Diseases of bones and joints / by Leonard W. Ely.

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LEONARD W. ELY, M. D.

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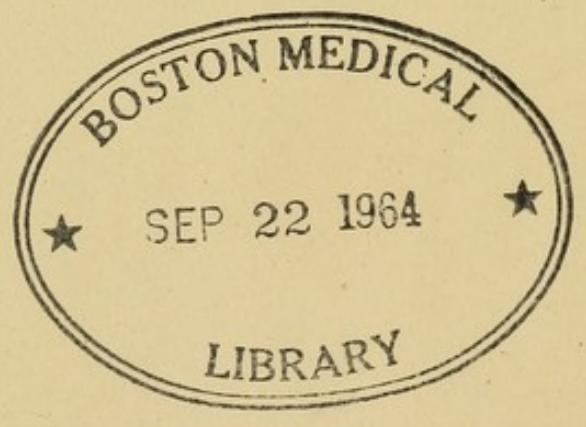

To My Wife.

"Dux femina facti"

PREFACE.

This book is put forward by its author, not without some misgivings. The whole subject of diseases of bones and joints is undergoing a rapid development, and is being transferred from the realm of empiricism to that of scientific study. While we must avoid going too fast, and taking up every idea without sufficient supporting evidence, we must not lag behind with those who complacently regard almost all arthropathies and osteopathies as manifestations of "rheumatism," and treat them in a manner that may be named most charitably as routine.

The work represents an effort to present the subject as briefly as possible, dealing with the various diseases, in the matter of space, very much as their relative frequency. It is intended primarily for the general practitioner, but, contrary to custom, instead of furnishing that long suffering and very important person with a mass of detail, and with many methods of treatment from which he may choose, attempts to lay down broad general principles, with the evidence on which they are based, and then to show how these principles may be applied. In other words, it demands as little faith as possible on the part of the reader.

The book upholds strongly the idea that a comprehension of the pathology of disease is a prerequisite for treatment. It derives whatever originality it possesses in this line from the study of about 120 specimens of bones and joints in the laboratory.

The author acknowledges his indebtedness to Doctors Joel E. Goldthwait, Robert B. Osgood, Frank Mathews and Aller G. Ellis for the loan of photographs and skiagrams, and to Professor Whitman, of the University of Colorado, for very valuable assistance rendered in many ways on numerous occasions.

"No man should try to allude to the greater part of what he sees in his subject, and there is hardly a limit to what he may omit. What is required is that he shall say what he elects to say discreetly, that he shall be quick to see the gist of a matter, and give it pithily without either prolixity or stint of words."

LANE HOSPITAL, San Francisco, 1914.

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CHAPTER I.

THE ANATOMY, PHYSIOLOGY AND PATHOLOGY OF BONES AND JOINTS: GENERAL CONSIDERATIONS.

Structure of Bones and Joints

For a proper understanding of disease in bones and joints, a knowledge of their structure and function is necessary. Without this knowledge we are hampered in our study, and are at the mercy of anyone who advances a therapeutic idea, no matter how absurd and fantastic. Not all that follows is as yet proved, but it represents the results of original investigation and clinical observation, and is submitted as a working hypothesis[1]. We shall confine our attention to the bones and joints of the extremities and of the spine.[2]

Six tissues interest us, namely: 1, Bone tissue proper; 2, Marrow; 3, Periosteum; 4, Cartilage; 5, Synovia; 6, Ligament.

Bone tissue is composed of lime salts and organic material, and is the same wherever it is found. It varies only in its arrangement and in its amount. It is divided into two classes, *i. e.*, dense or compact, and spongy or cancellous, but the bone in each is the same. Dense bone is found at the cir-

(1) See also MacEwen, Growth of Bone, The Macmillan Co., New York, 1912.

(2) The conclusions set forth are based, not alone on clinical experience, but on the examination of some 500 slides of bones and joints.

The Bone

cumference of the shafts of the long bones[1], and generally as a layer on the outside of all spongy bones or portions of bone. The dense bone of the shafts does not suddenly cease on its inner aspect, but is replaced by a small amount of open-meshed bone. The trabeculae become scantier and gradually cease. In other words, the shaft of the bone is not to be regarded as an external layer of dense bone, filled with a cylinder of marrow, but as a mass of bone and marrow differing in their relative amount from the circumference to the center. All through this dense bone are marrow canals. As it approaches the extremities the layer of dense bone tapers off, and is prolonged as a thin layer under the articular cartilage.

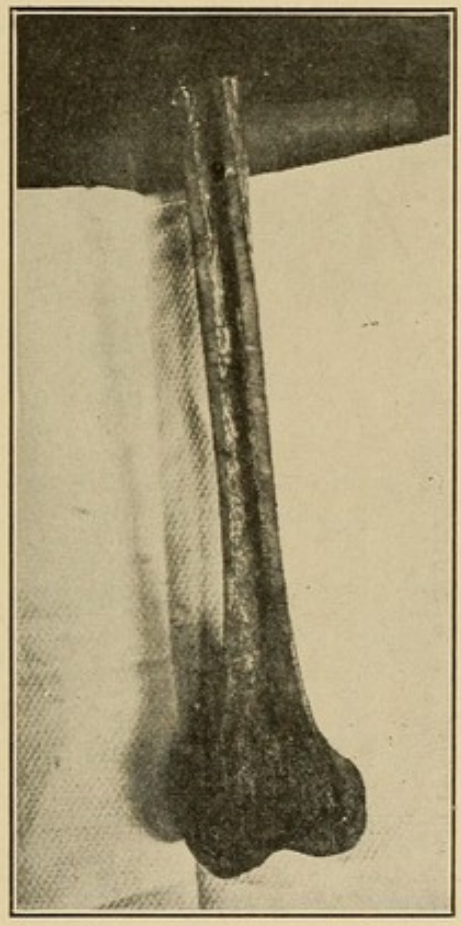
Cancellous bone makes up the bulk of the ends of the long bones, and the bulk of the short and flat bones, the cranial diploë, the vertebrae, the sternum, ribs, carpus, tarsus, etc. A layer of it lines also the shafts of the long bones .

Bone tissue is not subject itself to inflammation or actively to disease, and simply reacts to disease or change of its contained marrow. Usually a mild irritation of the marrow causes an hypertrophy of bone, a stronger irritation an atrophy. If the inflammation in the marrow be very severe, it kills the bone mechanically by shutting off its nutrition. Heretofore in almost all bone diseases the custom of studying the bone tissue itself has prevailed, but this tissue is really of minor importance, like the walls of a factory in which are carried on various activities, like a honey-comb, or the shell of an

(1) The metacarpals, metatarsals and phalanges are classed as long bones.

oyster. The only function of bone is a mechanical one.

All bone is covered by a tissue with which it is intimately connected, that is, by the periosteum or by cartilage. An endosteum has been described as lining the canals and spaces of bone. Of course,



Endosteum

Fig. 1.
Photograph of lower two-thirds of femur of young adult, frontal section. Observe the thin layer of cancellous bone internal to the dense cortex of the shaft.

anything within the bone is endosteum, but there is no special tissue to which this name should be given. Endosteum is simply marrow.

Constant change is going on in the bone, that is, its structure is always changing, and is adapting it-

self to the use to which it is put. If a part be put at complete rest, the bone atrophies, if great work is demanded, the bone hypertrophies, but both processes are passive, and are brought about by the action of the marrow and periosteum. In the long bones the bone tissue may be increased in amount in two ways, first by the ossification of cartilage, and second by productive osteitis. In the latter

Bone Growth

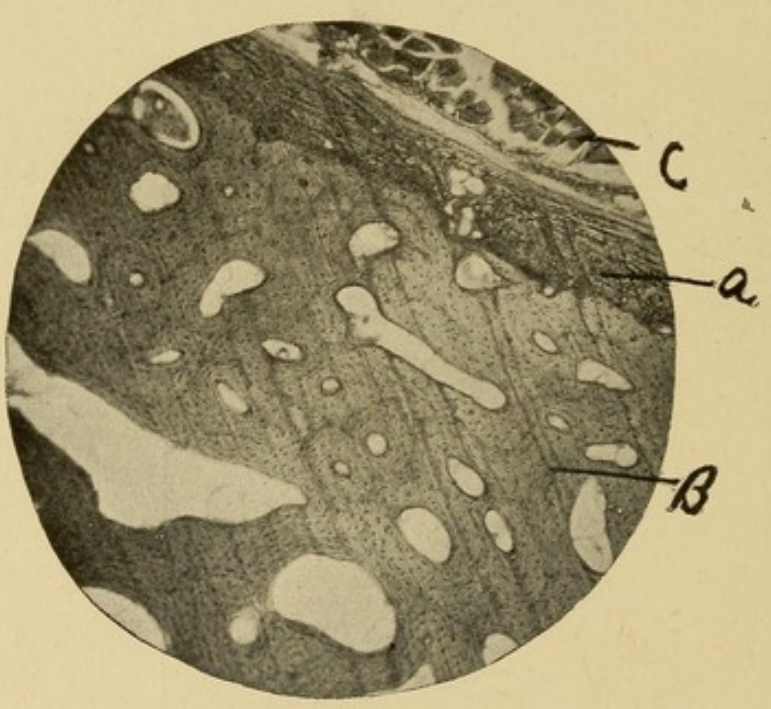


Fig. 2.
Low power photomicrograph. Normal bone and periosteum of lower metaphysis of adult femur. A—periosteum, B—bone, C—muscle. Note bone cells—small black spots in the bone—and the character of the bone near the surface.

case the bone trabeculae are seen under the microscope to be edged by small, round marrow cells—osteoblasts.

Bone tissue has been said to atrophy in either one of two ways, first by absorption of lime salts—halisteresis—and second by a rarefying osteitis. The best modern authority denies the existence of the former process. In the latter case one sees,

under the microscope, giant cells—megaloblasts or osteoclasts—lying in depressions in the bone trabeculae—Howship's lacunae—and eating the bone trabeculae away. When the bone has been killed by interference with its nutrition, these depressions can often be seen in the dead pieces, but the giant cells are no longer in them. Sometimes on one side of a trabecula, osteoclasts are devouring the bone,

Bone Rarefaction

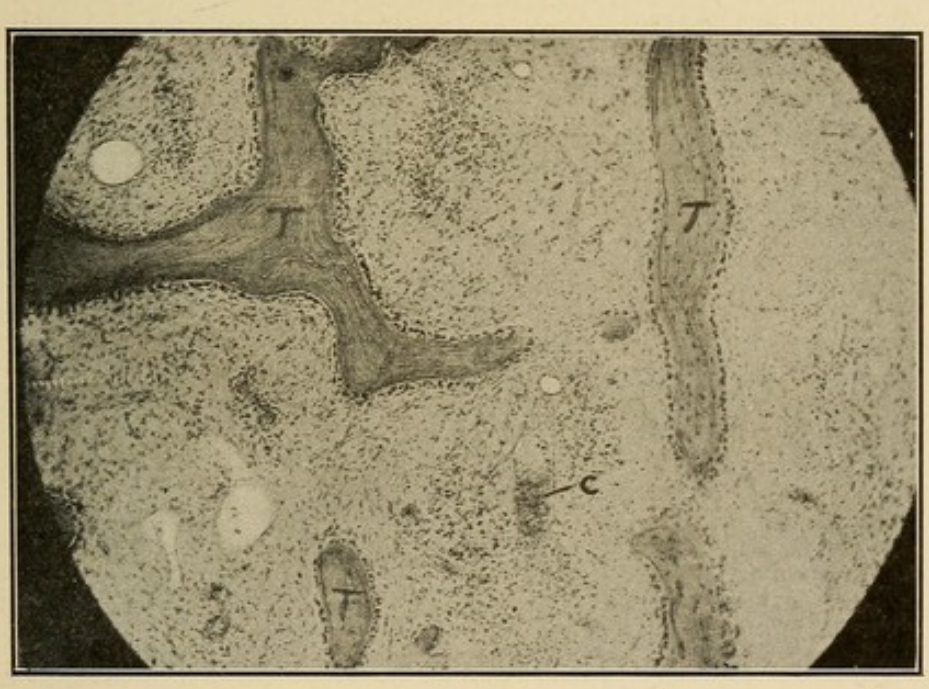


Fig. 3.

Photomicrograph of section from a case of acute suppurative arthritis of knee. T—bone trabeculae, along whose margins is a row of osteoblasts, showing proliferative osteitis. C—colony of pus cocci in a blood vessel.

Zeiss apochromatic 16 m.m., ocular 4. Tube length 49 cm.

and on the other side osteoblasts are laying down new bone. Rarefaction probably may take place, however, without the presence of osteoclasts. Dead bone is easily recognized by the absence of bone cells. Macroscopically dead bone is recognized by its roughness, its change of color, and by the fact that it does not bleed when divided.

MARROW.

The Marrow

Normal marrow is of two kinds, the red or lymphoid, and the yellow or fatty. The former is to be regarded as typical marrow. It consists of a reticulum of connective tissue, in whose meshes lie large numbers of most diverse cells. The derivation, function and destination of the cells are still

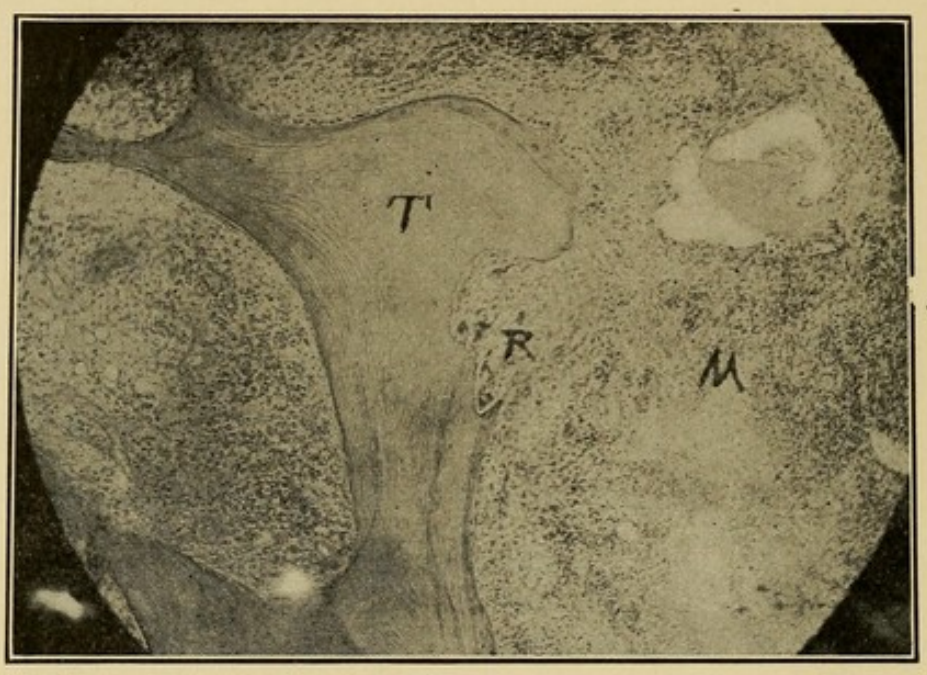


Fig. 4.

Photomicrograph of a section from a case of acute suppurative arthritis. T—trabecula; M—marrow. Most of the trabecula is dead, but its upper left hand and lower right hand borders show evidences of a productive osteitis and a deposit of new bone. At R. a piece of the trabecula is seen to have been eaten away by the osteoclasts lying in their lacunae. The dead bone is distinguished by the absence of bone cells. The marrow is the seat of an intense inflammation.

Zeiss objective 16 m.m., ocular 4.

only partially known[1]. Some are regarded as blood cells in embryo. The contained cells are what give this kind of marrow its name. Yellow marrow is marrow which contains much fat, and very

(1) See W. E. D. Dickson, The Marrow, etc., 1908. Longmans, Green & Co.

few other cells. In old age the bone marrow undergoes a myxomatous degeneration.

In adults lymphoid marrow is found in spongy bone, yellow marrow in dense bone. In children lymphoid marrow is found in the shafts, but, as

Distribution of the Marrow

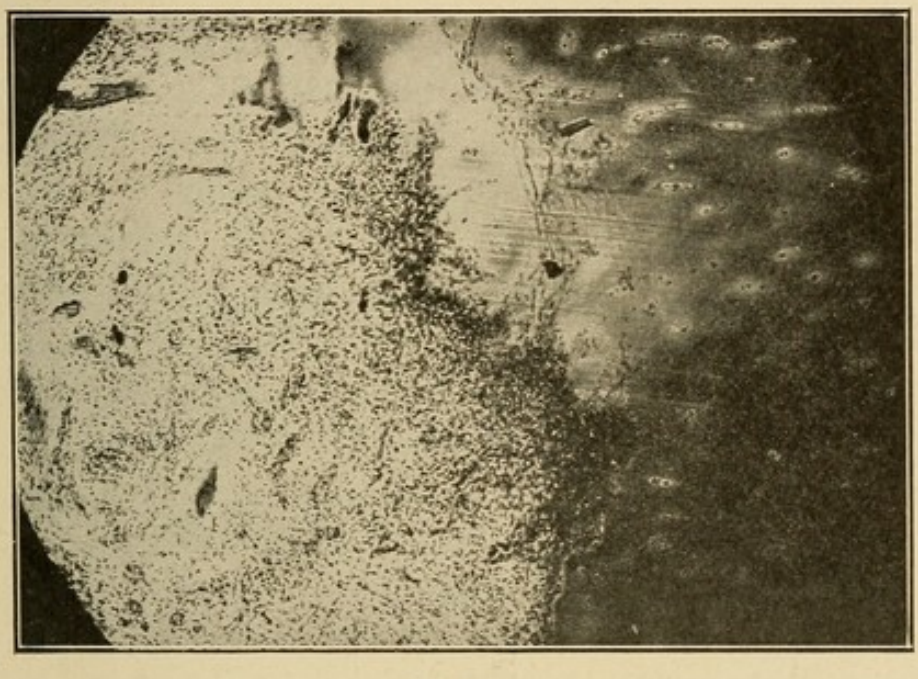


Fig. 5.

Photomicrograph of bone and cartilage: from a section of an acute purulent arthritis, following operation on a case of suppurative inflammation of the shaft of the tibia. Degenerating cartilage on the right. Bone marrow on the left. Observe how the inflammatory process in the marrow is eating its way into the cartilage, and how it has already consumed most of the bone. Only a few small bone trabeculae are left. Zeiss apochromatic 16 m.m. Ocular 4. Tube length 49 cm.

time goes on, it is replaced by fatty marrow. As has been said, the central canal of the long bones is filled with marrow, but so also is the dense bone of the cortex, to a less extent. Wherever there is bone there is marrow, and the marrow is not a passive filling for the bones, but their active constituent, building them up and tearing them down. In it can be discerned the customary evidences of disease. In tuberculosis the marrow exhibits the

Physiology of the Marrow

classic tubercules, in active inflammation it shows engorgement of blood vessels, pus cells, colonies of bacteria, etc., in chronic disease peculiar appearances later to be described.

Pathology of the Marrow

When we would find out why any disease locates itself where it does, we study the structure of the tissues in that locality. Thus, carcinoma often starts at a spot where the character of the epithelium changes, gonorrhea affects mucous membranes with a certain kind of cells. Following out this theory in bone and joint disease, if we recognize that the marrow is the essential factor in all bone diseases, and if we bear in mind the situations in which each kind of marrow is found, we shall readily understand why certain diseases affect the shafts of the long bones, certain others their extremities, and possibly still others, both shafts and extremities. We shall put aside such vague terms as "rapid growth predisposing to infection," "congestion following injury," and shall rather study the constituents of the marrow at various periods of life, and their vulnerability to certain kinds of infection.

THE PERIOSTEUM.

The Periosteum

The periosteum is composed of two layers, an inner or cellular, and an outer or fibrous layer. The inner, very thin in adults, thicker in children, bears a strong analogy to the marrow of the subjacent bone, has similar functions, and is vulnerable to the same diseases. It is to be regarded as a layer of marrow on the outside of the bone, and is not separate or distinct from the marrow on the inside. (See Fig. 2.) The two communicate by small channels or canals in the bone. Both nourish the

bone tissue. This is the reason why, when we strip the periosteum from the bone in operations, the bone does not die, but when the periosteum is stripped off and raised up in acute osteomyelitis, the bone usually dies, because its nutrition has been cut off by a simultaneous inflammation in the "internal" marrow.

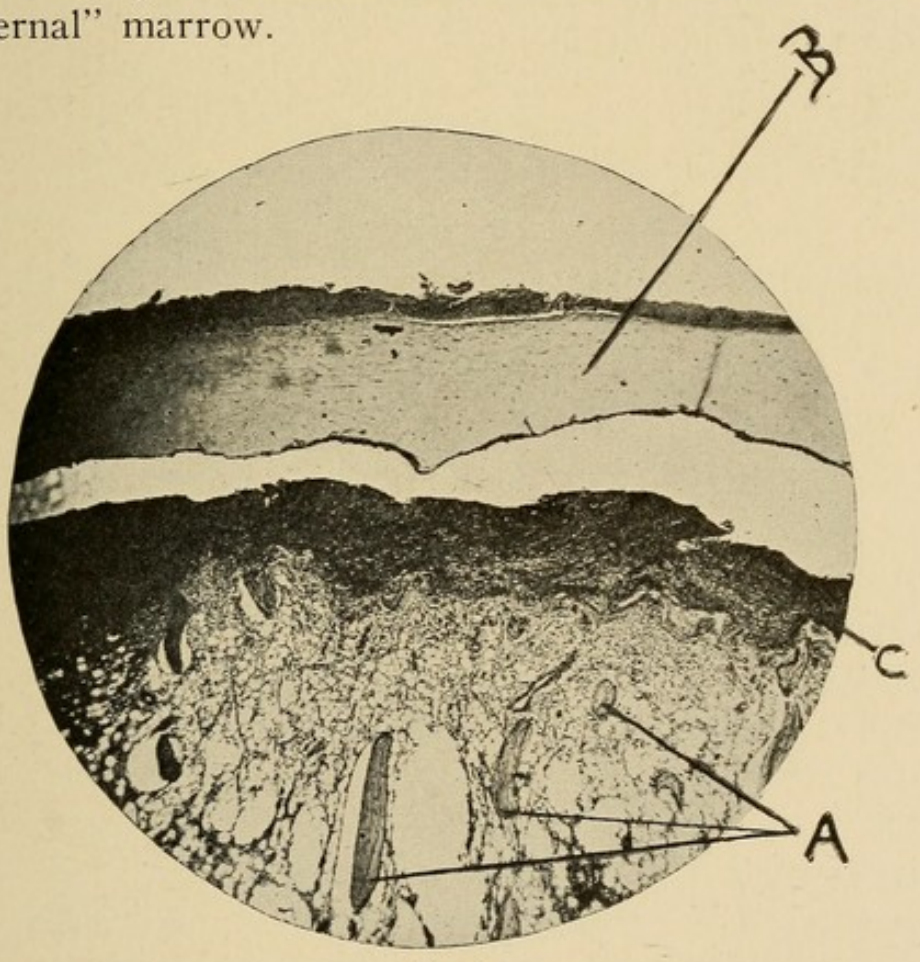


Fig. 6.
Photomicrograph showing tuberculosis of the marrow (C) directly beneath the cartilage (B). The cartilage has been lifted off *en masse*, and its surface is fibrillated. A—bone trabeculae.

The outer layer of the periosteum serves as an envelope for the bone, and may be disregarded except for its mechanical function.

At the ends of the long bones, where the joint cartilage begins, the fibrous layer of the periosteum is continuous with the ligament. For our purposes

the inner layer may be regarded as continuous with the synovia.

THE JOINT.

We now come to a consideration of the joint, and shall take for our example a simple joint between the ends of two long bones. All others are only modifications of this.

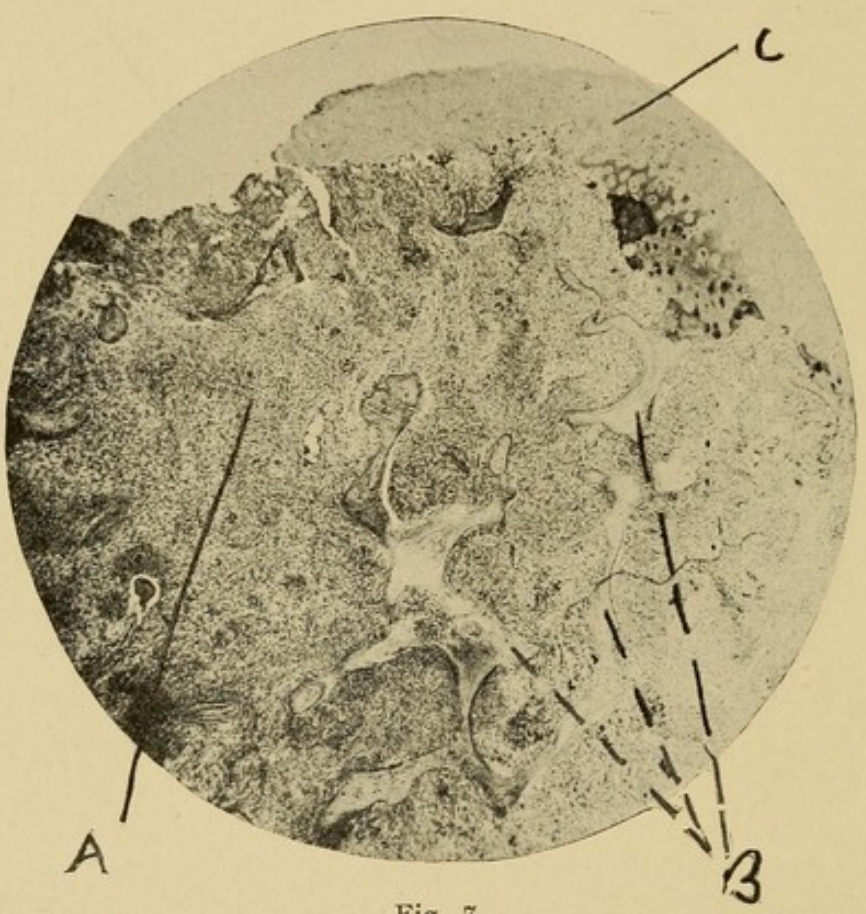


Fig. 7.

Photomicrograph showing intense tuberculosis inflammation of the marrow (A). B—dead bone trabeculae. C—degenerating cartilage. The inflammation is making its way into the marrow at the margin of the articular cartilage, destroying the bone and cartilage as it advances. Low power.

Definition

A joint is a closed cavity between two bones, bounded by two tissues, the cartilage and the capsule. Where one begins the other ends.

Cartilage may be regarded as bone in embryo and in potentiality. It is composed of cells and base-

ment substance. The latter appears to be homogeneous, but this homogeneity, due to its cement substance, is only apparent. If the nutrition of the cartilage is disturbed, or if the joint be put at rest, the real fibrous nature of the basement substance becomes evident. Deep in the cartilage the fibres are at right angles to the surface, but more superficially, parallel to it. I have never been able to identify a perichondrium of an articular cartilage.

The Cartilage

These fibres in the cartilage have often been mistaken for layers of fibrin in diseased joints. The importance and frequency of fibrin deposition in diseased joints has probably been exaggerated. A substance is occasionally seen precipitated on the surface of the synovia, that looks like fibrin, but does not take the fibrin stain, though in hemorrhagic joints fibrin is a natural constituent of the exudate.

The cartilage, like the bone, draws its nutrition from the marrow directly beneath it, and probably also, at the sides from the synovia (or periosteum). It contains no blood vessels of its own. Its role in disease is still a matter of debate. Some authorities maintain that it is subject to inflammation, others that it is not. Some ascribe to it a principal part in chronic joint disease. My personal opinion is that its role is largely a passive one. The only sign of activity I have been able to distinguish is a proliferation of its cells. The deposit in it of crystals of biurate of soda in gout is really a passive process. Mysterious chemicals floating in the blood stream must not be regarded as affecting the cartilage in any way. No fluid in the joint can have any effect upon it whatever.

The Role of the Cartilage

The joint cartilage forms an absolute barrier against the extension of disease from the bone into the joint, and *vice versa,* until it has been perforated by the cutting off of its nutrition.

Until this perforation takes place the only method of extension must be at the periphery of the cartilage, where the inner layer of the periosteum is continuous with the synovia. Much the same may be said of the epiphyseal cartilage.

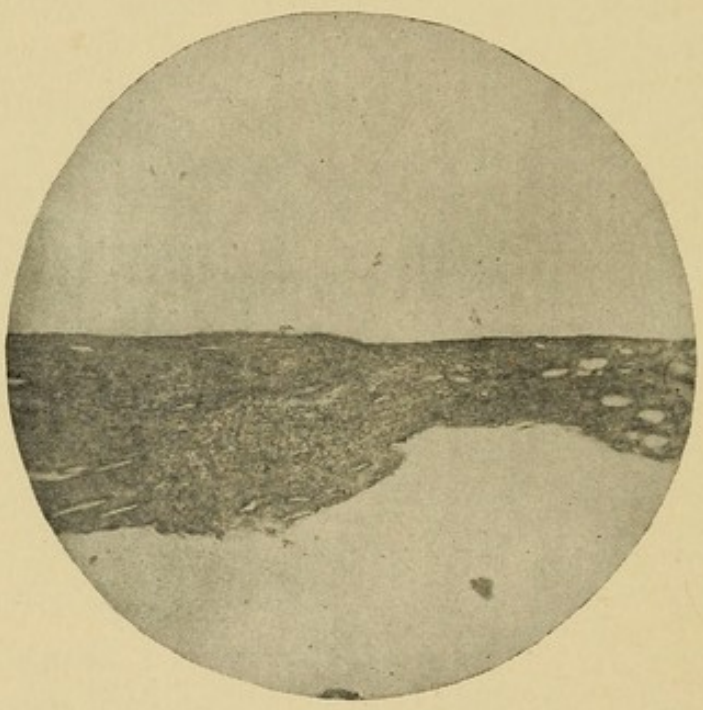


Fig. 8.

Capsule of a normal knee joint, low power. Joint surface above. Note the vascular spaces. Zeiss, objective aa. Ocular 3.

Changes in the Cartilage

The joint cartilage atrophies and hypertrophies very much as do the bone trabeculae, namely, as the result of disease in the bone marrow. In addition, the cartilage reflects changes in the synovia. Only on this hypothesis of the passive role of the cartilage can the changes in it be explained.

THE CAPSULE.

The Capsule

This, as we have seen, is to be viewed as the continuation of the periosteum, and, like it, consists of two layers, an inner or cellular, and an outer or fibrous layer. The former is known as the synovial membrane (or synovia), the latter as the ligament, but there is no sharp dividing line between them. They are not separate structures, in point of fact. The inner is simply a differentiated, cellular layer. For the purpose of study, however, it is well to regard them as distinct, for their reaction to disease is entirely different. The synovia is active, the ligament passive. As far as their reaction is concerned, the synovia is marrow, the ligament is periosteum.

THE SYNOVIA.

The Synovia

The synovia, defined by Quain as belonging to the lymphatic system, is, in its simplest form, a layer of round cells lining the capsule of the joint. It is not a true pavement endothelial membrane like the peritoneum. For much of its extent it is smooth, but in the recesses of the joint it is thrown into folds or villi, and has the appearance of epithelium. In synovial inflammations the round cells proliferate, sometimes enormously, and the synovia may undergo a general villous proliferation. The proliferation is to be regarded as the response of the synovia to an irritation. It may take place as the result of disease in the membrane itself or of any mechanical irritation in the joint cavity. The synovia then consists of a thick instead of a thin layer of cells, and resembles typical lymphoid tissue.

Such a proliferation is found in tuberculosis, and in certain other infectious processes.

A significant characteristic of the synovial membrane is the presence of lymph spaces in the connective tissue beneath.

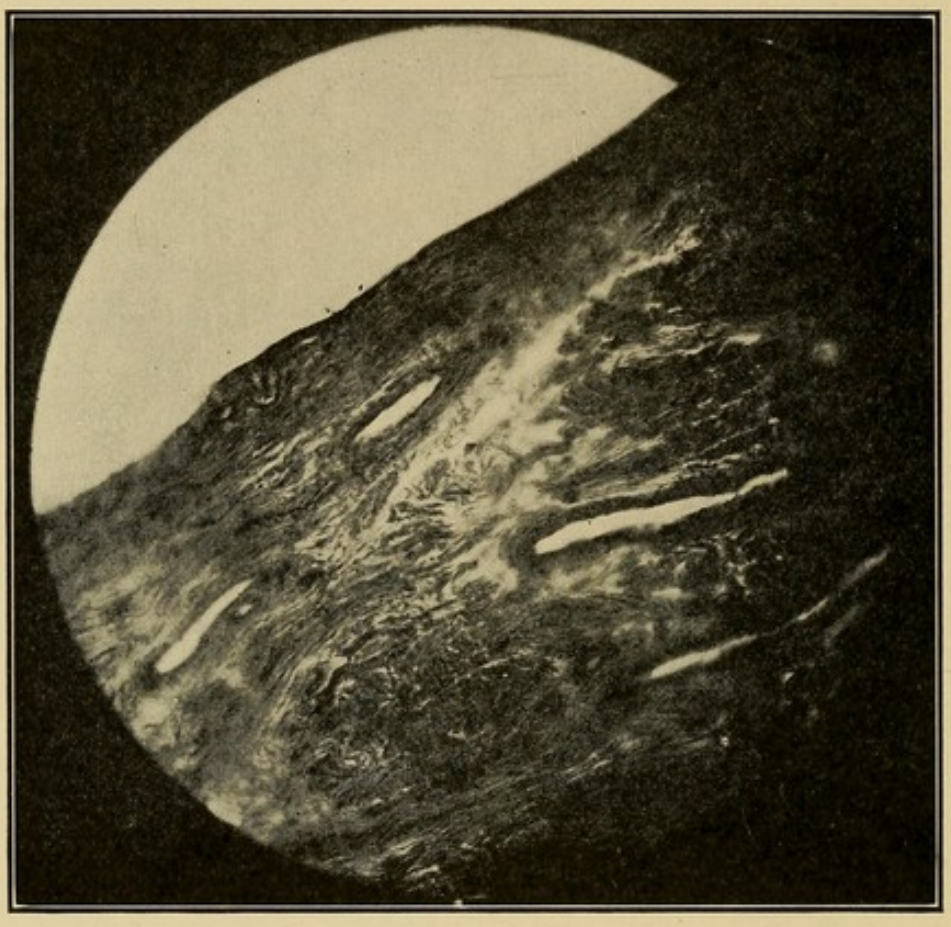


Fig. 9.

Photomicrograph of normal joint capsule, rather high power. Joint surface above. Note the vascular spaces.

The opinion formerly prevailed that the synovia was a closed sac, extending out over the cartilage, but this is not a fact. It is a section of a tube, ending at the border of the joint cartilage, with which it is, to a certain extent, continuous. The structure of one gradually shades off into that of the other. If one extends, the other recedes. When

the joint is immobilized, the synovia encroaches on the cartilage, as if automatically to obliterate the joint, but the joint is never actually obliterated thus, unless the cartilage be destroyed by disease. Probably no "adhesions" form, strictly speaking, in a normal joint that is immobilized for any reason. Ankylosis, in other words, is never caused by immobilization, but by the disease or injury for the treatment of which the immobilization is used.

Fig. 10.

Low power photomicrograph. Capsule from a normal knee joint of an adult man dead of alcoholism. The specimen is from a recess of the joint. Note the folds of the synovia. Zeiss objective, aa. Ocular 3.

Beware of Infection of the Synovia

In all operations on joints or in wounds of them, it is the infection of the synovial membrane that is to be feared.

THE LIGAMENT.

We have already discussed the ligament. It is composed of dense fibrous tissue, holds the ends of

The Ligament

the bones together, checks motion, and may be disregarded except for its mechanical functions.

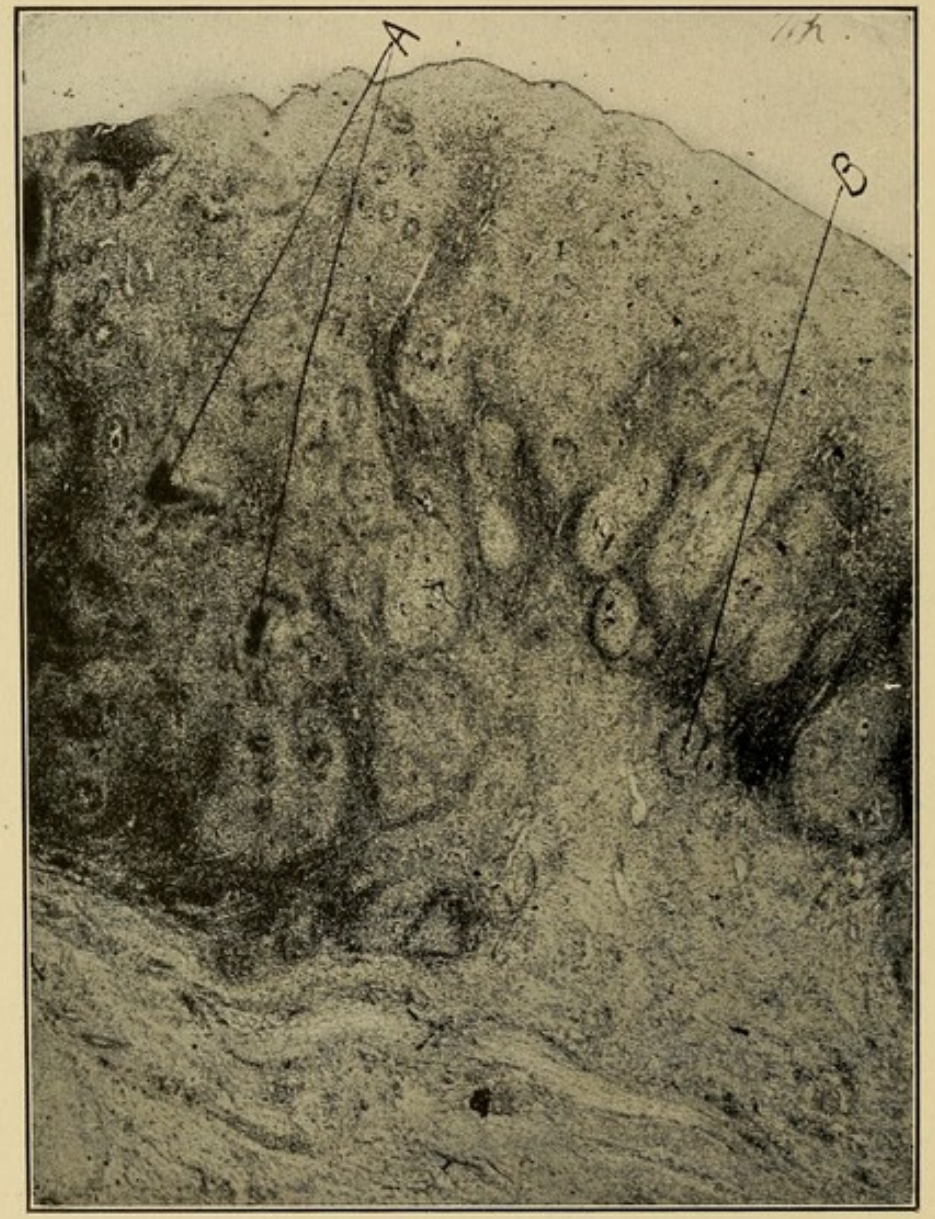


Fig. 11.

Synovial tuberculosis. Section from synovial membrane showing typical tuberculosis ("magnified") about 20 diameters. A—areas of necrosis. B—Giant cells.

We recognize, then, in bones and joints three active tissues, namely, the marrow, the synovia, and the inner layer of the periosteum; and two passive

tissues, the bone and the cartilage. The active tistues are continuous with one another and present similarity in structure and in their reaction to disease. The disease may start in one and spread to another. Certain diseases show a predilection for one, certain for two, and certain affect all three. Thus, "acute articular rheumatism" affects the synovia, syphilis the periosteum, the marrow, and

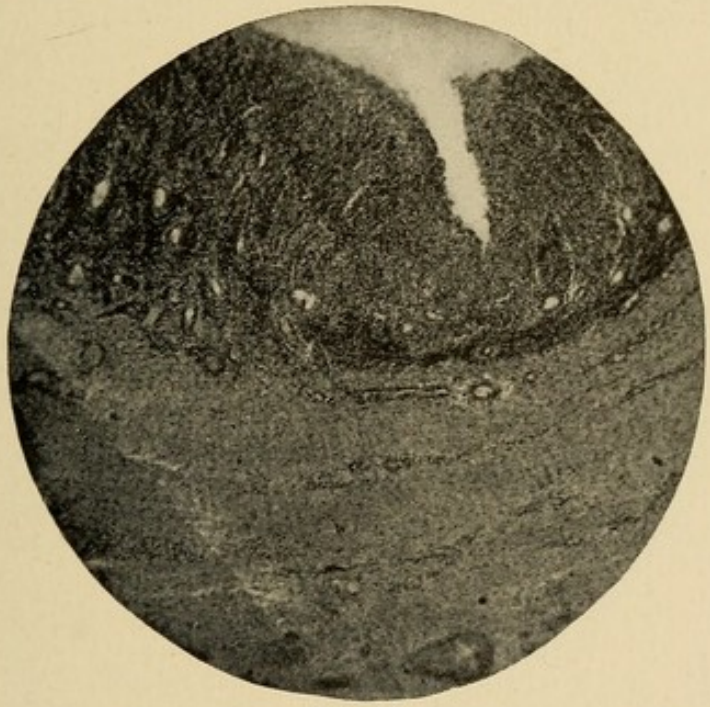


Fig. 12.

Photomicrograph of proliferating synovia. Zeiss objective aa. Ocular 2; from a case of probable syphilitic arthritis of the knee, wrongly diagnosed as tuberculosis, and secondarily infected following operation.

rarely the synovia, gonorrhea, the synovia, and rarely the marrow and the periosteum, tuberculosis and pus infections all three. The bone and the cartilage are continuous with each other, and their reaction to disease is similar also. They form the stroma, the others the parenchyma. The bone is the honeycomb which the bees have built up. As for the ligament and outer layer of the periosteum,

they are the hive with which neither the bees nor the honeycomb have any active connection.

Peculiarities of Disease in Bones

It must never be forgotten in dealing with bones that the morbid processes in them run their course in the marrow, locked up in a firm rigid case or shell. The effects of the disease are made manifest upon this shell, but one must not make the error of regarding these effects as the disease itself. Again,

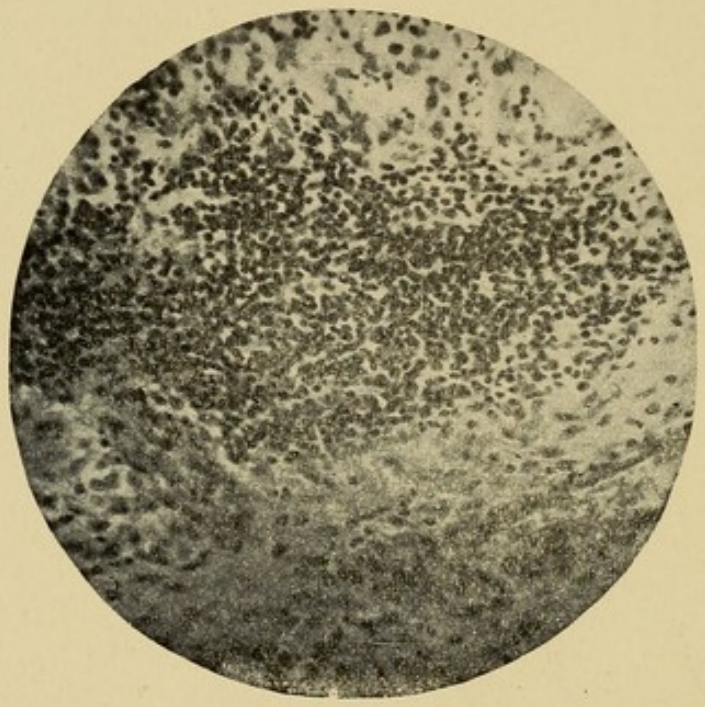


Fig. 13.

Proliferating synovia. Same case as Fig. 12. High power, Zeiss, D.D., ocular 1. Inflammation, round cell infiltration.

after an acute disease has run its course in the bone marrow, healing may be prevented by the mechanical obstruction afforded by the dead bone shell.

Synovial Folds

Certain joints have a structure more complicated than that we have described. Several bones may enter into their formation, or they may contain pouches and intra-articular ligaments. The intra-articular ligaments are covered by a synovial mem-

brane, which passes in from the side of a joint, but not over the cartilage. Folds of the membrane also pass across various joints, and in certain diseases these folds partially divide the joint off into pockets or compartments.

In the joints of children the epiphyseal cartilages may influence the spread of disease. They serve as a barrier to protect the joint from invasion in disease of the shaft or of the metaphyses[1], and *vice versa.*

Let not the "practical" surgeon dismiss the study of bone and joint pathology as not worth while. It is the most practical side of our subject. In those regions where it is neglected, the proportion of one-armed and one-legged persons will be found very high. The attempt to advance medical knowledge by means of clinical experience is like that to accumulate a fortune by speculation—*successful once in a while.*

(1) Metaphysis is the name given to that part of the expanded end of the long hones between the shaft and the epiphseal line.

CHAPTER II.

ACUTE ARTHRITIS.

By acute arthritis we mean an inflammation of the synovial membrane. In certain forms, as we shall see, this may be accompanied by an inflammation of the marrow of the bone ends entering into the formation of the joint. To separate acute from chronic inflammation is not always an easy task. It seems that in these lymphoid tissues, as in others, an acute inflammation may occur, and may persist until the cause of it is removed. Any sharp division is not practicable. Some cases are hard to classify properly.

Classification

Etiology. Two main classes are immediately apparent, *i. e.,* traumatic and infectious, and they differ so radically that they merit separate descriptions.

TRAUMATIC ARTHRITIS.

Under this head we shall consider all cases of joint inflammation caused by sprains, "strain," contusions, or by abnormal bodies in the joint, e. g., displaced fibro-cartilages, "joint mice," hypertrophied fringes. Absolutely clean penetrating wounds are also to be considered among the causes. An arthritis accompanies dislocation and intra-articular fractures, but sinks into insignificance as compared with the causal lesion.

MORBID ANATOMY.

The changes in the joint are the result of the presence of the fluid poured into its cavity from the wound in its capsule. This fluid may be serous or hemorrhagic. If the latter, then flocculi of fibrin may later be precipitated. The synovia is injected, reddened, and often succulent and thickened. More or less villous proliferation may be present as the result of irritation. If the joint be immobilized for any length of time, the articular cartilage may become somewhat fibrillated in its structure, and possibly the synovia may encroach upon it at its border. These are the essential features of the process. As long as no infection is added, the bone is usually not involved in the inflammation. Fractures and dislocations have their own pathology, which will not be discussed here. If the bone ends be injured, an aseptic necrosis of the bone and cartilage may be initiated, that possibly may be the starting point of a chronic joint disease[1], otherwise I do not believe that a chronic process ever results, unless the injury which has caused the arthritis continues to act, as with dislocated semi-lunar cartilages or loose bodies in the joint.

Reaction of the Joint Tissues

SYMPTOMATOLOGY.

Pain and restriction of function are the cardinal symptoms. The contour of the joint is wont to be changed, if the joint be superficially located. Fluctuation is easily detected in certain joints, e. g., the knee and elbow, with more difficulty in certain others, e. g., the hip and ankle. The whole pouch

(1) Vide Axhausen, Archiv für klinische Chirurgie.

on the front of the knee may be distended, causing the patella to float. In the elbow fluctuation is to be sought posteriorly at the sides of the triceps tendon. The pain is made much worse by motion, especially by motion which puts the injured structure on the stretch, and by pressure over the damaged structures. Usually the joint is held in semiflexion. Sometimes the synovia may be sensitive wherever it is accessible to the examining finger.

Ecchymosis is rare unless a fracture is present. The fracture may consist of the tearing off of a very minute spicule of bone.

Prognosis

The *prognosi* as a rule is good, unless the bone has been damaged. If the exudate have been hemorrhagic, some adhesions may remain as the result of the precipitation of fibrin. Usually the fluid is slowly absorbed, and the joint returns to its normal state.

The diagnosis is usually a simple matter. The symptoms supervene immediately on an injury, or a few hours later. The main thing to establish is that the joint was absolutely normal at the time of the trauma, in order that we may exclude a disease which has simply been aggravated by it. The second essential is to exclude fracture by careful examination and by an *x*-ray plate.

TREATMENT.

The first indication is to restrict as much as possible the formation of the exudate in the joint, especially in a joint that has a capacious cavity, like the knee. This we do by pressure, strapping, bandaging on a splint, etc. Heat and cold are also useful. These measures need not be continued more than a

few hours. Later the most important principle is rest, not necessarily rest for the whole joint, but rest for its damaged part. In the later stages, massage, baking, etc., may be necessary, but very rarely are.

The *knee* is usually damaged in its lateral ligaments, especially the internal, and *when it is in flexion.* Therefore our chief concern, after the acute symptoms have subsided, is to keep the joint in extension, in which position it is rarely injured. We accomplish this by diagonal criss-cross strapping with strips of adhesive plaster about ten inches in length and an inch or two in width, quite firmly applied over the front and sides of the knee, reaching from a point below the joint to one above the top of the quadriceps pouch. This strapping not only prevents flexion, but also exerts pressure. The patient is allowed to go about, preferably with a cane. His comfort is a good indication of the efficacy of the treatment. If he suffer pain the treatment is not sufficient. In the severer cases, we may be compelled to immobolize the joint at once. In such case subsequent massage and baking will be more frequently necessary.

Sprain of the Knee

If a semi-lunar be torn, we try these palliative measures for a while, in the hope that the lesion will heal. If the injury recur often, the torn piece of the semi-lunar or the whole semi-lunar should be removed. There are undoubted cases of damaged semi-lunar cartilage, however, that have ceased to occasion trouble even after years of recurrent attacks, and the knee joint should never be lightly invaded with a knife. If the patient be averse to an operation, a Campbell brace may be applied,

Torn Semi-Lunar Cartilages

allowing about thirty degrees of flexion—enough for comfortable locomotion, but preventing side strain.

Sprain of the Ankle

A sprained ankle is usually caused by a twist of the foot inwards, damaging the external lateral ligament. The treatment is simple and most satisfactory with the well-known Coterell strapping. The foot is held in dorsal flexion and abduction. The plaster, torn into strips about one inch wide, and of two different lengths, is to be ready at hand. The first strap starts just behind the metatarso-phalangeal joint of the little toe, runs around behind the heel and ends about the middle of the inner side of the foot. The second starts about the middle of the outer surface of the calf, passes down the leg, under the heel, and ends at about the level of the internal malleolus. The third overlaps the upper half of the first, the fourth the anterior half of the second, and so on until the external surface of the ankle and foot is covered. Finally the strapping is reinforced by short strips in various directions over the injured ligament and over the heel. The patient usually walks with comfort on the next day.

Repeated Sprain

If the ankle be sprained frequently, the foot should be strapped at right angles in extreme dorsal flexion, for the joint is not very vulnerable to a twist unless it is in plantar flexion.

Two long, wide strips pass from the upper part of the leg, down its external aspect, under the sole and up the inside. Over them a figure-of-eight is applied to the ankle with a narrow strip of plaster. The patient wears also a long, broad heel and a broad sole.

The elbow suffers rarely from traumatic arthritis, and is difficult to strap satisfactorily. A better plan is to put the forearm in a sling.

Criss-cross narrow strapping put on diagonally over the shoulder, usually avails to relieve the dis-

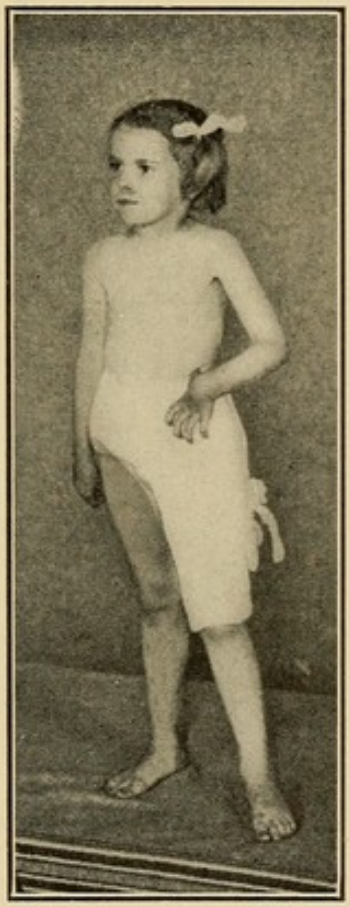


Fig. 14.

Short plaster spica. The tied bandage at the side is the "scratch" bandage, which passes next the skin under the plaster, and keeps the skin in condition. It is drawn up and down every day, giving the patient a dry rub.

comfort in mild cases of inflammation in this joint. The possibility of the presence of a Duplay's bursitis must not be overlooked.[1]

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(1) Codman, Boston Medical & Surgical Journal, May 31, 1906; May 18, 1911, etc.

A broad circular strap does well for arthritis in the wrist. If the sensitiveness be in the region of the carpal navicular, a skiagram of both wrists should be taken to rule out fracture of that bone.

Strapping is difficult to apply in the hip also. Probably rest and massage are our best expedients. If the inflammation be very severe, a short plaster of Paris spica may be necessary.

Finally, let a word of caution be said. If a supposed case of simple traumatic arthritis does not heal up completely in a few weeks, a diligent search should be made for the reason. It is most unwise to lull ourselves into a false security with a diagnosis of "rheumatism."

THE SPINE.

The joints of the spine are occasionally the seat of a traumatic arthritis, and the inflammation gives rise to what is generally regarded as lumbago. Lumbago is usually nothing more than a chronic sprain or "strain." The pain may be in the nature of a dull ache or of a "catch." If the lesion is a severe one, some lateral curvature may be present. Strapping, massage or ironing out with a hot flat-iron over several layers of blanket will usually give relief. If the symptoms persist, a light spinal brace may be worn in addition.

A traumatic arthritis of the lumbar spine or of the sacroiliac joint may be the cause of a sciatica. The treatment is much the same as for lumbago. If the sacro-iliac joint is subluxated, the displacement must be reduced. "Idiopathic" sciatica is very rare, if it ever occurs.

HEMOPHILIAC JOINTS.

These should really be classed with diseases of traumatic origin. Probably as the result of a slight joint injury in a person of the hemorrhagic diathesis a bloody effusion is poured out into a joint, and the changes in the joint tissues are due to mechan-

Haemarthrosis

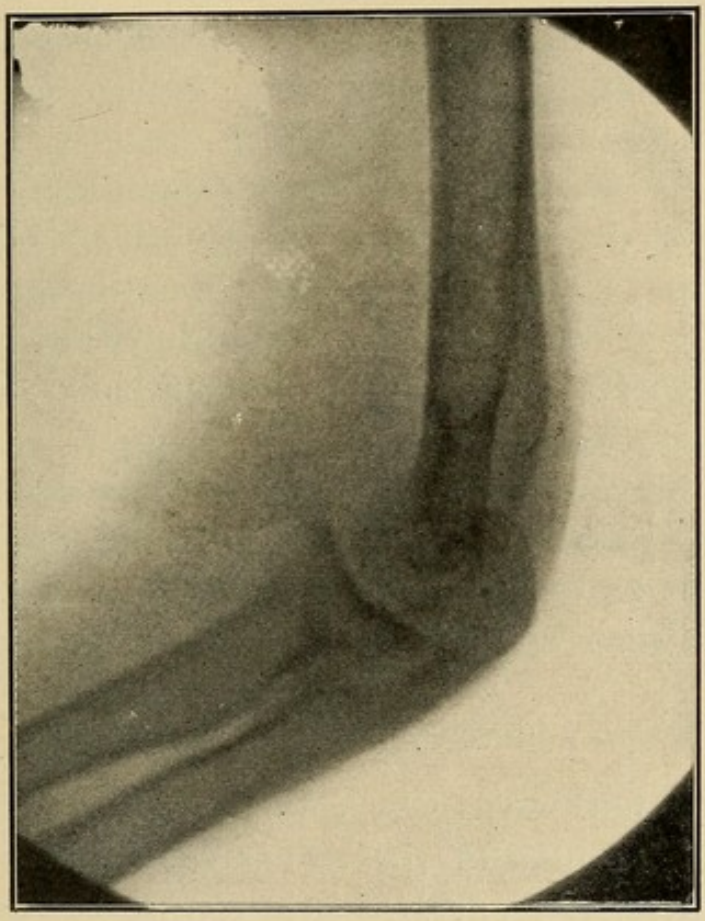

Fig. 15.

Old haemarthrosis of elbow. Several joints were involved in this patient. But for the history the case might easily be confused with tuberculosis. Note the irregularity of the joint outline, and the rarefaction of bone.

ical irritation by this effusion. The synovia proliferates, sometimes enormously, and its internal surface may be shaggy, with the enlarged synovial villi branching like moss on a rock. Later the precipitation of fibrin from the exudate gives rise to

dense and firm adhesions. The joint may be puffed up "like a balloon," as one patient expressed it. It fluctuates, and sometimes crepitates softly. Pain is usually insignificant. The chief complaint is the interference with function by the fluid.

A sudden, fluctuating, comparatively painless swelling of a joint, following a slight injury, should always awaken a suspicion of hemarthrosis, and should instigate a search for a family history of "bleeders."

Treatment: Rest, pressure and cold applications constitute the local measures. Injections of horse serum, in case of need, diphtheria antitoxin, rabbit serum, and normal human serum have their advocates. Direct transfusion of human blood has also been tried with success.[1]

In the late stages, when fibrous adhesions have formed, unless we are sure of our preventive measures against a recurrence of the hemorrhage, no mobilizing operations should be undertaken.

SCURVY, SCORBUTUS.

Scurvy

While not, strictly speaking, a joint disease, scurvy is akin to the preceding, and merits a few words here. As one sees it in civilization this disease occurs with a moderate frequency in bottle-fed infants, especially in those fed on sterilized or condensed milk. It causes hemorrhages beneath the periosteum, usually near the joints, manifest by exquisitely painful swellings. The child screams

(1) Steele, Yale Medical Journal, 1910-11, XVII, 326.
Wells, Boston M. & S. J., 1911, 716.
Moss, Johns Hopkins Hospital Bulletin, 1911, XXII, 272.
Weil, Presse med., 1905, XIII, 673.
Welch, Am. Journ. Med. Sciences, 1900, 800.
Lambert, Med. Record, 1908, Vol. 73, 885.
Murphy, Boston Medical & Surgical Journal, 1908, 865.

with pain when it is moved. Hemorrhages into the skin and mucous membranes, and swollen, bleeding gums may accompany the bone symptoms.

The treatment, eminently satisfactory, consists of giving raw milk, beef juice and fruit juice.

GOUT.

Gout

The joint inflammation occurring in gout is really a form of traumatic arthritis, caused by the deposit in the articular tissues of uratic material. The acute inflammation is the reaction of the synovia against the foreign material. Gout usually affects the metatarso-phalangeal joint of the great toe, comes on in attacks, and has its well-known constitutional symptoms and often its chalky deposits in other parts of the body, especially in the external ear. The poorly marked cases may occasion confusion with mild infections.

The treatment of gout belongs for the present in the domain of internal medicine.

ACUTE INFECTIOUS ARTHRITIS.

Acute Infectious Arthritis

Cause

The chief infective agents are ordinary pus cocci, the gonococcus, the typhoid bacillus and the pneumococcus. The streptococcus (or diplococcus) rheumaticus of Poynton and Paine will perhaps secure a permanent place as a causative agent. We find the disease occurring as the result of punctured wounds, compound dislocations, and compound intra-articular fractures, infection of tuberculous sinuses, as an extension of suppurative processes from the shafts by operation or spontoneously, etc. It may be a manifestation of a general pyaemia, and it may be a metastasis from some unknown source.

It sometimes follows the acute infectious diseases, particularly scarlatina, pneumonia and typhoid. In the last two the pneumococcus or typhoid bacillus may be present alone, or with the ordinary pus cocci. The same may be said of the gonococcus. According to the modern view, acute articular rheumatism is really an acute infectious arthritis.

Taken in connection with the blood-forming function of the marrow, it is a most significant fact that the infectious diseases characterized by leucocytosis are those that an arthritis follows. Typhoid fever has a leucopenia in the early stages, but a leucocytosis in the late stages, and it is in the late stages that arthritis occurs. Pneumonia may or may not have a leucocytosis according to circumstances. Mumps and malaria, as far as I know, are never complicated by arthritis.

In certain instances infectious arthritis is thought to be due to toxines rather than to bacteria themselves, but the possibility of the presence of the bacteria in the bone marrow alone must not be overlooked.

Morbid Anatomy. The disease may be confined to the synovia, or it may also involve the marrow. The synovia presents the signs of an acute inflammation—engorgement, redness, thickening, villous proliferation, etc. An exudate is quickly poured out into the joint cavity, serous, fibrinous, or purulent, as the case may be.

Pathology

Gonococcal and typhoid infections often remain confined to the synovia. Pneumococcus, streptococcus, and staphylococcus infections generally spread quickly to the marrow also, or they may first involve the marrow and then spread to the synovia.

The marrow of the bone ends may be involved either primarily or secondarily to the inflammation of the synovia. If primarily, then the inflammation quickly spreads to the synovia. Colonies of bacteria and abscesses may be seen in the meshes of the marrow. The bone shows in places productive osteitis, in places rarefying osteitis. In true suppurative inflammations the bone trabeculae are killed to a greater or less extent. The cartilage suffers in the same way. Its death leaves the bone end exposed. In the milder inflammations, especially the gonococcal, fibrous adhesions form in the joint, but in the severe ones the entire joint quickly becomes disorganized. The bursae communicating with the joint suffer also, and after the rupture of the joint capsule, the circumarticular tissues may become involved. The inflammation caused by the streptococcus, staphylococcus or pneumococcus is essentially a destructive one, that caused by the typhoid bacillus or gonococcus alone is distinctly less so.

Inflammation of the inner layer of the periosteum and of neighboring bursae and tendon sheaths is often added to a gonorrheal arthritis. Contractures and sub-luxations are frequent. One joint may be attacked alone or several. The knee is most frequently involved. After the acute stages have subsided the disease has a tendency to become chronic and to persist indefinitely.

Results of Acute Arthritis

The result of the morbid process varies according to the cause and to the timeliness and efficacy of the treatment, from a practical *restitutio ad integrum* to a fibrous or a bony ankylosis. Gonorrheal inflammations may heal completely, may become chronic,

or may result in fibrous ankylosis with distortions and atrophy of the bone. Even in the chronic cases, a cleaning up of the focus of infection may cure the joint disease. Typhoid arthritis may recover completely, but often results in dislocation, especially in the hip. If a suppurative inflammation be added, it may cause ankylosis. As long as an inflammation by pus cocci is restricted to the synovia, there is hope for a good functional result, but if the marrow be involved, a bony ankylosis is the best we can expect. In children the outlook for motion is more favorable than in adults.

SYMPTOMATOLOGY.

Symptoms

Pain and restriction of function are the two main symptoms, both varying in intensity as the cause of the infection and its virulence. Fluid is almost invariably present in the joint, except sometimes with gonorrheal arthritis. Motion is painful, and the synovia is sensitive to pressure. The joint is held in the attitude most comfortable to the patient—usually semi-flexion.

Prognosis

Prognosis. Simple gonorrheal or typhoid arthritis is in itself not dangerous to life. The prognosis in pneumococcic and suppurative arthritis is much graver. In the last two, death may quickly supervene unless prompt treatment be instituted, or even in spite of it. If the patient survive the acute stages he may die later of exhaustion. Relapses are frequent in gonorrheal arthritis unless the causal lesion has been cured.

Diagnosis. That we have to deal with an acute arthritis is easy to establish. Our main problem is to determine the nature of the inflammation.

In non-infectious traumatic arthritis, while there may be a slight rise in temperature (one degree), constitutional symptoms are conspicuous by their absence.

Acute inflammatory rheumatism has its acute pyrexia, its *fleeting nature,* its acid sweats. The view has always been maintained, by none more vigorously than by the writer, that this affection disappeared completely, leaving no trace, and was absolutely unconnected with chronic arthritis. If Poynton and Paine can succeed in establishing the truth of their contentions (and it must be admitted that they maintain their arguments with great force) we shall be compelled to change our opinions radically.

Diagnosis

Pneumococcic inflammation occurs in the late stage of pneumonia, or after the disease has run its course. It is wont to be very severe, and to be accompanied by grave constitutional disturbance.

Simple typhoid arthritis occurs in the later stage of typhoid, or in convalescence, is not very painful as a rule, and does not greatly aggravate the existing disease. It is most frequent in the hip.

Gonorrheal arthritis may occur in the florid stages of the urethral trouble, or long after the acute symptoms have subsided.

Other gonorrheal complications may also be present. It is most frequent in the third week and in adult males, and in their urinary tract may be discovered the evidences of specific inflammation. Pregnancy, labor, and the puerperium predispose in women. The real invasion of gonorrheal arthritis is often preceded for several days by pain in various joints. Possibly several joints may be lightly

involved. Then the inflammation seems to concentrate upon one or two joints, and expends its energies upon them. The temperature may run rather high, but not, as a rule, as high as in streptococcic or staphylococcic inflammations, nor is the constitutional involvement nearly as serious. The slower cases—those with a fibrinous exudate—occasion little difficulty in diagnosis, if the possibility of a gonorrheal lesion be borne in mind.

As a rule arthritis due to the pus cocci is severe from the start. The pain is very great, and the constitutional disturbance is severe. A penetrating wound, the pre-existence of scarlatina, measles or smallpox, the presence of a suppurative process elsewhere, all point to this type of infection. An acute inflammation in the joint of a young child is almost always a streptococcic or staphylococcic one, unless he has had pneumonia.[1]

Besides a hemophiliac joint, already described, if a joint swell up suddenly, almost painlessly, and without sufficient cause, a Charcot joint should be thought of, and search should be made for a spinal cord lesion.

Blood Count

The blood count will not be of great service. A leucocytosis may be present in any acute joint inflammation. Possibly it will be greatest in an ordinary streptococcic or staphylococcic arthritis. In acute inflammatory rheumatism it will be of the neutrophilic type.

Aspiration is a most valuable help, when the surgeon is in doubt. Pus cocci and pneumococci can be demonstrated, if they be present. Gonococci

(1) Rövsing maintains that many of the acute arthritides in young children are really cases of synovial tuberculosis.

can sometimes be found in gonorrheal joints, sometimes not. From typhoid joints the fluid may be sterile, it may contain typhoid bacilli, or typhoid bacilli and pus cocci.[1]

The Roentgen rays are not of great use in the diagnosis.

TREATMENT.

Acute articular (inflammatory) rheumatism has been considered as belonging in the domain of internal medicine. If the affected joints be immobilized during the acute stages of the disorder, the pain will probably be less. The wisdom of removing the possible source of the original infection (diseased tonsils!) after the attack has subsided, is apparent.

Acute Articular Rheumatism

Typhoid Arthritis. Rest, protection, and the avoidance by attitude of dislocation, so frequent in this affection, are our chief concern. The hip is the joint most frequently dislocated. The attitude to be avoided is one of flexion and adduction. If much fluid accumulate, the joint should be aspirated under strict aseptic precautions. If a pus infection be added, the symptoms become those of an ordinary suppurative joint, and the treatment will be that of the latter.

Typhoid Arthritis

Gonorrheal Arthritis. The causal lesion must receive prompt and thorough treatment. The joint itself should be put at rest, either by splints or by plaster of Paris.[2] If much fluid be present, it may

Gonococcic Arthritis

(1) A. G. Ellis, Journal of Infectious Diseases, 1909, Vol. 6, p. 181.

(2) When any diseased joint is put at rest by splints it should be put in the attitude most useful if an ankylosis take place; thus, the knee should be put up in full extension, the ankle and elbow in right angled flexion, the hip in extension and slight abduction, the wrist in slight super-extension.

be aspirated. Some authorities recommend washing out the joint.

The use of vaccines and of anti-gonococcus serum has been warmly debated. Many writers affirm their inefficacy, but in several of my cases the relief of the symptoms has been so marked after the administration of anti-gonococcic serum, that I should advocate its trial. A secondary infection by pus germs demands prompt and radical treatment, but fortunately it is not very frequent.

Pneumococcic Arthritis

Pneumococcic Arthritis. We must be governed here largely by the condition of the patient. If the joint contain very little fluid, or if the fluid be serous, and if the constitutional involvement be not great, we may temporize. Numerous cases of cure by simple aspiration and immobilization have been reported. If the joint be full of pus, and if the constitutional symptoms be severe, the sooner we resort to operation, the better. The joint must be laid open and thoroughly drained.

Suppurative Arthritis

Arthritis Caused by Ordinary Pus Cocci. Pus infection of a joint should always be avoided if possible. All operations on tuberculous joints and abscesses should be carried out with this idea in mind. Tuberculous joints should never be opened and drained. In all operations on the diseased shafts and metaphyses of the long bones, care should be taken to avoid penetrating the joint or approaching too near the joint cartilage. The normal synovial membrane has a very poor ability to resist infection, therefore all joint operations should be carried out with even more rigorous asepsis, if possible, than usual. Probably the safest course with a compound dislocation is to regard infection

as inevitable, and to attempt to forestall it by immediate resection. A stiff joint is better than an amputated limb, and either than death.

It has been fairly well established that even when aspiration reveals the presence of a few pus corpuscles in a joint, a conservative course is often permissible. If the course of the affection be not very severe, and if the constitutional involvement be not marked, we might be justified in immobilizing the joint, aspirating it, and possibly washing it out. The effect of injecting any substances into the joint, or of attempting to pull the articular cartilages apart by traction, is probably illusory, and the employment of these measures rests often upon an ignorance of pathology.

Joint Drainage

If the infection be a severe one, or if aspiration reveal streptococci or staphylococci, no time should be lost. We must immediately resort to radical measures. *The joint must be opened and thoroughly drained.* Drainage does not consist in making a small incision into the upper aspect of a joint, and letting the pus out. The joint must be drained in every part. The incisions must be free. Through and through drainage by counter-openings is best. I consider gauze far better than drainage tubes. I have had poor results from the latter.

In the hip an anterior and a posterior incision should suffice. In the knee long incisions at the sides of the patella, and another incision in the popliteal space may be used. If the infection be a very virulent one, a more thorough operation is necessary. The incision runs across the patella, the patella is sawn through, the fragments are reflected up and down (or the incision may be a horse-shoe

one below the patella, and the patella may be reflected upward), and the lateral and crucial ligaments are divided. When the joint is flexed, its recesses can be easily drained. It should be packed loosely with gauze—the quadriceps pouch should not be overlooked—and should be put up in marked flexion, but not flexion so extreme as to compromise the popliteal vessels. As the infection subsides the joint may be brought gradually into extension, a procedure comparatively simple if the tibial tuberosities have not been permitted to slip up back of the femoral condyles.

In disease of the ankle, excision of the talus will afford free drainage, and will leave a useful foot.

It is said that the punctured wound of the metacarpo-phalangeal joint received from an adversary's tooth in fistic encounters, almost invariably results in dangerous infection, and good authority is behind the advice to amputate the finger at the first sign of the infection, and thus forestall the inevitable here also.

THE SPINE.

Spinal Arthritis

The joints of the spine present certain peculiarities. They are rarely involved in an acute suppurative[1] or in an acute gonorrheal or pneumococcic inflammation. When they are so involved, the principles of treatment are much the same as in other joints. A typhoid infection gives rise to a condition that merits a special description.

TYPHOID SPINE.

The nature of this lesion was for a long time disputed, but it is now generally regarded with rea-

(1) Infectious Osteomyelitis of the Spine. Strong, The Lancet, 1912, II, 1576.

sonable certainty as due to infection of the vertebral joints by the typhoid bacillus. No case of a mixed infection is on record. A synovitis of the intervertebral joints would account for the symptoms. The kyphosis observed in some of the reported cases points to the occasional bony involvement.

Typhoid Spine

Symptomatology and Course. The affection, most common in vigorous young men, usually begins in the late stages of a typhoid fever or during convalescence, with pain and stiffness in the lumbar region. Trauma often plays a part, either a causative or an aggravating one. The patient complains of a persistent lumbago. Spasm of the muscles of the back, limitation of motion, and possibly some lateral curvature may be present. In some cases the lumbago gradually subsides, in others it grows more and more severe until the patient is absolutely unable to sit or to stand. The pain comes on in violent paroxysms, often shooting around the abdomen and down the legs. Women who have suffered say that it is worse than labor pains. Hysterical symptoms have often been noted, and rhythmic contractions of the abdominal muscles. The attacks of pain may be ushered in by a rise of temperature of three or four degrees Fahrenheit. The knee-jerk may be increased or diminished. A distinct kyphosis rarely forms. The functions of stomach and bowels may be disturbed. "Starting pains" have been observed at night.

Diagnosis. A severe lumbago, occurring late in the course of a typhoid, or during convalescence, means typhoid spine, but the possibility of a tuberculous lesion following typhoid must be remembered.

The disease never is fatal. The patient always recovers, usually completely, but the recovery is slow and tedious. A year is the average duration of the severe cases. Relapses sometimes occur. A kyphosis once formed is permanent. Suppuration is unknown.

The treatment is rest. The milder cases do well with strapping of the back to take the strain off the spinal joints. Severer cases demand a light spinal brace or a plaster jacket. For the most severe cases recumbency is necessary, preferably on a gas-pipe frame. The spine must be put completely at rest in order to prevent the attacks of pain. For the paroxysms morphine may be used, or a whiff of ether or of chloroform.

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CHAPTER III.

CHRONIC ARTHRITIS, TYPE I.

TUBERCULOSIS, SYPHILIS AND GONORRHEA.

The subject of chronic arthritis presents many unsolved problems, and to the superficial observer appears a most discouraging one, but, to him who will trace carefully the progress of investigation for the past fifty years, the prospect is bright with promise. One by one various diseases which may cause the joint lesions have been identified and carefully described, and, while much remains to learn, probably no obstacles exist that will not yield to patient work. It is not a field for dazzling exploits. He who wishes to achieve brilliant results without hard work should turn his attention to some other branch of medicine. Possibly it is this absence of the theatrical which causes the discoveries that have been made to attract so little notice.

DEFINITION. Chronic arthritis is the expression in the joint tissues of a long continued irritation. The irritation may be mechanical or chemical. In the last analysis both are very similar.

CLASSIFICATION.

Most if not all cases of chronic joint diseases are divisible into two main classes, the first characterized by a proliferation of the synovia and of the lymphoid marrow with a *resulting* atrophy of the cartilage and bone, and the second by an exactly

Classification

opposite condition, namely an inflammation and degeneration of the synovia and of the marrow, with a *resulting* hypertrophy of the cartilage and bone. If we regard these changes in the bone and cartilage as primary we are quite unable to comprehend or to classify them, but if we study the tissue from which the cartilage and bone draw their nutrition we seem to make a long stride toward a solution.

TYPE I.

Cases Characterized by Proliferation of the Synovia and Marrow, Followed by Atrophy of Bone and Cartilage.

Cases of Arthritis in Type I

Under this heading fall tuberculosis, chronic gonorrheal and syphilitic arthritis, and that large group of cases described by English writers generally as Rheumatoid Arthritis; by Goldthwait and others as Atrophic Arthritis, by Nichols and Richardson as the Proliferative Form, by Nathan and others as Metabolic Osteo-Arthritis, and by other writers by various other appellations. It includes also some of the cases recently described under the heading "Infectious Arthritis." As the view is gaining ground that all these joint diseases are probably of infectious origin, it seems unwise to separate a special class under that name, implying that others are of different origin.[1]

JOINT TUBERCULOSIS.

Etiology. The exciting cause is the tubercle bacillus. Heredity, environment, trauma and the acute

[1] "Certain writers would make another type which they would term 'infectious.' In the present condition of our knowledge it seems better to restrict this term to conditions associated with a definite organism."—McCrae.

infectious diseases have all been considered as contributing causes. The influence of trauma has probably been overrated, and, as we shall see in our study of the pathology, those cases which start in the bone marrow cannot be influenced in the be-

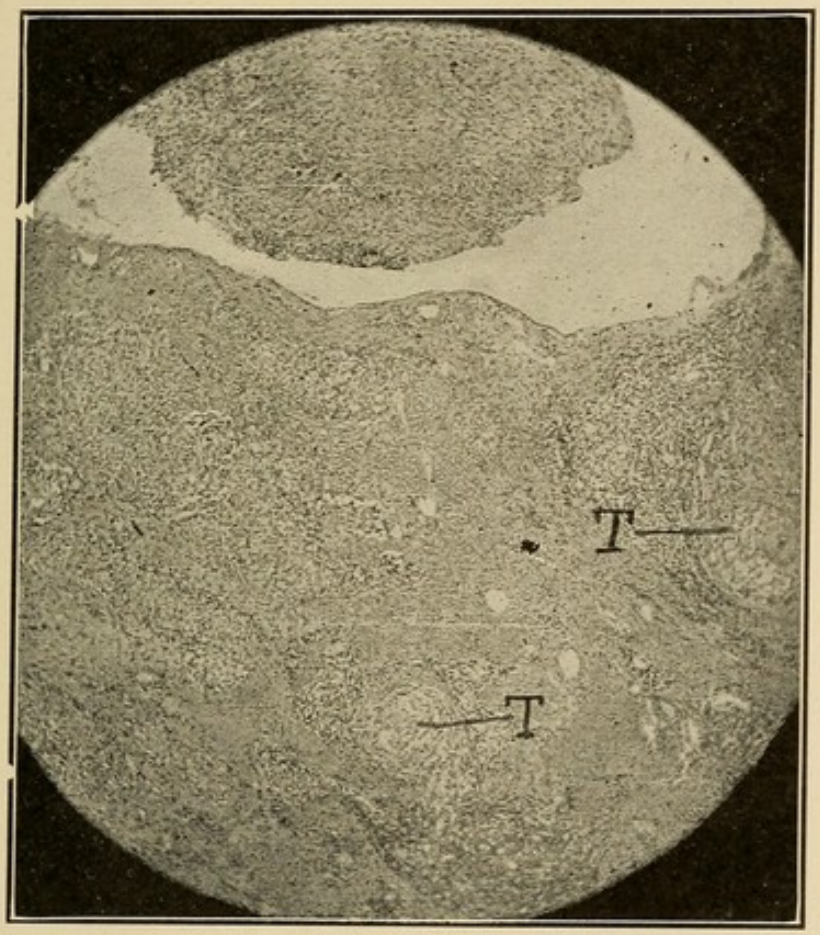

Fig. 16.

Photomicrograph of synovial tuberculosis of the adult knee—slow, chronic form, with well-marked production of fibrous tissue. The disease had existed with desultory treatment, for many years, and was completely cured by the author's resection three years ago. The patient now is in health. T—tubercles, well encapsulated by new fibrous tissue. The structure above is an hypertrophied synovial villus. Zeiss apochromatic 16 mm.

Trauma

ginning by trauma, for the marrow is shielded from any force other than fracture. Trauma may, however, bear a more direct causal relation to the cases starting in the synovia.

In a large proportion of instances one of the acute infectious diseases precedes the onset of joint tuberculosis, especially among children. Measles ranks first, then whooping cough, then scarlet fever and pneumonia.

Fig. 17.

Photomicrograph of synovial tuberculosis of the adult knee—rapid, destructive, diffuse form with almost no evidences of fibrous encapsulation. The patient died shortly afterwards of pulmonary tuberculosis. At A may be seen a typical giant cell, and just above it a large area of beginning necrosis. The entire synovial membrane was enormously thickened. Zeiss apochromatic 16 mm. objective.

PATHOLOGY.

Upon a proper understanding of the pathology of joint tuberculosis depend our ideas of the entire subject, and he who would treat the disease intelligently must master this aspect of it Otherwise his

treatment will resolve itself into a jumble of expedients that constantly must be changed to accommodate themselves to every new and extraordinary therapeutic measure. Let us start at the beginning and trace the entire process.

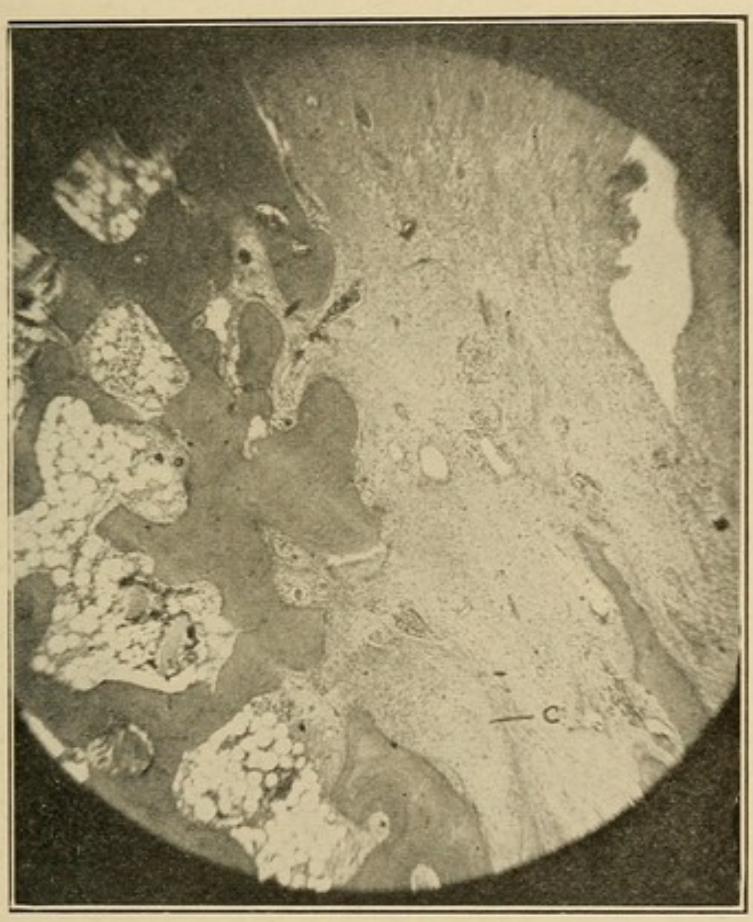

Fig. 18.

Photomicrograph of section of wall of old encapsulated tubercle in bone. At the left are fairly normal bone and marrow, then comes an almost complete wall of bone, then a layer of fibrous tissue. At one spot C can be seen a piece of cartilage, probably representing a stage of bone formation. Leitz objective No. 2.

Value of Animal Experimentation

It is obvious, in the first place, that too much reliance must not be placed upon experimental joint tuberculosis. Numerous investigators have injected the joint cavities of animals with a pure culture of tubercle bacilli, and by observing the resulting joint changes have tried to build up the pathology of the

disease, but in so doing they have reversed the actual pathological changes, for in the disease as it occurs in nature the joint cavity is affected from the joint tissues, not the joint tissues from the joint cavity. Other investigators have injected pure cul-

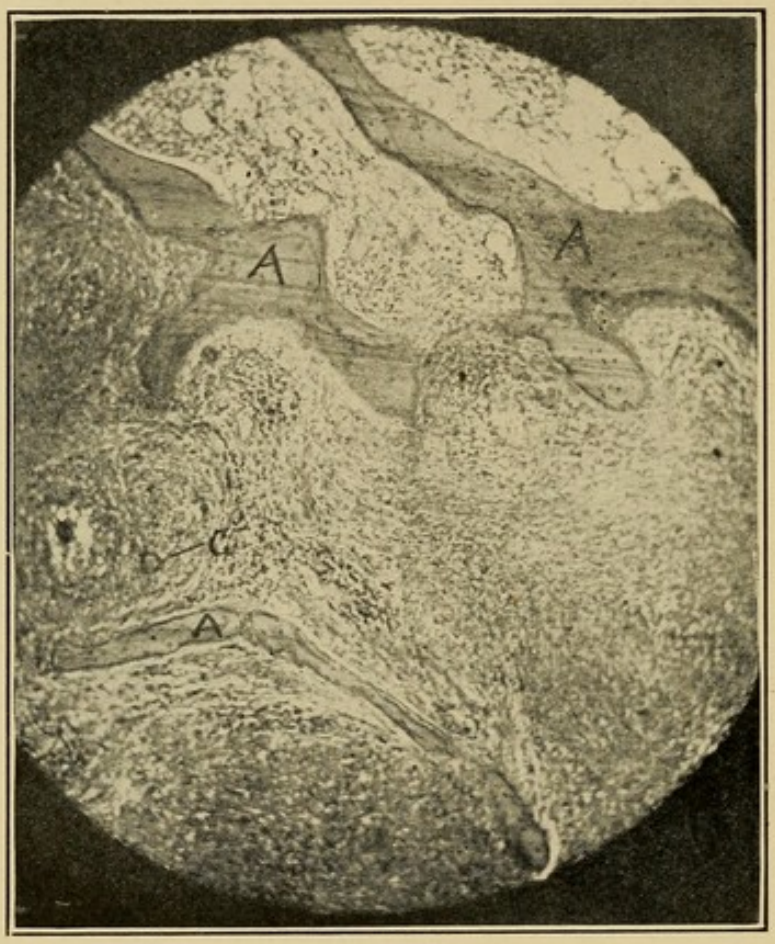

Fig. 19.

Photomicrograph of an intense tuberculosis of the marrow, near the cavity of a bone abscess. Above may be seen a milder inflammation of the marrow. A.A.A. bone trabeculae, the lower ones necrotic; G. Giant Cell. Zeiss apochromatic objective 16 mm., ocular 4.

tures into the nutrient arteries of the long bones, and, while their findings are of somewhat more value, still they are apt to lead us astray, for it is doubtful if a plug or embolus such as this might occasion, is often responsible for the beginning of

the disease. Here, as elsewhere in the body, the disease probably starts by the deposition of one bacillus or of several bacilli either in the embrace of a white corpuscle, or floating free in the blood stream.

Theories of Joint Localization of Tuberculosis

Immediately the question arises, "Why are these tubercle bacilli deposited in the neighborhood of the joints and rarely if ever in the shafts of the long bones?" This question for years has proved a stumbling block. Upon its answer much depends. Some say that the increased circulation necessary for the rapid growth in the region of the epiphyses is the explanation, but rapid growth and increased circulation are not causes of tuberculosis, and we do not find the disease ordinarily in other rapidly growing tissues. Again, the disease occurs frequently in adults, whose growth has ceased.

Traumatic Theory

Liability to injury has been advanced as the answer, but injury is also no cause of tuberculosis. A frequently injured muscle or ligament is never the starting point of the disease, and the joints most often injured, such as the hand and foot, are not most often involved. No injury but a fracture can effect the bone marrow in any way.

Lexer's Theory

The lack of anastomosis in the terminal blood vessels in the neighborhood of the epiphyseal line in children has been held responsible, and is still regarded so by many observers, yet joint tuberculosis occurs in adults, as we have said, in the ends of whose long bones the customary anastomosis exists. Again, this lack of anastomosis does not exist in the bones of the spine, and in the short bones of the carpus and tarsus, even in children, but tuberculosis is fairly frequent in these localities.

In adults the synovia is frequently involved primarily, and here again we can find no lack of anastomosis. On the other hand, when we look further and consider the various other organs of the body,

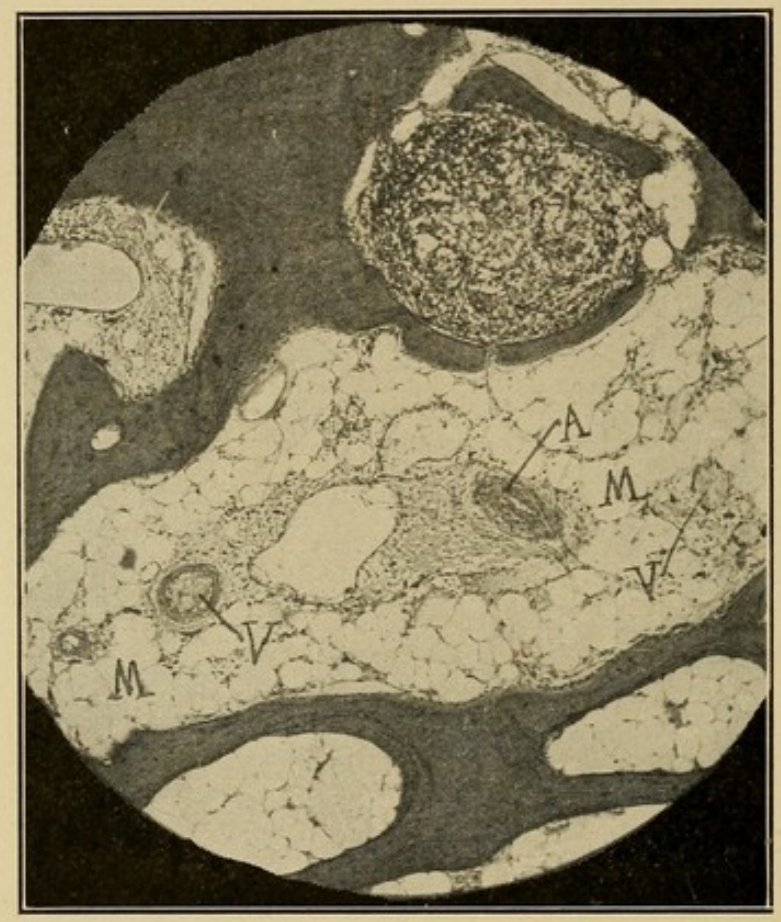

Fig. 20.

Same case as the preceding. Photomicrograph of a section of the bone at some distance from the abscess. This shows well the futility of any operation designed to remove the focus of disease. There is no known way of distinguishing the presence of this tubercle in life. Above can be seen a tubercle almost surrounded by bone trabeculae, then comes a strip of non-tuberculous marrow (M.M.) containing a thickened artery (A.) and engorged veins (V.V.). The dark bone trabeculae below show a well-marked productive osteitis. Zeiss apochromatic, 16 mm. Ocular 4.

the brain, for instance, we do not find that the presence of end arteries predisposes them to tuberculous involvement. If we accepted this view of the cause we should immediately be in difficulty to

explain why the tubercle bacillus was arrested by the end arteries, and involved the joint end of the bone, while the staphylococcus lodging at or near the same spot, involved the shafts, and produced the ordinary acute suppurative osteomyelitis. The

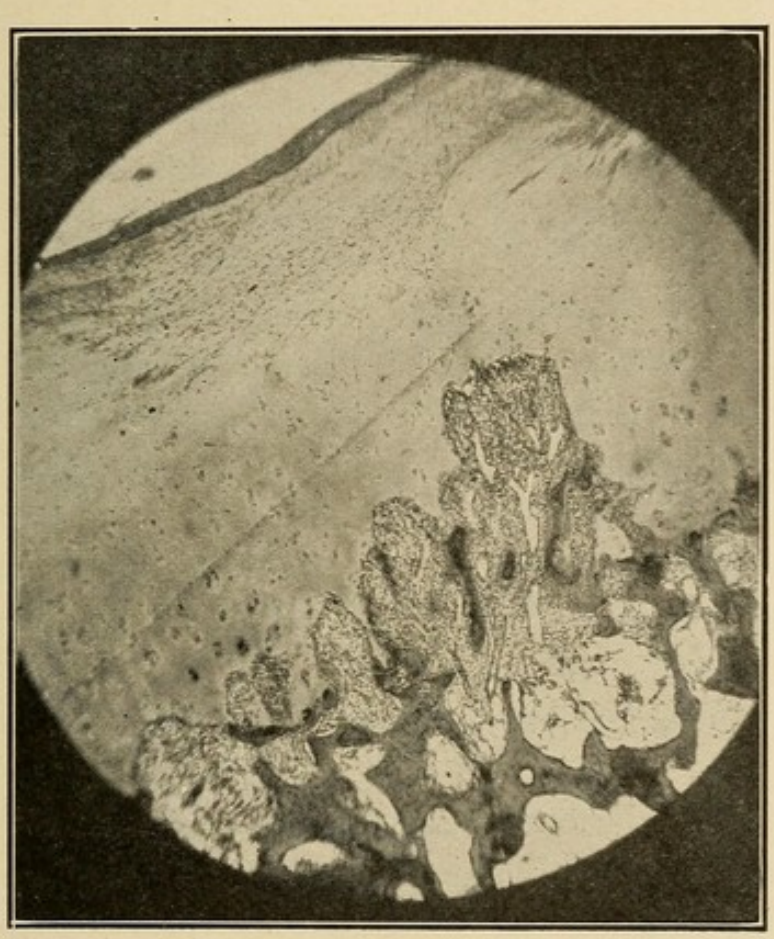

Fig. 21.

Photomicrograph of knee joint tuberculosis in an adult, Leitz objective No. 2. Joint surface above, bone marrow below. Osteoid tissue at the bottom of the articular cartilage. Note how the granulations are "eating into" the cartilage from below, and how the superficial portion of the cartilage is undergoing fibrillation.

same may be said of the claim that the "spongy nature of the bone," or the sluggish circulation in its capillaries permitted the deposition of the bacilli. The simple, easy explanation lying directly before our faces, has passed unnoticed. Tuberculosis

Sluggish Circulation Theory

manifests throughout the body a marked affinity for epithelial, endothelial and lymphoid tissues, and, *if unmixed with a secondary infection,* it rarely attacks directly any other tissues[1] whatever, but simply affects them by interfering with their nutrition.

The Author's Theory

In the region of the joints we find two lymphoid structures, the red marrow and the synovia. We find red or cellular or lymphoid marrow also in the short and flat bones, the vertebrae, sternum, cranial diploë, carpus, tarsus, etc., and we note that wherever this lymphoid marrow is found, the bone in that region is vulnerable to tuberculosis, and where it is not found, there the bone is invulnerable, or practically so, to a pure tuberculous invasion. The unmixed disease never occurs in the shafts of the long bones, where ordinary yellow or fatty marrow is found. Occasionally it is found in the shafts of the long bones of young children, but at this period of life lymphoid marrow is still present in the shafts. As age advances, less and less red marrow is present in the ends of the long bones, and bone tuberculosis becomes less frequent.

Changes in the Bone After Resection

Numerous authorities have found that after a complete bony ankylosis in a joint, the bone in that locality takes on the characteristics of the bone in the shafts,[2] and the lymphoid marrow in the adult changes to the fatty variety. This explains the cure of the disease after successful resections, for the most enthusiastic advocates of radical removal of diseased tissue could not maintain that they had eradicated it all, at least they could not maintain

[1] This rule may possibly have a very few exceptions, but if exceptions exist, they are so rare as to be practically negligible.

[2] Vide the chapter on ankylosis.

this if they studied their tuberculous joints under the microscope.

Joint tuberculosis is very rare in children under one year of age, because their bone ends are mostly cartilaginous. When the disease occurs at this period of life it is probably synovial.

The presence of lymphoid tissue is, then, the prime cause for the location of the disease in the neighborhood of the joints, and the process may start in the synovia or in the lymphoid marrow. In children the original focus is said to be almost invariably in the marrow, in adults, though good authorities differ on this point, the synovial form is probably of about equal frequency with the myeloid. At no time in the course of the disease, provided secondary infection do not take place, is any other joint tissue directly involved, except the inner cellular layer of the periosteum.[1] The ligament, the fibrous layer of the periosteum, the cartilage and the bone trabecula are all absolutely immune to an unmixed tuberculous infection, and merely suffer in their nutrition. The disease must find its pabulum in the two "lymphoid" tissues, and if we can cause the disappearance of these two tissues the disease cannot exist.

Original Focus

Too much stress cannot be laid upon this point. All our therapeutic ideas must be based upon it, and it will be found that all therapeutic measures which have survived the test of time are in consonance with this idea: *No function, no red marrow and synovia; no red marrow and synovia, no joint tuberculosis.*

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[2] Adami and others maintain that the disease may start in the periosteum, and this may be so in rare instances.

The tubercle bacilli, deposited in the synovia or in the lymphoid marrow, give rise to the initial tubercle, with its lymphoid, epithelioid and giant cells, and this tubercle, following the usual course, tends to spread at the periphery and to undergo necrosis at its centre, and to involve more vulnerable tissue in the neighborhood.

From the start of the process the forces of repair are also at work, and according as one or the other predominates, the disease tends to extension

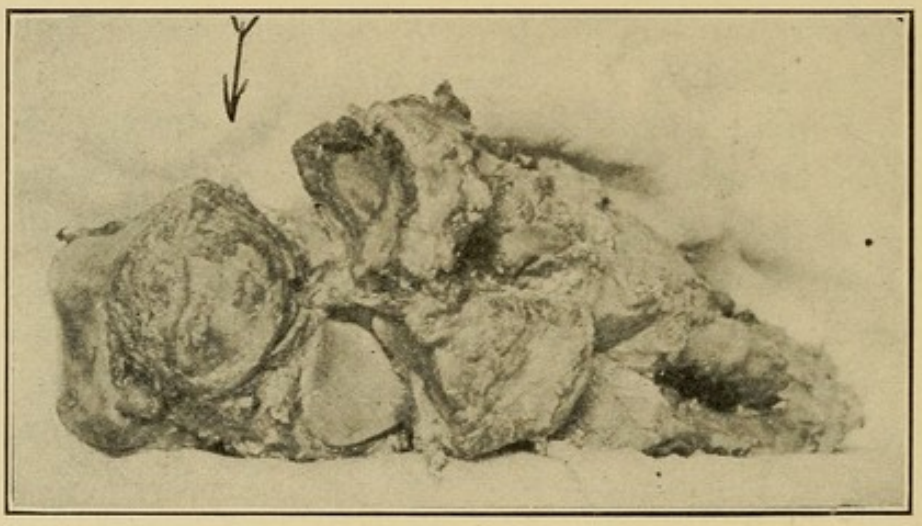

Fig. 22.

Tuberculosis of the talo-navicular joint in an adult. Note the erosion of the joint cartilage.

or to recovery. Sometimes the disease is progressing to destruction in one part of a joint and to repair in another. Sometimes the reparative processes are seen to be strong, sometimes feeble. Here as elsewhere in the body nature strives to cure by encapsulation, and walling off, and often a microscopical examination of the stained slide will give a good idea of the clinical history of the case. If the course be slow and chronic, much

Repair

fibrous tissue will be found; if rapid and acute, very little.

Tuberculous disease in the marrow may exist for a long time without involving the synovia, and *vice versa*. It cannot jump across the joint from one bone to another except by way of the synovia. An intact articular cartilage forms an absolute barrier to its progress.

The Joint Cartilage a Bar to Tuberculosis

Let us trace more minutely the process of the infection, observing its effect upon the various tissues, and taking up first the cases beginning in a so-called bony focus, or, better expressed, in a marrow focus.

The tuberculous inflammation in the lymphoid marrow—say in the upper end of the tibia—causes an irritation in its neighborhood, that may result in a temporary productive osteitis of the trabeculae, but, as the disease spreads, this soon changes to a rarefying osteitis, and to a destruction of the bone tissue. If the spread be rapid, a large piece of the bone will be killed at once, and a sequestrum may result, but the usual course is a slow spread and a destruction of the bone in small pieces, giving us the so-called "bone sand" so characteristic of the disease.

Course of the Morbid Process

The tuberculous granulations may extend downward to the limits of the lymphoid marrow. Here they stop. Outward the granulations may make their way to the periosteum. Here also they stop, unless an abscess be formed and the contents force their way through the periosteum into the tissues outside. The cartilage draws its nourishment from the subjacent bone marrow. When the tuberculous granulations reach the under surface of the car-

tilage they interfere with its nutrition, and it begins to degenerate. The first sign of this degeneration is often a fibrillation. Sometimes the tuberculous granulations can be seen under the microscope pushing their way through the cartilage. They may

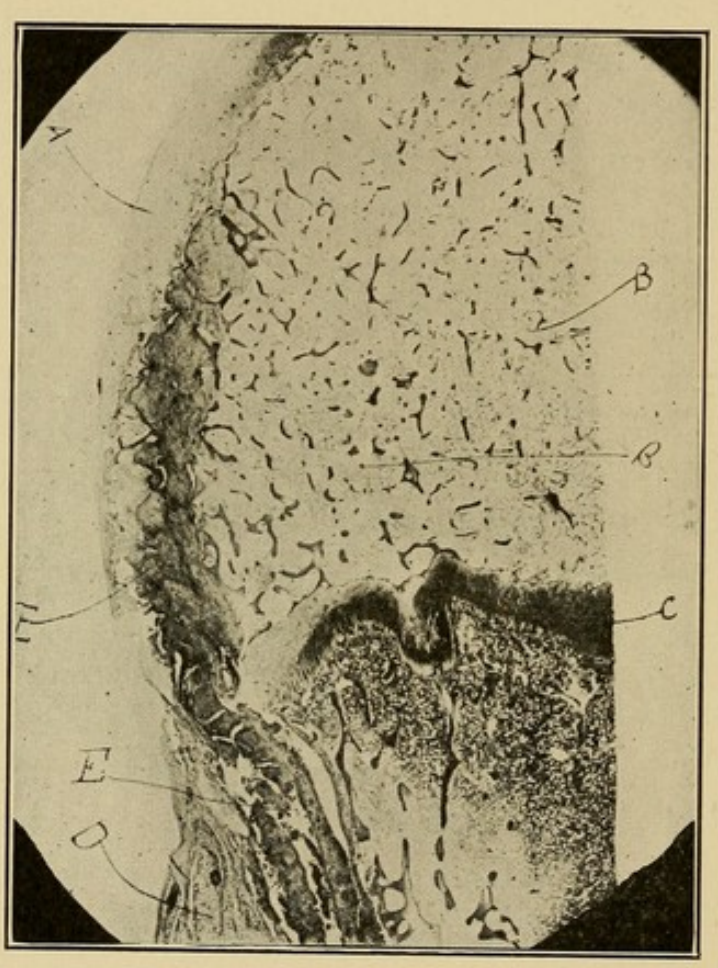

Fig. 23.

Low power photo-micrograph of a case of knee joint tuberculosis in a child about ten years old. The focus was located on the shaft side of the epiphyseal line. The tuberculous granulations made their way to the periosteum, and burrowed along under this, and then under the articular cartilage. A—articular cartilage, B—epiphysis, C—epiphyseal cartilage, D—periosteum, E—tuberculous granulations.

Damage to the Cartilage

break through at some one spot, or they may spread out and break into the joint at the margin of the cartilage. Sometimes they spread out under the entire cartilage and lift it off. When they gain the

joint, they infect the synovia on its surface, or, if the disease has reached the margin of the cartilage without breaking into the joint, then the synovia is probably involved by direct extension. The disease then attacks the other bone or bones of the

Fig. 24.

Low power photo-micrograph of a portion of a collection of rice bodies in their capsule, from a tuberculous knee joint in an adult.

articulation, entering them at or near the periphery of the joint cartilage.

Before the tuberculous granulations reach the joint a serious effusion is often formed in its cavity.

Reaction of the Synovia

The synovia shows the typical tubercles when it has been involved, on its surface or in its substance or both. It is thickened, succulent, and usually

presents greatly enlarged villi: again, it may be tough and fibrous. It may be involved throughout its extent or only in certain areas. It sometimes hypertrophies, and extends outward over the joint surfaces, hiding them more or less completely—synovial pannus. This pannus may be attached to the degenerated cartilages or it may not be. It does not "eat its way" into the cartilages, as some writers have affirmed, but the disease spreads from the synovia under the cartilages at their margin and causes them to degenerate by shutting off their nutrition.

Synovial Pannus

At any stage in the proceedings detailed above, serum and white blood corpuscles may be added to the necrotic bone tissue, cartilage and diseased marrow, and a tuberculous abscess may be formed in the bone, which later breaks into the joint: or the abscess may be formed in the joint itself. In rare instances the abscess breaks through the periosteum and never involves the joint at all.

If formed in the joint, or if it has reached the joint after formation in the head of the bone, the abscess may rupture through the ligaments and so, reaching the extra-articular structures, may make its way to the surface, or it may stay indefinitely in the extra-articular structures, or, never emerging from the joint, it may finally disappear entirely.

Abscess

Careful examination will often reveal in these abscesses a few tubercle bacilli, but not invariably.

The walls of the tuberculous abscess are formed by the necrotic tissues in which they lie. These walls *are probably themselves never tuberculous, unless the abscess be secondarily infected.* If

secondarily infected, then tubercles will be found in the walls.

If the abscess be opened or if it rupture spontaneously, then it will almost invariably become secondarily infected, and will remain open for a

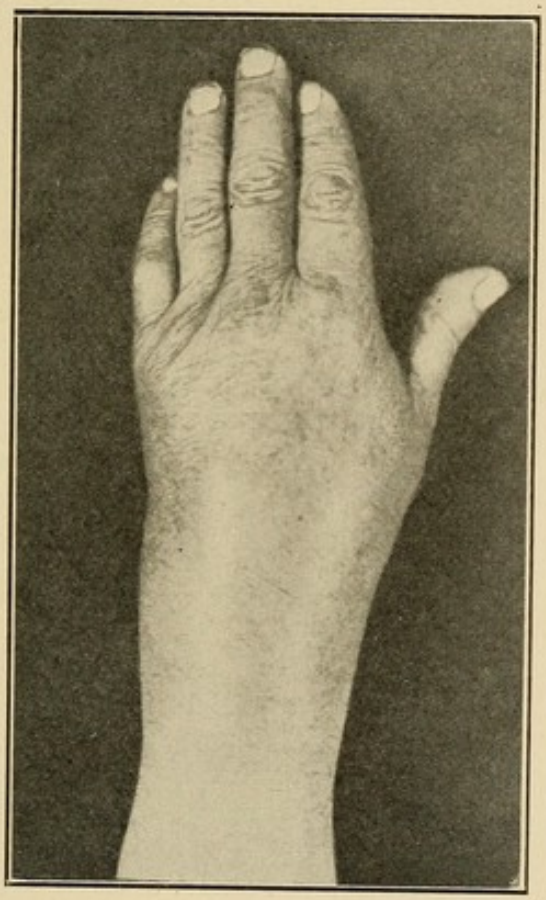

Fig. 25.

Tuberculosis of an adult's wrist. Note the swelling. This picture would do equally well to illustrate any joint disease in Type I. There is nothing characteristic about it. No one could possibly make a diagnosis from its inspection.

long time, communicating with the joint and with the surface by tracts called *sinuses,* whose walls, as well as the tissues of the joints to which they run, are lined with tubercles, and discharge large quantities of foul pus.

The Sinus

SYNOVAL DISEASE.

When the disease starts in the synovia the original tubercle is deposited in its lymphoid tissue, and the disease extends down to the fibrous capsule and over part of the membrane, or throughout it all,

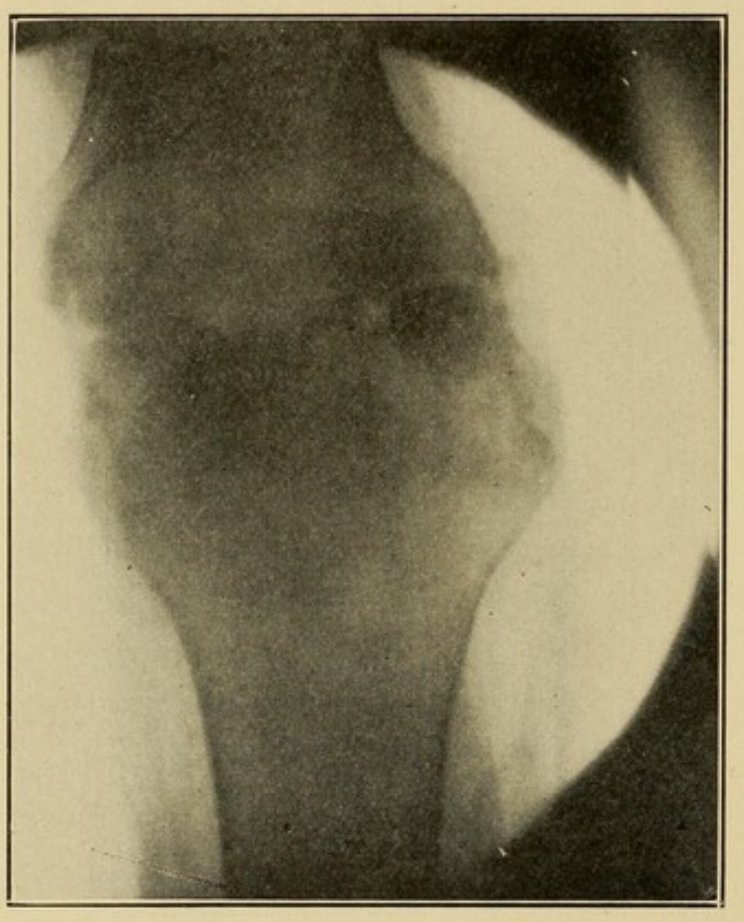

Fig. 26.

Knee joint tuberculosis in the adult, of about ten years' duration —the slow chronic form. Note the disappearance of the joint cartilage, the worm-eaten appearance of the bone, and the areas of bone condensation. An arthroplastic operation was done on this joint, but of course the wound broke down, suppuration ensued, and a subsequent resection was necessary.

possibly involving the bone by extension into its substance at the margin of the cartilage, where the synovia is attached. From this point there is no essential difference in the behavior of the two forms, bony and synovial.

On the other hand the disease may remain for years confined to the synovia and may never extend to the bone, unless this extension be brought about by unwise operative measures.

Necrosis in the Synovia

The surface of the synovia may be covered by necrotic tissue in places, and necrosis may also be detected in its substance. Layers of fibrin have been described by many writers as occuring on the surface. I have not been able to identify them. In one case, a careful staining showed what we had taken to be fibrin was pseudo-fibrin.

Picture of Joint Tuberculosis

Let us then hold in our minds a picture of a disease making its way through the lymphoid marrow and synovia, and changing their structure. The diseased areas always have a tendency to break down. The bony, cartilaginous and fibrous tissues are degenerating, breaking down, and crumbling away because their nutrition is cut off. Nature on her part is striving to stop the process by walling it in with fibrous tissues, and in the bone by building up thickened trabeculae in addition. All her efforts tend to destroy joint function.

Result

Tuberculosis joint disease, if no serious damage has been done to the articular cartilage, may turn back to practically complete recovery. The usual result is a more or less damaged joint with a restriction or abolition of motion. The ankylosis is always fibrous. *Bony union never takes* place except after resection, or in children after a mixed infection.[1]

RICE BODIES.

Rice Bodies

Rice bodies are small, hard, white, smooth, slippery bodies, sometimes found in tuberculous joints,

[1] See section on ankylosis.

especially in the synovial forms of the disease characterized by a slow extension and more or less effective efforts at repair. The nature of their origin has been a disputed point, but, inasmuch as in one joint in our possession a collection of them was found enclosed in a well-defined fibrous capsule

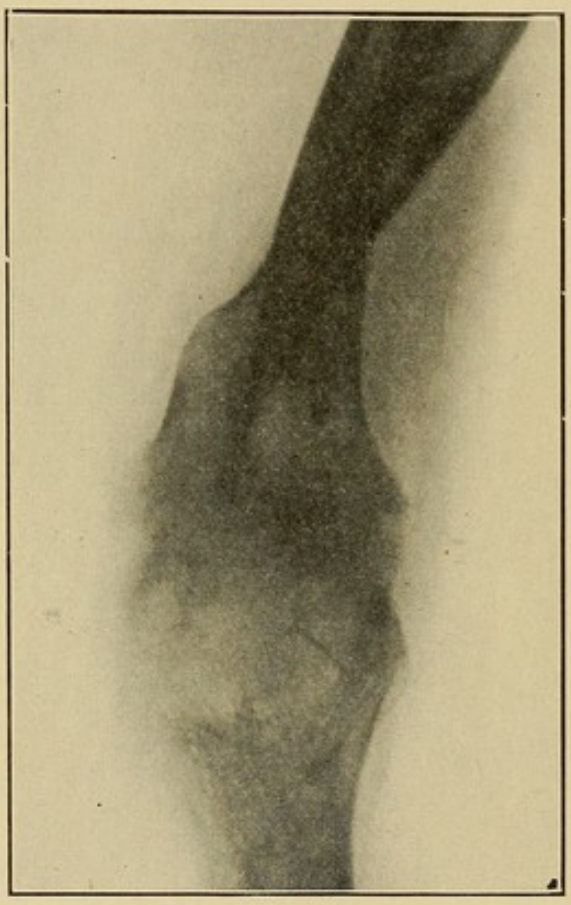

Fig. 27.

Tuberculosis of several years' duration in the elbow of an adult treated by conservative measures. Note the disappearance of the cartilage, and the atrophy of the bone. This picture illustrates well the futility of conservative treatment in the adult.

in the diseased synovia, we may infer that they are the result of a process of repair, possibly of the walling off and condensation of tuberculosis granulations. They must be set free in the joint—they are usually found free—by the bursting of their capsule.

The tissues in the neighborhood of a tuberculous joint take part in the disease to a limited extent. The tendon sheaths and bursae—synovial structures—are prone to direct involvement. The other tissues suffer from interference with their nutrition. Atrophy is a marked feature of tuberculous joint disease, and in this atrophy all the tissues of the limb take part—muscles, bones, etc. The atrophy above and below the joint accentuates the appearance of the joint swelling, and causes the bone ends to look enlarged, but this enlargement is only apparent. No exostoses ever form in tuberculous joints.

Involvement of Circumarticular Structures

MACROSCOPIC APPEARANCE.

There are some diseased joints that may be diagnosed as tuberculous with the naked eye, but there are many others that cannot. The synovia may be studded with tubercles, but again it may simply be hypertrophied and inflamed. Areas of necrosis may or may not be found in it. The joint may be the seat of a serous, or of a turbid, floccoulent exudate, or of a tuberculous abscess, or it may be a mass of fibrous adhesions.

Various Aspects of a Tuberculous Joint

The cartilages may be perfectly smooth, they may present one or more dimples, they may be eroded at their margins or on their surface, or they may be dead and may be found loose in the joint.

The bone tissue may or may not be necrotic. It may be hard and dense in spots, and soft and carious in others. Abscess cavities may be found in its substance. Sometimes sections of it will float in water. Often it undergoes a fatty change—the so-called fatty osteomalacia.

Fatty Osteomalacia

Attempts have been made to classify these tuberculous joints according to their gross appearances. Thus "Joint Fungus" was the name given to those cases in which an extreme proliferation existed in the synovia, while "Caries Sicca" was the type of

Joint Fungus
Caries Sicca

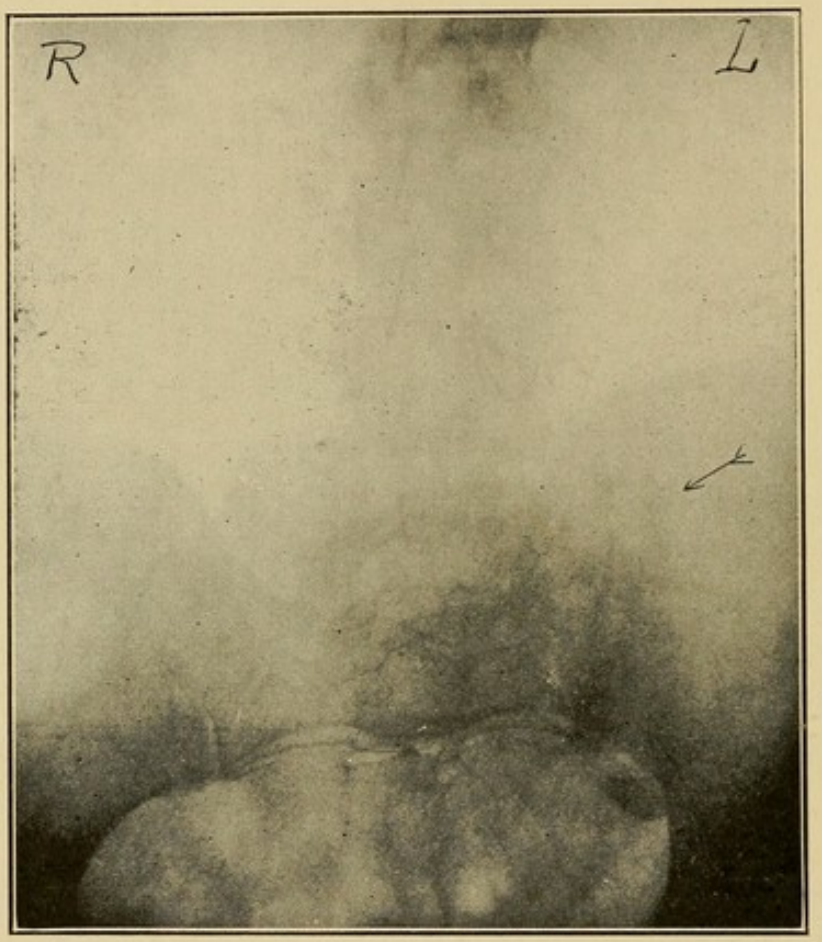

Fig. 28.

Tuberculosis of the left side of the sacrum in an adult. Note the irregular bone atrophy. The right sacro-iliac sychrondrosis is "sprung," and the chief complaint of the patient was a right sciatica.

disease characterized by a dry condition of the joint, and a preponderance of osteal involvement.

Such divisions are purely artificial and apparently quite unnecessary, as well as very confusing.

PATHOGENESIS OF BONE TUBERCULOSIS.

We have seen that the disease occurs in those portions of the bone only where lymphoid marrow is found, and that it disappears when the lymphoid marrow disappears. Now, according to the accepted relation of the lymphoid cell to the tubercle bacillus, this is exactly contrary to what we should expect, for why should the tubercle bacilli thrive in a tissue rich in cells (lymphocytes) whose function is to destroy them, and then disappear when these cells disappear? In the end we shall probably be compelled to revise our ideas on what Ziegler calls the "poetic" theory of phagocytosis.[1] We know that the red blood cell is the food of the malarial plasmodium, recent investigations point to the fact that the lymphocyte is the chosen domicile of the treponema pallidum[2], and it is more than likely that we shall conclude that the lymphocyte, instead of being the active foe of the tubercle bacillus, is, in fact, its food. Admitting this, then all we need to do to cure joint tuberculosis is to cause the disappearance of the lymphoid cell from the bone marrow and from the synovia.

SYMPTOMATOLOGY AND COURSE.

The onset of the disease is almost invariably slow. Often the exact date of the beginning of the symptoms is impossible to set. They are usually so mild at first that the patient disregards them or ascribes them to a "touch of rheumatism." Onset

The symptoms and physical signs common to other diseases of Type I. are found here—pain,

(1) See also Petrie, Journal of Pathology and Bacteriology, 1903, IX. 130.

(2) Vide J. E. R. McDonagh, *The Lancet*, 1212 II. 1011.

stiffness, swelling, limitation of motion, change in attitude, deformity, disturbance of function, and often a local increase in temperature.

In addition, in the cases with bone involvement two physical signs are more or less characteristic, namely, muscular spasm and early muscular atrophy. In point of fact, almost all the symptoms are worse in the bony cases.

Pain

The pain varies greatly in its severity. It may be very intense or comparatively insignificant, local or referred, constant or intermittent. It is almost invariably aggravated by motion. It is often worse at night, and wakes the patient up when the muscles holding the joint quiet relax—"starting pain." The

Night Cry

patient may wake up with a cry—"night cry"—and immediately fall asleep again.

The referred pain must always be remembered, and must not lead us astray in our diagnosis. In spinal disease the pain may be felt in the back, the chest, the arms, the abdomen, the hip or in the lower

Pain in the Knee in Hip Disease

extremities. In disease of the hip, pain in the knee is a frequent symptom.

Local sensitiveness to pressure can be elicited if the joint is superficial. In the early stages the pain is wont to be most severe when the patient begins to move about in the morning, and wears away during the day. The same may be said of the stiffness. It is due to the muscular spasm and to the inflammatory products.

Swelling

Swelling of the joint is a constant phenomenon, but in the deeper joints, such as the hip, it cannot be detected unless it is very great. The swelling may be boggy in the fungous type of synovia, or it may fluctuate. With the formation of an abscess

the swelling may reach an extreme degree. In the knee, elbow, and ankle especially it often takes on a peculiar fusiform shape, accentuated by the atrophy of the muscles above and below the joint: The overlying skin becomes blanched, and the superficial veins distended. This peculiar condition has received the name "White Swelling" or "Tumor Albus."

White Swelling

Limitation of motion is caused by the muscular spasm, and by the inflammatory products in the joint. That caused by the former disappears under an anesthetic, that by the latter does not.

Muscular spasm is essentially a conservative symptom, and represents nature's means of putting the joint at rest. If the needed rest be furnished by apparatus, the spasm abates. It is on guard during waking hours, but, as the patient falls asleep, relaxes and permits motion—hence the pain. It is partly responsible for the change in attitude of the joint. If this faulty attitude persist, the muscles become set in their contracted state and cause "contractures"—fixed faulty attitudes. The hip is flexed, adducted and rotated in, the knee flexed and the tibia subluxated backward and rotated out, the elbow semiflexed, the ankle plantar-flexed, etc.

Muscular Spasm

The change in attitude (deformity) and the functional disability are the result of the muscular spasm and of the inflammatory process in and about the joint. The attitude is due to the influence of certain muscles, as Jansen has pointed out.[1] At first it is determined by the contraction of the "uniarticular" muscles, i. e., those passing over the affected joint alone. The later contractures are due to the

Change in Attitude

(1) Jansen, Archiv für klinische Chirurgie, November, 1911.

contraction of the "multiarticular" muscles, i. e., those passing over more than one joint.

In a general way, semiflexion is the characteristic attitude of joint tuberculosis.

In untreated cases the deformity may be extreme, and may completely cripple the patient.

The disability is due to the inflammatory products, the pain, the muscular spasm, and the altered attitude. The patient spares the affected joint when he can. This results in a limp in the lower extremity, and in a vicarious use of the unaffected upper extremity.

Local Temperature

The local temperature varies somewhat as the severity of the disease. Usually it is not perceptibly raised in the ordinary cases, but it may be quite high in the face of a secondary infection.

Muscular Atrophy

Early atrophy of the muscles moving the joint is fairly characteristic of joint tuberculosis—but not of the pure synovial disease. It is partly the atrophy of disuse, but an added element in its causation exists that has never ben explained. The constant contraction of the muscle interferes with its nutrition (?). All the tissues of the limb, as time goes on, come to participate in the atrophy, so that, under certain forms of treatment, the limb is greatly withered. Any of the above symptoms or all may be present during the course of the disease. Us-

Outcome

ually the march is fairly steady, and, untreated, goes on to partial destruction of the joint. Abscesses appear, approach the surface, burst and become secondarily infected. The constitution becomes involved and the patient dies of septic intoxication, exhaustion, tuberculosis of some other

organ, or amyloid degeneration. On the other hand spontaneous cure may result.

Synovial Cases Milder than Bony

The purely synovial cases are distinctly milder in their course. They can exist for years without seriously damaging the joint. Tuberculosis is not to be ruled out because, after the disease has lasted for several years, the patient is in health, and his joint is simply moderately swollen, sensitive, and limited in its motion. It is not unusual for the surgeon to hear from an adult patient a year or two after examination that he is "practically" well without treatment.

The Roentgen Picture

The Röntgen picture of a tuberculous joint is what one would expect from a study of the pathology, and presents the features common to all cases in Type I, namely, a thinning, erosion, or disappearance of the articular cartilage, and a thinning and porosity of the bone. The bone often presents a worm-eaten appearance. In the later stages of the slow cases a thickening and condensation of the bone is apparent in areas. The shafts of the long bones also show an atrophy. In the short bones the bone trabeculae lose their distinctness. In children the epiphysis may have "furred" edges. A properly developed plate may show a thickening of the joint capsule.

POTT'S DISEASE.

Tuberculosis of the Spine

The disease in the spine presents certain peculiarities. The laminae and other processes, composed as they are of dense bone, as a rule escape its ravages, but the spongy bodies are eaten away, thus depriving the column above of its base of support. As the bodies crumble, the weight is assumed

by the articular processes located behind them, and the column above bends slowly forward, swinging on the articular processes as on a hinge, and giving to the spine at the affected region an angular convexity backward—kyphosis—one of the principal signs of Pott's disease. The angularity of the kyphosis distinguishes it from that formed as the result of most other diseases. It is wont to be prominent when it occurs in the thoracic region, and is an earlier sign in children than in adults. Above and below the kyphosis the normal spinal curves change.

The Kyphosis

Spasm of the back muscles is an early sign of Pott's disease, and causes the patient to assume a stiff, rigid attitude. The attitude varies according to the location of the disease. A familiarity with the normal attitude and with the normal spinal curves enables one to recognize any departure from them.

Pressure upon the roots of the spinal nerves causes the referred pains heretofore mentioned. It causes also various other disturbances, such as grunting respiration, cough, etc.

Pressure Upon Spinal Nerves

The inflammatory products about the cord sometimes exert pressure upon it, and interfere with its functions. Incontinence or retention of urine and feces may result. In certain cases a spastic paralysis develops in the lower extremities—Pott's paraplegia. This is seen most often in disease of the upper thoracic region, and in adults may be the cause of the patient's first visit to the surgeon.

Potts' Paraplegia

If the morbid process involve the lateral spinal articulations, a lateral curvature ensues.

The disease may follow the usual course, or it may exist for many years with progress so slow that a diagnosis is not made.

SPINA VENTOSA.

Tuberculosis of the Fingers and Toes

The finger and toe joints are occasionally affected in adults. In children the shaft of the bone is vul-

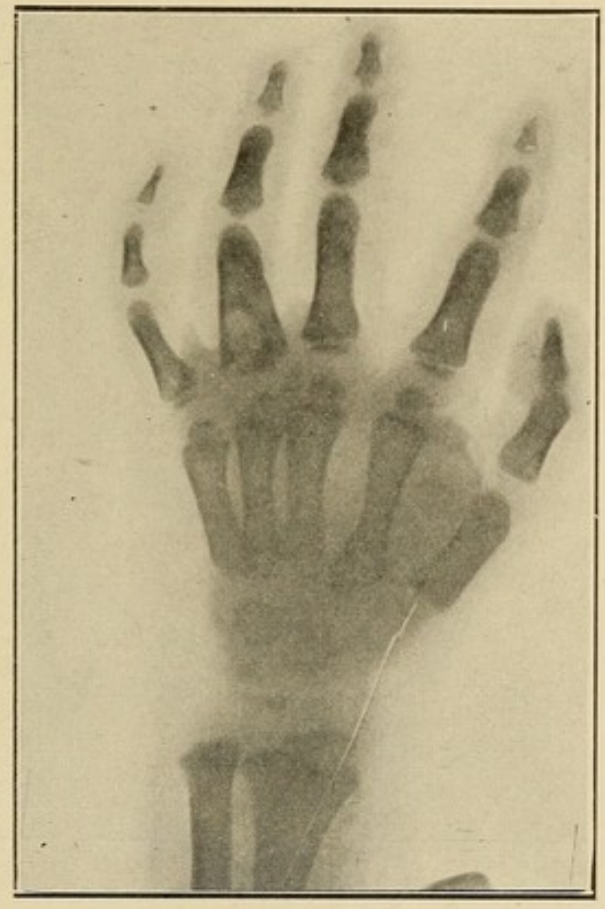

Fig. 29.
Tuberculosis of the phalanx in a child—spina ventosa.

nerable to the tuberculous affection. The disease eats away the interior of the bone, and at the same time a new proliferation of bone takes place at the periphery, giving to the shaft the swollen, puffed-up appearance which is responsible for the common

name of the disease. Strangely enough the joints in this form of disease show little tendency to involvement, and the diagnosis from syphilitic dactylitis may be impossible to make. Secondary infection is very frequent.

Disease of the Calcaneus

Tuberculosis affecting the calcaneus is met, with a fair degree of frequency, especially in children,

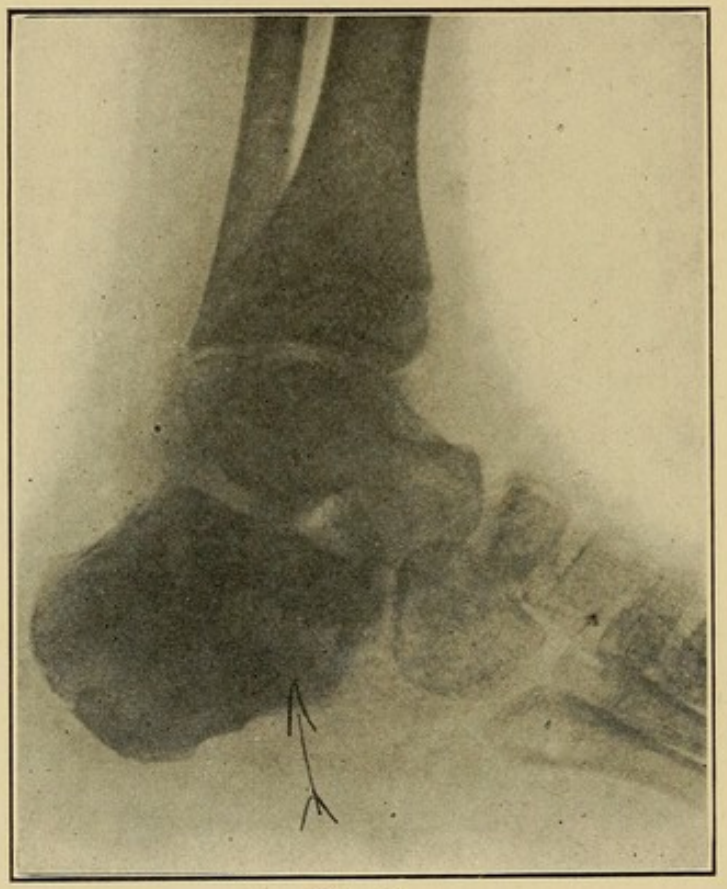

Fig. 30.
Tuberculosis of the calcaneus.

and presents one or two peculiarities. Its favorite site is the anterior portion of the bone, and it seldom if ever involves a joint. Its course is rather tedious.

ABSCESSES.

Abscess

Abscess formation is so frequent an accompaniment of joint tuberculosis that it should be classed

rather as a frequent symptom than as a complication. Its frequency is variously estimated. Some abscesses are so small as to escape detection. The abscess may be formed in the bone or in the joint cavity, and differs from the ordinary acute abscess in that it contains no pus germs. It is an accumulation of serum, a few blood cells, and broken down material, and does not affect the constitution nor produce any symptoms except mechanically by pressure. It may persist indefinitely, it may be absorbed, it may travel by gravity and the line of least resistance for some distance in the fascial planes, or it may become at any time infected by pus germs. In the last case its contents change in their nature, true pus forms, and the symptoms and signs become those of the ordinary abscess.

If the cold abscess rupture spontaneously, or if it be opened with a knife, its walls immediately fall together, and can be seen to be lined by shreds of necrotic material. Practically no swelling is present in the surrounding tissues, the opening into the cavity, called a "sinus," lies flat, and discharges a small amount of a serous fluid. In a day or two, if no precautions against infection be taken, sooner or later in almost every case, the surrounding tissues become inflamed and swollen, the opening becomes puffy and infiltrated and the discharge becomes profuse, purulent and often very foul. The cavity of the abscess now is lined by a porky granulation tissue, which on section is found to be filled with tubercules, and these porky granulations can be found lining the walls of every tract leading to the original focus in the joint.

Infection of the Abscess

A marked constitutional reaction accompanies this process. The temperature shoots up, and the patient becomes acutely sick. The constitutional symptoms persist for a while, and then slowly subside, but the sinuses and abscess cavity, once secondarily infected, may remain open for years, and in the end may be indirectly the cause of death.

Fever

Fever is not a sympton of tuberculous joint disease. If the patient be kept out of doors and in good condition his temperature may remain consistently normal. High temperature means the involvement of some other organ, or the occurence of a secondary infection.

COMPLICATIONS.

Complications

Tuberculous disease of the lungs is a frequent complication in adults, meningitis in children. The last is invariably fatal. Tuberculous adenitis is fairly frequent, tuberculosis of other organs also. Amyloid degeneration is a natural consequence of prolonged suppuration, and unless amputation remove the limb in the very earliest stage of the affection, it is also invariably fatal.

PROGNOSIS.

The prognosis as to life is good in children and in adults under proper treatment. Simple joint tuberculosis involves in itself absolutely no danger to life, except the cases located in the cervical spine, on account of the proximity of vital structures; but we fear the complications. A tuberculosis in a patient must bring these before our minds. A secondary infection immediately makes the prognosis much worse.

The various joints carry a different prognosis; the smaller the joint, as a rule, the better the outlook. Tuberculosis of the spine in adults has always been practically hopeless, but the recent advances in treatment threaten to change this. Tuberculosis of the sacro-iliac articulation is usually fatal. A prompt resection of an adult's joint often improves his general health. Many robust men are walking about, whose tuberculous knees have been resected. Much depends on the treatment, and upon the willingness of the patient to carry it out.

The Smaller the Joint, the Better the Prognosis

The functional result varies somewhat as the age. In children the result under the best treatment varies from complete bony ankylosis to good function. I have heard surgeons of experience say they would be satisfied with a perfectly stiff joint in a good attitude, and without shortening. Others claim excellent function in most of their cases. Personally I am inclined to side with the former.

Result

As to adults: Some years ago, as the result of clinical and pathological studies, I made the statement that painless motion in the adult tuberculous joint was an irridescent dream. Others have reached this conclusion before me. I have never yet seen anything to make me change the opinion, that a tuberculous joint cured by conservative means with good motion, in an adult, was simply a case of erroneous diagnosis. Patients disappear and send word back to the surgeon with some exhibition of glee, that his prognosis was erroneous, but in the end they must submit to the loss of their joint. I have yet to see a proved case of joint tuberculosis in an adult that recovered with motion, and this in

Good Function Very Rare in Adults

spite of the fact that all my training has been on conservative lines.

Children may recover with good function, with fibrous ankylosis, or, if a secondary infection has taken place, with bony union. In adults the result is always a fibrous ankylosis, and the old tubercles, more or less encapsulated, may be set free by trauma at any time.

DIAGNOSIS.

No Infallible Way to Make a Diagnosis

From other cases in Type I. tuberculosis often can be distinguished by its slow, more or less steady course, its almost invariable unarticular nature, by the frequent accompaniment of other tuberculous lesions in the body, by the general absence of acute exacerbation unless an abscess form, or a trauma be received. The tuberculin test is suggestive but not final. If a piece of the synovia were examined under the microscope it *might* be conclusive, but only if tubercles were found in it, and it must be remembered that tuberculosis might exist in the membrane and not in the specimen examined. It is rarely justifiable to open the joint for diagnosis, for the disease may be made worse by the trauma. Perhaps the best way to reach a positive diagnosis is by withdrawing some of the joint fluid and by injecting it into the abdomen of a guinea-pig. If the joint be tuberculous the animal will die of tuberculosis within six weeks. In case of doubt, the rule should be to wait and watch the joint for a few months.

When in Doubt, Wait

From cases in Type II. the disease is differentiated by its evidences of active inflammation, by the absence of bony growths about the joint, and by

the Roentgen picture. This shows a worm eaten appearance of the bone, rarefying osteitis in places, and if the disease is slow, areas of increased density.

An hysterical joint presents a total disproportion between the objective and subjective signs. While the patient complains of great pain, evidence of any anatomical change is lacking. If the patient's attention can be diverted, the affected joint can be manipulated without pain. A search will reveal hysterical stigmata.

New Growths

Sarcoma and *benign myeloma* occur in the metaphysis, *but almost never involve the joint.* The swelling and sensitiveness are above or below the joint in the end of one bone. The other bone is uninvolved. Motion is little restricted if at all, and then only by mechanical interference.

Scurvy

Scurvy in civilisation is seen in bottle-fed infants, comes on rather suddenly, and is characterized by a very painful swelling near the joint. The child screams with pain when it is moved, and often has bleeding gums, and hemorrhages into the skin and mucous membranes. Raw fruit juice causes the symptoms to disappear quickly.

"Sprains" may cause confusion in children. Immobilisation for a few weeks will cure all sprains.

Trauma

Intracapsular fracture, and separation of an epiphysis are distinguished by the Roentgen rays.

Charcot's joint is a local manifestation of tabes dorsalis or syringomyelia. The evidences of one of these diseases will be present. Pain is absent. Disorganization exists rather than inflammation. The joint is loose and "wobbly," grates and creaks and contains fragments of bone and cartilage. Motion is unrestricted. The onset of a Charcot joint is

usually sudden—it fills quickly and painlessly with fluid, and the subsequent swelling extends well beyond the limits of the joint cavity.

GENERAL TREATMENT.

Constitutional Treatment

As in any form of tuberculosis, so in this, constitutional treatment is most important. The presence of a tuberculous joint implies a poor resistance to the disease, and it is most important that the patient's general health be kept at the highest possible pitch. Good food, and plenty of fresh air for the entire day and night, are essential. The patient must sleep out-of-doors if he can. Sleeping in the open air is practicable for almost everyone. No form of climate is specific against the disease. The seashore agrees with children, but simply because it seems to improve their general condition. Adults with a complicating pulmonary lesion should live in a high, dry climate if they can. Rollier and his disciples have recently published remarkable results of treatment by heliotherapy.

Heliotherapy

The digestion must receive careful attention. Adenoids and diseased tonsils must be removed.

Drugs are useless except to meet special indications. Cod liver oil is an excellent form of fat for the winter months.

LOCAL TREATMENT.

In our study of the pathology and of the symptomatology we have learned two most significant facts, 1st. The existence of tuberculosis in the region of the joints is dependent upon the presence of the synovia and the lymphoid marrow. The presence of the lymphoid marrow and synovia is dependent

upon function. If function be removed, these two tissues disappear. If they disappear the disease dies out, because it has no food. It is starved out. All nature's efforts are expended to deprive the joint of function. 2nd. A secondary infection by pus germs greatly adds to the dangers of the disease. Therefore, our two main rules for treatment are: 1. Deprive the joint of function. 2. Avoid secondary infection.

Principles of Treatment

Radical operations on children's joints are often ineffectual, because lymphoid marrow is found in the shafts, and we have no way of telling just how far the disease has spread. These operations cause unsightly and crippling deformities by interfering with the centres of growth in the ends of the bones, and the testimony of those who have practised conservative treatment is to the effect that tuberculous joints in children can often be cured thus with function. Again, it is possible to carry through with a child a long course of treatment that would be impracticable for an adult. Therefore, in children the treatment is almost invariably conservative. We carry it through in the face of almost every obstacle until all hope of saving the limb is gone. Then we amputate to save life. Beginning ankyloid degeneration, or a prolonged septic absorption may demand amputation.

Conservative Treatment in Children

In adolescence we adopt conservative treatment until growth is finished, or almost finished. Then, if the disease be not well, we adopt the measures suitable for adults.

Conservative treatment is rarely if ever successful in adults, and is almost impossible to carry out. Under the best circumstances the only thing we can

possibly hope for is a stiff joint. This we can attain by operation in a few months, and can certainly cure the disease. Therefore, we maintain that the treatment of a tuberculous joint in an adult is almost invariably radical. Bearing in mind, however, the statement of some authorities that occasionally the milder forms recover under conservative treatment, and bearing in mind the fallibility of our diagnosis, it is often well to try conservative measures for a few months. Then, if the joint be not much improved, and if we be sure of our diagnosis, radical treatment is in order.

Radical in Adults

The object of conservative treatment is to deprive the joint of function temporarily; the object of radical treatment is to destroy the function in the joint, to deprive it of function permanently.

CONSERVATIVE TREATMENT.

This consists of immobilization by "braces," plaster of Paris dressings, etc., and of certain other measures in addition, that may be adopted to meet special indications. Whatever form of appliance be used, it should fulfill two conditions—it should prevent deformity and should deprive the joint of function. All deformity should be reduced before the apparatus is applied, by rest in bed with traction, by manipulation under ether, or by the application of several plaster bandages at intervals, correcting the deformity a little at a time. The second method should be done very carefully for fear of aggravating the disease. No patient should ever be sent to the brace-maker with the instructions to get a brace. To do this is to turn over to a mechanic the duties of the surgeon—duties the mechanic is

Reduce Deformity

not fitted to perform. If the surgeon be unwilling to prescribe the exact form of brace to be used, and to supervise its application, he should use plaster of Paris or refuse to treat the case. No practitioner would think of turning over the conduct of a case of pneumonia or appendicitis to the apothecary,

Do Not Permit a Brace Maker to Treat the Case

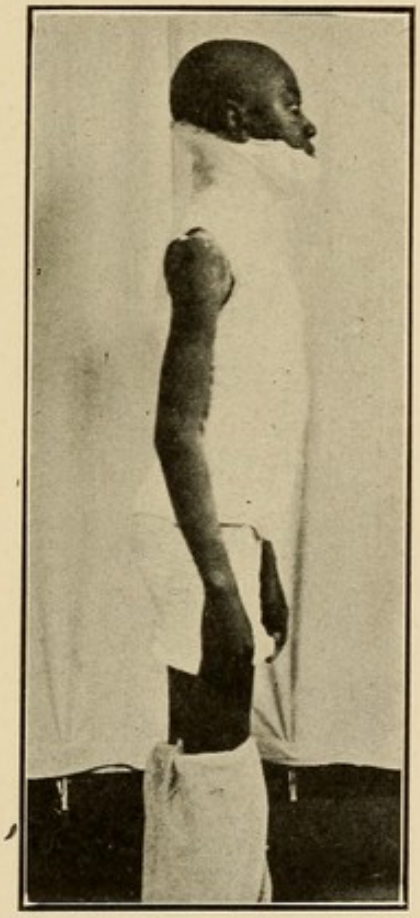

Fig. 31.

Grand Calot Jacket, an excellent dressing for high thoracic, or for cervical Pott's disease.

but unfortunately the other error, just as bad, is quite a common one.

Braces

A brace is a metal splint fastened on with straps and buckles, and sometimes reinforced with felt, celluloid, etc. It is an excellent means of treatment, if accurately fitted and carefully planned. It

must be as light as is consistent with strength, and must be comfortable. A child will usually loosen the apparatus if he can, and the problem of preventing this is often a difficult one.

Plaster of Paris

Plaster of Paris dressings are serviceable, efficacious, and, with a little practice, fairly easy to ap-

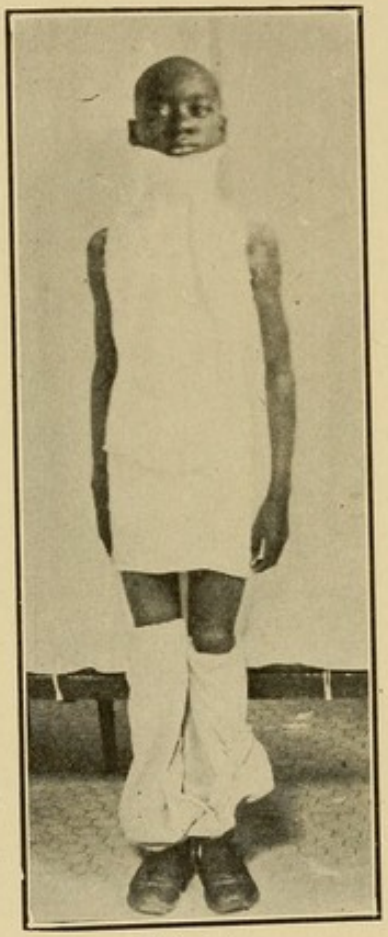

Fig. 32.

Grand Calot Jacket—front view.

ply. They should fit snugly, but not too tightly, *should reach well above and below the diseased joint,* and should be as heavy as is necessary to withstand the strain to which they are exposed, but no heavier. A dressing for the knee should reach from the perineum to the malleoli; for the hip, from

the waist to the knee; for the ankle, from the knee to the toes, etc. In other words the plaster must have a suitable leverage. Bony prominences should be carefully padded. Padding is not necessary over muscle bellies. If a tight seamless shirt or stocking (stockinette shirting) be put on the limb before the plaster be applied, the comfort of the patient will be subserved. A "scratch bandage" is a strip of linen or cotton next to the skin run longitudinally under the shirting, which is drawn up and down daily to keep the skin in condition.

Scratch Bandage

Ingredients of the Plaster Bandage

Fine dental plaster is used in making the bandages, and a brand of crinoline made up with starch but not with glue. With good materials the plaster sets quickly, and no salt in the water in which the bandages are soaked is necessary or advisable.

When the joint is immobilized it should be put up in the position most favorable for function if ankylosis ensue—the hip in extension and slight abduction, the knee in full extension, the ankle and elbow in right-angled flexion, the wrist in slight superextension.

Cessation of Brace Treatment Should Be Gradual

When treatment by apparatus is begun it should be continued without intermission until the disease is cured. Then it should be gradually discontinued, while the joint is carefully watched. First the apparatus is left off at night, then for one or two hours a day, etc.

Advantages of Plaster Dressings

Some surgeons prefer braces, some plaster of Paris. My personal preference is strong for the latter, when it can be used. It leaves nothing to the intelligence or care of the patient or of his parents except to watch for excoriations under it. These are manifest by a foul odor, and demand the

immediate removal of the dressing. Plaster of Paris fits more exactly than can any brace, and cannot be removed without the knowledge of the surgeon.

Tuberculin

Tuberculin Treatment. The general consensus of opinion seems to be that this is not of great value in joint tuberculosis.

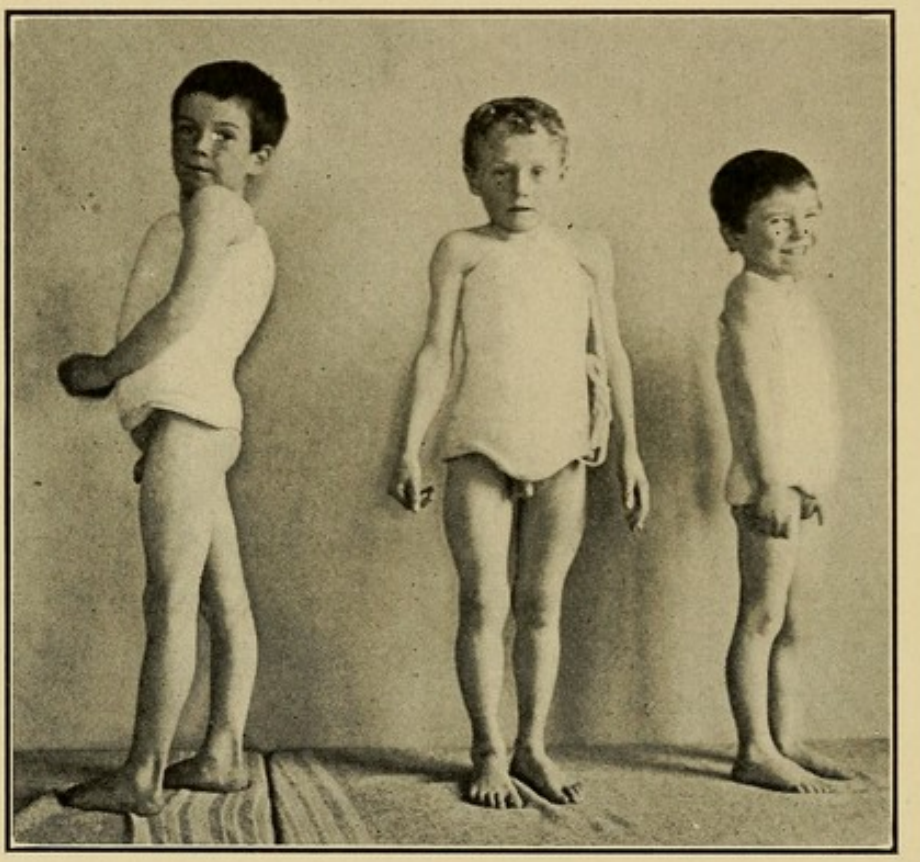

Fig. 33.
Ordinary plaster of Paris jackets.

Bier Treatment

"Bier Treatment," Passive Hyperemia. "Stauungshyperaemie" some years ago enjoyed quite a vogue, but seems not to have held its own. Some authorities still advocate its use, and in certain instances we may think it worth a trial.[1]

[1] Bier. Hyperaemie als Heilmittel. Leipzig. F. C. W. Vogel, 1907.
Klapp, Archiv für klinische Chirurgie, 1906, LXXX, 42.
Ely, *Surgery, Gynecology & Obstetrics,* January, 1910.

Injection Treatment

Treatment by Injections. The gamut has been run on all sorts of materials injected into tuberculous joints. Their employment does not seem rational. Nothing we can inject into a joint will produce any direct effect upon tubercles in the marrow or in the substance of the synovial membrane, or upon dead bone and cartilage. These injected substances may possibly act, however, as irritating the joint tissues, and so inducing the production of fibrous tissue. Any sterile, non-poisonous substance will serve about as well as another.

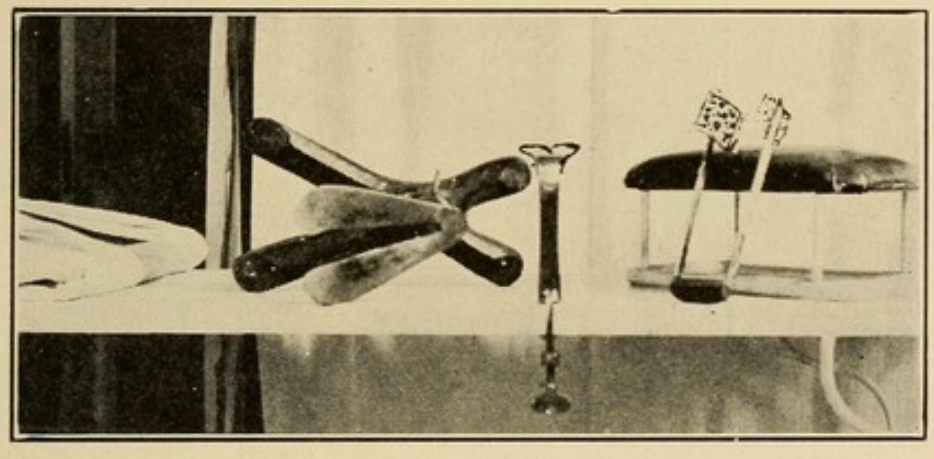

Fig. 34.

Apparatus for applying plaster spica. From left to right, Schultze pelvic rest, author's pelvic rest, head and shoulder rest. On the last is leaning a Lorenz stirrup for incorporation with the plaster, when the limb is to be deprived of weight bearing.

RADICAL TREATMENT.

The Knee

In almost every instance our object is to destroy function, and the simplest and easiest way to destroy function is by a resection. We do not attempt to remove all the tuberculous tissue, for we know that usually this is quite impracticable and unnecessary. We remove just enough of the ends of the bones to secure apposition of bare bone to bare bone, and then aim at bony union and healing

Object of Radical Treatment

of the wound by first intention. Given bony union without a secondary infection in an adult, the tuberculous disease is ended. Wire nails, screws, pegs, etc., have all been advocated in resections, but do not seem necessary. The bone ends can be held in apposition by the external plaster of Paris dress-

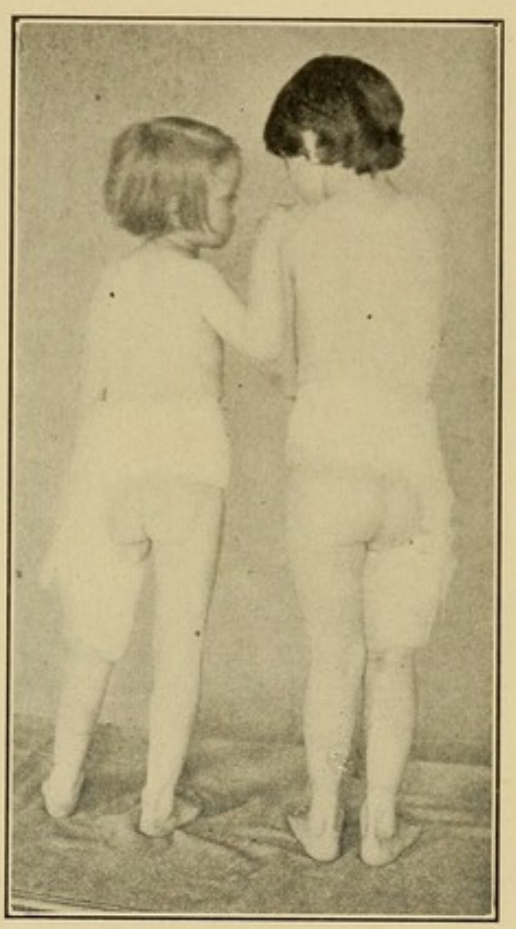

Fig. 35.

Short plaster of Paris spicas for ambulatory treatment of hip joint disease. Note the excellent general condition of the children, and the absence of muscular atrophy in the affected legs. For anterior view of spica see chapter on acute arthritis.

ing, and the fewer foreign bodies we put into the wound, the less danger there will be of secondary infection.

This rule has a few exceptions. In disease of the carpus and tarsus no operation can be done that

will cause the necessary change in the synovia and marrow. Therefore, unless we can get the case in the very earliest stages and remove the diseased carpal or tarsal bone before invasion of one of the extensive synovial membranes, we shall be obliged

Tarsal and Carpal Disease

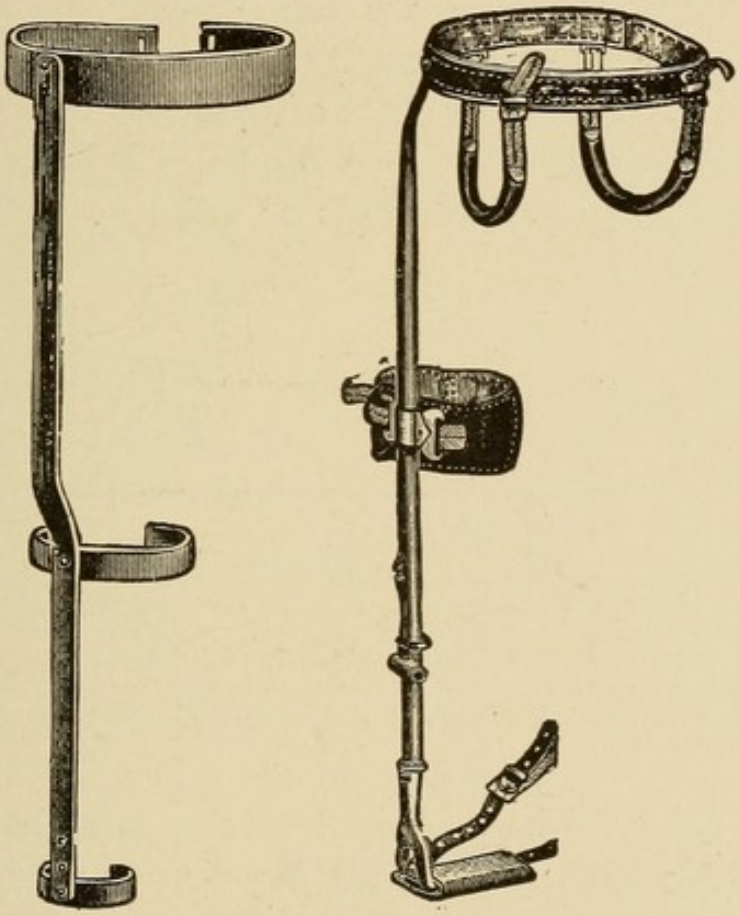

Fig. 36.

Hip splints, ordinary traction brace on right, Thomas brace on left.

to resect the entire wrist or tarsus. If once a secondary infection takes place in these joints an amputation will almost certainly be necessary.

Tuberculosis of the finger or toe joints in an adult is best treated by amputation.

Fingers and Toes

In the hip we can cure the disease by producing

The Hip

an ankylosis (Albee's operation[1]), or by removing just enough of the head of the bone to produce a dislocation. In either case the joint is destroyed. The former operation gives a result better for walking, the latter for sitting. A moment's consideration will teach the utter futility of attempting to remove all the tuberculous tissue from this joint. To do so would necessitate the removal of the entire acetabulum and a large part of the ilium.

The Elbow

In the elbow one of two results is sufficient for cure, namely, bony ankylosis or a fibrous union of the bone ends.

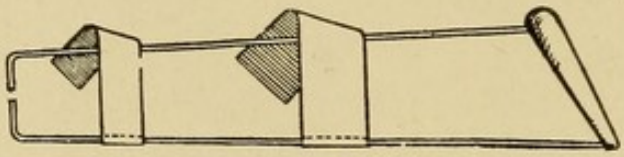

Fig. 37.

The Thomas brace for use in tuberculosis of the knee or ankle (Ridlon). The ends of the wire are usually welded together and shod with a foot piece of rubber or leather. When used for the knee the brace is held on by extension adhesive straps on the leg, when used for the ankle, the ring is provided with a strap of webbing which fastens on anteriorly and posteriorly, and passes over the shoulders.

The Spine

The spine. The problem here presented has until recently been too difficult for solution. The tuberculous disease, located anteriorly, in the bodies of the vertebrae, destroyed these in whole or in part, together with the intervertebral discs. To eradicate the diseased tissue is impossible, and operative attempts to do so simply made matters worse by carrying in a secondary infection. The operation of Albee[2] and that of Hibbs[3] promise good re-

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[1] Albee, *Surgery, Gynecology & Obstetrics,* March, 1910.

[2] Albee, *Journal of the A. M. A.,* 1911, LVII, 885, *N. Y. Medical Journal,* 1912, XCV, 469.

[3] Hibbs, *Journal of the A. M. A.,* 1912, LIX, 433.

Albee's Operation

sults, however, for they are founded on sound pathological principles. Both authors ignore the tuberculous disease in the bodies of the vertebrae, and direct their attention to securing a bony splint, by operations upon the spinous processes. Albee splits the spinous processes of the diseased verte-

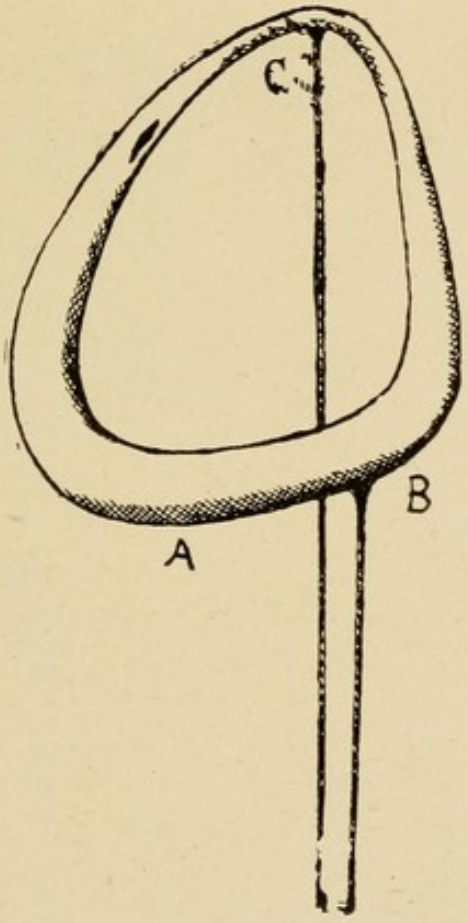

Fig. 38.

The ring of the Thomas brace (Ridlon). A—posterior portion, B—inner bar, C—outer bar.

brae, and of one or two above and below, and inserts in the interval a piece of the patient's tibia covered with its periosteum, sewing the wound up over the graft. The graft unites with the raw surface of the split spinous processes, and forms a bony splint.

Hibbs' Operation

Hibbs fractures the spinous processes, turning each one down on the fractured base of the one below.

ABSCESSES.

Treatment of Cold Abscess

Our great concern in the treatment of a cold or tuberculous abscess is to keep it from becoming

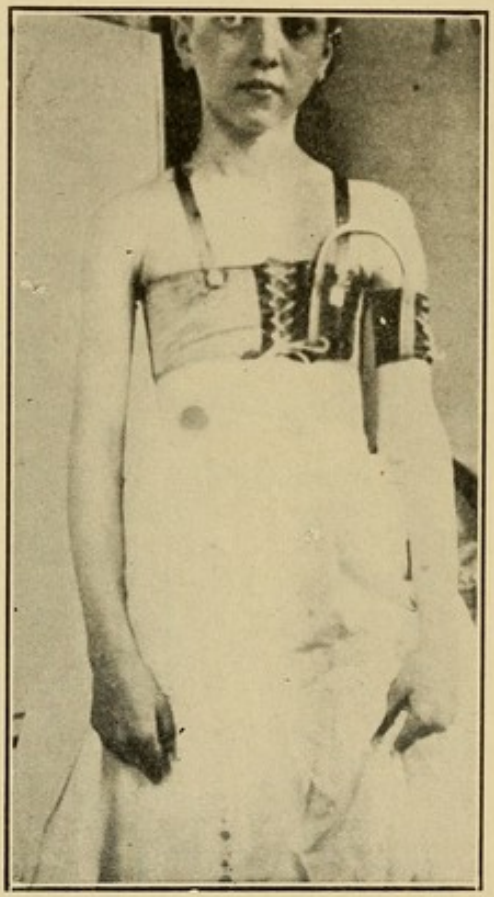

Fig. 39.
Author's brace for shoulder joint disease.

infected. Inasmuch, therefore, as we know that it will frequently be absorbed and disappear, if it be small, deep-seated and symptomless we let it severely alone. If larger, or more superficial, or if it cause pressure symptoms, it must be attacked, but in such a manner as to avoid secondary infection.

In spite of our efforts abscesses in the more superficial joints will rupture sooner or later almost invariably.

We have learned that there are seldom if ever any tubercles in the walls of a cold abscess, or anything else that needs removal. Therefore, we need not scrape the walls, or interfere with them in any way. Our sole object in interference is to let out the contents without carrying in an infection. Continuous drainage instead of providing for the exit of tuberculous tissue, really provides for the entrance of pus germs. The best treatment for a tuberculous abscess is to attack it when it reaches any considerable size or approaches the surface. It should be aspirated, and, when it fills up again, it should be aspirated again. After a few repetitions of the operation the abscess will usually cease to fill. A bottle aspirator is advisable to prevent the entrance of air. On account of the flocculi, and shreds of necrotic tissue, a very large aspirating needle is necessary.

Drainage Provides for Entrance of Pus Germs

Aspirate

Having emptied the abscess what shall we do next? Various authorities have recommended the use of injections of many different substances—bismuth paste, iodoform in vaseline or in ether, carbolic acid, etc.—for their effect on the tubercles in the abscess wall, but, as there are no tubercles in the wall, and as none of these substances has been proved to have any effect on the tubercles if they were there, we shall maintain an attitude of scepticism, and use empirically whichever we choose, or shall discard all, and simply apply gentle equable pressure with our sterile dressing.

Bier and Klapp recommend for the treatment of

these abscesses, small incisions and subsequent cupping.

Psoas Abscess

The psoas abscesses of Pott's disease should be attacked in the iliac region, before they make their way down into the thigh. The abscess can be distinctly palpated. It pushes the intestines upward, and is usually tapped at a point about one inch internal to the anterior superior spine, and one inch below it.

Postpharyngeal abscesses should never be opened through the mouth on account of the practical certainty of secondary infection. They are best attacked from the side of the neck.

INFECTED ABSCESSES.

Bismuth Paste

If we can conquer the secondary infection, we can often heal up the abscess. Following out Beck's idea[1] we inject the cavity with some form of paste, but as a number of fatalities has been reported from bismuth paste, we shall do well to omit the bismuth ingredient. Blanchard,[2] of Chicago, recommends

Other Pastes

the following mixture: white wax, one part; vaseline, eight parts; mix while boiling. The paste is heated in a water bath until it is fluid, and then it is injected with a glass syringe, with considerable force, into the opening of the sinus, so to fill up every nook and crevice of the tract. Sterile gauze is clapped over the opening quickly to prevent the exit of the paste until it has a chance to harden at the body temperature.

No paste should ever be used if the Roentgen rays show the presence of a sequestrum, nor does

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[1] Beck, Transactions of the Sixth International Congress on Tuberculosis, 1908.

[2] Blanchard, *Medical Record,* May 18, 1912.

any paste have any direct effect upon the tuberculous process in the joint. It simply sets aside the secondary infection.

Klapp's Treatment

Klapp recommends cupping of infected sinuses to draw out the secretion, using passive hyperemia (Bier treatment) in addition.

Pott's Paraplegia

Pott's Paraplegia demands rest in the recumbent posture for months, on a frame or in plaster.

Spina Ventosa

Spina Ventosa needs clean dressings and patience. Suction hyperemia will often be found useful. Bone grafting operations have been done with good results.[1] Amputation is rarely justifiable.

Calcaneal Tuberculosis

Tuberculosis of the calcaneus. If a sequestrum be present it should be removed with as little damage as possible to the surrounding bone. Under no circumstances should our ideas on the necessity of eradicating the disease lead us to carry the infection into a joint. The bone cavity may be filled with some non-toxic paste, or the Bier-Klapp treatment may be worth a trial. Occasionally resection of the calcaneus will be found necessary.

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Rogers, Boston Medical and Surgical Journal, June 8, 1911.
Fraser, Edinburgh Medical Journal, November, 1912.
Brandes & Mau, Deutsche medicinische Wochenschrift, 1913, XXXIX, 1137.

CHRONIC GONORRHEAL ARTHRITIS.

Because a gonococcic arthritis usually begins as an acute inflammation, we have described it under the head of acute arthritis, but unless the primary inflammation in the genito-urinary tract be healed,

(1) Ahrens, Berliner klinische Wochenschrift, 1909, No. 48. 2167.

the disease is wont to persist as a chronic inflammation and to extend for years, until it destroys the function of the joint. When this result is attained the inflammation subsides, as it does in any other organ.

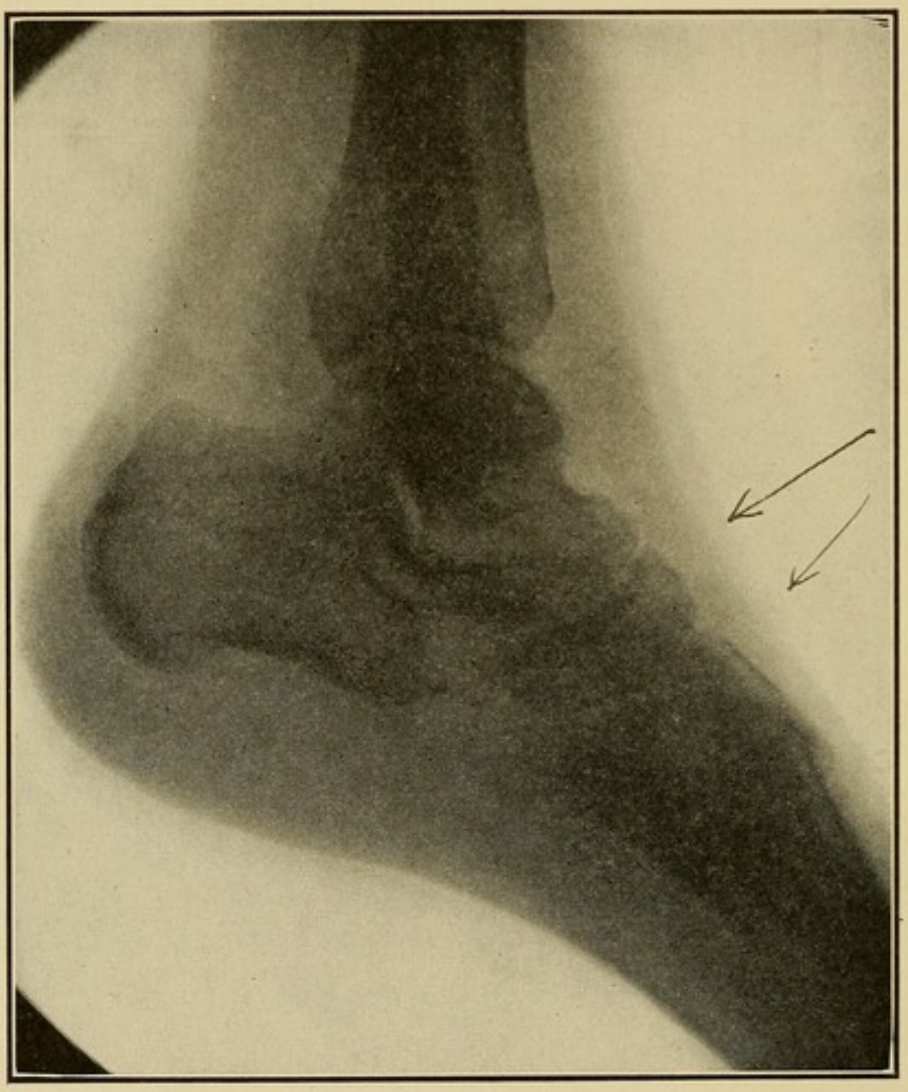

Fig. 40.

Chronic gonorrheal arthritis in a supposedly tuberculous (pulmonary) patient. The diagnosis was based on the condition of the urethra and prostate, the multiarticular lesions, the reaction to appropriate treatment and the relation of the clinical picture to the Röntgen ray findings.

Morbid Anatomy

Pathology. The synovia, after the early stage of proliferation, gradually undergoes a fibrous

change, and the joint becomes a mass of adhesions. Probably the cartilage also becomes fibrillated in its structure. Just how much part the lymphoid marrow takes in the process it is hard to say, owing to the difficulty of securing specimens with a definite and complete history, but from skiagrams and from

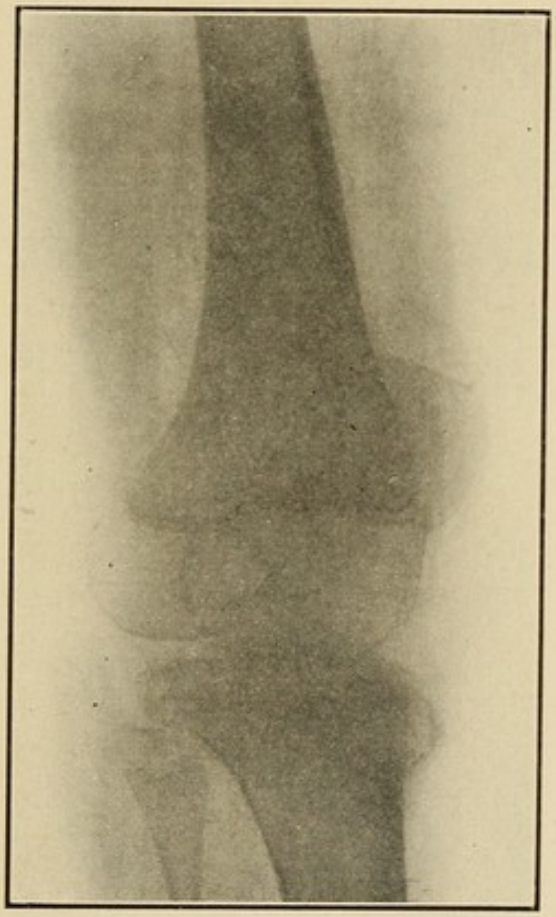

Fig. 41.

Hereditary syphilis of the knee joint of a child after treatment for two years with a diagnosis of tuberculosis, lateral view. Note the diseased tibial epiphysis, and the productive osteitis of the femur shaft about four inches above the joint.

bacteriological examination of material removed at operation, it is evident that the inner layer of the periosteum reacts to the disease, and produces a picture hardly to be distinguished from tuberculosis or from the other diseases of Type I.

Symptoms

Symptomatology. The joint is shrunken and stiff, and, as long as the morbid process is still active, painful. When the disease has reached its logical outcome—complete stiffness—the pain disappears. While a moderate degree of deformity may be present, the extreme contractures so char-

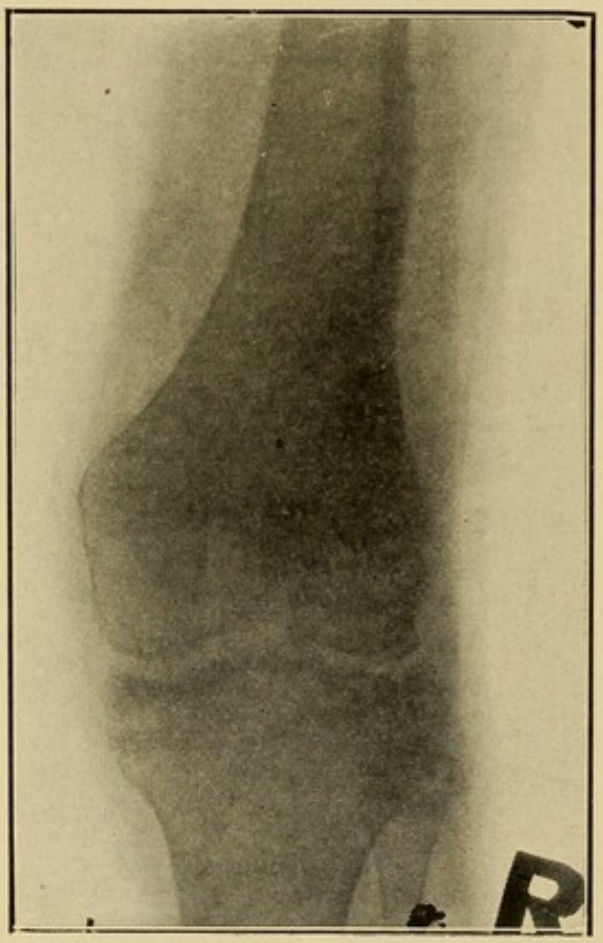

Fig. 42.

Same case as Fig. 41, antero-posterior view.

acteristic of joint tuberculosis are not seen. This is what one would expect in a disease so largely synovial and with so little destruction of bone. The muscles of the limb are atrophied. The joints most frequently involved are the knee, the foot (not the toes), and, in women, the wrist.

From the absence of bony outgrowths (usually), from the appearance of the skiagram and of the joint itself, and from the history, it is usually easy to determine that the disease belongs in Type I. To determine the exact nature of the process may

Diagnosis

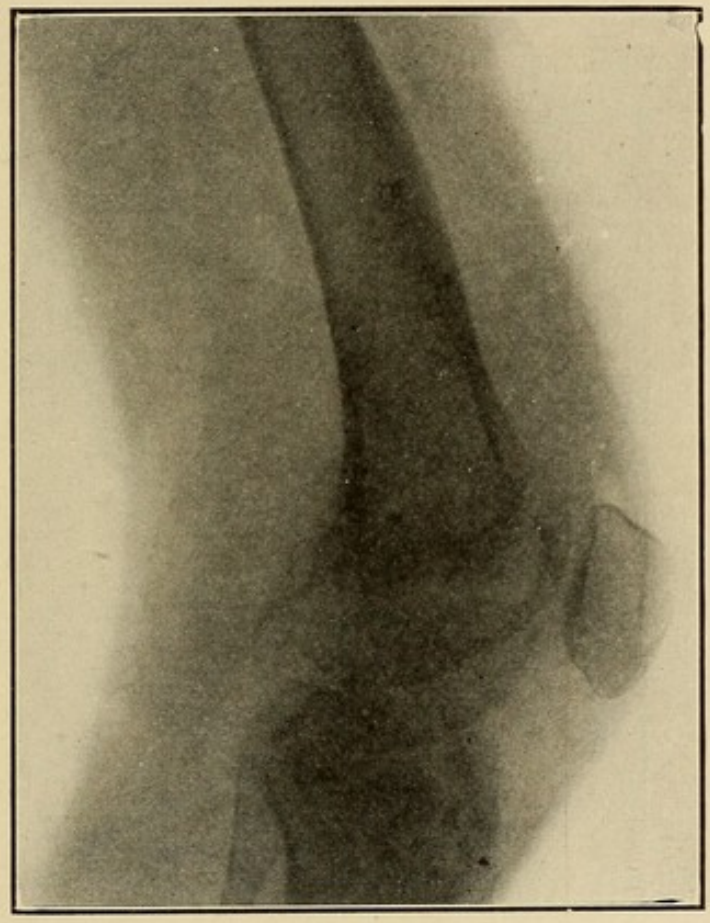

Fig. 43.

Same case as Figs. 41 and 42, after two injections of salvarsan and a course of mercurial inunctions. Note the improvement in the tibial epiphysis, and the gouged-out appearance in the femoral epiphysis. Skiagram taken four months after the preceding. The clinical improvement was marked, and the joint was practically well, except for a moderate restriction of motion, which was slowly improving.

be more difficult. If the patient were an adult male, and gave a history of a urethral inflammation at or about the time of the onset of the joint trouble, and if two or three joints were involved, the pre-

sumption of a gonococcic joint would be strong. If an examination of the urine revealed shreds (silver threads among the gold, so to speak) or gonococci, a tentative diagnosis of a gonorrheal joint would be justifiable. On the other hand we must not forget that a patient with chronic gonorrhea

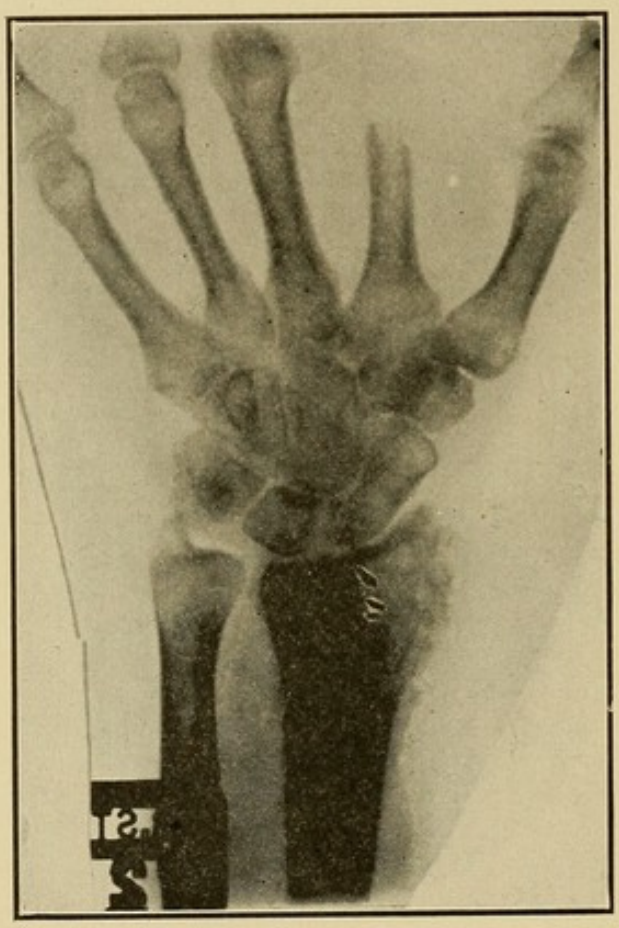

Fig. 44.

Syphilis of the wrist, erroneously treated as tuberculosis. Observe the stump of the amputated finger. For a full description of this case, see Medical Record, 1912, LXXXI, 1179. Skiagram taken Sept. 6, 1910.

might have a tuberculous joint, or that a patient with pulmonary tuberculosis might have a gonorrheal arthritis (see Fig. 40), or that even a child might quite innocently have a gonorrheal joint.

Syphilitic arthritis can often be diagnosed only by

finding some other evidence of syphilis in the body, by the presence of a broken-down gumma in the bone end (*chronic* gonorrheal arthritis probably never ends in suppuration unless as a result of operation) by the Wasserman reaction, or eventually by a therapeutic test.

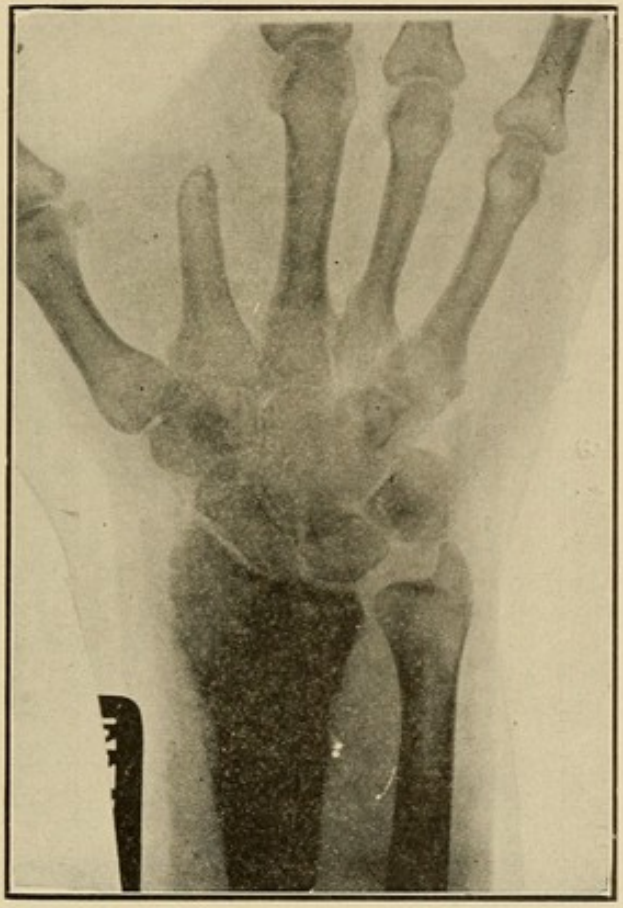

Fig. 45.

Same case as Fig. 44. Skiagram taken Feb. 7, 1911. Note the improvement under constitutional treatment alone. This never occurs in adult joint tuberculosis.

Treatment. The first requisite is to set the genito-urinary tract in order. After this is done, mobilising operations may be done if *necessary*, but until the source of infection is removed, it is evident that any attempts at mobilisation will only

Treatment

Passive Motion

aggravate the joint trouble. Perhaps the wisest course is to begin with vigorous manipulation under an anesthetic, and then to fix the joint with plaster of Paris in an entirely different attitude until the inflammatory reaction has subsided, when gentle

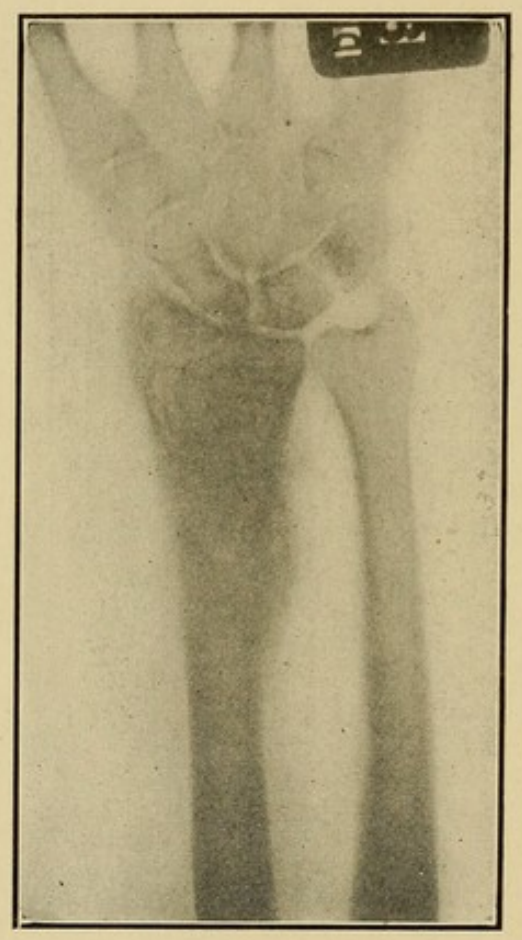

Fig. 46.

Same case as Figs. 44 and 45. Skiagram taken January, 1912, after about one year of desultory treatment by mercurial injections and the iodides. This treatment seems to have brought out the distinguishing marks of syphilis.

active and passive motion may begin. Possibly the operation might be repeated. For other treatment the reader is referred to the section on acute gonorrheal arthritis.

JOINT SYPHILIS.

Syphilitic arthritis has been considered rather rare. On the contrary, I believe it is fairly frequent, but often unrecognized and treated under a wrong diagnosis. I have frequently been guilty of the error and have detected it in other men's work.[1]

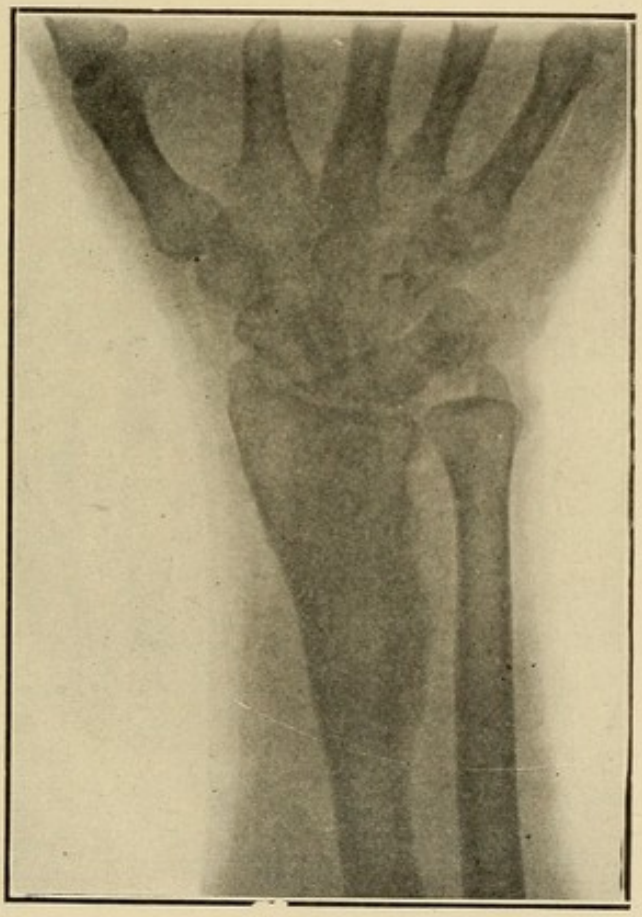

Fig. 47.

Same case as Figs. 44, 45 and 46, about one year after two doses of salvarsan. The patient is well.

Syphilis occurs in the joints in two well differentiated forms, which correspond to the synovial and bony forms of joint tuberculosis, and probably in two others, one of which will be considered later on under the head of the multiarticular,

[1] Ely, *Medical Record*, 1912, LXXXI, 1179.

progressive variety of Type I., and the other under the head of Charcot's joint.

Syphilitic Synovitis

The rarer form of joint syphilis is a synovitis which occurs in the tertiary stage. It is a slow, almost painless affection most frequent in the knee,

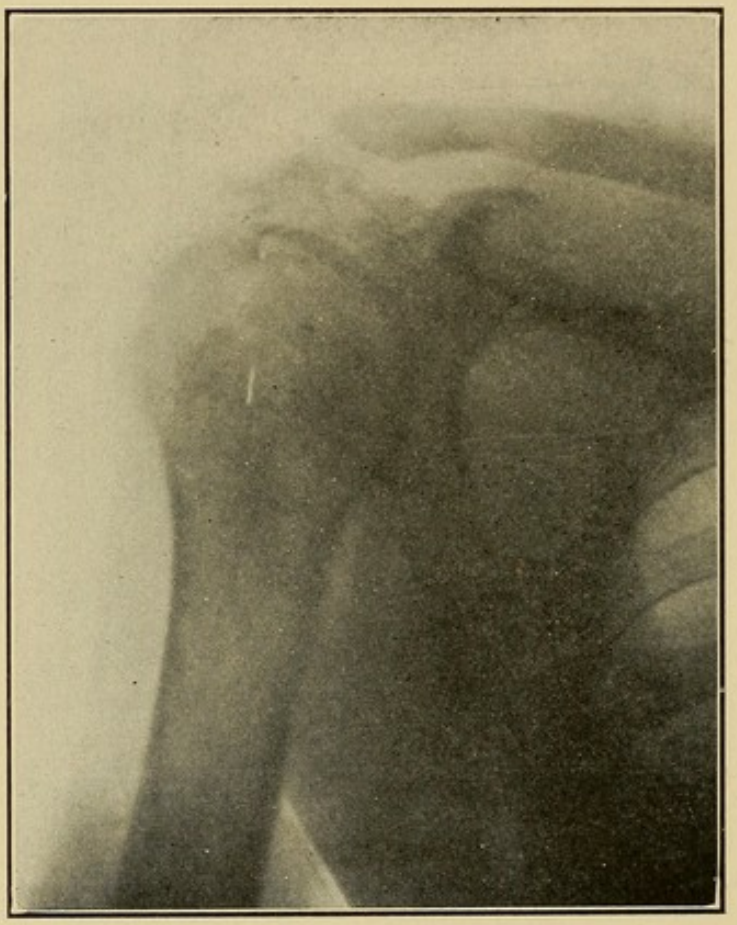

Fig. 48.

Skiagram of the shoulder joint of the multiarticular form of Type I. The patient was an adult of about 45 years. The cause was probably syphilis. Mark the rarefaction of the acromion and of the head of the humerus.

showing little if any tendency to involve the bone, unless as the result of unwise operative measures. The joint is swollen, contains fluid, and is but slightly disturbed in its function. The essential pathological lesion is probably a proliferation of the

synovia with a resulting effusion in the joint cavity. The disease may easily be confused with synovial tuberculosis.

The results of *treatment* by mercury are usually prompt and satisfactory.

Syphilitic Myelitis

A more severe and more frequent form of syphilis occurs as a proliferative inflammation of the marrow and of the inner layer of the periosteum of the bone end, to which an inflammation of the synovia may or may not be added. These, as we have learned, are the essential lesions of joint tuberculosis, and it follows that not only is the clinical picture of the two diseases often the same, but that even an expert Roentgenologist may not be able to differentiate them by a careful examination of skiagrams. Syphilis is often multiarticular, but a uniarticular form also occurs with almost equal frequency, and only by keeping a sharp lookout for other signs of syphilis can we detect its true nature. I believe that this peculiar lesion is often a manifestation of heredity syphilis—*syphilis hereditaria tarda*—and often is the only observed sign of the disease. It may occur in children or in adults, and is much more painful than the preceding, and often more damaging to the function. It may result in the breaking down of the gummatous infiltration and in the formation of a typical, sluggish, undermined syphilitic sinus; in which case the diagnosis is made easier.

Hereditary Syphilis

Treatment. Strangely enough treatment by immobilization is useless, even when continued for years. Mercury has been our sheet anchor, but often these cases, after improving for a while, come to a stand-still, and then clear up after one or two

doses of Salvarsan. In the healing process in children the skiagram may take on an appearance that I regard as diagnostic. The epiphysis presents at some part a large "cavity" (see Fig. 43). It is interesting to watch by skiagrams how the architecture of an apparently hopelessly damaged joint is re-formed under antisyphilitic treatment.

CHAPTER IV.

OTHER CASES UNDER TYPE I.

In the course of time the three foregoing diseases have been described and identified. There still remains in this type a group of cases which have hitherto eluded exact identification. Originally they were thought to be one definite clinical entity, and to form a large part of what was known as "arthritis deformans," but the conviction has been steadily growing that a number of different agents may be responsible for them. It is a fact that under great difference in their course and clinical behaviour many cases of chronic joint disease are seen whose pathology is very similar, and strongly resembles that of the three diseases already described—tuberculosis, syphilis and gonorrhea.

ETIOLOGY.

The exact cause of these diseases for the present must be left undecided. Some writers regard them all as infectious, some believe that some are infectious and that others are due to "faulty metabolism," some introduce the element of disturbance of a center of nutrition in the spinal cord. Evidence is constantly accumulating in favor of the infectious theory, enough for us to feel justified in accepting it as a working hypothesis. We shall tentatively adopt that view in what follows, and

Exact Cause Not Yet Determined

shall assume that every case of chronic arthritis, like every case of acute arthritis, is due to an infection or to trauma.

The arguments for and against the infectious theory may be sketched briefly as follows:[1]

Every Non-Traumatic Case of Arthrition is Due to an Infection

1. Every non-traumatic joint disease whose exact cause and pathology we have worked out is due to an infection, and the general pathological changes in most of the chronic ones are the same as those of this group. Gonorrheal arthritis was lately considered as due to reflex irritation of the urethral mucous membrane.[2]

Faulty Metabolism in Disease

2. Various observers[3] have isolated pure cultures of microorganisms from cases of this group, and in some instances have produced the disease in the joints of animals by injection. The organisms are not always identical, but this agrees with our hypothesis that these diseases may be due to a number of different causes.

3. No center of nutrition for the joints has ever been identified in the spinal cord.

4. "Faulty metabolism" should not be employed as a term to define the causation. It is a cloak to cover our ignorance, a modern equivalent of a "humor" or a diathesis, a result of disease, not its cause. Adami defines health as metabolic equilibrium, disease as a condition in which the equilibrium is wanting. Then we may say that disease is disturbed metabolism. It is seen, therefore, that to say a joint disease is due to faulty metabolism is to say that it is due to disease.

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[1] *American Journal of Orthopaedic Surgery*, November, 1912.

2 Allbutt's System of Medicine, 1901, Vol. III.

3 Schüller, Banantyne, Fayerweather, Poynton v. Paine, et al.

5. Some authorities, while admitting that the cases which begin acutely with glandular swelling, fever, leucocytosis, etc., are infectious, deny the infectious nature of the slow, chronic ones. Acuteness of onset, and leucocytosis are not characteristic of infectious diseases, as witness tuberculosis and syphilis.

Analogy to Tuberculosis

Joint tuberculosis may be aptly quoted as an illustration of many points. We know that it is infectious, and in joints in which we find the characteristic tubercle we make our positive diagnosis, but the synovia or the bone marrow may be diseased alone, or both together, the bone may be rarefied or thickened, the cartilage may be normal or diseased, the synovia may be hypertrophied, or atrophied and fibrous, the joint may be shrunken or swollen, full of fibrous adhesions, or the seat of one of several kinds of exudate. The bones in the neighborhood may be lengthened or shortened, nervous and psychic symptoms may be present or absent. The patient may have fever, night sweats, hemorrhages, rapid pulse or he may not have them. If he have them we say that they are due to the same cause as the joint lesion. Before the discovery of the nature of the tubercle, all these manifestations must have been most puzzling. Indeed, the survival of such names as *"caries sicca"* and *"joint fungus"* gives us an inkling of the confusion which existed.

Finally let it be said that in many of these cases of chronic joint disease a distinct source of infection has been found, and in some the removal of this source of infection has been followed by an improvement or a cure of the joint disease. The

Sources of Infection

more careful the search for the infection, the more frequently will be found a diseased tonsil, a suppurating tooth socket, ethmoid cell, or ear, a gastro-intestinal infection, a history of an old, perhaps forgotten chancre. We have in the neighborhood of the joints two tissues that, according to all our knowledge, should be vulnerable to infection, the lymphoid synovia, and the red or lymphoid marrow. The importance of one of these two tissues, the synovia, has long been recognized in the chronic arthritides, the study of the marrow has been comparatively neglected. The marrow is the only tissue ever directly involved in those bone diseases whose cause we know and whose pathology we have worked out, e. g., acute infectious osteo-myelitis and tuberculosis, and if we would understand the bone changes in them we simply study the behavior of the diseased marrow. From the marrow the bone and cartilage draw their nutrition. All through the bibliography of chronic arthritis, one sees cursory allusions to the changes in the marrow, and, in many instances, statements that the nutrition of the cartilage and bone seem to be impaired.[1] The obvious corollary of this is to study

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[1] Hale White, Guy's Hosp. Reports, 1902. Autopsy on an acute case of rheumatoid arthritis.

"Foci of inflammation were met with in the bone, the cancellous tissue of which was more open than usual. Microscopical section showed that these foci were the seat of a small-celled infiltration, while the fat cells were markedly decreased in number."

"The bone may, however, show foci of inflammation, and in these areas there is comparative absence of fat cells."—McCrae.

"The medullary tissue is principally fatty," e. g., in the hand joints of a patient with so-called rh. arth.—Preble & Hektoen, *Am. Journal of the Med. Sciences*, January, 1901.

"The lesions have more resemblance to those caused by some neurotrophic disturbance or some failure to provide the proper constituents to develop bone and cartilage."—Painter, *Boston Med. & Surg. Jour.*, Nov. 28, 1901.

carefully the changes in the tissue from which the cartilage and bone draw nutrition.

If we discover swelling and disease of lymphoid tissue anywhere in the body, we do not attempt to understand it by examining the reaction of the tissues in its neighborhood but we subject the lymphoid tissue itself to as exhaustive an examination as possible.

As will be seen from what follows, we have attempted to do this in the joints, and, while our researches are by no means complete, and while as yet we have little absolute proof to offer, we have learned that the marrow is something besides a passive filling for the bones, and that it is much more profitable to study the *active* tissue of the bone than what may really be termed the *passive* tissue.

Nature of the Infection

In regard to the nature of the infection: I believe that any micro-organism capable of causing an acute arthritis can cause a chronic arthritis. Whether the organism itself must be in the joint or whether, located in some other organ, it can elaborate toxins which cause the arthritis, is not settled. Possibly the organisms may dwell in the marrow, with attenuated virulence. Among the causes, therefore, are the typhoid bacillus, the diplo-streptococcus of Poynton and Paine and others, the streptococcus and staphylococcus pyogenes, and the pneumococcus.

Other Causes

Various other factors have been linked up in a causal relation to chronic arthritis, namely, exposure to cold and wet, strain, wear and tear of laborious occupation, and mental emotions.

Trauma probably plays a role in the causation of this class of cases, though not so important as in

cases in Type II.[1] It is not to be viewed in the nature of a contributing cause, that is, as preparing a *locus minoris resistentiae,* but as a direct exciting cause, in a similar manner as in the acute cases. The trauma in chronic cases is oft repeated and usually slight. Sometimes it is in the nature of a chronic strain.

Trauma

A form of ankylosing arthritis occurs very rarely as the result of a single severe trauma. After the injury the joint slowly stiffens until complete bony ankylosis takes place. No fracture can be demonstrated by the most careful examination.[2] Treatment is without avail.

Traumatic Arthritis

If chronic arthritis is ever due to auto-intoxication from the intestinal canal, the influence of mental emotion would be quite comprehensible, as disturbing the digestion.

Concerning the other factors, authorities differ radically. It is hard to speak positively on the subject. Pregnancy, labor and the menopause have been said to predispose to joint involvement. In this connection the frequent occurence of pyorrhea alveolaris in pregnancy should be noted.

Familial Predisposition

Certain families seem more or less predisposed to these diseases. Proof here is also somewhat difficult, but the evidence seems to point toward the positive side. Possibly the joint tissues are predisposed congenitally to invasion, but more probably the tissues or organs into which the infection orig-

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[1] See a most interesting article by Axhausen, *Archiv für klinische Chirurgie,* September, 1912. Axhausen produced many of the characteristic lesions of chronic joint disease by destroying with an electric needle small areas of the joint cartilage.

(2) Murphy, *Journal of the American Medical Association,* April 27, 1912.

inally makes its way are congenitally less able to resist it.

Ductless Glands

The influence of the ductless glands is considered by some authorities to be quite important in the causation.

Visceroptosis

Goldthwait[1] has recently called attention to the connection which exists between certain of these joint lesions and visceroptosis. Here again we come back to the influence of disturbed digestion and the possible absorbtion of the toxic material.

Age of Onset

No age is exempt except the earliest infancy (when the bone ends are cartilaginous). According to McCrae the time of onset is most often in the third and fourth decades of life. The two types differ in this regard. Type II is wont to come on rather later in life than Type I. The occurrence of Still's disease during a period of oral sepsis is at least suggestive.

The weight of opinion seems to favor the view that most of the diseases in our causative category can cause joint lesions of both types. Thus, joint tuberculosis in its active stage, as we shall see, belongs in Type I, but the late manifestations are often characterized by the formation of new bone. The lesions of either type can probably be caused by a number of agents, and possibly each one of these agents can cause lesions of either or of both types.[2]

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[1] *Boston Medical and Surgical Journal*, May 26, 1910.

[1] Charcot held that rheumatoid arthritis and osteo-arthritis were but different expressions of one and the same underlying morbid process.

McCrae says the two types, atrophic and hypertrophic, occur in the same patient.

Goldthwait says that more than one type may be present in the same individual.

SIMPLE FORM.

Let us start with the simplest form. A patient presents himself, complaining of a pain in a joint, or possibly in a bursa. The pain came on suddenly or gradually, is constant, or present only on use, and is accompanied by few constitutional symptoms or by none at all. We find a localized point of tenderness, or sensitiveness of the entire synovia where it can be reached. Motion may be limited much or little.

These signs may clear up after a few days, they may persist, or they may disappear to return later with renewed force. If they clear up and do not return, we dismiss the case from our minds, or we consider that any therapeutic measures we adopted were efficacious, and possibly we recommend them to others.[1]

It is difficult to put such a mild form of arthritis in its proper place. If it disappear promptly, we place it in the category of acute, if it persist, then in that of chronic arthritis. It may be said to be on the border line.

Origin of These Cases

If the symptoms persist or return, we go minutely into the history, and find that the patient has a slightly enlarged tonsil, perhaps with a thickening of the lymph-nodes below it, or gives the history of a severe attack of dysentery or gonorrhea, or of a suppurating tooth cavity or accessory sinus of the nose. We find further that no matter what local measures we adopt, no permanent benefit will ensue until we have removed the tonsil, have

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[1] McCrae says that many cases of chronic joint disease recover in the early stages.

washed out the colon, or have cleaned out the suppurating cavity or genito-urinary tract. Then at once the joint trouble disappears.

Here we have an inflammation of the synovia. Any changes that may have existed in the bone are so slight as to escape notice. The disease may possibly start in the marrow and spread to the synovia, or *vice versa.* Again either may be affected alone and show no tendency to involve the other.

Between this slight synovial involvement and the severest cases of chronic arthritis of this group the difference is merely one of degree, as the difference between a mild synovial tuberculosis and a destructive joint tuberculosis is merely one of degree. If, in such an arthritis the infection continue to act or if it be very potent, we shall see how the damage may involve many tissues, and even may involve many joints, at once or *seriatim,* but, on the hypothesis laid down, all the pathological changes can be readily undersood, while, if we regard each change as in itself of importance, and as representing a different clinical entity, we shall soon be at sea in a rising tide of divisions, sub-divisions and classifications. This way of regarding the matter is, of course, pure theory, but on this theory we can trace the disease in the joint tissues, and from what we know of joint pathology we can ascertain that many of the changes are what we should expect.

On this theory we are not necessarily compelled to admit that an acute disease can actually pass into a chronic one, but we simply point again to the analogy of the lymph-nodes. These may swell up and subside, possibly may swell up again repeatedly, or may swell up acutely or slowly and remain

swollen, depending on the virulence of the poison, or upon the amount and constancy of supply. We never think of denying the infectious nature of their involvement because this involvement is slow and chronic.

RUDIMENTARY FORM.

CASES CHARACTERIZED BY INFLAMMATION, OR PROLIFERATION, OF THE SYNOVIA—

SIMPLE SYNOVITIS.

Nature of These Cases

In this form one or two joints swell and become painful, either at the same time or one after another. The pain may be severe or slight, fluid may be present or absent. Sometimes the entire synovia is sensitive to pressure, at other times the sensitiveness will be centered at one spot. The joint may creak. Motion, while painful, is limited but little, if at all, and then usually by the mechanical obstruction caused by the fluid in the joint. There may be a slight rise in temperature at the beginning, but fever is not a characteristic of the process. Constitutional symptoms are present or absent according to the severity and the nature of the infection.

Under this head would come cases of traumatic or non-traumatic simple synovitis, intermittent hydrops,[1] etc. The process often involves the knees and no other joints.

The bone, the cartilage, and the fibrous structures about the joint show no gross tendency to in-

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[1] See an interesting article by Klemm, *Arch. f. klin. Chirurgie*, February, 1912.
The author believes that many cases of intermittent hydrops are due to a chronic infectious (pus) osteomyelitis in the end of the bone.

volvement. The disease shows no tendency to spread through the other joints. It involves one or two, and there it stops. It may clear up at any time and leave no trace, or it may recur or persist.

These are just as much cases of "arthritis deformans" as are the cases with extensive destruction of the bone and cartilage. The infectious agent here is feeble in its manifestation, and the disease expends its energy on the synovia.

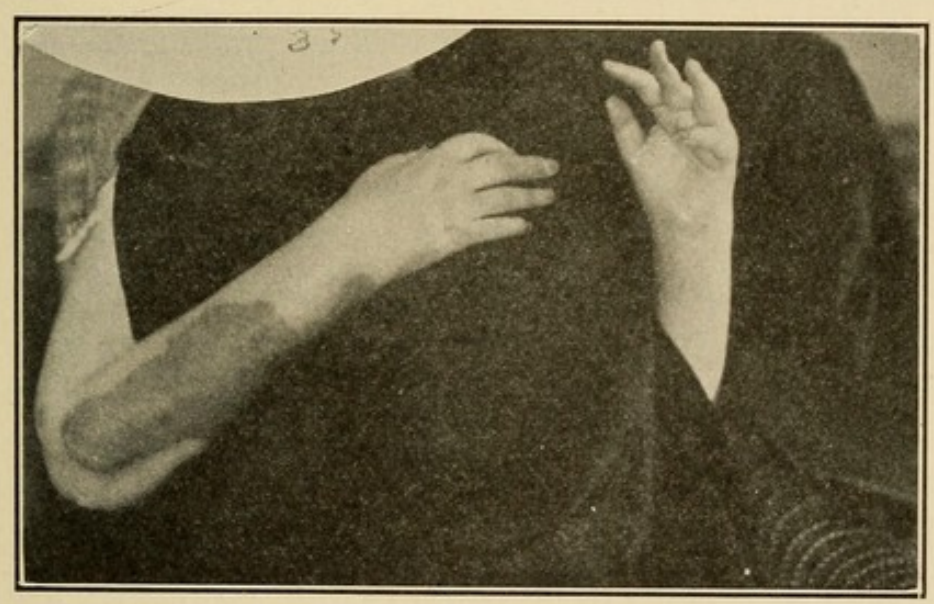

Fig. 49.

The multiarticular forms of Type I in the fingers. Note the swelling and the flexions (Goldthwait).

The diagnosis is to be made with great care, not in the haste of a five-minute office examination. In all these chronic arthritides our only hope of success is in thoroughness and in taking pains. The patient's family history and his own history from childhood must be known, his occupation, his habits, the dates of every infectious disease, and especially of venereal infection or typhoid fever, the possible sequence of his symptoms upon any of them, the

Necessity for care in the Diagnosis

exact course and chronology of his present disease, and all its subjective symptoms. Has he had a purulent otitis, or any symptoms in nose, throat, tonsils, gastro-intestinal canal, appendix, gall-bladder, or genito-urinary tract? Nothing is too insignificant to mention. A history of recurring trauma should also be sought.

All these facts should be noted down, before proceeding to the physical examination. The examination is to be as thorough as we can make it. One searches for every possible source of infection, and, if one lacks the knowledge to conduct any particular line of investigation, one calls in a colleague to conduct it. Every organ is interrogated.

Attention is last of all turned to the joint or joints. Keeping in mind the pathological lesions of this type, we look for their clinical manifestations.

With these data before us we are often in a position to make a diagnosis, though a day or two for their study is always desirable. A show of brilliance and diagnostic fireworks is quite out of place here. If we be still in doubt, a Wasserman or tuberculin test may occasionally be of assistance. Skiagraphy is of the greatest use to detect not only local lesions of the joints, but also misplacements of the viscera. Naturally a skiagram of a slight, purely synovial inflammation will be mainly useful as excluding cartilage and bone involvement.

DIFFERENTIAL DIAGNOSIS.

The lesions from which the disease with these manifestations must be differentiated are synovial tuberculosis, gonorrheal synovitis and the synovitis that occurs as a manifestation of tertiary syphilis.

Synovial Tuberculosis

From synovial tuberculosis the disease often cannot be differentiated without a microscopic examination of a piece of the synovia. The tuberculin tests are merely suggestive. An injection of fluid, aspirated from the joint, into a guinea pig's abdomen may clinch the diagnosis. Failure of these measures makes it advisable to view every slow case of uniarticular synovitis as possible tuberculosis.

Traumatic Arthritis

Acute traumatic synovitis follows immediately on an injury, and usually subsides on rest and protection. Often one may detect evidences of a torn ligament or of a displaced fibro-cartilage.

Syphilitic Arthritis

A syphilitic synovitis presents no difference from cases in this group. As it is, having determined that the disease is syphilitic, by the history and the concomitant phenomena, we immediately transfer it to the category of syphilitic joints, and treat it accordingly. The diagnosis is made in the well-known manner. Much the same may be said of a *gonorrheal joint.*

TREATMENT.

Remove the Cause

The treatment is, first of all, to remove the cause, if we can. Usually then the trouble will disappear of itself. In the meantime rest is indicated in the acute stage. The diseased appendix, gall-bladder, or tonsil must be removed. The ear, the nose, the intestinal or genito-urinary tract must receive the appropriate treatment. A syphilitic synovitis usually yields promptly to mercury or to Salvarsan. The iodides are not of so great value in joint disease. Traumatic synovitis demands the removal of the cause of the mechanical injury.

If symptoms persist the joint may be strapped with adhesive tape, it may be baked or massaged. Hydrotherapy or vibratory massage will often be found useful. The efficacy of any form of ultra-rays is doubtful. The salicylates often will relieve the pain temporarily, but they are not curative. Those cases dependent upon absorbtion of intestinal poisons will be benefitted by purging; hence the efficacy of many Spas.

If gross misplacements of the abdominal organs be present, they should be corrected either by an operation or more frequently by the use of apparatus and exercises to support the belly wall. The usual form of apparatus is a light spinal brace, with an apron over the lower abdomen. Goldthwait puts his severe cases to bed for a while on a hard mattress, with their shoulders low and a firm cushion in the back of their middle thoracic spine, with the idea of expanding their chests, pulling up the diaphragm, and thus making room for the displaced abdominal organs. His results seem excellent.

SEVERE OR MULTIARTICULAR GROUP.

This group is essentially multiarticular and progressive, involving several joints in succession. It occurs almost certainly as a late syphilitic manifestation.[1] The view has been advanced that a focus of infection exists somewhere in the body in every case—in the gall-bladder, the tonsil, the appendix, the genito-urinary tract, etc.—and that just as the gonococcus can cause an acute arthritis, and, when domiciled in the original host, a chronic arthritis, so can the typhoid bacillus, the pneumococcus,

Causative Agents

[1] *Colorado Medicine*, August, 1911.

the streptococcus, the staphylococcus, and possibly others. This view is largely theoretical, but evidence is constantly accumulating to support it.

An increased excretion of calcium salts has been detected in these cases. This is what one would expect when the bone is atrophying.

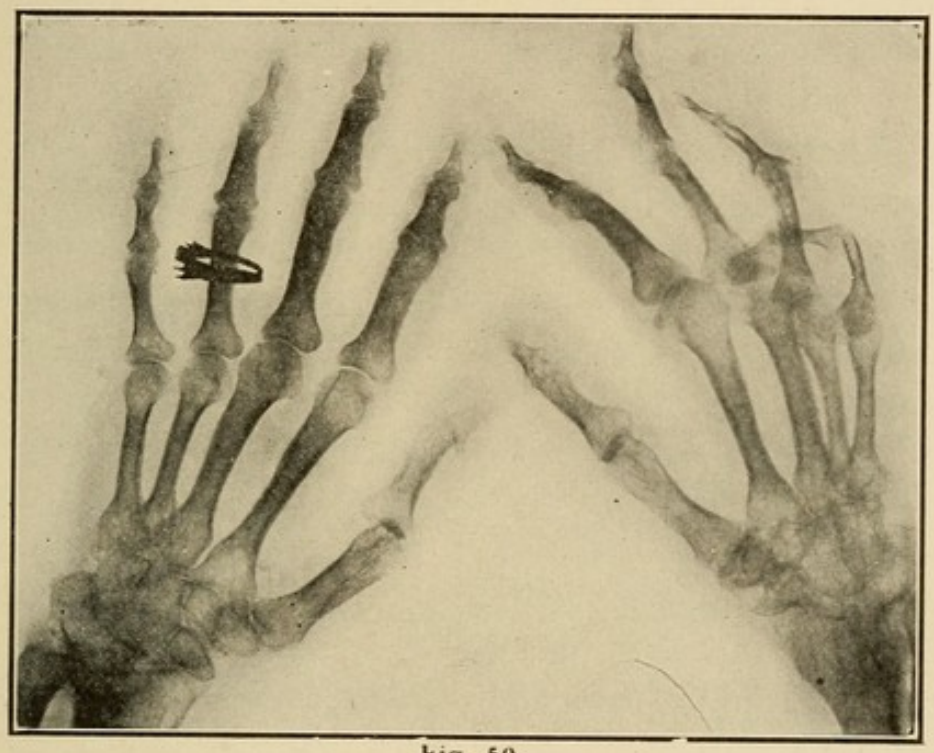

Fig. 50.

Skiagram of the multiarticular form of Type I. Note the flexions and the superextensions of the phalanges, the rarefactions and the destruction of the bone ends, and the bony ankyloses. Note also the involvement of the wrist bones and of the radii (Goldthwait).

PATHOLOGY.

Essential Features of the Pathology

The essential feature of this type of arthritis is a proliferation of the synovia, and to this in most instances is added a similar process in the lymphoid marrow.[2] The report of microscopic examina-

[2] W. Hale White, Guy's Hospital Reports, 1902. On the Pathology of Acute Rheumatoid Arthritis.

McCrae says that the first structure to show a change is the synovia, and that this change consists of proliferation. After this the cartilage is sometimes attacked—first eroded by the action of the synovia.

Nathan maintains that the changes in the bone can be perceived by the Röntgen ray before the symptoms are present in the joint.

tions of these joints in the early stages are unfortunately rare, and it is only from these reports that we can judge of the nature of the disease.

The synovia proliferates and is thickened, inflamed and thrown into folds. The bone marrow is in some instances at least, the seat of inflammatory changes. Hale White found foci of recent inflammation in the marrow, as shown by round cell infiltration.

Similarity to Tuberculosis

These are exactly the changes that are early apparent in joint tuberculosis,[1] and consequently one is not surprised by the statement that not only are the subsequent changes in the bone and cartilage much the same as those seen in the latter disease, but also that neither by the symptoms, nor by the physical signs can the two diseases be differentiated. If a single joint only were inspected, one could not make a diagnosis between the two diseases, even if the joint were opened for gross inspection. It is only by the demonstration of the tubercle, by its tendency to necrosis and to secondary infection, and by its uniarticular nature that a tuberculous joint can be recognized. In other words we cease to look hereafter for any peculiar effect of toxines or faulty metabolism upon the bone and cartilage, but knowing the sequence in tuberculosis, and identifying the changes in the synovia and in the marrow in this type of chronic arthritis, we can predict fairly well what will happen to the other tissues—the stroma, so to speak.

We shall not go here into the minutiae of all the changes in the various tissues. They have been set

(1) In Hale White's article appears an illustration of an enlargement of a phalangeal joint that lacks only the presence of tubercles to be characteristic of joint tuberculosis.

forth fully by other writers,[1] but we may well devote a few words to some of the essential features of the process.

The proliferation of the synovia may be extreme, and may result in the formation of tags, whose structure varies. The ligaments are usually thickened by increase of their connective tissue. Later in the disease the synovia may atrophy.

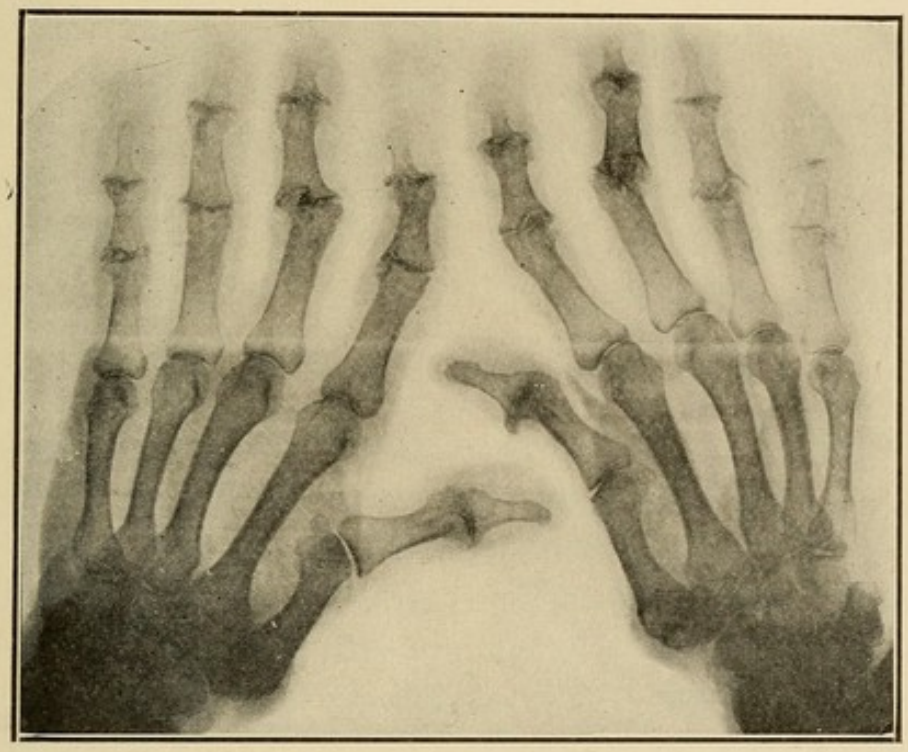

Fig. 51.

Skiagram of a case of multiple arthritis of Type II. All the interphalangeal joints are involved. Note the bony outgrowths and the irreguar line of the joints.

By reason of the lack of mobility in the joint and of the proliferation of the synovia, or of the marrow, or of both, the cartilage usually becomes fibrillated, and, suffering in its nutrition, degenerates.

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[1] Vide Nichols and Richardson. *Journal of Medical Research,* 1909, XXI, 149. Nathan, *American Journal Medical Sciences,* 1909, CXXXVI, p. 817.

Erosions appear on its surface. The inflammatory tissue from the marrow may break through the cartilage into the joint. The joint cartilage in this way may be destroyed entirely, or, degenerating in its upper and lower portions, may remain as a thin strip in the midst of fibrous tissue. Fibrous adhesions may form between the degenerated cartilage and its fellow of the other bone entering into the articulation, or between it and the synovia, or between both. Finally, in some cases a bony ankylosis may take place. Nichols and Richardson are authority for the significant statement that a subsidence of symptoms ensues on a bony ankylosis. They also speak of the "proliferation of the destroying connective tissue of the marrow."

CHANGES IN THE BONE AND IN THE MARROW.

Changes in the Marrow

"The change" (in the marrow) "frequently extends beyond the layer just beneath the joint cartilage and sometimes includes the marrow of the entire epiphysis, especially in the more rapidly progressing cases. The change consists chiefly of a proliferation of the connective tissue of the marrow, sometimes associated with a proliferation of the osteoblasts. The degree of proliferation of connective tissue varies a great deal. In some instances it is very marked, so that all evidence of normal marrow disappears, and the marrow spaces are filled sometimes with dense nonvascular fibrous tissue, in most cases with a very fibrous, loose-meshed, edematous connective tissue, which may or may not show infiltration with lymphoid and plasma cells."[1]

As opposed to these statements concerning the

[1] Nichols and Richardson.

structure of the bone, Hale White says: "The cancellous tissue is more open than usual." Nathan[2] says: "One finds the marrow more red and the trabeculae of the spongiosa diminished in number and thinner than normal. During the early stages the bone atrophy is distinctly subchondral, but it gradually advances and finally involves the whole epiphysis. In the majority of cases at an early stage one can discover with the naked eye, or at any rate with a low power magnifying glass, minute cavities situated in the subchondral region of the epiphysis within, or at the edges of the bone trabeculae. These are bone cysts[1] and they vary in size, being of course larger and more numerous as the disease advances."

This author regards the changes in the synovia as insignificant in comparison with those in the bone, a view diametrically opposite to that of other writers.

The lesions in this type of joint disease, then, may be summed up as a proliferation of the synovia and of the bone marrow, an atrophy of the bone (either a rarefying osteitis, or a resorption of calcium salts) and erosion and destruction of the cartilage. These result in subluxations, distortions, and in fibrous and bony ankylosis.

SYMPTOMATOLOGY.

Cases belonging to this type may begin suddenly or insidiously. Sometimes the onset is preceded by various local nervous and circulatory disturbances, —tingling, numbness, muscular cramps, stiffness,

Onset

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[2] *American Journal of the Medical Sciences,* Vol. 137, p. 835.
(1) Bone cysts occur also in Type 2.

weakness, hyperaesthesia, sweating, change in color, etc.—at other times its approach is unheralded. One joint after another may be attacked in rapid succession, or the disease may take years for its full development. The history of the disease in any given case may be a series of exacerbations and remissions. Fever and other constitutional symptoms may or may not accompany the invasion of the various joints. A persistently rapid pulse is characteristic of some cases. The lymph nodes are often involved, especially in children—Still's disease. This swelling of the lymph nodes is a strong argument in favor of the infectious origin. The spleen also may be enlarged.

Sequence of Joint Involvement

The disease usually attacks first the joints of the fingers, and travels toward the trunk—*centripetally*. Its general symmetrical tendency is more or less characteristic. On the other hand one of the larger joints may be attacked first, and the other joints throughout the body without any regular succession, but sooner or later the symmetry will be apparent. With the involvement of fresh joints, those previously involved may be the seat of an acute exacerbation. The spinal articulations may take part, or, they may be exempt. The hips often escape, though a special form of the disease affecting only the hips and spine is occasionally encountered.

Deformity and Cause

The tendency to deformity in the affected joints is also progressive, and with the successive involvement of the various articulations the patient becomes bed-ridden, and a hopeless cripple. Bed sores then form a serious and often dangerous complication. The patient may finally succumb to some intercurrent disease, to which the malnutrition,

often present, renders him an easy prey. Again, during the intermissions the patient's nutrition may be good, and constitutional involvement may be absent. If the thoracic spine suffer, death may be due to pneumonia or to pulmonary tuberculosis. Muscular cramps are especially frequent in cases with spinal involvement.

Nephritis, general arterial sclerosis, and gastro-intestinal diseases frequently accompany this form of arthritis, according to some authors; disease of the thyroid, and paralysis agitans more rarely. Psoriasis and eczema also are troublesome complications. Accompanying cardiac lesions are comparatively rare.

Accompanying Lesions

The skin is often dry and harsh, or glazed, and the subcutaneous tissues of the extremities may be thickened. Pigmented cutaneous areas are frequently present. The nails may be cracked and fissured.

The anaemia which so often accompanies this form of the disease is what might be expected in view of the blood-forming function of the lymphoid marrow. Some observers have noted a fall in the number of red blood cells, but as a rule the number is about normal. Leucocytosis may or may not be present. It may be said that the blood changes are not characteristic.

Anaemia

The reflexes are wont to be exaggerated, especially during the acute exacerbations.

The disease may progress to a fatal termination, as indicated above, or it may be arrested, and the patient may recover with a fair amount of motion in the affected joints. It may recur after a long period of quiescence.

Outcome

LOCAL SYMPTOMS AND PHYSICAL SIGNS.

The disease usually begins with stiffness, pain and swelling. The swelling is soft and often fluctuating, and this fluctuation may be caused by thickening of the synovia, or by fluid, or by both. The affected joints take on a fusiform appearance, and from the start are wont to be in flexion. The proximal interphalangeal, or metacarpo-phalangeal joints are often the first to be affected. Later on in the disease the swelling sometimes disappears and leaves the joint shrunken, often with a distinct constriction at the articular line. The constriction is due to the secondary shrinking of the synovia, and to the atrophy of the articular cartilage.

Motion is restricted almost from the start. In the late stages bony ankylosis sometimes takes place.

Flexions and subluxations of the diseased joints are often present, and may persist even after the disease has run its course. Frequently the contractures are most troublesome. The skin over the joint is often a pale, waxy color; again, it may be reddened. The Roentgen rays show, as one would expect, a thickened synovial capsule, a thinning and erosion of the cartilage, and an atrophy of the bone. These are much the same changes as those in a tuberculous joint, but tuberculosis is distinctly uniarticular. In the later stages the distortions are easily made out on the plate, but the thickened synovia will have disappeared.

Roentgen Picture

STILL'S DISEASE.

Still has described a special form of this disease,[1] occurring in children, before the second den-

[1] Allbutt's System of Medicine, 1901, Vol. 3.
See also Arthritis Deformans, Llewelyn Jones. Wm. Wood & Co., 1909.

tition, and accompanied by enlargement of the lymph nodes and of the spleen. The pathological process expends itself upon the synovia, and, according to Still, shows no tendency to involve the other tissues of the joint.

Other authors have contributed histories of cases since Still's original monograph, but some confusion still exists as to whether the disease represents a distinct clinical entity, or whether marked general lymphoid involvement is due to the greater tendency in children to disease of the lymph nodes. For the present it seems wiser to include it in the same category with similar disease in adults. None of Still's cases recovered. Subsequent writers have reported recoveries. The prognosis in children is now fairly good and even where considerable fixation of the joints accompanied by deformity has occurred there is good prospect of fair function.

All Still's Cases Were Fatal

DIAGNOSIS.

The diagnosis should occasion very little difficulty, for all these cases are peculiar in their progressive and multiarticular nature.

The *bony nodules* that occur in Type II. occur more often in the terminal interphalangeal joints, and can be distinguished by palpation; the deformity is often partly a lateral one. The X-ray in that type shows an outgrowth of bone, an exostosis, which seldom occurs in Type I. The obstruction to motion is mechanical only, and is due to the bony outgrowths and to the irregularity of the joint line. Bony ankylosis never takes place.

Tuberculosis is distinctively uniarticular, and presents little difficulty in diagnosis. The secondary

infections and abscesses, so frequent in tuberculosis, are never present in this group.

Gout is slower in its progress, and usually expends its energies upon one or two joints. Its chalky deposits are characteristic.

Acute articular rheumatism (inflammatory) might be confused at the outset with the cases of this type which begin acutely, but acute inflammatory rheumatism is essentially a more fleeting disease, usually leaves one joint before going to another, leaving no local damage behind. According to Poynton and Paine, however, the infection which is the cause of acute inflammatory rheumatism is quite capable of causing a chronic arthritis, and often does so. Endocarditis and pericarditis are frequent complications of acute inflammatory rheumatism.

PROGNOSIS.

Until recently the prognosis of cases in this group has been almost universally bad. Little could be done to arrest the onward march of the disease, and, except in the instances of spontaneous recovery, those afflicted with it could look forward only to a life of hopeless invalidism and suffering. As the result of recent discoveries the prognosis is now not so bad. Sometimes we can arrest the disease, and often can restore the patients to a state approximating health. In other cases it must be confessed, our efforts will probably be futile. The prognosis in any individual case will often depend upon the amount of care and work the medical advisor is willing to expend, as well as upon the persistence and coöperation of the patient and of his friends.

As in the preceding class, so in this, the treatment first of all consists in removing the cause if we can find it, and we refer the reader to what has already been said on that subject. Treatment

If, after careful study, we are unable to find the cause, we must needs fall back upon empiricism. Our first thought is of syphilis without other manifestations. We therefore will do well to carry through a vigorous course of anti-syphilitic treatment for at least six weeks. The iodides are of little use here. Mercury must be our main reliance, either in the form of intramuscular injections[1] or of inunctions.[2] Salvarsan should also be administered.

Failing in our antisyphilitic treatment, we may administer empirically extract of thymus gland, after the manner of Nathan. The powdered extract should be administered in doses of ten grains three times a day. Thymus Gland

If we fail here, we may possibly have better success with thyroid extract or with the extract of the supra-renals, or even with some preparation of milk that has undergone lactic acid fermentation.[3] Thyroid Extract

CONSTITUTIONAL TREATMENT.

During the acute periods, if high temperature and severe constitutional symptoms be present, we may possibly think well of a restricted diet, as in other febrile affections. Some writers maintain that it is

[1] A convenient form is salicylate of mercury 10 per cent in liquid abolene. This should be injected deep into the muscles, preferably the gluteal, under strict asepsis, at intervals of one week, first in one side and then in the other. The initial dose should be about 10 minims of the mixture, and this should later be increased to about 25 minims.

[2] Fifty per cent mercurial ointment.

[3] Andrews and Hoke, *American Journal of Orthopaedic Surgery*, July, 1907.

a mistake to restrict the diet of these patients *unless their gastro-intestinal apparatus be at fault.* They say that we should rather strive to keep their nutrition at a high point. The bowels should be kept open. A mild dry climate is considered of great benefit, but I doubt its efficacy.

LOCAL TREATMENT.

During the acute stages of the disease and during the exacerbations, the first indication is usually rest. This may be furnished by ordinary rest in bed, or, in addition, we may think it advisable to apply some sort of a splint to the more painful joints. It is most important to keep the affected joints in such a position that, if ankylosis result, they will be in the best attitude for function: Thus, the knees should be kept in complete extension, the elbows and ankles at a right-angled flexion.

The application of heat or of cold may also serve to allay the pain. The salicylates here often have a distinct effect, palliative but not curative.

During the remissions the patients may be permitted to go about, in fact they should be encouraged to do so. Exercise in moderation in the open air helps to keep up their nutrition. If necessary, the affected joints may be supported by some form of brace, which not only protects them from injury, but also permits motion through a limited arc. Passive motion to the limit of toleration often serves to retain what motion is possible. Massage and exercises carried out by the Zander apparatus have their advocates. High frequency currents have been praised.

The Bier treatment by passive congestion has

been urged, not only for the relief of pain but also to encourage motion, but this treatment has been advocated for so many different diseases, to meet so many different indications, that one hesitates to judge as to what its exact effects may be. It certainly seems to quiet pain in some cases, but whether this is due to the congestion or to pressure upon nerve trunks it is hard to say.

Operative treatment is rarely indicated, except possibly in those joints, such as the knee, where the disease has run its course, and has left behind it hypertrophied fringes which mechanically interfere with function. In these cases one may open the joint and trim away the fringes.

CHAPTER V.

TYPE II.

Cases Characterized by Inflammation and Degeneration of the Synovia, and Degeneration of the Marrow, and by Resulting Hypertrophy of Bone and Cartilage.

Under this heading are grouped cases heretofore described as osteo-arthritis, the hypertrophic form of Goldthwait, and the degenerative form of Nichols and Richardson. It includes such lesions also as Heberden's nodes, *morbus coxae senilis,* etc.

ETIOLOGY.

Concerning the etiology of this type we know nothing definite. We may believe that after the manner of all joint cases whose nature we understand, some of these cases are also infectious in their origin. We may believe that they are dependent upon a lesion of some trophic centre in the cord, which, after many years of search, has never been located, we may dismiss them as "metabolic," which again is simply to confess ignorance. We do know, however, that the changes found in this type are often simply exaggerations of those ordinarily taking place in the joints as age advances. Some writers maintain that the two types represent stages in the same process. If the two types ever are due to the same causes, the intensity of the morbid process in the marrow is more probably the reason of the difference in the secondary changes.

Numerous observers have noted that patients with this type of arthritis often suffer with flatulence and intestinal indigestion. This again would point to an infectious origin. Sometimes the onset of the disease is said to follow a "dysentery," which has been suddenly checked.

Osler affirms that Heberden's nodes are an evidence of longevity.

Trauma

The influence of trauma is probably greater in this type than in Type I. An internal derangement of a joint, such as a displaced semilunar cartilage, an intra-articular fracture healed with marked deformity, may occasion an arthritis that will persist until the cause is removed, and according to the views held by certain writers, even then will leave behind it the lesions of a chronic arthritis. Axhausen produced many of the characteristic lesions of this type of joint disease by puncturing the joint cartilages of animals with an electric needle.[1] A severe single trauma often appears clinically to be the starting point of the disease especially in the hip. Axhausen's experiments show well the manner in which the trauma acts. Again, persistent recurring traumata, received in certain occupations cause many of the cases. In this class would come the stiffened spines found in persons pursuing laborious occupations, carrying heavy weights, or doing heavy work in a bent attitude. According to Arbuthnot-Lane[2] and others the changes in the joints of such persons are the result of Nature's efforts to relieve the muscular strain. Lane quotes the coal-trimmer's elbow, often seen in England, as another example.

[1] Axhausen, *Archiv für klinische Chirurgie,* September, 1912.
[2] Lane, *Lancet-Clinic,* Cincinnati: 1913. CIX, 32.

PATHOLOGY.

Chief Morbid Changes

Roughly speaking, the changes observed in this type are just the reverse of those noted in Type I. As the result of well marked degenerative processes in the bone marrow, in the deeper layer of the periosteum and in the synovia, the bone and cartilage hypertrophy. Hence, instead of the synovia and marrow encroaching upon the bone and the cartilage, the cartilage and bone may be regarded as encroaching upon the bone marrow and on the synovia. This is the same process that is often observed in other organs of the body. As their essential elements, their parenchyma, degenerate, the connective tissue stroma proliferates, so that the degeneration and proliferation go hand in hand.

The following hypothesis to account for the joint changes is put forward tentatively:

The articular cartilage proliferates throughout its entire extent, and masses of new bone and cartilage are formed at its periphery. At the same time there is a production of new cartilage and bone underneath the articular cartilage, diminishing the space for the marrow. The articular cartilage derives its nutrition from the subjacent marrow, and when this marrow is replaced by bone, the cartilage fibrillates, degenerates, and wears away, leaving the thickened bone bare, and grooved in the line of joint motion. The cartilage near the periphery, however, deriving its nutrition largely from the synovia and from the periosteum, does not undergo so soon this degeneration and wearing away, but, maintaining its irregularity, hypertrophies, and causes the joint to become flattened and distorted. The hypertrophied masses of cartilage may persist as such, or they may

be transformed into bone, causing spurs and exostoses, which interfere with motion. In ball-and-socket joints, the new bone formations render the head of the bone too large for its socket, and subluxations may result. In the hinge joints, the irregularity of the bone formation causes lateral distortions as well as flexions. These lateral distor-

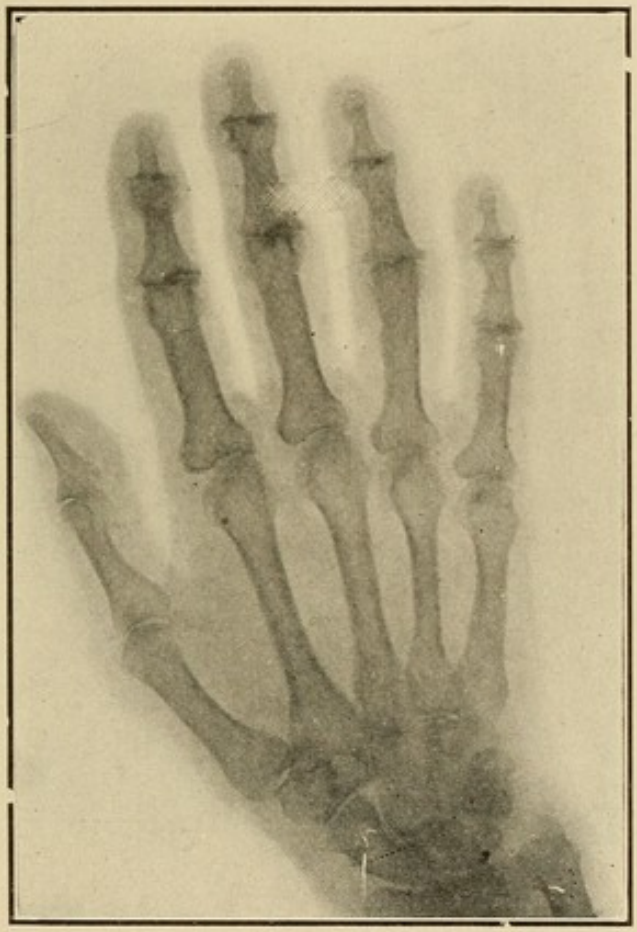

Fig. 52.
Skiagram of multiarticular lesions of Type II (Goldthwait).

tions are characteristic of the disease. The nodular bone formations in the terminal phalangeal joints were described many years ago by Heberden, and have received the name of "Heberden's Nodes."

Heberden's Nodes

Portions of the proliferated cartilage may be torn loose and may form foreign bodies in the joint.

The synovia in this type may appear comparatively normal, or it may proliferate in spots. According to Nichols and Richardson the localised proliferation may result in the formation of papillary masses, composed of granulation or of dense

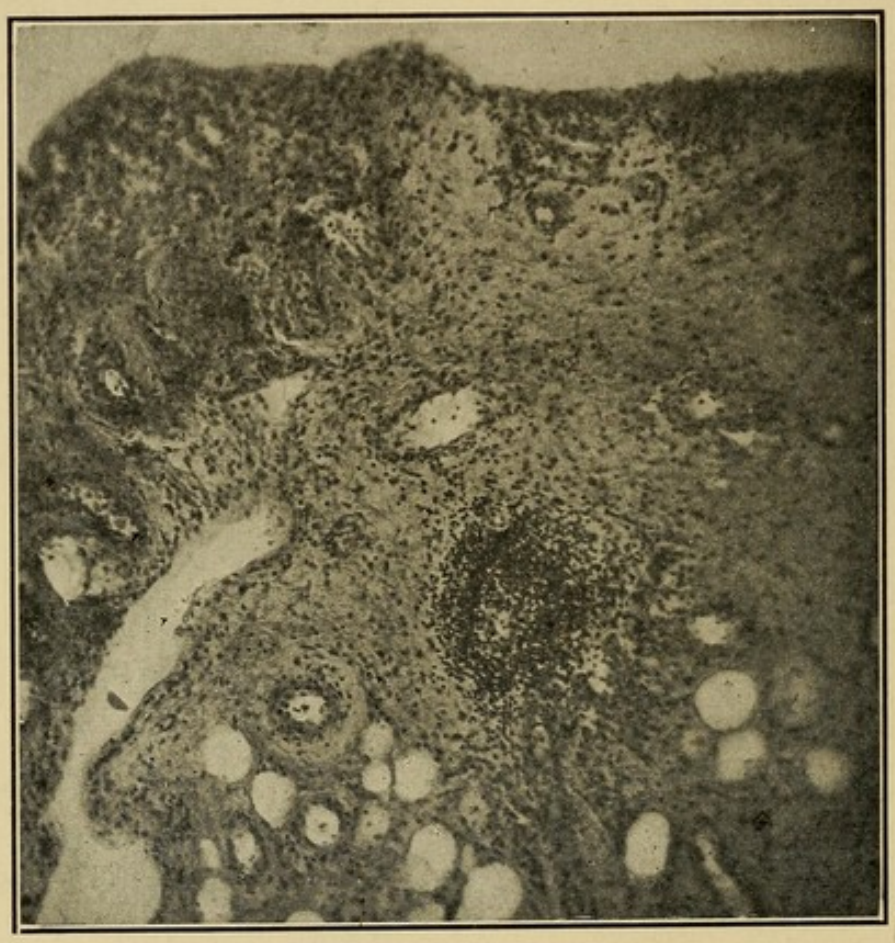

Fig. 53.

Photomicrograph of synovia from a case of arthritis of the hip in an adult male, Type II. Note the collection of round cells about the blood vessel, and the fibrous structure of the membrane. Zeiss apochromatic 16 mm. objective.

connective tissue. These masses also may be torn loose later and form "joint mice."

Adhesions never form between the opposing joint surfaces, and bony or fibrous union never takes place. We have seen in Type I. that the adhesions between the bone ends are due to the pro-

liferation of the synovia and of the marrow through the gaps in the cartilage, but in this type the proliferation, if it exists at all, is not marked. The obstruction to motion is purely mechanical. It will be remembered that in old fractures, where eburmation of the bone ends has taken place, union is impossible.

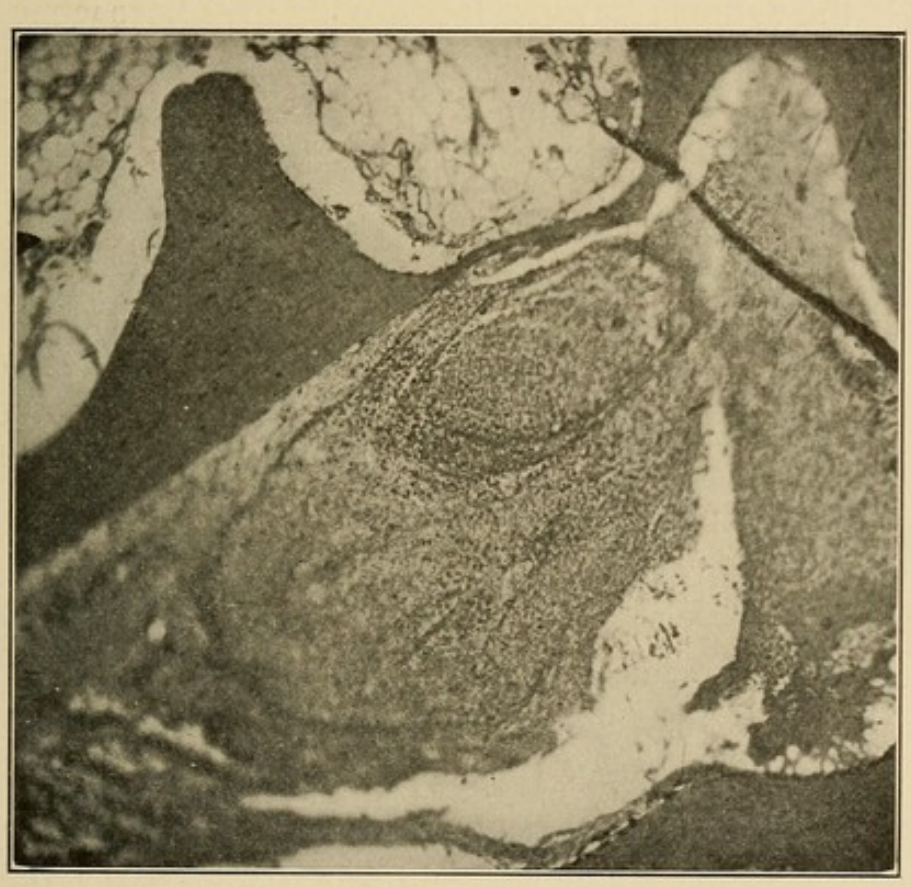

Fig. 54.

Type II. Photomicrograph of an inflammatory focus in the marrow. Same case as the preceding. Note the productive osteitis of the trabeculae. Zeiss 16 mm. objective.

HISTOLOGICAL PATHOLOGY.

The changes in the bone and cartilage have hitherto been regarded as the essential lesions of this disease, and to the naked eye they appear to be most important, but their study has been almost

barren of results. If we examine carefully the behavior of the bone marrow from which these tissues draw their nutrition, and of the synovia, we shall immediately detect significant alterations in structure, and possibly in the end we may come to regard the bone and cartilage changes as merely the results of these alterations.

If the synovia be examined under the microscope, areas of round cell infiltrations will be seen, especially in the neighborhood of the blood vessels. The arteries are often thickened and the veins widened. On the surface of the membrane there may be a slight increase of the lymphoid elements, but the membrane itself, while often thickened, can not be said to be proliferated, but rather degenerated. One sees areas of fat cells scattered through it and much new fibrous tissue. The pathological process in the synovia may be interpreted as localised inflammation followed by degeneration. Possibly the signs of inflammation may disappear in the late stages of the disease, but they are certainly present in the active period.

Localized Inflammation of the Synovia Characteristic

The Marrow. Of the changes one meets in the marrow it is hard to say which are characteristic. Perhaps a fatty change is met most frequently. The normal marrow may be almost replaced by new fibrous tissues in whose meshes the marrow cells may be distinguished. Again, the marrow is packed with cells; in spots it appears practically normal. The marrow spaces are less capacious than normal, on account of the production of new bone. Islands of cartilage in the marrow probably represent the early stages of new bone trabeculae cut across. Evidences of a productive osteitis may be seen espe-

Marrow Changes

cially in those areas where marrow changes are prominent. Cysts are sometimes present, and apparently, when situated immediately beneath the cartilage may rupture and perforate it. The arteries show thickened walls.

New Production of Bone

The bone is decidedly increased in amount. The trabeculae are thicker than normal, and in larger

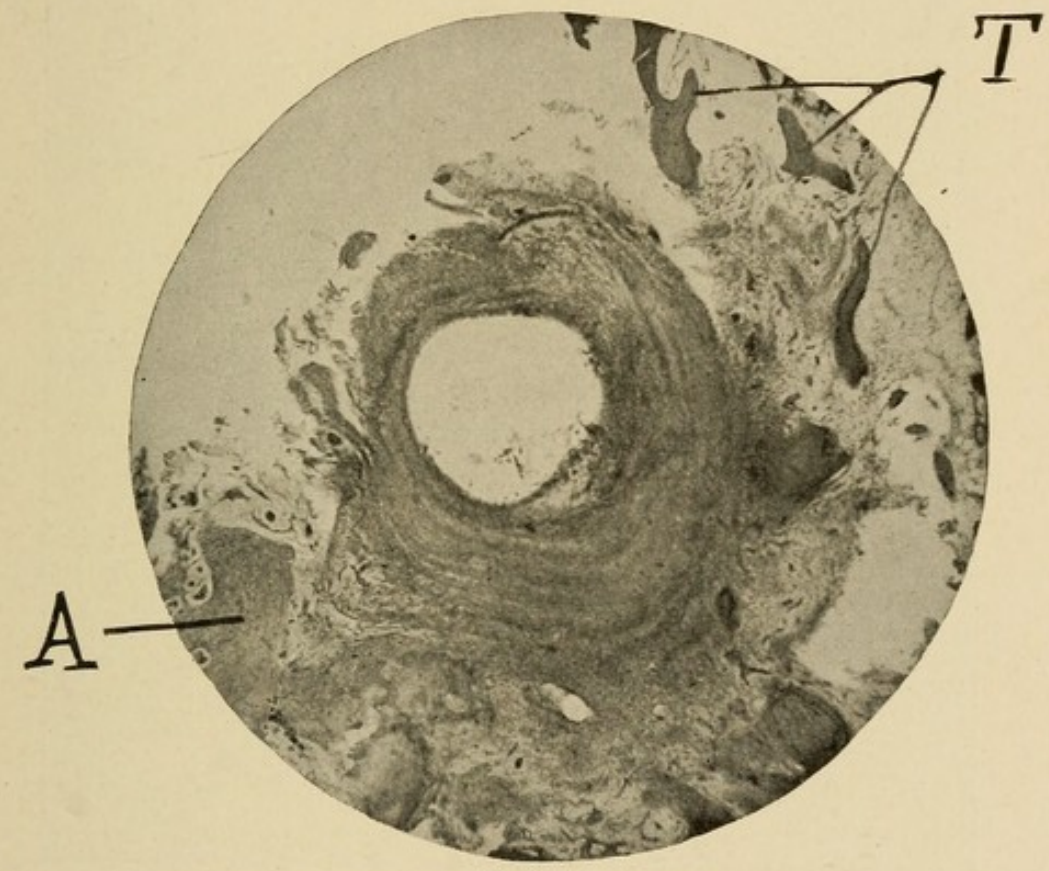

Fig. 55.

Photomicrograph of a bone cyst from a case of arthritis of the hip—Type II. The patient was a young woman. This cyst was located directly beneath the level of the articular cartilage of the femur, which had been perforated at that spot. T—trabeculae; A—cartilage. Zeiss objective aa, no ocular.

numbers. Some of them are composed partially of osteoid tissue. The production of new bone dominates the picture.

The cartilage is thickened and in various stages of degeneration. Its appearance is often bizarre

in the extreme. Almost always its structure is more or less fibrillated and broken up. Its surface may be roughened. A thin layer of fibrillated cartilage may be superposed on a thick layer of osteoid tissue containing clusters of large cartilage cells staining deeply with haematoxylin. Granular ma-

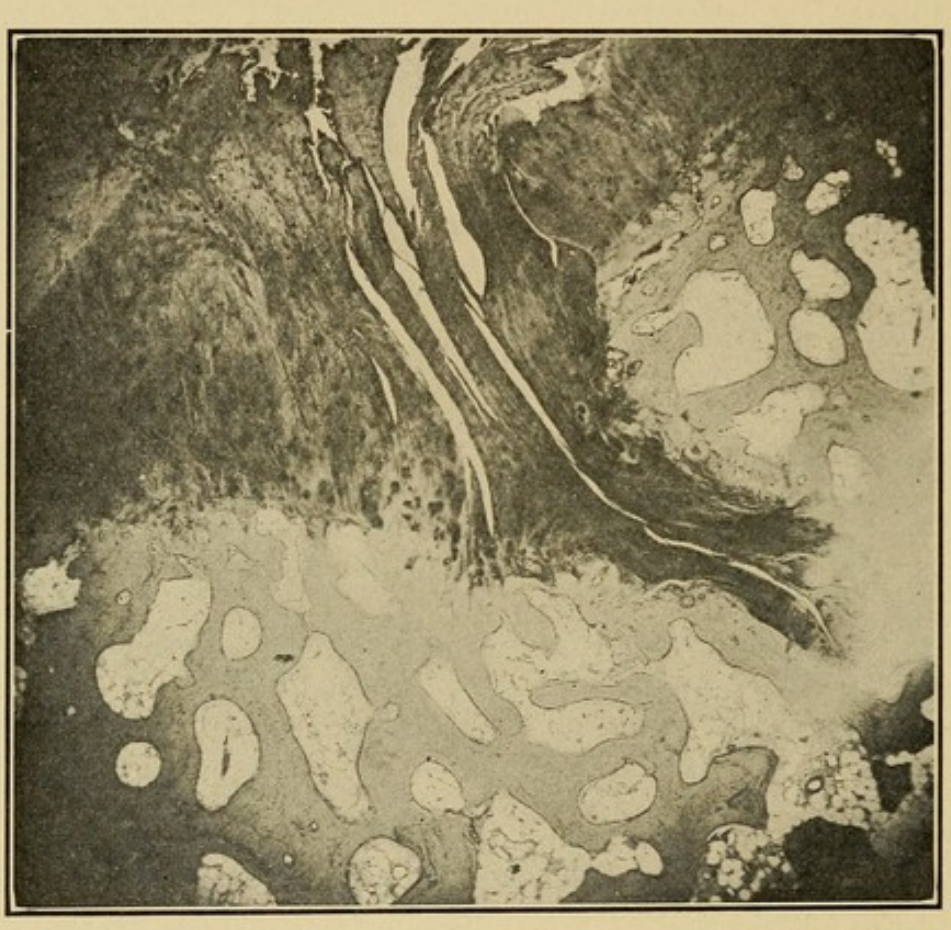

Fig. 56.

Photomicrograph from a case of chronic arthritis in the hip of a young woman, Type II. Degenerated cartilage above, bone below. Note the band of fibrous tissue running down into the bone. Cartilage cells can be seen in it—fibro-cartilaginous tissue. Leitz objective No. 2, no eye piece.

terial similar to that found in the marrow may be seen in the gaps in the cartilage. A ball of cartilage, found loose in one joint, was composed of densely packed cartilage cells with scanty matrix, and was

surrounded by a well-marked capsule of fibrous tissue.

SYMPTOMATOLOGY.

The manifestations of this type of joint disease are not as troublesome by any means as are those of the preceding. Constitutional symptoms and changes in other tissues are not conspicuous, though occasionally the patients suffer from indigestion.

Pain and restriction of function in the affected joints constitute the chief cause of complaint. The pain is usually decidedly worse when the joint is used, and often is of small account as long as it is kept quiet. When the lower extremity is affected the patient suffers from a limp. The knee is wont to be in semi-flexion, the hip in flexion, abduction and external rotation. More or less atrophy of the limb is usually present. The restriction of motion, always present, is due to mechanical interference, and not to muscular spasm. Grating and cracking may be perceived on palpation or on auscultation.

Restriction of Motion Due to Mechanical Obstruction

Herberden's Nodes manifest themselves as small hard nodules on the sides of the terminal phalanges, not painful, as a rule, except during the process of formation. They are accompanied by lateral distortions and slight flexions of the end phalanges.

DIAGNOSIS.

In the early stages of the disease, especially of the deeper joints, a positive diagnosis may be impossible; hence the advisability of conservatism. Many of these joints have been resected as tuberculous. In the later stages the bony and cartilaginous outgrowths, the "lipping" of the joint circumference,

the bony thickening, with the wearing away of the cartilage in places, should clinch the diagnosis.

The cases accompanied by the formation of Heberden's nodes should occasion no difficulty.

Tuberculosis is uniarticular and begins insidiously also, but is more painful as a rule, is accompanied

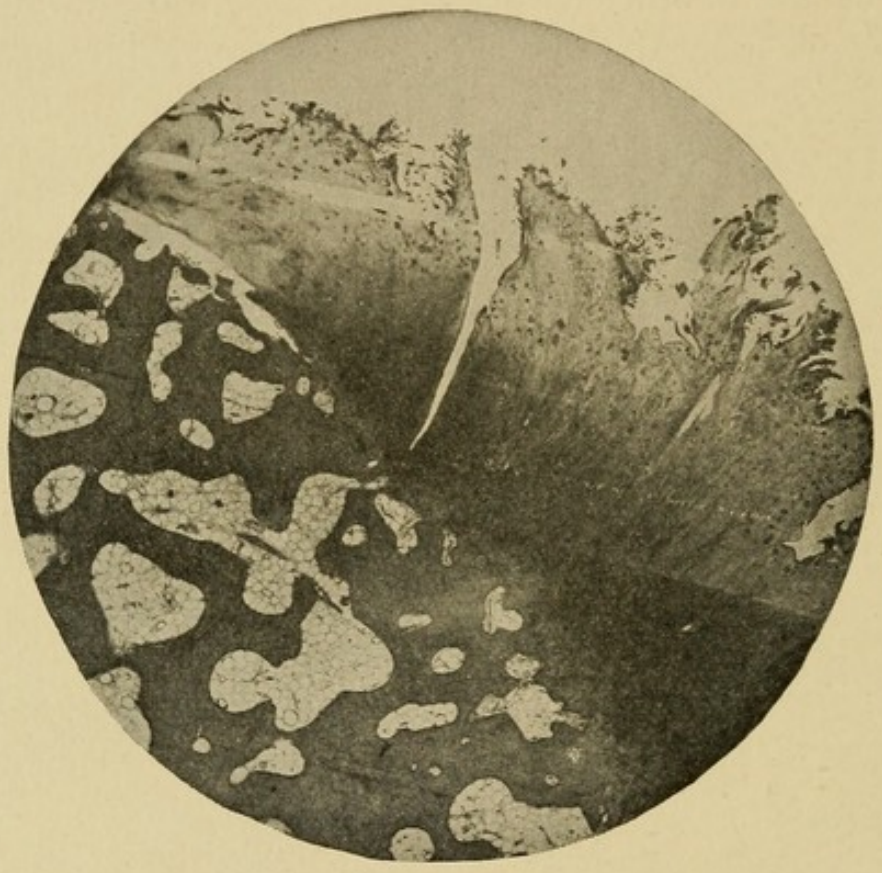

Fig. 57.

Photomicrograph of the same case as the preceding. This picture shows well the degenerating cartilage—its fibrillation. Note the cartilage "tatters." Zeiss objective aa, no ocular.

by muscular spasm, and shows in a skiagram an early rarefaction of the bone. The limitation of motion in tuberculosis is caused by muscular spasm as well as by the products of inflammation in the joint, while in this type of arthritis the obstruction is almost exclusively mechanical. Bony and car-

tilaginous outgrowths do not occur in tuberculosis, though the bone tissues may be increased in density in the later stages.

Other cases in Type I, as we have seen, are characterized by bony atrophy, thickening of the synovia, and atrophy and erosion of the articular carti-

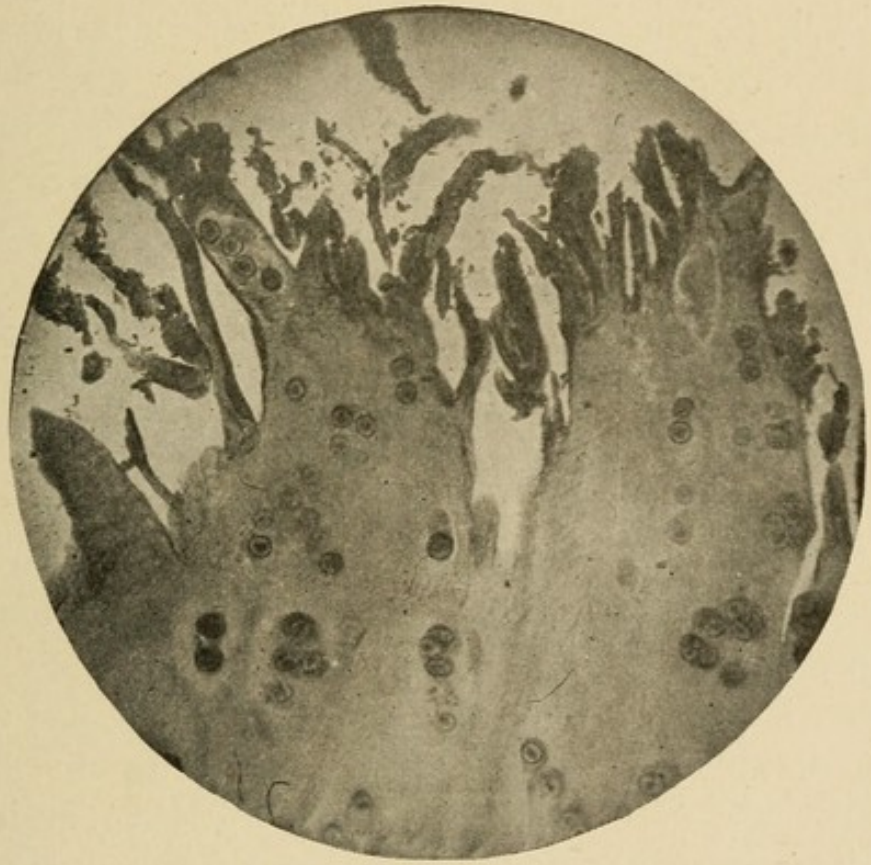

Fig. 58.

Higher power of the preceding—cartilage tatters. Zeiss objective C, ocular 1.

lage. The signs of pain and of active inflammation are more pronounced.

The lesions of the fingers in Type II. are usually of the terminal phalangeal joints, are nodular, and cause lateral deformities. Those of Type I. are of the metacarpophalangeal or proximal phalangeal

joints, are fusiform, later often shrunken, and cause flexion deformities.

Gout manifests constitutional symptoms, comes in acute attacks, recurring often in the same joints, usually the great toe, is very painful and leaves chalky deposits behind.

PROGNOSIS.

The prognosis *quoad vitam* is good. The disease does not affect the constitution in any way. We must not, however, expect a return to painless function, except in the very mildest cases. Severe damage once done to the bone and cartilage will never be repaired. If the disease be complicated by involvement of the spine, as will hereafter appear, the prognosis becomes graver, for here the functions of vital organs may be compromised.

TREATMENT.

The constitutional treatment consist largely, as far as can be ascertained, in promoting elimination through the gastro-intestinal canal, either by cathartics, or better by flushing of the colon, and to a moderate extent by hydrotherapy,—baths, etc. The daily use of phosphate of soda has been praised. If derangement of the viscera be detected, it should be rectified. If any other source of infection be discovered, it should be removed, as in Type I. The same may be said of any mechanical injury.

Passive Motion Harmful

Local treatment. Passive motions, mechanotherapy, etc., in contradistinction to Type I, are absolutely harmful. If we picture to ourselves the pathological condition of the joint we can easily see how the only effect of passive motion will be to injure the joint directly, by grinding together the

rough bony and cartilaginous surfaces, and by wounding the soft parts.

All our efforts should be directed to rest and protection. A brace which permits motion within painless limits is advisable. The fingers may be splinted with thick gloves, possibly reinforced by

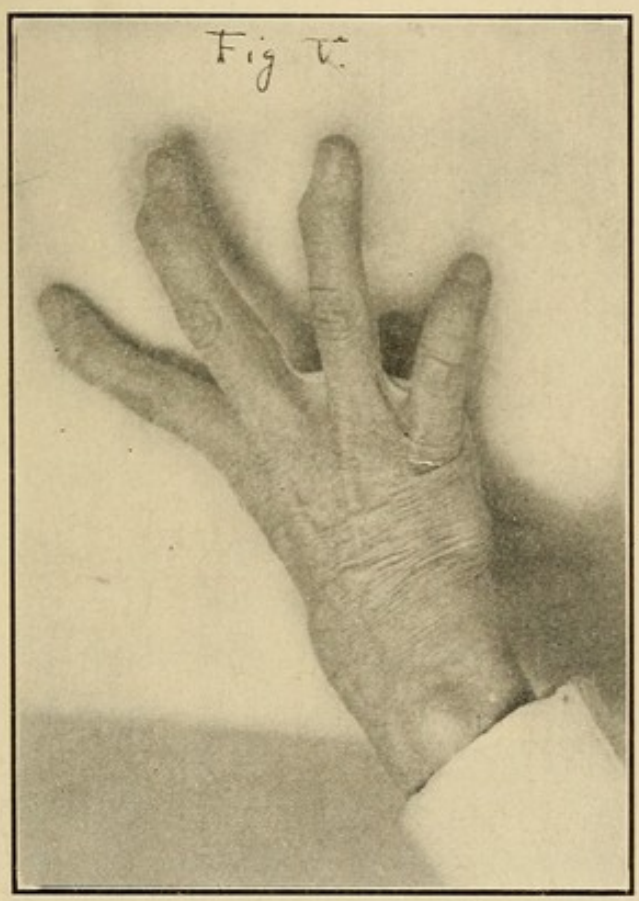

Fig. 59.

Multiarticular finger lesions of Type II.—Heberden's Nodes.

light metal splints. If the joints be very painful they may be immobilised for a while with plaster of paris. Baking may be found of service.

Operative treatment is occasionally advisable. If the disease has run its course, and a well marked spur interfere with motion, an arthrotomy may be done, and the spur may be removed. Oftentimes

a stiff, firmly ankylosed, painless joint is much to be preferred to a slightly movable, painful one. In such a case a resection should be done with the sole idea of stiffening the joint. Albee claims excellent results for an operation of his devising, upon the hip[1]. He opens the joint through the anterior incision, chisels off the upper portion of the head of the femur in a line about parallel to the femoral neck, and a corresponding piece from the upper part of the acetabulum. This procedure results in a practical subluxation of the hip. Then as much as possible of the cartilage is removed from the head of the femur and the acetabulum, the wound is sewn up, and a long plaster spica is applied, with the extremity in slight flexion and abduction.

CHRONIC ARTHRITIS OF THE SPINE.

Chronic Rheumatism of the Spine, Rheumatoid Arthritis and Osteo-Arthritis of the Spine, Spondylose Rhizomelique, Von Bechterew's Type, etc.

While various writers have described various types of chronic arthritis of the spine, and have emphasised their clinical distinction from the disease as found in the other joints of the body, the truth of their contentions has not been established, and the weight of opinion is that the disease, as manifested in the spine, can be generally divided into the two types hitherto described. Type II in the spine has the same multiarticular characteristics as in the fingers. It is quite probable also that many of the cases hitherto regarded as lumbago and fascial rheumatism are examples of the rudimentary form of chronic spinal arthritis.

[1] Albee, *Surgery, Gynecology and Obstetrics,* March, 1910.

Occurrence

The disease occurs with overwhelming preponderance in the male sex; Goldthwait and others affirm that it follows exposure to chilling of the surface, especially among those, who, like firemen and engineers, are exposed to sudden changes of temperature. This were better regarded as a predisposing cause, lessening the resistance of the individual to infection, as is the case with many infectious diseases, or better yet, viewed in the light of repeated trauma, as a direct exciting cause.

A fairly safe attitude for the present is to view the disease in the spine as due to the same causes as in other joints. Possibly certain forms are merely early manifestations of the chronic ankylosing process so often seen in apparently normal persons as age advances.

PATHOLOGY.

On account of the inaccessibilty of the spinal bones and joints we are compelled to trust for our knowledge of the pathology to the specimens on the post mortem table and in museums, a very unsatisfactory state of affairs, for we can observe merely the results of the pathological processes, and not the pathological processes themselves.

In the spine there are three sets of joints which may be involved, those between the bodies of the vertebrae through the medium of the fibro-cartilages, those between transverse processes, and the costo-vertebral articulations. The finer changes in these joints during the course of the disease are largely a matter of surmise. Sometimes a part of the spine is affected, and sometimes the entire column. The intervertebral fibro-cartilages may de-

generate and be absorbed, or they may also be involved in the ossifying process, and then the spine is converted into a solid bony column. Masses of new bone are formed on the anterior surface of the column, often more on one side than on the other, welding the vertebrae to one another. In some in-

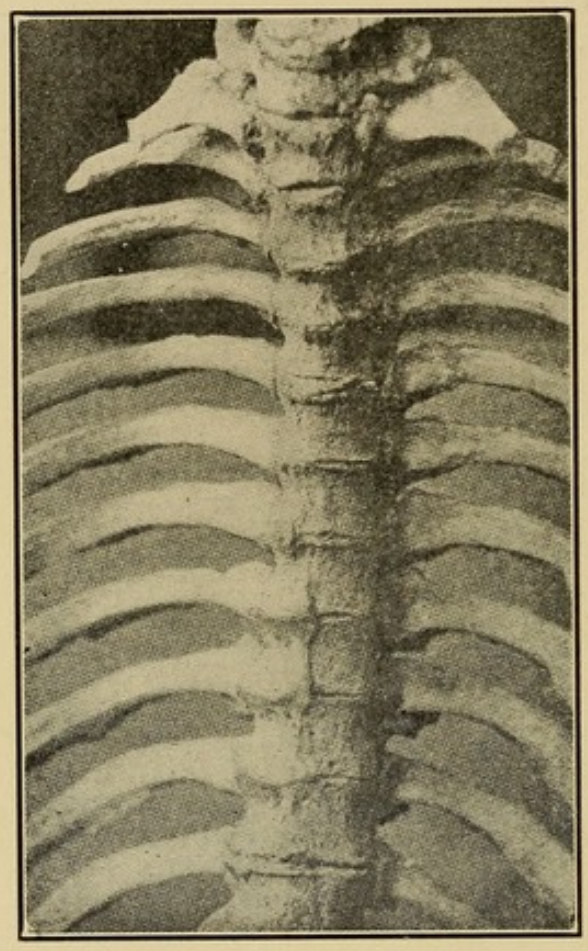

Fig. 60.

Chronic arthritis of the spine. Note the bony ankylosis. (Goldthwait.)

stances, while solid bony union appears to be present, on opening the spine the intervertebral joints will be found persisting at their centers. Bony "lipping" takes place at the edges of the vertebrae. The new bone formation may extend out on the ribs, and at times may encroach upon the spinal

canal and upon the foramina of exit of the spinal nerves.

SYMPTOMATOLOGY.

Symptoms of pain, stiffness, disability and deformity are present here as in the other joints.

The pain may be severe or mild, it may be localised in the back, especially in the lumbo-thoracic segment, or it may shoot down the extremities.

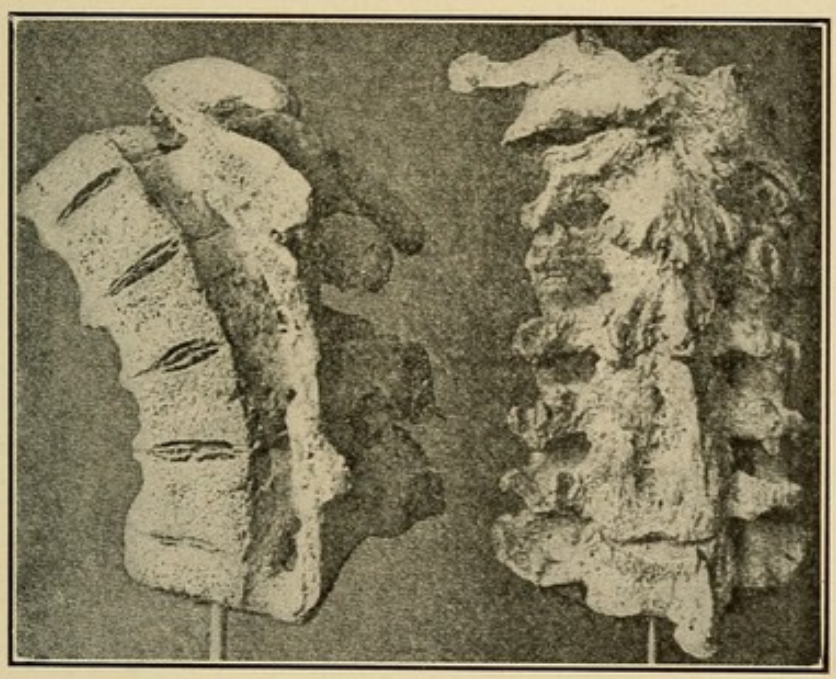

Fig. 61.
Chronic spinal arthritis.

Many cases of "lumbago" and "sciatica" are due to spinal arthritis. If the "sciatica" be one-sided, it will often be accompanied by atrophy of the extremity on that side.

The stiffness may vary in degree and in extent. Sometimes segments of the column may be restricted in motion, sometimes the entire column. The uppermost joints often escape involvement. If the invertebral discs have atrophied, the resulting

deformity will be in the nature of a long, round posterior curve. If bony union have taken place before the discs have atrophied, this curve will be absent, and the deformity may then consist of an obliteration of the normal curves, giving an approximately straight spine—the "poker back." The long posterior curve with stiffness is an exaggeration of the deformity that is wont to be present as age draws on, in the healthy human body. Lateral deformity may or may not be present.

DIAGNOSIS.

The diagnosis as a rule need occasion little difficulty. Pott's disease in its early stages might be confused with the localised form. If a segment of the spine be affected which is accessible to the Roentgen rays, bony outgrowths may be made out in Type II. in contrast to the rarefaction of Pott's disease. A kyphosis in Potts' disease is more angular than in these cases of chronic arthrities, and *is not often complicated by disease of other joints.* As a rule it is more localised. Abscesses are frequent in Pott's disease, absent in most other forms of spinal disease.

Kyphosis After Injury

A slowly developing kyphosis sometimes appears after spinal injuries in which no fracture can be made out. It appears some time after the injury, and gradually increases. Pain is not a prominent feature[1].

TREATMENT.

The general principles of treatment are those already laid down, namely, removal of the source of

(1) Mauclaire et Burnier, *Archives Generales de Chirurgie,* March 25, 1912.

infection, if that be possible, plus rest and protection. A well fitting spinal brace or a jacket should be worn to relieve the pain and to prevent deformity. It is important that the development of the long posterior curve should be prevented, and this can often be done by the constant wearing of a spinal support. Heat, massage and vibration may relieve the pain. Attempts at forcible mobilization are distinctly out of place, and may do harm.

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CHARCOT'S JOINT.

TABETIC OR NEUROPATHIC JOINT.

In the course of certain spinal diseases, notably tabes dorsalis and syringomyelia, a peculiar form

Fig. 62.

Charcot's Joint. T, T, T, bone trabeculae, M, M, marrow F, fibrous tissue at joint surface. The articular cartilage has completely disappeared. Low power photomicrograph.

of joint lesion occasionally is seen, that has baffled all attempts at explanation. Charcot, who first described it, thought it was due to a lesion of the

trophic centers in the anterior horns of the spinal cord; Virchow, Von Volkmann and others maintained that it was simply a form of "arthritis deformans" modified by the traumata incident to the lack of sensation produced by the nerve lesions. Others again say that the anesthesia alone is responsible.

Until more light is thrown on the subject we are free to hold any opinion that seems to us most rational. For myself, I prefer to regard the disease, as I regard other joint diseases, as primarily a lesion of the lymphoid marrow and synovia, probably produced by the late syphilitic toxines. A similar affection in the shafts of the long bones results in fractures. These fractures have two peculiarities, viz., their painlessness, and the apparent inadequacy of their cause.

Tabetic Osteoarthropathy may occur in any stage of tabes, preataxic or late. Sometimes it is seen years before the onset of other symptoms. It is more frequent in men than in women in about the same ratio as the causal disease, and is seen much more often in the lower than in the upper extremity. The reverse is true of the lesions in syringomyelia.

MORBID ANATOMY.

The pathology of Charcot's joint is somewhat as follows: A low grade inflammation takes place in the marrow of one of the articulating bones, which gradually eats away the bone trabeculae. This inflammation is not general but seems to occur in certain areas. In other places the marrow is largely fatty. In time the cartilage is destroyed, the inflammatory process has access to the joint, and the

Marrow Changes the Most Striking

synovia also becomes involved. The joint now becomes filled with fluid. The bone is killed in larger

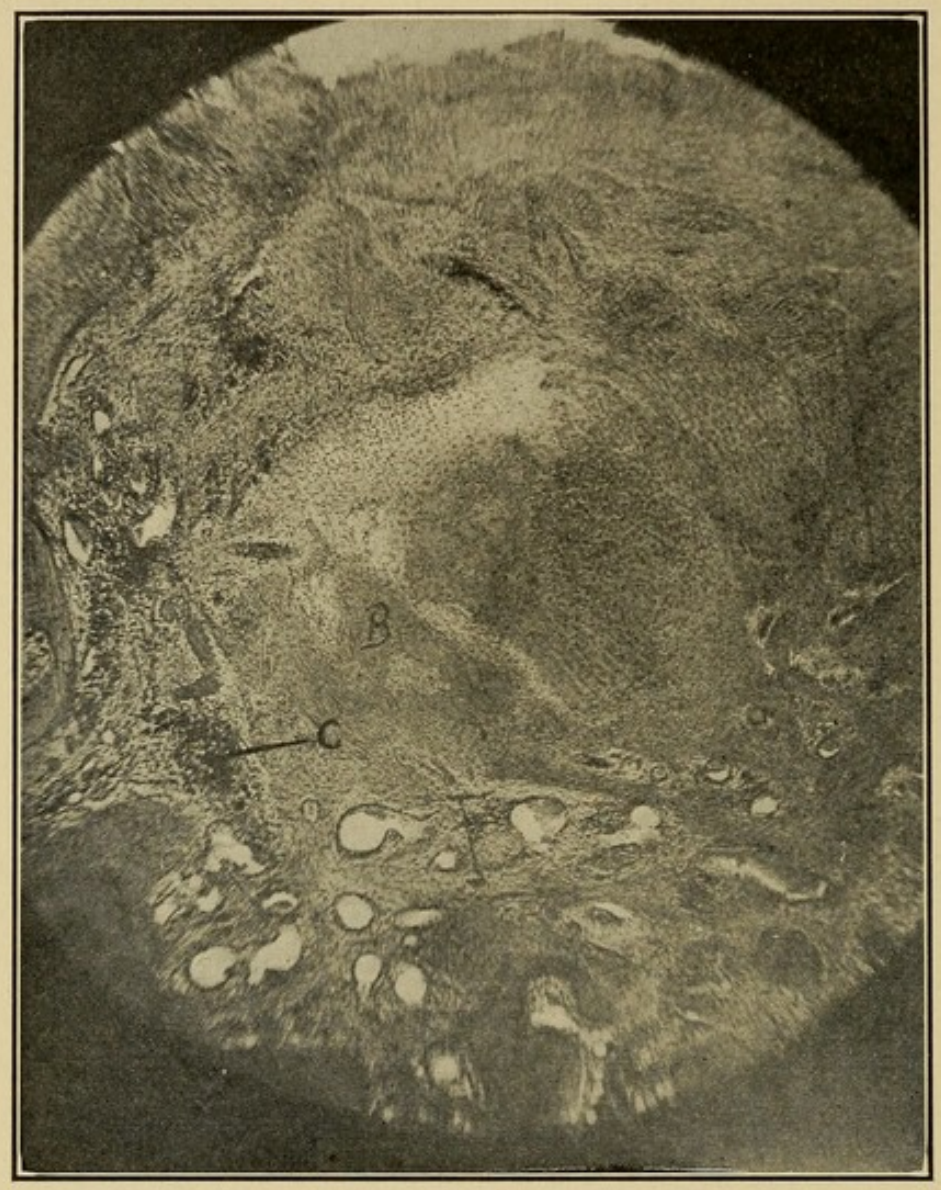

Fig. 63.

Low power photomicrograph of peculiar "focus" found at the joint surface of the bone in a Charcot's knee. It was composed largely of fibrous tissue, continuous with that covering the head of the bone. Indications of new bone formation can be seen at B. Portions of the enclosing bony wall of this "focus" were thickened. Bone trabeculae may be seen on the left of the picture, and bone and marrow indistinctly below. C, collection of round cells.

and smaller pieces, which are thrown out into the joint cavity, and occasion the creaking and

"crumbling" so characteristic of this disease. There is apparently some effort at repair of the damage by nature at the spot where it has occurred, but the effort is ineffectual. Nature's efforts at repair are largely confined to producing a compen-

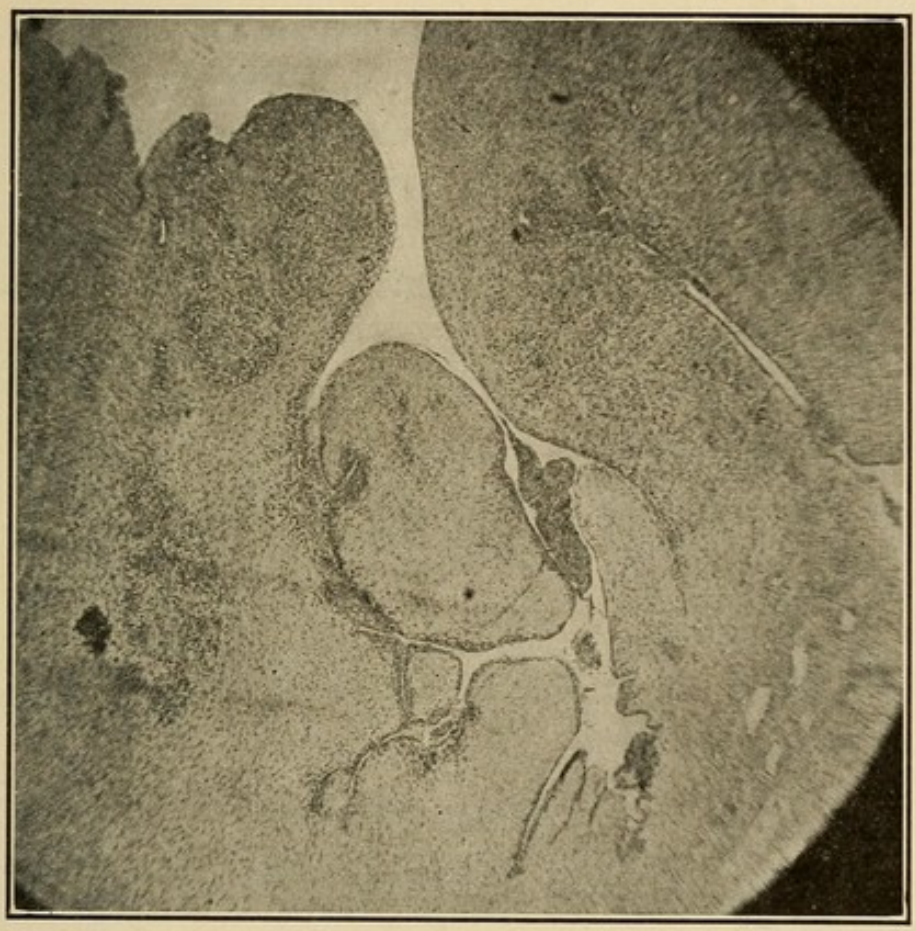

Fig. 64.

Photomicograph of synovia from a case of Charcot's joint. The membrane is greatly thickened and villous. It is largely fibrous in its structure. Low power.

satory hypertrophy of bone in other places, and in covering over the denuded bone ends with fibrous tissue.

In certain areas the fibrous tissue covering the bone end is continuous with the new fibrous tissue in the marrow; as if the disease had burst from the

marrow into the joint. In certain places rarefying osteitis may be seen, especially under the replacing fibrous tissue of the articular surface. At other places a productive osteitis is more prominent.

Often the destructive changes predominate in one articulating bone, and the productive in another. Again the ends of both bones may be largely destroyed.

There is no regularity in the morbid process. The bone ends are eaten away in large "bites," and roughly buttressed up in spots as if in an unskillful attempt to repair the damage. The joint becomes loose, subluxated and very "wobbly." Its component parts may be recognised with great difficulty in the laboratory.

Degeneration of the nerves of the limb has been observed, but not greater on the affected than on the sound side.

The synovia shows a moderate degree of proliferation. The resulting villi are composed largely of fibrous tissue.

SYMPTOMATOLOGY.

Course of a Tabetic Osteoarthropathy

The onset is peculiar. With slight cause or without known cause the joint suddenly fills with fluid. On our hypothesis, this sudden swelling of the joint represents the irruption of the disease from the marrow. The swelling extends far beyond the joint limits. This swelling is hard and oedematous. It may slowly disappear, and the joint may return to a practically normal state, to be affected again later, or the process rapidly may result in the disorganisation of the joint. Sometimes the course of the disease takes years. In spite of the suddenness and

severity of the onset, pain is practically absent. Crepitation can often be felt on motion, and loose bodies can be distinguished in the joint cavity. All sorts of distortions may be present. The most prominent feature of the disease is the total disproportion between the gravity of the anatomical lesions, and the amount of discomfort the patient suffers from them.

The symptoms of the causal trouble—the pupillary changes, the absence of the knee jerk, the ataxia, etc.—can be easily elicited.

DIAGNOSIS.

Little difficulty in diagnosis exists, if the possibility of a Charcot's joint be kept in mind. A haemarthrosis at first might present similar symptoms, but the haemarthrosis is not caused by a cord lesion. Only through carelessness can one err. When the spine is affected, the pressure of the bones upon the cord may give rise to physical signs unusual in tabes dorsalis.

Haemarthrosis

TREATMENT.

Conservative treatment is usually best. A well fitting brace will often enable the patient to go about with comfort for years. Resections have been done with good effect in the ankle; with rather poor results in the knee. Amputations of the leg have also been successful. In the thigh they are much more dangerous.

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CHAPTER VI.

ANKYLOSIS.

True and False Ankylosis

The word ankylosis is derived from the Greek word *agkulos,* crooked, but the modern meaning attached to ankylosis is joint stiffness. The stiffness may reside in the joint tissues themselves—true ankylosis—or in tissues outside the joint—false ankylosis. The term contracture is gradually replacing the latter.

Ankylosis may be due to abnormal connection between the ends of the bones entering into the formation of the joint, or it may be due to the obstruction caused by bony or cartilaginous growths, or by misplaced pieces of bone. The former is divided into complete or bony, and incomplete or fibrous. There is probably no such thing as intercartilaginous ankylosis. As long as the cartilages are intact ankylosis does not occur. When they are destroyed in whole or in part, the resulting ankylosis is fibrous or bony.

Although any diseased joint may present stiffness more or less great, yet ankylosis has come to mean generally the stiffness remaining after the disease has run its course.

A word may probably be said here concerning the fear of ankylosis from putting a joint at rest with retentive splints or bandages. It is doubtful if immobilisation, even for long periods, ever avails to cause anything more than temporary changes in a

normal joint, which soon disappear when motion is again permitted. The restriction of motion lies in the muscles and tendons outside the joint. In other words, ankylosis following the application of splints is due, not to the immobilisation, but to the disease or injury for which the immobilisation was applied.

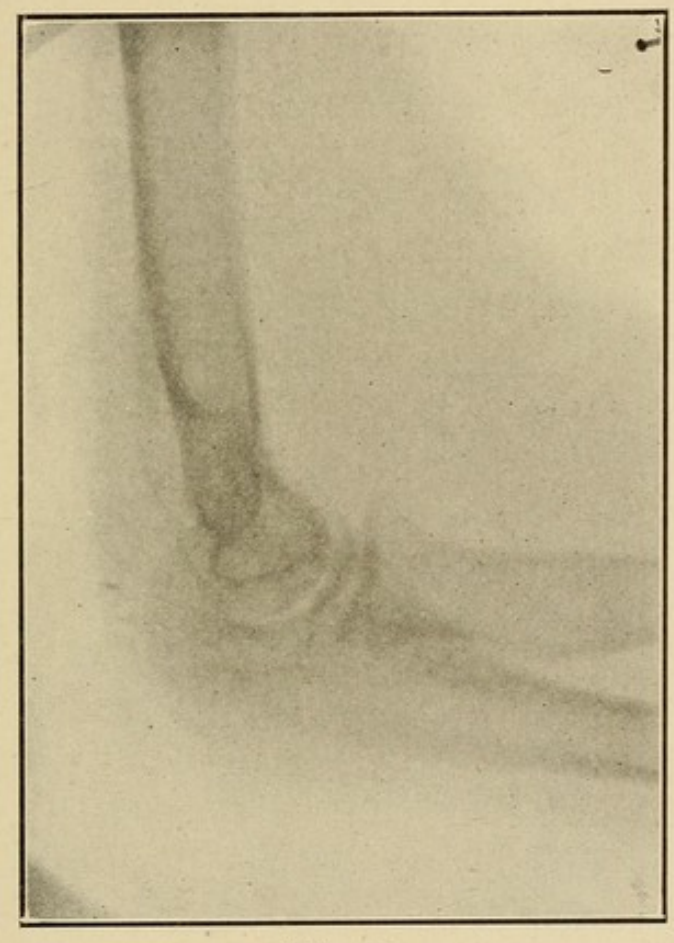

Fig. 65.

Fibrous union following a hemarthrosis in a hemophiliac.

The first duty of the surgeon, to whom a patient with any ankylosed joint presents himself, is to find out the original cause of the ankylosis. His subsequent action will be governed accordingly, but he should always observe the cardinal rule, never to adopt any measures whatever to mobilise a joint that is the seat of active disease. Such measures

are not only quite useless, but may be dangerous as well.

The second problem is to ascertain exactly the nature of the ankylosis, whether bony or fibrous union be present, or whether the obstruction be due to distorted and deformed bone ends. We decide

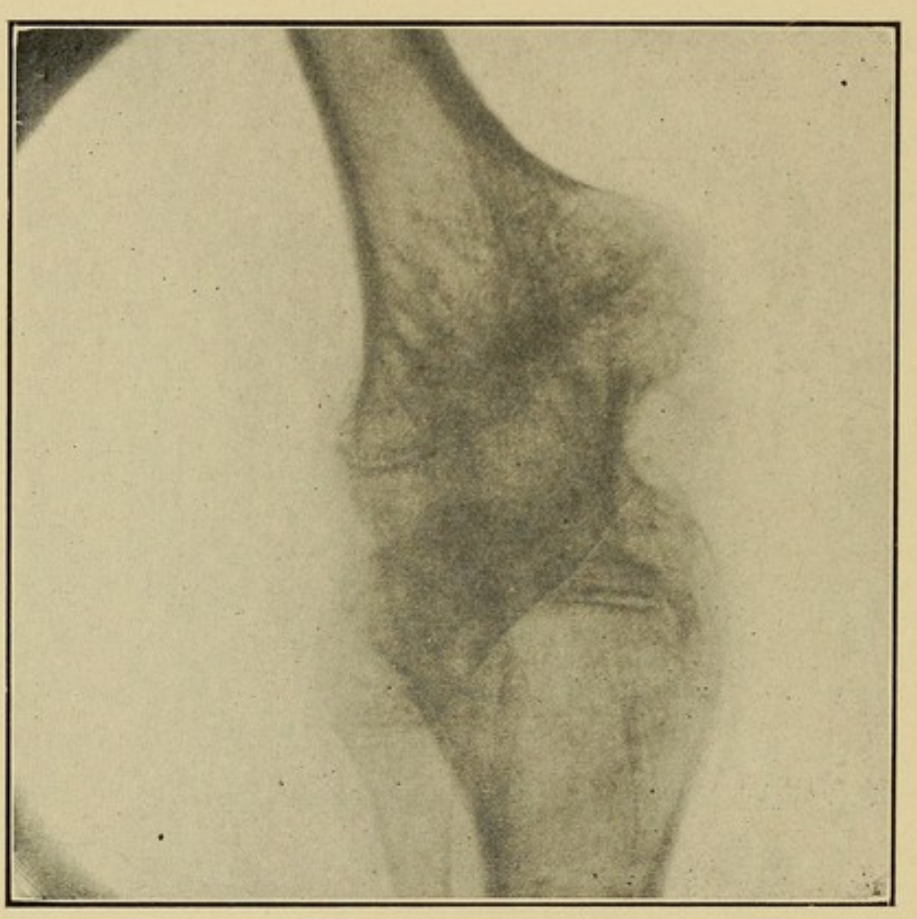

Fig. 66.

Old healed knee joint tuberculosis in a child of about thirteen. Conservative treatment. Bony union following a mixed infection. Note the remains of the epiphyseal lines, and the irregularity of the new bone between the tibia and femur.

as to the nature of the ankylosis by a study of the history, by good skiagrams, and by a careful examination of the joint, under ether if necessary. If any motion be present, the ankylosis is not bony. Ankylosis from distorted bone ends results espe-

cially from diseases of Type II. of chronic arthritis, and from intra-articular fractures. In such cases the joint motion may possibly be improved by chiseling away the bony obstructions, but it is quite evident that any attempt at passive motion will only do harm by injuring the joint tissues. Anky-

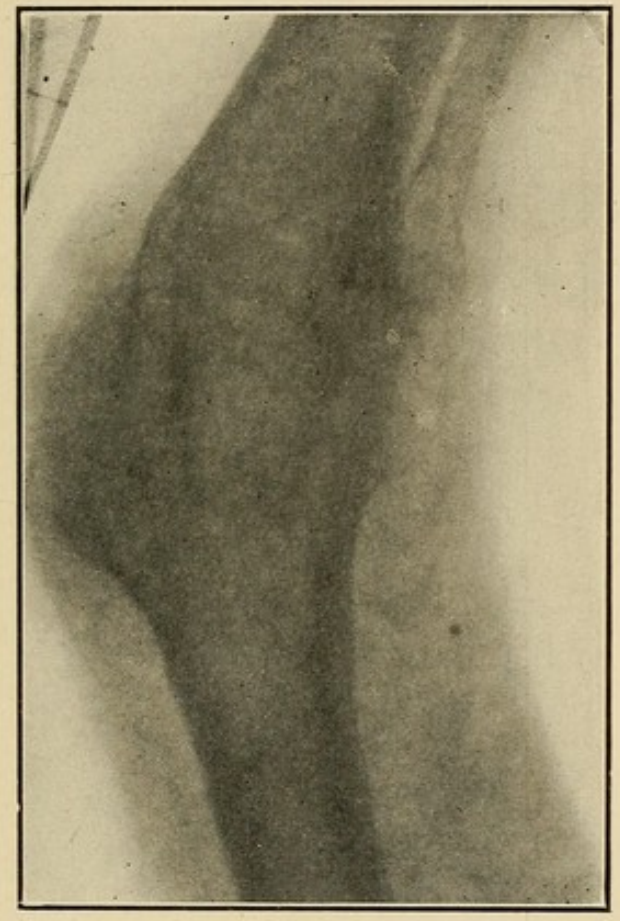

Fig. 67.

Same case as Fig. 66. Skiagram taken two years later. Note the smoothing off of the bony prominences, the canalization of the bone at the former site of the joint.

losis by bony union may also follow intra-articular fractures.

FIBROUS ANKYLOSIS.

This may follow acute arthritis or any of the chronic arthritides of Type I, and its treatment will

vary as the cause. If it be due to tuberculosis, and if the joint be in a good attitude, and painless, our wisest course is to let it severely alone. Our study of the pathology of this disease has taught us that encapsulated foci may lurk in the bone marrow and in the intra-articular adhesions for years, ready to

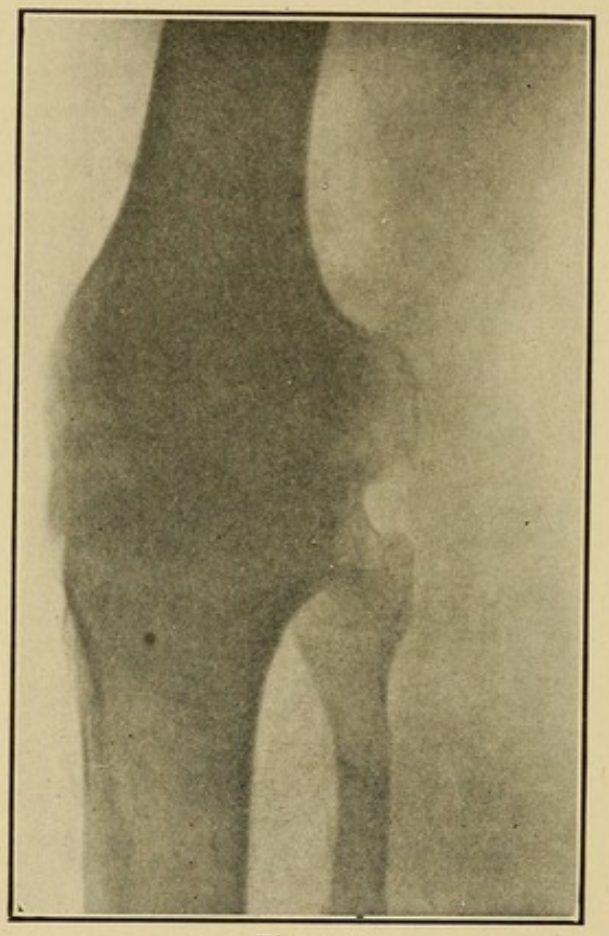

Fig. 68.

Old knee joint tuberculosis in a young adult. Bony union two years after a resection done on the author's theory. Note the density of the bone at the old level of the joint. The patient is well.[1]

light up afresh if motion be resumed, and while an occasional old tuberculous joint may be successfully mobilised, the risks of the operation should condemn it.

[1] For a history of this case, see Ely, Joint Tuberculosis, Wm. Wood & Co., 1911, page 92.

If an old tuberculous joint is ankylosed (by fibrous tissue) in a faulty attitude, the deformity should be reduced as gently as possible, and the joint should then be immobilised by plaster in the correct attitude for a number of months. The reduction may be made by a number of plaster ban-

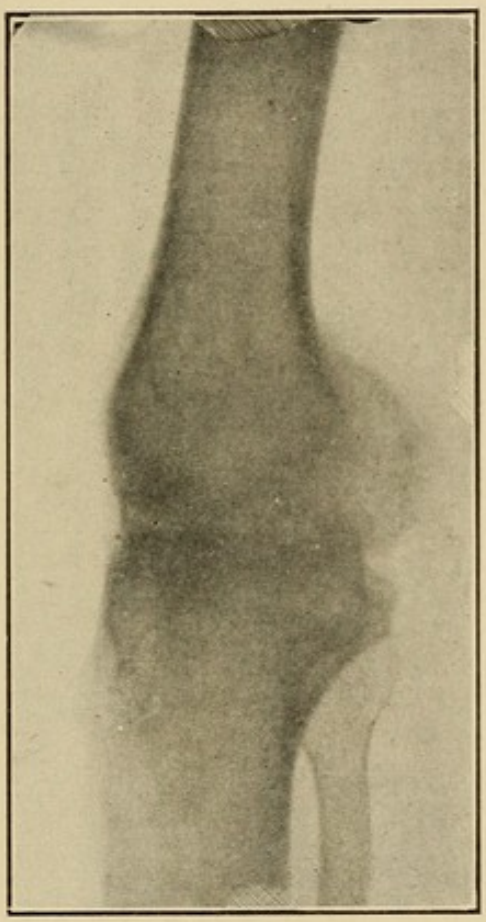

Fig. 69.

Same case as the preceding. Canalization is advancing very slowly. Skiagram taken four years after resection.

dages, correcting the attitude a little at a time, or it may be made under ether at one sitting, dividing the contracted tissue if necessary. If the latter, then great care should be exercised not to use undue force. Some good authorities use prolonged traction by apparatus, while the patient is kept in bed.

For the milder cases of fibrous ankylosis following the other diseases, baking, massage, vibratory massage, passive hyperaemia, and active and passive motion will often accomplish all that is necessary. For the severer forms we shall often do well to

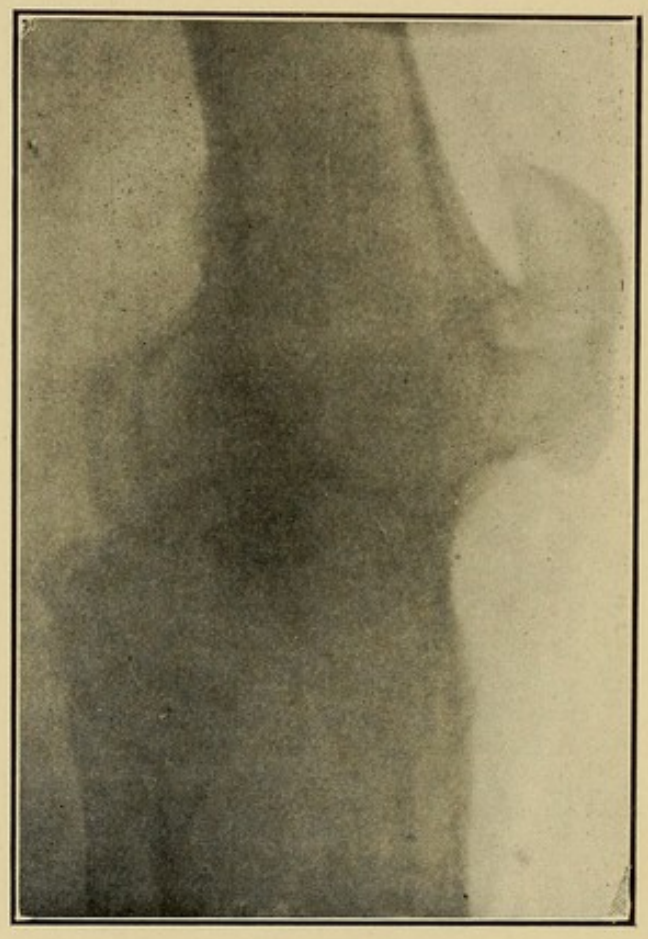

Fig. 70.

Old knee joint tuberculosis in the adult. Skiagram shows the bony union two years after a resection done according to the author's theory. Note the area of dense bone at the former site of the joint, and the beginning canalization.

break up the adhesions under ether, and then put the joint up in plaster in an entirely different attitude for a couple of weeks, following this with the treatment recommended for the milder forms. Possibly a repetition of the operation may be ad-

visable once or twice, with a changed attitude each time.

For the most severe cases even these measures may fail, and we may be forced to consider in an adult the advisability of an arthroplasty.

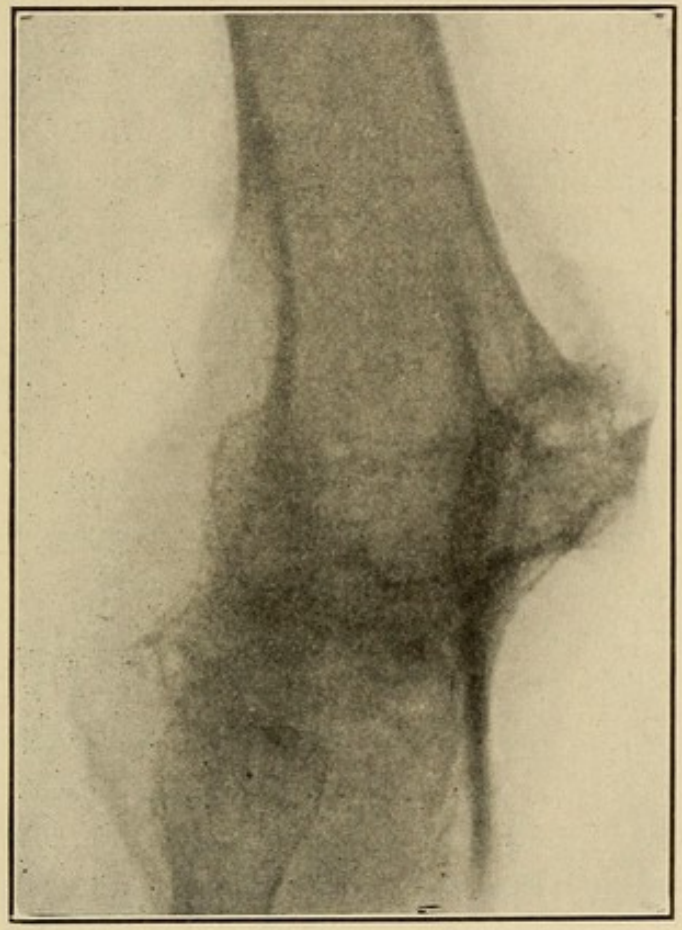

Fig. 71.

Same case as the preceding. Note the advancement of the process of canalization. The patient is well.

BONY ANKYLOSIS.

Bony ankylosis in the limbs may follow an intra-articular fracture, a pus infection, and certain of the chronic arthritides of Type I. I believe that it never follows an unmixed tuberculous, gonorrheal, or typhoid arthritis.

When the joint is ankylosed in a faulty attitude, we have the choice of an osteotomy and correction, or of an arthroplasty. An arthroplasty for a bony ankylosis following a mixed tuberculous infection is probably safer than that for a fibrous one.

ARTHROPLASTY.

The credit of successfully making a new joint seems to belong to Helferich. Murphy has done much to popularize the operation in this country.[1] The formation of a false joint following the interposition of muscle between the fractured ends of a bone originated the idea. Sumita has recently demonstrated upon dogs the histological changes in the joint following the operation.[2]

Arthroplasty Occasionally Successfully

Various substances, such as chromicised pig's bladder (Baer), silver foil, etc., have been employed to prevent the reunion of the divided bone, but none has been demonstrated superior to autoplastic flaps of fat, fascia or muscle.

The operation is rather difficult, demands an elaborate technique, and is not applicable to a great number of cases. In young, vigorous adults with the pecuniary recourses and the will power to carry out the after-treatment, it may afford relief. Its outlook seems to be best in the elbow.

Payr,[3] who has done a great deal of work along this line, reports nine successful results out of thirty operations. Many other operators have been even less successful.

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[1] Murphy, *Journal of the American Medical Association*, 1905, XLIV, 1573.

[2] Sumita, *Archiv für klinische Chirurgie*, October, 1912.

[3] Payr, *Münchener medicinische Wochenschrift*, 1910, No. 37, 1921. *Archiv für klinische Chirurgie*, October, 1912.

JOINT TRANSPLANTATION.

This operation has been put forward by Lexer,[4] and may eventually find a place in recognized procedure, but at present it is on a more or less experimental basis. To transplant a joint is comparatively simple, but to transplant the tendons attached near the joint, and giving it its function is a much more serious problem.

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[4] Lexer, Medizinische Klinik, 1908, 817, Centralblatt für Chirurgie, 1908, No. 38, H. 2. See also Axhausen, *Archiv für klinische Chirurgie*, August, 1912.

CHAPTER VII.

DISEASES OF THE SHAFTS.

ACUTE OSTEOMYELITIS OR MYELOPERIOSTEITIS.

As in other organs of the body, so in the bone, an inflammation may be caused by a number of different infectious agents, and in the shafts the principal infective agents are the pus cocci—staphylococcus and streptococcus—typhoid bacillus, and the treponema pallidum of syphilis. Hence we find the disease occurring as a metastasis from an unknown focus of infection, and as the result of compound fractures, etc., as a complication of typhoid and as a manifestation of syphilis. Uncomplicated tuberculosis is said to occur in the shafts of children's bones only, and is extremely rare. The fatty marrow of the adult bone affords no field for invasion by the tubercle bacillus. Some authorities maintain that the gonococcus may cause an osteomyelitis. I believe that this is correct. A pus infection may be grafted on other forms of bone marrow infection. A secondary infection of a tuberculous arthritis permits an extension of the disease to the shaft marrow also.

Occurrence

SUPPURATIVE OSTEOMYELITIS, OR SUPPURATIVE MYELO-PERIOSTEITIS.

This form of inflammation of the marrow and of the inner layer of the periosteum usually goes

by the name of acute infectious osteomyelitis, but the term is not descriptive, for a myelitis or periosteitis caused by the typhoid bacillus, by the gonococcus, or by the tubercle bacillus is infectious also.

The cause has already been mentioned. The staphylococcus pyogenes aureus is the usual infective agent, more rarely the streptococcus and the staphylococcus-pyogenes albus. The disease occurs most frequently in children and adolescents. Trauma and chilling of the surface have been said to predispose. The acute infectious diseases, which frequently precede an arthritis, may precede a myelitis of the shaft also.

PATHOLOGY.[1]

The essential features of an inflammation of the marrow of the shafts are much the same as those of an inflammation of the marrow of the ends of the long bones. The results are modified by the structure of the bony and periosteal envelope of the diseased marrow and represent the solution of a mechanical problem.

The Diffuse Form

In the more common form (the diffuse) the original focus usually forms in the marrow of the metaphysis (most often of the femur, tibia, or humerus) near the epiphyseal line, and spreads in the meshes of the spongy bone until it reaches the large central marrow canal, the marrow compartments of which communicate freely with one another. The disease spreads, in other words, in the direction of least resistance, for the epiphyseal cartilage offers a barrier to its progress toward the

[1] See also E. H. Nichols, *Journal of the American Medical Association*, 1904, XLII, 439.

joint. Streptococcic myelitis is said to involve the joint oftener than staphylococcic.

According to some writers the start of the process in a large proportion of the cases is in the marrow of the central canal.

Death of Bone

The violent inflammation kills the marrow tissue, which breaks down, and the bone, deprived of its nutrition, dies also. Pus forms. The process spreads through the marrow canals in the cortical bone and involves the deep layer of the periosteum Pus forms here also, raising up the periosteal membrane from the bone, and running along beneath it. Coincident with all this the disease may have sought the surface of the bone in the metaphysis, made its way through the thin, dense layer of bone and, gaining the under layer of the periosteum, may have spread along shaftwards. In its typical and well developed expression the morbid process results in the death of the bone from one epiphysis to the other, and the dead shaft lies bathed in the broken-down and suppurating marrow and inner layer of the periosteum, the dense outer layer of the periosteum enclosing the whole in a firm envelope, preventing the exit of the pus, and thus adding to the severity and destructiveness of the inflammation. The cortical bone of the shaft also serves to bottle up the inflammatory products in the central canal in spite of the minute marrow canals running through it, and thus helps the spread along the central canal.

The pus may make its way through the cortical bone and periosteum and gain the surface, but in the severer cases, unless artificial aid is rendered,

death from septic absorption may take place before this occurs.

When an operation is undertaken in the early stage, the incision through the periosteum opens up the abscess beneath it. The bone has lost its connection with the periosteum, and exudes pus through the minute openings on its surface. If the bone be trephined, the central canal will be found full of pus, containing fat droplets that give it an oily appearance.

The complete and typical course is by no means invariable. The inflammation may spread along under the periosteum and kill a portion of the cortical bone, possibly never invading the central canal, or it may involve a section of the central marrow canal and shaft, and leave the rest uninvaded. In this case the healthy end of the bone is wont to be walled off from the diseased end by a mass of dense bone.

The neighboring joint may be the seat of a serous exudate. In some instances the inflammation may make its way into the joint, causing a suppurative arthritis.

Marrow Changes

Under the microscope the marrow is seen to be the seat of an intense inflammation, and contains nests of pus cocci, abscesses, areas of round cell infiltration, etc. In places the marrow undergoes a fibrous change.

As the disease spreads, the bone trabeculæ in places show productive, in places rarefying inflammation. Many of them are dead. Some of the dead trabeculæ in the less active cases are lined by osteoid tissue, showing the effort of the still living marrow to repair the damage done. When the

morbid process has run its course, then all signs of activity cease in the dead structures.

When an acute purulent inflammation has run its course in the soft tissues, healing is accomplished by a filling of scar tissue, but in osteomye-

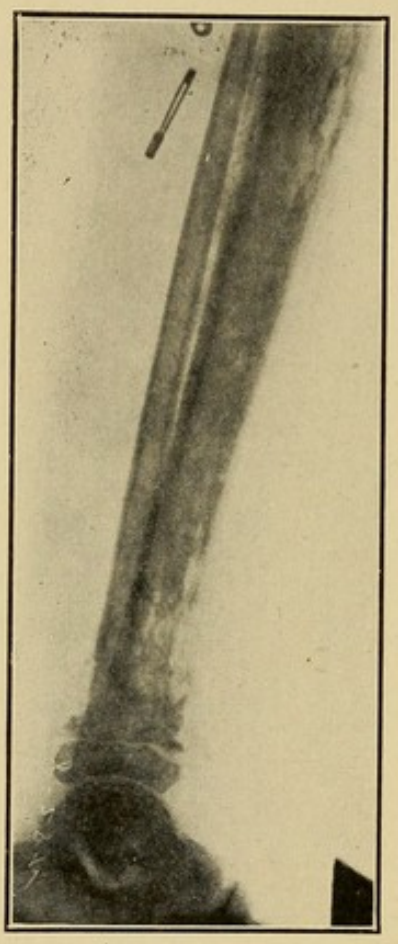

Fig. 72.

Acute suppurative osteomyelitis, diffuse form, in a boy of 7.

litis this is impossible. Whether the pus have been let out with a knife, or whether spontaneous discharge have taken place, the piece of necrotic bone shaft lies in its fibrous periosteal envelope, with which it may be adherent in places, attached at one or both ends to living bone. In time nature will separate the dead from the living bone, but if the

destruction have been extensive, the dead sequestrum cannot be thrown off through the comparatively small openings in the periosteum, but remains indefinitely locked up in a shell of new bone (the involucrum) built up around it by the activity of the inner layer of the periosteum. This is the so-called chronic stage of osteomyelitis, but in point of fact there is no active marrow inflammation. The disease has run its course, and the wound is prevented from healing by a mechanical obstruction.

Mechanical Obstruction to Healing

CIRCUMSCRIBED FORM.

Another form of the disease occurs which shows little tendency to spread far, or to do much damage. The focus is located as a rule near the epiphysis. The bone breaks down in a small area, and around this area the bone may be enormously thickened. The course may be rapid or very slow. Sometimes the collection of pus may remain for a long time. According to Klemm[1] many cases of chronic arthritis are due to circumscribed suppurative osteomyelitis in the bone end.

SYMTOMATOLOGY.

The onset of an acute suppurative osteomyelitis is usually sudden, with chill, fever, etc. Pain is an early and constant symptom, and is very severe. It has been described as "boring." Sensitiveness of the affected bone to pressure is extreme, and the location of this sensitiveness is wont to be in the neighborhood of a joint, but not in the joint itself. Reddening of the skin, and edema may be present, and the edema may involve the portion of the limb

[1] Klemm, *Archiv für klinische Chirurgie,* February, 1912.

distal to the diseased area. Fluctuation can rarely be elicted in the early stages. Even after the process has spread up into the central marrow canal, the physical signs may still be most severe in the metaphysis. Delirium, extreme prostration and marked constitutional involvement may be early signs. A leucocytosis is the rule.

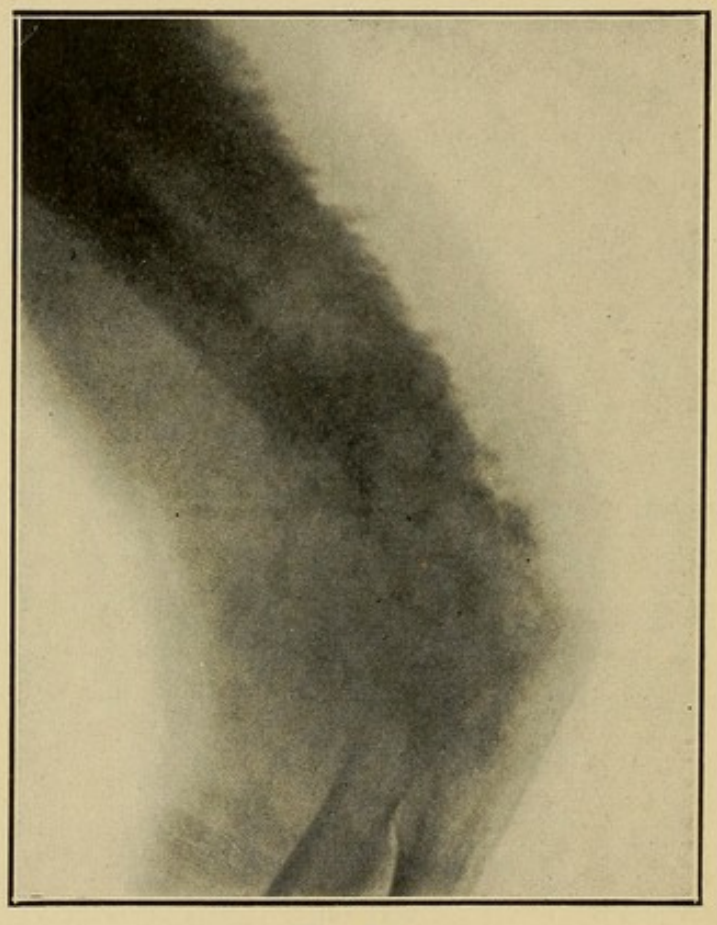

Fig. 73.

Acute suppurative osteomyelitis, in the stage of repair (Osgood).

The Roentgen rays in the earliest stages will probably show no characteristic changes. Later in the course of the disease, they are of great assistance in determining the extent of the destructive process. The dead bone is more permeable, and shows as dark areas on the plate.

The circumscribed form is not so severe as the diffuse. It also starts in the metaphysis, but does not spread up into the shaft. It may be rapid in its course, or very slow, lasting for years. The latter class is characterized by more or less pain and sensitiveness, which may be intermittent. The neighboring joint may show evidences of inflammation.

The Roentgen rays are one of the most valuable means of diagnosis in all these circumscribed cases. They will often show the cavity near the end of the bone, possibly with thickened walls.

DIAGNOSIS.

In the very early stages, suppurative osteomyelitis might be confused with a joint inflammation—"rheumatic" or other—but, while the patient is most eager to keep the limb quiet, motion in the neighboring joint is but slightly restricted. The edema of the limb is characteristic.

Tuberculous inflammation of the shaft marrow is very rare, and is decidedly slower in its course.

Syphilis, typhoid and gonorrheal marrow inflammations are not so destructive, nor so rapid, nor is the constitutional involvement so severe, as in suppurative myelitis.

If we will steer clear of the error of calling any deep-seated disease in the extremities "rheumatism" and dosing the patient with salicylates as a routine measure, we shall usually not be very long in reaching a correct diagnosis.

Avoid the Term Rheumatism

The thickened shaft, the discharging sinuses, etc., make the later stages quite plain.

PROGNOSIS.

This is usually good if the disease be recognized early, and if proper treatment be carried out, bad otherwise. Death may quickly result from septicemia. The mechanical obstruction to healing

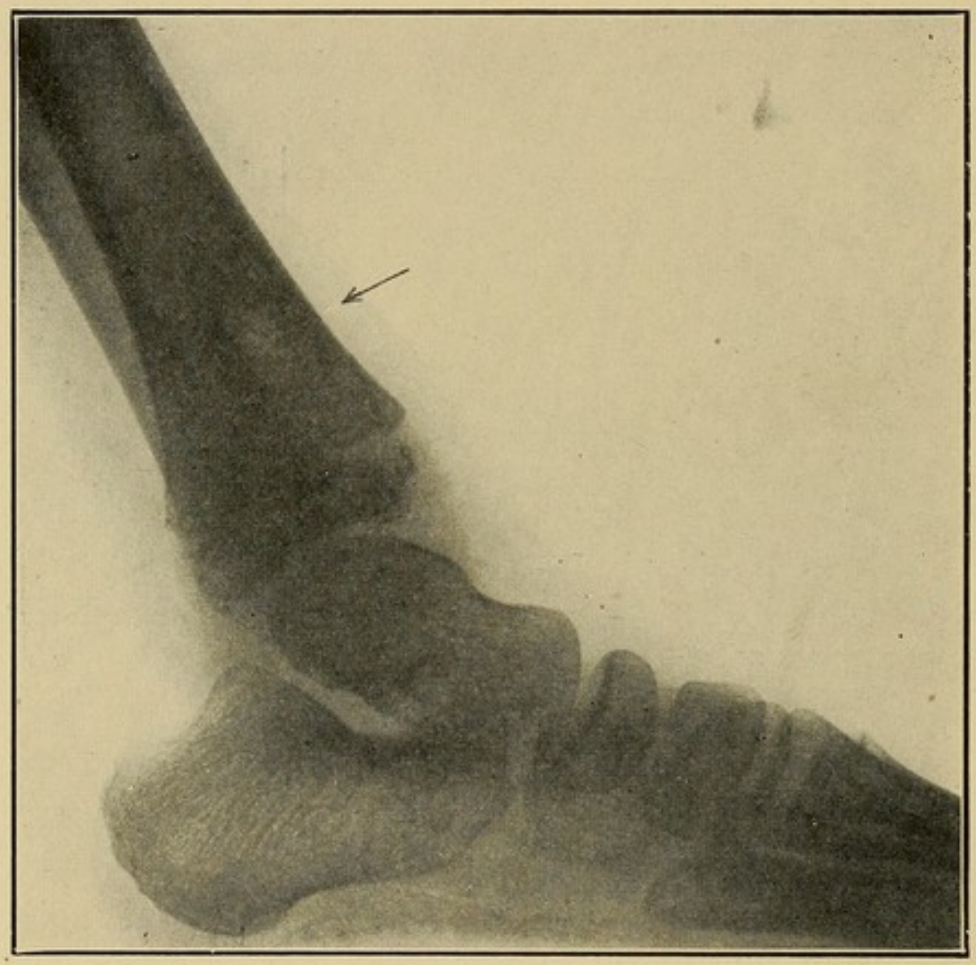

Fig. 74.

Suppurative osteomyelitis, circumscribed form (Osgood).

caused by the presence of dead bone or by Nature's unaided efforts to repair the damage, may result in a suppurating cavity that may persist for years. The dense eburnated bone walling off the diseased area from the rest of the shaft may prevent normal bone production, and the union of the two ends

of the bone. Two eburnated bone ends seldom if ever unite spontaneously, probably because they are practically without marrow. Nichols maintains that if the sequestrum be allowed to remain too long the periosteal bone is incapable of filling the gap.

TREATMENT.

For the circumscribed forms, incision and drainage constitute the best treatment.

Method of Operating

For the diffuse form incision and drainage are our first care also, but here we must remember that when we have incised the periosteum and have let out a collection of pus under it, our task, except in the most superficial cases, is only begun. We must follow the infection as far as it has gone, and must drain, not only the superficial bone, but the central canal. The periosteum should be split up as far as it is stripped from the bone. Wherever the periosteum is loosened we are justified in assuming the death of at least the superficial layer of the bone cortex.

The next step is to ascertain the condition of the marrow of the central canal. If this is infected, and it usually is, it must obviously be drained. With a hammer and chisel an opening is made. If pus be found, the opening is enlarged longitudinally until the limits of the disease are reached, by removing a strip of the cortex a quarter to a half an inch wide. Some authorities recommend scraping out the diseased marrow, some condemn the procedure. It is apparently unnecessary. The curette has little place in bone surgery. Bone scraping is, at best, a poor operation.

Theoretically the dead bone should be removed,

but most authorities condemn this procedure. Le Conte[1] maintains that if this operation be undertaken within 96 hours after the onset of the disease, it should be possible to remove all the dead bone and obviate absolutely the necessity of a later operation. His position seems quite logical, and his example is well worth following.

To guard against the too early closure of the wound, we sew the skin to the periosteum or to the deep fascia, pack the wound with sterile gauze and apply our dressing.

As a rule, the symptoms abate as soon as the pus has been evacuated, and after a few days the patient returns to a state approximating health.

When to Remove the Dead Bone

The almost universal rule has prevailed to abstain from further interference until nature has loosened the dead piece of bone, and has accomplished all that was possible, but Nichols[2] has demonstrated rather conclusively that this is an error. Not only does the dead shaft or fragment adhere to the periosteum in places for long periods, but also the presence of the large sequestrum causes, as we have seen, an eburnation of the ends of the "box" of new bone in which it eventually comes to lie. The new periosteal bone formation may be quite active, and may result in a thick shell at the circumference of the new shaft, but when the sequestrum is removed, the periosteum is quite unable to bridge in the gap in the interior, and a suppurating wound may remain for years.

For this reason Nichols recommends that the sequestrum be left in its place just long enough to

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[1] Le Conte, *Annals of Surgery*, 1912, LVI, 150.
[2] Nichols, Loc. cit.

provide for the laying down by the periosteum of sufficient new bone to hold the limb in shape after the sequestrum is removed. This period is roughly eight weeks in the case of a bone that has a companion parallel bone, such as the tibia and radius, and sixteen weeks in other bones. In the former class, the thickness of the new periosteal bone is determined, not only by radiograms, but by thrusting a needle through the periosteum near the opening of the sinus. If the bone crackles like an egg shell when the needle passes through it, it is strong enough to justify the removal of the sequestrum.

Through an incision in the periosteum the dead bone is laid bare along its entire extent. Dead bone is dead-white or dirty gray or yellowish, and does not bleed through its minute "pores" when the periosteum is stripped off. If the periosteum be adherent in places, it is separated, and the dead shaft or portion of shaft is removed, by grasping it with a pair of forceps and twisting it out. If it be still attached at its ends to the viable portion of the shaft, it may be chiseled away.

The divided edges of the periosteum are then sewn together for most of their extent, leaving an opening for drainage.

If one elects to follow the older methods, one allows enough time for the dead bone to become loosened—six months to a year—and then, chiseling a hole in the dense involucrum, inserts forceps, and twists the sequestrum out. As a rule the shaft re-forms, and the wound slowly heals.

The treatment of osteomyelitis following compound fractures consists in early incision and

drainage, followed some time later by the removal of the dead bone.

Bone Regeneration

When the shaft of a bone, or portion of the shaft, has been removed, it is replaced, as a rule, by growth upward and downward from the sound bone at its extremities, and by growth of the periosteal bone. This growth is slow and may require a year for its completion. Therefore as long as the *x*-rays show that it is actually going on, no need exists for interference. If the growth stop, before complete repair of the shaft, the gap may be bridged by grafting in a piece of bone, preferably a piece of another bone from the patient—autoplastic graft. It is necessary that the graft be inserted at each end into sound bone. If the ends of the receiving bone be eburnated, they must be trimmed off.

Bone Grafting

The employment of this graft in cases of nonunion following simple fracture is well worth a trial.

In the old osteomyelitis of the tibia Codman and Stone,[1] instead of taking an actual graft from another bone, transfer the fibula into the dead tibia, doing the operation in two stages, first implanting one end of the fibula, and when this has united, the other.

For filling in cavities in bone that will not heal, various expedients have been recommended, some of them on purely theoretical grounds. Small pieces of sponge, decalcified bone chips, aseptic blood clot, are some of the materials that are now rarely employed. Nichols' plan of subperiosteal

[1] Codman and Stone, *Annals of Surgery*, October, 1907.

resection of the affected portion of the shaft seems the most logical of all, making sure of the complete removal of all sclerosed or eburnated bone, in order that the marrow may have a chance to build up the vacancy.

Mosetig-Moorhof[1] advocates filling in these cavities with a mixture of iodoform 40 parts, and of oil of sesame and spermaceti 30 parts each. After the cavity has been made as aseptic as possible and perfectly dry, the iodoform mixture is heated to a temperature of 80 degrees centigrade and is poured in.

Bone-Wax

Some surgeons have had good results with this paste, others have not. Its effect is probably purely mechanical. The only specific effect of iodoform here or elsewhere is probably a bad smell, or in rare cases severe symptoms of poisoning.

To bismuth subnitrate paste a specific action has been also attributed, but here, also, an occasional case of bismuth poisoning has caused the adoption of a more harmless mixture. (See the section on joint tuberculosis.)

Other Pastes for Filling

A procedure which has met in some instances with success is the chiseling away the sharp, abrupt edges of the bone cavity, followed by nailing the skin to the bone.

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1 Mosetig-Moorhof, *Surgery, Gynecology and Obstetrics*, 1906, 111, 547.

CHAPTER VIII.

CHRONIC INFLAMMATIONS IN THE BONE SHAFTS

SYPHILITIC OSTEOMYELITIS AND PERIOSTEITIS.

Syphilitic bone lesions may occur in the hereditary form of the disease, and in the late secondary or in the tertiary stage of the acquired form. Certain of the manifestations are characteristic, and enable us to recognize the disease almost at a glance; others are not so characteristic. Here as elsewhere in the body, protean is the best adjective to describe the evidences of lues.

PATHOLOGY.

Syphilis may affect the central marrow, the marrow of the cortex, or the periosteum. Sometimes all three suffer. A frequent result of the morbid process is the formation of larger or smaller collections of a mucilaginous material and the local destruction of the bone. Around this collection of dead bone and mucilaginous material the bone may be sclerosed. Sometimes in the interior of the bone these collections may attain the size of a nut. When they lie near the surface they may become secondarily infected, forming abscesses, which later burst, and communicate with the surface. A common situation for them is immediately underneath the periosteum. The openings of the sinuses

Syphilis Affects Marrow and Periosteum

upon the surface are wont to be dark red, undermined and ragged, not pale and puffy as is the case with tuberculous sinuses. What gives to syphilitic disease of the shafts its peculiar and almost diagnostic appearance is the thickening of the periosteum—a production of new bone, sometimes

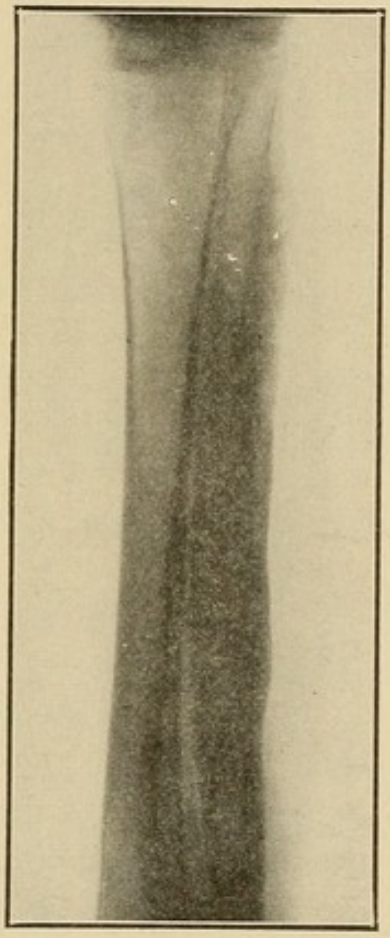

Fig. 75.
Syphilitic myelo-periosteitis, hereditary.

spindle shaped and sometimes more diffuse, and extending for a considerable distance. This bony thickening may be very painful. Especially in congenital syphilis the tibiæ become thickened and curved anteriorly, taking on a peculiar "saber shape."

Saber Tibiae

Another and rarer effect of congenital syphilis is an irregularity of the epiphyseal line. It is toothed and notched instead of being straight or slightly arched. Occasionally disease in the neighborhood of the epiphyseal line causes a dislocation of the epiphysis.

Syphilitic Dactylitis

Congenital syphilis is also responsible for the fairly frequent disease of the metacarpals and phalanges, and a condition results hardly to be distinguished from tuberculous dactylitis. Secondary infection and sinus formation is frequent in this syphilitic dactylitis, and a portion of the end of one or more of the finger bones may be destroyed. Owing to the thin covering of these bones, the skin over them is usually discolored when they are involved in a syphilitic process.

SYMPTOMATOLOGY.

Pain is wont to be a prominent feature, but it may be insignificant or absent. When present, it is usually worse at night. Usually a periosteal thickening is palpable, and is apparent in the skiagram. The Roentgen rays may show also areas of rarefaction in the shaft. When the lesion is located near the end of the bone it may show in the skiagram as if it had been bitten out. If the bone be superficial, reddening and infiltration of the skin are present, and often small fluctuating areas may be detected.

Fracture in Syphilitic Bones

Syphilitic sinuses have already been described. The course of a bone lesion in the limbs is almost always slow, and its appearance is sluggish. Other signs of lues should be sought for—bone lesions in other parts of the body, enlarged liver, spleen or

lymph nodes, etc. The Wassermann or Noguchi reaction will usually be present.

Fractures are said to occur occasionally in syphilitic bone disease. That they are not more frequent in the cases with localized areas of rarefaction is probably because of the thickening of the bone cortex.

DIAGNOSIS.

The diagnosis is sometimes easy, sometimes quite difficult. It may be made on the signs of syphilis elsewhere in the body, as well as upon the symptoms and skiagrams. The spindle-shaped enlargement is very suggestive when it is present.

Pus Osteomyelitis

An osteomyelitis due to pus germs is wont to be more acute in its course, and to be accompanied by more severe constitutional symptoms, though the slow, "sub-acute" cases may resemble bone syphilis. Sarcoma may sometimes occasion confusion, but the rarefaction of the bone as shown by the *x*-rays is very prominent in central sarcoma, and the production of new bone is insignificant. Periosteal sarcomata are often distinguishable from syphilis by the irregular growth at the site of the disease.

TREATMENT.

Avoid Operation

Our chief reliance should be on anti-syphilitic drugs—mercury, salvarsan and the iodides. Operation should be avoided if possible, though, if secondary infection have taken place, incision and drainage may be justifiable. If a sequestrum be present in such a case, it should be removed, but all bone-scraping operations are quite useless.

Secondary infection almost always makes the healing of the lesion much more tedious, and it

may be said in a general way that the longer the disease has lasted the slower will be the recovery.

GONOCOCCIC OSTEOMYELITIS.

Gonococcic osteomyelitis is generally considered to be rare. Gonorrheal periosteitis on the other hand is said to be fairly frequent in certain locations, especially under the calcaneus. In this situation spurs result from the irritation. Baer and others have obtained good results by chiseling off these bony spurs.

It should be remembered that in normal feet a spicule of bone often extends forward from the calcaneus at the attachment of the plantar fascia.

A peculiar condition of sensitive feet, found occasionally in patients with chronic gonorrhea, is probably due to a mild degree of periosteitis. The patients walk as if they were treading on eggs. The affection yields to strapping and to treatment of the urethra, as does often the more definite lesion mentioned above.

Typhoid Osteomyelitis

Typhoid Osteomyelitis or periosteitis is fairly frequent. It develops usually in the late stages of the disease, in convalescence, or after recovery. The typhoid bacillus alone may be the causal organism or it may be joined by ordinary pus cocci. Abscess formation is frequent. The bone is killed in greater or smaller pieces. The fistulæ remaining after the abscess has opened often persist for a long time.

The treatment is conservative where possible. If secondary infection occur, incision and drainage are necessary. Bone scraping here as elsewhere is quite useless, though the various expedients for healing up bone cavities may meet with success.

OSTEITIS DEFORMANS—PAGET'S DISEASE.

PAGET'S OSTEITIS DEFORMANS.

CHRONIC DEFORMING OSTEOMYELITIS.

The exact pathological process at the bottom of this disease is a matter of debate. It is usually considered to be a form of chronic inflammation

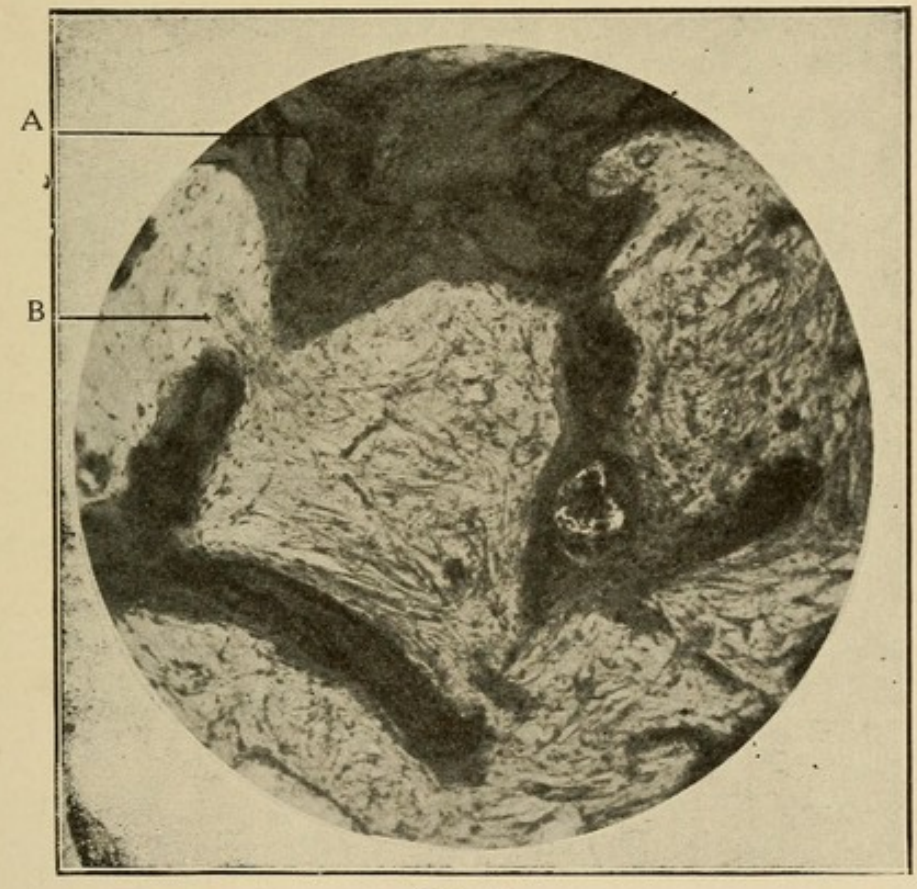

Fig. 76.

Paget's Deforming Osteomyelitis. Photomicrograph of section of femur near marrow canal. A. Osteoid tissue. B. connective tissue (Ellis).

of the bone, but as, according to the ideas already advanced, the bone itself is not subject to inflammation, we shall adopt the generally accepted view that the disease is essentially an inflammation, and

Probably a Chronic Osteomyelitis

shall class it with the chronic inflammations of the marrow, regarding it, that is, as a *chronic osteomyelitis*. Indeed, although some of the marrow changes resemble those of giant cell tumor (myeloma), the general pathological process often seems to bear a marked resemblance to that observed in bone syphilis.

The disease most often is observed after 40 years of age, and occurs about twice as frequently in men as in women.

Syphilis as a Factor

Etiology: This is unknown. The morbid changes bear a certain resemblance to those observed in syphilis, and certain cases are said to have reacted favorably to antisyphilitic treatment. From these facts (or theories) some observers maintain that syphilis is a causal factor. Paget, after whom the disease takes one of its names, denied this. Of eight of his cases traced to the end, five died of cancer. Some later writers have not been able to trace this relationship, or coincidence. "Trophic" disturbances, as in many other bone and joint diseases, have been held responsible.

Bartlett's case had a very large pituitary gland. The disease seems at times to manifest an hereditary nature. Disturbance of the thyroid has been observed in several cases. Since good authorities differ so radically, we shall not be criticised if we adopt the view that some obscure infection of the bone marrow and of the periosteum is responsible for the disease.

Paget's Description

Pathology: The gross pathological changes, as described in Paget's original article, are as follows:

"Periosteum not visibly changed. The outer surface of the walls of the bones irregularly and

finely nodular, as with external deposits or outgrowths of bone, deeply grooved with channels for the periosteal blood-vessels, finely but visibly perforated in every part for the transmission of the enlarged small blood-vessels. Everything seemed

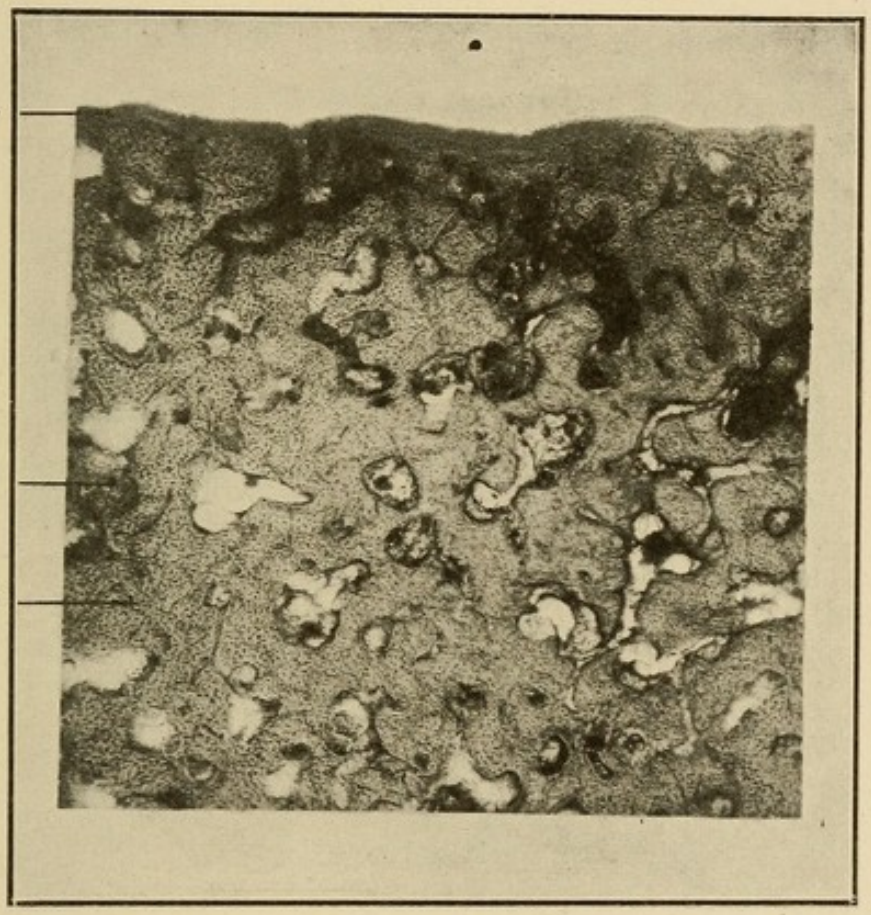

Fig. 77.

Paget's Osteomyelitis. Photomicrograph of the femur at the surface. A. Periosteum. B. Connective tissue containing giant cells. C. Cellular osteoid tissue (Ellis).

to indicate a greatly increased quantity of blood in the vessels of the bone.

"The medullary structures appeared to the naked eye as little changed as the periosteum. The medullary spaces were not encroached upon.

"The compact substance of the bones was in

every part increased in thickness. In the greater part of the walls of the shafts of the bones the whole construction of the bone was altered into a hard porous or finely reticulate substance like very fine coral. In some places there were small, ill-

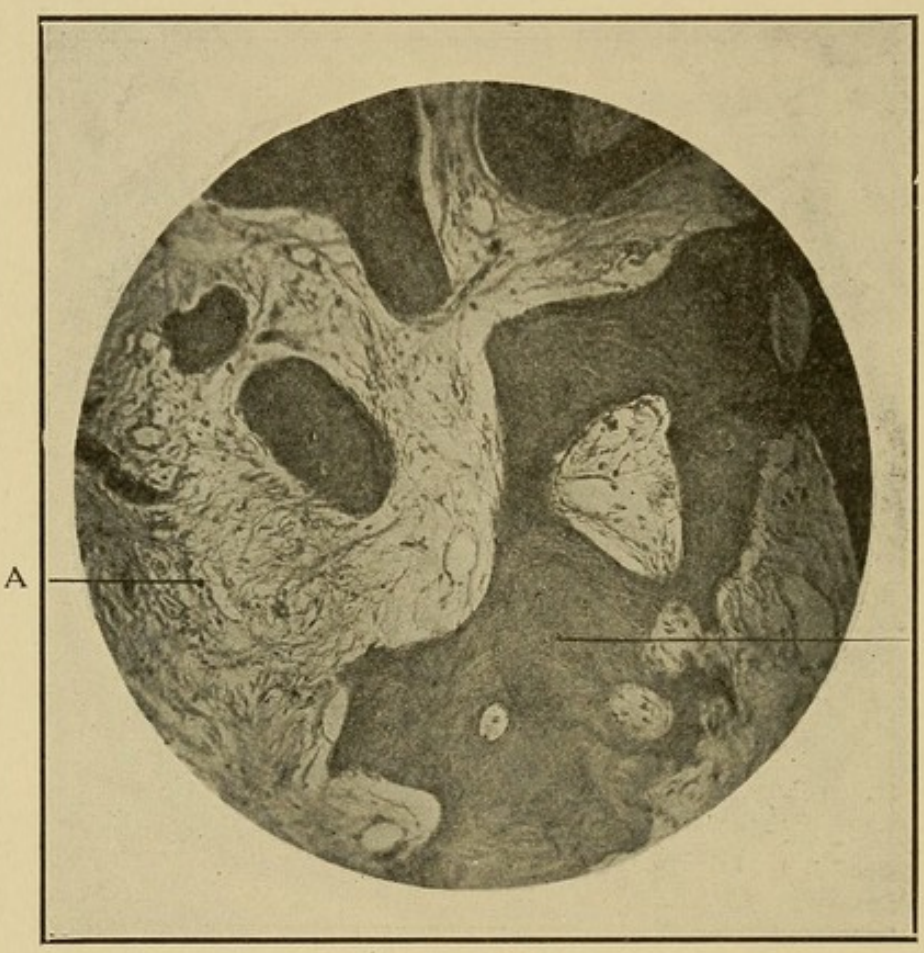

Fig. 78.

Photomicrograph of section of skull, near inner surface. A. Loose connective tissue. B. Osteoid tissue, slightly fibrillated (Ellis).

defined patches of pale, dense and hard bone, looking as solid as a brick.

"In the compact covering of the articular ends of the long bones the increase of thickness was due to encroachment of the cancellous texture, as if by filling its spaces with compact porous new-formed bone."

Packard, Steele and Kirkbride sum up the pathological changes thus: "Osteitis deformans is a disease affecting the skull, vertebrae, and certain of the long bones. Its essential pathological characteristics are:

"(a) Absorption of the compact substance causing enlargement and confluence of the Haversian canals.

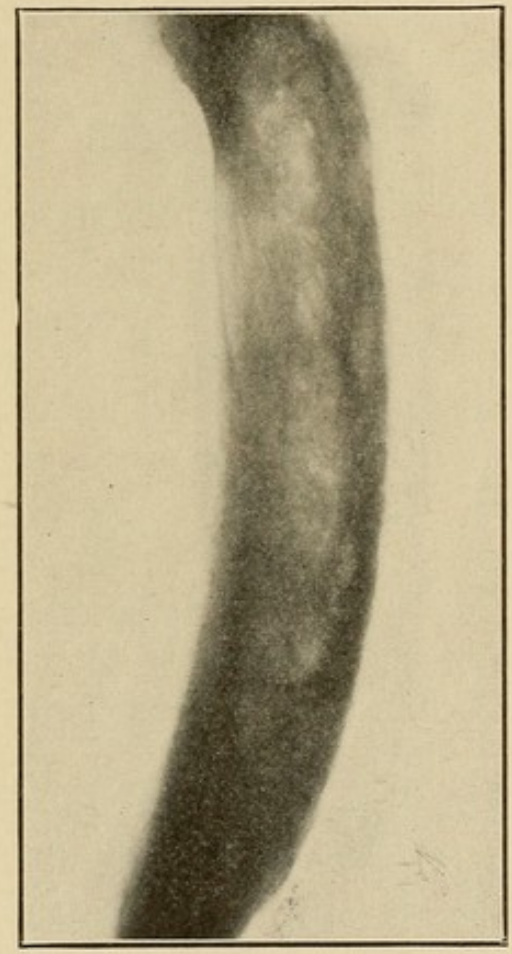

Fig. 79.

Skiagram of tibia in advanced case of Paget's deforming osteomyelitis. Note the rarefaction.

"(b) Formation of new bone which runs diffusely through the affected and the adjacent healthy portions. This new bone remains uncalcified, and is in turn reabsorbed.

"(c) The conversion of the medullary substance into a vascular connective tissue containing fat cells, giant cells, and leucocytes. In a small proportion of the reported cases cysts filled with gelatinous material and giant celled sarcomata occur in the medulla."

If (c) be placed before (a) and (b) the process can be better understood.

As a consequence of these three processes, the ordinary relations of the compact substance and medulla are destroyed. The bones become exceedingly thickened and asymmetrical, but since the new bone tissue remains uncalcified, its elasticity permits of great deformity of the long bones from the weight of the body, and fractures do not occur.

Ellis

Ellis says: "Essentially there is resorption of the bone and the production of an enormous excess of a substance which, probably from the lack of a better term, we designate fibro-osteoid tissue. Fibrous tissue, generally loose and cellular, in instances more dense, is a conspicuous figure. . . . The giant cells, with their basophilic protoplasm, are I think safely to be regarded as destructive in function."

Sypmtomatology: Paget's disease is usually slow and insidious in its onset, so that the time of its invasion can be set only approximately. Pain may or may not be present.

The disease manifests itself essentially by these physical signs: The legs and thighs are bowed outward, the former also forward. The lower extremities as a whole seem too large for the trunk. The spine possesses a long stiff anterior curvature, not unlike that of an ordinary spinal arthritis. The

skull is enlarged, the head carried forward, and the hips widened. The bones of the arms and forearms may be affected, as a rule not so severely nor so early as are the bones of the lower extremity. The skull and the tibiæ are usually the first bones to show the morbid changes.

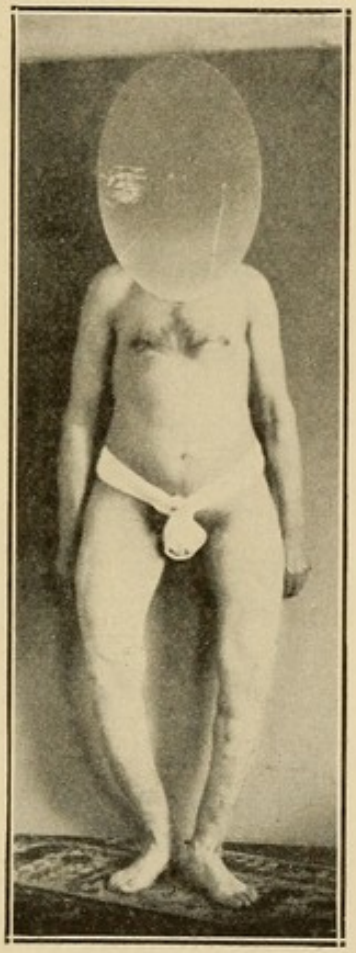

Fig. 80.
Paget's Osteomyelitis (Osgood).

Goldthwait, Painter and Osgood publish a skiagram of a case in which the bones of the forearm were affected. Some cases show a marked preponderance of changes in the left side of the body. Arteriosclerosis is a frequent accompaniment.

Roentgen rays show the thickened bones, the distortions, and often the rarefied bony areas.

Fractures Do Not Occur

Diagnosis: The identification of a typical case is usually easy. Milder cases may occasion confusion for a while. The saber tibiæ of congenital syphilis make their appearance in childhood. The peculiar changes in the other bones are not present in congenital syphilis.

The fractures which are so frequent in osteomalacia do not occur in Paget's disease, nor is there any production of new bone at the periphery in osteomalacia.

Prognosis: The disease may exist for a long time, but is considered incurable. Death may be due to some intercurrent disease, or, in a certain proportion of cases, to malignant growths.

Treatment: Treatment is palliative. So-called "antirheumatic" drugs are probably useless. Antisyphilitic drugs may be tried. Search for some source of infection might be worth while.

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RICKETS—RHACHITIS.

Rickets is a constitutional disease whose cause is unknown. It manifests itself largely by morbid

changes in certain of the lymphoid tissues of the body. Unhygienic surroundings predispose to it; faulty feeding and syphilis also stand in some sort of a causal relationship. Rickets is a disease of infancy and of early childhood. Fetal rickets is generally considered a misnomer. The existence of late rickets, that is, in adolescents, is still a matter of dispute. The disease is more common among certain races than among others, and in certain climates than in others. People from southern climates, transplanted to northern, seem especially affected, possibly from the fact that they live more indoors in their new home. Rickets is common in England and in Germany, and in our northern cities many cases are seen among Italians and negroes. It is less common in the country than in the city. Rickets has been held to be an infectious disease, but proof of this view is lacking. Possibly the marrow changes may be due to any one of a number of infectious agents, and the intestinal symptoms, so frequently observed, point to the intestinal canal as a possible source of infection. Changes in the long bones are among the most prominent evidences of rickets.

Occurrence

Rickets in Bone Is Possibly An Infectious Osteomyelitis

The marrow, according to Nichols, becomes a very vascular, myxomatous tissue. Islands of osteoid tissue are found in it. Kassowitz maintains that one of the earliest changes is a chronic inflammation in the marrow. The Haversian canals, and the so-called marrow spaces, are enlarged. The most marked changes take place in the region of the epiphyseal line. Masses of the abnormal marrow push in among the columns of cartilage cells in the zone of provisional calcifica-

Changes at the Epiphyseal Line

tion on the diaphyseal side of the epiphyseal line. The zone of provisional calcification disappears, and the epiphyseal line becomes irregular and broadened. On its diaphyseal side masses of osteoid tissue, instead of normal bone, are laid down. Islands of cartilage persist in this osteoid tissue. In other words ossification is halted. Calcification

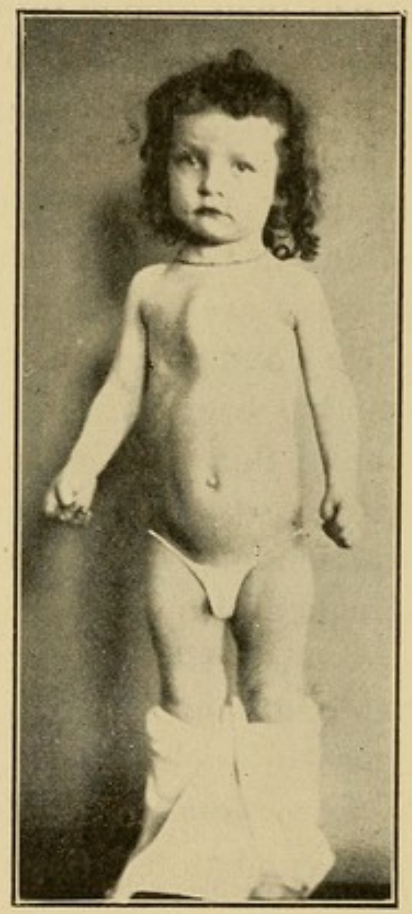

Fig. 81.
Rickets.

does not take place. Layers of osteoid tissue form on the trabeculæ of the diaphysis and of the metaphysis also, and the trabeculæ themselves may revert to osteoid tissue. Osteoclasis of the trabeculæ of the metaphysis and diaphysis takes place.

The periosteum is thickened, and its inner layer deposits layers of osteoid tissue instead of true bone. Rarefaction of the cortex may be carried on by the marrow cells. Thus the cortex may be thinned and weakened from within and from without.

Gross Changes

In gross the bones become soft and bent. They are broader in the region of their epiphyses. Fractures are fairly frequent. Vascular, spongy, soft, grayish-red masses of tissue are seen in the medulla and on the cortex. The epiphyseal line is broadened, and instead of being sharply defined and approximately straight, is irregular. Medullary and osteoid tissue extends into it. The central medullary canal is widened.

Eburnation

When rickets has run its course, calcification of the osteoid tissues takes place, and the bones become denser than normal—sclerosed. The deformities may persist or they may disappear completely.

Deformities of Rickets

Symptomatology: The bones of the extremities become bent and distorted, and thickened in the region of their epiphyses. Bow-legs and knock-knees are extremely common. The fontanelles remain open abnormally long, the head is large and square, the forehead prominent, the bones of the skull are soft. Teething is late, and the child does not begin to walk until after the usual time. Sweating, especially about the back of the head, is profuse. The sternum is often prominent, the chest flattened laterally, and knobs form at the junction of the ribs with their cartilages—the "rhachitic rosary." The spine often possesses a long, rounded, posterior curve, occasionally a more or

General Appearance

less angular bend. The abdomen is prominent, the spleen is frequently enlarged. Anemia is always a symptom, probably on account of interference with the blood-forming function of the marrow. The patients are pasty-looking and weak. Gastro-intestinal symptoms are wont to be marked, convulsions and attacks of false croup occur in many

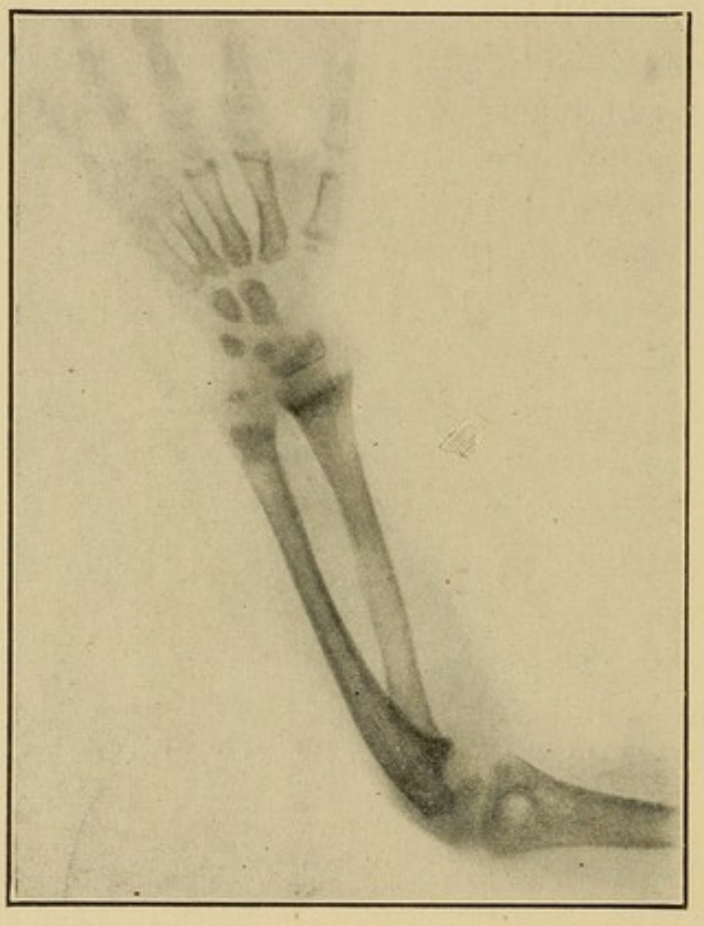

Fig. 82.

Rickets. Skiagram of hand and forearm.

cases. Pain and sensitiveness of the epiphyses are often observed.

DIAGNOSIS.

The milder cases may occasion some difficulty, but the severer ones can be recognized at a glance. Scurvy has its acute, exquisitely painful swellings

in the neighborhood of the joints, and its hemorrhages into the gums and elsewhere. A mild degree of chronic hydrocephalus produces a change in the skull slightly resembling that of rickets, but it, like scurvy, lacks all the constitutional symptoms of the latter. Osteopsathyrosis shows evidences of numerous fractures.

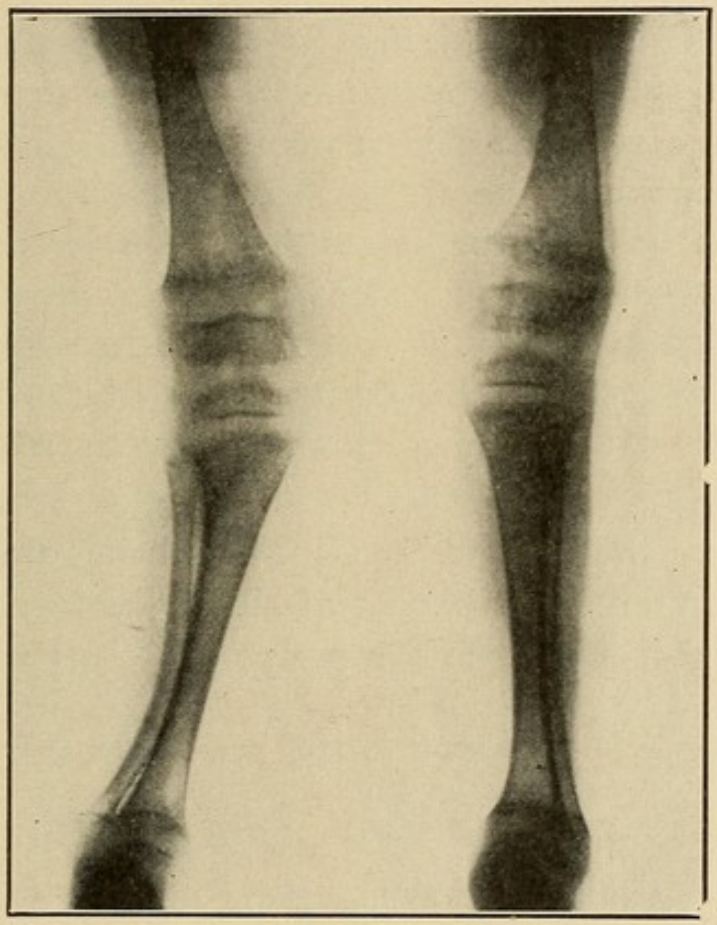

Fig. 83.

Rickets. Skiagram of lower extremities (Osgood).

PROGNOSIS.

Rickets itself involves little danger to life, but children who suffer with it are prone to intercurrent disease, and their mortality is rather high.

TREATMENT.

Treatment Constitutional and Local

With our present knowledge, the treatment is largely constitutional. Attention to the diet, and plenty of fresh air are essential. Cod liver oil has been praised. The lime preparations are probably not of much value. A child with severe rickets should be kept off his feet as much as possible, in order to prevent deformity. Braces and manipulations are very valuable for curing the deformities, as long as the bones are soft—roughly up to 4 years of age. When the bones become sclerosed, all apparatus is useless. For the severe deformities operation is usually necessary, but never while the bones are still soft. The tendency of the lighter degrees of deformity to spontaneous recovery should be remembered.

(Since this was written Jos. Koch has produced the essential bone changes of rickets in dogs by injection of cultures of "streptococcus longus.")

OSTEOPSATHYROSIS IDIOPATHICA.

FRAGILITAS OSSIUM, OSTEOGENESIS IMPERFECTA.

Osteopsathyrosis means bone fragility. It is observed in a number of different diseases, notably in malignant growths, osteomalacia and tabes. Osteopsathyrosis idiopathica is the name given to a rare condition (it can hardly be called a disease) in early life, characterized by the occurrence of multiple fractures, especially of the long bones. Its essential nature is still unknown, as is its cause. In some instances the bones are fractured in intrauterine life. The patients are almost invariably quite young. If they reach mature age the condi-

tion disappears.[1] Many of the patients are still-born, others die at an early age. An hereditary element has been observed. The fractures take place from the least possible force, sometimes apparently without cause—spontaneously. They are sometimes many in number. Chaussier counted 113 in a new-born child.

PATHOLOGY.

Absence of True Bone Formation

The characteristic of the condition is a general deficiency in the formation of bone. The cortex of the long bones often consists of a series of platelets instead of a definite continuous layer. The laying down of new bone is deficient both in the diaphysis and in the metaphysis. The details of the process are differently described by different authors. The epiphyseal cartilage is normal, in contradistinction to chondrodystrophia fœtalis. Aseptic necrosis of the new-formed trabeculæ has been observed. If this observation is correct, the condition consists not only in a lack of formation of new bone, but also in a destruction of bone already formed. The Haversian canals may be widened to form large open spaces.

Marrow Disease the Chief Factor

The abnormal bone formation is probably due to disease of the marrow and of the deep layer of the periosteum. In Lovett and Nichols' case the marrow near the epiphyseal line and in the cortex was changed to an "edematous, myxomatous, connective tissue." Döring observed a fibrous degeneration of the marrow in his case, and inflammatory changes in the neighbourhood of the necrotic bone.

[1] In Biggs' case the disease appeared at the age of 20, and ceased at 30.

SYMPTOMATOLOGY.

The chief clinical manifestations of the disease are the abnormal friability of the bones and the multiple fractures. The fractures may occur *in utero* or after birth. Callus can be felt at the location of the older ones. The skull is wont to be

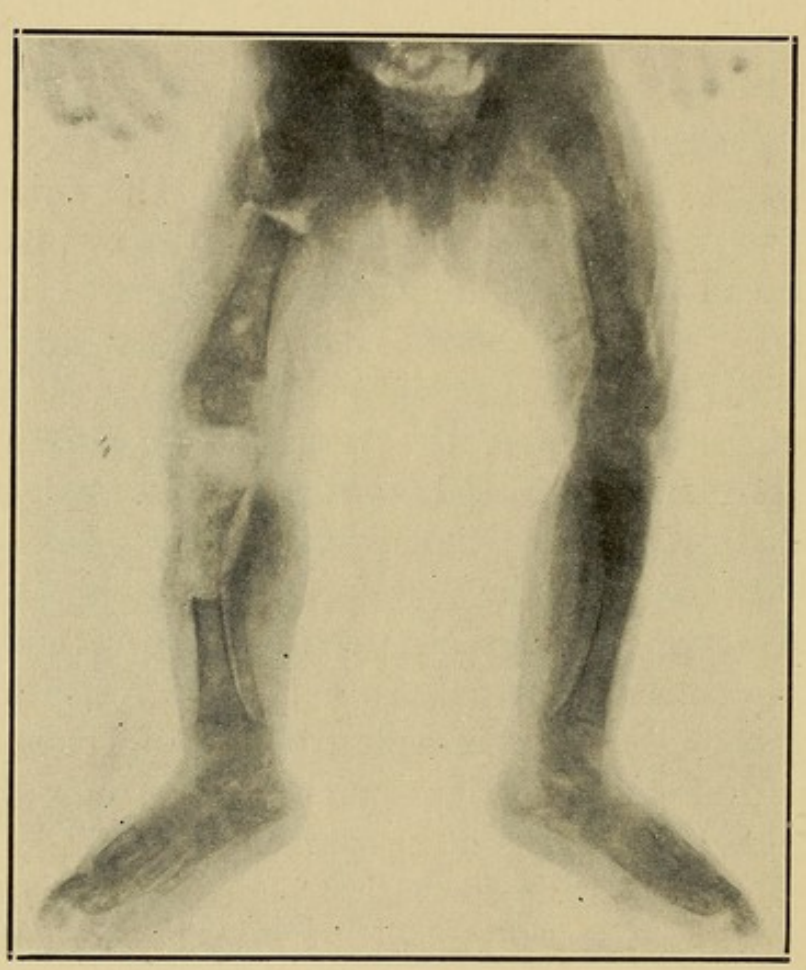

Fig. 84.

Osteopsathyrosis Idiopathica. Skiagram of lower extremities (Osgood).

large, and the fontanelles remain open until very late. Teething is also much retarded. According to Nathan the skin and subcutaneous tissues are thickened, giving an appearance similar to that of chondrodystrophia.

The diagnosis is made on the multiple fractures,

occurring usually in a new-born baby or in a young child.

The only known treatment is to keep the child as carefully as possible, with the idea of preventing further fractures. Treatment

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OSTEOMALACIA.

Osteomalacia means a softness of bone. It is a convenient term, but rather an unfortunate one. The disease in its pathological manifestations is somewhat akin to Paget's deforming osteomyelitis, but without the cortical bone production of the latter. In certain features it also resembles rickets and osteopsathyrosis, but these affect children's bones, while osteomalacia is essentially a disease of adult life.

Etiology: This is unknown. The disease occurs more often in certain districts than in others, and about nine times more often in women than in men. Frequent child-bearing seems to predispose. The early changes in the marrow point to an obscure infection as a possible cause.

PATHOLOGY.

The essential lesion in osteomalacia is an inflammation and degeneration of the red and of the yel-

Marrow Changes Chief Factor here also

low marrow. In the early stages hemorrhages into the marrow are said to take place, which leave behind collections of pigment. Liquefaction and cyst formation are later observed. Decalcification and then disappearance of the bone result. The bones soften, so that they can easily be cut or broken.

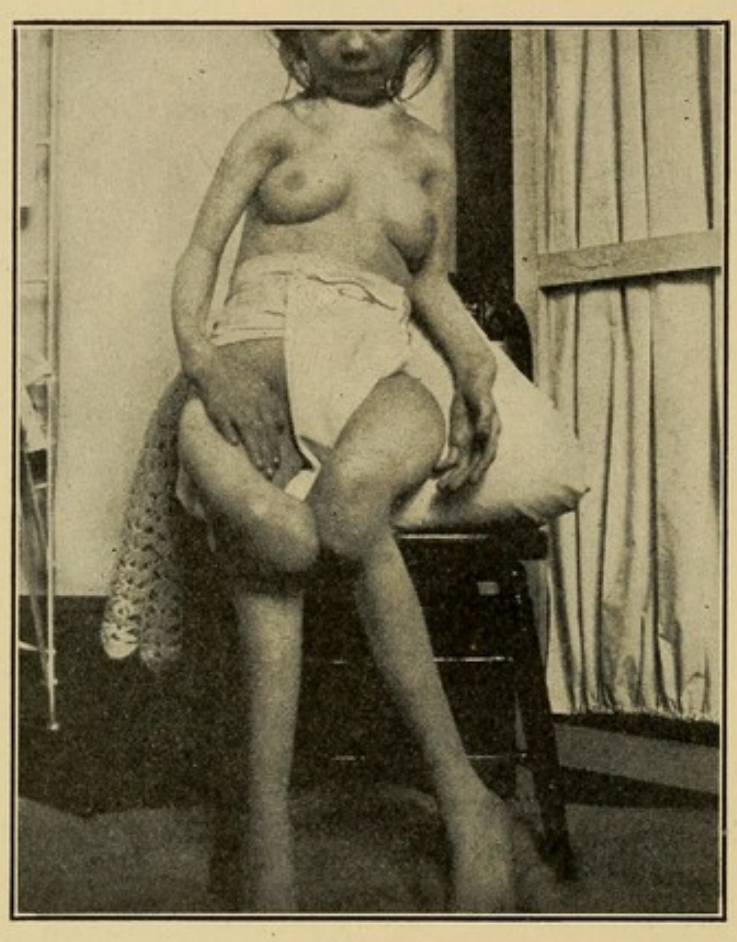

Fig. 85.
Osteomalacia.

Spontaneous fractures occur. In the later stages of the severe cases the shaft of a long bone may disappear entirely, leaving only the periosteum filled with broken-down material. No true compensatory bone production takes place in osteomalacia. The attempt by nature to produce bone after spontaneous fracture, results only in osteoid

tissue. The bones of the head and face suffer little if at all.

In rare cases the disease in the marrow subsides, the bone-forming function returns, and the patient recovers.

Symptomatology: The course of the disease is

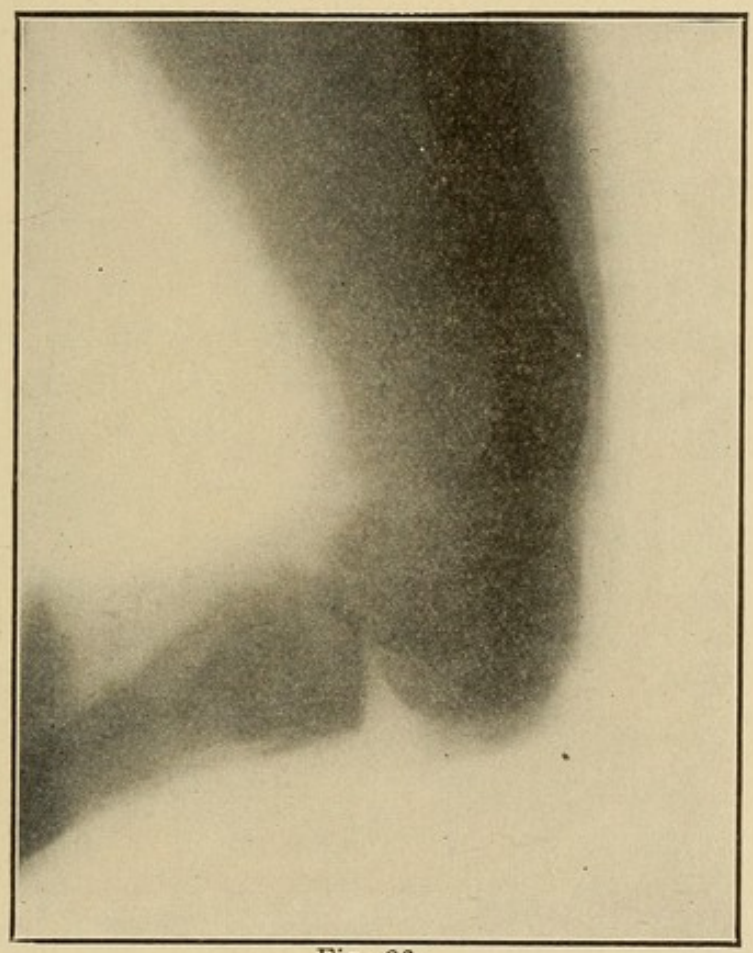

Fig. 86.

Skiagram of osteomalacic bone (Osgood).

usually slow. Pain may be present or absent. The bones of the lower limbs, as they bend, are unable to support the body, and walking becomes at first waddling and difficult, then impossible. The various deformities caused by the bending of the bones, and by their fracture, appear. Death may

take place from exhaustion, or from complicating pulmonary disease.

Treatment Unsatisfactory

Treatment is often fruitless. Pregnancy must not be permitted. In some instances removal of the ovaries and tubes has been followed by recovery.

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CHAPTER IX.

NEW GROWTHS IN BONE.

OSTEOMA.

An osteoma is a tumor which consists of bone tissue. Osteomata usually occur on the skeletal bones, but may occur in other tissues. A small osteoma arising from a bone is called an *osteophyte,* a larger one an *extosis*. Extoses may be single or multiple. In the latter case they are usually situated on the ends of the long bones, or at the insertion of tendons, or in both localities. At times they seem hereditary in their nature. Sometimes cartilage is mixed with bone in an osteoma—osteochondroma. An extosis may be covered by the periosteum alone, or by a layer of cartilage in addition—exostosis cartilaginea.

Cartilaginous Tumors

Cartilaginous tumors occur most often in the bones of the hand and on the extremities of the long bones. Occasionally they are found in the medullary canal.

The only treatment for a bony or cartilaginous tumor is removal. Recurrence is fairly frequent, especially if removal be not complete, but these tumors are not malignant.

NEW GROWTHS OF THE MARROW AND PERIOSTEUM.

These may be either benign or malignant. The chief member of the former class is the so-called

benign myeloma. The latter class includes the various types of sarcomata and carcinoma.

BENIGN MYELOMA.

Synonyms: Giant Cell Sarcoma, Giant Cell Myeloma.

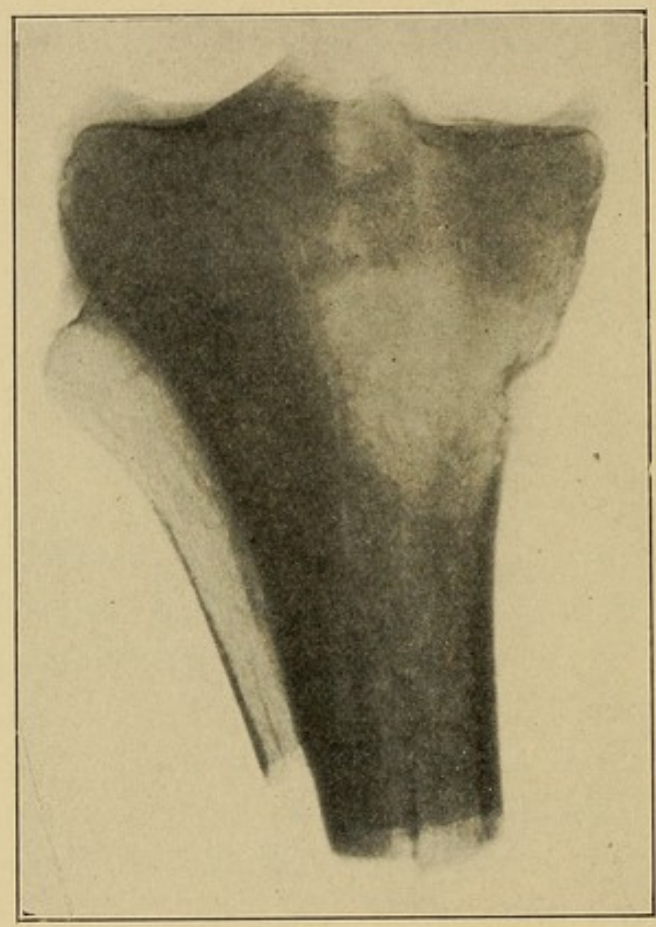

Fig. 87. Myeloma of Upper Metaphysis of Tibia—Mathews.

Until recently regarded almost universally as a variety of sorcoma, and treated as such, this new growth is regarded by most modern authorities as distinctly a benign growth, with a tendency to local recurrence if not thoroughly removed, but possessing no other attributes of malignancy.* Unfortu-

* J. Clark Stewart (Surgery, Gynecology and Obstetrics, July, 1913), maintains, however, that these growths are really giant cell sarcomata, and that if they break through into the surrounding tissues they possess all the elements of malignancy.

nately the diagnosis of its nature is somewhat difficult, and consequently its treatment generally is still that of more malignant growths, namely, amputation of the limb.

As to the cause, nothing definite is known. Trauma, as with practically every other bone and joint disease, has been held responsible in certain cases.

Trauma

Occurrence. According to Bloodgood myelomata occur usually between the ages of 20 and 60. Mathews maintains that most of the patients are below 25. The favorite site of growths is in the metaphysis of the long bones.

Pathology. The tumor is essentially a new growth in the marrow, consisting of spindle and round cells, with a stroma of connective tissue. In addition are found great numbers of large multinucleated cells, the so-called giant cells which are responsible for the common designation of the tumor. The blood supply is very free, and hemorrhages often take place into the substance of the growth. Spreading throughout the marrow the new-formed tissue causes a destruction and absorption of the bone, and more or less of an expansion of it. The growth is usually rather circumscribed, does not break through the periosteum until its late stages, and does not form metastases.

Symptomatology. Benign myeloma usually appears as a more or less sensitive swelling in the metaphysis of the long bones, especially at the upper end of the tibia, and at the lower end of the femur, the radius or the ulna. Pain may or may not be severe. Joint symptoms are usually absent. The bone shell expands over the growth, but there is

little if any production of new bone. The tumor may increase in size rapidly or slowly. "Egg-shell crackling" may be present over it.

The Roentgen rays show a rarefied area in the metaphysis, covered over as a rule by a bulging shell of bone, and this bulging growth is wont to

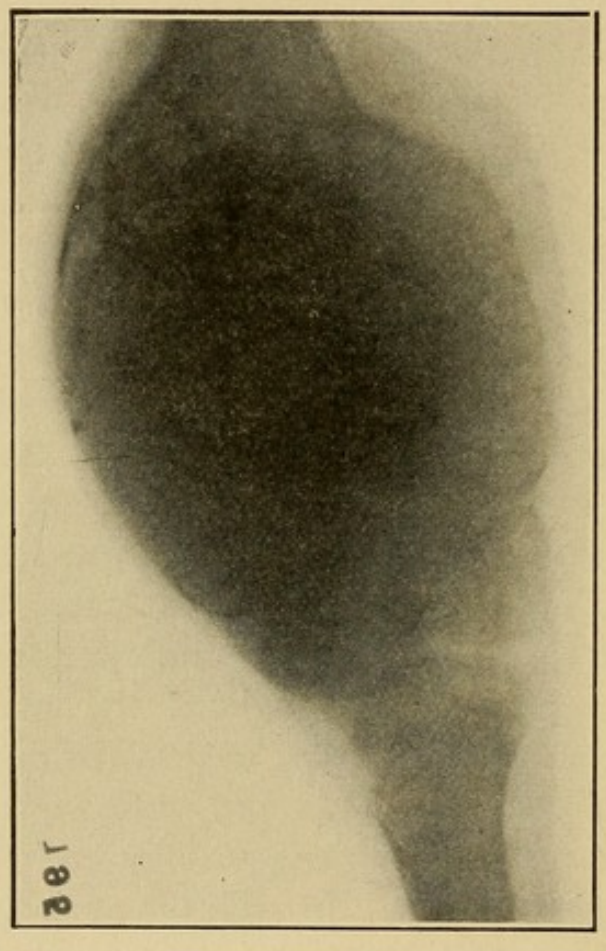

Fig. 88. Myeloma of Lower End of Femur—Mathews.

be circumscribed and well defined. The bony and periosteal shell usually persists. The myeloma usually does not burst through it as does a bone sarcoma, to invade the surrounding tissues. Blood-good says that the picture cannot be absolutely distinguished from that of a bone cyst.

Diagnosis. A rather slowly growing single bone tumor with the appearance and signs described above, occurring in the metaphysis, is usually a benign myeloma. According to Bloodgood the positive diagnosis from other growths cannot be made

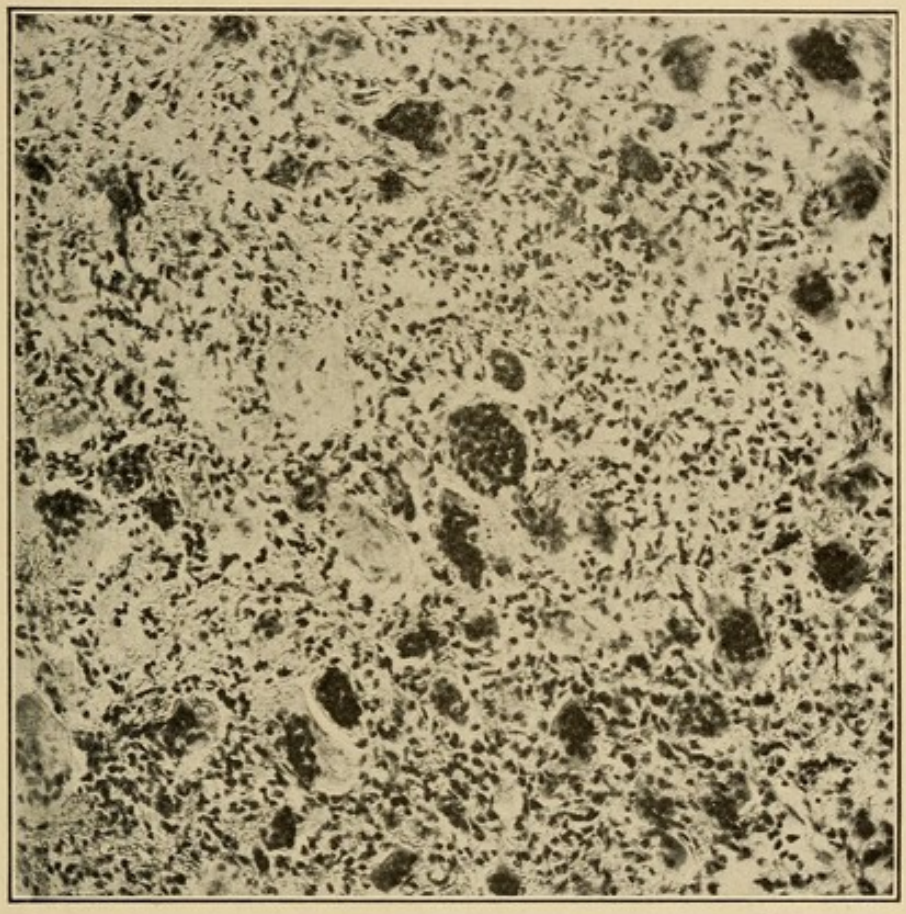

Fig. 89. Photomicrograph of Myeloma—Mathews.

until operation. Then the appearance is characteristic. I quote Bloodgood's words:

"It differs from the periosteal and medullary sarcomata of the spindle, round, or mixed cell type by the color. It is distinctly vascular; it resembles to a certain extent young granulation tissue. The appearance can be well described as that of currant jelly, and I find this term employed by

Currant Jelly

many others in the literature. Mixed with the red areas, there may be white areas, and areas mottled, white and red. The second characteristic feature is the consistency of the tumor. There seems to be a complete absence of any supporting stroma. It can be broken up with the finger or curette into

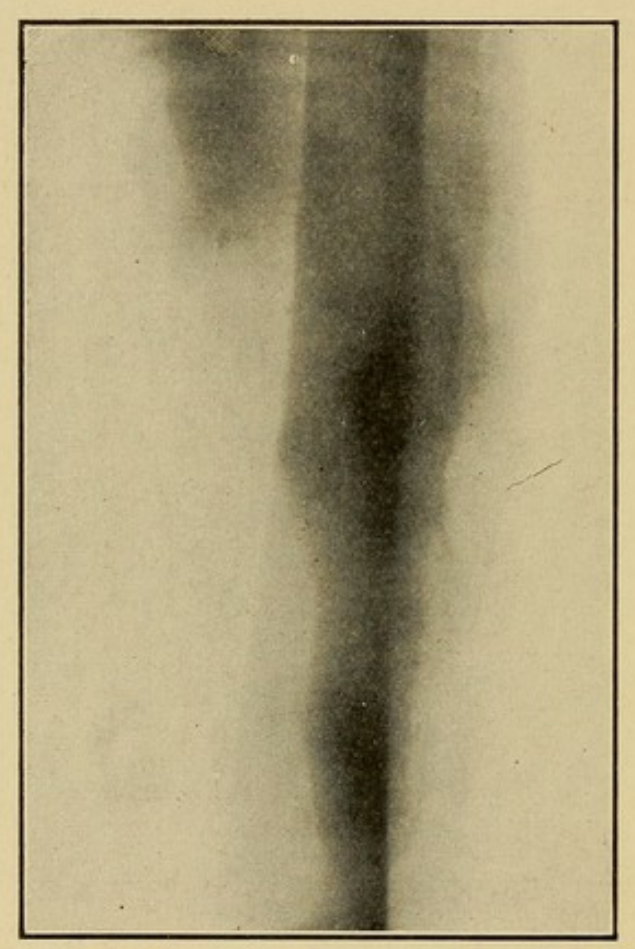

Fig. 40. Round Cell Periosteal Sarcoma of Lower End of Femur—Osgood.

small pieces, and one sees not even the finest connective tissue holding the pieces together, yet it does not break up into finely granular masses characteristic of the very cellular round-cell sarcoma. The consistency resembles "Schmierkaese." In a few instances the white areas are quite firm and fibrous. Then we have an admixture of

osteitis fibrosa . . . The giant cell sarcoma tissue can be removed from its bony shell as easily as the connective tissue lining of a bone cyst."

Frozen Section

If any doubt exists, a frozen section will show the characteristic giant cells in large numbers.

Syphilis is often multilocular in its bone manifestations, shows the other signs of syphilis, and a combination of rarefaction and bony proliferation. The "cavity" in the metaphysis, with its well-defined and thin bone shell is not present in syphilis.

Tuberculosis should occasion no difficulty, if one avoids the error of regarding every disease in the bone end as tuberculosis.

Treatment. Under artificial ischemia the myeloma must be opened and *every bit of the diseased tissue must be removed with a curette.* Bloodgood insists on the importance of swabbing out the cavity with carbolic acid followed by alcohol. In more extensive cases a resection may be necessary, subperiosteal or other. If the tumor returns, as it often does, the operation may be repeated several times if necessary. Bone grafting may be done to fill in the defect. Amputation should be done only as a last resort.

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SARCOMA.

Sarcoma of the long bones is usually of the spindle cell or round cell variety, and takes it origin from the marrow or the periosteum. It may start

in the metaphysis or in the shaft, possibly in rare instances in the epiphysis. Its cause is unknown.

Morbid Anatomy. The growth presents essentially the same characteristics as does sarcoma in any other region of the body, but it is modified by its bony environment. When the sarcoma starts in the marrow, it causes an absorption of the bone in the neighborhood, and spreads in all directions.

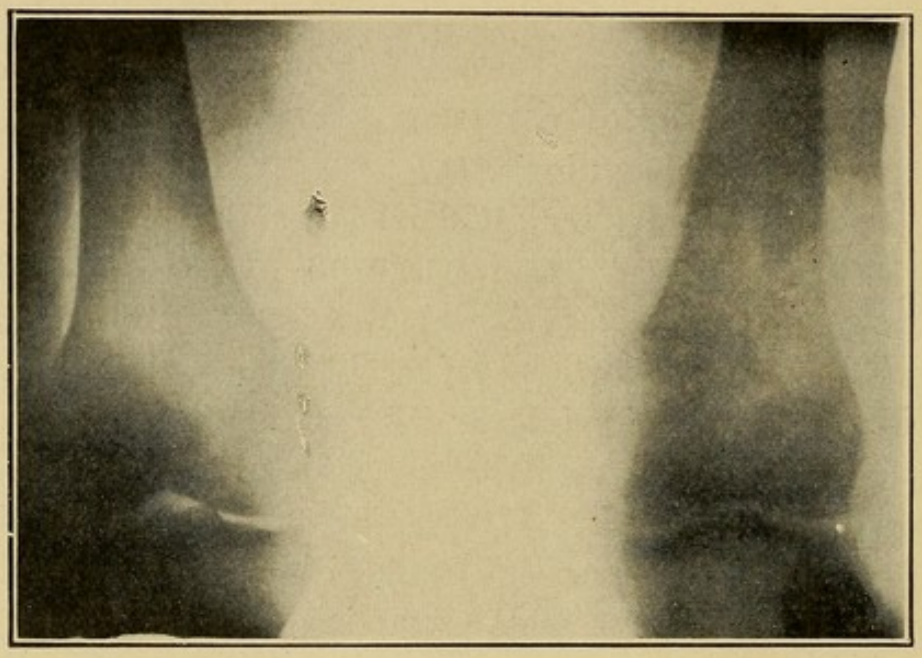

Fig. 91. Sarcoma of Shaft of Femur—Osgood.

The periosteum may proliferate, and lay down a shell of new bone at the periphery. Then the growth is said to be "expansile." As times goes on, this new bone is perforated and destroyed, and the sarcoma invades the surrounding tissues. Pathological fracture is common.

In the tumor tissue itself retrogressive changes are common, e. g., hemorrhages and cysts. Occasionally masses of cartilage and bone are formed—chondro-sarcoma, osteo-sarcoma.

A sarcoma starting in the periosteum may spread through the Haversian canals into the marrow, and, in the later stages of the disease the origin of the

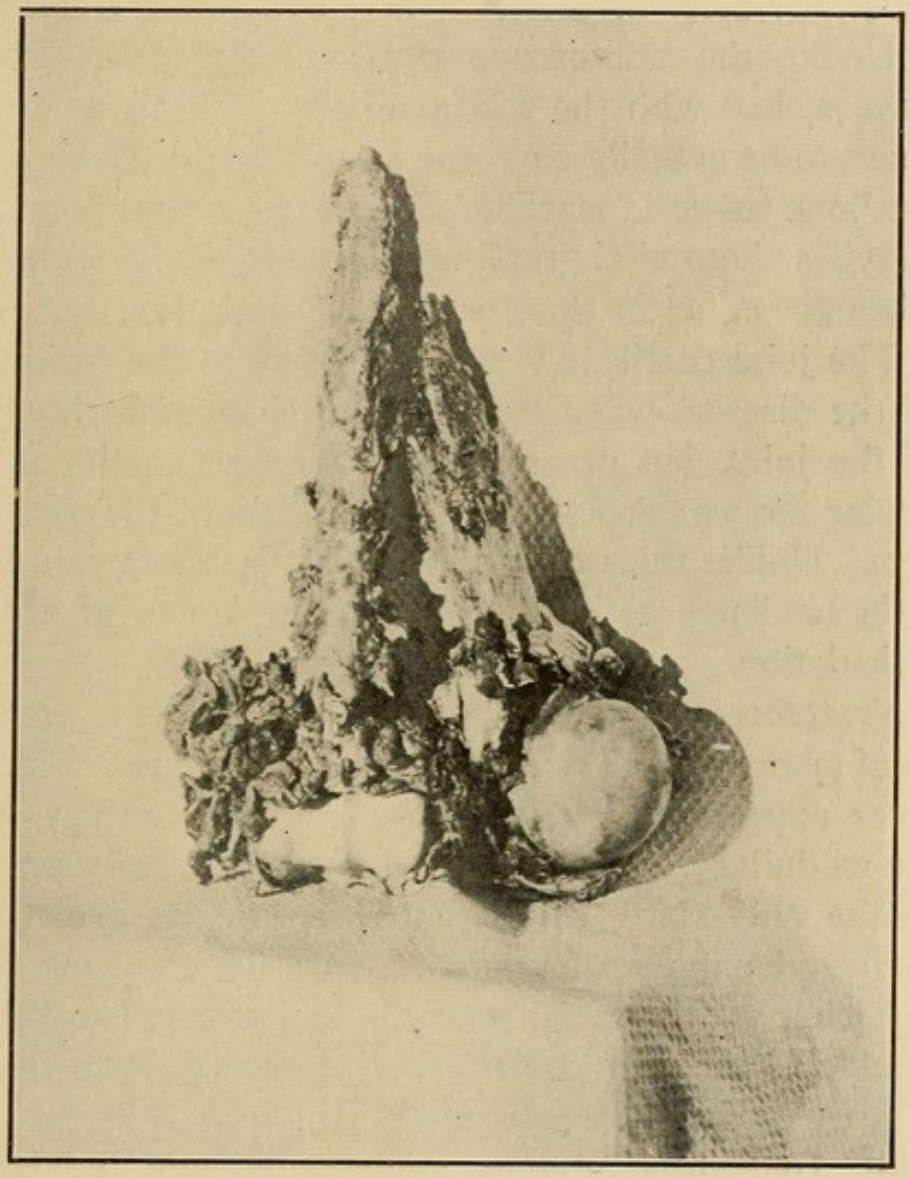

Fig. 92. Preparation showing pathological fracture of shaft of femur in an adult male. The only premonitory symptoms had been vague "rheumatic" pains. The fracture occurred while the patient was walking in the street. Amputation through shoulder joint.

growth often cannot be determined. Nichols holds that new bone formation is common in the periosteal sarcoma, Lexer holds the contrary. Invasion of the surrounding tissues is fairly early.

Wherever the growth starts, tendency to the destruction of the entire bone in its vicinity is marked. The progress is wont to be fairly rapid, especially after invasion of the surrounding tissues.

Under the microscope the marrow spaces are seen packed with the sarcoma cells. The bone appears to be actually consumed. Aseptic necrosis of the bone tissue is marked. Reference to the accompanying photomicrograph will serve to explain what takes place, better than pages of description.

Joint Cartilage Resistant

The joint cartilage is most resistant to the spread of the disease, when it occurs in the neighborhood of the joint, but occasionally extensions to the articular tissues takes place at the margin of the cartilage. Unlike tuberculosis, however, sarcoma shows little tendency to involve the other bones of the articulation.

Symptomatology. Pain and swelling are the chief symptoms of sarcoma of the long bones. The latter appears earlier in the periosteal type than in the medullary. A vague history of local pain may be the only thing to call attention to the growth until suddenly a spontaneous fracture awakens a suspicion of its true nature. A "parchment-like crackle" has been described as palpable over the growth in some instances. Bone sarcoma is seen most frequently among adolescents and young adults.

Parchment Crackle

Spontaneous Fracture

The Roentgen rays show a rarefaction of the bone, and sometimes a proliferation of bone at the circumference.

Diagnosis. Suppurative osteomyelitis is usually accompanied by marked constitutional disturbances and is rapid in its course. The circumscribed forms

may lack these characteristics, but their very circumscribed nature itself will differentiate them from sarcoma.

Bone syphilis may imitate sarcoma closely, not only in its symptoms but also in the Roentgen picture. Other signs of syphilis, and the reaction to anti-syphilitic treatment will clear up the doubt.

Spontaneous fracture occurs quite frequently in the osteopathy of tabes dorsalis. A search for the

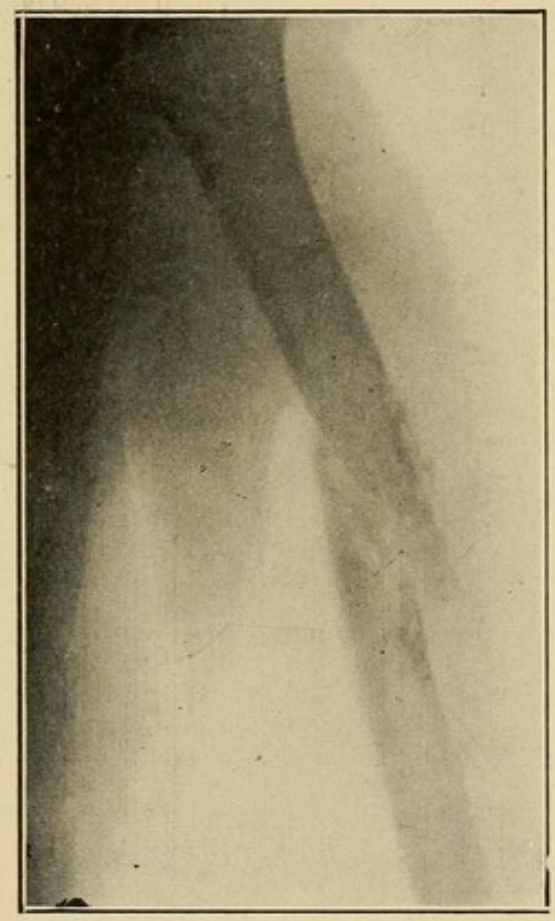

Fig. 93. Skiagram of bone shown in Fig. 6. Diagnosis, sarcoma.

symptoms and physical signs of this disease will make the nature of the process evident. The bone lesions of tabes are almost invariably painless.

Benign Myeloma. From the fact that this disease

has been included hitherto with sarcoma, the differential diagnosis is still somewhat obscure. It usually occurs in the metaphysics of the long bones. "Egg-shell crackling" is frequent over it. It is probably slower in its growth than is sarcoma, and does not break through into the surrounding tissues

Fig. 95. Photomicrograph of portion of shaft of bone shown in Figs. 6 and 7. A typical round cell sarcoma. Note the multitude of small round cells replacing the normal marrow, and the marked absorption of the bone. The occurrence of fracture can be understood easily.

until very late if at all. The diagnosis may be impossible until the time of operation. Then the currant jelly appearance, possibly mottled with lighter areas, and the "Schmierkäse" consistency will point to the nature of the growth. If any doubt remains,

the examination of a frozen section will reveal large numbers of giant cells.

Prognosis. This is bad, doubly so if involvement of the surrounding tissues have taken place. Metastases almost invariably occur under any form of treatment.

Treatment. The best treatment probably consists of amputation. The operation should be not an amputation through the affected bone, but a disarticulation above it.

CARCINOMA.

Carcinoma in bone occurs only as a metastasis of the disease in some other organ. It affects the marrow primarily, and causes a destruction of the bone in much the same manner as does sarcoma. Pain, swelling, and fractures are prominent features of the disease.

MULTIPLE MYELOMA.

This is a rare disease which causes multiple tumors in the bone. Albumose is found in the urine. The outcome is invariably fatal.

COCCIDIOIDAL GRANULOMA* "BLASTOMYCOSIS."

Oidium Coccidioides, a fungus, very rare in its bone localization, causes in the bone marrow an infectious granuloma, with endothelial hyperplasia, breaking down of tissue, and cold abscess formation. The pus burrows, forming sinuses. The

* Rixford and Gilchrist, Johns Hopkins Hospital Reports, 1896, I, 209. Ophuls, Journal of the A. M. A., 1905, XLV, 1291. Whitman, Journal of Infectious Diseases, 1913, XIII, 85.

lesions much resemble those of tuberculosis, and the disease in the bone is usually diagnosed tuberculosis. The correct diagnosis is usually made by finding in the pus large, doubly-refracting yeast bodies.

Coccidioidal Granuloma when occurring in the bones is fatal. No known treatment is of much avail. Iodide of postassium in one case is said to have effected a cure.

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