ANNALS of SURGERY

VOL. LXXX OCTOBER, 1924 No. 4

# THE EFFECT OF PRESSURE ON ARTICULAR SURFACES IN PYOGENIC AND TUBERCULOUS ARTHRITIDES AND ITS BEARING ON TREATMENT*

BY DALLAS B. PHEMISTER, M.D.
OF CHICAGO, ILL.

IT WAS recognized that pressure played a rôle in the destruction of articular surfaces in arthritis long before the bacterial nature of infection was known. Since the time that pyogenic and tuberculous arthritides were differentiated bacteriologically, the differences in the effects of contact and pressure on the articular surfaces in the two conditions have not been fully elaborated. In fact, it is too generally considered that they are much the same in the two conditions, that the effects are destructive, and that in both the articular surfaces are destroyed first and most extensively at the points of contact and pressure of opposing articular surfaces. Examination of a series of specimens showing each disease in its various stages demonstrates that this is not the case. The changes as influenced by contact and pressure are more accurately described for pyogenic than for tuberculous arthritis. Koenig's [1] work on tuberculous arthritis is the most extensive and exact, but inaccuracies may be found in it pertaining to the persistence of cartilage longer at certain points than at others and to bony invasion and necrosis. Also, knowledge as to proteolytic activities in pyogenic and tuberculous infections has not been utilized in explanation of the changes seen in articular cartilages in pyogenic and tuberculous joints.

In studying the effects of contact and pressure, a distinction should be made between those produced on the articular cartilage and those produced on the underlying bone. In acute pyogenic arthritis it is found that when there is an effect from contact and pressure, it is to help to destroy articular cartilage in the regions of contact of opposing articular surfaces. On the other hand, in tuberculous arthritis the effect, except in the later stages, is protective, and articular cartilage is usually preserved longest at the points of contact and pressure, while the first evidences of destruction are found over the free surfaces. The differences are present because the agents which attack and destroy articular cartilage are very different in the two processes. These observations have been made mainly on adults and on the knee-joint, where, because of large areas of both free and opposed articular surfaces, conditions are favorable for contrasting the effects of pressure and lack of pressure. They hold in varying degrees for other joints and for children. In both

* Read before the American Surgical Association, April 18, 1924.

pyogenic and tuberculous infections the articular cartilage is involved secondarily, the primary infection being either in the synovia or in the bone.

*Pyogenic Arthritis. Effects of Pressure on Cartilage.*—In pyogenic arthritis the articular cartilage may not become involved if the infection is mild, but if the infection is severe, cartilage will be killed, and it is generally killed first and most extensively at the points of contact and pressure of opposing articular surfaces. This is undoubtedly related to the unfavorable conditions for nutrition produced by pressure. The amount of necrosis on surfaces that are not pressed upon varies. In some cases it is slight, while in others the entire cartilage may be killed. Whatever happens on one side of the joint usually happens on the other. If cartilage is killed in the apposed regions on one side, it is usually killed to the same extent on the other side. Dead cartilage in pyogenic arthritis is usually destroyed in a comparatively short time. It is broken down partly by the absorptive action of granulation tissue, partly by erosion of opposing articular surfaces, and partly by the digestive action of proteolytic ferments in the exudate of the joint. These ferments are derived very largely from the polymorphonuclear leucocytes, to a slight extent from broken down bacteria. Where cartilage is killed in its entire thickness, granulations from beneath its attached surface and rapidly detach it by absorption of both cartilage and bony cortex at their junction. Where only a superficial layer of cartilage has been killed in unopposed regions, it is removed mainly by digestion, but granulations may also grow from the margins over the unopposed surfaces and absorb the dead layer.

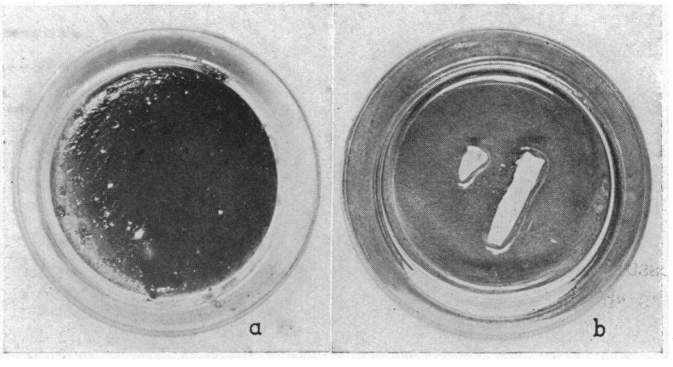


FIG. 1.—Equal amounts of articular cartilage in staphylococcus pus (a) and in tuberculous pus (b) after incubation at 55 degrees C. for ten hours; cartilage digested in (a) and undigested in (b).

A series of experiments has been performed to test the rapidity with which articular cartilage is digested by a pyogenic exudate *in vitro*. When pieces of articular cartilage are immersed in pus produced by any of the pyogenic microörganisms and the mixture incubated at a temperature of 55 degrees C., so that proteolytic action is augmented, the cartilage is digested in from three to twenty-four hours, depending on the concentration of the pus. Figure 1 shows two watch glasses, each of which at the beginning of the experiment contained two pieces of fresh articular cartilage and underlying bony cortex of the size shown in (b). Staphylococcus pus was added to (a) and pus from a tuberculous cold abscess to (b). After incubation for ten hours, the articular cartilage in (a) was completely digested and particles of bone sand were the only solid materials remaining. The pus was more liquid than at the beginning of the experiment, because of the breaking down of its proteids and of the leucocytes themselves. At a temperature of 55 degrees C., bacterial action is suspended, so that the digestion was produced by existing ferments in the pus. That the ferments are derived very largely or wholly from the polymorpho-

nuclear leucocytes in the exudate is shown by the fact that relatively little or sometimes no cartilage is digested, when it is incubated in a suspension of pyogenic bacteria. Experiments have demonstrated that a variable amount of proteolytic ferment is formed by different organisms, as shown by their ability to break down tissues and to liquefy such media as gelatine and blood serum. According to Bittrolff,[2] it is small in amount with most pathogenic forms. Heavy suspensions in normal salt solution of some strains of staphylococcus aureus were found not to digest articular cartilage, while others digested it to a slight degree. In the experiment shown in Fig. 2, equal sized pieces of articular cartilage were placed in the tubes. Tube (1) contained normal salt solution, tube (2) a suspension of staphylococci in a concentration of 15 millions per cubic millimeter, and tube (3) a suspension of 40 millions per cubic millimeter. They were incubated at 55 degrees C. for six days and there was no reduction in size of the pieces of cartilage. Microscopic examination of sections showed cartilage well preserved in the piece incubated in salt solution and only very slight breaking down of cells and vacuolation of intercellular substance in the pieces incubated in the staphylococcus suspensions. The experiments of Bittrolff,[2] Cacace[3] and others have shown that proteins may be split by the action of bacterial ferments into albumoses, peptones and amino-acids.

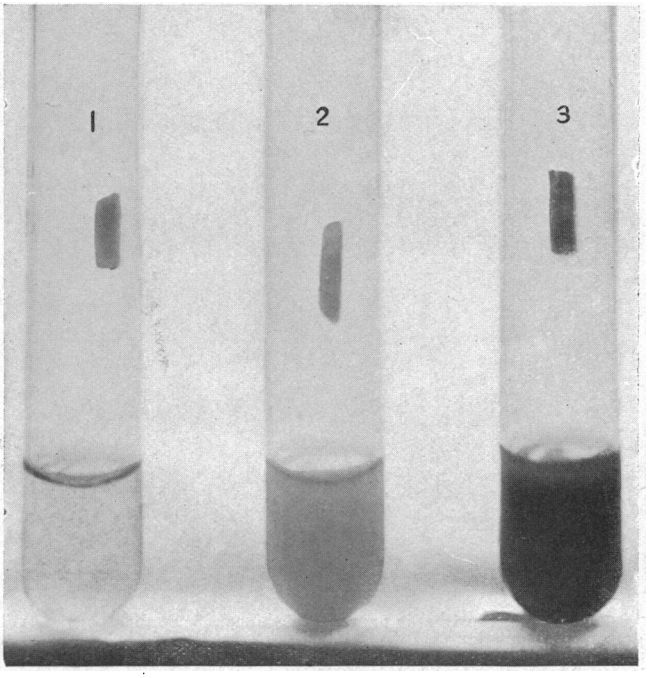


FIG. 2.—Equal sized pieces of articular cartilage incubated at 55 degrees C. for six days in normal salt solution (1) and staphylococcus suspensions (2) and (3). No change in (1) and only slight microscopic changes in (2) and (3), showing little digestion by the bacteria.

*Effects of Pressure on the Bone.*—In pyogenic arthritis the changes in the bone bordering on articular surfaces vary according to the point of primary infection. In primary arthritis with secondary involvement of cartilage, the articular cortex of bone is nearly always destroyed in those regions where the entire thickness of articular cartilage is broken down. Consequently it is destroyed oftenest and most extensively at the points of pressure. An inflammatory reaction is seen in the adjacent layer of spongy bone of these regions, but deep invasion producing osteomyelitis and sequestration, even at the points of pressure, is rare. In infected penetrating wounds of joints with associated joint fracture and osteomyelitis, death of detached bony fragments is common. When the primary infection is an osteomyelitis which spreads into the epiphysis and the joint, necrosis and sequestration of bone bordering on the articular surface is not uncommon, and even here, as in the head of the femur, it may be greatest in the weight-bearing region. When bone and overlying articular

cartilage are both killed, the cartilage rapidly disappears by the digestive action of ferments, but the dead bone with its layer of articular cortex may persist and be separated as a sequestrum.

Figure 3 shows a photograph of the articular surfaces of the bones of the knee and ankle, which were involved in arthritis by direct extension from staphylococcus osteomyelitis of the entire shaft of the tibia in a twelve year-old boy. The infection in the knee was purulent and was drained on the fifty-fourth day, when the patient was admitted to the Presbyterian Hospital. That in the ankle was less severe and had subsided without drainage when the limb was amputated above the knee on the seventy-fifth day of the illness. Articular cartilage and cortex of bone were almost completely destroyed at the points of contact and pressure of os calcis (a) with the tibia (b), but were preserved about the sides of the joint on os calcis, tibia and fibula, where there was little or no pressure. At the knee, where the arthritis was severe, the destruction of articular surfaces was more marked. The infection had involved a part of the upper epiphysis of the tibia and had killed articular cartilage and underlying cortex of both tibial tuberosities (c). The cartilage had been destroyed by digestion, but portions of articular cortex were present as disc-like sequestra. The one on the internal tuberosity is shown *in situ* at (y) in Fig. 3. The femur (d) showed complete disappearance of articular cartilage and of underlying cortex on the condyles at the points of contact and pressure with the tibia and partial destruction of cartilage and cortex at the point of contact with the patella, which latter structure had also lost its cartilage, excepting remnants about the periphery. The cartilage was largely preserved on the remaining free surfaces anteriorly between the points of contact with patella and tuberosities, and posteriorly on the condyles, but its surface was mostly uneven from superficial destruction.

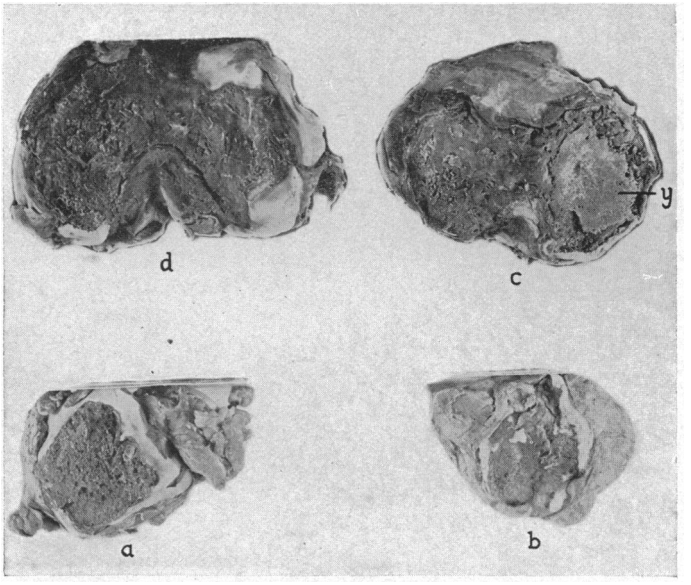


FIG. 3.—Joint surfaces in pyogenic infections of knee and ankle. Cartilage destroyed at pressure points and largely preserved where not compressed on astragalus (a), lower end of tibia (b) and femur (d). Cartilage all destroyed on upper end of tibia (c). Cortical sequestrum (y).

This case illustrates well the disappearance of cartilage and cortex where pressed upon by opposing articular surface, the survival of cartilage in the regions of the joint that are free from pressure, and the preservation of bony cortex and disappearance of cartilaginous covering on an articular sequestrum. A comparatively mild acute arthritis may involve articular cartilage at the points of greatest pressure, which will lead to bony ankylosis, unless measures are introduced to combat it.

## EFFECT OF PRESSURE ON ARTICULAR SURFACES

Figure 4 shows a photograph of the articular surfaces of the right knee five months after the onset of a hæmatogenous arthritis secondary to erysipelas of the left thigh. There was an effusion at the onset and aspiration yielded a turbid serous fluid, from which a hæmolytic streptococcus was grown. The infection subsided spontaneously after six or seven weeks, leaving the joint with only a small range of motion. Extension was not applied to the limb, nor was the joint mobilized. The limb was amputated because of recurrence of an old staphylococcus osteomyelitis of the upper end of the femur with coxitis. Dissection showed destruction of articular cartilage and cortex at the points of contact of external condyle (a) and external tuberosity (b), which is the region of greatest pressure in the knee-joint, and a bony bridge was in process of formation here. The articular cartilage was preserved elsewhere on the tibia and femur and on the patella, but there was extensive fibrous ankylosis present in these regions. The extreme degree of destruction of cartilage and fibrous ankylosis resulting from the comparatively mild arthritis was no doubt related to the fact that the knee was neither mobilized nor extended during any part of the course of the infection.

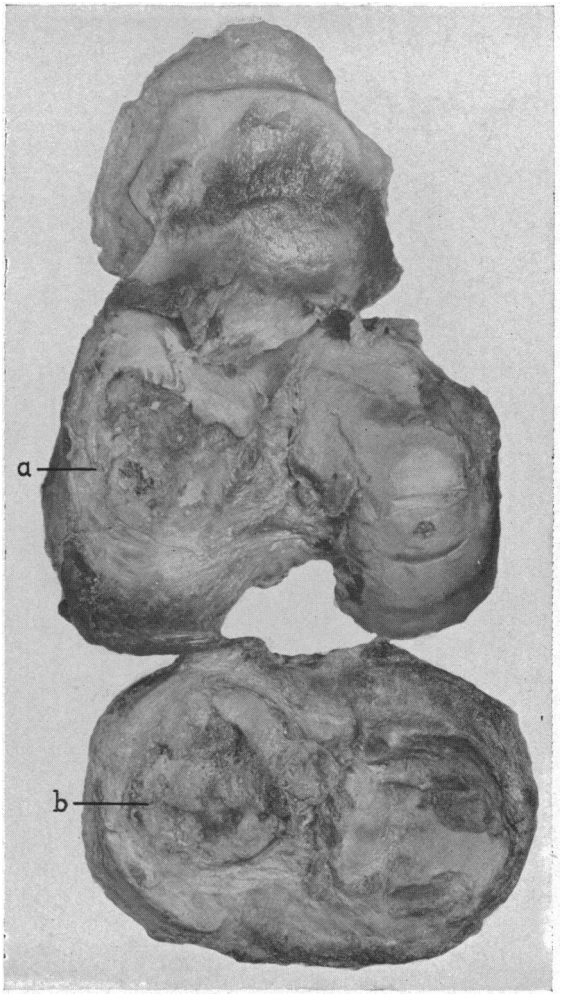


FIG. 4.—Ankylosis five months after mild undrained arthritis of knee. Cartilage destroyed only at points of pressure of external condyle (a) on external tuberosity (b). Fibrous ankylosis elsewhere.

In moderately severe pyogenic arthritis, it may rarely happen that articular cartilage and underlying cortex are destroyed at the points of pressure on one side of the joint and persist on the other. The presence of cartilage on one side lessens the liability to the development of ankylosis, and there may be healing with preservation of considerable motion. The area of destroyed articular surface is repaired by the outgrowth of granulation tissue from the underlying bone, and it may overgrow the surrounding cartilage, producing a ridge about the margins of the defect. These granulations change into fibrocartilage and may ossify in their deeper portions, producing osteophytes on the articular surface. Figure 5 shows the articular surfaces of a man's knee, resected one year after spontaneous healing of a mild seropurulent arthritis, resulting from extension of a staphylococcus osteomyelitis of the tibia. The articular surfaces had been destroyed at the points of contact on the condyles of the femur, but not on the tibia, where they were somewhat protected by the marginal support of the semilunar cartilages. The

damaged areas on the condyles had been repaired by a layer of fibrocartilage, the margins of which were raised and in places overhanging. A sagittal section through the lateral condyle showed that articular cartilage and cortex had been destroyed at the point of greatest pressure and that reparative tissue had grown over from the underlying bone, filling the defect and overlying the margins of surrounding cartilage. (Fig. 6.) The superficial portion of this tissue was fibrocartilage, while the deeper portions had ossified.

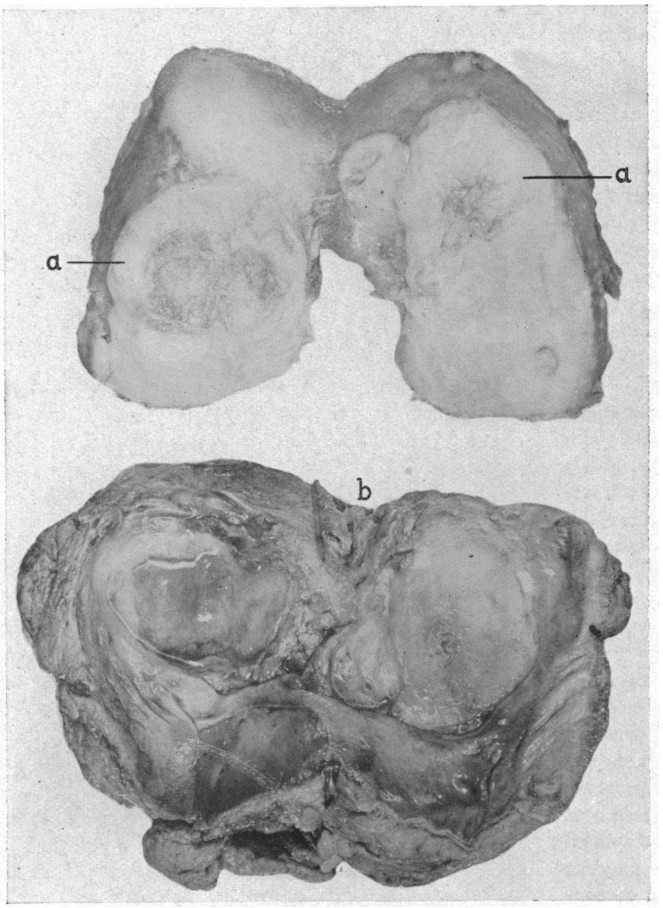


FIG. 5.—Healed undrained pyogenic arthritis of knee. Roughened healed areas where cartilage is destroyed at points of pressure on condyles of femur (a); cartilage on tibia (b) little changed.

The shadow cast in the röntgenogram by the new bone overlying the remaining articular cartilage gives the appearance of a bony disc interposed in the cartilage space of the joint between condyle and tuberosity (Fig. 7). If cartilage and cortex break down on both sides of the joint, it is extremely difficult to avoid the occurrence of bony ankylosis.

*Tuberculous Arthritis. Effects of Pressure on Cartilage.*—In tuberculous arthritis the point of primary infection is either in the bone or in the synovial lining, and the articular cartilage becomes involved secondarily. The inflammatory reaction is not sufficiently severe to kill articular cartilage *en masse* in the early stages of the disease, as is so often the case in pyogenic arthritis. Tuberculous synovitis usually runs for months and is well established before there is any sign of destruction of articular surfaces. The articular cartilage is then first killed and absorbed by the direct attack of tuberculous granulations, which grow onto it from the surrounding synovia. They attack it first along its free surfaces and about its margins, where they can readily get at it. The cartilage is protected from surface attack of tuberculous granulations in the regions of contact and pressure of opposing surfaces in the joint, and the

destruction may be very marked along the free surfaces and about the margins before there is any change in the cartilage of the contacted regions.

This is illustrated by Fig. 8, which shows the articular surfaces of the resected femur and tibia in tuberculosis of the knee of about fifteen months' standing in an eighteen year old boy. There was marked tuberculous synovitis and granulations had attacked the cartilage of the tibia about its margins, but the central portions of cartilage on either tuberosity were free from attack, as were the surfaces of the condyles of the femur with which they come in contact. The surfaces of contact of patella and femur were also free. Granulations covered the free cartilaginous surface of the femur between its points of contact with patella and tibia and on the posterior surface of the condyles, and had absorbed most of the thickness of cartilage. The layer of granulations is seen intact between patellar surface and internal condyle, but it has been removed, leaving a grooved and roughened surface, between the patellar and external condylar surfaces.

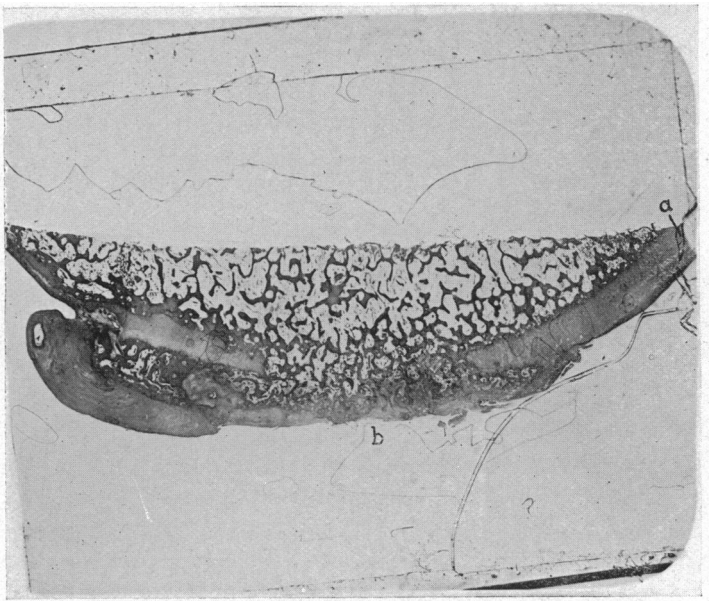


FIG. 6.—Section through external condyle shown in Fig. 5, showing layer of articular cartilage (a) destroyed at point of greatest pressure and overgrown by partly ossified reparative tissue (b) growing out from the bone.

Destruction of cartilage in the regions of contact and pressure is usually brought about first by undermining subchondral granulations, which may be tuberculous in nature about the periphery of the cartilage, but which are non-tuberculous beneath the more centrally located portions of cartilage. There they consist of capillaries, fibroblasts and round cells, and in the capillary loops that invade and absorb the cartilage, polymorphonuclear leucocytes may be seen. They gradually detach the cartilage by absorbing articular bony cortex and the deeper portions of cartilage. As in pyogenic arthritis, whatever happens on one side of the joint usually happens on the other. If cartilage is preserved or eroded or undermined on one side the same condition obtains at points directly opposed.

These changes are well illustrated in Fig. 9. It shows the articular surfaces of the femur and tibia of a man whose knee was resected because of tuberculous arthritis of two and one-half years' standing. Cartilage is attached and preserved on the central portions of the surfaces of contact of femur with patella and of lateral condyle with lateral tuberosity, which is the point of greatest pressure in the joint. This is the region in the knee-joint where cartilage usually persists longest. It is detached and thinned by under-

mining granulations on the mesial condyle and tuberosity. It has been destroyed everywhere else in the joint except in these regions of contact, and they show marginal absorption. Figure 10 is a side view of the same specimens, with a sagittal section through the lateral condyle. It shows the preservation of cartilage and of articular cortex of bone at the points of contact of patella and of tibia with femur and the loss of cartilage and of articular cortex in the unopposed regions. The preservation of cartilage space and of articular cortex in the opposed regions is demonstrable in the röntgenogram taken before operation and shown in Fig. 11.

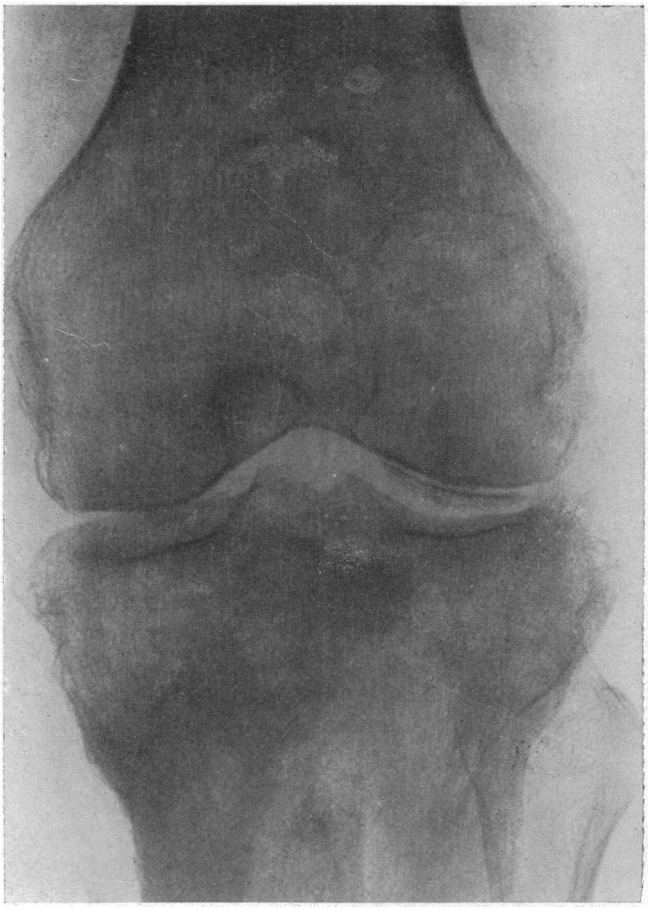

FIG. 7.—Röntgenogram of knee shown in Fig. 5. The ossified reparative tissue overgrowing the articular cartilage about margins of the regions of destruction cast disc-like shadows in cartilage spaces between condyles and tuberosities.

The first röntgenographic evidences of destruction of articular cortex in tuberculosis of the knee are usually seen along the free surfaces and about the margins of the articular surfaces. Preservation of both the normal width of cartilage space and shadow of cortex in the regions of contact and pressure is in favor of tuberculous arthritis, while loss in those regions with preservation elsewhere in the joint is in favor of pyogenic arthritis.

In those joints whose articular surfaces have like contour and in which cartilage fits snugly against cartilage throughout, leaving little or no unopposed surface, the loosening and destruction of articular cartilage is carried on mainly by the action of undermining granulations. This is true of the hip and ankle-joints, where, on opening the joint at the right stage, the cartilages may be found completely detached and considerably thinned from beneath, while there is little or no evidence of destruction along their free surfaces. After the disease is well advanced and destruction is extensive in the unopposed regions of the joint, the remaining cartilage in the opposed regions

may become necrotic. Cartilage is also killed *en masse* when the undermining granulations destroy its bony connections. In fact, the explanation of the development of the non-tuberculous subchondral granulations may be the necrosis of the cartilage without the entrance of tubercle bacilli into the subchondral regions. The granulations then form as a foreign body reaction to absorb and sequestrate the dead cartilage.

The experimental work of Nussbaum [4] supports the theory that articular cartilage, except the deepest layer, receives its nutrition from the synovial fluid. If the joint fluid continues to be the source of nutrition for the cartilage in tuberculous arthritis, it is understandable how, after the disease is well advanced, the altered exudate no longer furnishes adequate foodstuffs for cartilage and it dies *en masse* from lack of nourishment, as well as from the effect of tubercle toxins.

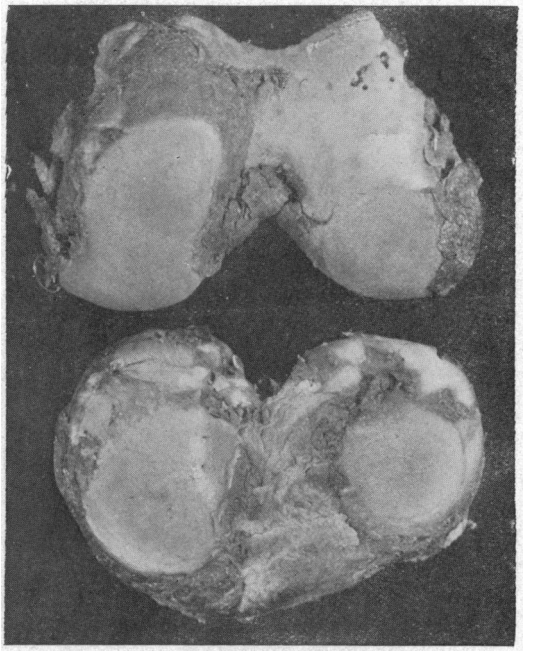

FIG. 8.—Tuberculosis of knee. Cartilage preserved where patella and tuberosities come in contact with femur and partly destroyed along unopposed surfaces of tibia and femur.

Dead cartilage, whether attached or detached, may persist in the joint for incredibly long periods, because of the absence of active proteolytic ferments in the tuberculous exudate. Pressure and motion erode and destroy dead cartilage. But when these are absent or slight, detached discs may persist for many months and dead cartilage may stand for years on articular sequestra, since granulations cannot get at its base to absorb it and there are no proteolytic ferments in the exudate to digest it. Edward Mueller,[5] Jochmann,[6] Opie and Barker,[7] and others have shown that exudates in tuberculous processes, including cold abscesses, contain practically no active proteolytic ferments, and we know that tuberculous granulation tissue is killed by coagulation necrosis, coagulins being formed by the tubercle bacilli (Ruppel,[8] Schmoll [9]) which precipitate the soluble colloids of the cells. The caseous areas persist for extremely long periods, because of the absence of active digestive ferments.

No published studies were found on the effect of tuberculous exudates on dead articular cartilage in relation to the persistence of cartilage in tuberculous joints. The digestive action of tuberculous exudates on cartilage was tested *in vitro*. Cold abscesses were aspirated and cartilage was incubated

at a temperature of 55 degrees C. in the fluid obtained. The tuberculous fluid coagulated in a short time, due to its richness in albuminous substances, and both articular cartilage and coagulum persisted after several days of incubation without any signs of digestion. This shows the absence of proteolytic ferment in the exudate. The tubercle bacillus is found to contain no proteolytic ferments and its toxins destroy the autolytic enzymes of the dead tissues. Consequently there is no autolysis of the dead cartilage. The leucocyte present in the tuberculous granulations and exudate is mainly the large mononuclear cell. It contains some proteolytic ferment, which is active in acid media, but not in tuberculous exudates, which are always alkaline in reaction (Opie and Barker [7]).

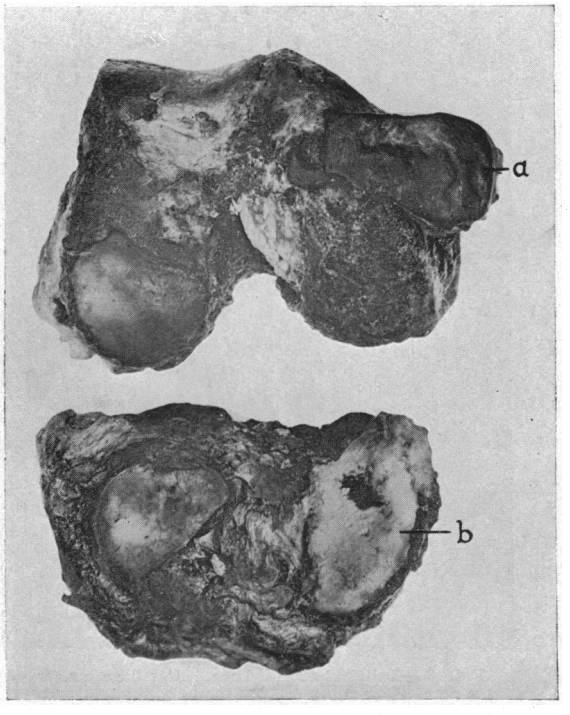


FIG. 9.—Tuberculous knee of two and one-half years' duration. Cartilage largely preserved at points of contact of patella and tibia with femur and destroyed elsewhere in joint. Contacting cartilage undermined on mesial condyle (a) and mesial tuberosity (b).

Figure 1 (b) shows cartilage unchanged and tuberculous fluid coagulated after ten hours of incubation. Figure 12 shows test tubes, 1, 2, 3 and 4, in which were placed equal sized pieces of cartilage. To them were added, respectively, salt solution, tuberculous pus, staphylococci suspended in salt solution and staphylococcus pus. They were incubated for 40 hours at 55 degrees C. The cartilage remained unchanged in the salt solution and in tuberculous exudate, and the latter coagulated; cartilage in the bacterial suspension was slightly reduced in size, while that in the staphylococcus pus was completely digested. The pus in tube 4 was much more liquid than it was at the beginning of the experiment.

*The Effect of Pressure on the Bone in Tuberculous Arthritis.*—Secondary invasion of the bone in tuberculous arthritis does not usually occur as long as the articular cartilage remains little disturbed. The non-tuberculous granulations which undermine the cartilage as the disease advances are superficial and rarely invade the bone to any appreciable extent. The tuberculous granulations which undermine the margins of the cartilage and attack the bone in regions where cartilage has been completely destroyed may invade the bone to some extent, absorbing it, with resultant pits and grooves in the bony surface. Secondary invasion of bone resulting in necrosis and sequestra is of rare occurrence in the regions of the joint that are not subjected to

pressure. While cartilage is usually protected and preserved longest at the points of pressure, the bone in these regions, after joint cartilage is largely or entirely destroyed, does not fare likewise. On the contrary, it is more subject to involvement than that in any other part of the joint. When cartilage has disappeared, the pressure and friction of bony surfaces may produce extensive bony erosion, as of femoral head and acetabulum at the hip, with pathological dislocation. Occasionally in weight-bearing joints, particularly the knee, there may be sclerosis of the bone to some depth and polishing of the bare bony articular surfaces at the points of weight-bearing. At an advanced stage there is not infrequently extensive secondary invasion of bone in the zones of pressure, either before or after their coverings of cartilage have been completely destroyed. Undoubtedly the damaging influence of pressure is instrumental in producing this invasion, and if the bone is invaded on one side of the joint, it is apt to be invaded on the other side. This results in necrosis of bone, and the areas are so large that the dead bone is usually not absorbed, but is gradually sequestrated. When the condition is bilateral, it produces "kissing sequestra." If cartilage is still present on the articular surface at the time of invasion, it will die and may subsequently be removed by erosion, or it may stand for a very long time, as granulations cannot readily get at it and the exudate does not break it down by proteolysis. The articular cortex is always preserved on the sequestrum, if cartilage is still present at the time of bony invasion. The sequestra usually show some evidence of bone atrophy, which had occurred from disuse before invasion and death of the bone. After death their density remains stationary, while that of the surrounding living bone is gradually reduced as the result of continued atrophy and absorption. In the röntgenogram such sequestra are recognizable by their density, which is greater than that of the surrounding living bone, and by

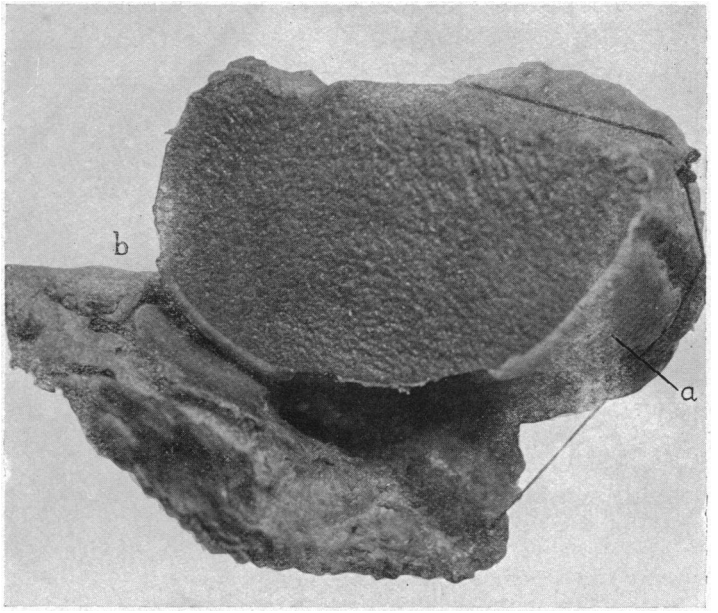


FIG. 10.—Lateral view of joint in Fig. 9 with sagittal section through lateral condyle, showing cartilage preserved in regions where femur came in contact with patella (a) and tibia (b). Cartilage destroyed elsewhere.

the presence of a shadow of articular cortex and sometimes of a zone of demarcation. If articular cartilage and cortex have been destroyed before bony invasion, there may be seen in the röntgenogram no sharp outline of the articular surface of the sequestrum. If bony sclerosis and polishing of the surface develop before bony invasion and death, the shadow of cortex and of underlying bone in the sequestrum may be very heavy. In fact, sclerosis may very rarely result in density which is greater than that of normal bone of the region. Occasionally there may be calcification of the necrotic tissue of the cancellous spaces of the dead bone, which may cause the sequestrum to cast a heavier shadow than normal bone of the region.

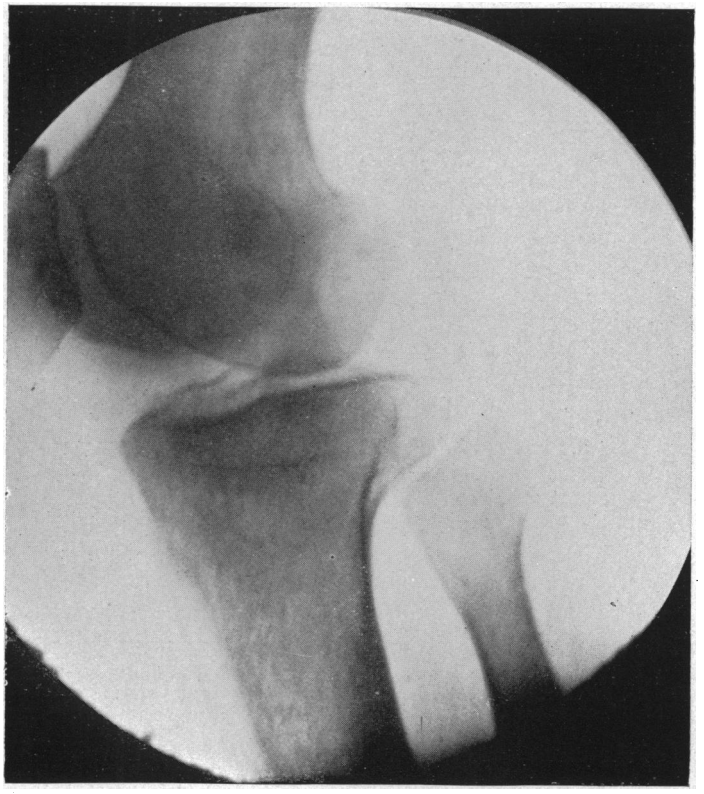

FIG. 11.—Side view of röntgenogram of joint in Fig. 9, showing cartilage space and articular cortex preserved in regions of contact of external condyle and tuberosity and of patella and femur, and articular cortex absent elsewhere.

Figure 13 is of a röntgenogram taken three years after the onset of tuberculous coxitis in a man, which had produced marked symptoms and had been treated by immobilization during the previous six months. At the top of the joint, which is its point of greatest pressure, there is an area in the head and one opposed to it in the ilium, which cast heavier shadows than the surrounding living bone and are separated from it by zones of demarcation. A definite, sharp shadow is cast by articular cortex on each dense area, while the shadows of articular cortex in the rest of the joint are absent. At operation by Doctor Ryerson extensive tuberculous coxitis was found. In the regions casting denser shadows there were two kissing sequestra. On each the articular cortex was preserved, and there was a thin layer of cartilage about the periphery of the much more extensive articular surface of the sequestrum from the head (Fig. 14). The findings indicate that articular cartilage was present on both areas when they were invaded and killed, and that the dead cartilage in the regions of contact of the two surfaces was destroyed by erosion, while that on the unopposed margins of the larger surface of the head was only partly destroyed. On microscopic examination evidence of slight atrophy of disuse was found in both sequestra, which indicates that an interval of time separated

the bony invasion from the onset of the disease. The presence of atrophy within the sequestra, their equal density, and their situation at the point of greatest pressure in the joint are conclusive evidence that they arose from simultaneous secondary invasion.

The presence of opposing sequestra in the knee-joint has been mentioned by Koenig [1] and Krause,[10] but an incorrect explanation of their development was given. Koenig assumed that one sequestrum represented the area of primary infection and that the other arose by secondary invasion across the joint after tuberculous arthritis had been established. No mention is made of the fact that they develop only in the regions of greatest pressure in the joint, nor was any comparison made of the pathological changes presented by the two sequestra. In a study of a comparatively limited number of joints operated on for advanced tuberculous disease, I have met with eight instances of kissing sequestra or opposed areas of necrosis in which sequestration was not yet complete. Seven were in the knee and one, the case above reported, in the hip. Detailed röntgenological and pathological examinations show that in every instance the bony invasion occurred simultaneously and secondarily on the two sides of the joint. That the lesions developed simultaneously is shown by the fact that the pathological changes are exactly the same on the two sides. They each present the same degrees of density and of destruction. If articular cortex is present on one, it is present on the other also. If dead articular cartilage is found on one, it is found on the other, unless the articular surfaces are of unequal size, in which case cartilage may be absent from erosion where the surfaces of the sequestra come together, but present on the unopposed portion of the larger surface.

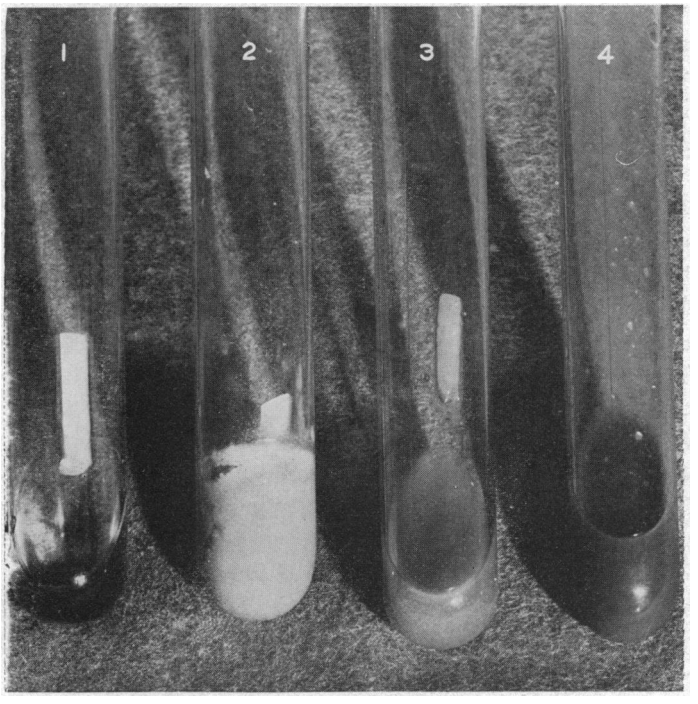


FIG. 12.—Cartilage digestion experiment. Equal sized pieces of cartilage incubated at 55 degrees C. for forty hours in normal salt solution (1), tuberculous pus (2), staphylococcus suspension (3) and staphylococcus pus (4). Cartilage unchanged in (1) and (2), slightly changed in (3) and completely destroyed in (4). Tuberculous pus (2) was coagulated and staphylococcus pus (4) was liquefied.

That both sequestra arose secondarily is shown by a number of facts. Since the pathological evidence indicates that the two lesions arose simultaneously, it is evident that they are either both primary or both secondary osseous involvements. It is inconceivable that bacteria from the blood stream would so often lodge at the same time in the bone underlying directly opposed surfaces of the joint, and always in the regions of greatest pressure. Microscopic examination of the sequestra showed evidences of atrophy of the bone before its death. In some cases the atrophy was slight, while in others it was very marked and equal to that in the surrounding living bone. The necrotic bony areas may be found attached to the surrounding living bone, with few signs of absorption and sequestration about their margins. This is evidence of recent invasion, and when the dead bone is also atrophic, the secondary nature of the lesions is definitely established.

The shape of an area of necrosis is variable. It may be that of an oval or often that of a low cone with its base bordering on the joint. Since Koenig's publications it has been the custom to regard all cone-shaped areas of necrotic bone with their bases on the articular surface as the result of embolism, clumps of tubercle bacilli, either alone or in tuberculous debris, lodging in end arteries of the epiphysis and infecting the area supplied by the obstructed artery. The recent work of Nussbaum,[11] showing that the arteries of the epiphysis, unlike those of the metaphysis, are not end arteries, has cast doubt on the correctness of this theory. It is readily apparent that areas of necrosis from secondary invasion of the bone have been confused with those from primary hematogenous invasion. Some authors, as Nichols,[12] have claimed that practically all tuberculosis of the joints arises by extension from a primary focus in the adjacent bone, and they cite the presence of sequestra and necrotic bony areas as the most important evidence in favor of the contention. The mere presence, along the articular surface, of a sequestrum or of a cavity remaining after necrotic bone has been absorbed, is not proof that the primary infection was in the bone. Careful examination from the standpoints of location, density, bilateral involvement and amount of sequestration will show that many of these bony lesions are the result of secondary invasion.

The following is another case in point: Male, age sixty-eight, had mild tuberculous arthritis of the knee for fourteen years, during which time he received no treatment and worked on the limb continuously. The symptoms then became markedly aggravated, and Fig. 15 shows the appearance of the joint in the röntgenogram eight months later. The joint was then resected, and two opposed areas of bony necrosis were found in the region of contact of mesial condyle and tuberosity, as shown in Fig. 16. That the bony invasions were approximately simultaneous and of recent date is evident from the fact that the density of the bone in the two necrotic areas and in the surrounding living bone is the same and is considerably less than normal. Gross and microscopic examinations of the regions involved showed only partial sequestration of the dead bone and an equal degree of atrophy in the dead and living bone. The röntgenographic features of tuberculous and pyogenic arthritis have been described elsewhere in greater detail. (American Journal of Röntgenology and Radium Therapy, July, 1924.)

In case of cold abscess and fistula formation in tuberculous joints, there may be invasion and infection by pyogenic organisms, greatly complicating the pathological picture. After pyogenic arthritis has been engrafted on tuberculous arthritis, the destructive effects of the former may be seen in the regions of contact and pressure and all articular cartilage killed *en masse* by the tuberculous process may be rapidly destroyed. However, tuberculous joints with fistulæ of even long standing very frequently show no evidence of secondary pyogenic infection.

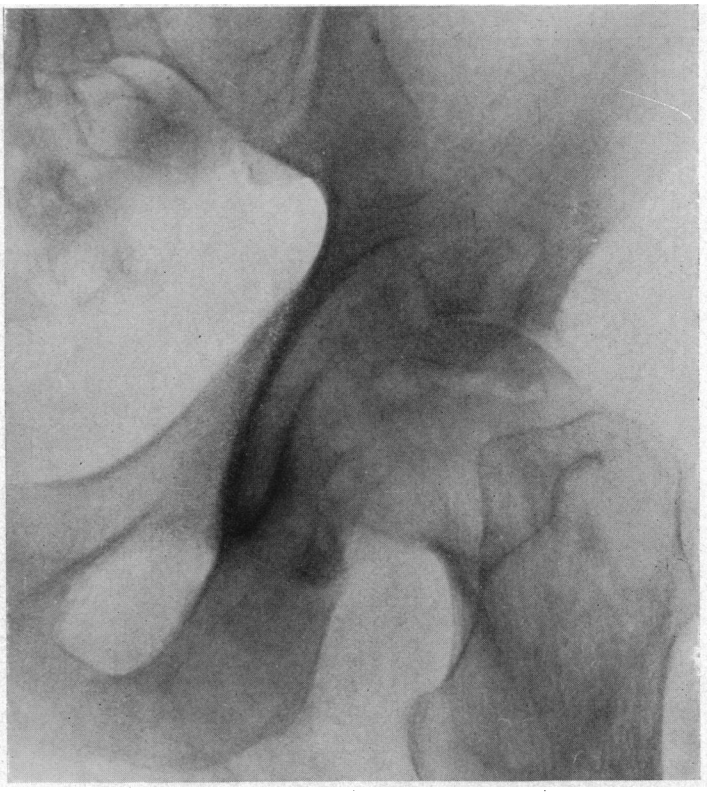

FIG. 13.—Tuberculosis of hip, showing two kissing sequestra at point of greatest pressure in joint.

*Bearing of Pressure on Treatment.*—It is readily seen from the pathological changes in articular surfaces that contact and pressure have a bearing on treatment, and that it is different in pyogenic from what it is in tuberculous arthritis.

In pyogenic arthritis the aim of treatment should be to limit and to overcome the infection and to preserve motion. The agencies which assist in the realization of one of these aims may be helpful, indifferent or harmful in the realization of the other. Pressure exerts an unfavorable influence on the spread of the infection, inasmuch as it disposes to invasion and destruction of articular surfaces at points of contact; and destruction of articular surfaces disposes to the development of ankylosis. Pressure is increased by weight-bearing and by marked effusion in the joint, distending the capsule and forcing articular surfaces together. Pressure may be relieved to some extent by drainage and by extension. Drainage acts beneficially by permitting the escape of noxious and of necrotic substances, to a slight extent by the relief of pressure. Incision should be adequate for the degree of the infection, and should be

resorted to oftener than is commonly practised in the milder cases. The danger of aggravating a severe turbid serous or seropurulent arthritis by arthrotomy plus careful post-operative dressings, except in the presence of severer neighboring infection, as osteomyelitis, has been exaggerated. Limitation of motion following loss of articular surfaces at the points of pressure only may be lessened or obviated in this way. The insertion of a drain is unnecessary where the overlying soft parts are thin, but where thick, as at the hip or shoulder, a tube should be inserted and its end sutured to the synovia. Excepting in very mild cases, pyogenic arthritis should be treated during the active stage of the disease by extension, unless it is impractical because of the location of the joint or the presence of neighboring disease. In view of the pathological changes that result from pressure, it would seem good practice to diminish or if possible entirely relieve it by extension, unless there are very strong arguments to the contrary. The main argument to be advanced against extension is that it interferes with motion. Moving the joint acts beneficially by increasing drainage and by obviating continuous pressure of opposed articular surfaces in one region. Willems [13] has advocated motion and when possible weight-bearing at every stage of arthritis, because they assist in drainage. It should be remembered that, in general, motion is harmful to infected tissues and that friction and pressure of opposing articular surfaces favor cartilage destruction. Does the advantage of drainage derived from pressure by weight-bearing outweigh the advantage of protection of articular cartilage derived from extension, and is the increased drainage produced by motion more beneficial than rest? It is a curious fact that almost no mention is made by Willems of the pathological changes that may occur in the articular surfaces and of the influences they may have on the therapeutic result, particularly as concerns mobility of the joint. It is impossible to conclude from Willems'

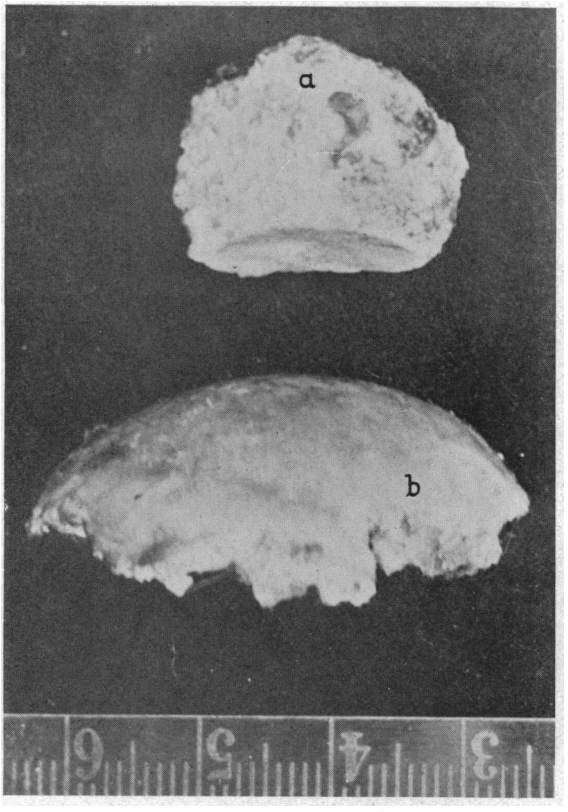


FIG. 14.—Photograph of kissing sequestra shown in Fig. 13, (a) from ilium and (b) from head.

writings whether he holds that if one follows his methods there will be no pathology in the articular surfaces or that, regardless of the extent of changes from the infection, his methods will give a better functional result than any other.

It would appear that rational management of pyogenic arthritis consists in early and free drainage and, whenever possible, extension of the joint. Mobilization with the weight lifted should be inaugurated as soon as one feels that it will be tolerated. Whether or not it should be carried out from the beginning of treatment is a question that is still open for debate. Mobilization in arthritis is often very difficult to carry out, because of the severity of the general condition, as sepsis, or of the regional condition, as osteomyelitis, of which it is a complication. In general, mobilization should be started only after the peak of the acute infection has been passed, and extension should be continued along with it until general symptoms have partially subsided and the discharge has become slight and has lost its purulent nature. By that time the danger of augmenting by pressure the infection of articular surfaces has passed and motion for the purpose of restoring function may be pushed to the limits of toleration. Röntgenograms of joints in continuous extension show that it is difficult to procure any appreciable amount of separation of articular surfaces unless considerable weight is applied. With light weight extension pressure between apposed joint surfaces may still be sufficient to cause greater breaking down there than elsewhere. Thus in a case of suppurative arthritis

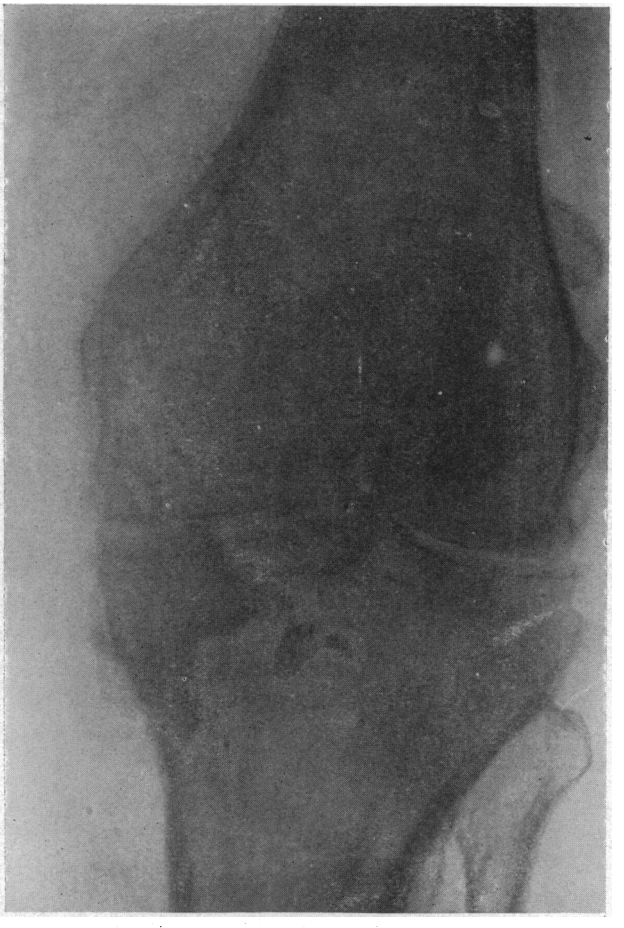

FIG. 15.—Tuberculosis of knee of fourteen years' standing in sixty-eight year old man. Secondary kissing sequestra, seen in Fig. 16, in mesial condyle and tuberosity, not recognizable in röntgenogram because both the dead and the surrounding living bone were equally atrophic.

of the hip-joint drained two weeks from the onset (during which time there must have been considerable necrosis of joint surfaces) there was later röntgenologic evidence of progressive destruction of the upper part of the head and of opposed acetabulum despite the fact that an eight pound weight extension was kept up during the ensuing six weeks.

In the treatment of tuberculous arthritis there is more variation of opinion, both as to aims that should be sought and as to the methods of achieving them, than is the case in the treatment of pyogenic arthritis. By operative treatment one generally aims to obtain healing with bony ankylosis, which, once established, is the surest safeguard against recurrence. By non-operative measures the majority of surgeons try to obtain healing with the greatest possible limitation of motion, preferably with ankylosis, while a few strive for healing in some cases with preservation of motion. Pressure plays a rôle in the non-operative treatment, in that it modifies the time and manner of cartilage destruction, and after cartilage is destroyed it disposes to erosion or extensive invasion and necrosis of bone to which it is applied. By leading to extensive necrosis of bone followed by sequestration, it may create the necessity for operative interference in the course of conservative treatment.

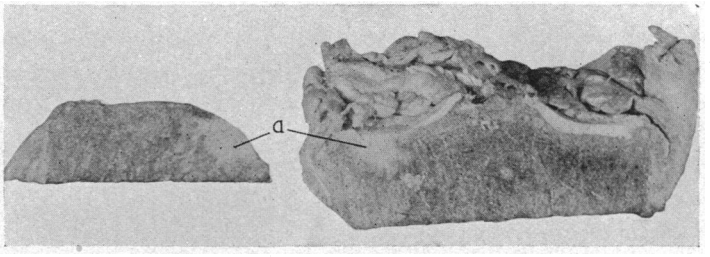


FIG. 16.—Resected specimens from joint in Fig. 15. Coronal section through tibia and sagittal section through mesial condyle, showing kissing sequestra (a) where it and mesial tuberosity were in contact. The necrotic areas of bone atrophic and partially sequestrated, showing recent development.

Immobilization without extension of the joint in the earlier stages of tuberculous arthritis should theoretically be the best method for preservation of cartilage at the points of contact and pressure. Extension would tend to pull the surfaces apart and in that particular would enable the granulation tissue to get at and destroy more of the articular cartilage. As previously stated separation by extension is difficult of accomplishment. At the same time extension brings into play the favorable factor of fixation, which tends to lessen the extent of the tuberculous changes. This makes it difficult to estimate the separate effects of extension and of fixation on the articular cartilage. If preservation of articular cartilage is what is desired, there are in the pathological findings certain grounds for believing that it is more likely to be realized from fixation alone than from fixation plus extension. Surely, those who argue that if extension is not applied, articular cartilage will soon be destroyed in the regions of contact and pressure are in error. There seem to be perhaps equally good grounds for arguing that if extension is applied, articular cartilage will be destroyed earlier than usual in those regions, because then the granulations would have a better chance to get at its surfaces. It is

even possible that some of the beneficial effects derived from extension have come from this more rapid loss of cartilage, which puts the joint in a more favorable condition for the occurrence of ankylosis. Theoretically, it would appear that healing with motion is more apt to come from the treatment of early tuberculous arthritis by immobilization alone than by extension, because immobilization alone is more apt to preserve cartilage, and preservation of cartilage is a prerequisite for motion. Lorenz and others claimed improved results from treatment by immobilization plus weight-bearing in tuberculous arthritis of the lower extremity. If this is right, it should be worth while to investigate whether the extra pressure of weight-bearing is the beneficial factor, and, if so, whether it acts by hastening the destruction of articular cartilage or by assisting in its preservation. Once articular cartilage is destroyed in a case of tuberculous arthritis, extension would seem a rational procedure, if the disease is progressive, since it should then protect the bone from erosion or extensive invasion with sequestration at the points of pressure.

Further röntgenological and clinical observations made with the anatomical changes herein noted kept in mind, should throw additional light upon the influence which pressure has upon the therapeutic results in both pyogenic and tuberculous infections of joints. They should be made on patients treated with and without extension and with and without weight-bearing.

## CONCLUSIONS

1. In pyogenic arthritis articular cartilage is killed and broken down first at the points of contact and pressure of opposing articular surfaces.

2. In tuberculous arthritis articular cartilage is not killed first, but is protected at the points of contact and pressure of opposing articular surfaces. Cartilage is extensively destroyed first along the free surfaces, where the tuberculous granulations can grow onto and remove it. It usually disappears last in the regions of contact and greatest pressure in the joint, where it is detached and killed by undermining granulations and is then partly eroded by pressure of opposing bony surfaces.

3. Proteolytic ferments derived largely from polymorphonuclear leucocytes assist greatly in the rapid removal of necrotic cartilage in pyogenic arthritis. Proteolytic ferments are absent in tuberculous arthritis, and masses of dead cartilage may persist for months or years, showing few signs of progressive destruction.

4. In pyogenic arthritis the infection rarely invades secondarily the deeper portions of the bone at the points of pressure.

5. In tuberculous arthritis invasion of the bone at the points of pressure is of common occurrence after the articular cartilage has been largely or wholly destroyed. The invasion is frequently on both sides, in which case it may lead to the formation of kissing sequestra at the points of greatest pressure in the joint.

6. Weight extension should be applied during the active period of pyogenic

arthritis to lessen the amount of invasion and destruction of articular surfaces at the points of contact and pressure.

7. On the other hand, it would appear that extension for preventing the destruction of articular cartilage in tuberculous arthritis is not indicated. But when articular cartilage has already been destroyed, extension should lessen the tendency to erosion or invasion with sequestration of bone at the points of greatest pressure in the joint.

## LITERATURE

[1] Koenig, F.: Die specielle Tuberculose der Knochen und Gelenke. Berlin, Aug. Hirschwald, 1896, vol. ii, Das Hüftgelenk, p. 13.
[2] Bittrolff: Beitr. z. path. Anat., 1915, vol. lx, p. 337.
[3] Cacace, E.: Centralbl. f. Bakt., 1901, vol. xxx, p. 244.
[4] Nussbaum, A.: Beitr. z. klin. Chir., 1924, vol. cxxx, p. 495.
[5] Mueller, E.: Zentralbl. f. inn. Med., 1907, vol. xxviii, p. 297.
[6] Jochmann and Müller: Münch. med. Wochenschr., 1906, vol. liii, p. 2002.
[7] Opie and Barker: Jour. Exp. Med., 1909, vol. xi, p. 686.
[8] Ruppel: Zeit. physiol. Chem., 1898, vol. xxvi, p. 218.
[9] Schmoll: Deut. Arch. klin. Med., 1904, vol. lxxxi, p. 163.
[10] Krause, F.: Die Tuberculose der Knochen und Gelenke. Stuttgart, F. Enke, 1899.
[11] Nussbaum, A.: Beitr. z. klin. Chir., 1923, vol. cxxix, p. 244.
[12] Nichols, E. H.: Trans. Am. Orthop. Asso., 1898, vol. xi, p. 353.
[13] Willems, C.: Surg., Gynec. and Obst., 1919, vol. xxviii, p. 546. Bull. Acad. de Med. Paris, 1917, vol. lxxvii, p. 394.