop in temperature (in patients with pneumonia) than sulfathiazole. Its only known disadvantage is that it penetrates erythrocytes as does sulfapyridine and therefore may rarely cause acute hemolytic anemia.

Sulfadiazine is readily absorbed from the gastrointestinal tract, disappears from the blood slowly and is not excreted as readily as sulfathiazole. Its concentration in peritoneal and pleural fluids approximates that of the blood.

Experimental studies have shown that sulfadiazine is more effective against experimental streptococcal or pneumococcal infections in mice than sulfanilamide, sulfapyridine or sulfathiazole. Effective blood concentrations are reached in two to four hours after oral administration; it disappears slowly from the blood so that appreciable quantities remain in the blood forty-eight hours after oral administration of 3 grams of the drug. The drug enters the pleural and peritoneal cavities readily in concentrations only slightly less than blood.

Sulfadiazine has been found to be of marked value in the treatment of **meningococcic infections.**[20,27,28,58,87] Higher death rates still occur in elderly patients even with sulfonamide therapy.[8,46] Although sulfonamides are effective in the treatment of meningococcic meningitis, this disease is extremely variable in severity.[19,77,85,88]

Lepper, Sweet and Dowling reported cures in 86 of 96 patients with sulfadiazine therapy in **meningococcic meningitis** and only 2 deaths in 22 patients following sulfamerazine. Approximately 25 per cent of all patients admitted in coma died, while all patients who were conscious on admission lived. Almost 25 per cent of the patients over the age of forty died. It thus appeared that coma and the age of the patient were the two most important factors in prognosis. Other unfavorable prognostic signs were the presence of numerous organisms or a very low dextrose level in the initial spinal fluid. *Sulfadiazine has been used prophylactically in soldiers with the resultant reduced incidence of infection, reduced carrier rate* and without serious toxic effects.[57]

Dosage: In adults with pneumococcic pneumonia, an initial dose of 3 or 4 grams is given by mouth, followed by 1 gram every six hours until the temperature is normal for forty-eight hours together with clinical improvement (unless toxic symptoms occur); total dosage for the period of treatment is approximately 20 to 30 grams.[32]

If rapid elevation of the blood level is desired, the sodium salt may be given as a 5 per cent solution (0.06 gm. per kilogram of body weight) in sterile triple distilled water intravenously; 3 grams of sulfadiazine sodium given intravenously may give a free blood level of 10 to 12 mg. per cent within fifteen minutes, with maintenance of a concentration above 6 mg. per cent for at least eight hours.

Sulfathiazole

Sulfathiazole has a therapeutic effect in mice equivalent to that of sulfapyridine against infections due to pneumococci,[81,89] meningococci and beta hemolytic streptococci and against the agent of lymphogranuloma venereum, E. coli, Staph. aureus and B. proteus. **Sulfathiazole appears to have a greater bacteriostatic activity for Staph. aureus than sulfanilamide or sulfapyridine.** Experimentally and clinically sulfathiazole appears to be as effective if not more so than sulfanilamide or sulfapyridine in *urinary tract infections.* Sulfathiazole readily kills strains of B. proteus and ærobacter ærogenes[2,44] and has a bactericidal action on strains of Streptococcus fæcalis while sulfanilamide and sulfapyridine have no bactericidal action against the latter organism.

Use of Sulfathiazole in Staphylococcic Sepsticemia: Sulfathiazole was the most effective therapeutic agent in the treatment of this disorder until the introduction of penicillin. Spink and Hansen reported recovery of all of 15 patients with staphylococcic septicemia following use of sulfathiazole in sufficient amounts to maintain a blood concentration of 4 to 5 mg. of free sulfathiazole per 100 cc., together with adequate drainage of the focus or foci promptly.

Urinary Tract Infections: Sulfathiazole is of value in urinary infections due to Staph. aureus, Proteus ammoniæ, Strep. fecalis, E. coli, Ærobacter ærogenes and Pseudomonas æruginosa. In adults, ½ to 1 gram of sulfathiazole may be given every four hours for three to five days, following which this can often be reduced. In urinary tract infections in patients with severe renal damage 60 to 120 mg. (1 or 2 grains) of sulfathiazole given four or five times a day may have a marked bactericidal effect.[43,45]

Sulfanilamide

Sulfanilamide is seldom used at present except in the prophylaxis of rheumatic fever, for local application, and occasionally in individuals with renal damage.

Sulfanilamide has been used prophylactically in some instances. Kolmer advised the prophylactic use of sulfonamides in the following conditions:[56]

A. Cases of difficult delivery and to puerperal women exposed to streptococcic infection at or near delivery.
B. Prevention of recurrence of rheumatic fever[15,86] (given only during periods of quiescence of the disease); the drug is not of value therapeutically in the disease, and may make the patient worse if used during active rheumatic fever. This is discussed in detail later. Sulfanilamide has been the drug of choice for this purpose because of its solubility and absence of renal complications. This is discussed in detail in the chapter on Rheumatic Fever.
C. Prevention of flareup of an arthritis, iritis, nephritis, etc., following removal of an infected focus.
D. Prevention of peritonitis following rupture of an appendix or open intestinal wounds.
E. Patients exposed to gas gangrene.
F. Epidemics of sore throat from milk borne streptococci.
G. Prevention of extension of hemolytic streptococcal infection to the mastoid cells, middle ear or meninges in severe cases of sinusitis.
H. Prevention of hemolytic streptococcal infection after pricking a finger at autopsy or in operating upon infected areas.

Mode of Action of Sulfanilamide.—Lockwood has shown that sulfanilamide has a *bacteriostatic* and to some extent a *bactericidal* action *in vitro* against numerous organisms (such as hemolytic streptococci, colon bacilli, pneumococci, etc.). It produces an environment in the body tissues and fluids which is unfavorable for bacterial multiplication, thus limiting the course of an infectious lesion.[60,61] Depression of the capacity of the bacteria to multiply diminishes their invasiveness and reduces their vitality to the point at which the natural defenses of the body are able to dispose of the surviving organisms. *Sulfanilamide probably acts by interfering with the nutritional requirements of susceptible bacteria.*[52]

Lockwood has shown that this drug is most effective in the early stage of infections where there is little suppuration; *it is often ineffective when well established abscesses are present and in wounds with much necrotic tissue.* If abscesses are present, these must be drained even though sulfanilamide or other sulfonamide preparations are being used.

Absorption of Sulfanilamide.—Following its oral administration, it reaches its maximal blood concentration in approximately four hours. Some of the drug is then **conjugated** (*acetylated*) in the liver into acetylsulfanilamide; this latter is a practically inactive form of the drug. *The free (non-acetylated) form of the drug is the therapeutically active substance.*

Diffusion Into Body Tissues and Fluids.—*Sulfanilamide diffuses readily to all fluids and tissues of the body in relation to their vascular supply;* this is deficient in areas of chronic infection, necrotic tissue and bone. It has been found in exudates, transudates, cerebrospinal fluid, pleural, peritoneal and pericardial fluid,[70] saliva, bile, pancreatic juice and in fetal blood in the same concentration as in maternal blood. Although it is present in human milk in a concentration equal to that of the blood, there are apparently no toxic effects upon the nursing infant.

The distribution of sulfanilamide is essentially the same in all of the body tissues except fat and bone.[64] *Due to the increased permeability of capillaries which accompanies acute inflammation, there may be an increased level of sulfonamides in such areas as in pleural and joint effusions in the presence of infection.*[39]

Excretion of Sulfanilamide.—Free and acetylated forms are *rapidly excreted in the urine* except in patients with nephritis with nitrogen retention. Sulfanilamide is excreted almost entirely in the urine.

Methods of Administration of Sulfanilamide.—In most cases, the drug can be given by mouth. If necessary, it may be given as an 0.8 per cent solution subcutaneously, by rectum, intravenously or intraspinally; it has also been used intramuscularly but this is usually not indicated. Sulfanilamide is well absorbed from the rectum. The solution of 0.8 per cent sulfanilamide may be sterilized by boiling this three minutes at 15 pounds pressure.[6] Sulfapyridine, sulfathiazole and sulfamethylthiazole are poorly absorbed after rectal administration; the absorption is slightly better when the sodium salts of these drugs are used.[84]

Blood Levels of Sulfanilamide.—The effective agent is the "free sulfanilamide" present in the blood stream; in some cases, it is advisable to check

the blood level of free sulfanilamide from time to time (as this varies in different individuals depending upon intestinal absorption, renal excretion, etc.). In very severe infections, it is usually advisable to attempt to maintain a blood level of approximately 10 to 12 mg. per 100 cc. of blood (or of from 5 to 10 in moderately severe infections).

Because the fluid intake per kilogram of body weight is greater in children than in adults, children require a greater amount of sulfanilamide per kilogram to establish adequate blood levels.

Dose of Sulfanilamide

This varies considerably depending upon the disease being treated, the severity of the infection, age of the patient, presence of renal disease, etc. A general working suggestion (but by no means a rule) is as follows: *In adults of 110 pounds or more:*

1. *Mild infections* (susceptible to sulfanilamide), 0.6 gm. (10 gr.), every four hours, day and night (if possible).
2. *Moderately severe infections*, 1 gm. (15 gr.), every four hours day and night.
3. *Very severe infections*—initial dose of 5 gm. (75 gr.) and then 1.3 gm. (20 gr.) every four hours day and night; follow the blood concentration of free sulfanilamide if the patient does not improve.

Dose of Sulfanilamide Given Parenterally (0.8 Per Cent Sulfanilamide in Physiologic Saline)

In severe infections or in those patients who cannot swallow or retain the drug by mouth, parenteral administration may be required for the first several doses.

A. *In moderately severe infections:* 0.75 cc. per pound of body weight each six hours (by hypodermoclysis or intravenously).
B. *In very severe infections:* 1.2 cc. per pound of body weight each 6 hours.

Frequency of Administration.—Inasmuch as the drug is absorbed from the intestinal tract and reaches its highest blood level within four hours, it is usually advisable to administer this drug at intervals of four hours (day and night in severe infections).

Dose of Sulfanilamide in Children

A. *Oral:* Initial dose, 30 mg. (½ gr.) per pound (maximum, 4 gm. [60 gr.]). Subsequent doses, 10 to 15 mg. (⅙ to ¼ gr.) per pound every four hours (maximum dose, 1.3 gm. [20 gr.]).
B. *Subcutaneous:* 0.8 per cent sulfanilamide in physiologic saline. Initial dose, 4 to 5 cc. per pound (maximum dose, 500 cc.). Subsequent doses, 3 to 4 cc. per pound at intervals of eight to twelve hours.
C. *Intravenous:* Maximum dose, 3 cc. per pound total in twenty-four hours of the 0.8 per cent solution of sulfanilamide in physiologic saline.[11]

Use of Other Drugs During the Course of Sulfanilamide Therapy.—Other drugs may be given safely, if needed, during the course of sulfanila-

mide therapy. It is advised that the patient receive no magnesium or sodium sulfate because of the possible increased formation of sulfhemoglobin.

Exposure to sunlight or ultra-violet rays may produce a dermatitis during sulfanilamide therapy because of photosensitization.

Cause of Cyanosis During Sulfanilamide Therapy: This is due to the formation of methemoglobin (and rarely sulfhemoglobin).[42]

Sulfapyridine

Although sulfapyridine has been very effective in pneumonia due to the pneumococcus,[1,36] it has been superseded by the newer derivatives, sulfathiazole, sulfadiazine and sulfamerazine (which drugs retain the therapeutic effectiveness of sulfapyridine without the unpleasant gastrointestinal irritation caused by the sulfapyridine).

Sulfapyridine was found to be an effective chemotherapeutic agent in experimental infections in mice due to pneumococci, streptococci, meningococci, staphylococci, Friedländer's bacilli, Welch bacilli and other organisms. Clinically, therapeutic success has been achieved in all types of pneumococcic lobar and bronchial pneumonia, meningococcic, gonococcic, staphylococcic and other infections. It is apparently equally effective against all types of pneumococci.

Sulfaguanidine and Sulfasuxidine

These drugs are fairly soluble in water but unlike most other sulfonamide preparations *are very poorly absorbed from the digestive tract.* Most of these substances, when given by mouth, stay in the lower alimentary canal. They are among the least toxic of the sulfonamides.[65]

In view of its properties, a high concentration of *sulfaguanidine* may be obtained in the intestine and a low concentration in the blood and tissues. It was suggested that this substance may prove useful in the treatment of infections mainly localized in the intestine, and in lowering the bacterial count in the intestine prior to surgery of the bowel.

The drug is conjugated, rapidly penetrates the tissues except the brain and passes into the spinal fluid more slowly than sulfanilamide or sulfapyridine. It is less effective in mice against beta hemolytic streptococci than is sulfanilamide, but is as effective as sulfapyridine against pneumococcic infection in mice.[66] Marshall and his associates showed that the concentration of coliform bacteria of the feces of mice could be reduced by its administration. The drug is rapidly excreted.

Sulfasuxidine has proved superior to sulfaguanidine in the treatment of bacillary dysentery and as a preoperative drug (for gastrointestinal surgery). Some investigators now believe that sulfadiazine by mouth is the choice of therapy in bacillary dysentery.

MISCELLANEOUS CONDITIONS

1. **Actinomycosis:** Dobson and his associates have recently reported cure of 3 cases of actinomycosis; therapy included sulfanilamide, roentgen-rays and iodides.[21] Others have obtained cures in a few cases by therapy which has included sulfanilamide or sulfapyridine.

The value of thymol in actinomycosis is now generally accepted; potassium iodide is ineffective in true actinomycosis. Surgical excision of the diseased tissue has been of greater importance than thymol. Numerous authors observed recovery from actinomycotic infections following sulfonamide therapy.[62]

2. **Agranulocytosis:** Dameshek and Wolfson reported on the successful treatment of 2 cases of severe **agranulocytosis** with large doses of sulfathiazole, in addition to transfusion, pentose nucleotides and liver extract.[18] It was believed that death in this disease was due not to the lack of neutrophils *per se* but to the overwhelming sepsis. It was hypothesized that if the invading organisms could be held in check by the drug while natural processes in the bone marrow concerned with leukopoiesis might become reëstablished, the patient might recover. It must be remembered that cases of agranulocytosis occasionally occur during sulfonamide therapy and have been thought to be due to the depressing effect of sulfonamides on the bone marrow; however, if the leukopenia is due to an infectious process, sulfonamides may result in an increase in the leukocyte count and assist in curing the disease.

3. **Chancroid:** Powdered sulfathiazole applied locally twice a day to lesions about the penis may clear old Ducrey infections in four days.[12] Chancroid has been cured rapidly by the oral administration of sulfanilamide. In adults, one may employ 1.3 gm. (20 gr.) four times a day for five days, followed by 0.6 gm. (10 gr.) four times a day for an additional ten days. Fluids are restricted to about 1 liter per day.

4. **Dental Extractions:** From 0.15 to 0.3 gm. (2½ to 5 grains) of sulfanilamide may be packed into sockets following extractions. This has resulted in a markedly lessened incidence of postoperative infection, dry sockets and delayed hemorrhage.

5. **Gas Bacillus Infections:** Polyvalent tetanus-gas gangrene antitoxin should be given prophylactically (5500 units) or therapeutically (20,000 to 40,000 units or more intravenously) if indicated.

Sulfonamides have been advocated for prophylaxis and treatment of gas gangrene.

Crystalline sulfanilamide has been applied locally.[13] Penicillin is also of great value in gas gangrene, in addition to proper surgical measures.

6. **Impetigo:** A favorable response may be seen within forty-eight hours after local application of sulfathiazole powder or 5 per cent sulfathiazole as an ointment to the weeping lesions. Impetigo has been eliminated in some nurseries by putting sulfathiazole powder in the bath soap.

7. **Infected Wounds:** Sprinkling sulfathiazole powder into infected wounds or abrasions of the skin is of definite value (as most of these are infected by the staphylococcus).

Key reported that the implantation of sterile sulfanilamide or sulfathiazole powder in clean operative wounds was a safe procedure and decreased the incidence of postoperative infections. Such local use of the drug lessened the incidence and severity of infection in contaminated wounds whether these were sutured or left open.[53,55] In acute pyogenic infections, adequate drainage was established and sulfathiazole was used locally and by mouth in full doses.

Sulfanilamide has been used locally in compound fractures as a prophylactic measure with excellent results;[82] infections were eliminated by introducing from 5 to 15 gm. of powdered sulfanilamide after thorough débridement. The sulfanilamide was readily absorbed from the wound.

Adults with crushing injuries may be given sulfamerazine prophylactically 1 gm. every eight hours in an effort to prevent serious complications.

8. **Malaria:** Various authors have reported on the effectiveness of sulfonamides in the treatment of malaria; however, when atabrine or quinine are available, there seems to be no advantage of sulfonamide therapy in this disease.[49]

9. **Nose and Throat Infections:** Livingston has discussed the clinical use of sulfonamides in otolaryngology. Sulfonamides have almost eliminated mastoid surgery. Arnett[4] has incorporated *sulfadiazine in paraffin* to chew in cases of tonsillitis and pharyngitis. Approximately 0.3 gm. was incorporated in paraffin. Saliva levels of 20 to 25 mg. per 100 cc. were found four hours after chewing the paraffin containing sulfadiazine. The medicated paraffin sticks are given to patients after meals and are chewed until the next meal; two or three chews per minute seemed sufficient.

10 **Sulfonamide compounds** appear to have **no effect on** most virus diseases such as the common cold, chickenpox, smallpox, measles, poliomyelitis and epidemic influenza.

11. Succinyl-sulfathiazole (**sulfasuxidine**) and phthalylsulfathiazole have been found to be without value in the treatment of experimental trichinosis.[5]

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CHAPTER LX

PENICILLIN

Penicillin is without question the most powerful antibacterial agent known for the treatment of infections caused by Gram-positive microörganisms and the gonococcus and meningococcus. It has greatly lowered the fatality rates in these diseases, as well as shortened the total days of illness; it has been a great boon to mankind, and especially to injured soldiers. We still know next to nothing of its mode of action. We hope soon to know more of its chemical structure so that it can be produced synthetically on a large scale. The great need at present is for new antibacterial agents for the Gram-negative organisms and the viruses.

Penicillin is effective primarily against Gram-positive cocci and rods and against Gram-negative cocci. Effective sulfonamide therapy is available for many infections caused by these organisms. In some individuals, sulfonamides cannot be given because of sensitivity, profound anemia or renal insufficiency.

The amount of penicillin necessary to kill staphylococci is considerably greater than that required to kill other pyogenic cocci.[18] *Gonococci and meningococci appear to be the most sensitive,* while strains of streptococcus viridans require much larger doses (as for staphylococci).

Penicillin disappears rapidly from the circulating blood after intravenous injection. About 75 per cent of the injected material disappears from the blood stream within fifteen minutes after intravenous injection and 90 per cent disappears at the end of thirty minutes. The blood concentration reaches a maximum within fifteen to thirty minutes following intramuscular injection and remains more or less stationary for the next thirty minutes before gradually falling off. *Penicillin can be demonstrated in the spinal fluid at the end of twenty-four hours after intrathecal administration;* the same occurs in joints and the pleural cavity after injections into these. In normal persons, little or no penicillin is excreted in the cerebrospinal fluid but detectable amounts may be found in active meningeal inflammation.

Advantages of intramuscular injection of penicillin are that a higher concentration of the drug is maintained for a longer period of time even though the initial blood level is not as high as that obtained by intravenous administration; the technic of intramuscular injections is simple and can be carried out by any trained nurse, and the injections are better tolerated by the patient (inasmuch as these cause no resulting pain if the final dilution contains ½ per cent procaine).

For local treatment, penicillin has been given *intra-articularly, intrapleurally* and *intrathecally.* It has also been used in *irrigation of sinus tracts* and *deep wounds* and has been applied as *wet dressings* to wounds, directly

to the eye as baths, and instilled into a tracheotomy tube in acute laryngotracheitis in an infant.

Most of the penicillin which is used clinically is prepared *in vacuum packed ampoules* containing 100,000 units. This will dissolve readily in physiologic saline, 5 per cent dextrose or distilled water. *In most instances, the drug is now given intramuscularly with injections being made every three hours.* Because of the pain which follows injections of penicillin, it has been our custom to dilute the 100,000 units with 8 cc. of 1 per cent *procaine* and 8 cc. of physiologic saline, with a resulting solution of 16 cc. of ½ per cent procaine in saline, containing the penicillin. Fortunately, procaine does not inhibit the action of penicillin, and completely relieves any pain which may have followed the injection. Two cc. of the solution is then given intramuscularly (gluteal or deltoid muscles) every three hours day and night. In severe infections it may be necessary or desirable to use as much as 200,000 or even 300,000 units per twenty-four hours. If 200,000 units are to be given, each 100,000 units is dissolved in 8 cc. of ½ per cent procaine in physiologic saline; 2 cc. is given every three hours. The solution is kept in the ice box. To date, we have seen no instance of local infection following the intramuscular injections.

In **overwhelming infections**, such as staphylococcic bacteremia or subacute bacterial endocarditis, where it may still be advisable to give the drug by continuous intravenous drip twenty-four hours per day, each 100,000 units of penicillin is dissolved in a liter of physiologic saline, which may then be run into the vein at the desired speed, usually about 2 cc. per minute (when approximately 3 liters is being given per day, with a total of 300,000 units of penicillin). The penicillin may be diluted further, if desired. Some individuals have given intermittent intravenous injections every three hours.

The *three most important developments in the history of penicillin* were (1) *Fleming's* discovery; (2) the work of *Florey* and his associates who obtained penicillin in a relatively pure form and who demonstrated its clinical use, and (3) *American success in large scale production of the drug.*

The fascinating subject of penicillin therapy carries with it a great deal of drama. Penicillin has been known at least in name since 1929, when Alexander Fleming, now Sir Alexander Fleming, bacteriologist at St. Mary's Hospital in London, became interested in the colonial characteristics of bacteria as they age in growth on artificial media. He observed that one of the plates was contaminated with a mold. This threatened to ruin his experiment, but he kept the plate and noticed that as the colony of mold increased in size, the colonies of staphylococci decreased in size and changed in colonial characteristics. He isolated the mold and grew it; after ten days, the broth contained a substance which inhibited the growth of many bacteria.

Fleming studied the toxicity of this substance on the skin, conjunctiva and other tissues and found that this extract was relatively non-toxic. He was very much discouraged as the crude extracts were unstable and it was impossible to produce the substance in large quantities. Fleming recorded his observations and wrote that the substance could be used as an antiseptic, but that its chief use was to include it in cultures to suppress the growth of Gram-positive organisms and to allow the Gram-negative organisms to flourish. He did not permit his original observations to disappear and he used this material for several years as an aid in isolating the influenza bacillus in cultures.

Despite the recent introduction of penicillin, factors inhibiting bacteria were known forty-five years ago.[27,28] The medical profession has become interested in these in the past several years because of the advent of penicillin. Waksman and his associates have reviewed the literature on the antagonistic relations of microörganisms,[73,74] and Schmitt has recently presented a thorough review of the literature on penicillin until 1944.[67]

Penicillin was used first in the laboratory to prevent the growth of certain organisms in mixed cultures.[16,33] In 1932, Fleming noted that the main uses of penicillin were to isolate hemophilic bacteria (as B. pertussis and B. influenzæ) from streptococci and pneumococci; to demonstrate bacterial antagonisms; to separate Gram-negative cocci from other mouth organisms and to isolate a partially insensitive microbe in pure culture from a mixture of more sensitive organisms. Except for its use in the laboratory, it was not until 1940 that the drug was used in man.

The Penicillium mold which Fleming studied was a member of the great number which rot vegetables, fruit, grain and meat and which were so conspicuous in various stale, decaying matter that they constituted the common conception of "mold."[30] Fifteen years ago, the usefulness of this mold was confined to its ripening effect upon Roquefort and Camembert cheeses and its utilization in the laboratory in isolating one bacterium from another.[31,34,69] The mold was identified as the Penicillium notatum Westling and it was later found that only this particular mold had any antibacterial effect.

From 1932 till 1940 no important contributions appeared concerning the usefulness of penicillin. In 1940 and 1941, **Florey, Abraham, Chain** and others reported in detail on their investigations of penicillin.[2,13] Prior to this, **Dubos** had published his stimulating work.[21–25]

In 1939, a survey was made of antibacterial agents. Workers were able to produce an extract of penicillin from broth cultures and developed a method to extract a fraction which was non-toxic and non-pyrogenic in animals. Five or six patients were treated and it was suggested that penicillin would be a remarkable antibacterial agent for the treatment of infections in man.

Several years ago, **Florey** came to this country and worked in Peoria, Illinois. He consulted **Dr. A. N. Richards**, of the University of Pennsylvania, and, as a result, penicillin began to develop rapidly in the United States. To Dr. Richards, Vice-President in Charge of Medical Affairs of the University of Pennsylvania, more than anyone else, belongs the credit of developing penicillin in the United States and in stimulating interest and in bringing it to its present position in this country.

In 1942, there was sufficient penicillin available in the United States to

treat 40 patients; in 1943, enough of the drug was on hand to treat 500 patients. Now, sufficient penicillin is available to care for all of the casualties in our armed forces and much is available for civilian use. This is a truly remarkable achievement.[17,43,52]

Schmitt has reviewed the method of growth, media, yield, extraction and purification of penicillin.[67] These factors are not of great interest to the general practitioner. It has been possible to obtain penicillin in the form of a free acid and as various salts of calcium, sodium, potassium, etc. *Penicillin is very soluble in water.* Most of the salts of penicillin lose their activity rapidly on exposure to air. Boiling for a few minutes does not produce loss of activity but autoclaving for twenty minutes at 115° C. completely destroys it.

Various substances related to penicillin have been described. These include *penicillin B*,[65,72] *penatin*,[46–48] *notatin*,[74] *penicidin*,[4,5] *penicillic acid*,[26,56] *penicillamine*[3] and *various esters*, including methyl, ethyl, n-butyl and benzohydryl esters.[54,55] Various workers have reported that penicillin B, penatin and notatin were probably identical.[36] Penatin was found to be bacteriostatic in dilutions of 1 to 400 millions and also to be antagonistic to certain Gram-negative organisms such as the E. coli and Brucella abortus. It was reported that notatin was active against S. aureus, Streptococcus hemolyticus, pneumococci, Bacillus anthracis, E. typhosus and paratyphosus, Vibrio cholera and B. proteus vulgaris.

Factors which must be determined for penicillin therapy are *dosage*, the best *method of administration* and the *duration of therapy;* the study of more cases will provide an answer to these.

Herrell concluded that 40,000 units of penicillin per day was sufficient for the treatment of most cases of infection due to Staphylococcus aureus, gonococci, streptococci, actinomyces and micrococci.

Penicillin is usually furnished as the sodium salt—a brown or yellow powder, put up in sealed glass ampoules. This is extremely soluble in water, saline or dextrose solution. *One cc. of fluid readily takes up 10,000 units or more of penicillin.*

Penicillin—General Considerations

1. Obtained from the mold—Penicillium notatum.
2. *Fleming,* in 1929, isolated this mold and observed that it inhibited the growth of many bacteria; in 1940 and 1941, *Florey* and his associates reported their pioneer work on the therapeutic value of penicillin in man. *Dr. A. N. Richards* of the University of Pennsylvania was largely responsible for developing penicillin in the United States and bringing it to its present position.[64]
3. Penicillin is a powder, available in ampoules containing 5,000, 10,000, 25,000, 100,000 and 1,000,000 units. It is prepared as the sodium, calcium or ammonium salt.
4. Penicillin sodium or calcium are very soluble in distilled water, physiologic saline or 5 per cent glucose.

Penicillin—General Considerations—(*Continued*)

5. At present, the activity of penicillin is measured in Oxford units; a *unit* is the amount of penicillin which regularly inhibits the growth of a known inoculum (usually the Staphylococcus aureus). This may later be changed to measurement by milligrams.
6. *Cost* of penicillin varies widely, depending upon the manufacturer (from $1.50 to $2.50 per 100,000 units). It is hoped that with large scale production, this will be reduced 50 per cent or more.
7. Although solutions of penicillin were at first thought to lose their potency by standing at room temperature, recent evidence suggests that if necessary, solutions may be maintained at room temperature for four days without appreciable loss of potency. Ampoules are always stored in the refrigerator.
8. *Modes of administration:*
   (*a*) *Intramuscular*—preferred at present. As much as 50,000 units can be dissolved in 1 cc. of water or physiologic saline, if necessary; one day's supply is usually made up in advance so that 2 cc. is given intramuscularly every three hours day and night. Small amounts of procaine may be added to avoid pain following injections. Penicillin has also been given by continuous intramuscular infusion.[38]
   (*b*) *Intravenous*—dissolve penicillin in physiologic saline or 5 per cent dextrose. Dissolve half of the twenty-four hour dose in 1000 cc. The first 100 to 200 cc. is given fairly rapidly, following which the rate is reduced to 30 or 40 drops per minute. The second 1000 cc. is attached to the continuous intravenous system about ten hours later.
   (*c*) *Subcutaneous injections*—seldom employed, but if necessary, 100,000 to 200,000 units of penicillin can be given in 1000 cc. of physiologic saline or 5 per cent dextrose by hypodermoclysis.
   (*d*) *Intrasternal drip*—same preparation as for intravenous injections.
   (*e*) *Lumbar, cisternal and ventricular routes* may be employed if necessary. Recent evidence suggests that such routes may be unnecessary in meningitis.
   (*f*) *Injection into joint or pleural cavities and locally in wounds.* In joint or pleural cavities, from 20,000 to 50,000 units of penicillin may be given dissolved in 10 to 40 cc. of physiologic saline. *For local use on wounds,* employ an isotonic solution containing *250 units of penicillin per cc.*
   Penicillin may be given orally if properly buffered and taken on an empty stomach; rectal administration is ineffective because of inactivation by *penicillinase* produced by E. coli.
9. *Penicillin disappears from the circulating blood rapidly after intravenous injection,* 75 per cent of the injected material disappearing from the blood stream within fifteen minutes and 90 per cent disappearing within thirty minutes. *Following intramuscular injection, a higher concentration of the drug is maintained for a longer period of time,* although the initial blood level is not as high as that obtained with intravenous administration. It is excreted mainly by the kidneys.
10. *Toxic reactions*—extremely uncommon. These include urticaria (in from 1 to 3 per cent of cases), and occasionally, faintness, flushing, headache, diarrhea, and following intravenous administration—thrombophlebitis (locally) and chills and fever in some instances.

The solution of the sodium salt of penicillin, kept in the ice box in a closed container at a temperature of 5° C., loses very little if any potency in a month.[42] One should certainly not discard solutions of penicillin that have been kept for only a few days at ice box temperatures. If solutions of either the sodium or calcium salt are kept at room temperature, they lose potency rapidly. The sodium salt is not very stable even in the dry state at room temperature; the calcium salt can be kept at room temperature for approximately two months with very little loss of potency.

The *calcium salt* of penicillin has been found to be non-hygroscopic and is *relatively stable. Herrell was unable to detect any loss of activity of the calcium salt in the dry state in sealed ampoules stored at room temperatures for fifty-six days.*[40] Intramuscular injections of the calcium salt have resulted in no evidence of irritation of tissue.

Organisms Susceptible to Penicillin

1. **Gonococci**
2. **Meningococci**
3. **Hemolytic streptococci**
4. **Pneumococci**
5. **Staphylococci**
6. *Streptococcus viridans*
7. *Cl. tetani*
8. *Cl. welchii*
9. Cl. diphtheriæ
10. Treponema pallidum
11. Some strains of Actinomyces bovis[39]
12. B. subtilis
13. Vibrio septique
14. Vibrio comma
15. Cl. histolyticus
16. Cl. botulinum
17. B. sporogenes
18. B. œdematiens
19. B. sordelli
20. Lactobacillus
21. B. anthracis
22. Micrococci
23. Leptospira icterohemorrhagica
24. Spirillum minus
25. Psittacosis virus
26. Ornithosis virus
27. Streptobacillus moniliformis
28. Borrelia novyi (spirochete of relapsing fever)

The gonococcus and meningococcus are the only two Gram-negative organisms which are susceptible. The most susceptible organisms are placed in order beginning with number one (from numbers 1 to 10).

Organisms Not Susceptible to Penicillin

1. Enterococcus[12]
2. *E. coli*
3. *B. typhosus*
4. S. paratyphi
5. *S. dysenteriæ*
6. H. influenzæ
7. Pertussis
8. Cholera
9. B. proteus
10. *B. pyocyaneus*
11. Friedländer bacillus
12. B. melitensis (or at most, a very weak action)
13. *M. tuberculosis*[66]
14. B. pestis
15. P. tularensis
16. S. enteritidis
17. P. fluorescens
18. P. prodigiosus
19. Monilia albicans
20. Monilia krusei
21. Monilia candida
22. Plasmodium vivax[49]
23. Bacillus prodigiosus
24. Streptococcus fæcalis
25. Toxoplasma
26. Blastomyces

The calcium salt appears to be superior to the sodium salt for local therapy because of its greater stability and ease of handling. Bacterial infections may also be treated locally by sulfonamides, gramicidin and the synthetic quaternary ammonium compounds.

Penicillinase: Although Gram-negative bacilli are not susceptible to the action of penicillin, pyogenic cocci and the diphtheria group are very susceptible. When B. coli are crushed, an extract is produced which, upon incubation at 37° C. destroys the growth-inhibiting properties of penicillin on S. aureus. This extract evidently contains an inhibiting enzyme, which has been named **penicillinase.** Penicillinase apparently does not destroy penicillin, but merely inhibits its action. Another source of penicillinase is the paracolon bacillus. Penicillinase prepared in this latter method actively destroys penicillin.

Mode of Action: The action of penicillin is as a *bacteriostatic* agent[20] but under certain conditions it may be *bactericidal.*[44] It has been shown that **penicillin is unaffected by the presence of pus** *or by the products of tissue destruction.* It is active in the presence of large numbers of bacteria or in the presence of para-amino benzoic acid. It is not a detergent and is not hemolytic.[32] Its action is not inhibited by plasma or erythrocytes.[62]

Resistance to sulfonamides does not indicate resistance to penicillin.[15,57] Strains of pneumococci which are resistant to sulfonamides are susceptible to the action of penicillin.[53,71] Prolonged exposure is necessary to induce resistance of organisms to penicillin; resistance to sulfonamides is attained much more readily.

Penicillin	Sulfonamides
Less active orally than parenterally. Usually administered parenterally	Very effective if given orally. Given parenterally only occasionally (at the onset of severe infections or if not tolerated orally)
Effective locally	Effective locally, but local action persists for only short periods
Action is stronger (several times more potent)	Weaker acting
Influenced to only a minor extent by number of bacteria present	Effect definitely influenced by number of bacteria present
Active in presence of pus or hydrolytic protein products	Inactivated by pus or hydrolytic protein products
Low toxicity to cells	More toxic
Very few untoward complications	Reactions may be severe
Acts mainly against Gram-positive organisms (except meningococcus and gonococcus)	Acts also against B. coli and malarial plasmodia
Effective in treatment of early and late syphilis	No action in syphilis
Not affected by procaine	Inactivated by procaine

Penicillin Is Inactivated by:[60]

1. Hydrochloric acid of the stomach.
2. Colon and paracolon bacillus (sources of penicillinase).

Administration: Saliva does not inactivate penicillin. If penicillin is introduced by a duodenal tube (by-passing the stomach), penicillin is absorbed from the intestine. Unfortunately, hydrochloric acid in the stomach destroys the active portion of penicillin unless the latter is properly buffered. The drug cannot be administered by rectum because of its destruction by penicillinase in the colon.[1]

Following intravenous administration of penicillin, the serum level of the drug rises rapidly; traces of penicillin remain in the blood stream for one-half to three and a half hours after intravenous injection. The blood level rises rapidly after intramuscular administration, although usually not to as high a level as following intravenous administration. It is hoped that in the near future, newer and more efficient penicillin or related compounds may be developed or that the penicillin may be linked with substances (such as protamine) which will delay its absorption and spread this over a longer period. Recent work has shown that the simultaneous administration of para-aminohippuric acid (PAHA) will prolong the retention of penicillin in the body.[8] Penicillin is also absorbed following introduction into body cavities, and following intrathecal or intracisternal injection.[6,61] Penicillin has been given as the solid drug directly into a brain abscess without toxic effects.[58] It is not detected in the spinal fluid after intravenous administration in healthy subjects. Recent studies suggest, however, that penicillin administered intravenously or intramuscularly in adequate doses may be effective in the treatment of meningitis without supplementary intrathecal therapy as penicillin is found in the spinal fluid in patients with meningitis following its intravenous or intramuscular injection.

The degree of antibacterial action is usually proportional to the concentration of penicillin in the blood serum.[63] The leukocytes remain active in any concentration which is reached after intravenous administration of the drug.

It is not known where or how much penicillin is destroyed in the body. From 37 to 99 per cent is *excreted unchanged in the urine,* mostly within an hour after its injection. The concentration of penicillin in the blood stream is increased in individuals with renal failure.

Penicillin is considered to be non-toxic in doses far exceeding that required for therapeutic purposes. No deaths have been reported from penicillin itself.

Different organisms vary greatly in their sensitivity to penicillin and different strains of the same organism may show wide variations in susceptibility. In general, the **gonococcus and meningococcus are most sensitive** to penicillin, followed by the pneumococcus, hemolytic streptococcus (group A), Staphylococcus aureus, Streptococcus viridans, Staphylococcus albus and Gram positive rods. Some strains of staphylococci may be highly resistant. Whenever possible, one should test the sensitivity of the infecting strain to penicillin if prompt improvement does not occur.

Diffuse and phlegmonous lesions usually respond much more promptly than localized infections. Infections in well vascularized soft tissues usually respond to concentrations of penicillin which are apparently ineffective in relatively avascular hard tissues such as bone.

Very little is yet known concerning the minimal concentration of the drug that is effective in various types of infection.

Dose of Penicillin in Infections

1. No exact minimal dose has yet been determined. Doses given below are approximate. Although some individuals still prefer continuous intravenous administration of the drug, it appears that intramuscular injection is usually preferable except possibly in severe sepsis such as staphylococcic septicemia.
2. If *intramuscular injections* are employed, these are usually given *every three hours* day and night.
3. 100,000 units of penicillin can be dissolved easily in 16 cc. of solution of physiologic saline containing 0.5 per cent *procaine;* the latter eliminates any pain which might otherwise follow injections of penicillin.
4. *Injections may be made into the deltoid or gluteal muscles.* If the total dose for twenty-four hours is made up in 16 cc. of solution, 2 cc. may be injected every three hours. If desired, 200,000 units may be dissolved in 16 cc. of the above solution. The solution is always kept in the ice box when not in use.
5. **Pneumococcic infections:**
   (*a*) **Pneumonia**—50,000 to 100,000 units intramuscularly daily for two or three days (in divided doses every three hours).
   (*b*) **Empyema**—as in (*a*) plus aspiration of the pus and *instillation of 20,000 units intrapleurally* in 30 or 40 cc. of physiologic saline daily for two or three days.
   (*c*) **Meningitis**—as in (*a*) and in addition, *20,000 units daily intrathecally* for several days; if immediate improvement does not occur, a similar dose may be given daily intracisternally.
6. **Gonococcic infections:**
   (*a*) **Gonorrheal urethritis**—100,000 units in divided doses every three hours for one day; most cases will respond with cure with even half of this dose. *The urethra can be sterilized in 99 per cent of patients within twenty-four hours by the use of 50,000 to 100,000 units of penicillin given in divided doses every three hours intramuscularly.* The gonococcus is the most susceptible organism affected by penicillin.[35,41]
   (*b*) **Gonococcic arthritis**—100,000 units per twenty-four hours intramuscularly in divided doses for three or four days; if a large joint is involved (such as the knee), 10,000 to 20,000 units may be injected in solution directly into the joint cavity daily for two or three days.
7. **Meningococcic infections:**
   (*a*) **Meningitis**—20,000 units intrathecally daily for two or three days; similar quantity intracisternally if prompt response does not occur. In addition, at present, 100,000 units are usually given each twenty-four hours in divided doses intramuscularly every three hours for three or four days.
   (*b*) **Blood stream infection**—100,000 units daily in divided doses intramuscularly continued for forty-eight hours after the temperature has returned to normal.
   (*c*) **Arthritis**—same as dose in gonorrheal arthritis.

Dose of Penicillin in Infections—(*Continued*)

8. **Streptococcal infections:**
   (*a*) *Drain circumscribed infections* and apply penicillin locally as wet dressings, *q. v.*
   (*b*) In *hemolytic streptococcal systemic infections*, give 50,000 to 100,000 units of penicillin per twenty-four hours in divided doses every three hours intramuscularly, and gradually decrease the dose (but not the time interval of injections) as improvement occurs.
   (*c*) **In subacute bacterial endocarditis,** give *300,000 units per twenty-four hours, preferably by continuous intravenous drip*, or by divided doses every three hours intramuscularly for at least fourteen days. The blood stream will probably be sterile within twenty-four to forty-eight hours after institution of this therapy in those cases with strains of Streptococcus viridans which are not resistant to the action of penicillin. The dosage following this initial two weeks is not yet determined. If therapy is discontinued at this point, the disease usually manifests itself again. We have preferred to *gradually decrease the dose after the initial two weeks* of therapy, giving the penicillin intramuscularly following this. The dose is reduced to that level which can be given daily without reappearance of fever, general malaise or evidence of blood stream infection. We have found that a continuance of 50,000 units per twenty-four hours will often be sufficient to maintain this state. Some patients have been maintained on such doses for months. It is too early to know whether subacute bacterial endocarditis can be "cured" by penicillin therapy, or whether such therapy will have to be continued for very long periods, or possibly indefinitely to maintain the individual in a bacteria-free stage. Temporary sterilization of the blood stream and decrease in fever may occur and permanent improvement may occur in a few; only 3 of 55 cases treated with penicillin are alive after one year of study.

9. **Staphylococcal Infections With Bacteremia:**
   (*a*) *Drain the focus of infection.*
   (*b*) Give penicillin, by continuous intravenous drip, 150,000 to 300,000 units per day, reducing this to 50,000 units per day as the patient improves. With improvement, the drug may be given intramuscularly every three hours day and night in divided doses instead of intravenously. Some individuals prefer the intramuscular route from the beginning.

10. **Staphylococcic Infections Without Bacteremia:**
   (*a*) **Empyema.** Give intramuscular injections every three hours day and night (from 100,000 to 200,000 units per twenty-four hours); remove daily as much pus as possible by aspiration through a large bore needle, and inject following this from 20,000 to 40,000 units of penicillin in saline directly into the pleural cavity (each day for two or three days). This may be followed by a moderate amount of pleural pain which persists for five to twenty minutes.
   (*b*) **Acute osteomyelitis, acute abscess formation, acute phlegmonous cellulitis:** 50,000 to 200,000 units per twenty-four hours in divided doses intramuscularly every three hours until forty-eight hours after the temperature has returned to normal (usually approximately five to ten days of treatment).

Dose of Penicillin in Infections—(*Continued*)

   (*c*) **Chronic osteomyelitis** and **chronic abscess formation:** *remove all foreign material, sequestra and necrotic tissue;* give 100,000 to 200,000 units per twenty-four hours by intramuscular injection every three hours. Duration of therapy varies with the extent and severity of the process and the clinical response; usually at least five to nine days of treatment are required. In some cases, use *moist dressings dipped in a solution of 250 units of penicillin per cc.* of saline applied directly to the open lesions of osteomyelitis.
   (*d*) *Genito-urinary infections* of susceptible organisms: 50,000 units per twenty-four hours in divided doses every three hours intramuscularly for three to five days.
   (*e*) **Arthritis or meningitis**—penicillin intramuscularly—100,000 units per twenty-four hours in divided doses intramuscularly every three hours, in addition to local injection of 20,000 units daily for two or three days into the joint cavity or subarachnoid space, continued until the temperature has been normal for at least forty-eight hours. A smaller dose may then be continued intramuscularly for several days.

11. **Syphilis:**
   (*a*) **The primary lesion becomes dark field negative within twenty-four hours after the injection of penicillin. Present dosage schedule** for early syphilis is 40,000 units intramuscularly every three hours day and night for a total of seven and a half days (2,400,000 units total in the seven and a half days).
   (*b*) Penicillin is of value in neurosyphilis.

12. **Biliary Infections:**
   (*a*) Penicillin may be of assistance in biliary infections as a small amount of penicillin may be excreted in the bile.

13. **Actinomycosis:**
   (*a*) Some strains of actinomyces appear to be affected by penicillin. Further work is needed in this disease.

14. **Topical applications:**
   (*a*) Sinus tracts may be irrigated or wet dressings may be applied daily with solutions containing from 100 to 250 units of penicillin per cc.

15. **Burns or pyogenic infections:**
   (*a*) Wet dressings dipped in solutions of 250 units of penicillin per cc. of physiologic saline or penicillin in a neutral cream may favorably influence the course.[10,14]

16. **War wounds:**
   (*a*) Surgical intervention is usually necessary as penicillin alone does not control the polymicrobial character of the wounds.[70]
   (*b*) Penicillin is used intramuscularly, or intravenously and locally (*q. v.*).

Methods of Administration: **Oral** doses of penicillin are poorly absorbed unless properly buffered, because of the inactivating effect of the acid in the stomach; rectal administration is not effective because of its inactivation by penicillinase produced by E. coli.

Penicillin is not found in normal subjects in the spinal fluid, saliva or tears when given intravenously.

If penicillin is given **intravenously,** it is dissolved in physiologic saline,

unless it is undesirable to use the latter in that particular patient; in such instances, the drug may be dissolved in 5 per cent dextrose although this may produce venous irritation at times. Half of the twenty-four hour dose is usually dissolved in 1000 cc. of physiologic saline. From 100 to 200 cc. of the material are given fairly rapidly following which the rate of injection is reduced to 30 or 40 drops per minute. The second 1000 cc. is attached to the continuous intravenous system about ten hours later.

As much as 500,000 units of penicillin has been given in 100 cc. of water intravenously over thirty minutes and 40,000 units in 5 cc. of physiologic saline intrathecally without significant untoward reaction.

Subcutaneous injections are seldom given. Although it is not particularly desirable, penicillin can be given by continuous **subcutaneous infusion** (hypodermoclysis) with 100,000 to 200,000 units of penicillin being dissolved in 1000 cc. of physiologic saline or 5 per cent dextrose.

Penicillin has also been given by **intrasternal** drip in cases in which intravenous injections were extremely difficult or impossible.[9]

It has been demonstrated that **lumbar, cisternal and ventricular routes** are all practical methods of injecting penicillin in meningitis.

Such injections have been given in doses of from 10,000 to 20,000 units (diluted in saline so that there is not more than 5000 units per cc. of saline) without harm.[7]

Although it is not advised, penicillin (as the solid drug) has been introduced into a cerebral abscess without deleterious effect.

It is hoped that newer advances will result in a penicillin which is absorbed more slowly.

Because of the poor diffusion of penicillin, it has been extremely useful **locally in wounds** and infections of **joints** and the **pleural cavity.** *The powder itself or concentrated solutions are not employed topically because they are irritating.*[45] At present, most clinics employ **for local use** *an isotonic solution containing 250 units per cc.* This has been used by immersing gauze into it or using this as a spray. In this dilution, it may be used *in the mucous membranes of the nose or nasal sinuses.*[11] Clark and his associates prepared a cream for burns consisting of castor oil, lanette wax, penicillin and water.

A **snuff** of 94 parts lycopodium, 5 parts menthol and 1 part penicillin has been used in an ineffectual attempt to control the common cold, although this did reduce the number of pyogenic organisms.[19]

Assay: Penicillin cannot be standardized by chemical means but is assayed by its biologic effect; such assay is still cumbersome and unsatisfactory.

A unit of penicillin is the amount which under the conditions of assay gives an inhibition zone of 24 mm. in diameter—or the amount of penicillin which regularly inhibits the growth of a known inoculum (usually the Staphylococcus aureus). Sherwood, Falco and De Beer have described a *rapid, quantitative method for the determination of penicillin* in which the penicillin is placed on sterile, absorbent paper discs on the surface of inoculated nutrient agar.[68] The paper acts as a reservoir from which the antibiotic substance diffuses into the agar where it inhibits the growth of

the test organism, resulting in a clear zone surrounding the disc. The diameter of this zone of inhibition is proportional to the amount of penicillin present.

Failures of Penicillin Therapy

1. Inadequate treatment.
2. Late treatment.
3. The presence of an endocarditis with localization of bacteria on the heart valve.
4. Association with other debilitating diseases.

Failure of Penicillin Therapy: *Some strains of staphylococci are extremely resistant to penicillin;* this is not the case with hemolytic streptococci, gonococci, meningococci or pneumococci.

Penicillin is not effective against Gram-negative bacilli as some of the Gram-negative rods such as the *E. coli,* secrete an enzyme, penicillinase, which destroys penicillin.

The *B. pyocyaneus* seems to thrive in wounds despite penicillin treatment. Although this organism has a high nuisance value and may retard wound healing, it causes little real concern.

Penicillin has not proved to be of value in *malaria.*

The acquisition of resistance to penicillin is rare. Although penicillin may rid the body of pathogenic bacteria, the individual may die despite this. In severe meningococcal meningitis, in some instances death may occur even though penicillin has apparently rid the spinal fluid and meninges of all bacteria (as shown by necropsy studies). Maintenance of proper nutrition, fluid and salt balance and other factors are still of utmost importance despite the use of penicillin.

Toxic Reactions From Penicillin

1. *Fever and chills.* A febrile reaction coming on from the third to fifth day of a continuous infusion of penicillin may indicate contamination; the set should be changed.
2. *Thrombophlebitis* at the site of an intravenous injection.
3. Faintness, flushing of the face, headache, diarrhea.
4. *Urticaria.* This occurs in 3 per cent of cases if penicillin is continued for ten days or more. This usually appears between the fourteenth and twenty-second day after institution of therapy. This may be due to some impurities in the preparation or to actual hypersensitive reaction to the penicillin.
5. Transient increase in blood urea nitrogen.

It is generally agreed that penicillin is essentially non-toxic for man. Penicillin is devoid of toxic effects in concentrations far beyond those necessary for therapeutic purposes. Prolonged administration does not lead to development of sensitivity or intolerance.

There has been demonstrated no injury to kidneys, liver, bone marrow or brain following penicillin.

Untoward reactions from penicillin (some batches) which are thought to be *due to impurities include* chills, fever, eosinophilia (up to 30 per cent), pain at the site of injection, headache, faintness, flushing of the face, tingling in the testes, muscular cramps and femoral phlebothrombosis.

The commonest complications following penicillin administration are **urticaria, fever,** *transient increase in the blood urea nitrogen* and **thrombophlebitis.** Urticaria is the commonest single complication and may occur in approximately 5 per cent of cases. It may occur as early as the first day or as late as the fourth week of therapy; it has developed as late as nine days after treatment has been stopped. The wheals are distributed widely over the body. The face and eyelids become swollen and joint pains may occur in the hands with swelling of the fingers. Fever, abdominal crampy pain and eosinophilia may occur together with the urticaria. The course of the urticaria seems to be independent of cessation or continuance of treatment and usually continues for from three to five days. It is usually benefited by epinephrine or ephedrine.

Fever and abdominal cramps may be associated with urticaria. The fever does not often exceed 101° F. In a few patients, fever may be noted without urticaria during the first three to five days of treatment.

Thrombophlebitis occurs frequently at the site of constant intravenous injection, and may lead to chills and fever. This complication is often avoided by changing the position of the needle daily.

As penicillin has been further purified, thrombophlebitis has become less common following intravenous administration. *If continuous intravenous administrations are given, changing the needle each day seems to lessen the incidence of this complication.* Thrombophlebitis is a local affair and does not occur following intramuscular injection of the drug.

No significance has been attached to the slight transient rise in blood urea nitrogen which may occur occasionally during penicillin therapy. Albuminuria does not occur but hyaline cases are noted in the urine occasionally. Slight tubular damage has been reported in rats following injections of penicillin.

Staphylococcic Infections: Penicillin has a powerful effect upon **staphylococcus aureus infections**; sulfonamides have but little effect upon such infections. Penicillin is the best agent available today for staphylococcus aureus infections.[4]

Staphylococcus empyema has cleared following the local use of penicillin instilled directly into the pleural cavity after aspiration of as much pus as possible. From 20,000 to 30,000 units of penicillin instilled daily for several days usually results in a sterile pleural cavity.

Acute breast abscesses heal rapidly after incision and drainage, followed by the use of parenteral penicillin, from 100,000 to 200,000 units daily.

The value of penicillin in *hemolytic staphylococcus aureus bacteremia* and *pneumonia* is outstanding.[37,75] In severe infections, the blood may be cleared of bacteria in forty-eight to seventy-two hours, or less. Staphylococcus pneumonia was a common complication in the recent influenza outbreaks and in infectious tracheobronchitis in infants and children. *The mortality rate has been lowered from 80 to 15 per cent in cases of staphylococcus bacteremia with pneumonia by use of penicillin.*

Good surgical treatment must still be used together with penicillin in the treatment of purulent collections if good results are to be obtained.

Acute osteomyelitis of the sacro-iliac joint with staphylococcus aureus bacteremia has been followed by complete healing without recurrence following the use of parenteral penicillin (in doses of 100,000 to 150,000 units daily for several weeks).

In some instances, osteomyelitis in the acute stage can be controlled without surgical intervention by the use of penicillin. *Surgery is still necessary in almost all cases of chronic osteomyelitis,* together with penicillin.

Patients suffering from *multiple staphylococcus aureus subcutaneous abscesses* for several years have witnessed clearing of their skin lesions and healing of the infected sinuses following the administration of 100,000 units of penicillin daily for three weeks.

A striking feature in *chronic staphylococcus infections* after penicillin therapy is that the constitutional symptoms recede very rapidly and the individuals gain weight very quickly.

Penicillin has been used locally in the *preparation of the site for skin grafting,* as after incision or excision of a carbuncle in the neck in a diabetic.

The presence of a *Staphylococcus aureus endocarditis* in an individual with staphylococcus bacteremia renders the prognosis much worse even with the use of penicillin. Fortunately, some cases of survival have now been recorded following the use of penicillin in staphylococcus aureus endocarditis.

Another use of penicillin is in individuals with *staphylococcus aureus endarteritis,* where surgery may be necessary as in *patent ductus arteriosus with staphylococcus aureus infection.*

Streptococcic Infections: Hemolytic streptococci are much more susceptible to penicillin than are staphylococci. Blood cultures may become sterile in less than thirty-six hours following the use of penicillin in individuals with hemolytic streptococcus blood stream infection.

Chronic osteomyelitis of the lumbar spine and ilium, persistent for seven months—due to a hemolytic streptococcus and staphylococcus aureus—has been reported cured following the administration of penicillin.

Hemolytic streptococcal *brain abscess* and *meningitis* may also respond to penicillin. If the brain abscess is walled off so that penicillin may not reach it, it will not be sterilized by parenteral administration of penicillin. In localized infections, one should make every effort to drain the abscess and then treat it locally with penicillin.

In patients with hemolytic **streptococcal tonsillitis,** excellent prompt results follow the administration of penicillin; unfortunately, in some of these cases, the streptococci may reappear in the throat after five or six days and the patient may remain a carrier. Hemolytic streptococci are decreased in number so rapidly in the throat following the use of penicillin,

the hemolytic streptococcus being so susceptible to penicillin, that the body has little opportunity to develop antibodies against the streptococci. Thus, treatment of such cases should be carried on somewhat longer despite the marked local improvement in the throat.

In **subacute bacterial endocarditis,** large amounts of penicillin are needed over long periods of time in an effort to alter the course of the disease. At present, penicillin is the best agent available for bacterial endocarditis. Many strains of streptococcus viridans are susceptible *in vitro* to penicillin. In some cases, it has been possible to sterilize the blood stream and maintain this condition for months. Recurrences of blood stream infection have been reported in numerous instances when penicillin therapy is discontinued. The usual dose in this condition has been 300,000 units per day for a period of two weeks. Instead of discontinuing therapy at this time as has been the custom in the past experiments, therapy is now being continued with smaller doses, reducing this to 100,000 or less units per day, and maintaining that dose which is necessary daily to keep the blood culture sterile. It may be necessary to continue the drug for months or longer in an effort to "cure" this disease.

Pneumococcic Infections: **Pneumococci** are very sensitive to penicillin. Within twenty-four hours after the intramuscular, intrathecal and intracisternal injection of penicillin, in cases of *pneumococcic meningitis,* there may remain no pneumococci either in the blood or spinal fluid. One must remember that even though the spinal fluid and blood are sterilized, the patient may still die of other factors. Thus, it is still necessary to treat the patient as a whole and not to merely institute penicillin therapy and believe that this will do away with adequate nursing care, fluids, nourishment, etc. In such patients, 10,000 units of penicillin have been given intrathecally, 10,000 units intracisternally, and 100,000 units intramuscularly each day. **The mortality in pneumococcic meningitis has been reduced from 99 per cent to 40 or 50 per cent by the use of penicillin.** Unfortunately, a number of patients who recover have so much brain damage that they remain mentally defective. It is possible that the *combined use of sulfonamides and penicillin* may produce still better results *in pneumococcic meningitis* than when either is used alone.

The mortality rate in *pneumococcic pneumonia* treated with penicillin is approximately 4 per cent; in those who die, there is usually an associated disease. Penicillin is extremely effective in the treatment of pneumococcic lobar pneumonia. One can avoid thoracotomy in some cases of *empyema* by the aspiration of purulent fluid and instillation of penicillin into the pleural cavity. From 20,000 to 40,000 units of penicillin may be instilled into the pleural cavity daily for three or four days if needed to clear the infection.

Gas Gangrene: The pathogenic clostridia are sensitive to penicillin. Penicillin has been shown to be a most valuable agent in **gas grangrene** with the B. welchii if *used in combination with antitoxin and proper surgical treatment.* The Welch bacillus thrives in dead tissue and in ischemic lesions. **Penicillin will not remove dead tissues, nor will it neutralize tetanus toxin. One must use antitoxin and surgery in combination with penicillin in these cases.** Penicillin does prevent the growth of the Welch bacillus. In these cases, penicillin has been employed in doses of 100,000 units per day in divided doses; penicillin can be used together with sulfonamides.

Gonorrhea: The *gonococcus* is the most susceptible organism known to be affected by penicillin. *The urethra can be sterilized in 99 per cent of patients with gonorrhea within twenty-four hours by the use of from 50,000 to 100,000 units of penicillin* intramuscularly in divided doses every three hours.

Meningococcic Infections: Good results have been obtained following penicillin therapy in **meningococcus meningitis.** Patients with *meningococcemia* respond more quickly than those with meningitis; in the latter, treatment should be continued for at least six to ten days.

Surgical Infections: Lyons reported the results of *penicillin therapy of surgical infections in the U. S. Army,* summarizing the data from several general hospitals within the Zone of the Interior.

The powdered sodium salt of penicillin is too irritating for general use locally; the usual preparation used locally is diluted to contain not more than 250 units per cc. *The antibacterial activity of these solutions has been demonstrated in exudates for twenty-four hours after one local application. A single daily application is usually sufficient to keep the wound clean and free from pyogenic cocci.*

Penicillin has a definite place in the management of chronically infected gunshot fractures.

In **septic gunshot wounds,** penicillin is of definite value but foreign bodies or sequestra must be removed. Dramatically successful results may be achieved by a combination of penicillin, blood transfusions and conservative surgery.

Syphilis: *In early syphilis, spirocheta pallida disappear from surface lesions in from six to fifteen hours following penicillin therapy,* and the primary lesions heal within one or two weeks.[50]

The **optimum dose of penicillin in the treatment of early syphilis** is not yet known. The two schemes which are being tried at present include the 1,200,000 units in four days (in divided intramuscular doses every three hours) and 2,400,000 units in eight days.[51,56] It is probable that results will prove superior with the higher dose. Intramuscular administration of penicillin in early syphilis appears to be five to six times more effective than intravenous administration, judging by relapses. The present **Army-Navy standard dose of penicillin in cases of early syphilis** *is not less than 2,400,000 units in seven and a half days* (given preferably every three hours by the intramusular route).

Under penicillin therapy, **early meningeal neurosyphilis** shows an excellent response.[69] In simple *demented paresis,* 80 per cent show some improvement; 50 per cent of those treated improve 50 per cent. In *tabes dorsalis,* 20 per cent are improved approximately 50 per cent or more. Improvement also occurs in *meningovascular neurosyphilis, optic papillitis,* and recent *ocular nerve palsies.* In **symptomatic and asymptomatic neurosyphilis,**

present evidence suggests that the total course of penicillin be approximately 4,000,000 units (with the daily dose of from 200,000 to 320,000 units).

Penicillin has completely overshadowed *intensive arsenotherapy* which has been coming to the fore during the past decade and which was being advanced by newer knowledge of detoxicants (in an effort to lessen the toxicity of the combinations of arsenic and heavy metals). It is possible that future work may lead to *combined therapy with penicillin* and a short course of *mapharsen*.

Rheumatoid Arthritis.—We have treated 6 patients with rheumatoid arthritis with penicillin. This was given intramuscularly every three hours in a dose of 100,000 units daily for as long as four weeks; several of these patients were given 200,000 units daily for two weeks, followed by 100,000 units daily for two additional weeks. We did not note any striking improvement in the arthritis. The sedimentation rate was lowered in 1 of these patients. Several of the patients stated that they felt stronger and less tired after the course of penicillin. Our experience to date does not suggest that penicillin will produce rapid improvement in rheumatoid arthritis although further study must be done in this respect.

Miscellaneous: Penicillin was shown to be effective against *relapsing fever* produced experimentally in mice.

Tyrothricin: **Tyrothricin** is an extract, first isolated by Dubos, obtained from Bacillus brevis, a Gram-positive, aërobic, spore-forming soil organism. Tyrothricin consists of at least **gramicidin** and **tyrocidin**. Organisms that show some degree of susceptibility to tyrothricin include species of pneumococci, streptococci and staphylococci. This compound retards growth and causes lysis of the bacteria against which it is effective.

Tyrothricin is applied locally and is ineffective when given orally. **It should not be given intravenously.** It has been reported to be of value in the treatment of superficial indolent ulcers with predominantly Gram-positive organisms, mastoiditis, empyema and some other wound infections. It apparently has no effect unless it comes into direct contact with the organisms.

Tyrothricin is still in the experimental stage. It is applied locally after diluting with sterile distilled water to form an isotonic solution in a concentration yielding 500 micrograms of the drug per cc.

Gramicidin exhibits great selective activity as to microbial species and is most active, in general, against Gram-positive organisms although gonococci and meningococci are somewhat susceptible. *Gramicidin is very toxic for erythrocytes* and its hemolytic property rules out its use in the treatment of systemic infections.

Lysozyme: Work on **lysozyme**, a *potent antibacterial enzyme* found in most body tissues, was a factor in recreating interest in penicillin. Lysozyme occurs in high concentration in human tears, saliva and in egg-white.[29] Prior to the development of gramicidin, lysozyme was probably the best known of all the antibacterials of cellular origin. Other bacterials of cellular origin include *pyocyanin, actinomycin, streptothricin* and *bacteriophage.*

Streptomycin: **Streptomycin** is one of the newer antibiotic agents first described by Waksman and his associates in 1944. It has been reported to be active against a variety of bacteria.

Streptomycin was isolated from the genus Streptomyces, which includes the soil actinomycetes. It is effective against some pathogenic Gram-negative bacilli, including Eberthella typhosa and it also controls experimental infection in animals with Mycobacterium tuberculosis, Klebsiella pneumoniæ, Pasteurella tularensis and a mixture of anaërobic streptococcus and Proteus vulgaris. It has not been determined as yet whether streptomycin is effective in arthritis caused by these organisms. Streptomycin has also been of value clinically in acute and subacute non-specific ulcerative colitis.

A unit of streptomycin is the amount necessary to inhibit the growth of a strain of Escherichia coli in 1 cc. of broth or agar.

Streptomycin has been prepared in sterile ampoules for intramuscular or intravenous use following which it appears in the blood and urine and also for oral administration following which only traces appear in the blood and urine and most of the drug is excreted unchanged in the feces. The dose is still a matter of trial and error and varies from one million units upwards per twenty-four hours, being administered every three hours day and night.

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CHAPTER LXI

SUMMARIES OF CASE REPORTS

Gonorrheal Arthritis.—(*a*) Colored girl, aged nineteen years, illegitimately seven months pregnant.

(*b*) Chief complaint: Severe pain and swelling of the right knee for seven days; temperature, 101° to 103°; multiple joint pains for several days at the onset of illness.

(*c*) Past history: Joint pains for several months at the age of fifteen—diagnosed rheumatic fever; no evidence of cardiac damage at any time.

(*d*) Right knee moderately swollen; severe pain on slight motion; heart negative; other joints apparently negative.

(*e*) Blood count and urine normal; blood Wassermann reaction negative; Gc complement fixation negative.

(*f*) Aspiration of the right knee revealed cloudy yellow fluid, filled with polys; gonococci cultured from this fluid.

(*g*) Therapy: Sulfanilamide, 1.3 gm. (20 gr.), every four hours day and night (with 0.6 gm. [10 gr.] of sodium bicarbonate at each dose); after one week, sulfanilamide was decreased to 1 gm. (15 gr.) every four hours; after three additional days, this was decreased to 0.6 gm. (10 gr.) every four hours. Sulfanilamide was discontinued after sixteen days. Additional care: posterior molded plaster splint to the right leg; the splint was removed daily for slight motion (short of severe pain) to prevent formation of dense adhesions; heat to the knee, exercises, etc., after subsidence of the acute process.

(*h*) *Results:* Subsidence of fever within forty-eight hours; subsidence of joint pain when the posterior splint was applied. Almost full motion in the knee in three weeks. Weight bearing (asymptomatic) in four weeks.

Clinical Cure.—(Note: At present, penicillin is the drug of choice in gonorrheal arthritis, although sulfadiazine or sulfamerazine are effective in most cases.)

Subdeltoid Bursitis.—1. Female, aged forty years, with severe pain in the right shoulder increasing to its maximum within twenty-four hours. Pain unbearable, forbidding motion of the arm and sleep. Pain radiating down the upper arm in the region of the deltoid muscle and occasionally passing down to the hand. Arm held along the side of the body constantly because of pain. Unable to dress, comb her hair, brush her teeth, etc.

2. Similar attack one year previously, diagnosed "neuritis," treated with heat and rest; discomfort persisted for three months.

3. Very slight relief with heat in present attack. Some relief with large doses of dilaudid.

4. Examination: Excruciating tenderness over the subdeltoid bursa; no pain on anteroposterior motion of the arm; extreme pain on abduction

of the arm or attempting to place the arm behind the back. Diagnosis: Subdeltoid bursitis.

5. Roentgen-ray: Area of calcification in the region of the subdeltoid bursa.

6. *Treatment:* Roentgen therapy, 100 r produced increased pain; a similar dose on the following day gave almost complete relief of pain within four hours. A third similar treatment two days later was followed by absence of any pain; slight achiness persisted for an additional week. Shoulder exercises were instituted. Full painless motion of the arm was obtained in ten days. Roentgen examination three months later showed no evidence of calcification. (NOTE: Calcified deposits occasionally disappear spontaneously over a period of several months.)

Gout.—1. Male aged twenty-nine years. Intermittent attacks of joint pains with free intervals since the age of nine years; at that age he had acute swelling of the right metatarsophalangeal joint, responding to rest in bed.

2. Excessive beer drinker for the past three years; from 8 to 15 glasses per night.

3. Present attack: Acute exacerbation of pain in multiple joints, including swelling of the big toe; onset two days after appendectomy. (Postoperative arthritis suggests gout.)

4. Examination: Swelling and tenderness of the right metatarsophalangeal joint; pain and tenderness on motion of several other joints; right olecranon bursitis (also suggestive of gout); one tophus on left ear (monosodium urate crystals demonstrated microscopically); blood uric acid—6.5 mg. per cent. Diagnosis: Gout.

5. *Treatment:* Colchicine, 0.5 mg. ($\frac{1}{120}$ gr.), every hour for 7 doses produced relief of pain and very slight diarrhea; thereafter, colchicine, 0.5 mg. ($\frac{1}{120}$ gr.), four times a day for one week, and then three times a day for four successive days each week for one month. No joint pains since first day of therapy. Low purine diet. Advised to use alcohol only moderately or not at all.

Rheumatoid Spondylitis.—1. Male aged thirty years. Well until one year ago when he developed pain and tightness in the low back after a soccer game. Within several months—pains in both shoulders; swelling, redness and pain in the left big toe; swelling and pain in the right knee. Slight pain and swelling of the proximal interphalangeal joints of the hands; stiffness and soreness on turning the head.

2. Had visited various physicians during this year; treatment had included salicylates intravenously and by mouth, diathermy, iodides, manipulations of the back without anesthesia and reassurance—no benefit. Numerous chiropractic treatments with no improvement. Loss of 27 pounds in weight in the past year.

3. Examination: Blood-pressure 96/60; weight 106 pounds; thin, underweight, very poor posture, marked kyphosis, narrow subcostal angle, inability to abduct the left shoulder because of pain; right knee flexed with moderate amount of swelling; low back rigid—with almost no motion· feet flat; tenderness over the second and third metatarsal heads of both feet.

4. Hemoglobin—67 per cent; erythrocytes 3.6 million; differential count—normal; sedimentation rate very rapid. Roentgen examination: atrophic changes in the right knee, both hips, both sacro-iliac joints and left shoulder; calcification of the longitudinal ligaments in the dorsal and lumbar regions; grade 1 spondylolisthesis and spina bifida of the fifth lumbar segment. The joint spaces at the sacro-iliac joints were completely obliterated.

5. *Therapy:* Rest in bed for two weeks, using a firm non-sag mattress; positions to hyperextend the dorsal spine several times each day; high caloric, high vitamin diet; several small transfusions, 250 cc. each at intervals of three days; intensive physical therapy; warm bubble baths twice a week (98° to 102° for ten to twenty minutes each), followed by dry full pack for twenty minutes; thirty minutes of general massage each day; baking, massage and passive movements to the affected parts (left foot, right knee, left shoulder, back and pelvic region); roentgen therapy—to the entire spine and left shoulder; ferrous sulfate, 0.2 gm. (gr. iii), four times a day. With improvement, graduated exercises, first in bed, then in a sitting and finally in an upright position; corrective oxfords with anterior felt pads ($\frac{1}{4}$ inch); Taylor back brace with shoulder straps.

6. *Result:* Patient was markedly improved within six weeks; had gained 16 pounds in weight; swelling of the hands and knee had disappeared; no foot pain on walking; much less deformity of the back with very slight discomfort in the back; blood count within normal limits; no houlder pain—motion markedly increased. Mental outlook much improved.

Degenerative Knee Joint Disease.—1. Male aged sixty years, well until eighteen months ago when he developed pain below the right patella, which disappeared following massage. Six months ago, stiffness of the right knee developed; improved following use of heat lamp; improved at night while in bed. Worse in the past six months.

2. Examination: Weight 215 pounds; height 5 feet 8 inches; intra- and periarticular swelling of the right knee; creaking of the right knee; pain on motion of the right knee.

3. Laboratory data: normal complete blood count; normal sedimentation rate; Gc complement fixation test negative; blood Wassermann reaction negative; uric acid normal. Roentgen examination: Lipping and osteophyte formation, small amount of fluid in the right knee; impression-degenerative joint disease.

4. *Treatment:* Further questioning revealed that several months prior to the onset of pain in the knee, the patient was forced to seek gainful occupation; he obtained a position which required driving a large automobile for long distances daily. Patient was now given one week's vacation; daily periods of rest in recumbency; use of a Castex cylinder for the right leg during the night. Low caloric diet. Daily baking and massage

of the right knee. Exercises to improve body posture; attention to the feet (longitudinal arch supports, exercises and periods of rest).

5. *Results:* Marked diminution in pain in the knee, disappearance of effusion; improved sense of well-being following a loss of 15 pounds of weight within two months; occasionally slight stiffness of the knee. (Such therapy of course will not remove osteophytes but will materially improve the patient symptomatically and decrease the tendency to further damage.)

Rheumatoid Arthritis.—1. Female aged thirty-five years, who had been treated by conservative means for more than one and a half years. There was swelling and limitation of motion of the proximal interphalangeal joints, wrists, knees and ankles—together with flexion contractures of both knees. The patient was run down, tired, lacked pep.

2. *Laboratory data:* Very rapid sedimentation rate; moderate hypochromic anemia; negative: urinalysis, blood Wassermann reaction, Gc complement fixation test. Normal blood uric acid. Roentgen examination of affected joints revealed narrowing of joint spaces, demineralization of bones, soft tissue swelling.

3. *Treatment:* Rest in bed with Buck's extension applied to both lower legs in the line of the deformity—gradually increasing the weights as tolerated; high caloric, high vitamin, anticonstipation diet; splinting of hands and forearms in positions of physiologic rest (cock-up splints with the hands in dorsiflexion and the fingers in moderate flexion). Splints were removed each day and at least two or three painless motions were performed at each splinted joint. Muscle setting exercises were begun at once especially for the quadriceps and gluteal muscles. After several days, molded posterior plaster splints were applied from high on each thigh to the tips of the toes with the foot at a right angle and the knees practically straight. Two blood transfusions were given.

Paraffin dips were used daily for the hands and bubble baths were prescribed every second day, alternating with baking and massage of the knees, wrists and hands. Graduate exercises were begun after the first few days in bed and these were gradually increased within the limits of the patient's tolerance (avoiding fatigue and persisting pain).

Gold therapy was begun with intramuscular injections given each week.

The molded plaster splints were removed from the legs daily and several painless motions were performed at each joint splinted.

After a total of from three to four weeks in bed, the patient was fitted with caliper leg braces with orthopedic oxfords and got out of bed increasing amounts each day. Walking was performed gradually with the aid of a walker, increasing from fifteen minutes per day to several hours per day.

An optimistic attitude was maintained at all times and the patient was greatly encouraged by the elimination of her flexion contractures of the knees, gradual decrease in joint swelling and the return of her sedimentation rate to normal.

4. *Result:* The patient gradually gained in weight and strength. Joint pain and swelling gradually decreased and after ten months, there was only a slight residual swelling of the fingers with no discomfort in the knees except with marked change of weather.

The caliper leg splints could be discarded after five months, but were continued in use each night as splints to prevent the return of flexion contractures of the knees.

The patient was given two courses of chrysotherapy, with a free interval of eight weeks between courses. Complete blood counts and urinalyses were made each week before each gold injection was given. No toxic signs appeared. The patient maintained her asymptomatic state when last seen one and a half years after conclusion of the chrysotherapy.

Low Back Strain (Chronic).—1. Female aged thirty-eight years. First pregnancy eight years ago; pain in the low back following this, persisting off and on since then. Worse with each successive pregnancy and with the menses; aggravated by heavy household duties. No acute pain. No pain while in bed at night; no stiffness on arising in the morning. Overweight.

2. Marked tenderness over the lumbosacral joint and sacro-iliac joints; poor posture; flat feet. Laboratory studies—unimportant.

3. Therapy: Periods of rest in bed on a firm, non-sag mattress; use of a low back support with paraspinal supports; baking and massage of the low back several times each week. Instruction in good posture; postural exercises. Orthopedic oxfords; exercises for the feet. Low caloric diet.

4. *Result:* Complete relief of pain and discomfort in the low back.

Hysteria.—1. Male twenty-six years of age, with severe pain, tenderness and limitation of motion in all joints of both hands. No other joints in the body were involved. Unable to dress or feed himself. Duration of discomfort—three months.

2. Additional history unimportant except breaking off of relations with girl friend shortly before onset of joint pains.

3. Treatment during the past several months included salicylates, various injections by several physicians and tonsillectomy. No relief was obtained.

4. Examination: No swelling of affected joints; no muscular atrophy; extreme pain on motion of the hands except when the patient's mind was distracted. No fever.

5. Laboratory tests: Sedimentation rate normal; blood Wassermann reaction negative; blood count and urinalysis negative. Roentgen-rays of the hands—negative.

6. *Treatment:* Marked persuasion that all joint pain and tenderness would disappear following one or two electrical (static) treatments. Treatment was given only once.

7. *Result:* Complete disappearance of pain, tenderness and limitation of motion following several minutes of electrical therapy. No return of joint symptoms (follow-up two years later—patient "cured" of joint pains). Diagnosis—Hysteria.

Brucellosis (Undulant Fever).—1. Male thirty years of age, with pains in multiple joints intermittently for the past five months, low grade fever,

tachycardia, loss of 20 pounds in weight during this period and bouts of marked sweating.

2. Patient had received multiple drugs, diathermy to the affected joints twice a week and injections of sulfur; several teeth had been extracted.

3. Examination: Slight pain on motion of multiple joints, small effusion present in each knee joint, evidence of loss of weight and palpable spleen (2 fingers breadth below costal margin).

4. *Course:* Low grade fever, tachycardia, enlarged spleen and bouts of copious perspiration suggested diagnosis of Brucellosis; patient had drunk unpasteurized milk. Agglutination with B. abortus was positive in a titer of 1:800.

5. *Treatment:* Fever therapy with typhoid vaccine—first dose—25 million organisms intravenously; the resulting temperature reached 103° F. The temperature remained over 101° F. for eight hours. Four days later, 50 million dead organisms were given, with a similar result. The joint pain lessened markedly and joint swelling disappeared. Two additional injections of typhoid vaccine were given intravenously (100 and 150 million organisms respectively) at five-day intervals. Following subsidence of the fourth injection, the patient's temperature returned to normal, his pulse gradually fell to normal, joint pains did not recur and the patient felt much improved (although somewhat fatigued). Discharge from the hospital; placed on a high caloric, high vitamin diet. Within two weeks following his discharge, the patient declared that he was feeling fine; follow-up one and two years later—no return of joint symptoms. Diagnosis: Brucellosis.

Traumatic Arthritis.—1. Female forty-four years of age, with severe pains in the right knee for the past two months. No fever, loss of weight or tachycardia; no other joints involved. Patient slightly overweight.

2. No relief from drug therapy; some alleviation of pain by salicylates.

3. Examination: Negative except slight tenderness over the lower margin of the patella and slight pain on motion of the right knee; posture good; no flat feet; no varicose veins.

4. Laboratory tests: Blood count, urinalysis and sedimentation rate normal; blood Wassermann test—negative. Roentgen-ray of affected knee —slight roughening of lower margin of patella.

5. *Treatment:* Further questioning revealed that for the past three months, the patient had used the right knee to push against a heavy front door (which was very difficult to close), several times each day. This was now forbidden and patient rested one hour each morning and afternoon; hot fomentations were applied to the right knee for thirty minutes each morning. The patient improved markedly within one week; complete relief of joint pain in one month; no recurrence within a year.

Rheumatic Fever.—1. Boy twelve years of age; gradual onset of joint pains and swelling following acute tonsillitis (one week previously). History of rheumatic fever at the age of seven (with cardiac valvular damage). Both wrists were swollen and tender. Temperature 101° to 103° F., pulse 110 to 130, marked sweating, general malaise.

2. Laboratory examinations: Moderate secondary anemia with slight neutrophilic leukocytosis; very rapid sedimentation rate.

3. *Course:* Involvement of multiple joints in succession at intervals of three to five days with the inflammations subsiding in one joint as new ones become involved: knees, ankles, elbows, hips; loss of weight; poor appetite; listless. Examination showed the affected joints to be swollen, hot, red and tender; the heart was slightly enlarged with loud apical systolic and diastolic murmurs.

4. *Treatment*: Rest in bed with splinting of the affected joints; salicylates for relief of pain, 1 gm. (15 gr.) of sodium salicylate with an equal amount of sodium bicarbonate four or five times a day; liquid diet with forced fluids until the temperature returned to normal (which occurred within four days following onset of the salicylate therapy); decrease in salicylate dosage, 0.6 gm. (10 gr.) of sodium salicylate and of sodium bicarbonate t.i.d., for an additional week; ferrous sulfate, 0.2 gm. (3 gr.) t.i.d.; cod liver oil, 15 cc. (½ ounce) t.i.d.; cevitamic acid, 50 mg. t.i.d.; brewer's yeast—1 tablet q.i.d.; high caloric diet, when tolerated.

5. *Course:* Joint pains and swelling rapidly subsided following rest in bed, splinting and salicylates. Low grade fever (99° F.) remained for six weeks; sedimentation rate improved slightly after two months. The patient remained in bed. The pulse rate continued to be elevated somewhat (90 to 100). Two small blood transfusions were given (150 cc. each) one week apart. The sedimentation rate slowly returned toward normal; the pulse gradually fell to normal (six months after onset of attack). The pasty color in the cheeks disappeared and the patient gained weight and strength. Daily graduated exercises were performed in bed during the period of convalescence; massage was given each day to the muscles of the legs, arms and back. A small cradle was placed over the feet to prevent foot drop (from pressure of the bed clothes). When the sedimentation rate had returned to normal, the patient was allowed out of bed for increasing periods each day. There was no leukocytosis, fever or tachycardia at this time. Graduated exercises were performed until, three months later (*i. e.*, nine months following onset of attack), the patient was leading a normal life (except participation in competitive athletics). Tonsils were removed at this time with uneventful postoperative course; sulfamerazine is now given for several days prior to and following operation (in an attempt to prevent a rheumatic flare-up).

Joint Malignancy.—1. Male aged sixty-four years, with a history of fleeting joint pains and chronic low back pain, not severe, for one year; loss of 31 pounds (135 to 104) in the past six months; a stiff swollen left ankle for six months requiring him to walk with crutches; swelling and limitation of motion of both elbows for three months. All teeth were removed three months ago with no benefit to the joint pains.

2. Treated by injections and by a chiropractor without relief.

3. On examination, there was generalized adenopathy, markedly swollen, hot, tender elbow joints, swelling of the left ankle joint, beaking of the

nails, signs of pulmonary pathology, hepatic enlargement and moderate prostatic hypertrophy.

4. *Roentgen-rays* revealed metastatic lesions of bone (pelvis, left clavicle, ribs, bones of forearms and upper arms and legs) with an infiltrative lesion of the left lung extending out from the mediastinum. Bony lesions were multiple osteolytic patches, scattered diffusely.

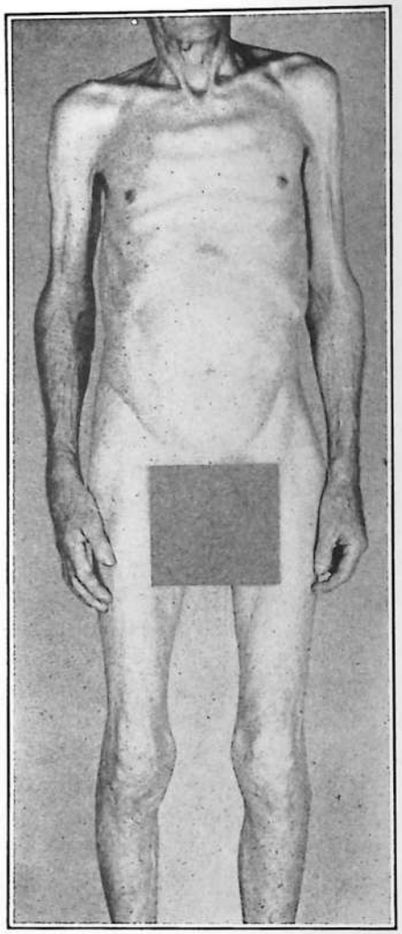

FIG. 326.—Metastatic carcinoma in bones at both elbows in a male, aged fifty-eight years. Patient had noticed marked loss of weight, fever, and bilateral swelling, pain and tenderness at the elbows. Multiple bones showed osteolytic process. Primary tumor was a bronchiogenic carcinoma. Other figures show close-up of elbow joint and roentgen-ray appearance; see Figures 327 to 328.

5. Other examinations: negative skull roentgen-ray; no Bence-Jones protein in the urine; rapid sedimentation rate; normal blood calcium, blood phosphorus, B.U.N., blood sugar; serum proteins 5.9 per cent; uric acid 3 mg. per cent; hemoglobin 57, red blood cells 3.4 million per c.mm., nor-

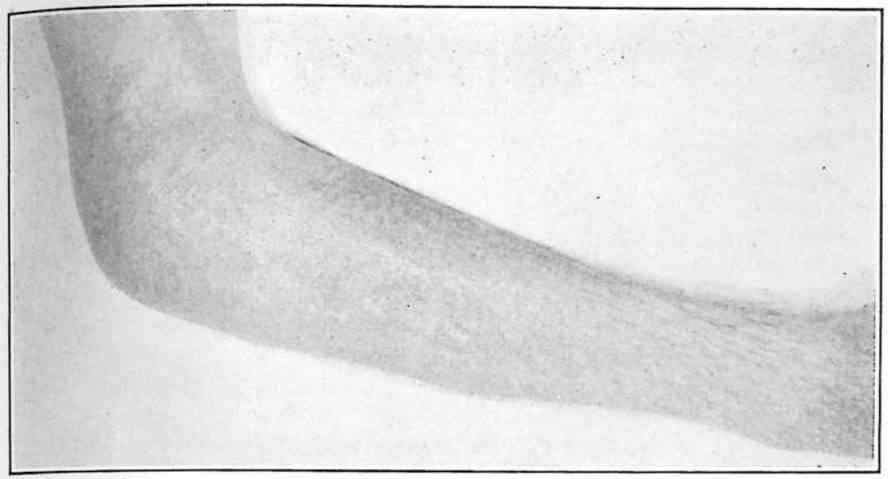

FIG. 327.—Forearm of patient seen in Figure 326, showing marked swelling of elbow joint; this was bilateral.

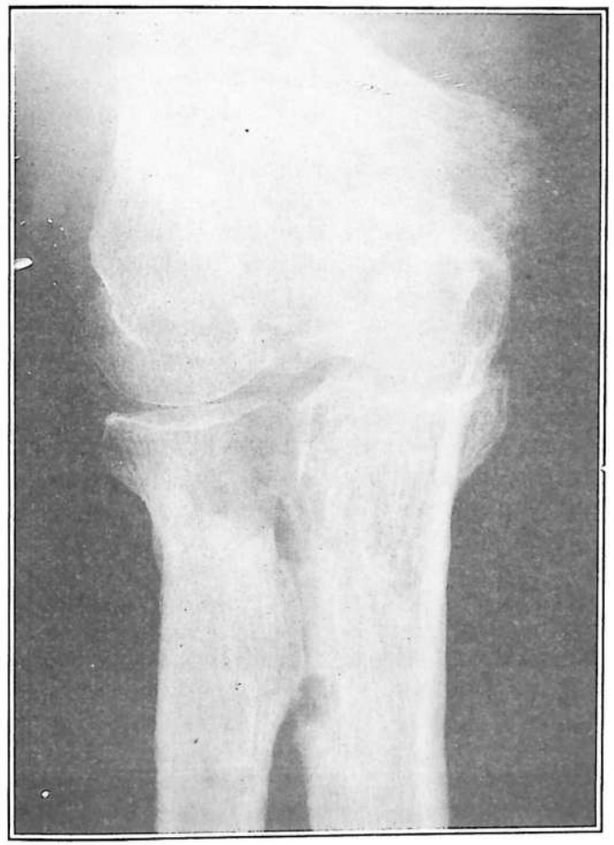

FIG. 328.—Metastatic carcinoma with marked joint swelling. There are multiple osteolytic lesions in the bones of the forearm and arm.

mal white count and differential pattern; gastric analysis normal; negative urinalysis; B.W.R. negative.

6. Final diagnosis: *Primary bronchiogenic carcinoma with metastases to multiple bones.* Marked emaciation of patient with lymphadenopathy and joint swellings suggested rheumatoid arthritis. See Figures 326, 327 and 328 for photographs of patient, close-up of an involved elbow joint and roentgen-ray of bones at an elbow joint.

Periarteritis Nodosa.—1. Female aged sixty-three years, with a history of stiff joints in 1933, relieved following tonsillectomy. In 1939, the patient again developed swelling of the wrists, hands and legs; in 1940 there was involvement in addition, of the ankles, knees and hips, with pain on motion, and sensitiveness of the legs to pressure. Dyspnea on exertion appeared in 1939; ankle edema was first noticed in June, 1940. From June to September, 1940, the patient lost 20 pounds.

2. Physical examination showed evidence of marked loss of weight (present weight 90 pounds), edema of the feet and legs, cyanotic, cold hands and feet, swelling and marked tenderness of the proximal interphalangeal joints of the fingers and tenderness to palpation of the feet and calves of the legs.

3. Roentgen-ray examinations of the hands and feet were negative; electrocardiogram was essentially normal. Normal values were obtained for blood: cholesterol, phosphorus, calcium, sugar, serum proteins, uric acid, urea nitrogen; B.W.R. was negative.

4. Because of the tenseness of the skin over the hands and feet, together with its shiny appearance and the subjective pain, a tentative diagnosis of sclerodactylia following years of Raynaud's disease was made. The patient was improved following a program of rest, high caloric, high vitamin diet and physical therapy.

5. The patient returned to the hospital in January, 1941, complaining of recurrence of pains in the legs and shooting and pulling pains in the palms. She had become weak, had lost her appetite, was dyspneic on exertion, and had a slight morning cough and nocturia. Examination now revealed the shiny, stretched skin over the hands and feet, swollen stiff fingers and toes, evidence of underweight, peripheral sclerosis, cheilosis and systolic murmurs over the precordium.

6. The patient developed a fever (ranging from 100° to 102° F.) which persisted off and on for two weeks. No known cause was found for this at the time. After some improvement on a high vitamin intake with forced feedings, the patient developed what appeared to be a diffuse bronchopneumonia and died within three days. Although she had diffuse patches of râles over her lungs and a Type 15 pneumococcus in her sputum, she showed no fever during her final week of illness.

7. Many other laboratory examinations done throughout the course of her illness were negative except: there was usually a persistent leukocytosis—from 18,000 to 25,000 white blood cells during the final four months of illness with from 68 to 85 per cent of polys; just before death, the total white count rose to 63,000 cells per c.mm. The urine during the final four months of illness usually contained a moderate amount of albumin, a few casts and a moderate number of white blood cells. Blood pressure averaged 150/90.

8. Autopsy revealed typical lesions of diffuse *periarteritis nodosa.* At no time during her course had the patient shown an eosinophilia. Although this disease is uncommon, it must always be considered in atypical joint pains with muscular tenderness, urinary findings, leukocytosis (often with eosinophilia), irregular inexplicable febrile paroxysms, nodules in the subcutaneous tissues especially along the course of medium sized arteries, gastro-intestinal complaints (crampy abdominal pains, diarrhea, vomiting), and rash (erythematous, purpuric or maculopapular). This disease is not often (about 25 per cent of cases) diagnosed antemortem; biopsies will greatly increase the percentage of correct diagnoses.

CHAPTER LXII

ARTHRITIS AND ALLIED CONDITIONS IN TROPICAL DISEASES

By Julia Morgan, M.D.,* and Bernard I. Comroe, M.D.

Arthritis is a well-known complication in a few tropical diseases, notably *bacillary dysentery, undulant fever, the fungous infections,* coccidioidomycosis, madura foot, and in *filariasis* due to Wuchereria bancrofti. *Arthritis may occur occasionally,* incidentally or as a result of extension by contiguity from the more typical lesions elsewhere, in a number of other diseases: the spirochetal diseases, *yaws, bejel, relapsing fever and rat-bite fever;* the fungous diseases, *sporotrichosis and histoplasmosis;* the nematode infections, *onchocerciasis* and *dracontiasis;* in *leprosy, smallpox, scurvy* and in *tropical ulcer.*

Arthralgias are common and *occur in* the rickettsial group in *typhus, Rocky Mountain spotted fever* and *tsutsugamushi,* in *trench fever, Schara boil, Oroya fever* and *verruga peruviana;* in the virus group in *yellow fever, dengue* and *sand-fly fever;* in the spirochetal diseases, *yaws, relapsing fever* and *Weil's disease;* in the *filaria infections* with Wuchereria bancrofti and Loa loa; in *onchocerciasis* and *dracontiasis*; in *hookworm infection;* also in scurvy, epidemic dropsy, *malaria,* African trypanosomiasis, and sometimes after antimony injections.

Pains in the limbs are described in a still larger group of diseases, frequently at the onset of the fever, and often associated with headache, backache or generalized body pains. In three groups of diseases, the spirochetal group, the rickettsial diseases and the viral diseases, the pains are severe and so regularly present as to be a helpful diagnostic aid. Most of the other diseases and conditions described may at times give pain in the limbs of greater or lesser degree.

Diseases which *seldom or never produce arthritis, joint pains* or other pains likely to be confused with joint pains are *amebiasis* and the *leishmania* infections.

Rheumatic diseases may differ somewhat as to their occurrence in temperate or in tropical regions. A number of observers find that although rheumatic fever may occur with its usual frequency among European residents in many tropical areas, in the native races in the tropics both rheumatic fever and rheumatoid arthritis are comparatively uncommon. Among Chinese patients in North China the situation with regard to arthritis and allied conditions seems to be analogous to that in our own southern states. Rheumatic heart disease with mitral stenosis is commonly seen, though acute attacks of rheumatic fever are observed infre-

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quently and a history of such an illness can be elicited in very few of the cardiac patients. Some experienced workers in India, Malaya, South China and Central Africa report never having seen rheumatic fever or endocarditis in a life-long experience. Chorea is reported to be rare among children in East Africa. However, among natives in the Gulf of Guinea region, "rheumatic arthritis" is said to be the commonest joint disease. Gonorrheal arthritis is commonly reported in Ceylon, India and China, though actual statistics are incomplete and difficult to obtain. Tuberculosis of joints is more common in the tropics probably because tropical regions are largely unsanitated and the incidence of tuberculosis there is, in general, very high.

In addition to the forms of arthritis seen in temperate climates, the tropics present a variety of joint affections associated with the distinctively tropical diseases, as well as bodily aches and pains of various sorts occurring in connection with such diseases which must be differentiated from true arthritis.

Some of the diseases discussed are found in the tropics only; others are exotic but not exclusively tropical while many occur in our own country but are commoner in tropical regions. Though they might be included in this last category, tuberculosis, typhoid and the venereal diseases are discussed in previous sections.

BACTERIAL DISEASES

Bacillary Dysentery.[7,11,21,49,50,62,77,84]—Arthritis is a well-known complication of bacillary dysentery.[30] It occurs in approximately 3 per cent of the cases, and is *more common in Shiga infections.*[83] It usually appears as the acute symptoms subside or within the first three to four weeks, but may develop up to three months or more.[5] As a rule, the *large joints are affected,* especially the knees, on one or both sides. One or multiple joints may be involved.[58,59,66] Next in order of frequency are the ankles, elbows, fingers, shoulders and wrists. Some observers place hip joints next in order to the ankles.

At the onset there is fever and pain, followed by effusion into the joint cavity of straw-colored, slightly viscid and usually sterile fluid. *Shigella organisms are occasionally cultivated from the joint fluid* and agglutinins are present, sometimes in higher titer than in the blood. Joint symptoms persist for a few days to six months but eventually there is complete recovery in practically every case.[3,31] Suppuration and resultant ankylosis have occasionally been noted.[35]

In the diagnosis one must beware of confusing dysenteric arthritis with that due to recent serum therapy. This complication of dysentery is not prevented by sulfonamides. Various authors have discussed the rôle of dysentery in Reiter's disease.[15,45]

In contrast to its occurrence with bacillary dysentery, *arthritis is seldom if ever found with amœbic dysentery.*[77,86]

Leprosy.—The *onset* of symptoms in leprosy may be accompanied by fever with generalized aching and pains in the limbs and back so severe that

the patient may be unable to move.[26,50,57,62,77] Early in the course of the disease there may be stiffness and numbness of the extremities and *pain and swelling of hands and feet.*

Chaulmoogra oil has been tried in the treatment of rheumatoid arthritis partly because of the supposed immunity of lepers to arthritis.[51] In a recent analysis it was found that 4.5 per cent of 425 patients at the Carville, Louisiana, Leprosarium had had a previous diagnosis of "arthritis," "rheumatism" or acute rheumatic fever.

Contractions and distortions of fingers and toes may occur fairly early in leprosy. Later, as infiltration of nerves proceeds, *trophic disorders of bones and joints* as well as of muscles become apparent. *Contractures* of the hand are likely to assume the main-en-griffe appearance, with function perhaps little impaired. Sometimes only the fourth and fifth fingers show contracture. In the feet, contractures are less common. There may be excruciating pains in the toes, especially the big toe. Trophic ulcerations may combine with secondary infection to penetrate and disorganize bones. By *x*-ray one finds partial absorption and necrosis of bones.

The pseudo-arthritis of lepers is mainly an osseous destruction; disintegration of the epiphyses and diaphyses follows destructive changes in the phalanges and metacarpal and metatarsal bones due to trophic changes. Eventually there may be spontaneous amputation of fingers or toes in whole or in part. Even without ulceration, the bones of the digits may become rarefied and absorbed so that bones and joints disappear and the finger nail may be found riding back on the knuckle. With certain joints, wrists and ankles especially, an actual *neuropathic process similar to a Charcot joint is occasionally seen,* with erosion and disorganization of cartilage and bone.

Bubonic Plague.—Prodromal pains occur in the back and limbs. As the mental state deteriorates, the pains are less evident. The site of the developing bubo is tender and painful even before the bubo appears. With a *bubo* in the groin the patient tends to lie with his *leg flexed;* if it develops in the axilla, the arm may be held abducted for comfort.[14,50,53,62,77]

Tularemia.—The onset is likely to be sudden with headache, backache and generalized body aches.[38,50,77] Pains may be fleeting or they may persist two to four weeks. They may settle in the shin bones. In the chronic disease localized pains may recur at intervals for as long as a year.

Undulant Fever (Brucellosis).—*During the course* of the disease rheumatic complaints are common, occurring in more than half of the patients infected with B. melitensis and in a smaller proportion of those infected with B. abortus.[12,14,49,50,62,77] Periarthritis or bursitis may occur.

The large joints are most often affected, the hip, knee, shoulder, ankle, wrist, fingers, toes, elbows, intervertebral joints, being involved in order of frequency. The onset is usually sudden with pain on active motion, local tenderness and heat without redness, along with swelling and effusion from which Brucella can frequently be cultivated.[32,34,36,60,72] The effusion may be thin or thick but is not purulent. Signs persist a day or more and may then subside promptly and completely. Another joint may be affected a day or two later. Signs may shift from joint to joint until in some cases almost all the joints in the body, including costosternal, costochondral, costovertebral and temporomandibular, have been involved, with a return finally, perhaps, to one of the original joints. Pain may be severe, especially in the vertebral or sacro-iliac joints. Lumbar pain or a stiff spine may be the outstanding symptom, the pain being worse at night and tending to assume a sciatic radiation.

In the sacro-iliac cases effusion is usually absent. These cases are especially prone to chronicity. *X*-ray of involved joints may show blurred contours of the joint margins. Mild or moderate pains may continue into convalescence and the joint may remain stiff for weeks or months without, however, becoming ankylosed.

Joint involvement in Brucellosis is discussed in detail in Chapter XLV (Miscellaneous Forms of Arthritis).

Cholera.—*Serofibrinous synovitis* has been reported; general aches and pains may occur in the muscles of the extremities, then spreading upward and increasing in intensity. Similar symptoms, usually in milder degree, may occur in severe acute dehydration from any cause, as in acute bacillary dysentery and acute food poisoning.[62,77]

SPIROCHETAL DISEASES

Bejel, the native, endemic, non-venereal treponematosis of the Euphrates Valley, *frequently exhibits bone changes.* There is periosteal or endosteal proliferation with or without areas of rarefaction.[37,52,65] Gumma-like lesions involving the medulla may extend to the end of the bone and joint and produce there localized destruction of an articular cartilage, with irregular erosions in the underlying epiphysis without involvement of the contiguous joint surface. Proliferative changes may surround the area of bone destruction. The ordinary osteochondritis as seen in syphilis does not occur in bejel, nor is joint neuropathy of the Charcot type found.

Yaws.—*Joint pains occur in all three stages of yaws.*[14,20,29,50,61,62,77] In the initial *incubation* period, along with slight fever, there may be headache, pain in the loins, rheumatic-like pains, worse at night, in the joints and long bones. In the *secondary* stage, six to twelve weeks after the primary lesion has appeared, there may again be irregular fever with headache and pains in the muscles, bones and joints. *In late yaws, bones and joints are affected in a fashion reminiscent of syphilis.* With diffuse osteitis and periostitis there are severe bone pains not always distinguishable from pain in the joints.

The *joints may be attacked* with *hydrarthrosis* in some, and in others, chronic infiltration of perisynovial or synovial tissues, tenosynovitis or chondrosynovitis or perhaps disorganization of the whole joint. *Ankylosis may result in a useless finger, hand, large joint or extremity.*

Symptoms are often far fewer than one would expect from the extent of the lesions. A less direct joint involvement is seen in the *bow contraction of the little finger,* or less commonly the fourth finger, that is typical of late

yaws. Primary contraction of the skin is followed by shrinkage of the joint capsule. *Subcutaneous nodules* are common in yaws especially about an ankle or leg. They may soften and lead to lesions of the bone and deformities with deep ulceration and scarring, especially where the bone is close to the skin. Starting from this, joints may be affected at the shoulder, elbow, wrist, ankle or feet. *Juxta-articular nodes* may appear placed symmetrically in relation to joints, as over the olecranon or the lower end of the femur. They are movable at first, later becoming fixed. *Bone and joint sequelæ are important in yaws and in some regions occur in 20 per cent of the patients.* Severe pains may be an outstanding feature of the disease.

Relapsing Fever.—The *onset* is usually with pains so severe as to constitute the outstanding symptom. They may be very persistent throughout the course of the fever and recur when the fever recurs; they may persist for a time after the fever subsides. There are headaches, often frontal, general body aches, gnawing pains at the nape of the neck, pains in the back and loins and in the muscles, bones and joints of the limbs.[14,38,46,49,50,62,74,76,77] There may be shin bone pains and severe pains about the knee. The pains closely resemble those of dengue. Though there is usually no joint swelling, actual *arthritis is reported in certain epidemics and polyarthritis may complicate convalescence.*

Infectious Jaundice or Weil's Disease.—The onset is usually sudden with headache, pain and tenderness in the back and limbs, sometimes agonizing in severity, especially in the calves. There may be joint as well as muscle pains. Severe muscle pain and tenderness are so characteristic as to suggest the diagnosis.[14,16,49,50,62,77]

Rat-bite Fever.—In the spirochetal form of rat-bite fever *severe joint pains often occur* with the fever.[10,14,50,77] True arthritis is uncommon in the naturally occurring disease, but has been found in the artificially induced disease, most often in the elbow and ankle. This is discussed in Chapter XLV (Miscellaneous Forms of Arthritis).

Other spirochetal diseases associated with pains in the muscles are seven-day fever where the onset is abrupt, accompanied by pain, and the pseudodengue of Java which gives severe pain especially in the legs.[77]

PROTOZOAL DISEASES

Trypanosomiasis.—In the early stages of African sleeping sickness there are headache, neuralgic pains especially in the joints, and cramps.[19,50,62,77] *Kerandel's symptom* may be present; this is a deep hyperesthesia with severe pain, often delayed following slight trauma over a bony prominence. Painful swelling may occur about the hands, feet or other joints, sometimes disappearing after a few days, but may reappear elsewhere. In the *later* stages of the disease there may be pains and cramps and a shuffling gait with weakness of the legs.

Leishmaniasis.—True arthritis is not part of the picture in any of the leishmania infections.[8,50]

Malaria.—With the high fever of a malarial attack the patient commonly experiences severe headache and *pains in the back and in the joints,*

sometimes suggesting dengue. Associated *muscle soreness* may be present. Rheumatic pains often occur even in the premonitory stage of the disease and in undiagnosed cases of malaria, headache, back pains and painful joints may be present and may not respond to any therapy until malaria is found and treated.[6,17,19,28,49,50,62,77]

Black Water Fever.—This may be accompanied by lumbar pain and headache.

VIRAL DISEASES

Dengue.—Prodromal symptoms may occur a few hours before the temperature rises, with twinges of pain in the joints of the limbs.[2,71] *As the fever rises* more generalized pains appear, especially severe in the head, back and limbs, the latter being referred to the joints. *All of the larger joints may be affected.*[14,49,50,62,70,77] Pain is perhaps most intense in the knees, hips and back, and may be agonizing, increased by the slightest active motion though unaffected by passive manipulation.

Actually *the painful points are in the tendinous insertions about the joints rather than in the joints themselves.* With remission of fever, the pains often recede, perhaps to recur in less severe form as the temperature again mounts. These *limb pains referred to the joints occur in over half of the patients,* mild in some epidemics, intense enough in others to merit the name "*break-bone.*"

The joints usually show no sign of inflammation. An occasional patient develops periarthritis of a knee or ankle which may give symptoms for months. The convalescent patient may continue to have pains for a month or more, worse in the morning and after a rest. Locomotion is difficult and walking is stilted, hence the name "dengue," a probable corruption of "dandy." The pains may persist in the hands and soles. Eventually complete recovery usually occurs.

Yellow Fever.—The onset is often sudden with severe headache and pain in the neck, back and limbs which may continue to increase for several days.[14,44,49,50,62,77]

Rift Valley Fever.—This is found only in Africa; like certain other viral diseases, this causes pains in the back, shoulders and legs.[50]

Psittacosis.—In the early stages there are generalized aches and pains.

Sand-fly Fever.—The symptomatology is similar to that of dengue.

Smallpox.—The onset is often characterized by generalized aches and pains, headache and aching limbs and extremely severe backache which may continue for several days. Actual serous or purulent arthritis with resultant ankylosis and deformity occurs occasionally.[14,43,50,74]

RICKETTSIAL AND ALLIED DISEASES

Typhus.—The *onset* or stage of invasion is often accompanied by headache, pains in the limbs, especially in the calves,[14,22,52,56,62,74,77] and severe back pains. During the *course* of the disease the patient often continues to be tormented by excruciating pains in the back and limbs. These are among the commonest symptoms of the disease. The pains may be

migratory. There is usually no redness or swelling of the joints, but purulent joint effusion has been observed in one instance. *During convalescence* pains in the legs and feet are common.

Rocky Mountain Spotted Fever and Tsutsugamushi.—Pains similar to those of typhus are usually present. Hyperesthesia may occur.[1,80]

Q Fever.—This may cause pains in the back and legs.[19]

Trench Fever.—At the *onset* there is frequently headache, backache, pain in the back of the neck and in the limbs; these pains may continue, shift or come and go.[14,39,41,82,85] The outstanding symptoms of the disease, however, are pains in the shin bones which are dull, aching, stabbing or boring, milder in the daytime but so severe at night that the patient may keep all his companions awake with his groans. They may regress during apyrexial periods, and recur when the fever rises again. *Hyperesthesia is marked* and may be so severe that even the weight of the bed clothes is intolerable. *Joint pains occur especially in the knees and ankles.* In the chronic stage lumbar pain is likely to persist. Any thought of malingering is excluded by the presence of fever and the fact the pains are worse at night.

Oroya Fever.—In the *chronic mild type* of this disease and in the *febrile type* joint pains are common; in the latter type, headache and bone pains also occur. In the *Verruga peruana* stage there is an onset with rather severe joint pains, especially in the knees, ankles and wrists, along with fever. Constitutional symptoms subside with the appearance of skin lesions.

VITAMIN DEFICIENCY DISEASES

Scurvy.—Pains in the legs are an outstanding symptom. Hemorrhage may extend from the calf or posterior thigh over the regions of the knee and ankle causing pain on motion of the joints. A knee and ankle may be edematous and painful without obvious hemorrhage. These cases of scurvy arthritis are found in a small proportion of cases in large epidemics.[55,77] Occasionally the knee, ankle or hip may be acutely painful without other local signs and pain sometimes subsides rapidly overnight.

In children the limbs may show the well-known subperiosteal hemorrhages with resultant swelling and tenderness and with violent pain on active or passive motion. *X*-ray shows the typical changes of periosteal detachment, more readily recognized after some calcium has been laid down; other findings may include subepiphyseal slipping or separation. *X*-ray changes are seen less frequently in adults.

Pellagra.—Pellagra may give muscle cramps, shooting pains in the limbs and backache, often with tenderness over the dorsal or lumbar spine. The gait is often peculiar, the legs being separated and bent and the patient shuffling along with the feet barely raised from the ground. The gait may suggest that of spastic paralysis.[50,62,77]

Beriberi.—The calf muscles are sensitive to pressure. There may be burning of the feet which is made worse on walking. This, added to the weakness or actual paralysis due to neuritis makes walking difficult.

Reduced or absent leg reflexes and tenderness on squeezing the calves are important diagnostic signs.

Osteomalacia.—Osteomalacia is common in the tropics in pregnant or lactating women. The patients experience severe pains in the back, pelvis and legs and may become completely bedridden. The softened bones become bent and deformed with weight bearing. Typical pelvic trifoliate deformity, along with bowing of the back and deformity of the legs may appear. The history of inadequacy of diet and sunshine in a pregnant or lactating woman directs attention toward the correct diagnosis, especially if the symptoms are repeated and increased in severity in successive pregnancies.

FUNGUS DISEASES

Coccidioidomycosis.[13,64,79]—*The acute respiratory phase* or the *inapparent form* of coccidioidomycosis may be followed by pains, often severe, in muscles and joints.[73,77] Any joint may be affected. "*Desert rheumatism*" is one term applied to this stage of the disease. In the *later* progressive stage there is sometimes a miliary dissemination which may involve bones and joints, particularly the ankle with abscesses, simulating tuberculosis of the joint. This is discussed in detail in Chapter XLV on Miscellaneous Forms of Arthritis.

Madura Foot.—This chronic inflammatory disease *usually attacks the foot* but may involve the hand, knee, thigh or buttock. There is marked swelling or deformity of the foot or hand with progressive disintegration of all the tissues including the bones and joints, and with granulomatous nodules on the surface with sinus openings. Amputation is the recognized treatment but penicillin is now being tried.

Sporotrichosis.—Sporotrichosis occasionally involves muscles, bones or joints with its gummatous or ulcerative lesions.

Actinomycosis.—Actinomycosis of the spine is not uncommon.[40,54] (See Chapter XLV on Miscellaneous Forms of Arthritis.)

Histoplasmosis.—Histoplasmosis may affect a joint, producing a chronic painful swelling, with thick purulent joint fluid. The joint tissues are invaded by large mononuclear cells containing many of the causative fungi. By *x*-ray, bony erosion is evident. This is discussed in Chapter XLV on Miscellaneous Forms of Arthritis.

HELMINTH INFECTIONS

Filariasis.—In filariasis due to Wuchereria bancrofti, the filarial invasion is often accompanied by synovitis. Synovitis and arthritis may occur in the hip or knee; in some cases a purulent effusion is seen.[49,50,67] Surgical interference may be indicated. Where the hip joint is involved, removal of the inflamed iliac glands may afford relief. Fibrous ankylosis often results.

Loiasis.—In loiasis the first symptom may be *transient swellings about the joints followed by stiffness.* The swellings may be painless or there may

be aching and burning along with the swelling in an ankle, arm, hand or wrist.[33,77]

Onchocerciasis.—In onchocerciasis acute arthritis has been described, with microfilaria in the joint fluid.[50,77,81] More commonly one finds small or large subcutaneous tumors (even up to the size of a pigeon's egg), often near the joints, in the popliteal space, knee, over the trochanter, in the axillary or shoulder region, about the elbow or over the vertebral column. In the incipient stage these nodules may be quite painful, those near joints being the more easily inflamed. If the nodule acts as a purulent focus, there may be pain and fistula formation with scarring and loss of function; the trochanters are a frequent site for such scarring.

Dracontiasis.—In endemic areas, a bullous blister or sinus in the foot or leg is almost certainly due to the **guinea worm**, which often presents at the ankle, knee or foot.[39] There may be more than one sinus. When the worm presents near a *joint*, that region becomes red, swollen and painful, and locomotion is difficult.[19,24,50,62,77] *Synovitis* and *arthritis* often develop, with a serous or purulent joint effusion. This is usually sterile.

Fibrotic changes and contraction of the tendons, as the Achilles tendon or the hamstrings, may occur, with or without bony ankylosis. *Permanent deformities may result* from secondary bacterial infection, especially after long recumbency. Such bacterial infection of a joint may appear before there is any sinus tract. *Arthritis may result from invasion of the joint by the worm itself or the nearby presence of a calcified worm;* a live worm burrowing deep between fascial planes may give pains without actual joint involvement.

Hookworm infestation is sometimes accompanied by joint pains.

Other helminth infections that cause body pains, often more generalized, are:

Trichinosis, where the beginning of the period of larval migration into muscles is often associated with mild or severe muscle pains, sometimes with tenderness or periorbital edema. Pain on mastication may be a symptom. An erroneous diagnosis of "arthritis" or "rheumatism" is frequently made.

Cysticercosis.—Cysticercosis may be accompanied by *myalgias* during the active life and development of the parasite.

In **schistosomiasis** there may be toxic symptoms at the end of the incubation period including persistent headache and pains in the back and limbs.

There are several *disease entities of unknown etiology* where joint involvement may be present or be simulated:

Tropical ulcer usually attacks the lower part of the leg or the foot. An ulcer on the dorsum of the toes may burrow down to involve tendons or even a joint cavity but those on the plantar surface seldom extend deeply.[9,23,50,62]

Pyomyositis, of unknown origin, is especially found on the African Gold Coast, and usually attacks the quadriceps or gluteal muscles.[25,77] It must be distinguished from cellulitis and septic arthritis.

In **Ainhum** a circular groove appears at the base of one or both little toes, less often the fourth toes, deepens gradually and in the course of several years proceeds to the severance of the toe, usually without ulceration or pain.[47,50,77] *X*-ray shows absorption of the ungual tufts and of other parts of the bones, especially in the neighborhood of the constriction.

Big Heel, occurring on the African Gold Coast, begins with tenderness in one or both heels with enlargement of the os calcis, resulting in marked pain on walking.

Lathyrism.—Lathyrism, in India, Africa and elsewhere, has symptoms similar to spastic paralysis with ataxia. At the onset there are pains in the back and weakness of the legs.[50,75,77] The disease is thought to be due to eating bread made of flour from the chick-pea, Lathyrus sativus, or possibly a contaminating weed, Vicia sativa. Deleterious effects are evident only when a considerable amount is consumed.

Epidemic Dropsy, somewhat similar to beriberi, thought to be caused by toxic properties of oils used in the diet, has as its prominent symptoms aches in joints, muscles and bones.

Drugs.—Various drugs used in the treatment of certain tropical diseases may, on occasion, give rise to generalized pains or pains in muscles or joints:

The milder symptoms of **aspidium** poisoning include muscle cramps.[75]

Several of the drugs used in treating African trypanosomiasis have frequently caused headache and generalized body pains, *viz:* **antrypol** (the English equivalent of *Bayer 205*,[18,27,48]), **tryparsamide,** or the combined treatment of antrypol followed by tryparsamide.[48] With these drugs the reactions are usually mild but sometimes severe enough to necessitate discontinuance of treatment.

In the treatment of kala-azar or oriental sore with **antimony** preparations intravenously, immediate reactions often include a feeling of *muscle stiffness.* Toward the end of a course of therapy *severe pains in joints and muscles with cramps in the calves* may occur four to five hours after an injection and may last as long as twelve hours.[50,69,77] Such pains must be regarded as danger signals. They are less common with the less toxic pentavalent antimony compounds than with the trivalent sodium or potassium antimony tartrate.

In this connection one must mention the pain in the legs so bitterly complained of by many *opium addicts when the drug is withdrawn.*[75] Such pain sometimes persists for days or weeks after all other symptoms of craving have disappeared.

The sulfonamides are finding their place of usefulness in treatment in certain tropical diseases and the occasional appearance of joint pains (sulfatoxic arthritis) due to their use must be mentioned.

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CHAPTER LXIII

MISTAKES IN THE DIAGNOSIS AND HANDLING OF PATIENTS WITH ARTHRITIS AND ALLIED CONDITIONS

THIS chapter presents in brief summary form some of the more common mistakes of omission and commission in the diagnosis and handling of arthritic patients. In all instances, the subjects are discussed in detail in the preceding chapters and those individuals desiring more detailed information upon any particular subject mentioned in this chapter should refer to the Index. Although opinions concerning the handling of arthritic patients vary considerably, most rheumatologists are in general agreement concerning the mistakes mentioned in this chapter.

MISTAKE 1 *is to be pessimistic concerning the outlook in the arthritic patient.* The physician must manifest an honest degree of optimism in the handling of these patients.

MISTAKE 2 *is to tell the arthritic patient that he will be much improved within a few days or weeks.* The treatment of rheumatoid arthritis at present requires months and years of careful management.

MISTAKE 3 *is to think that there is yet a specific remedy (pill or capsule) for the cure of rheumatoid arthritis.* As yet, such a drug has not been reported.

MISTAKE 4 is to consider merely that the patient has "arthritis." There are many different types of arthritis, some of which are very amenable to therapy within short periods of time.

MISTAKE 5 is the *failure to take a complete history, perform a careful, complete physical examination and a few simple laboratory tests* on the patient with joint disease. *Rheumatoid arthritis may be simulated by many diseases* including other forms of arthritis as well as syphilis, hypothyroidism, malingering, leukemia, Raynaud's syndrome, scleroderma, Parkinsonism, trichiniasis, periarteritis nodosa, lupus erythematosus, dermatomyositis and many others.

MISTAKE 6 is *unwillingness to alter the course of therapy* in a patient. Do not hesitate to alter treatment from time to time when indicated.

MISTAKE 7 is *to promise the patient with rheumatoid arthritis a cure.* Never promise a cure in rheumatoid arthritis; however, the patient can usually be told that he has about nine chances in ten of being definitely benefited and about seven chances in ten of being markedly improved.

MISTAKE 8 is to treat only the arthritis. *One must always treat the patient, and not only his disease.*

MISTAKE 9 *is to think that the patient with rheumatoid arthritis will have constant improvement without interruption;* usually, such is not the case. With intelligent handling of the individual, the general trend in most cases will be favorable but with changes in weather, emotional upsets and other unknown factors, exacerbations and relapses of the disease may occur despite every effort to avoid these.

MISTAKE 10 *is to neglect the patient's comfort and general health.*

MISTAKE 11 is to expect dramatic changes in the patient with rheumatoid arthritis within a short period.

MISTAKE 12 is to become discouraged or to allow the patient with arthritis to become discouraged.

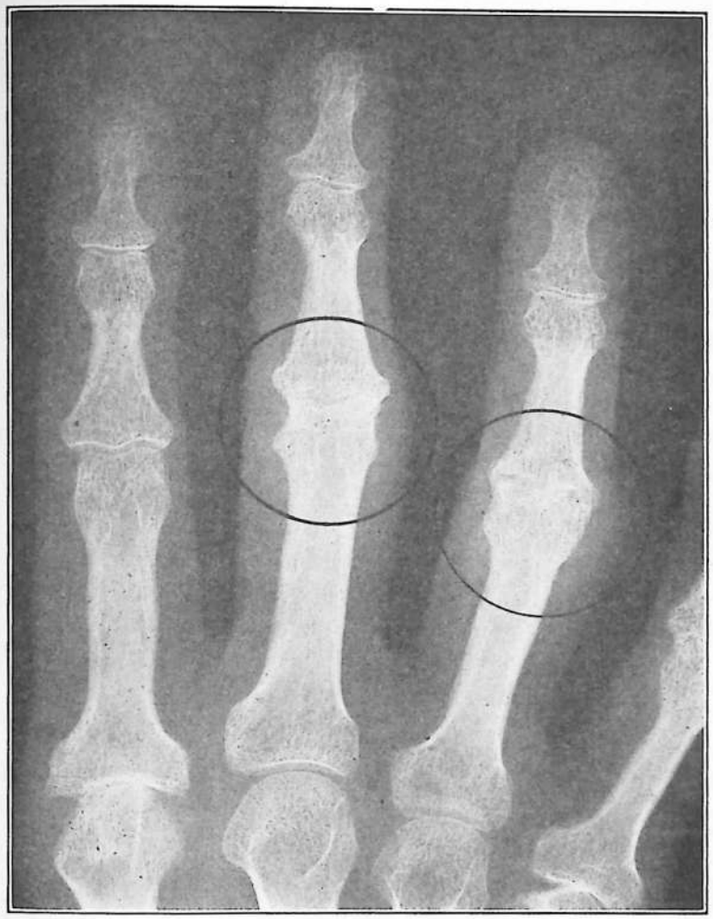

FIG. 329.—Rheumatoid Arthritis. Note narrowing of joint spaces and demineralization

MISTAKE 13 *is to depart from recognized remedies.* If the physician departs from recognized remedies or methods of treatment or diagnosis, he does so at his own risk and peril and if such departure results in injury to the person treated, the physician is liable for the damages no matter how good his intentions may have been.

MISTAKE 14 is to omit the *value of psychotherapy* in most of these patients.

MISTAKE 15 *is to depend upon analgesics to relieve the discomfort of rheumatoid arthritis.*

MISTAKE 16 *is to keep the rheumatoid arthritic in bed without adequate massage and muscle setting exercises.* If the patient lies in bed like a lump of putty much harm can be done.

MISTAKE 17 is to avoid rest of the joints in rheumatoid arthritis. The joints must be rested in favorable positions and good body mechanics must be preserved.

MISTAKE 18 is to forget that *marked loss of weight* may occur in rheumatoid arthritis. Conversely, one should be alert to the possibility of concomitant disease in the individual and not necessarily attribute the marked loss of weight to rheumatoid arthritis without a careful history and examination of the patient.

MISTAKE 19 is to forget that *rheumatoid arthritis may have an acute onset in about 8 per cent of cases,* so that it may be difficult to differentiate this from rheumatic fever without the passage of considerable time in some instances.

MISTAKE 20 is to forget that low grade *fever frequently accompanies rheumatoid arthritis;* this may be as high as 102°, 103° or more in some cases.

MISTAKE 21 is to forget that the *joint pain and swelling in rheumatoid arthritis may be temporarily relieved by pregnancy or jaundice.*

MISTAKE 22 is to advise the rheumatoid arthritic to become pregnant in an effort to relieve the joint phenomena inasmuch as the arthritis returns in full intensity in most cases within six weeks following delivery.

MISTAKE 23 is failure to remember that the *spleen may be palpable* in as many as 10 per cent of patients with rheumatoid arthritis.

MISTAKE 24 is failure to remember that *marked muscular atrophy may occur* with rheumatoid arthritis; many of these patients have been misdiagnosed as primary muscular diseases.

MISTAKE 25 is failure to recall that *generalized lymphadenopathy* may occur in patients with rheumatoid arthritis.

MISTAKE 26 is failure to remember that *valvular lesions occur in as many as 35 per cent or more of patients with rheumatoid arthritis* who come to autopsy and that clinical evidence of valvular heart disease may be found in 10 per cent or more of patients with rheumatoid arthritis.

MISTAKE 27 is failure to remember that pain referred to the medial aspect of the knee and anterior thigh may be the first symptom of arthritis of the hip joint.

MISTAKE 28 *is to think that any special vaccine will cure various forms of arthritis* (except Brucellosis). Carefully controlled experiments fail to show the specific value of any vaccine in arthritis, although the psychologic value of "any injection" is unquestioned.

MISTAKE 29 is in believing there is any specific value in *sulfur, chaulmoogra oil or bee venom* in rheumatoid arthritis. *The psychic effect of any harmless material is undeniable*; from our experience, we can not recommend any of these substances in the treatment of rheumatoid arthritis.

MISTAKE 30 is to forget that the wave of enthusiasm for sulfur therapy which was so popular ten to fifteen years ago in the treatment of various forms of arthritis has now died down to a trickle.

MISTAKE 31 is to consider that the cystine content of the nails indicates a sulfur deficiency in rheumatoid arthritis.

MISTAKE 32 is in believing that there is any sulfur deficiency or abnormality of sulfur metabolism in the patient with rheumatoid arthritis.

MISTAKE 33 *is the belief that any vaccine will straighten out chronically deformed ankylosed joints.*

MISTAKE 34 *is to give narcotics* except as an emergency measure in patients with arthritis. If *narcotics are needed over long periods of time, the diagnosis should be checked.*

MISTAKE 35 is to feel that *iontophoresis* is of any specific value in the treatment of arthritis. Most rheumatologists are not impressed with the results from iontophoresis with histamine or choline preparations in the treatment of rheumatoid arthritis.

MISTAKE 36 is to forget that to date, the use of *chaulmoogra oil* in the treatment of rheumatoid arthritis has been entirely empirical.

MISTAKE 37 is to believe that *parathyroidectomy* will relieve the rheumatoid arthritic. There is no proved relation between arthritis in any form and primary hyperparathyroidism and *parathyroidectomy is not indicated in the treatment of any form of arthritis.*

MISTAKE 38 is to believe that *splenectomy* is of therapeutic value in rheumatoid arthritis.

MISTAKE 39 is to believe that *foreign protein therapy* is of any lasting value in the great majority of patients with rheumatoid arthritis.

MISTAKE 40 is to attribute value to *snake venom* in arthritis. *There is lack of scientific evidence for the use of any venom in the treatment of rheumatic conditions.* No snake venom has an established place in the treatment of arthritis.

MISTAKE 41 is to attribute specific value to *iodides* in the arthritic armamentarium. The rôle of iodides in most forms of joint disease is very questionable.

MISTAKE 42 is to consider that *crude liver extract* by injection will cure rheumatoid arthritis.

MISTAKE 43 is failure to take an adequate history concerning *drug ingestion* before the onset of joint pain inasmuch as the latter occasionally occurs after the ingestion of various drugs (sulfonamides, bismuth, phenobarbital, etc.).

MISTAKE 44 is to prescribe *aspirin* in solutions; aspirin should be prescribed in dry form as it is insoluble in water.

MISTAKE 45 *is to give too little aspirin when this is indicated.* A small dose will usually not relieve pain and will thus leave the patient pessimistic concerning his joints.

MISTAKE 46 is to forget that *sensitivity to aspirin* is extremely common.

MISTAKE 47 is to prescribe *aminopyrine* without remembering that it may cause agranulocytic angina.

MISTAKE 48 is to prescribe *cinchophen* or its derivatives without thinking that *liver damage* such as acute yellow atrophy and cirrhosis may follow its use.

MISTAKE 49 is in administering *sulfonamides per se* in the treatment of *rheumatoid arthritis*. As yet, sulfonamides have not been found to be of specific value in the treatment of rheumatoid arthritis.

MISTAKE 50 is to forget that painful joints (*sulfatoxic arthritis*) may develop during sulfonamide administration.

MISTAKE 51 is to forget that *fever therapy* (using bouts of high fever) is seldom used in the treatment of rheumatic disease at present (except in brucellosis and chorea).

MISTAKE 52 is to consider that *jaundice* is a cure for arthritis. Jaundice is merely one of Nature's methods of temporarily ameliorating the condition. Production of artificial jaundice for the treatment of rheumatoid arthritis is not yet advised.

MISTAKE 53 is failure to remember that the *sedimentation rate* is increased during the acute attacks of gout and that although the rate is usually normal in uncomplicated degenerative joint disease (osteo-arthritis), an individual with the latter disease may have a rapid sedimentation rate from many other causes such as a chronic sinusitis, chronic bronchitis, low grade pyelitis, etc.

MISTAKE 54 is to forget that the physiologic state *of pregnancy* is usually markedly antagonistic to the continuance of the symptoms of rheumatoid arthritis.

MISTAKE 55 is to forget that the sedimentation rate is increased in pregnancy, and that it is frequently lowered in allergy.

MISTAKE 56 is to forget that a **normal sedimentation rate does not rule out organic disease.**

MISTAKE 57 *is to depend too much upon the sedimentation rate alone in the diagnosis of joint disease.* Although this is one of our most important tests, it should only be used in conjunction with a carefully taken history, a thorough physical examination, blood count, urinalysis, Wassermann test, joint *x*-rays and common sense. The sedimentation rate is not infallible and if this is not in agreement with the diagnosis, the sedimentation rate should be repeated. If disagreement still persists, either the diagnosis should be questioned or one should search for additional disease processes in that patient. The sedimentation rate is usually increased (rapid) in the active stage of rheumatoid arthritis, rheumatic fever and gout, and is usually approximately normal in degenerative joint disease and fibrositis without complicating diseases. However, one must not depend upon a single determination.

MISTAKE 58 *is to advise the patient with rheumatoid arthritis to give up his job, sell his belongings and move to the Southwest with the hope that he will be cured by this procedure.* Change in climate alone will not usually result in marked improvement in rheumatoid arthritis. It has been found that *chronic arthritis may continue as a sad and crippling disease even on the Arizona Desert.* Most patients should battle their arthritic problems at home and should not be encouraged to endanger their economic status or disrupt their social ties in a vain attempt to seek a climate that cures. However, the patient with rheumatic fever will generally be distinctly improved by residence in the tropics or southwestern part of the United States, Puerto Rico and similar regions. **Tropical climate will not cure rheumatic heart disease and will not remake damaged valves,** nor will it influence cardiac decompensation. Activity in rheumatic fever subsides much faster in the southern climates.

MISTAKE 59 is to believe that *Florida* or other southern states are a panacea for arthritis.

MISTAKE 60 is to think that a stay of a month or two in Florida will "cure" the arthritis. Some patients feel worse while in Florida, others feel somewhat better and still others note no change. Despite the location of the individual with rheumatoid arthritis, his disease needs intelligent handling. Contractures will not disappear spontaneously in the sunshine of either Florida or Tucson.

MISTAKE 61 is to believe that patients with various forms of arthritis react alike in their *response to changes in weather. Cold, dampness and chilling* have been condemned for centuries by patients and physicians as predisposing to the onset or aggravating rheumatic diseases.

MISTAKE 62 is to inform the patient that a series of *21 baths at some spa* will cure his arthritis. Many individuals are improved by proper hydrotherapy and by a vacation away from home. However, the physician must not mislead the patient with rheumatoid arthritis to make great financial sacrifice unnecessarily.

MISTAKE 63 is the *lack of sufficient funds for investigative work and lack of adequate hospital facilities for the care of arthritic patients in this country.* **There are not more than 200 free beds in the United States for the care of rheumatic patients as contrasted with 100,000 free beds for the care of tuberculous patients** (despite the fact that arthritis and allied conditions are more common than the sum of all tuberculosis, cancer, diabetes and heart disease). There should be hundreds of institutions scattered throughout the United States under expert supervision—devoted solely to the care of arthritis and allied conditions.

MISTAKE 64 is to believe that any common form of arthritis develops as a result of a particular *vitamin deficiency.* Vitamin B or C deficiency are not important factors in the causation of rheumatoid arthritis.

MISTAKE 65 is to consider that *massive doses of vitamin D* are specific in rheumatoid arthritis. The Council on Pharmacy and Chemistry of the American Medical Association stated that **clinical evidence did not warrant the claim that massive doses of vitamin D were of benefit in chronic arthritis.**

MISTAKE 66 is to use *vitamin D* in massive doses without realizing that *toxic symptoms* may arise. There is a definite risk of *renal damage* from vitamin D. In one series, 86 per cent of the patients had some toxic symptoms.

MISTAKE 67 is to give large doses of vitamin D without other therapy in rheumatoid arthritis. Although some workers feel that vitamin D may be of value in rheumatoid arthritis, it is not without danger in massive doses and the patient should be checked periodically.

MISTAKE 68 is to restrict certain *foods* or classes of foods in the arthritic in the belief that particular foods in themselves prevent healing or cause further joint damage. The *diet* in most instances should be sensible, high caloric, high vitamin and should contain anticonstipation factors in individuals with rheumatoid arthritis.

MISTAKE 69 is to tell the patient that he can be cured by a *specific diet.* There is no specific diet which will cure rheumatoid arthritis. In most instances, one employs a sensible high caloric, high vitamin, anticonstipation regimen which includes at least a pint of milk, several fresh green vegetables, fresh citrous fruit or juice, adequate protein (meat, fish, fowl, eggs, etc.) and other foods daily.

MISTAKE 70 is to believe that *gold salts* are the final answer to the treatment of rheumatoid arthritis. Gold may be toxic and relapses occur when gold therapy is stopped in a fair number of individuals; furthermore, not all patients with rheumatoid arthritis respond to gold therapy.

MISTAKE 71 is to consider gold therapy without danger. **Gold therapy in rheumatoid arthritis is still too dangerous for common use by the general practitioner without previous special instruction and study.**

MISTAKE 72 is to use large doses of gold salts in rheumatoid arthritis. Present evidence suggests that results are almost as good when individual doses do not exceed 25 mg. of the gold salt.

MISTAKE 73 is to employ gold salts in rheumatic diseases other than rheumatoid arthritis. **Gold salts appear to be of no value in rheumatic disease except rheumatoid arthritis.**

MISTAKE 74 is failure to *stop gold therapy if an untoward reaction occurs.* The unpredictable nature and time of the toxic reactions following gold therapy are the chief hazards in their use.

MISTAKE 75 is failure to tell the patient or his family (in advance of treatment) the *possibility of toxic reactions from gold.*

MISTAKE 76 is believing that one can predict which patients will develop signs of toxicity or complications from gold.

MISTAKE 77 is the giving of injections of gold by the general practitioner without first making a careful study of the whole problem of gold therapy.

MISTAKE 78 is in overlooking the diagnosis of rheumatic fever in the armed forces.

MISTAKE 79 is in forgetting that *rheumatic fever may simulate rheumatoid arthritis* and that only the passage of time and subsequent developments permit a correct diagnosis in some cases.

MISTAKE 80 is in forgetting that *in children, the onset of rheumatic fever is often much more insidious than in adults. Joint pains and swellings may be entirely absent in children with rheumatic fever.* Fatigability and failure to gain weight in rheumatic children may be the only evidence of a carditis.

MISTAKE 81 is in believing that *systolic murmurs* always indicate cardiac damage. Soft systolic murmurs may be associated with fever, anemia, hypertension, nervous excitement, tachycardia and hyperthyroidism without any demonstrable evidence of organic heart disease.

MISTAKE 82 is in forgetting that a *normal sized heart may be the seat of marked disease.*

MISTAKE 83 is to forget that **rheumatic fever may occur in adults.** Rheumatic fever has been an extremely important problem in the Army in World War II.

MISTAKE 84 is to mistake leukemia in young children for *rheumatic fever.*

MISTAKE 85 is in diagnosing rheumatic fever merely because fever and joint pains disappear following the administration of salicylates. Fever and joint pains may occasionally disappear even in acute leukemia in children following salicylate therapy.

MISTAKE 86 is in believing that *salicylates in rheumatic fever* merely lower the temperature and decrease or abolish joint pain. Although judgment must be withheld at present, recent work has suggested that in *some cases large doses of salicylates may prevent the development of a carditis.*

MISTAKE 87 is to administer *sulfanilamide* or other sulfonamides *during the active stage of rheumatic fever* in an attempt to cure the rheumatic fever. These drugs may be harmful in rheumatic fever; they are useless during the acute stage of rheumatic fever and must not be used except for the treatment of an associated infection (or in the prevention of recurrent bouts of rheumatic fever after the acute attack has subsided).

MISTAKE 88 *is to tell the patient that tonsillectomy is insurance against recurrence of rheumatic fever.*

MISTAKE 89 is to overlook an active rheumatic fever in the absence of fever. *An active stage of rheumatic fever may be present with a normal temperature.*

MISTAKE 90 is to permit recurrence of *rheumatic fever* in children when this can be prevented in most instances by *sulfanilamide prophylaxis.* The use of sulfanilamide (and possibly of other sulfonamides) seems to be the best form of prophylaxis against recurrences of hemolytic streptococcal respiratory infections and recurrences of rheumatic fever.

MISTAKE 91 is to permit *foot drop* in any patient confined to bed for long periods.

MISTAKE 92 is to permit *focal infection* to remain in the patient with rheumatoid arthritis. It is true that we do not yet know the rôle of focal infection in most cases of rheumatoid arthritis; however, it seems unwise to allow any definite focal infection to remain in the patient's body if this can be eradicated without detriment to the patient. Removal of focal infection *per se* seldom produces marked improvement in rheumatoid arthritis, but may lessen the load which the patient is carrying.

MISTAKE 93 is for a medical man with very little knowledge concerning the teeth to tell the dentist which teeth must be extracted. *Most*

medical men know very little about the teeth and their disorders; however, they do not hesitate to give orders to the dentist. *The dentist should be considered as a consultant, and not merely as a mechanic.*

MISTAKE 94 is to omit taking a *full mouth x-ray* in the patient with rheumatoid arthritis.

MISTAKE 95 is to believe that a negative roentgenogram of a *pulpless tooth* eliminates the tooth as a possible source of infection.

MISTAKE 96 is to order extraction of all pulpless teeth in an arthritic. *There is no more evidence to warrant extraction of a pulpless tooth in a patient with rheumatoid arthritis than in a patient without arthritis.*

MISTAKE 97 is to order *cholecystectomy* for the "cure" of rheumatoid arthritis unless the pathologic process in the gall bladder necessitates its removal despite the arthritis.

MISTAKE 98 is to think that removal of any focus of infection will cure rheumatoid arthritis. Definite foci of infection should be removed to lessen the load which that patient is carrying but removal of focal infection *per se* will result in marked improvement in the joint phenomena in only a very small percentage of patients.

MISTAKE 99 is failure to discontinue or modify the *application of heat* if this makes the patient worse.

MISTAKE 100 is failure to use *utmost care in applying heat* to the diabetic, to any one with loss of sensation, scars or impairment of circulation.

MISTAKE 101 is failure to explain the dangers of *hot paraffin applications* to the patient in detail.

MISTAKE 102 is failure to remember that the effects from *diathermy* or short wave therapy are due to general effects of heat and not to any specific biologic or bactericidal action.

MISTAKE 103 *is the use of chairs or tables with metal parts for diathermy treatments.*

MISTAKE 104 is to permit the patient to regulate the current in diathermy treatments.

MISTAKE 105 is to permit the patient to have any unpleasant or painful heat sensation during the diathermy treatment.

MISTAKE 106 is to permit metal to come in contact with the patient or the machine during diathermy treatments.

MISTAKE 107 is to apply the diathermy electrodes to the broken skin.

MISTAKE 108 *is to believe that physical therapy alone cures arthritis.*

MISTAKE 109 is to omit physical therapy in the treatment of the average arthritic patient.

MISTAKE 110 is to believe that *sunlight per se* cures rheumatoid arthritis. Although the incidence of rheumatic fever and rheumatic heart disease may increase as exposure to the sun decreases, no one has yet been able to attribute any specific beneficial effect to sunlight in rheumatoid arthritis.

MISTAKE 111 is to omit *underwater exercises* if such treatment is available for the arthritic.

MISTAKE 112 is to believe that buoyancy is present only in water at certain spas. **Buoyancy is present in any water, whether at the most expensive spa or in the cheapest home made pool or bath tub.**

MISTAKE 113 is for the *physical therapist* to make hasty, unguarded statements to the patient.

MISTAKE 114 is to believe that *mineral baths or applications of mud* are particularly beneficial because of absorption of minerals or salts from these. *There is yet no good proof that appreciable amounts of any element are absorbed from mineral waters or muds.*

MISTAKE 115 is to avoid *proper exercise;* the latter will decrease the tendency toward muscular atrophy and aid in preventing the formation of dense adhesions in the joint.

MISTAKE 116 is the *massaging of* painful or swollen bones or joints.

MISTAKE 117 is in permitting massage to be painful. *Proper massage should not cause pain.*

MISTAKE 118 is in *over-exercising* affected joints. *Joints should not be exercised to the point of persisting pain or fatigue.*

MISTAKE 119 is to prescribe physical therapy several times a week in the arthritic patient. If required, *physical therapy should be given daily,* the office or hospital treatments being supplemented by home therapy.

MISTAKE 120 is to tell the patient to exercise the affected joints almost continuously in an effort to keep these from becoming stiff. Acutely inflamed joints should be kept at rest, except for several full movements each day to prevent the formation of dense adhesions. Too much motion of such joints may be deleterious.

MISTAKE 121 is the performance of exercises rapidly. *Muscle exercises should be performed slowly* because rapid motion fatigues muscle quickly and sudden, rapid, jerky movements may produce further damage in arthritic joints.

MISTAKE 122 is the lack of knowledge of most physicians concerning *muscle setting exercises.*

MISTAKE 123 is to *manipulate* acutely inflamed joints; this should not be attempted.

MISTAKE 124 is to operate upon joints during the acute stage of rheumatoid arthritis; this should not be done.

MISTAKE 125 is to manipulate the elbow in rheumatoid arthritis; manipulation of the elbow usually leads to further stiffness and is inadvisable.

MISTAKE 126 is to omit *occupational therapy* in the arthritic patient who is confined to bed or to a chair.

MISTAKE 127 is to believe that heavy *casts* dry quickly. *Heavy casts may remain wet for days in wet, humid climates* (so that it is advisable, if possible, to use a cast dryer with a free circulation of air around the cast).

MISTAKE 128 is failure to realize that if the position of a cast is changed after it has been applied that the cast may be weakened.

MISTAKE 129 is to place *thick padding beneath a cast* as this is frequently a cause of a weakened cast.

MISTAKE 130 is to make wedges or large windows in green casts if this can be avoided.

MISTAKE 131 is to bring *Castex* near an open flame as this is inflammable until thoroughly dry.

MISTAKE 132 is to be unwilling to *learn to use plaster of Paris. Medical men should learn and be willing to use plaster in patients with arthritis.*

MISTAKE 133 is to avoid *splinting of the inflamed rheumatoid arthritic joints;* there is less danger of ankylosis if joints are properly splinted than if they are constantly moved.

MISTAKE 134 is to maintain the splints for long periods of time without removal. *Splints should be removed daily and the affected joints moved through at least two or three full motions to avoid the formation of dense adhesions.*

MISTAKE 135 *is to use orthopedic measures only as a last resort in the arthritic patient rather than as an aid at the onset of treatment.*

MISTAKE 136 is to forget that *various forms of arthritis may manifest the same basic alterations as seen in the x-ray.* The roentgen and pathologic response of joints to various insults is too similar to permit dogmatic interpretation of the roentgenogram.

MISTAKE 137 is to diagnose lumbago or "sciatica" when the basic lesion may be a malignancy of the vertebræ or pelvic bones.

MISTAKE 138 *is to accept the roentgen diagnosis of an arthritic condition as final.* If the roentgen diagnosis does not agree with the clinical impression, the clinician should go over the films with the roentgenologist and should not accept merely a typewritten report.

MISTAKE 139 is failure to *x*-ray the sacro-iliac joints early in *rheumatoid arthritis of the spine.*

MISTAKE 140 *is to rule out gout in acute cases by a negative roentgenogram.*

MISTAKE 141 is to believe that there are any pathognomonic roentgen signs of *gonorrheal arthritis.*

MISTAKE 142 is to rule out *tuberculous arthritis* in the early stages because of a negative *x*-ray.

MISTAKE 143 is to omit *roentgen therapy in* the treatment of *rheumatoid arthritis of the spine.*

MISTAKE 144 is to tell the patient that the joint is normal inasmuch as the *x*-rays are negative. **Marked limitation of motion may occur in the absence of obvious x-ray changes.**

MISTAKE 145 is to rule out the possibility of rheumatoid arthritis because of apparently negative roentgen findings. *The earliest roentgen findings occasionally may not appear for years after the onset of joint symptoms in rheumatoid arthritis.*

MISTAKE 146 is to forget that *punched-out areas in bones* may be caused by many diseases; such punched-out areas at the ends of the bones are not pathognomonic of gout but may occur, for example, in rheumatoid arthritis, some cases of degenerative joint disease, etc.

MISTAKE 147 is failure to remember that in the early stages of rheumatoid spondylitis (rheumatoid arthritis of the spine), symptoms may include only aches and pains in the extremities with very little stiffness or pain in the low back. Roentgen findings may be minimal in the early stages but definite changes will usually be seen in the sacro-iliac joints.

MISTAKE 148 is to overlook *narrowing of the cervical intervertebral foramina* with subsequent pressure on nerves; *x*-ray may not detect this unless oblique views are taken.

MISTAKE 149 is to forget that *osteoporosis of bones* may be caused by many diseases.

MISTAKE 150 is to forget the diagnosis of *gout* when a joint becomes markedly swollen, red and tender within a short period of time (in the absence of a history of trauma or gonorrhea).

MISTAKE 151 is to consider that all patients with an *elevated blood uric acid* have gout. Elevation of the blood uric acid occurs in many other diseases such as in glomerulonephritis, cardiac decompensation, leukemia, polycythemia, lobar pneumonia and some other acute febrile diseases, etc. However, in the presence of joint pain and swelling and an elevated blood uric acid, the diagnosis of gout is practically always correct.

MISTAKE 152 is the ready acceptance of the diagnosis of *gout in a female.* One must be extremely careful in such a diagnosis as *approximately 98 per cent of gout occurs in males.*

MISTAKE 153 is to wait until tophi appear to diagnose gout. *Tophi rarely appear before the tenth year of the disease except in very severe cases.*

MISTAKE 154 is to forget to consider gout when a great toe is involved in an arthritic process.

MISTAKE 155 is to pass over the diagnosis of gout because of negative roentgen findings. **X-ray findings in gout may be negative for at least several years in some cases.**

MISTAKE 156 is failure to remember that *poor posture* and *improper shoeing* may result in chronic pains in the legs and low back.

MISTAKE 157 is to place pillows under the knees of a patient with arthritis of the knees. If arthritic involvement of the knees is present, every attempt must be made to keep the knees extended; **pillows must never be placed under arthritic knees if one wishes to avoid contracture deformities.**

MISTAKE 158 is to permit the patient to walk on a deformed, flexed knee joint when this can be straightened; walking with the knee flexed may produce distortion in both lower extremities and in the trunk.

MISTAKE 159 is to permit weight bearing if the feet are involved in an arthritic process. Weight bearing should not be permitted until pain and swelling have subsided.

MISTAKE 160 is to forget that *poor posture* is one of the most important causes of strain of the back, foot, knee and hip.

MISTAKE 161 is to forget that most deformities in arthritis are those of flexion, and that joints are invariably flexed during sleep in these individuals to relieve pain.

MISTAKE 162 is to permit the arthritic patient to spend many hours sitting in a chair as this permits the affected joints to remain for long periods in positions of the common flexion adduction deformities.

MISTAKE 163 is failure to prevent deformities. *It is always easier to prevent a deformity than to correct one.*

MISTAKE 164 is to forget that the period between an injury and the production of a traumatic arthritis may vary from days to several months.

MISTAKE 165 is to forget that *in individuals of fifty years or more, degenerative joint disease is almost always present independent of any occupation*, especially in the lumbar, cervical and sacro-iliac region.

MISTAKE 166 is to rule out injury as the cause of joint discomfort in the absence of evident anatomic changes; **years of pain and limitation of motion may follow a single strain or contusion.**

MISTAKE 167 is to forget that *modern bullets* traveling at a high velocity may go *through a joint* causing little disturbance and no appreciable infection.

MISTAKE 168 is to forget that *hyaline articular cartilage does not regenerate* although it may be repaired somewhat by the formation of fibrocartilage.

MISTAKE 169 is to believe that joints remain normal for more than several decades following birth.

MISTAKE 170 is to forget that joint disease may be due to trauma.

MISTAKE 171 is to forget that *arthritis* may be *gonococcal* in origin, despite the individual's rank in society.

MISTAKE 172 is to believe that *spur formation on the os calcis* is usually due to gonorrheal arthritis. Such spurs are commonly found in the absence of the latter disease.

MISTAKE 173 is to consider that symptoms referable to the genito-urinary tract must be present in patients with *gonorrheal arthritis.*

MISTAKE 174 is to believe that the sternoclavicular, temporomandibular or spinal joints are frequently involved in gonorrheal arthritis.

MISTAKE 175 is to forget that *conjunctivitis* is more common *in gonorrheal arthritis* than in other forms of joint disease.

MISTAKE 176 is to forget that *tenosynovitis* is more frequent *in* patients with *gonococcic arthritis* than in any other type of arthritis.

MISTAKE 177 is to forget that bilateral hydrops of the knees in children is often due to syphilis (*Clutton's joints*).

MISTAKE 178 is to forget the possibility of a Charcot joint, although these should become rare with institution of penicillin therapy for syphilis.

MISTAKE 179 is to think that *Charcot joints* always develop very gradually; these occasionally develop with great suddenness.

MISTAKE 180 is to forget the possibility of a neuro-arthropathy if joint disease occurs without heat, pain or tenderness.

MISTAKE 181 is to omit the *blood Wassermann test* in any individual with joint aches or pains. This should be done routinely.

MISTAKE 182 is failure to consider syphilis in all patients with joint pain.

MISTAKE 183 is failure to consider a *tuberculous* etiology in the case of a chronic monarthritis (in the absence of trauma and gonorrhea).

MISTAKE 184 is to forget that arthritis may accompany *chronic ulcerative colitis.*

MISTAKE 185 is to forget the possibility of *periarteritis nodosa* which may simulate rheumatoid arthritis.

MISTAKE 186 is to forget that *angina pectoris* may cause pain radiating to the hand, and that spondylitis may result in precordial distress that may simulate angina.

MISTAKE 187 is to omit the possibility of *Parkinsonism* in individuals with deformities of the hands and feet, rigidity, tremors and changes in the skin.

MISTAKE 188 is to forget the possibility of *brucellosis* (undulant fever) in an individual with bizarre aches and pains, and a low grade, undulating fever.

MISTAKE 189 is failure to remember that *trichiniasis* may cause widespread muscular aches and pains.

MISTAKE 190 is failure to remember that *hysteria* may mimic any known form of arthritis.

MISTAKE 191 is to forget that the *hypothyroid* patient may exhibit numerous joint and muscle aches and pains.

MISTAKE 192 is to forget the possibility of *scleroderma* in any individual with pains in the hands and tightness and thickening of the skin.

MISTAKE 193 is failure to remember that a *positive Frei test may persist for years* after apparent healing has occurred in *lymphopathia venereum.*

MISTAKE 194 is to frequently diagnose "allergic arthritis." *No one has been able to set up any one form of arthritis as allergic arthritis*, nor has any form of arthritis been shown to be solely the result of bacterial or food allergy.

MISTAKE 195 is to forget that lupus erythematosus may exhibit temporary joint pain and swelling, associated with fever, loss of weight and the other signs of that disease.

MISTAKE 196 is to permit actinic therapy in a patient with *lupus erythematosus.* This may greatly aggravate the condition (as may removal of focal infection during the active stage of the lupus erythematosus).

MISTAKE 197 is to forget the possibilities of *periarteritis nodosa, lupus erythematosus or dermatomyositis* in individuals with bizarre muscular pains and aches, loss of weight, irregular periods of fever, muscular atrophy and joint pains.

MISTAKE 198 is to overlook the uncommon syndrome, *Milkman's disease* when *x*-ray discloses multiple spontaneous idiopathic symmetrical fractures.

MISTAKE 199 is to forget that *malignancy* may be primary or metastatic in the joint.

MISTAKE 200 is to forget that *coronary occlusion* may be followed by a *painful shoulder* and in some instances by pains in the hands and the development of thickening of the palmar fascia, resembling *Dupuytren's contracture.*

MISTAKE 201 is to forget that *80 per cent of patients with pain about the shoulder probably have the lesion commonly called subdeltoid bursitis* (with a frequently associated calcification in the supraspinatus tendon).

MISTAKE 202 is to omit the diagnosis of subdeltoid bursitis because of negative roentgen findings. *Roentgen findings (such as supraspinatus tendon calcifications)* are present in not more than 50 per cent of patients with this syndrome.

MISTAKE 203 is to think that *pain in the shoulder* is usually due to arthritis. *Arthritis involving the shoulder joint alone, without involvement of other joints, is uncommon.*

MISTAKE 204 is to forget that regardless of the method of treating subdeltoid bursitis, after the acute painful stage has passed, one should always follow this with *exercises, local heat and massage.*

MISTAKE 205 is to forget that any painful lesion about the shoulder may excite *spasm of the scalenus anticus muscle* with its characteristic train of symptoms, including pain in the shoulder and neck region and pain or numbness radiating to the elbow or hand.

MISTAKE 206 is to rule out **degenerative joint disease** because a negative roentgenogram is found. *Many of these joints may show no roentgen abnormalities despite gross pathologic changes which may be present in articular hyaline cartilage.*

MISTAKE 207 is to believe that one must have *articular lipping* as a prerequisite for degenerative joint disease.

MISTAKE 208 is to think that *Heberden's nodes* occur in uncomplicated rheumatoid arthritis.

MISTAKE 209 is to tell the patient that Heberden's nodes never cause discomfort. These nodes may appear rather abruptly in some instances and cause from slight to moderate discomfort with redness and swelling.

MISTAKE 210 is to fear that the patient will develop severe disability because of the appearance of *Heberden's nodes.* These latter *usually worry the patient more because of the unsightly appearance than because of any great degree of disability.*

MISTAKE 211 is to believe that all hypertrophic changes in the spine cause symptoms. *Extensive hypertrophic changes may occur here without producing symptoms.*

MISTAKE 212 is to fail to reassure the patient with degenerative joint disease that he will not progress to a stage of helpless invalidism.

MISTAKE 213 is to await roentgen changes in the vertebræ before diagnosing *rheumatoid spondylitis.* The early diagnosis should be suggested on clinical evidence.

MISTAKE 214 is in forgetting that *mixed forms of arthritis may occur.* The individual with degenerative joint disease may develop a superimposed rheumatoid arthritis or gonorrheal arthritis. Any mixture of joint diseases may occur.

MISTAKE 215 is to forget that *ligamentous and muscular strains cause the majority of all backaches.*

MISTAKE 216 is to forget that *50 per cent of patients with architectural weakness of the spine have no symptoms resulting therefrom.*

MISTAKE 217 is to forget that the *lumbosacral junction* is the most vulnerable portion of the spine and is subjected to the greatest strain.

MISTAKE 218 is to forget that the *symptoms of lumbosacral strain may be out of all proportion to the trauma.*

MISTAKE 219 is to forget that *more than one-third of all spines x-rayed for any purpose show congenital abnormalities;* most of these do not cause symptoms.

MISTAKE 220 is to forget that **chronic low back strain is the most common cause of low back pain.** Although herniated disks are extremely popular at present, these cause only a small proportion of all low back pain.

MISTAKE 221 is to attribute many instances of backache to gynecologic disorders.

MISTAKE 222 is to forget that *degenerative joint disease of the spine is present in practically all patients over the age of fifty;* in only a few of these is the condition severe enough to cause symptoms.

MISTAKE 223 is to forget the possibility of *vertebral malignancy* (usually metastatic) in any painful, progressive spondylitis.

MISTAKE 224 is to be satisfied with a diagnosis of **sciatica.** *Sciatica is not a disease but is merely a symptom*—designating pain in the distribution of the sciatic nerve. It is no more a diagnosis than is headache.

MISTAKE 225 is to forget the diagnosis of **herniated intervertebral disk** in an individual with recurring severe low backache with sciatic pain (in attacks) down the back of the leg.

MISTAKE 226 is to manipulate a spine if a protruded disk is suspected.

MISTAKE 227 is to forget to *x-ray the* persistent or *recurring painful back.*

MISTAKE 228 is to forget that *many abdominal operations have been performed upon patients because of pain due solely to spinal arthritis.*

MISTAKE 229 is to forget that a *correct diagnosis is essential to the successful treatment of low back pain.*

MISTAKE 230 is to apply the same treatment for all patients with low back pain.

MISTAKE 231 is to forget that *shortening of one leg and improper weight balance may be important causes of persisting low back pain.*

MISTAKE 232 is to forget that *chronic foot strain* is probably the most common cause of painful feet.

MISTAKE 233 is to fit the foot to arch supports rather than to remember that *arch supports, if needed, should be made to fit the individual foot.*

MISTAKE 234 is to buy shoes because of their beauty or quality. *If shoes are uncomfortable or painful, they should not be used.* **The proper shoe should fit.**

MISTAKE 235 is to permit shoe men to take over the care of the feet even though they lack the fundamental knowledge of that intricate mechanism.

MISTAKE 236 is to forget to tell the patient that the size, width and correct fit of a shoe is usually much more important than the trade name.

MISTAKE 237 is to warm *immersion feet;* the injured immersion feet must be kept cool (while the body is being warmed). Do not rub these feet or paint with a strong disinfectant. In immersion feet, recovery is usually complete with proper treatment even though massive gangrene from the ankle down may appear imminent.

MISTAKE 238 is to forget that *vascular disease is a common cause of pain in the foot in the aged.*

CHAPTER LXIV

RECENT ADVANCES IN ARTHRITIS AND ALLIED CONDITIONS

Gold Salts.—One of the important therapeutic agents introduced within the past fifteen years in the treatment of rheumatoid arthritis is *gold.* Gold therapy in arthritis is discussed in detail in Chapter XXII. It has been shown that injections of certain gold salts in proper dosage are followed by definite subjective and objective evidence of improvement in the majority of patients with rheumatoid arthritis. **Gold salts are of value in no rheumatic disease except rheumatoid arthritis.** However, gold salts are not the ideal drug for which we are looking in rheumatoid arthritis inasmuch as they are toxic; not all patients with rheumatoid arthritis respond to injections of gold salts; and the arthritis recurs in a moderate percentage of patients following cessation of chrysotherapy.

As yet, gold salts are not advised routinely at the onset of rheumatoid arthritis. Conservative therapy results in improvement in some patients with rheumatoid arthritis. If the rheumatoid arthritic obtains no benefit from conservative measures over a period of three or four months, or if he goes downhill rapidly even before this, gold therapy is advised if it can be given by one thoroughly acquainted with this subject. Gold therapy in rheumatoid arthritis is still too dangerous for common use by the average general practitioner.

Toxic reactions may occur following even small doses of gold salts; however, these are usually much less severe and of much shorter duration than those reactions which may follow large doses. Present evidence suggests that *results are almost as good when individual weekly doses do not exceed 25 mg. of the gold salt* (such as Myochrysine or Solganal-B Oleosum). Serious reactions may be avoided by questioning the patient before each injection concerning pruritus, sore mouth or gastrointestinal disturbances; examining the skin, gingiva and scleræ at each visit for dermatitis, stomatitis, purpuric spots and icterus; and performing a white blood count, differential count, hemoglobin estimation and urinalysis before each injection.

Gold salts should not be given to any patient with a history of agranulocytic angina, purpura, exfoliative dermatitis, renal or hepatic disease or during pregnancy. It is inadvisable to give gold salts to a diabetic unless the diabetes is extremely mild and the patient has a severe rheumatoid arthritis which is going downhill despite all attempts at conservative care; gold salts may aggravate the diabetic state.

Gold salts should be stopped if an untoward reaction occurs. If this is agranulocytic angina, purpura hemorrhagica, exfoliative dermatitis, hepatitis or severe renal damage, the gold should never be resumed. Following the subsidence of mild reactions, gold may be resumed in smaller doses.

Conservative care should be continued during chrysotherapy; this includes mental and physical rest; splinting of affected joints where indicated; an adequate sensible, well-balanced diet; physical and occupational therapy; orthopedic attention; psychotherapy and symptomatic care.

Rheumatoid arthritis has been reported to be definitely benefited by injections of gold salts by such authorities as *Freyberg, Dawson, Boots, Hartung, Cecil, Snyder* and others. Other eminent rheumatologists are not impressed by the results of gold therapy. *Bauer,* several years ago, observed that only 4 of 30 patients treated with gold showed marked subjective and objective improvement. In his experience, the results were no better than the usual treatment with rest, high caloric, high vitamin diet, analgesics and simple physical therapy. He believed that within ten years, gold would go the way of many previous rheumatic remedies and would not be considered the meritorious antirheumatic remedy it is hailed today. On the other hand, *Freyberg believes that gold therapy is the best treatment now available to arrest or lessen the inflammatory process in rheumatoid arthritis,* while Dawson, Boots and Tyson concluded that chrysotherapy offered greater promise than other forms of therapy in the treatment of rheumatoid arthritis. **The unpredictable nature and time of the toxic reactions following gold therapy are the chief hazards in their use.** In the series of 51 cases treated by Dawson, Boots and Tyson, the disease was apparently rendered inactive in 22 with persistently normal sedimentation rates following treatment. These cases are the most convincing argument in favor of chrysotherapy as similar results have not been obtained with any other form of therapy to date.

Therapeutic effects are not usually observed until after six or eight injections. *The mode of action of gold salts in arthritis is not known* although it has been shown that gold salts have marked chemotherapeutic properties against hemolytic streptococcal infection in mice, comparable to that obtained with the sulfonamide derivatives. Gold is excreted chiefly through the kidneys and to a lesser extent through the intestinal and bronchial mucosa. Gold may be retained in the body for long periods and has been found in the blood and urine as long as a year after treatment has been stopped. No one yet knows how, where or why gold salts result in improvement in rheumatoid arthritis.

Colloidal gold preparations have not been as effective therapeutically because of the rapid phagocytosis of the particles of gold by the reticulo-endothelial system with insignificant plasma gold concentrations.

Toxic reactions may appear at any time after the first injection up to several months after cessation of therapy. There is no reliable method of predicting which patients will develop signs of toxicity or complications. The factors upon which toxic reactions depend are unknown.

It is possible that the frequency of relapses following chrysotherapy may be decreased by continuing smaller doses of gold after completion of a series of injections so that the treatment is not discontinued abruptly. (See Chapter XXII on Gold Therapy for details.)

Penicillin.—Penicillin is definitely of great value in the treatment of certain specific infectious forms of arthritis such as the pneumococcal, streptococcal, staphylococcal, gonococcal and meningococcal types. In these individuals, at present, penicillin is administered parenterally (usually intramuscularly in divided doses every three hours day and night for a total of 100,000 units or more per twenty-four hours) as well as intra-articularly (introduction of 20,000 units or more of penicillin directly into the affected joints daily for several days, when possible). There is no indication for penicillin in degenerative joint disease or traumatic arthritis.

Penicillin has now been adequately tried in patients with rheumatoid arthritis. We have noticed no marked subjective or objective changes in the arthritis and no marked change in the sedimentation rate in 6 patients with typical rheumatoid arthritis who were given penicillin for a period of at least three weeks (using 100,000 units intramuscularly per twenty-four hours in divided doses every three hours in 3 patients and in 3 others, 200,000 units daily); 2 patients were treated for four weeks. Several patients thought that they felt stronger and had less general malaise.

The indications, dosage, method of administration and other details concerning penicillin are given in detail in Chapter LX on Penicillin.

Sulfonamides.—Sulfonamides have been shown to be of value in some of the specific infectious forms of arthritis (due to the pneumococcus, hemolytic streptococcus, meningococcus, gonococcus, etc.) but have been of very little if any value in rheumatoid arthritis except as a pre- and post-operative medication when focal infection is removed (in an attempt to avoid a flare-up in the arthritis). *Sulfonamides may increase the severity of rheumatic fever if given during the active stage of the disease* and are thus contraindicated at that time. Sulfonamides are not of value in the treatment of uncomplicated degenerative joint disease, fibrositis, gout or traumatic arthritis. Lymphopathia venereum may also be benefited by sulfonamides.

One of the important uses of sulfanilamide in the rheumatic diseases is as a *prophylactic measure in the prevention of recurrent attacks of rheumatic fever and hemolytic streptococcus sore throat.* The use of sulfanilamide daily seems to be the best form of prophylaxis against recurrences of rheumatic fever in susceptible individuals (giving approximately 0.6 gm. [gr. x] morning and evening, particularly in the period from October until June, although some workers continue this all year round in susceptible children for as long as from two to five years). Sulfadiazine has recently been tried in the same manner, giving from ½ to 1 gram daily in adults for periods as long as three months. The details of such therapy are given in Chapter XLIII on Rheumatic Fever.

Visceral Lesions in Rheumatoid Arthritis.—The recent findings at autopsy of the *high incidence of cardiac involvement in patients with rheumatoid arthritis* have been extremely interesting. Cardiac lesions indistinguishable from those of rheumatic fever have been found in a large percentage of patients (56 per cent in one series). In addition, from 35 to 40 per

cent of patients with rheumatoid arthritis in one series showed evidence of rheumatic heart disease by clinical examination, suggesting that rheumatic fever and rheumatoid arthritis bear some relationship to each other. Dawson thought that it was futile to speculate on the precise nature of this relationship as the etiology of neither disease has as yet been definitely established. This subject is considered in detail in Chapter VIII on the Visceral Lesions Associated With Rheumatoid Arthritis.

Vitamin D in Arthritis.—Much controversy has appeared concerning the effect of massive doses of vitamin D in rheumatoid arthritis. Much of this has centered around the preparation "**Ertron**"—which has been advertised extensively to physicians. The Council on Pharmacy and Chemistry of the American Medical Association has stated that *clinical evidence did not warrant the claim that massive doses of vitamin D were of benefit in chronic arthritis.* Freyberg concluded that massive doses of vitamin D should not be used with a comfortable feeling that great benefit is certain to result. He reported that **the course of rheumatoid arthritis was seldom favorably altered by Ertron.** The results of his study of vitamin D therapy in arthritis were far from impressive in that the drug was beneficial in only a minority of cases and when improvement did occur, this was usually temporary and only symptomatic. At the Mayo Clinic, Slocumb felt that *there was a definite risk of renal damage from vitamin D;* he concluded that massive doses of vitamin D were of very little, if any use in producing lasting improvement in the patient with infectious arthritis.

Some investigators have reported beneficial results from massive doses of vitamin D, using from 150,000 to 300,000 units per day. Physicians using this form of therapy must realize that there is definite danger attached to it and the patient must be watched carefully for signs of toxicity. At present, *most investigators at teaching hospitals have concluded that massive doses of vitamin D are of very little permanent value in rheumatoid arthritis* (although they may cause a temporary subjective improvement, increase in appetite and gain in weight). This subject is discussed in detail in Chapter XXVI on The Rôle of Vitamins in Arthritis.

Paraneuritic Nodules.—Leichentritt and his associates found *small nodules in the perineurium of peripheral nerves in patients with rheumatoid arthritis.* They considered these lesions to be specific for rheumatoid arthritis. The nodules varied from 0.14 to 0.2 mm. in diameter and appeared to the naked eye as bluish, round or oval shaped lesions when stained with hematoxylin eosin. Microscopically, they showed a peripheral ring of lymphocytes and plasma cells with a middle zone of epithelioid-like cells and an inner pale and acellular center. Other workers have confirmed this finding. (See Chapter VI, Pathology of Rheumatoid Arthritis.)

Procaine Injections in Arthritis.—Procaine has been employed by various workers in the field of arthritis. In traumatic arthritis, procaine has been suggested actively and prophylactically. Procaine is widely used in the treatment of *sprains and contusions.* Some individuals have reported encouraging results by infiltrating the ligaments and muscles about joints which are affected by degenerative joint disease Others have employed

procaine in the treatment of rheumatoid arthritis, bursitis, neuritis and spondylitis with reports of freedom from pain in 70 per cent of cases after an average of three or four injections. *The use of procaine in sprains, contusions, some forms of bursitis and around joints in degenerative joint disease (as a symptomatic measure) is accepted by most physicians.* The use of procaine in *fibrositis* with injections into tender nodules is also well known. Procaine has not been used widely in rheumatoid arthritis. (See Chapter XX, Miscellaneous Items of Therapy.)

Procaine Injections in Sprains.—*Manpower shortage has been minimized in wartime by the use of local anesthetics injected directly into the most tender area soon after sprains or injuries when there is no accompanying open wound.* Such injections greatly reduce the pain and permit the injured individual to remain active.

The affected part, usually an ankle, is washed with soap and water, followed by the application of iodine and alcohol. One or 2 per cent procaine is injected directly into the most tender subcutaneous tissues, withdrawing the barrel of the syringe before injection to prevent against injection into the general circulation. From 10 to 30 cc. of procaine may be used to inject all tender areas in the injured ligament as well as tender areas in other ligaments until no tender or painful areas remain on palpation or on motion of the ankle or foot.

An elastic bandage is then wrapped snugly around the ankle and the patient is instructed to use the foot and ankle normally except that no running or jumping is done. The individual is requested not to keep the foot immobile in one position for even short periods. The bandage is removed and rewrapped in an hour or two to prevent possible circulatory constriction. The success of the injection depends upon complete adequate initial intraligamentous injection to produce total elimination of pain and tenderness and then, continued use and motion of the foot and ankle. McMaster reported excellent results by this method in all patients.

Rheumatic Fever.—*Rheumatic fever has been found to be an important military disease.* Prior to the World War II, it was observed that in the great majority of cases, first attacks of rheumatic fever appeared before the age of fifteen. However, one must always remember that rheumatic fever is relatively common in adults during wartime particularly in susceptible individuals (by heredity) who develop hemolytic streptococcal respiratory infections in certain areas (varying with climate and meteorology) such as the northern states or the central Rocky Mountain area. Rheumatic fever is uncommon in Army camps in the southern part of this country. However, large numbers of young adult soldiers may be attacked by the disease in certain areas of the United States. The incidence of rheumatic fever may be reduced in susceptible areas where hemolytic streptococcal infections are present if all of the men are given sulfonamides such as 0.5 to 1 gm. of sulfadiazine daily for periods up to three months.

Although all of the factors concerned in the **etiology of rheumatic fever** are not known, it appears that *important rôles are played by heredity, hemolytic streptococcal respiratory infections and climate and meteorology*—as well

as by other as yet unknown factors. This subject is discussed in Chapter XLIII on Rheumatic Fever.

Sulfadiazine Prophylaxis in Rheumatic Fever Control and in the Prevention of Respiratory Tract Bacterial Infections.—The United States Navy has been engaged in a long-term program for the *control of streptococcic infections and their disabling sequelæ.* One of the most important components of this program involves *mass prophylaxis with sulfadiazine.* Coburn reported (from observations on 30,000 men at three camps) that the **continuous daily ingestion of 1 gram of sulfadiazine was adequate to protect 85 per cent of susceptible recruits from implantation with bacterial respiratory pathogens, was sufficient to check a well-advanced streptococcic epidemic and to check a streptococcic outbreak at its onset.** It was also suggested that a daily dose of 0.5 gm. of sulfadiazine was almost 85 per cent effective in preventing implantation by the Streptococcus hemolyticus. About 0.5 per cent of the men involved in the mass sulfadiazine prophylaxis showed evanescent rashes and dangerous constitutional disturbances appeared in only 0.01 per cent.

Sulfadiazine prophylaxis also appeared to be effective in preventing rheumatic fever. *The incidence of rheumatic fever among men receiving chemoprophylaxis was only 15 per cent of the incidence in control groups.* It was concluded that approximately 85 per cent of the expected rheumatic fever cases appeared to be eliminated by sulfadiazine.

Holbrook emphasized that in the Army Air Forces, acute rheumatic fever occurred in high incidence and in every instance was preceded by a high incidence of hemolytic streptococcus infections. In his experience, a 50 to 75 per cent reduction in the incidence of respiratory diseases and streptococcic infections was accomplished by the use of sulfadiazine prophylaxis under carefully controlled conditions and on a significantly large troop population, without the occurrence of serious drug reactions. The reduction in rheumatic fever paralleled that of respiratory and streptococcic diseases.

This is extremely important in times of war as rheumatic fever has become a very serious problem in members of the armed forces. *In 1943, the United States Army of 7 million troops spent in excess of 14 million days in the hospital because of common respiratory diseases,* in addition to the inevitable number of complications and deaths and additional millions of man days lost from duty. If the recent experience with sulfadiazine prophylaxis holds true, from 50 to 75 per cent of this tremendous loss may be avoided. In some instances, sulfadiazine prophylaxis was continued for as long as three months.

Diet and Sunshine as Predisposing Factors in Rheumatic Fever.—*In a recent study, it has been observed that the incidence of acute rheumatic fever and rheumatic heart disease increased as exposure to the sun decreased. A very low incidence of rheumatic infection is said to exist in those countries having an abundance of protein and fish oils in the diet.*

Peete believes that *poverty,* and not dampness *is an important predisposing factor in rheumatic fever.* Holland has a low incidence of rheumatic heart disease despite its damp, cold climate. The incidence of rheumatic infection was found to be lowest in the private schools, next lowest in rural schools and highest in public schools. *Recurrences of active infection were very few when families were able to correct dietary deficiencies and add cod liver oil to the diet.* Peete has emphasized the importance of adequate amounts of vitamins A and D, milk, protein and the value of sun bathing in the prevention of rheumatic fever and its recurrences. These conclusions are extremely interesting but will require further confirmation.

Streptococcus Hemolyticus Filtrate.—A difference of opinion exists at present concerning the value of this therapy. However, Wasson thought that immunization of children who have had one or more attacks of rheumatic fever with a stock filtrate of Streptococcus hemolyticus appeared to prevent further attacks and improved the well-being of the individual. After nine years of experience, Wasson and Brown concluded that the therapy was definitely beneficial. In 42 subjects so immunized there was no rheumatic attack during a nine month period as compared with a 33 per cent incidence of attacks in 33 controls. On the other hand, Coburn and Pauli found no evidence that the course of immunization increased resistance to streptococcal infections or rheumatic fever. This subject is presented in Chapter XLIII on Rheumatic Fever.

Salicylates in Rheumatic Fever.—Several newer developments have appeared in this field. Coburn and Moore gave from *4 to 6 gm. of sodium salicylate daily for a month to rheumatic subjects who developed pharyngitis and in whom Group A hemolytic streptococci were cultured from the throat. Only 1 of 47 patients so treated developed rheumatic fever.* Schlesinger reported fewer rheumatic recurrences in children and milder recurrences when these appeared in those rheumatic children who were given from 0.5 to 0.8 gm. of aspirin three times a day at the onset of a pharyngitis. On the other hand, Sheldon was not convinced that a course of aspirin begun at the time of onset of a pharyngitis in patients with inactive rheumatic fever was beneficial, but suggested further trials.

Recent work by Coburn has also suggested that *large doses of salicylates may (in some instances) prevent the development of carditis in rheumatic fever.* He has suggested the use of large amounts of salicylates daily, including intravenous salicylate therapy in an effort to quickly modify the activity of the disease. Previously, it was considered that salicylates merely relieved the pain and fever associated with the disease and had no effect upon the development of cardiac lesions. *Eighteen patients who were treated with daily doses of 10 gm. or more of sodium salicylate by mouth or vein all showed rapid clinical recovery and a progressive fall in the blood sedimentation rate.* This study suggested that although 3 to 6 gm. daily was adequate to relieve pain and fever, it failed to suppress the inflammatory reaction. The salicylate plasma content was followed in these patients. This is presented in detail in Chapter XLIII on Rheumatic Fever.

Prevention of Recurrent Attacks of Gout.—Bartels reported the use of *a low fat, low purine, high carbohydrate diet together with cinchophen (in decreasing doses) in an attempt to reduce the number and severity of further*

attacks of gout. Cinchophen was given beginning with 0.5 gm. (7½ gr.) three times a day for three days a week. These patients were advised as to the symptoms of cinchophen toxicity and the drug was discontinued if signs of toxicity appeared. As the blood level of uric acid fell, the dose of cinchophen was decreased. The use of a high carbohydrate diet was an added precaution against liver damage. If liver function tests showed a decreased function, cinchophen was stopped. The use of alcohol was discouraged because of the possible toxicity from a synergistic or intensified action of alcohol and cinchophen on the liver. The blood uric acid level was followed every month or two; when this was lowered, the dose of cinchophen was reduced to one or two days each week, or omitted entirely when the reading was approximately normal. The high carbohydrate, low purine, low fat diet was continued indefinitely, but was liberalized if the blood uric acid level returned to normal and continued there. Vitamins A and B were added because the diet was low in these.

When the diet and cinchophen were adhered to, the blood level of uric acid fell appreciably and no major attacks of gout occurred; minor attacks were very few in number. Such a plan has definitely resulted in a marked decrease in the incidence of gouty attacks.

Use of Penicillin in Syphilis.—The recent reports on the beneficial effects of penicillin in early and late syphilis are extremely important. Moore, Mahoney and their associates showed that **penicillin had a profound immediate effect in early syphilis** with disappearance of surface organisms from open lesions, healing of lesions and a trend toward serologic reversal. The optimum time-dose relationship of penicillin in early syphilis is not yet known. *At present, in primary and secondary syphilis,* **one should administer 300,000 units per day for seven and a half days (a total of 2,400,000 units)**; this is given in divided doses intramuscularly at intervals of three hours, day and night. It is possible that a combination of seven and a half day of penicillin and simultaneous administration of mapharsen may give fewer relapses.

A favorable effect has also been found from penicillin in early asymptomatic neurosyphilis, acute syphilitic meningitis, early syphilis treatment resistant to arsenic and bismuth and infantile congenital syphilis.

Stokes and his associates found that the lesions of benign gummatous syphilis of the skin and bones healed with a daily dose of approximately 300,000 units in twelve to forty-six days. In all types of syphilis, penicillin produced a reduction in syphilitic reagin titer in the blood in from 50 to 60 per cent of late cases. Although improvement occurred in a high percentage of cases of simple demented paresis and in about 20 per cent of those with tabes dorsalis, *Charcot joint was unaffected by penicillin,* a new one developing during therapy.

Abuse of Rest in Bed.—Gold has recently emphasized some of the *dangers of prolonged rest in bed* (such as the effect upon morale, gastrointestinal problems, constipation, distention, the issue of the bedpan, urinary retention, muscle cramps, hypostatic congestion, etc.). Dock has noted that man differs from other mammals by sleeping on his back and lying recumbent when ill. Up to the Nightingale era, the sick patient usually got up several times each day for elimination and in some cases for meals.

Prolonged rest in bed may lead to decalcification of bones with an immediate severe, negative calcium balance; weakened vasomotor tone; wasting of voluntary muscles, etc.

Pulmonary embolism is rare in ambulatory patients or in those who lead wheel chair lives. *Rest in bed presents very slight hazards to children but does become increasingly dangerous with advancing years.*

However, rest of injured parts and of diseased bodies is one of the oldest and most valuable of all methods of therapy, except possibly psychotherapy. It is extremely interesting, therefore, to find a symposium on "The Abuse of Rest in the Treatment of Disease" presented before the annual session of the American Medical Association in June, 1944. During this symposium, Harrison suggested that persons with congestive heart failure be allowed out of bed for several hours a day, as soon as severe dyspnea at rest has subsided and that following myocardial infarction, recumbency should not be prescribed for a longer period than two to three weeks after the more acute and alarming symptoms have subsided. He further emphasized that the recumbent position should not be enforced on patients who are more comfortable sitting. Powers stated that rest is fraught with hazard and that prolonged periods of recumbency in bed are anatomically, physiologically and psychologically unsound. He favored early postoperative activity and walking.

Ghormley stated that he thought that when an arthritic patient gave up and went to bed, he became a total cripple. However, Krusen pointed out that much of the confusion with regard to the abuse of rest in the arthritic was due to failure to recognize the difference between complete immobilization of the individual and the avoidance of fatigue. Hench noted that some individuals have stressed rest so much that we may be in danger of overresting our patient. He thought that the dangers of overrest in arthritis were as great or more so than the dangers of overexercise inasmuch as the patient promptly knows he has done wrong when he has overexercised because of increased pain, but when he overrests the results creep up silently and maliciously until serious contractures develop.

During this symposium, Katz emphasized that rest does not consist of merely putting a patient to bed, but that *emotional rest* must also be obtained.

Powers stated that all of his surgical patients are now ambulatory on the first day after all major operations, except in the presence of generalized peritonitis.

One of the most important questions raised at the conference was *whether patients with rheumatic fever could be allowed out of bed safely without waiting for the sedimentation rate and all other factors to return to normal.* This question was left unanswered and Harrison concluded that he did not believe that anyone knew the extent to which the activity of a rheumatic process was influenced by the degree of inactivity of the patient.

One should secure an optimum rest exercise balance which will prevent the development of contractures and atrophy in most cases. Most patients with rheumatoid arthritis should be kept ambulatory except during the most severe exacerbations of the disease, and physical therapy, occupational therapy and physical reconditioning must be used intensively.

Arthritis Produced by Desoxycorticosterone.—*Selye produced,* under certain experimental conditions, by *overdosage with desoxycorticosterone acetate, a polyarthritis resembling histologically that seen in acute rheumatic fever. The joint lesions were produced more readily by desoxycorticosterone in adrenalectomized or thyroidectomized than in intact rats, especially if they were exposed to cold.* Unilateral nephrectomy and sodium chloride treatment increased the sensitivity of rats to many overdosage effects of desoxycorticosterone. The marked similarity between these experimentally produced lesions and the manifestations of rheumatic fever were interpreted as indicating that the adrenal cortex may play an important rôle in the pathogenesis of rheumatic fever. (See Chapter XXVI, Rôle of the Endocrine Glands in Arthritis; and Chapter V, Etiology of Rheumatoid Arthritis.)

Palindromic Rheumatism.—Hench and Rosenberg have described a *frequently recurring form of arthritis which produced no permanent damage to the joints and was associated with practically no abnormal laboratory findings The outstanding features of this syndrome were* multiple afebrile attacks of acute arthritis and periarthritis (and sometimes of para-arthritis) with pain, swelling, redness and disability usually of only one, but sometimes of more than one small or large joint in an adult. The attacks appear suddenly, develop rapidly, usually last only a few hours or days and disappear completely. Little or no constitutional reaction or abnormality is seen by laboratory tests. The great frequency of irregularly recurring short attacks, each with a complete symptomatic remission was the striking feature of the disease. Even after scores of attacks and years of the disease, no significant functional, pathologic or roentgen residues occur despite the transitory presence in at least some of the cases of inflammatory polymorphonuclear exudate in the articular tissues and cavity. (For details, see Palindromic Rheumatism, Chapter XLV, Miscellaneous Forms of Arthritis.)

Vitallium and Leucite Arthroplasty Cups.—*Vitallium, leucite and cellophane have been used successfully as interposition substances in arthroplasty in patients with rheumatoid arthritis.* Vitallium (65 per cent cobalt, 30 per cent chromium and 5 per cent molybdenum) is one of the best metals to employ in bone surgery. It results in no irritation of the surrounding soft tissue or bone and no discoloration or staining of the structures. Leucite and vitallium cups have been used with marked success in arthroplasty of the hip. Tantalum, a little known metal (the seventy-third element in the periodic table) is superior to all other surgical metals except vitallium. It is as strong as steel, resists corrosion and can be stamped, machined and drawn into wire. It appears to be inert in the human body. (For details, see Chapter XVII, Orthopedic Management of Arthritis.)

Hypersensitivity Reactions.—Various individuals have repeatedly suggested that the lesions of rheumatic fever may be due not to a direct action of bacterial toxins or invisible viruses, but may represent the effects of a hypersensitive reaction to bacterial products. Rich and his associates showed that *generalized vascular lesions identical with those of periarteritis nodosa could result from hypersensitivity reactions of the serum sickness (anaphylactic) type.* Acute rheumatic fever has many features in common with the anaphylactic reaction of serum sickness. Recent studies of Rich and Gregory have shown that a type of *carditis similar to that of rheumatic fever may occur in experimental serum sickness.* Others have suggested that *disseminated lupus erythematosus may be primarily a manifestation of hypersensitivity or allergy.* This is discussed further in Chapter XLIII on Rheumatic Fever and in Chapter XXXII on The Rôle of Allergy in Arthritis.

X-ray Therapy in Rheumatoid Spondylitis.—*X*-ray therapy has been used for years in the treatment of rheumatoid arthritis. Usually, little permanent improvement follows such therapy when this is directed at peripheral joints. It has been shown, however, that **roentgen therapy is extremely valuable in the treatment of rheumatoid arthritis of the spine** (rheumatoid spondylitis). Roentgen therapy is the treatment of choice in the early stages of this disease, resulting in marked lessening of pain and stiffness and in permitting the great majority of these individuals to return to work. (See Chapter XL, Spondylitis.)

Pregnancy or Jaundice in Rheumatoid Arthritis.—Hench has re-awakneed interest in the *ameliorating effect of pregnancy upon chronic arthritis, fibrositis and intermittent hydrarthrosis.* He concluded that the physiologic state of pregnancy was markedly antagonistic to the continuance of the symptoms of rheumatoid arthritis. Relief from pains was noticed usually between the fourth and sixth weeks of pregnancy. *In most instances, the arthritic symptoms recurred some weeks following delivery as severely (or more so) than prior to the pregnancy.*

The cause of the relief by pregnancy in some cases of arthritis is speculative. *Such remissions are not unlike those found in patients following the development of a toxic or obstructive jaundice.* A large percentage of patients with rheumatoid arthritis will note a marked lessening of the joint phenomena should an intrahepatic or obstructive jaundice intervene. Unfortunately the rheumatic manifestations usually reappear when the jaundice has cleared. The remissions may be sudden and complete and pain may be ameliorated within a few days after visible jaundice has supervened. These observations again demonstrate that rheumatoid arthritis is not a hopeless, uncontrollable disease.

Various workers have attempted to reproduce a harmless form of jaundice by numerous procedures. (See Chapter XXVII, Effect of Jaundice on Arthritis; and Chapter XXVI, Rôle of the Endocrine Glands in Arthritis.)

Psychogenic Factors in Rheumatic Diseases.—It has been suggested that *anxiety, worry and emotional shock may play an important rôle in the etiology of rheumatoid arthritis.* The term **psychogenic rheumatism** has

been applied recently to indicate those states in which the stiffness, pain, swelling or limitation of motion in muscles or joints is caused, intensified or continued by mental influences. In one recent report, *"psychogenic rheumatism" was claimed to be the most frequent cause of disability in 450 consecutive cases diagnosed as arthritis or an allied organic condition previous to admission to an Army General Hospital.*

Cobb and Bauer concluded that poverty, grief and family worry bore more than a chance relationship to the onset and exacerbations of rheumatoid arthritis.

Psychotherapy, whether used consciously or unconsciously, is often essential to the proper handling of these patients with rheumatoid arthritis, as they are especially amenable to suggestion.

Emotional upsets may cause upsets in the body including pain, stiffness and limitation of motion. Many patients with aching and stiffness in joints and muscles who are considered to have fibrositis are victims of what has been termed psychogenic rheumatism. (See complete discussion in Chapter XXIV, Psychogenic Factors in Rheumatic Diseases.)

Herniated Intervertebral Disks.—During the past ten years, *the subject of herniated intervertebral disks has sky-rocketed to the fore in discussions of low back pain and sciatica.* This has progressed to the point that Dandy recently stated that **recurring attacks of low lumbar backache, with sciatica in the back of the leg, are almost pathognomonic of defective intervertebral disks.** In most instances, the backache and sciatica are *intensified by sneezing and coughing* during the periods of acute pain. Dandy advises that in those individuals whose backaches are frequent and severe, operation is indicated as the latter is without risk and the results in his hands are said to be "excellent and permanent."

With progress of our knowledge concerning the symptoms of herniated disks, these *can be diagnosed from the symptoms and signs alone in almost 95 per cent of cases.* Roentgen examination will differentiate this from *spondylolisthesis* and *congenitally defective vertebræ* (which produce similar symptoms). *In half of the cases, roentgen examination of the lumbar spine will reveal narrowing of an intervertebral space—objective evidence of a defective disk.* It is not infrequent (80 per cent of the cases) to find two or three lower disks to be narrowed. Dandy advises against the use of contrast myelograms in these individuals because the diagnosis can be made more frequently without the myelogram. The main difficulty in differential diagnosis is with spinal tumors whose symptoms may be identical with those of herniated disks (but which occur only about one-hundredth as often as herniated disks). If one suspects a *tumor of the cauda equina,* xanthochromic spinal fluid will usually be found.

Dandy believes that *recurrence of symptoms following removal of a defective disk indicates* either recurrence of the original disk, an additional disk (which was missed at operation) or a tumor of the spinal cord or cauda equina. At present, he advises *removal of the disks as completely as possible with curets, baring the bone;* following this, the joint narrows and the cavity

fills in with connective tissue so that three months later the joint is firm, obviating the necessity of fusion by spinal grafts.

In a recent communication, he stated that recurring attacks of low lumbar backache without sciatica are also due to defective intervertebral disks and should receive the same therapy. This subject is discussed in detail in Chapter LVII on Sciatica.

Postmenopausal Osteoporosis.—Albright and his associates have shown that the *most common etiologic factor in generalized osteoporosis in women is the postmenopausal state.* Postmenopausal arthritis involves particularly the spine and pelvis, with less involvement of the long bones. The skull is almost never attacked. (See Chapter XLII, Radiology in Arthritis; and Chapter XXVI, Rôle of the Endocrine Glands in Arthritis.)

Fall in Prothrombin Level Following Prolonged Salicylate Therapy.—Henigsberger reported the development of nosebleed in 3 cases after taking large doses of aspirin and the frequent occurrence of bleeding in patients with rheumatic fever while they are receiving large doses of salicylates. Experimental evidence suggested that *if salicylic acid is given over a long period to rats or human beings, hemorrhages result with a fall in the prothrombin level. This fall in prothrombin level can be prevented if vitamin K is administered simultaneously with the salicylic acid.* It may thus be advisable to give prophylactic doses of vitamin K if salicylates are to be administered over a long period in large doses. (See Chapter XX, Miscellaneous Items of Therapy.)

Prostigmin.—Trommer and Cohen recently reported *relaxation of muscle spasm in* 13 of 19 cases of *rheumatoid arthritis following the administration of prostigmin.* When given subcutaneously, this acted rapidly and in some cases the action persisted for several days. It was thought that the prostigmin relieved the muscular spasm in rheumatoid arthritis and thereby relieved pain. Small amounts of atropine were given together with the prostigmin to lessen any untoward effects. *Prostigmin does not cure rheumatoid arthritis* and further work now in progress will determine the value of prostigmin in this connection. Related drugs, such as physostigmin, are also being investigated in a similar manner. (See Chapter XX, Miscellaneous Items of Therapy.)

Vitamin E in Fibrositis.—Steinberg reported on the use of vitamin E in fibrositis. He concluded that it was of value in primary fibrositis but that it resulted in very little relief to individuals with secondary fibrositis. Unfortunately, his results have not been substantiated by other workers. (See Chapter XLI, Fibrositis.)

Ultra-violet Blood Irradiation Therapy.—Following the publication in 1934 by Knott and Hancock of the use of ultra-violet irradiation of autotransfused blood, several individuals have employed this therapy in bronchial asthma and in various forms of arthritis. In brief, the method consists of the withdrawal and citrating of approximately one-twenty-fifth to one-thirtieth of the patient's blood, immediately passing it to a chamber with a quartz window against which a high intensity ultra-violet lamp is

held, and then returning the irradiated citrated venous blood to the venous circulation of the patient. It has been claimed that the effects of such ultra-violet irradiation are bactericidal, detoxifying, etc. It is stated by several workers who have employed such apparatus that the therapy is safe and efficient. *There are no controlled observations on the use of this method of therapy so that it cannot be recommended at present except as an experimental procedure.*

Dupuytren's Contracture.—Although Dupuytren's contracture has been known to clinicians for many years, it has recently been noted that *this syndrome appears not uncommonly as a sequel to coronary artery occlusion*, along with pain in the shoulder. It should be remembered that *complete excision of the palmar fascia will usually produce good and uniform results.*

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