THE RECOGNITION OF DEAD BONE BASED ON PATHOLOGICAL AND X-RAY STUDIES *

By Dallas B. Phemister, M.D.
of Chicago, Ill.

When bone dies rapidly and in appreciable quantity from infection in osteomyelitis, compound fractures, tuberculosis and rarely in lues, it is at first indistinguishable either by gross or röntgenologic appearance from the adjacent living portions. Only after the occurrence of further changes in the living and the dead bone can its extent be determined. A detailed knowledge of these changes is essential for arriving at a diagnosis, especially by means of the X-rays, and for planning suitable and properly timed operations.

The changes in the dead bone are of great importance in establishing its identity. There are changes in color which are of assistance at operation. Dead compact bone turns white from the loss of circulation and of soft parts, but usually it requires some time for their absorption and the difference in appearance early between it and the pinkish living bone is insufficient to make it a reliable guide for removing the dead bone before the line of separation has begun to form. Necrotic spongy bone is frequently dark brown or red, due to the presence of old blood and necrotic marrow in the cancellous spaces where it is sheltered from attack. Granulations may grow into and be removed with a spongy sequestrum, giving it a reddish color which is sometimes difficult to distinguish from living spongy bone. By holding the sequestrum under the tap, blood and granulations are readily washed out, leaving the white cancellous bone.

Granulation tissue soon attacks the dead bone, but its activity becomes most marked after the acute inflammatory stage subsides. The tasks for the granulations are to separate the dead bone from the living, to reduce its volume or break it up by absorption and to extrude it from the field through the discharging sinuses.

Separation of dead from living bone is accomplished more rapidly than the other changes. The granulations attack both living and dead cortex along the line of junction, from both periosteal and endosteal sides, forming two irregular tortuous grooves which are gradually deepened until they meet. This results in the formation of a jagged irregular zone of demarcation two to five mm. in width, depending on the thickness of the sequestrum. These grooves are frequently seen at early operation, or in the X-ray picture as a nick or uneven dotted line in the cortex, before separation is complete. In the separation of dead

* Read before the American Surgical Association, May 5, 1920.

spongy bone, because of its loose structure, a zone of granulation tissue forms simultaneously along the entire line of junction which results in early sequestration. The time required for separation is extremely variable according to the density and thickness of the bone involved. It ranges all the way from five or six days with very thin cortex or spongiosa of the small bones to five or six months with the thickest portions of the shaft of an adult tibia or femur.

Reduction in volume of the dead portion occurs from lacunar absorbtion by the granulations along its surfaces. There is no diffuse internal loss of lime salts from dead bone, so that any remaining portions retain

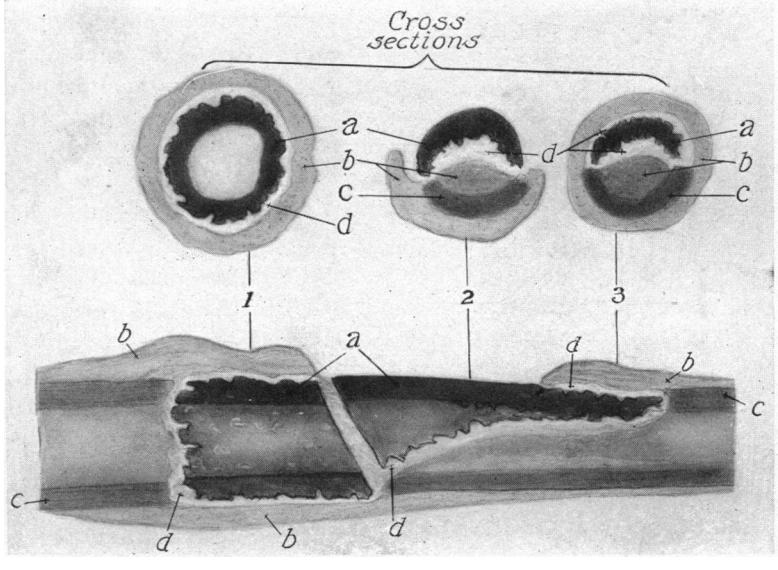

FIG. 1.—Sketch of X-ray of old infected fracture showing atrophy of surviving cortex, involucrum formation and two sequestra with well-preserved fracture lines and external surfaces that are eroded where adjacent to and little or uneroded where remote from living bone; *a*, dead bone; *b*, new bone; *c*, atrophied old bone; *d*, zone of demarcation. Cross sections show sequestra with erosion along 1, external surface; 2, internal surface; and 3, both internal and external surfaces. Differences in density well shown.

their original density. The rate of surface destruction varies greatly in different portions and is dependent largely upon the relation of the surfaces to surrounding living bone and to the channels of purulent discharge. Where living bone is in close contact with dead portions granulations springing from and supported by it attack the dead bone, producing an uneven worm-eaten surface in a comparatively short time. But those surfaces that lie at a considerable distance from living bone may show little or no signs of erosion even after long periods of time. Granulations arising from a soft part's covering attack dead bone slowly, and where they spring from the walls of a bone cavity will not bridge a wide gap and vigorously attack a relatively small enclosed sequestrum. Thus the periosteal surface of dead cortex will become extensively eroded in

those portions surrounded by involucrum, but will remain smooth or little eroded where involucrum fails to form. Similarly the condition of the endosteal surface will vary with the degree of involvement of the shaft at any level. If less than half of its circumference dies, endosteal

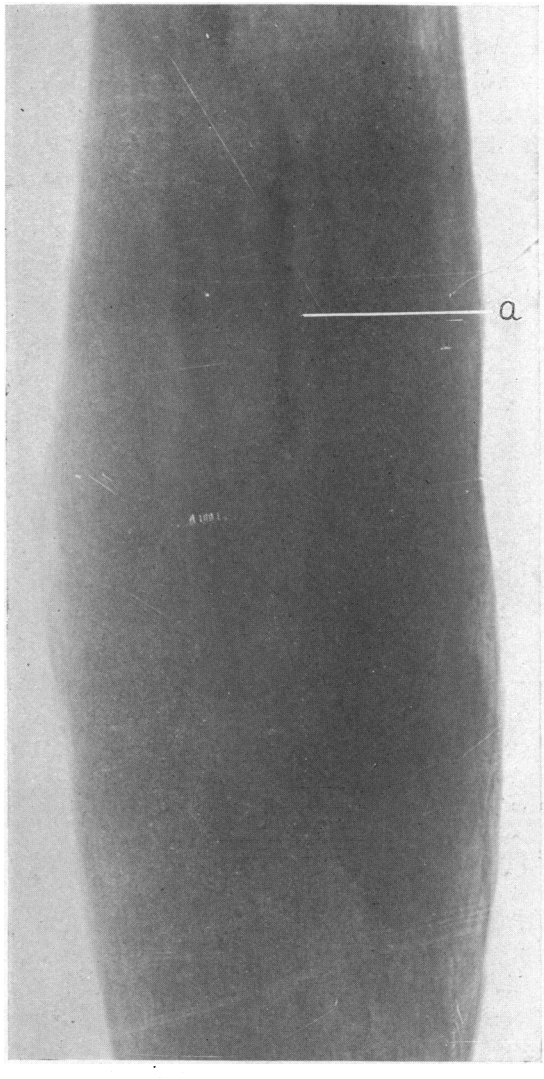

FIG. 2.—Almost stationary sequestrum in osteomyelitis of seven years' standing. *a*, Broad space between sequestrum and involucrum.

new bone will form from the surviving portion and maintain granulations in contact with the dead bone, destroying it from the endosteal surface. But if more than half the circumference is destroyed there will be frequently little or no erosion of the deeper portion of the endosteal sur-

face. When the entire circumference is dead, the endosteal surface will remain unchanged for months or even years, except at the ends of the dead tube where granulations will invade the canal for a short distance. Where an involucrum is present destruction from the periosteal side may

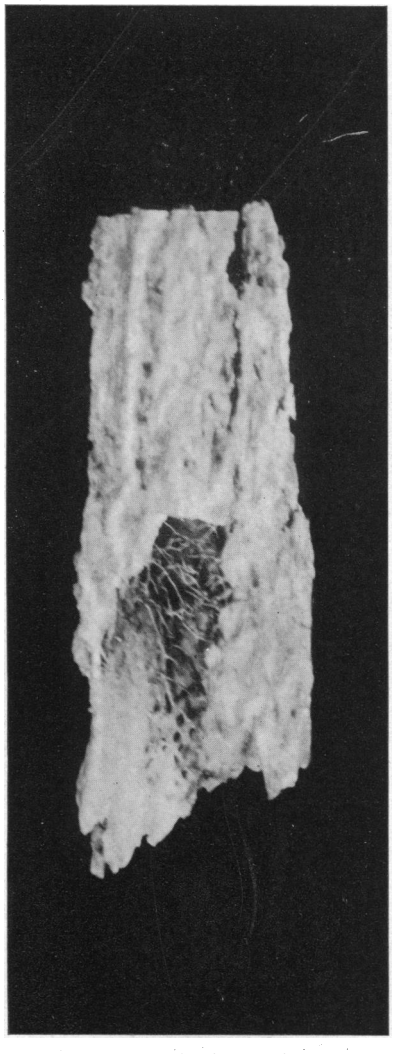

FIG. 3.—Photograph of part of sequestrum from Fig. 2. Periosteal surface markedly eroded, but endosteal surface undisturbed, as shown by presence of cancellous bone seen through window.

finally lead to perforations of the dead cortex, after which endosteal erosion from invading granulations may occur. These features are illustrated in Fig. 1, which is a composite sketch of the X-ray picture in the later stage of infected compound fracture showing different conditions of

surfaces in sequestra according to their relation to living bone. Fig. 2 shows an X-ray and Fig. 3 is a photograph of a seven-year-old sequestrum of the entire circumference of the shaft of the femur resulting from osteomyelitis at the age of ten. While markedly eroded externally, it shows through a window endosteal surface and spongy bone that had not been touched by granulations.

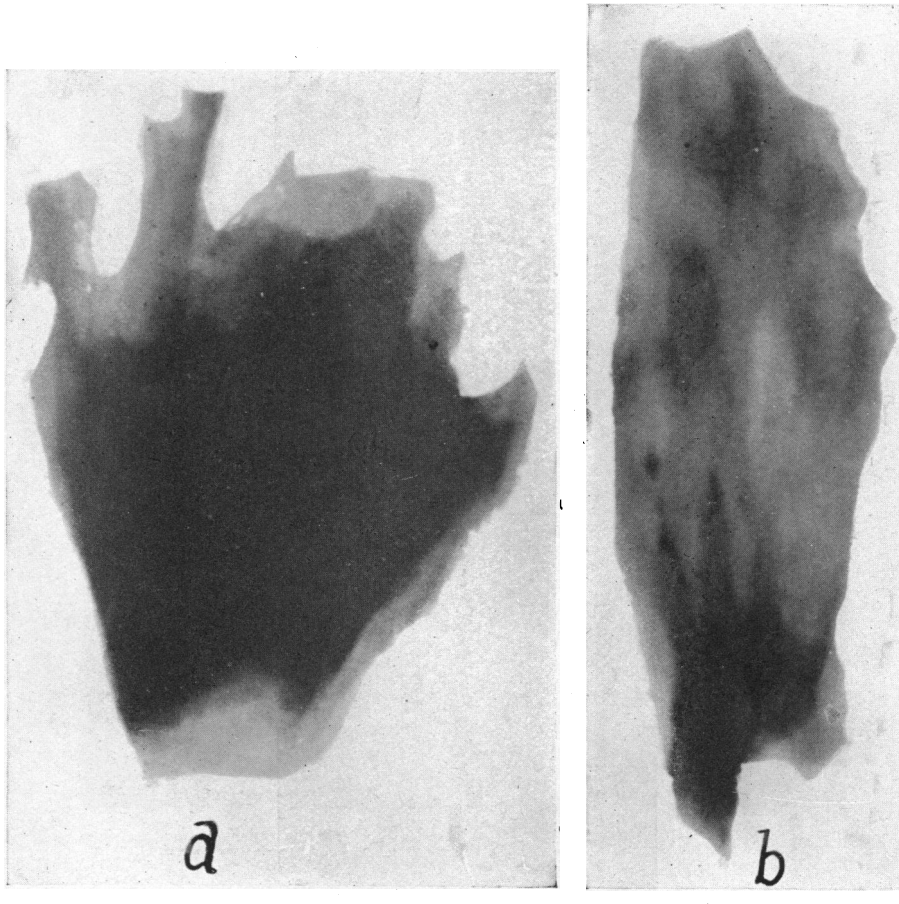

FIG. 4.—Cortical sequestra from gunshot fracture six months old. *a*, possesses original density because uneroded along periosteal or endosteal surface; *b*, eroded by granulations and density unevenly reduced.

There is little destruction of necrotic bone that occupies or borders on discharging channels, as the discharging pus in which it is bathed keeps the surrounding granulation tissue unhealthy. This is frequently seen in infected fractures where the fracture surfaces of the dead ends remain sharp and uneroded for months partly because of the restraining action of pus escaping from the deeper portions (Figs. 1 and 12–4).

Because of this unequal action of granulations upon its surface there may be marked variations in outline of different portions of a sequestrum.

Where unattacked the surface will be unchanged and the volume and density of the sequestrum will be what it was at the time death occurred (Figs. 4 *a* and 12–3). But where extensively eroded with deep pockets and sharp, irregular projections, the density will be unevenly reduced (Figs. 4 *b* and 5 *b*). Any portion that remains will have its original internal structure. These are all points of the greatest value in the X-ray diagnosis of dead bone.

The rate of destruction is greater while the dead piece is still at-

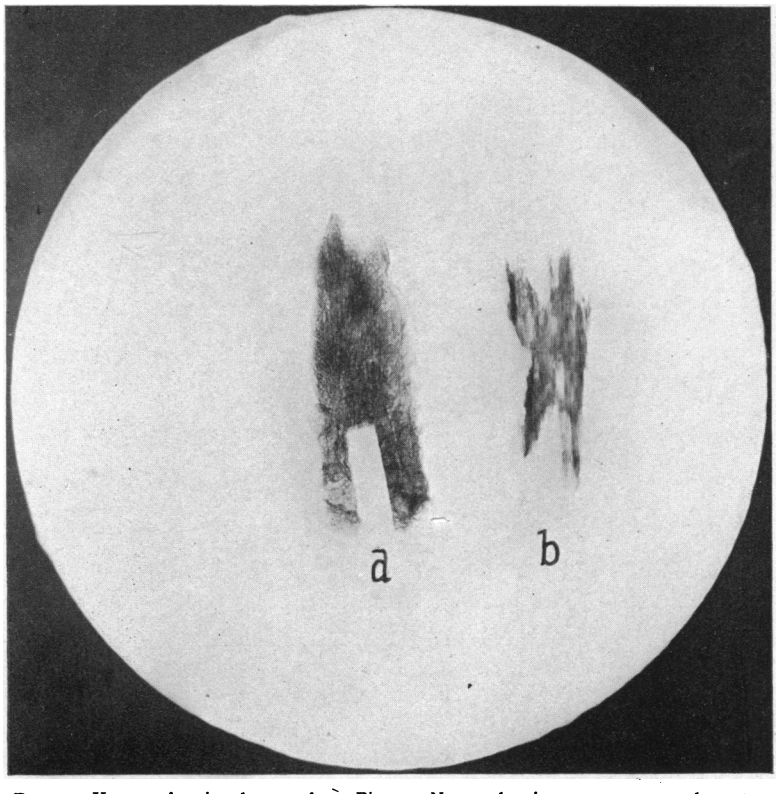

FIG. 5.—X-rays of *a*, involucrum from Fig. 13, No. 5, showing even, spongy character. *b*, sequestrum eroded, showing reduced uneven density but compact character.

tached to or incarcerated by living bone. Once it is loosened and the sequestrum moves into a freer position, especially if into a large bone pocket, destruction proceeds at a much slower rate. Splinters killed at the onset in infected fractures may be found little eroded at operation months afterward. In case of death of the entire circumference with complete encirclement of involucrum, the periosteal surface of the shaft is rapidly attacked at first, and where cortex is thin, especially in young children, it may be eaten through and fragmented, after which the pieces may be destroyed or extruded through the sinuses. But where cortex is thick, continued concentric erosion gradually leads to the development of

a wide space between sequestrum and involucrum as the latter becomes dense and does not fill in about the dwindling dead piece. This retards the action of the granulations, and such loosely enclosed sequestra may then stand for years with only slight reduction in size. This is illustrated by Fig. 2, showing a practically stationary sequestrum with a wide space between it and sclerotic involucrum.

Destruction of dead cancellous bone occurs more readily than of cor-

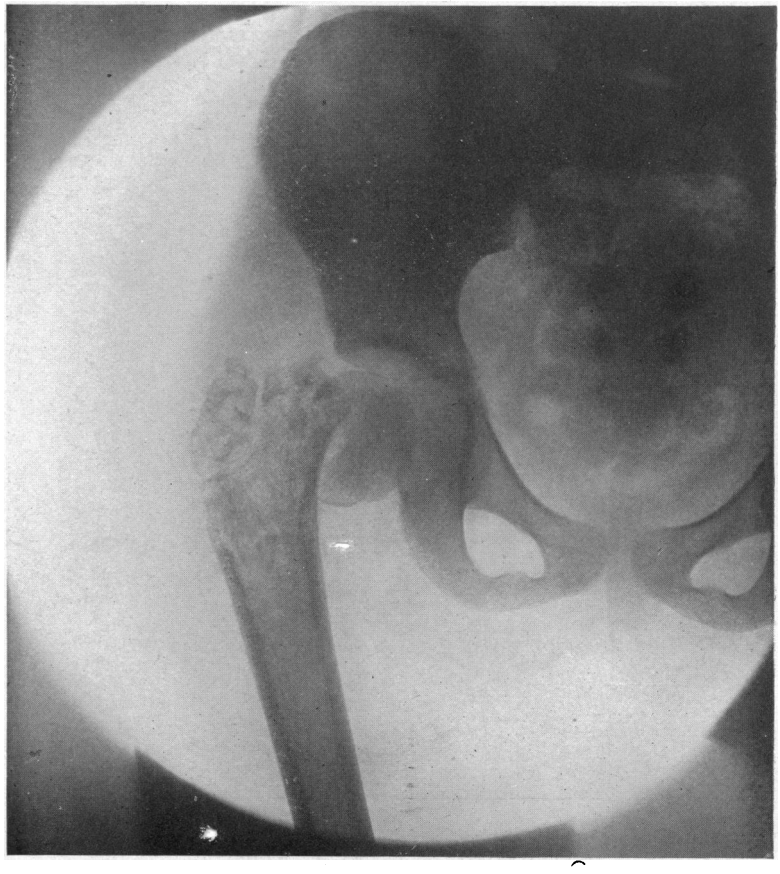

FIG. 6.—Scattered destruction in osteomyelitis of spongy portion of bone; an indirect sign of the presence of sequestra.

tex which usually gives rise to the first changes shown in the X-ray. In acute osteomyelitis of the end of the shaft the entire cancellous portion may become necrotic, but that is not the rule. Usually the dead bone is irregularly distributed and granulations developing from the adjacent surviving portions produce signs of scattered destruction. Fig. 6 shows such a condition in an eight-year-old child with osteomyelitis of the upper end of the femoral shaft of five weeks' standing and a pathological fracture of the neck. More sequestra than could be identified in the X-ray

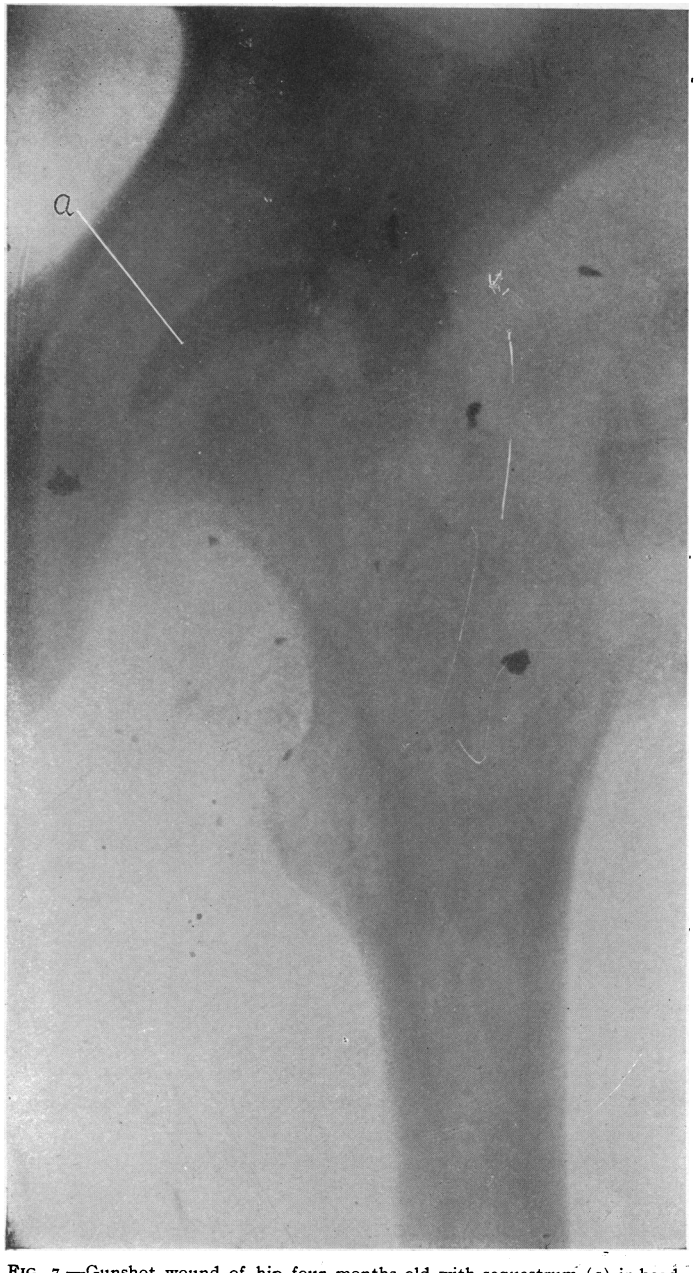

FIG. 7.—Gunshot wound of hip four months old with sequestrum (*a*) in head casting an evener and heavier shadow than atrophied surrounding bone. Articular surface on sequestrum preserved.

were found at operation throughout the region presenting signs of destruction. Smaller areas are usually completely broken down and it is rare to see large sequestra persist in this location. But in infected com-

minuted fractures of the ends of the bones, especially from gunshot wounds, large sequestra may form and persist with little more destruction than comes from formation of a wide zone of demarcation. When bordering on the joint cartilage in the presence of a complicating arthritis, the sequestrum stands out prominently because of the preservation of its original density and of the rim of bone supporting the articular cartilage, which on account of its inaccessibility to granulations, is not destroyed;

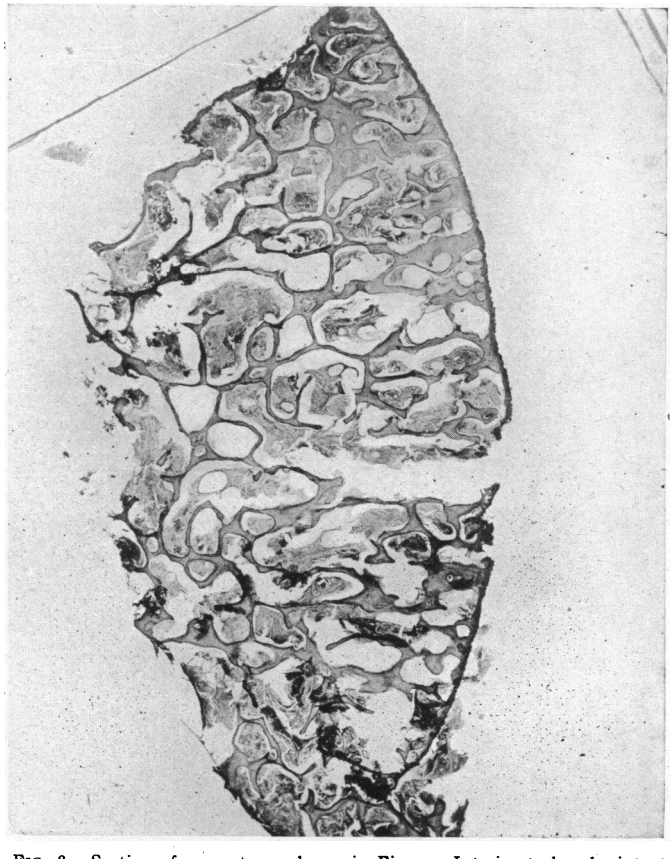

FIG. 8.—Section of sequestrum shown in Fig. 7. Interior trabeculæ intact and articular surface unbroken except in one place.

whereas that over the remaining living cartilaginous surface is irregularly broken down (Figs. 7 and 8).

A cone-shaped area of necrosis of considerable size is not infrequent in tuberculosis of the metaphysis or epiphysis and is usually broken down, leaving a cavity. When bordering on the articular surface of the joint, such a sequestrum is more apt to persist and maintain its original density. Occasionally calcification will occur in its cancellous spaces and thereby further increase its density so that it may cast a heavier shadow than the corresponding normal area on the other side.

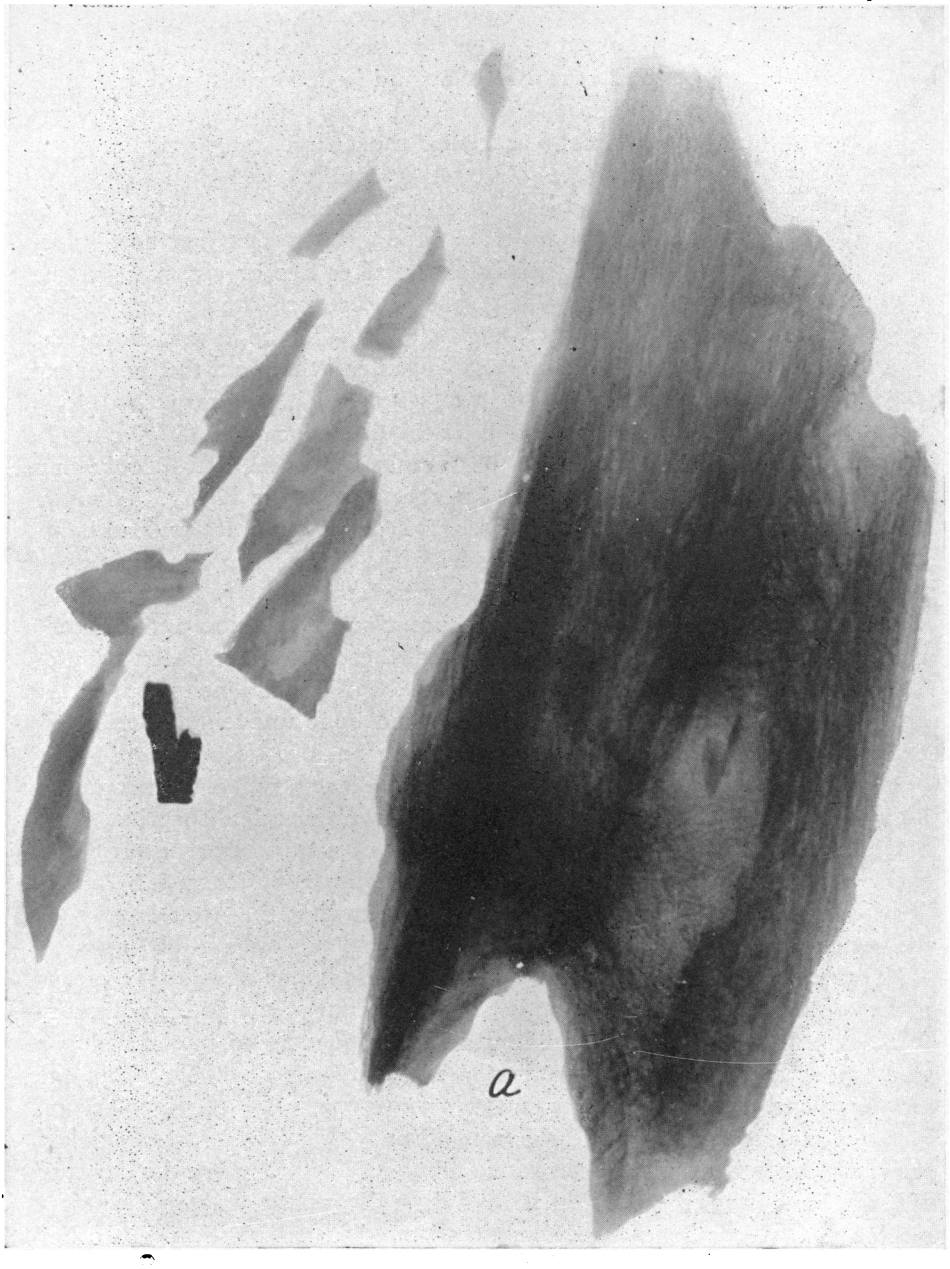

FIG. 9.—X-ray of excised wall of tunnel (*a*), sequestra and shell fragment in gunshot fracture of femur six and one-half months old. Atrophied cortex of most of wall is longitudinally streaked by dilated canals: sequestra compact.

Changes in the Living Bone.—Changes in the living bone consist in local absorption and regional atrophy and transformation of preëxisting bone, and in new bone formation.

Local absorption of living bone bordering immediately on the dead

was observed by John Hunter to be of greater importance in the process of sequestration than absorption of the dead bone itself.

Regional atrophy results from disuse and is dependent on the degree and duration of loss of function. A limited osteomyelitis producing partial loss of function for a comparatively short time, may produce little atrophy. Extensive osteomyelitis causing marked and prolonged loss of function, produces much atrophy. Infected fractures, because of prolonged and complete loss of function produced by the fracture, infection, and immobilization, frequently show, after five or six months, the highest degree of atrophy. In the case of cortex, it occurs by diffuse absorption of lime salts, mainly along the course of the haversian canals, and is slightly more marked on the endosteal than on the periosteal side. This produces an even loss in density which, when marked, may be striated longitudinally by the lines of dilated canals (Fig. 9). In cancellous bone there is reduction in numbers and size of trabeculæ in a way that frequently gives a spotted appearance in the X-ray. Atrophy of the living bone usually occurs faster than destruction of the dead bone, hence after a length of time varying with the size of the bone, the dead portion casts a heavier shadow in the X-ray than the living (Figs. 1 and 12). This relation obtains until there is resumption of function and increase in density of the atrophied portion, or until there is further destruction of the dead bone. Then the two portions may be of equal density, but in the living bone it will be evenly and in the dead bone unevenly distributed.

In case of cancellous sequestra bordering on joints this process may be reversed, as the texture of the dead bone is even, while the surrounding living bone shows areas of spotted atrophy and absorption from osteomyelitis. This is illustrated in Fig. 7, of a gunshot wound of the right hip of four months' standing with sequestrum formation in the head of the femur. Fig. 8 is of a section showing the even texture of the sequestrum.

Transformation of preëxisting bone is of less importance in the recognition of dead bone. In osteomyelitis with sequestrum and involucrum formation, the old cortex at the limits of the sequestrum may develop a greater degree of porosity than does the remaining living bone from the atrophy alone. It also gradually shifts to align itself with the involucrum, leaving the dead cortex in its original position. This shifting occurs fairly early, especially in the thin bones of children, but so late, where thick cortex of large bone is concerned, that it is of little diagnostic value. Transformation occurs late in infected fractures healed in malposition, but other signs make it possible to recognize the dead bone much earlier.

New Bone Formation.—New bone formation occurs along the course of the dead bone from the periosteum, unless there is also death of its osteogenetic elements, which is frequently extensive in infected fractures, but limited in osteomyelitis. It also occurs from the endosteum of the affected level unless the entire circumference has been killed. It extends back on the

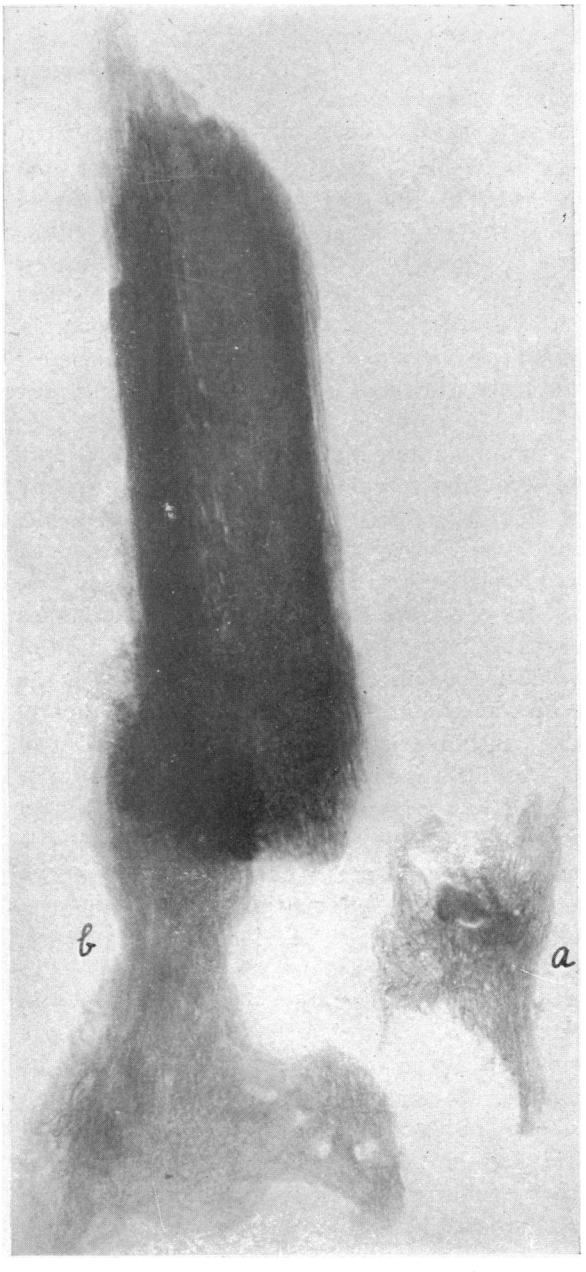

FIG. 10.—Secondary sequestrum (*a*) from wall of tunnel (*b*) in gunshot fracture of femur seven months old. Sequestrum resulted from denudation at operation five weeks before it and tunnel wall were recovered. Both have same density.

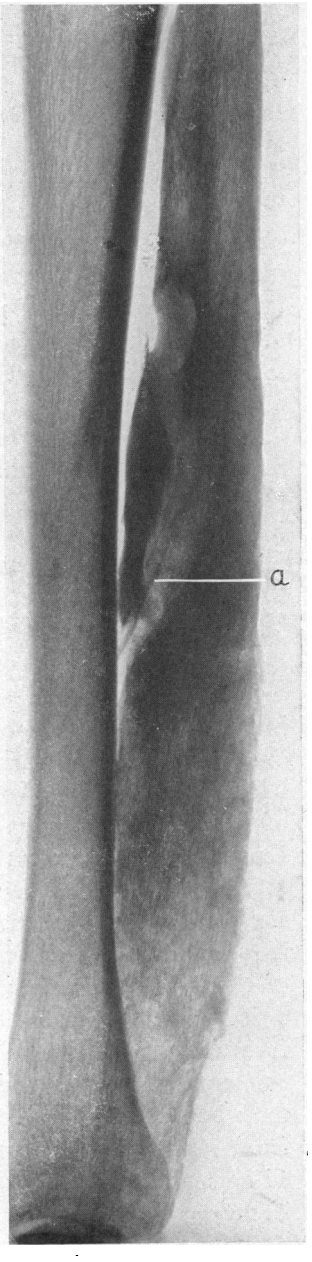

FIG. 11.—Old hypertrophic osteomyelitis of fibula, cancellous except about sequestrum. Acute recurrent osteomyelitis three and one-half weeks old in lower half producing extensive irregular absorption. At operation extensive irregular areas of necrosis and absorption found.

living shaft at the limits of the dead bone and gradually tapers off at a distance, depending on the cause of the infection. The newly formed bone is spongy early, but gradually increases in density (Fig. 5 *a*). With the resumption of function it slowly assumes a lamellated character. But if there is protracted disuse, as in ununited gunshot fractures, it may in turn undergo atrophy, vacuolation, and absorption.

There is a definite line of demarcation between the newly formed bone and dead bone, but none between it and old living bone. However, peripheral callus on the ends of living cortex may be laminated with an inner spongy and an outer compact layer, which occasionally may simulate involucrum and dead space about a sequestrum. Careful inspection of good X-ray plates will show the presence of a spongy shadow forming a narrower inner layer than is represented by the dead space about a sequestrum.

Irregular osteophytes and islands of new bone develop, especially as a result of gunshot fracture and secondary to operation with displacement of osteogenetic elements. The islands are frequently hard to distinguish from displaced sequestra, but, as a rule, have a more even density with dull fading margins, and their spongy nature can be made out in the X-ray. Displaced sequestra are usually derived from cortex and have a compact texture with sharp irregular outlines.

Secondary Bone Necrosis.—Secondary necrosis is not uncommon from a flare-up in the course of chronic osteomyelitis or from the spread of infection following operation in which extensive fresh-cut or denuded bony surfaces have been created. It differs from primary necrosis in that it usually occurs in atrophied old bone, spongy new bone, or a combination of the two. As a rule, large sequestra do not form since the infection is limited. Also further atrophy does not ordinarily occur in the surrounding living bone as is the case after primary bone necrosis. Consequently, the shadow cast by the new sequestrum is not heavier than that of the surrounding living bone and may be even fainter, as increase in density of the latter can occur from resumption of function during the time required for sequestration. Very small secondary sequestra frequently form from the cut or denuded surfaces produced at operation in chronic osteomyelitis or infected fractures. Because of their reduced density it may be difficult to distinguish them from islands of new bone formed from stripped-off osteogenetic elements or from small chips. The zone of separation forms faster in secondary than in primary bone necrosis because of the more porous character of the necrotic portion. Fig. 10 shows a secondary sequestrum *a,* which separated four weeks following operation, from one wall *b* of a tunnel, in a seven-months-old gunshot fracture of the femur. The wall was removed two weeks later and the sequestrum, placed alongside its defect, casts the same density as the portion from which it was separated.

Shaft that has first hypertrophied from old osteomyelitis and then become

porous from years of quiescence of the infection may become reinvolved from lighting up of a neighboring focus. In this case the infection will spread in the spongy hypertrophied portion and produce irregular areas of necrosis similar to that in cancellous bone of the ends of the shaft and epiphysis. The X-ray appearance is quite similar in the two conditions. This is well illustrated in Fig. 11. There had been an old osteomyelitis of the lower four-fifths of the fibula beginning nineteen years before, at the age of twelve. It discharged intermittently from the middle and lower portions for four years at which time dead bone was removed at operation and the lower portion healed. But a discharging sinus leading to the middle portion persisted. There had not been an acute exacerbation of the infection and no interference with function until three and one-half weeks before, when an acute osteomyelitis developed in the lower half leading to extensive abscess and fistula formation. X-ray shows a markedly enlarged lower four-fifths of the fibula with three distinct types of change. The upper one-fourth is enlarged and fairly evenly porous with a smooth surface. The second fourth is hypertrophied and contains an irregular canal with dense walls casting a heavy shadow in which lies a sequestrum, *a,* the lower end of which protrudes through a cloaca. The old fistula leads down to this. The surface in this region is also smooth. The lower half is enlarged and spongy, but throughout there are numerous small irregular areas of reduced density and marked irregularity of the surface, indicative of bone destruction. The entire hypertrophied portion except the external malleolus was excised subperiosteally, and the lower one-half found to be the seat of diffuse acute osteomyelitis with irregularly distributed areas of necrotic bone and bone absorption. The upper one-fourth was cancellous, but free from acute disease. No doubt the lower one-half possessed a similar but more marked cancellous structure, which permitted the acute infection, starting from the neighboring chronic focus, to spread throughout its entire extent.

Diagnostic Points in Septic Necrosis.—To sum up, the points by which we distinguish between dead and living bone are density, demarcation, and contour. These are best determined from a practical standpoint by means of the X-rays, and, after advent of the period when they may be of assistance in the management of the condition, can be expressed as follows:

The density of dead bone is greater than that of an equal volume of surrounding living bone. It retains its original compact texture. Living old bone has its density evenly reduced by atrophy and is occasionally streaked from dilated longitudinal cannular markings. Newly formed bone is of low density and spongy in texture. These are well illustrated in Fig. 12, showing a gunshot fracture of the femur seven months old. Differences in density are striking. Eight sequestra were removed. Fig. 13 is a photograph of the four large ones that could easily be identified in the X-ray and of a piece of involucrum (5) that encased sequestrum No. 2, the surface of which is markedly eroded, while that of Nos. 1 and 3,

which were not covered by involucrum, are smooth. No. 4, presenting a flat surface, was identified by its sharp fracture lines.

There are numerous variations from these general statements. Dead

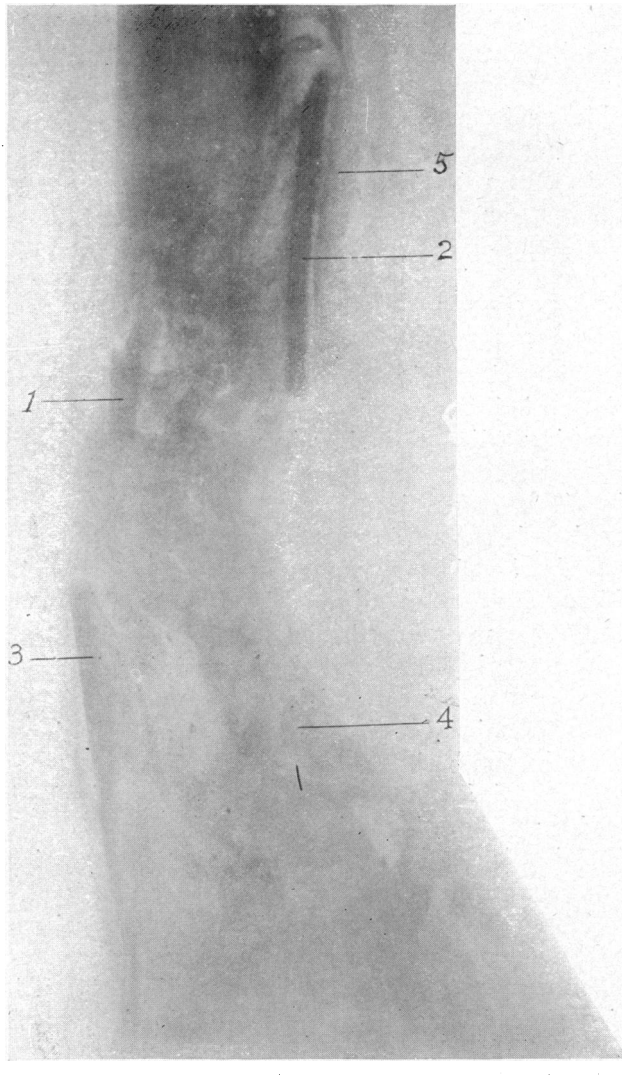

FIG. 12.—Gunshot racture of l. femur seven and one-half months old, showing extreme variations in density of dead bone, new bone and old living bone. Dense uneroded sequestra, 1 and 3, uncovered by involucrum; eroded sequestrum. 2, covered by involucrum; 5, thin sequestrum; 4, seen on flat and identified by its fracture line. Photograph in Fig. 13.

bone when extensively eroded has its shadow density reduced, which may be equal to or below that of the living bone, but is distinguished from the latter by its blotchy uneven character. Secondary sequestra usually show no variation in density from the adjacent living bone. The

line of demarcation between dead and living portions is usually sufficiently wide and clean cut to be of great value in diagnosis, but any oblique or tortuous portions, especially when overlapped by heavy living bone, may be indistinguishable or very imperfectly made out. Notches

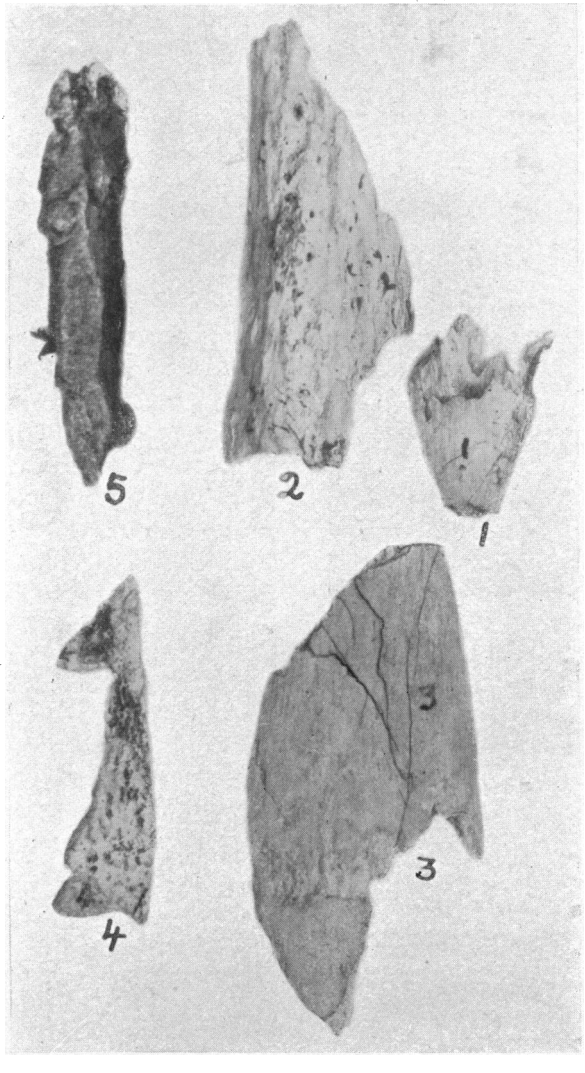

FIG. 13.–Sequestra from case shown in Fig. 12. Numbers same, but pieces reversed.

or unevenly streaked or dotted lines may indicate incomplete separation of the dead piece.

The outline of the sequestrum is of great diagnostic value. Its surface is smooth, sharp, and straight where unattacked, but irregular and jagged where erosion has occurred. Sharp spicules, especially about the

ends, are frequently to be made out. Preservation of the smooth curved cortical rim in sequestra bordering on an articular surface and of clean-cut fracture lines late in infected fractures are points of value. The compact texture of dead bone gives its outlines a sharpness that the less

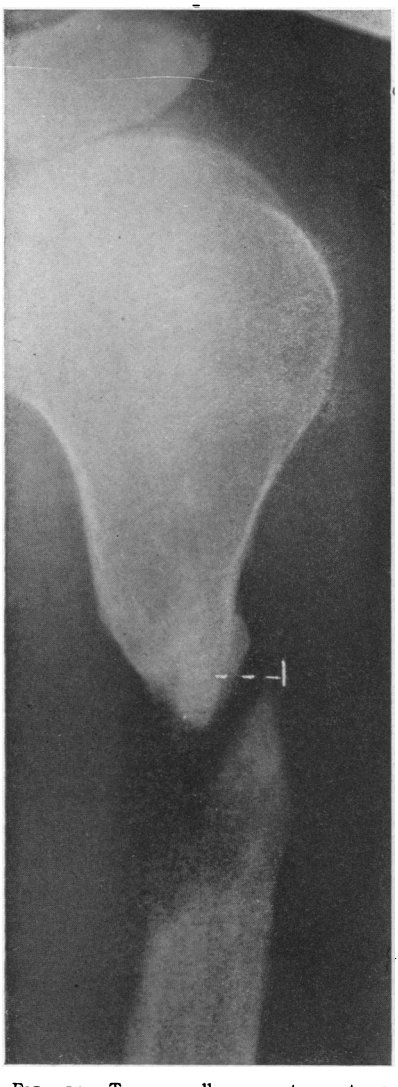

FIG. 14.—Two small sequestra at 1. Wound healed for three months.

FIG. 15.—View at a right angle to Fig. 14. Sequestra in fork of upper fragment.

dense and frequently growing living surfaces do not possess. Evidence of irregular destruction of spongy bone at the ends of the shaft in osteomyelitis is indirectly a pretty safe sign that dead portions are present even though their outlines cannot be determined.

There are many difficulties in distinguishing dead bone in the X-ray,

the greatest of which results from overlapping of shadows of necrotic and living portions which obscures the details of each. This can usually be obviated by obtaining views from different angles. Thus a line of de-

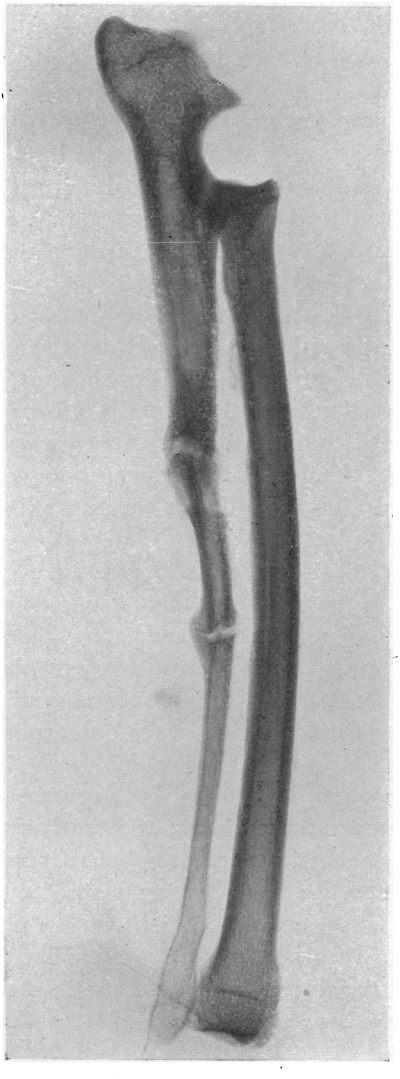

FIG. 16.—Transplant of ulna ten weeks old in adult dog. Casts heavier shadow than atrophied cortex above and below.

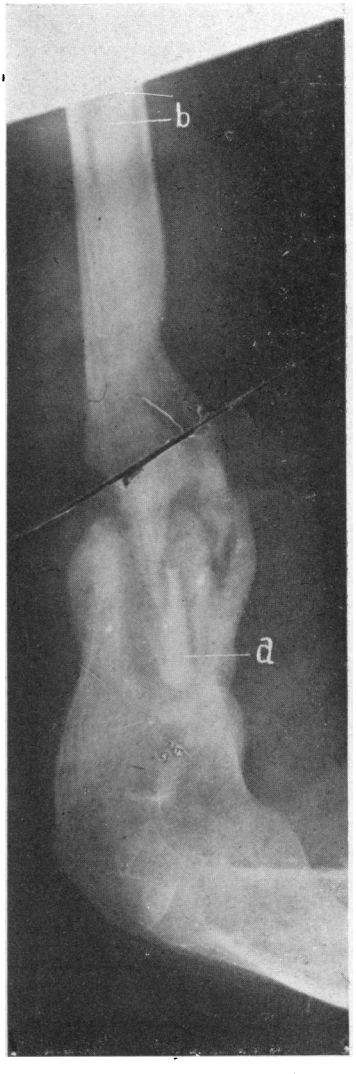

FIG. 17.—Tibial transplant *a–b* three months old in non-union of humerus. Lower 2 cm. (*a*) infected and separated as sequestrum casts a heavier shadow than the rest of the transplant which took and has been transformed.

marcation, a sharp point, or worm-eaten surface, may show plainly at one angle and faintly or not at all at another, and a flat piece of sequestrum seen from the side may show a low density and be unrecognizable, but seen on edge is easily recognized by its greater density. Thick overly-

ing soft parts frequently obscure the finer details of internal structure of different types of bone.

The presence of dead bone can nearly always be diagnosed, but frequently the exact number of pieces can not be determined, especially when they are small. It is a not uncommon experience to find, at operation in osteomyelitis and infected fractures, twice as many sequestra as were suspected from the X-rays.

Fistulæ usually persist as long as dead bone is present, because bacteria invade its canals and cancellous spaces, rendering sterilization impossible and keeping up a discharge. In marked contrast, projectiles in gunshot wounds usually heal in after subsidence of the acute infection, as their interior contains no bacteria and surface sterilization takes place. In old gunshot fractures it is common to see several pieces each of dead bone *with* sinuses at the seat of fracture, and missile fragments *without* sinuses in the adjacent soft parts. Wounds containing small sequestra may heal and remain closed indefinitely, but eventual lighting up of the infection usually occurs. Ununited fractures and defects requiring bone transplantation should be scrutinized, especially for the presence of tiny healed-in sequestra, and if such be found they should be removed and the transplantation postponed until the wound has been sufficiently long healed.

Fig. 14 shows the X-ray of an ununited gunshot fracture of the humerus healed for three months. The two small, sharp, dense spots (1) in the shadow of upper fragment suggested dead bone, consequently, a view at right angles was obtained (Fig. 15). It shows two small shadows in the fork of the upper fragment which at operation were found to be produced by two wheat-grain sized sequestra that were surrounded by moist granulations.

Density of Transplants.—The difference in density between dead and living bone in septic necrosis suggested the possibility of a similar occurrence in aseptic necrosis, such as takes place in uninfected bone transplants. Histological studies have shown that nearly all of the transplanted compact bone undergoes aseptic necrosis which, after reëstablishment of the circulation, is gradually replaced, through a process of creeping substitution, by new bone formed from the surviving unossified osteogenetic elements of the transplant in case the latter takes, or growing in from the surrounding bone where it does not take.

Atrophy would be expected to occur more rapidly in the adjacent living bone than in the transplant, because time would be required for the reëstablishment of circulation and the beginning of absorption in it with replacement of the dead cortex by new and less dense bone. Experiments on dogs, details of which will be published later, show this to be the case. A section of ulnar shaft two-thirds to one and one-half inches long, excised and reimplanted, is denser and casts a heavier shadow from the fourth to the tenth week than the adjacent atrophied fragments. This is well shown in

Fig. 16 of a ten-weeks' experiment. After this time the density of the transplant gradually approaches that of the fragments.

That a difference in density gradually develops between the infected and uninfected portions of a human transplant is illustrated by the following case: A tibial inlay graft was inserted for ununited gunshot fracture of the lower end of the humerus. Mild infection occurred with fistula formation at the seat of the fracture. Fig. 17 shows the X-ray at the end of three months. Two centimetres of the lower end of the graft underwent septic necrosis and separated as a sequestrum. It casts a heavier shadow than the rest of the graft which took and has undergone considerable transformation.