

LEWIS'
PRACTICE
OF
SURGERY

WALTMAN WALTERS

BONES AND JOINTS (Cont'd)
MUSCLES, TENDONS
NERVES
BRACHIAL PALSY
SYMPATHETIC NERVES
LYMPHATICS
AMPUTATIONS



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LEWIS' PRACTICE OF SURGERY

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CHAPTER ONE

TUBERCULOSIS OF THE BONES AND JOINTS

C. LESLIE MITCHELL, M.D.

Tuberculosis of the bones and joints is a local manifestation of a constitutional disease caused by the *Bacillus tuberculosis*. The condition is encountered much more frequently in children than in adults and is characterized by gradual destruction of bone and cartilage with little or no bone production. The primary tuberculous infection is often difficult to discover but is usually in the lungs, pulmonary or intestinal lymph nodes. The secondary infection in the bones and joints is the result of haematogenous implantation in the cancellous bone and synovia. Bone and joint tuberculosis is one of the most benign forms of the disease and the infection may be present for months or even years before symptoms are manifested.

Long before Koch detected and isolated the tubercle bacillus in 1882, pathologists and surgeons had recognized the fact that "scrofular strumous disease" was of a like nature to pulmonary consumption. Hippocrates and Galen noted the frequent association of chronic pulmonary disease and spinal caries. Wiseman in 1676 used the term "white swelling" for chronic tuberculous disease of joints and especially of the knee joint. In 1802 John Herdman of Edinburgh discussed the differential diagnosis of scrofulous and rheumatic joints. Rokitanski in 1844 introduced modern pathologic ideas when he definitely stated that there was similarity between chronic disease of joints and tuberculous disease of other organs, but Koster in 1869 was the first to demonstrate the existence of a tuberculous nodule in joint disease.

Etiology.—INFECTIOUS AGENT.—There are two main types of tubercle bacilli—the human type and the bovine type. These bacilli, although closely related, are not identical and can be differentiated by laboratory methods. From a public health standpoint it is important to know the causative agent, but clinically the differentiation is of little importance, as the pathologic processes produced by the two organisms are essentially the same. So far as adult human beings are concerned, the bovine bacillus seems to be a negligible factor, only a few cases infected with this type of bacillus having been reported. In children, however, and especially in children under five years of age, the percentage of cases of tuberculosis due to the bovine bacillus is quite high.

THE MANNER OF INFECTION AND IMMUNITY TO INFECTION.—Tuberculous infection comes from two sources: first, other human beings who are infected and who deposit tubercle bacilli about, to be taken up by nontuberculous persons; second, the dairy products of tuber-

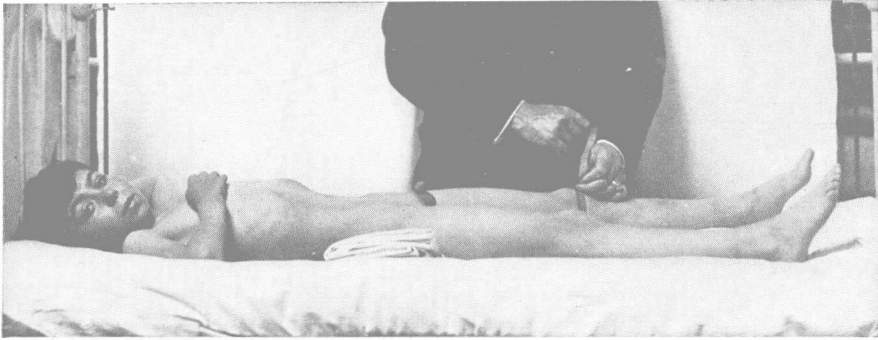


FIG. 1.—Routine physical examination in diagnosis of joint disease. Measuring the circumference of the lower extremity at the knee joint, to be compared with measurements taken on the nonaffected side.



FIG. 2.—Routine physical examination in diagnosis of joint disease. The use of the goniometer to determine the angulation of deformed positions of the extremities. Measuring the amount of flexion deformity of the knee.

culous cows, marketed in the form of milk and butter which contain tubercle bacilli and are ingested as food. Thus the respiratory tract and digestive tube are exposed to the infection. Having entered these portals, the organisms gain a foothold in the glandular tissues and either spread from here by direct extension or by haematogenous implantation in other organs. Pasteurization of milk, elimination of tuberculous cows, and segregation of individuals with active pulmonary tuberculosis, have done much in the past few decades to decrease the incidence and death rate of tuberculosis.

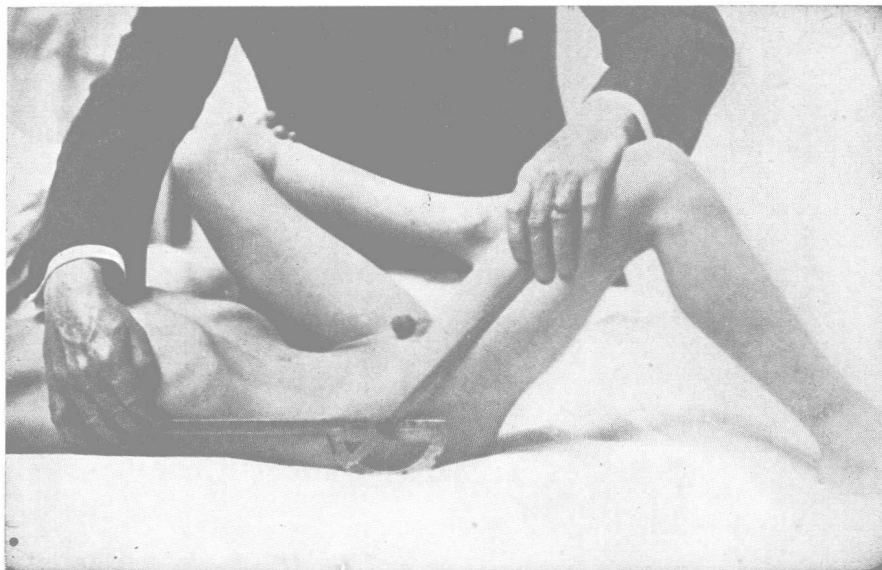


FIG. 3.—Routine physical examination in diagnosis of joint disease. The use of the goniometer to determine the angulation of deformed position of the extremities. Thomas test for flexion at the hip. The opposite thigh is flexed on the abdomen to place the pelvis in normal relationship to the spine.



FIG. 4.—Routine physical examination in diagnosis of joint disease. Palpation of the knee in order to determine the presence of excess joint fluid. With the forefinger of one hand ballottement of the patella may be discovered. If the patella "floats" there is excess fluid in the joint.

Immunity to tuberculosis balances very delicately with susceptibility. It is apparent that in an infection which is as widely spread as tuberculosis, most individuals have had an infection at one time or another. This is borne out by the high percentage of positive reactions to tuberculin tests in adult individuals and by the postmortem findings of healed tuberculous lesions in persons who never had clinical tuberculosis during life. Little is known of the factors governing immunity to tuberculosis. Rich has demonstrated that with tuberculosis, allergy and immunity may, and frequently do, coexist; but that they always coexist or that they always parallel each other in degree is certainly not the case. Allergy, as denoted by reaction to tuberculin tests, does not indicate immunity, and conversely, immune individuals are not necessarily allergic.

PREDISPOSING CAUSES.—In general it can be stated that any causative factor which lowers the power of resistance of the individual predisposes him to tuberculosis. Although the tuberculous organism itself cannot be inherited, it has been shown that children of tuberculous parents have an increased susceptibility to the disease. In many cases the predisposition is acquired. Thus the disease occurs more frequently in cold, damp climates than in hot, dry climates. It is also more frequent at lower than higher altitudes. The disease occurs more frequently in older thickly populated countries than in sparsely settled newer lands. The lowering of resistance, as a result of poor living conditions, including improper food, poor nutrition, lack of fresh air and sunshine, predisposes to tuberculosis. Thus the disease is always more prevalent in the tenement districts of large cities, where people live in crowded quarters with poor sanitation, improper ventilation and little or no sunshine. That there is a racial lack of immunity to the disease is proved by the high percentage of infection that occurs in those races such as the American Indians and Eskimos, who have not had contact with the disease.

Tuberculosis occurs at all ages but is pre-eminently a disease of the growing period. The vast majority of cases occur in the period between the third and tenth years. In more than 80 per cent of all patients with this condition, the disease has its onset before the age of fourteen. Infection is rare before the age of two. Tuberculosis has no predilection for either sex; it is slightly more frequent in males than females but not more than the normal relative preponderance of male over female children.

EXCITING CAUSES OF TUBERCULOSIS OF THE BONES AND JOINTS.—Trauma is unquestionably the most frequent exciting cause of the localization of tuberculosis in the bones and joints. This is well borne out by the fact that the infection occurs much more frequently in the

weight-bearing joints of the lower extremity than in the upper extremity. Although it is difficult to evaluate the importance of the history of preceding trauma in children who are constantly subjected to injuries, it seems certain that in many cases the development of a tuberculous lesion in a bone or joint following injury is not a pure coincidence. There is also experimental evidence to the effect that in tuberculous animals the injured joints develop tuberculosis much more readily than those uninjured. In the writer's series of 200 cases of bone and joint tuberculosis, the spine was involved in 60 per cent; the hip joint in 20 per cent; the knee joint in 10 per cent; the ankle joint in 3 per cent; and the joints of the upper extremity in 7 per cent.

The acute exanthemata, especially measles and scarlet fever, are often followed by the appearance of bone and joint disease in tuberculous individuals. Miliary tuberculosis is also frequently encountered following these infections, probably due to lowered resistance of the individual. Similarly, although not as frequently, tuberculosis of the bones and joints is seen following whooping cough, influenza, colds, etc. Any change in body metabolism, which lowers the general resistance, renders the individual more susceptible to tuberculous infection.

Pathology.—Tuberculosis of the bones and joints is a disease process caused by the tubercle bacilli, which have been carried to these tissues by the blood stream from a primary focus elsewhere in the body, usually in the lungs or alimentary tract.

Tuberculous lesions usually begin in the bone rather than in the synovial membrane of the joint. That part of the growing bone that has the richest blood supply, namely, the bone near the epiphysis, is usually the site of the primary infection in the long bones. In the short bones, such as the phalanges of the fingers, the primary lesion is commonly in the diaphysis. Tuberculous lesions of the vertebrae are usually primary in the bone marrow of the vertebral body.

There is as yet no unanimity of opinion regarding the site of the primary lesion. For many years it was felt that a primary focus in the synovial lining never occurred. More recent studies, however, by Peabody, Finder, Phemister and others appear to show that primary synovial involvement is more common than heretofore supposed. Controversy has also existed as to whether the primary lesion, when in bone, is in the epiphysis or metaphysis. Phemister states: "In children there is some variation in the changes from those seen in adults. In late childhood and adolescence, the picture gradually approaches the adult form. The disease is perhaps primary in the synovia in a higher percentage of cases than in adults. When the primary focus can be identified as osseous, it is more frequently situated in the me-

taphysis than in the epiphysis. These statements are truer for younger children than for older ones."

Allison analyzed the situation as follows: "The skeletal structures that furnish the soil upon which tuberculous infection may become localized and develop are these: bone of the cancellous and cortical

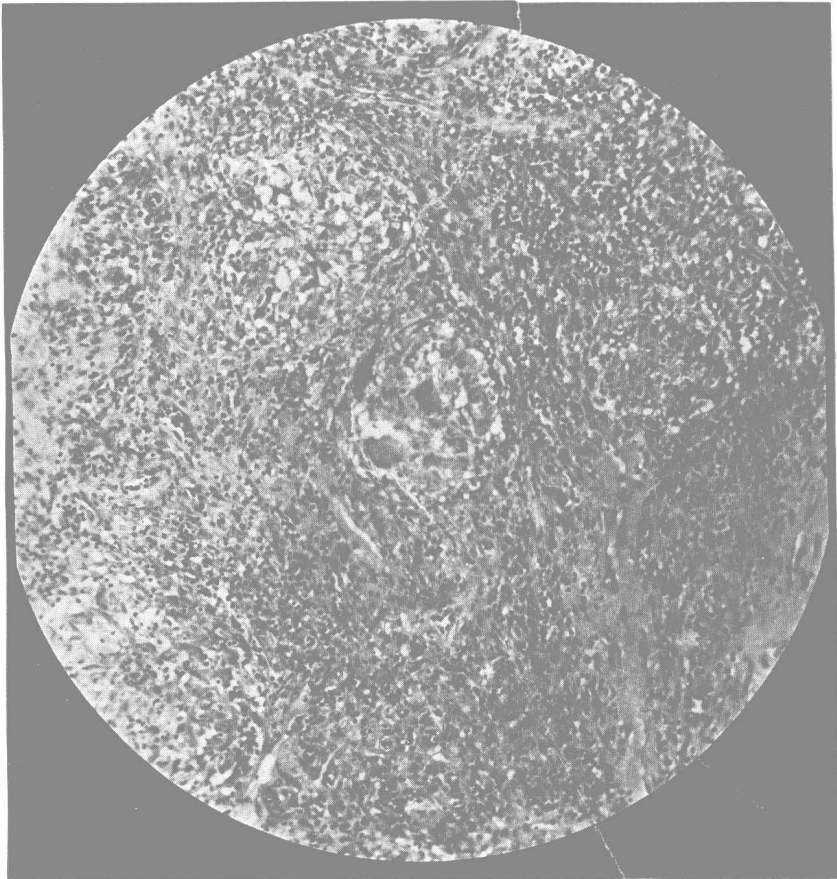


FIG. 5.—Photomicrograph of typical lesion of tuberculosis. Histologically, this lesion is known as a "tubercle." There is a typical focal collection of cells with giant-cell formation and evidences of caseation.

types; periosteum; epiphyseal cartilage; articular cartilage; the epiphysis; the metaphysis; the diaphysis; bone marrow; the joint capsule; the ligaments and the synovial membrane. Tuberculous infection, gaining a foothold in any one or in several of these structures, behaves in a manner which corresponds to the structure involved. There is no warrant, pathologically or clinically, to consider these lesions from the standpoint of contrasted bone or joint involvement. In most instances, joints are involved in the process. In practically every instance there is involvement of the bone. One cannot be sure in what

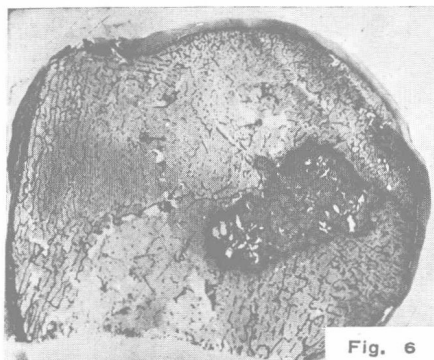


Fig. 6



Fig. 7

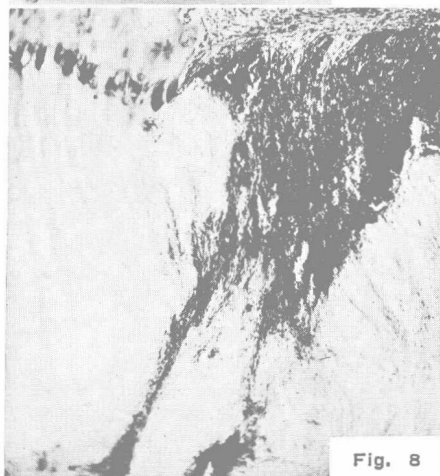


Fig. 8

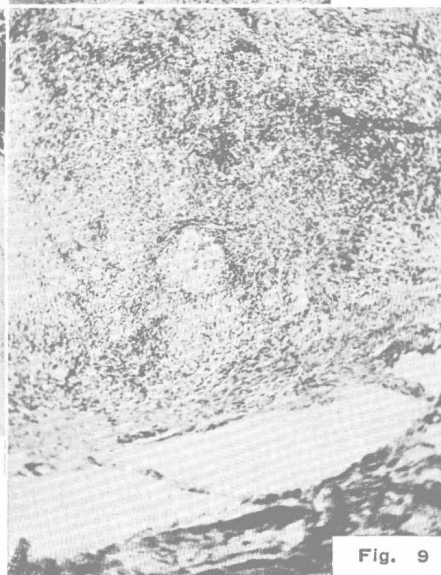


Fig. 9

FIG. 6.—Section of the head of a humerus in the midfrontal plane showing the site of the original tuberculous focus.

FIG. 7.—Microscopic section at the area of focus (Fig. 6) demonstrating the tuberculous granulation tissue surrounding the trabeculae. The trabeculae are being broken down in a rapid manner by osteoclasts and caseation is spreading rapidly.

FIG. 8.—Section through the cartilage (Fig. 6) showing erosion of the cartilage of the joint from below, and a "break through" into the joint cavity by tuberculous granulation.

FIG. 9.—Thickened synovial membrane with extensive tuberculous involvement.

tissue the primary infection takes place in all cases. One may be sure that in all of them the bone becomes the chief seat of the infection."

Tubercles which form in bone or synovial tissue present the same characteristic arrangement of cells that they do elsewhere. Tuberculosis, when primary in bone, usually begins in the marrow of the epiphysis of the long bones. The original lesion either becomes encysted and localized or infiltrates the tissue, depending on the viru-

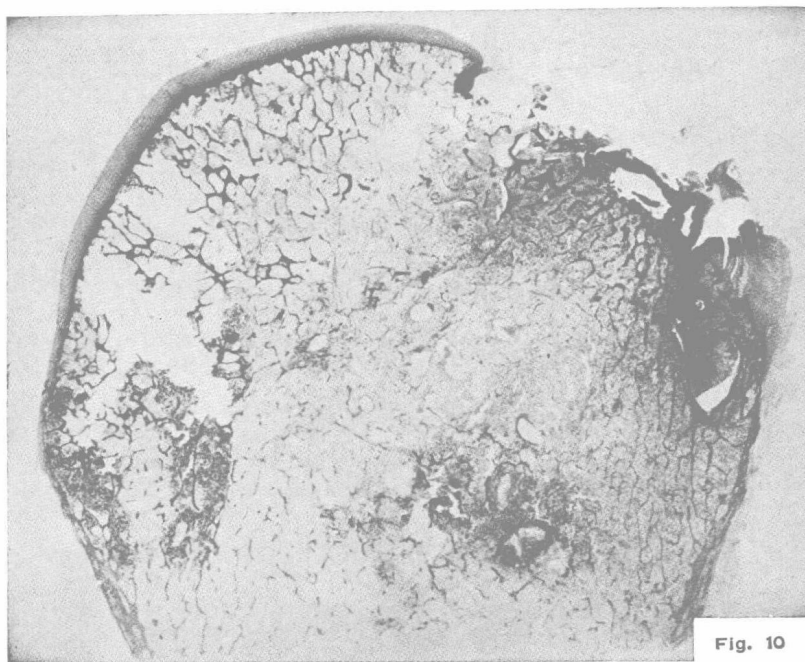


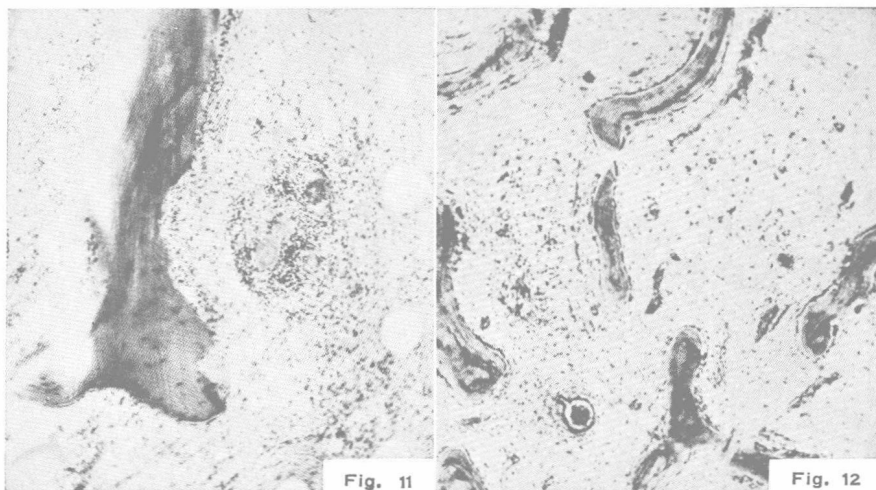
Fig. 10

FIG. 10.—Tuberculosis of shoulder. This demonstrates the extensive atrophy of the cancellous bone of the upper end of the humerus, with cartilage erosion and tuberculosis of the synovial membrane.

lence of the infection and the amount of individual resistance of the host. The lesion advances by the formation of conglomerate tubercles and diffuse granulation tissue which erode the bony trabeculae and replace the marrow. Frequently, during the process of invasion, the nutrition of a large portion of bone is interfered with either by tuberculous granulations or formation of an embolus, and as a result a large V-shaped sequestrum forms with its base usually on the surface of the joint. Occasionally the disease spreads to the joint by erosion and perforation of the articular cartilage, but in the majority of cases secondary joint involvement takes place by extension of the disease process to the joint margin at the point of reflection of the synovial membrane from the bone.

Following involvement of the synovial tissues, tuberculous granulations or pannus overgrows the marginal articular surfaces, destroying the underlying cartilage. Phemister has shown that in tuberculous in-

fection, articular cartilage is destroyed first along free surfaces where the tuberculous granulation tissue comes onto it and removes it. The cartilage persists longest where it is protected by contact and pressure of two opposing surfaces. In contrast to pyogenic infection of joints, the articular cartilage in tuberculosis is destroyed very slowly. In pyogenic infection the polymorphonuclear leukocytes elaborate a ferment which dissolves the articular cartilage. In tuberculous infection the lymphocytes do not elaborate this ferment, so that the cartilage



FIGS. 11 and 12.—Tuberculosis of hip of many years' duration. Section through the head of the femur showing various stages of the disease process. Figure 11 shows marked atrophic and infiltrating changes. The trabeculae are being rapidly destroyed by osteoclasts, low vascularity and caseation. Figure 12, on the other hand, shows power of repair; the marrow spaces are filled with fibroblasts, there is increased vascularity, the trabeculae are surrounded by osteoblasts, and there is definite new bone formation.

lasts until granulation tissues destroy it. As a result of this difference of behavior of the tissues, ankylosis is slow to develop in tuberculosis and rapidly develops in pyogenic infection, the presence of articular cartilage being the bar to bony ankylosis.

In many cases of tuberculous arthritis at the synovial margin, a thin layer of granulation tissue is formed which progresses between the cartilage and the bone toward the center of the joint. It had been formerly supposed by Phemister, Allison, Ghormley and others that these subchondral undermining granulations were tuberculous at the periphery but not tuberculous under the central portion of the cartilage. Brav has recently demonstrated evidence of tuberculosis in central and marginal portions of subchondral granulations in 29 per cent of the cases studied.

Abscesses are frequently encountered with tuberculosis of the bones and joints. These are known as "cold abscesses" because there is no increased local heat or redness. They consist of pus, caseous material and detritus, and travel usually following the pull of gravity, often reaching the body surface at a point far distant from the diseased area.

The abscess frequently perforates the skin, discharges, becomes secondarily infected, and a tuberculous sinus results. Under treatment, however, the abscess often becomes absorbed without breaking through the surface. It is difficult to demonstrate the tubercle bacillus in smears from the pus but its presence can usually be proved by guinea pig inoculation.

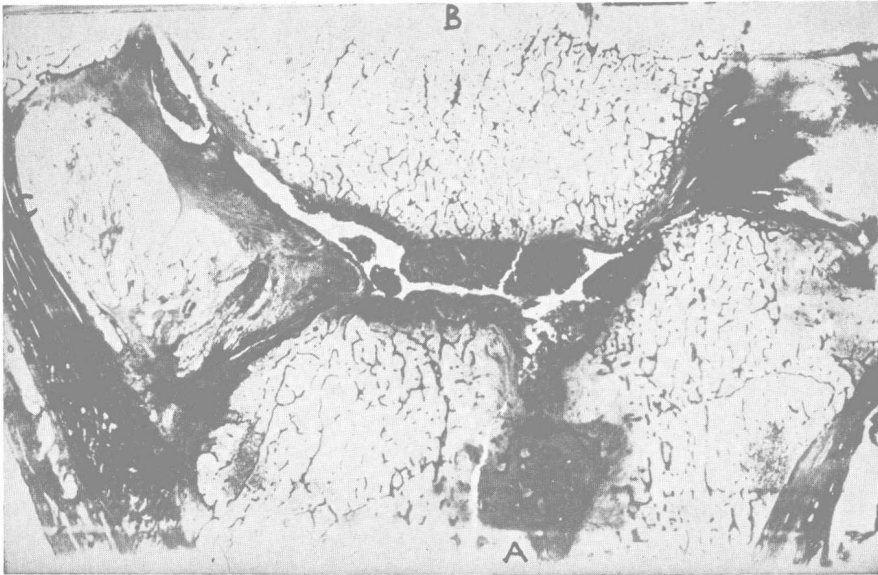


FIG. 13.—Mid-sagittal section of tuberculosis of the knee.

A represents the upper end of the tibia.

B represents the lower end of the femur.

C represents the inferior margin of the patella.

It will be seen that there is a large focus in the head of the tibia which has broken through into the joint, destroying the cartilage and invading the synovial membrane. The diseased process is apparently of long duration, as is shown by the atrophy of the cancellous bone. The synovial membrane and cartilage have been replaced by tuberculous granulations.

Bone Tuberculosis Without Joint Involvement.—Tuberculosis of the shaft of the long bones is seen rarely, and especially so in adults. Zumsteeg reported its occurrence in 0.9 per cent of all cases of bone and joint tuberculosis, and Miltner found four cases (0.4 per cent) in a total of 1000.

Tuberculous lesions of the shaft may simulate the osteomyelitic lesions produced by pyogenic infection, and therefore it is believed that the mode of infection is the same in either condition. Koenig first emphasized the embolic theory of skeletal tuberculosis.

Fraser, in describing the gross pathologic varieties of osseous tuberculosis, classified them as follows: (1) The encysted tuberculous



Fig. 14

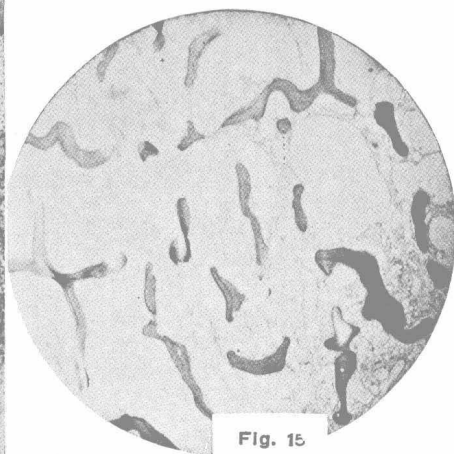


Fig. 15

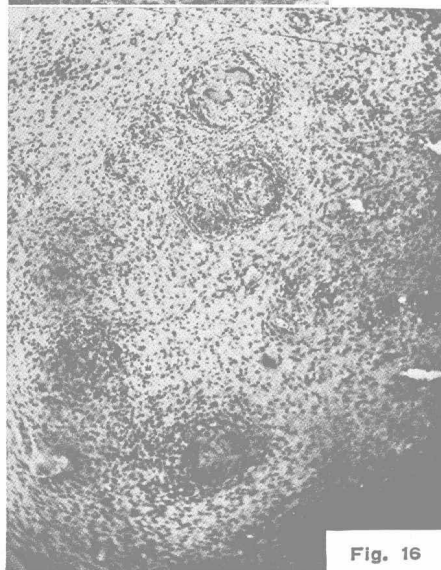


Fig. 16



Fig. 17

FIGS. 14-17.—Tuberculosis of knee. Figure 15 is a photomicrograph of the specimen. It shows the gross changes that were observed. The articular surfaces of the femur, the tibia and the patella are covered with a thick granulation tissue. Very little cartilage remains, and that in small islands; but the original focus of bone involvement is shown in the tibial head, in direct communication with the joint cavity. Microscopic study of the sections reveals extensive tuberculous involvement of cancellous bone (Fig. 14), marked atrophy of bone (Fig. 15), extensive tuberculosis of the joint capsule (Fig. 16) and involvement of the periosteum with proliferation of new bone on the tibial surface (Fig. 17).