A SYSTEMATIC STUDY OF THE DEGENERATION OF ARTICULAR CARTILAGE IN BOVINE JOINTS *

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In a previous study concerning the cytology and nitrogen content of normal synovial fluid of cattle,[1] constant differences were noted in the fluid removed from the carpometacarpal and astragalotibial joints. Synovial fluid obtained from the carpometacarpal joints was more viscid, contained more nucleated cells per cubic millimeter and showed a higher total protein content than did synovial fluid obtained from the astragalotibial joints of the same animal. At that time, occasional macroscopic and microscopic examinations of these joints revealed constant areas of degeneration in the medial articular cartilages of the carpometacarpal articulations. The astragalotibial joints which were examined [1] did not reveal similar lesions. A brief comment concerning these areas of degeneration in cartilage was made. The constant differences in the synovial fluid obtained from these carpometacarpal joints as compared to the synovial fluid aspirated from the astragalotibial articulations was explained by their presence.

The present investigation was undertaken with the purpose of studying these degenerative changes in the articular cartilage from their beginning through all the stages of development and if possible of assigning the causes for their occurrence. It was also hoped that a detailed study of the initial lesions might enable us to understand better the earliest pathological changes which occur in diseases of the articular cartilage in man.

A study was made of the carpometacarpal joints in a series of embryos in order to determine whether or not constant differences in development or peculiarities in vascular supply played any part in the production of these lesions.

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Material and Methods

In all, two hundred and seventeen carpometacarpal joints of bovine embryos, calves and cattle were examined. The joints of eight embryos ranging from 5.2 cm. to 70 cm. in length were subjected to macroscopic and microscopic examination. Thirty joint specimens obtained from slaughtered calves were examined grossly. Histological studies of selected ones were made. The joints of one hundred steers and heifers between 1 and 5 years of age and of fifty older milch cows were examined macroscopically. Finally, thirty-seven specimens which showed the minimal to maximal sized lesions in the two types of animals, young steers and heifers (beef cattle) and the older milch cows, were selected. All of this latter group of specimens were used for macroscopic and microscopic study. From them, the gross and microscopic illustrations were made.

The specimens were all fixed in 10 per cent formaldehyde solution. Large or complete transverse or anteroposterior blocks of cartilage and subchondral bone were taken. These blocks were decalcified in a 5 per cent nitric acid solution and embedded in celloidin. Sections were stained with hematoxylin and eosin. A few of the embryo specimens were embedded in paraffin, as were the synovial membrane specimens. Serial sections were made from a few of the celloidin blocks, employing a modification of the technique described for use in frozen sections.[2]

Development of Articular Cartilage in the Carpometacarpal Joint

Macroscopic Description: The carpometacarpal joints of a 5.2 cm. embryo were too small to warrant gross description. In a 15 cm. embryo, the metacarpal articular cartilage measured 3.5 mm. in width by 2 mm. in depth. In the larger embryos, the articular cartilage had increased sufficiently in size, so that in the oldest (70 cm.) embryo, it measured 30 mm. in width by 3 to 3.5 mm. in thickness. Small blood vessels just visible to the unaided eye were seen in all the larger specimens of articular cartilage.

The macroscopic examination of the carpometacarpal joints of thirty-five calves which were approximately 6 to 12 weeks of age failed to reveal any evidence of articular cartilage degeneration. The surface of the cartilage was smooth and glistening (Fig. 1). Occa-

sional small blood vessels could be seen in the depth of the cartilage by superficial inspection. After shaving off the surface cartilage, a rich blood vessel and capillary network was seen readily with the aid of a hand lens. Vertical transverse cross-sections showed the articular cartilage to average approximately 1 mm. in thickness. The metacarpal articular cartilage in animals older than those already described was reduced to a fraction of a millimeter in thickness. It should be emphasized that in the metacarpal bones of the cow there is no epiphysis. Thus, the thick articular cartilage of the embryo gradually becomes transformed into a thin adult articular cartilage.

Microscopic Description: A longitudinal section through the fore leg of the smallest (5.2 cm.) embryo showed the bulk of tissue to consist of cell-poor embryonic connective tissue. In the area where the carpometacarpal articulation later develops, there had been an accumulation of mesenchymal cells. This compact group of cells had differentiated, in its central portion, into avascular embryonal cartilage, while the peripheral zone of cells appeared to consist wholly of fibroblasts. No cleavage into a joint space had occurred in the region of the carpometacarpal joint, although a well formed articular cavity had developed at the radiohumeral joint. With the exception of the humerus, where some bony matrix had been laid down, the bone anlage consisted wholly of avascular embryonal cartilage. A few blood vessels, however, were noted in the surrounding cellular connective tissue (periosteum).

Longitudinal sections through the carpometacarpal joints of embryos 15 to 59 cm. in length revealed well developed bones, articular cartilages and joint cavities. In these specimens the cartilage had differentiated into three zones: (1) a superficial or perichondral, (2) a middle or vascular, and (3) a deep or proliferating zone (see Fig. 2). These three zones merged one into another without sharp lines of distinction. Numerous mitotic figures were found in all three zones. The surface zone of cartilage consisted of three to five layers of cells, the most superficial layer of which had the morphology of fibroblasts. The middle zone was composed of irregularly shaped and placed embryonal cartilage cells. Several medium sized blood vessels were present in this zone. These blood vessels, which usually consisted of artery and accompanying vein, could be traced in all sections except two to the larger vessels in the perichondrium and periosteum at the margins of the joints and the line of fusion between the meta-

carpal bones. The deepest zone of cartilage consisted of columns of flattened cells; in the upper two-thirds the cells were rapidly dividing, whereas in the lower one-third the cells were undergoing degeneration, prior to bone formation.

Embryos of 59 and 70 cm. length showed articular cartilages which had markedly increased in width. Evidences of rapid growth were still present in the form of fairly numerous mitotic figures and incomplete ossification of the subchondral bone trabeculae. Blood vessels of large size were seen entering from the margins of the cartilage into its middle zone where they immediately branched into many smaller divisions. One noted in the study of these serial sections that many of the blood vessels just above the line of ossification were obliterated. No blood vessels were seen to enter from the subchondral bone.

Sections obtained from growing calves showed a thinner cartilage which was mature from the standpoint of both cells and matrix (see Fig. 3). Fewer blood vessels were present. The three merging layers of cells were less distinct. The cartilage cells were nearly always in pairs or groups of cells and were arranged in an orderly manner. No mitotic figures were seen. A more abundant matrix was present and the subchondral bone was more completely calcified. The line of ossification in all preceding specimens was even and (in brief) consisted of buds of capillaries growing into the degenerating columns of cartilage cells. Oval, elongated and polyhedral cells (osteoblasts) accompanied these blood vessels and a pink-staining homogeneous osteoid matrix was laid down about them. This matrix was built up into regular, evenly spaced bone trabeculae. In the specimens obtained from young calves, the subchondral bone had encroached sufficiently upon the vascular zone of cartilage, so that occasional blood vessels were being surrounded by bone above the line of ossification (Fig. 3). This overtaking of the vascular zone of articular cartilage by subchondral bone suggests the process by which adult cartilage becomes avascular. In all the embryo specimens, the transition of cartilage into bone was by direct replacement from below. In calves, a calcified layer of cartilage had begun to form (Fig. 3). In older animals the deepest layer of cartilage was calcified, as indicated by its staining reaction (Fig. 4).

Degenerative Lesions of Articular Cartilage

Macroscopic Examination: In order that the stages of development of these degenerative lesions of cartilage might be more briefly and clearly described, the specimens have been grouped into four classes for both gross and microscopic study: (1) early lesions as illustrated in gross photographs, Figs. 5, 6, 7 and 8; (2) medium sized lesions as in Fig. 9; (3) large lesions of older beef cattle as in Figs. 10 and 11, and finally, (4) defects seen in old milch cows as illustrated in Figs. 12, 13 and 14.

Gross examination of the carpometacarpal articulations of six steers and heifers (beef cattle), which because of the absence of any permanent teeth were assumed to be under 2 years of age, revealed the earliest lesions. These changes are illustrated in Figs. 5, 6, 7 and 8. They consisted of slightly depressed, roughened areas of cartilage, or small areas in which cartilage was completely absent (Fig. 8). These small erosions always occurred in the concave surface of the medial articular cartilage.

Larger and deeper areas of degeneration in articular cartilage were found in the other carpometacarpal joints collected. Fig. 9 illustrates a degenerative lesion of average size and depth for young beef cattle. The area of degeneration in this joint measured 10 by 11 mm. It was sharply outlined by an irregular margin of overhanging cartilage. The base of the defect extended into subchondral bone 1.5 mm. in the deepest portion.

Figs. 10 and 11 illustrate the largest degenerative lesions observed in beef cattle. These lesions measured 20 by 17 by 2.5 mm. and 25 by 7 by 5 mm. respectively. In both instances they had extended deeply into the subchondral bone. The similarity of the size, shape and location of the lesions in the carpal cartilage as compared to the defects in the metacarpal cartilage is well illustrated in Fig. 11. It should be emphasized that these degenerative changes always occurred on the medial half of the joint cartilage and were present in both the carpal and metacarpal cartilages. Usually one defect was the mirror image of the opposing degenerative area and striking similarities between right and left side were noted (see Fig. 10).

In old milch cows, the degenerative lesions, while located in the same areas, presented slightly different appearances. The margins were sharper and less irregular in outline. The surrounding articular

cartilage was a pale yellow in color. Various sized lesions in old milch cows are illustrated in Figs. 12, 13 and 14. The bases of several of the larger defects appeared on macroscopic examination to be covered by organizing fibrin. Histological study, however, failed, with one exception, to show any fibrinous exudate.

In occasional joints, at the site of the future degenerative lesions, minute, hard yellowish elevations were found. These nodules did not usually project more than 1 mm. above the cartilage surface and were seldom more than 2 mm. in diameter.

Macroscopically there was little evidence of synovial membrane pathology. No villous fringes were seen and for the most part the synovial membrane was smooth and glistening.

Microscopic Examination: A systematic study of sections from twenty-two joints was made. These sections showed all stages of development of the articular cartilage lesions.

The normal portions of the adult articular cartilage showed a thin layer of uniform hyaline cartilage, which was 0.7 to 0.9 mm. in thickness. The articular surface was smooth. At the surface the paired cartilage cells, which were usually two to four layers deep, were so arranged that their long axis was transverse to the vertical axis of the bone. Beneath the surface layer, the cells were grouped within lacunar spaces in clusters of from two to fourteen cells. Many of the cells were in rows and columns. The cartilage matrix appeared homogeneous, purplish in staining reaction and showed no evidence of fibrillation. The matrix at the surface and just above the calcified layer stained slightly more intensely than in the middle zone. Just above the subchondral bone was a layer of calcified cartilage, the upper surface of which was smooth, the lower border forming an irregular line of union with the bone trabeculae below. This zone of calcification was about one-third the thickness of the entire articular cartilage. Blood vessels from the intertrabecular spaces very frequently were found within this zone. The subchondral bone consisted of thick trabeculae which enclosed small spaces containing fat, numerous capillaries and small blood vessels. Normal adult metacarpal articular cartilage and subchondral bone is illustrated in Fig. 4.

The earliest constant histological change noted was a thinning of the layer of calcified cartilage. The affinity of this layer for basic stain was decreased. On the surface of the articular cartilage one

frequently observed small light staining elevations at the margins of the beginning depressions. Most of these elevations were partially covered by fibroblasts and were composed of lightly staining matrix which enclosed scattered and distorted groups of cartilage cells. In many instances, the surface cells of these elevations closely simulated the connective tissue (perichondrium) seen at the margins of the articular cartilage (Fig. 15). Associated with the above changes, one frequently observed varying degrees of fibrillation of the cartilage matrix. In its earliest stages this was shown by a basic stain-streaking of the matrix between columns of cartilage cells. The streaking and later fibrillation of the hyaline matrix was almost always in the vertical axis (see Fig. 16). Varying degrees of distortion of the rows and columns of cartilage cells were usually associated with the fibrillation. In a few instances actual vertical splitting of the matrix had occurred. Examination of blood vessels in the subchondral bone beneath these early lesions showed no constant changes. In some instances, they were dilated and engorged; in other instances they appeared deficient in number. As a result of the thinning of the calcified cartilage layer, the subchondral blood vessels were nearer to the articular cartilage proper. It should be emphasized that we never found important inflammatory cell infiltration or necrotic foci in the subchondral bone or marrow spaces.

The small, hard, yellowish elevations noted on the articular surface, when examined histologically, were found to be enlarged overgrowths of articular cartilage such as have already been described (see Fig. 16). Some of these localized outgrowths had as a result of pressure spread out to form overlapping "mushroom-like" margins. The cartilage cells in these elevations were distorted, the matrix was fibrillated and vertical clefts had formed. In some of these protuberances lime salt deposition was observed.

In slightly more advanced lesions, shallow depressions and definite thinning of the articular cartilage had occurred. The calcified zone was markedly thinned, distorted and absent in many places. The surface of these altered areas of cartilage was covered by several layers of cells which could not be distinguished from fibroblasts (see Fig. 17). In more advanced lesions, the calcified layer of cartilage had completely disappeared. All of the remaining cartilage appeared as fibrocartilage and had been invaded by blood vessels from the intertrabecular spaces of the subchondral bone (see Fig. 18). Thus,

one may state that complete degeneration of articular cartilage and repair by granulation tissue does not necessarily precede vascularization of cartilage. The disappearance of the layer of calcified cartilage appeared to be of fundamental importance.

In the medium sized lesions, as illustrated grossly in Fig. 9, the articular cartilage had completely disappeared in the sharply demarcated area of degeneration. Subchondral bone had disappeared for a depth of from three to five times the thickness of articular cartilage. At the margins of the defects there was an abrupt change from normal hyaline cartilage into fibrocartilage and fibrous tissue. This change was first apparent on the articular surface and only later in the depth of the cartilage. The bases of the defects were lined by fibrous tissue in which fairly numerous small blood vessels were seen. The underlying bone trabeculae showed evidence of atrophy and rearrangement, but no evidence of osteoclasis. The marrow spaces were filled largely with fat but showed no fibrous tissue proliferation (Fig. 19).

The largest lesions studied gave little additional information. More bone trabeculae had been resorbed so that the subchondral bone defects were six times the thickness of articular cartilage. The margins were sharp and sometimes overhanging (Fig. 20). In one instance, definite crushing of the bone trabeculae beneath the articular cartilage was observed. In occasional sections, the connective tissue lining the areas of cartilage degeneration had become very much thickened, extremely vascular, and numerous fat cells had replaced the fibrous tissue (Fig. 21).

In the old cows there was more hyalinization of the connective tissue which lined the depressed lesions. The adjacent cartilage matrix was often more intensely stained with basic dye (Fig. 22).

No important pathology was found in the associated synovial membranes. There were numerous branching small blood vessels in the subsynovial tissue, many of which showed thickened hyalinized walls. There was also a varying degree of chronic inflammatory cell infiltration in parts of the synovial membrane and subsynovial tissues. This feature, however, was never very marked. No pannus formation or synovial villi were observed in any of the joints examined.

Comment

Relatively few studies of the pathology of spontaneous joint diseases of animals have been reported. Such observations have been recorded for the most part in publications which do not readily come to the attention of those interested in the study of human arthritis. The value of an intensive study of arthritis in domestic animals is well illustrated by the work of Hare [3] who, from a careful and extensive pathological study of rheumatoid arthritis in horses (the human proliferative type of Nichols and Richardson [4]), was able to describe in detail the changes in all portions of the involved joints. While areas of degeneration on opposing cartilage surfaces were observed by Hare, they were always associated with other important joint changes which were thought by him to follow inflammatory processes in the vascular connective tissue of the joints and tendons.

Chronic arthritis specifically involving the carpal articulation of horses has been described by Cherry [5] and Krüger.[6] Although Krüger mentioned areas of degeneration of cartilage on opposing articular surfaces, there were other associated articular changes. These changes were lipping, osteophyte formation, proliferation and inflammation of the synovial membrane.

In papers dealing with the disease process termed spavin of the tarsometatarsal articulation of horses and cattle, one finds an occasional brief description of pathological changes comparable to those that are the subject of this paper. However, the process appears to have been entirely different in that various workers [7, 8, 9] have directed attention to the inflammatory nature of the disease and the tendency to ankylosis of the involved joints.

Lesions such as are described in the present study are not mentioned by Hutyra and Marek [10] who, in discussing articular rheumatism of domestic animals, stated that it was most frequently seen in cattle. In cattle this disease affected delicate milkers most commonly, less often oxen, and was scarcely ever encountered in grazing cattle. It was assumed that the disease described (articular rheumatism) was due largely to bacterial infection.

The present investigation deals with progressive lesions of the articular cartilage of cattle which occur on the opposing articular surfaces of the medial side of the metacarpal and carpal bones. These

lesions were self-limited and not accompanied by any important pathological changes in other portions of the involved joints.

Possible etiological factors concerned with the degenerative lesions of the carpometacarpal articulations have been considered. The gross and microscopic examination showed that they were of a degenerative nature. No lesions were ever found in the carpometacarpal joints of calves slaughtered at the age customarily used for veal. The earliest lesions were found in young beef cattle (under 2 years of age), and the more extensive lesions occurred in some of the older animals. It is important to emphasize that the lesions did not necessarily progress once they had developed. Having reached their maximum size, they remained as depressed areas with little or no attempt to repair.

The degenerative changes in articular cartilage appeared to be unrelated to vascular changes as factors of causation. From the study of embryo and calf specimens it was observed that the blood supply to both the medial and lateral articular cartilage disappeared at the same time, yet the areas of degeneration occurred only on the medial cartilage. These blood vessels had all disappeared in the beef cattle before the age of 2 years. Examination of the blood vessels in the subchondral bone beneath the developing defects in cartilage did not reveal any abnormalities which were constant or of histological importance. The arterioles in the fibrous tissue lining the areas of degeneration (once formed) were often thick-walled and hyalinized. This factor was thought to be secondary to the alterations already present rather than to be in any way responsible for them.

Bacterial infection may be dismissed as a causative factor because of the constant occurrence of this lesion in all animals, the constant involvement of one localized area of the articulation, and because there is no histological or cytological evidence of infection to be found in the synovial membranes, articular cartilage, subchondral bone, or synovial fluid.

Gout as an etiological factor was ruled out since deposits of sodium urate crystals never were found upon articular surfaces or in the articular cartilage of either early or late joint lesions.

The constant occurrence of the localized articular cartilage degeneration in young western beef cattle and older milch cows * ap-

* These cattle were obtained largely from the New England states.

pears to rule out differences of activity and habitat as being of importance in causation.

The study of embryo joints failed to reveal anything unusual in the development of the carpometacarpal joints. It was learned from these specimens, however, that the articular cartilage rapidly decreased in thickness in the early months of life. When large transverse sections of the metacarpal articular cartilage and subchondral bone of embryos, calves and cattle were mounted in series on large glass plates and studied with a hand lens, interesting structural changes in the subchondral bone were noted. In the embryos and calves, the subchondral bone trabeculae were evenly distributed beneath the articular cartilage (Fig. 23). In the older animals one noted a rearrangement of the trabeculae and an increase in the thickness of the bone cortex. There was a marked difference in the manner in which the compact cortical bone expanded into the subchondral bone trabeculae of the medial and lateral sides (Fig. 24). The lateral cortex of the metacarpal bone flared sharply from the vertical axis, so that the vertical subchondral bone trabeculae supported adequately the entire lateral articulating surface, whereas the vertical subchondral bone trabeculae branching from the mesial cortex supported only the medial one-half of the medial articular surface. The central area of the entire articular surface was well supported by the dense bone which resulted from the fusion of two metacarpal bones. This rearrangement of bone trabeculae resulted in an area of rarefied subchondral bone which was directly beneath the site of these cartilage defects. These areas of rarefied subchondral bone were continuous with the marrow spaces of the bone metaphysis. The fact that the articular cartilage defects occurred directly over these areas of rarefied bone suggests that they are potential weak spots and therefore of great etiological importance. Such a deduction is further substantiated by the fact that the few remaining bone trabeculae underlying the larger lesions had been so arranged that they paralleled the surface cartilage. The latter structural arrangement was apparently a compensating phenomenon.

The gross structure of the carpometacarpal articulation of the cow seemed equally as important etiologically as the structural rearrangement of the subchondral bone. For this reason, the corresponding joints of the horse were incorporated in this study. Such specimens were not similarly involved even though the majority of them were

the seat of marked arthritic changes. From the mechanical standpoint, the carpometacarpal joint of the horse is more nearly perfect than is the corresponding joint of the cow. In the case of the former animal there are more articulating concavities and convexities which would aid in preventing anteroposterior or lateral slipping and the posterior portion of the joint is strengthened by two very strong articular ligaments. The metacarpal articulating surface of the cow is comprised of two large flat surfaces which are divided by a single narrow anteroposterior ridge situated just lateral to the midline. Only one articular ligament connects the carpal and metacarpal bones in cattle. In the cow one finds a considerable degree of genu valgum of the fore leg. Because of this factor, it would appear that the greatest weight is borne on the medial aspect of the carpometacarpal joint. Repeated traumatic injury applied to the articular surfaces of a weakly constructed joint might well explain the lesion in question, particularly since such lesions occur regularly over the area of least bony support. A probable source of repeated trauma is found in the manner in which a cow uses the front knees (carpometacarpal articulations) in the process of lying down and getting onto her feet from the recumbent position. In lying down, the cow drops the weight of the fore quarters upon one sharply flexed carpometacarpal joint and then upon the other. In rising, the front legs are folded under the thorax and the weight of the fore quarters of the animal is carried on the front knees until the hind quarters are upright, when with great exertion, the fore quarters are lifted from one front knee at a time. This series of movements is entirely different from those of the horse where the fore legs are extended forward and the weight of the fore quarters lifted onto the fully extended fore legs with the aid of forceful pushing by the rear extremities. If the above observations are indicative of the stresses applied to the carpal and metacarpal bones of the cow, then these articular cartilage lesions may well be considered as traumatic in origin.

It has been previously stated that the degeneration involved similarly the two opposing cartilage surfaces. This was true in both the early and the more extensive lesions. While this fact is apparently in agreement with a theory of traumatic origin, it does not explain why the two opposing depressed areas extended deeper, once formed. This phenomenon may be explained as follows: Once the lesions are formed, the weight is carried on the surrounding intact articular sur-

face, where the underlying bone is more directly continuous with the solid cortical shaft. Atrophy and resorption of the bone underlying the cartilage lesions might well take place because the pressure stimulus had been removed.

The histological and gross study showed that the lesions under discussion were not the same as the pathological changes described in any type of human arthritis. The fact that these lesions began as areas of articular cartilage degeneration makes the process more nearly comparable to the degenerative arthritis of Nichols and Richardson [4] than to the proliferative type described by them. They described the degeneration of articular cartilage on one joint surface and a compensatory overgrowth of cartilage and later bone on the opposing joint surface. This compensatory overgrowth of cartilage allowed continued apposition of the involved joint surfaces. In the lesion found in the cow, the degeneration occurred on opposing areas so that continued apposition of the defects was not possible. Nichols and Richardson described thickening and eburnation of the underlying bone. Such changes were never encountered in the joints of cows under study. The degenerative type of arthritis is a disease of older individuals and more common in women. Sex and age (with the exception of the necessary first two years of life) certainly play no part in the incidence of this disease in cattle.

Following the thinning of articular cartilage and rearrangement of bone trabeculae of the metacarpal bones in young cattle, degenerative lesions developed. The sequence of pathological changes leading to degeneration of cartilage appeared to begin with thinning and disruption of the layer of calcified cartilage. Fibrillation of the cartilage matrix followed and was accompanied by replacement of the surface articular cartilage with connective tissue. Disappearance and distortion of cartilage cells was then noted, together with more marked fibrillation of the articular cartilage matrix. Splitting of the fibrillated matrix followed in some instances. Blood vessels grew into the altered cartilage through gaps in the calcified zone, entering from the intertrabecular spaces of the subchondral bone. The altered articular cartilage eventually completely disappeared and varying degrees of subchondral bone resorption followed. The cartilage and bone defects became lined with a vascular connective tissue which resembled synovial membrane to a certain extent.

Summary

1. Constant differences in the synovial fluid of the carpometacarpal and astragalotibial articulations of the cow have been described in a previous publication.[1] The finding of areas of degeneration in the articular cartilages of the carpometacarpal articulations of all cattle over 2 years of age would appear to be an adequate explanation of the synovial fluid differences observed.

2. These areas of progressive degeneration in articular cartilage have been studied systematically and the successive changes have been described and illustrated.

3. The development of the carpometacarpal articulations was studied in a series of bovine embryos and calves. The vascular articular cartilages of embryos and calves became avascular before the animals attained the age of 2 years. Pronounced rearrangement of the subchondral bone trabeculae resulted in a relatively deficient bony support of the medial articular cartilage where the degenerative lesions occur.

4. The possible etiological factors of such cartilage lesions have been discussed. It was concluded that they were probably due to repeated trauma in weakly constructed articulations. Deficient subchondral bone support was thought to be an important predisposing factor.

5. The type of cartilage lesion described in this paper is not wholly similar to any of the joint lesions described in human arthritis.

Note: We wish to thank the New England Dressed Meat and Wool Company for the material used in this study.

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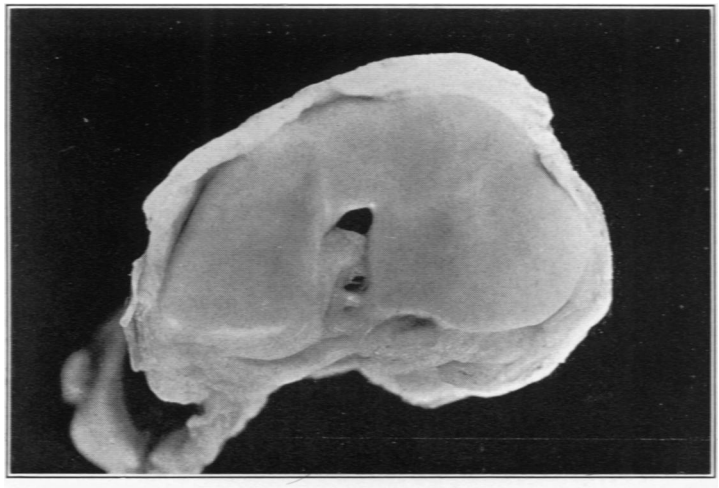
DESCRIPTION OF PLATES

PLATE 70

FIG. 1. A natural size photograph showing the normal metacarpal articular cartilage of a young calf.

FIG. 2. A photomicrograph of very low magnification showing the entire thickness of cartilage of a 59 cm. bovine embryo. Note the three merging zones of cartilage: (*a*) superficial or perichondrial; (*b*) middle or vascular; (*c*) deep or expanding. × 32.



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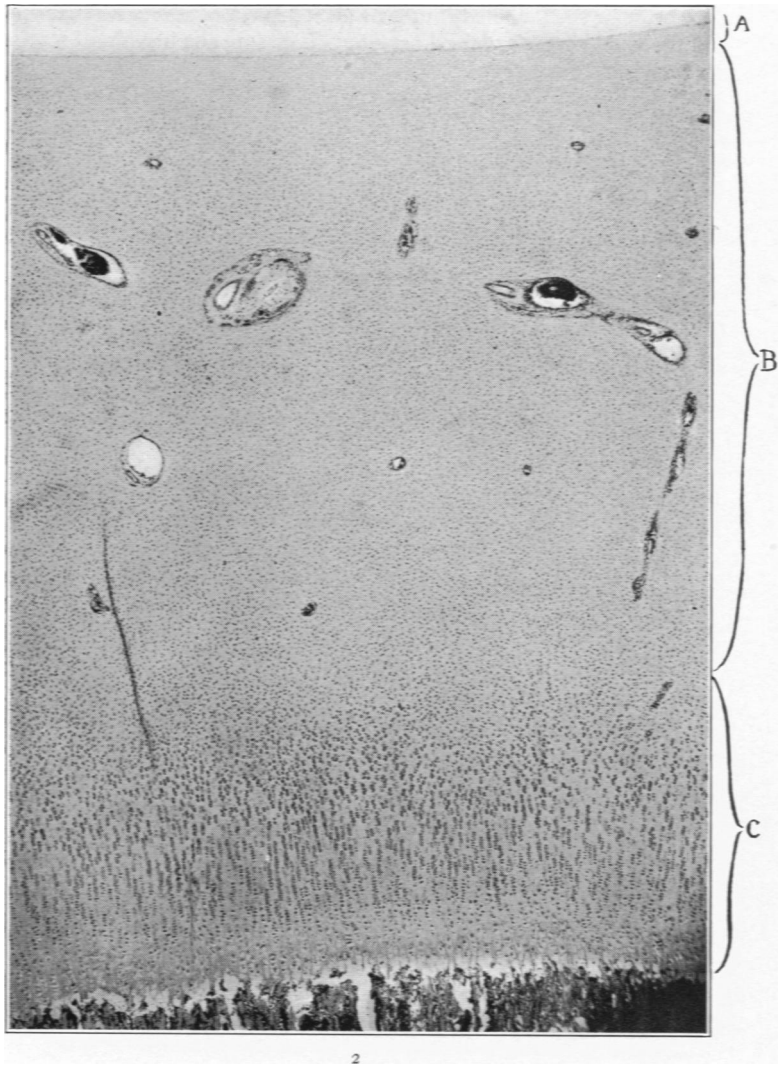

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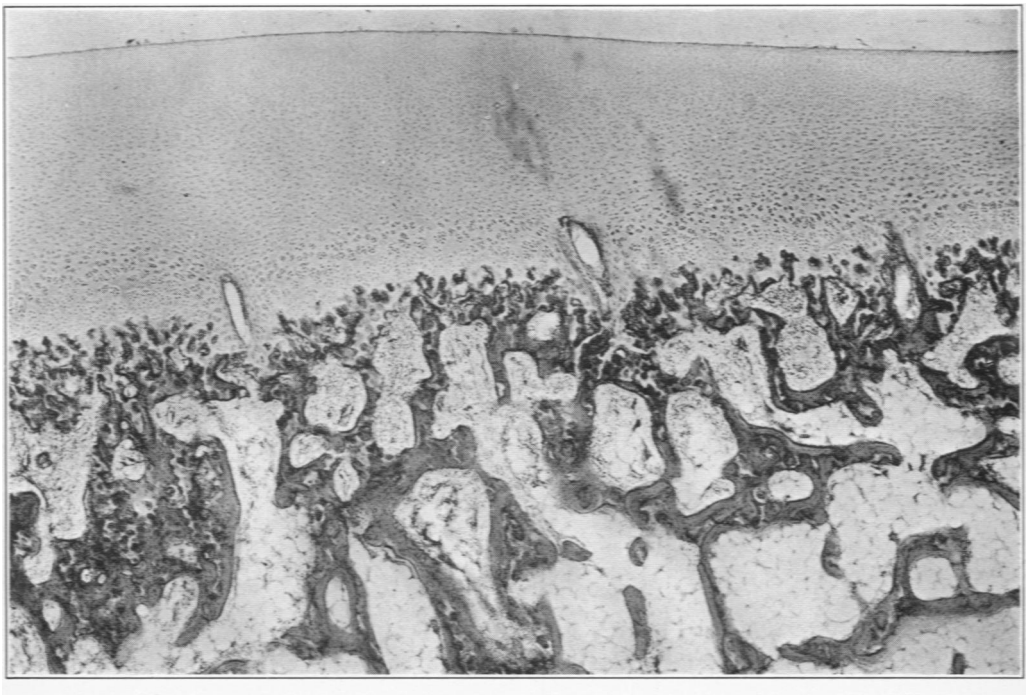
PLATE 71

FIG. 3. A low power photomicrograph of the metacarpal articular cartilage of a calf. Note the reduced thickness of cartilage (as compared to Fig. 2), the prominent perichondrial border, and the incorporation of three blood vessels by subchondral bone growth. The layer of provisional calcification is well formed. × 37.

FIG. 4. A photomicrograph of a normal portion of adult articular cartilage of the cow. One should note the orderly arrangement of cells in lacunar spaces, and the wide zone of calcified cartilage. The subchondral bone trabeculae are broad and very dense. × 84.



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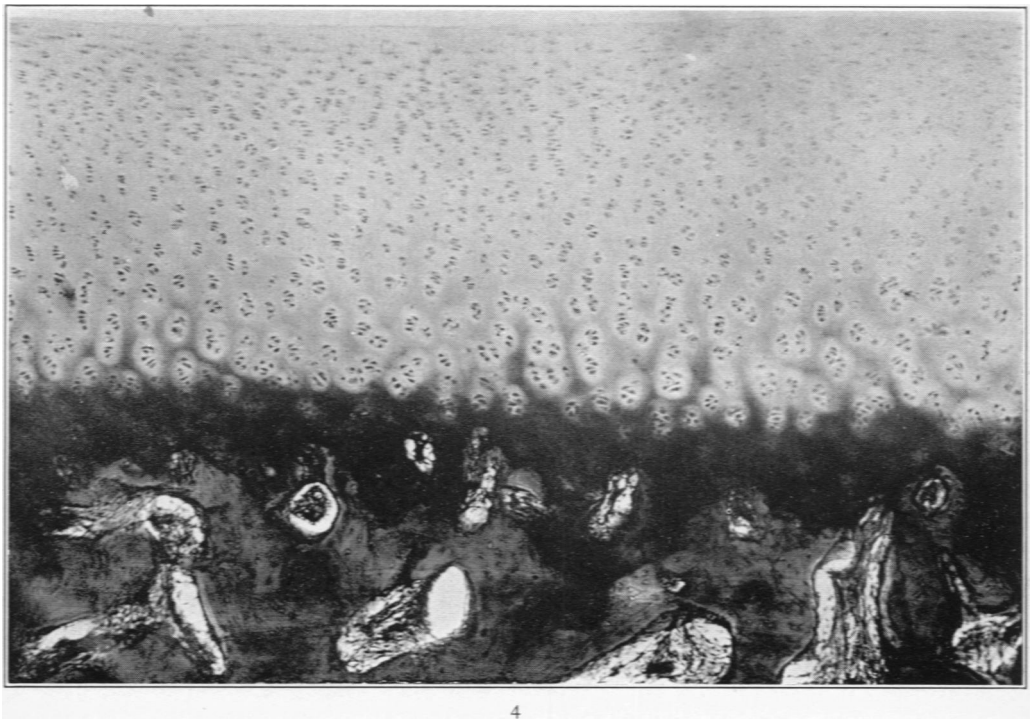

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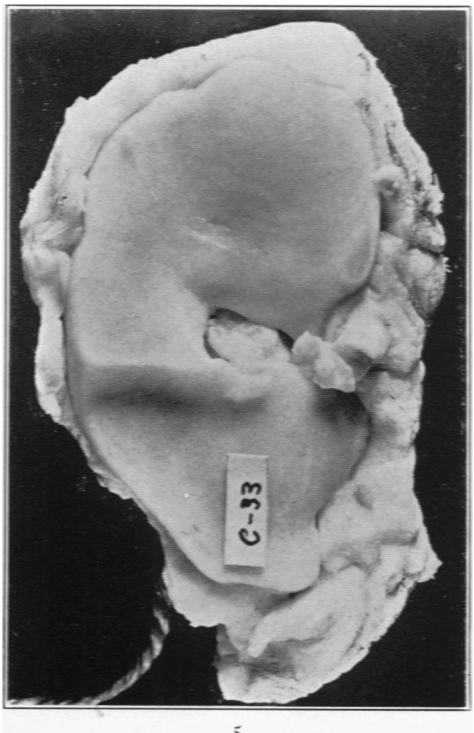
PLATE 72

FIG. 5. A metacarpal articular cartilage of a young steer (under 2 years of age). Note the early roughening of articular cartilage in the center of the medial surface. Natural size.

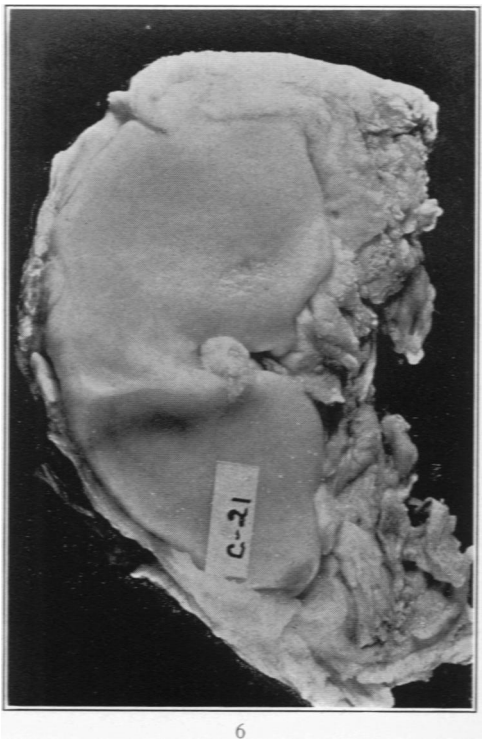
FIG. 6. The medial articular cartilage of this joint shows an area of roughening and thinning which is slightly larger than the defect in Fig. 5. Natural size.

FIG. 7. The central area of the medial articular cartilage is more depressed and the cartilage has been more completely destroyed than in the earlier illustrations. Natural size photograph.

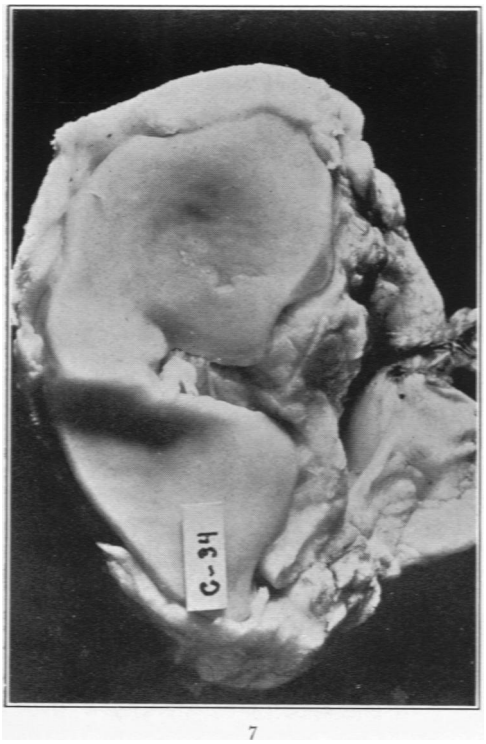
FIG. 8. A natural size photograph of a slightly larger area of degeneration in articular cartilage. This lesion extended down to subchondral bone in a few areas.



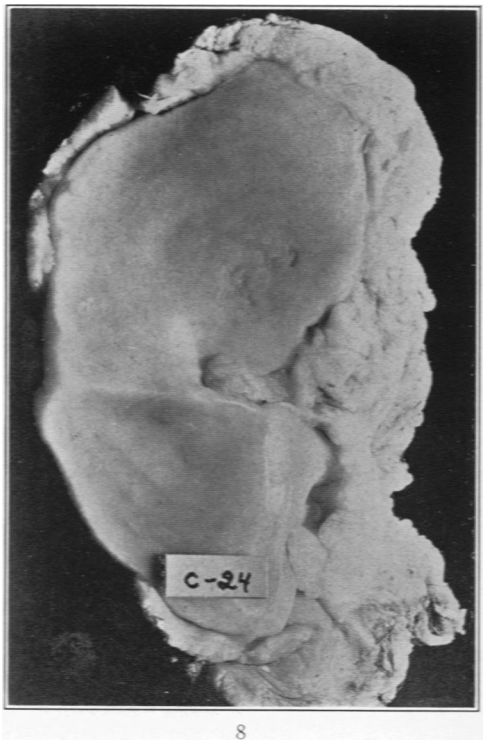
5



6



7

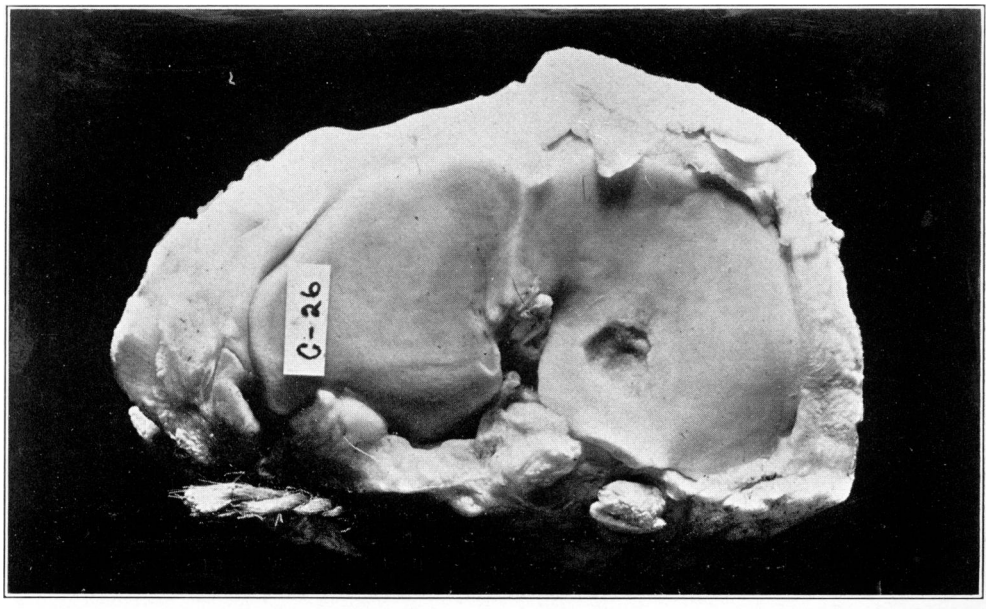


8

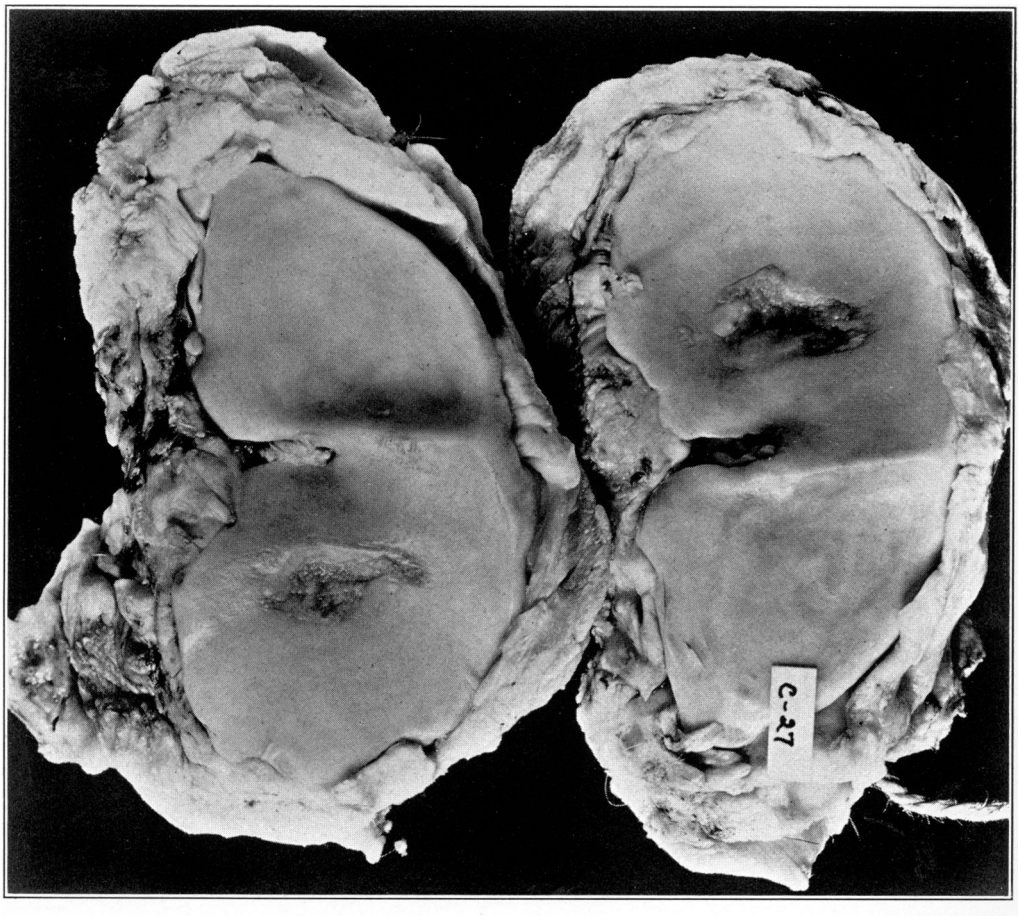
PLATE 73

FIG. 9. A natural sized photograph showing an average sized lesion in articular cartilage which extended into subchondral bone. Note the sharp overhanging margins.

FIG. 10. The articular cartilage surfaces of the right and left metacarpal bones of the same steer. Note the similarity in size, shape and location of the degenerative lesions. Natural size.



9

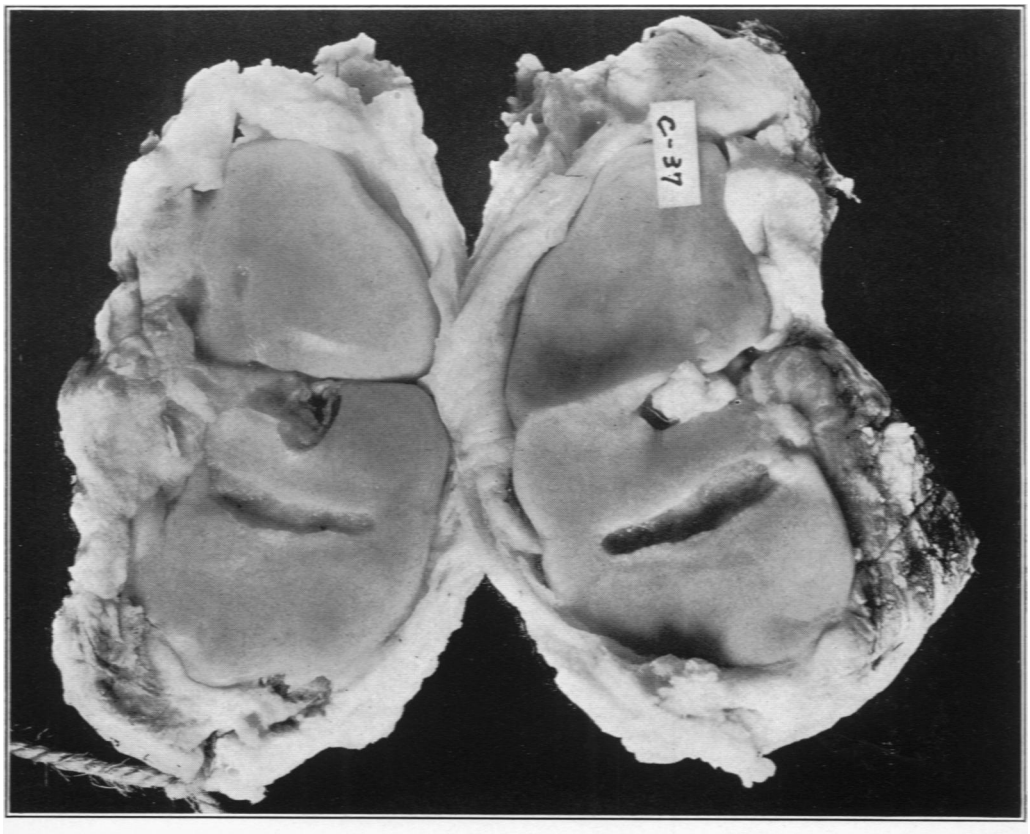


10

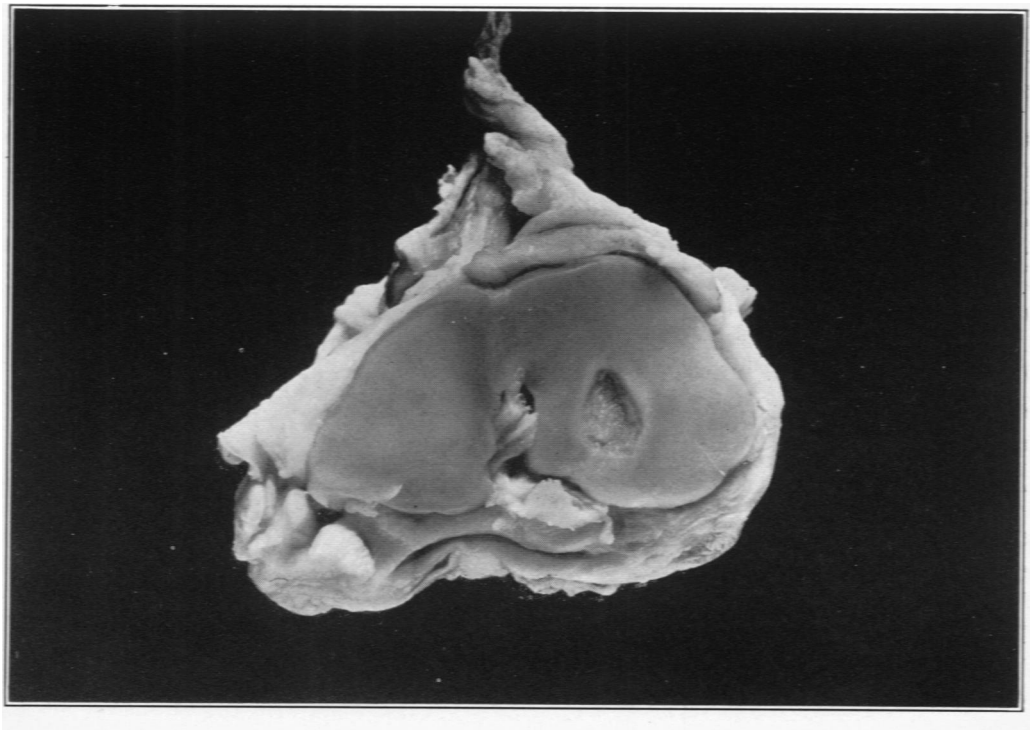
PLATE 74

FIG. 11. A carpometacarpal articulation opened in such a way as to show the opposing carpal and metacarpal articular surfaces. The similarity of size and type of degenerative lesion is apparent. Natural size.

FIG. 12. An average sized area of degeneration in cartilage and subchondral bone of an old milch cow. Note the light marginal halo produced by a yellow coloration of the articular cartilage at the margin of the lesion. This feature is peculiar to the lesions in older cattle. Natural size.



11

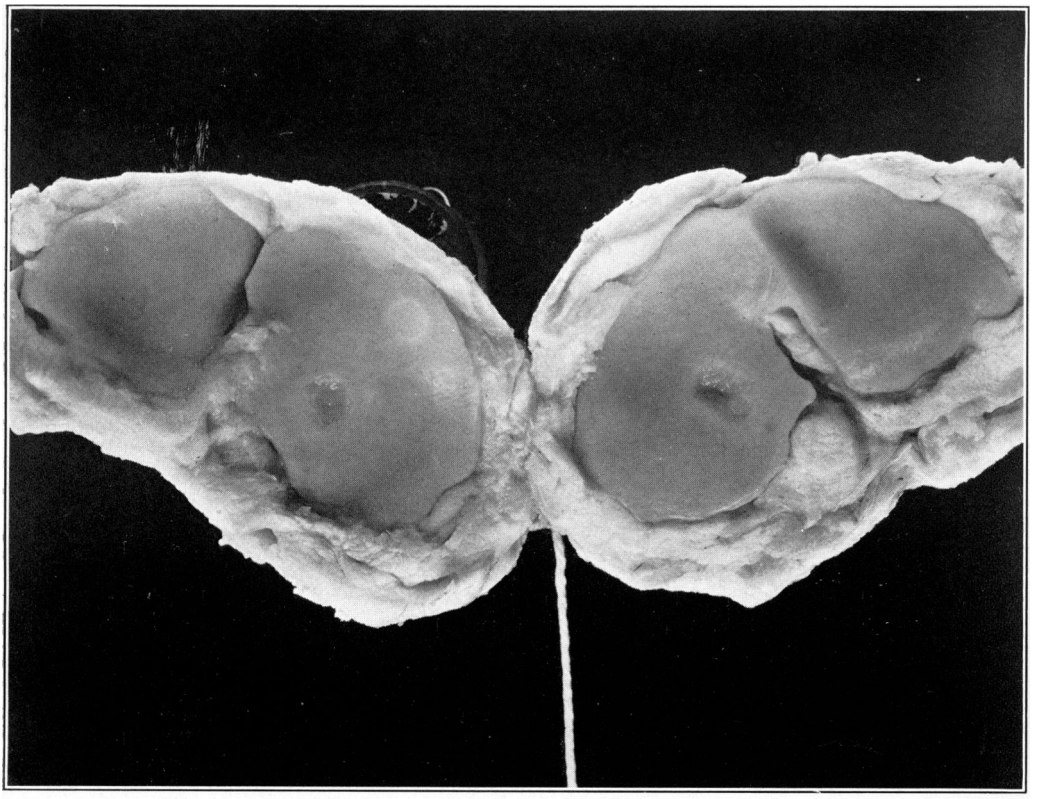


12

PLATE 75

FIG. 13. Similar lesions on corresponding surfaces of the carpal and metacarpal articular cartilages of an old milch cow. Natural size.

FIG. 14. The articular surfaces of the carpal and metacarpal bones of an old milch cow, showing the striking similarity of the lesion on opposing surfaces. Natural size.



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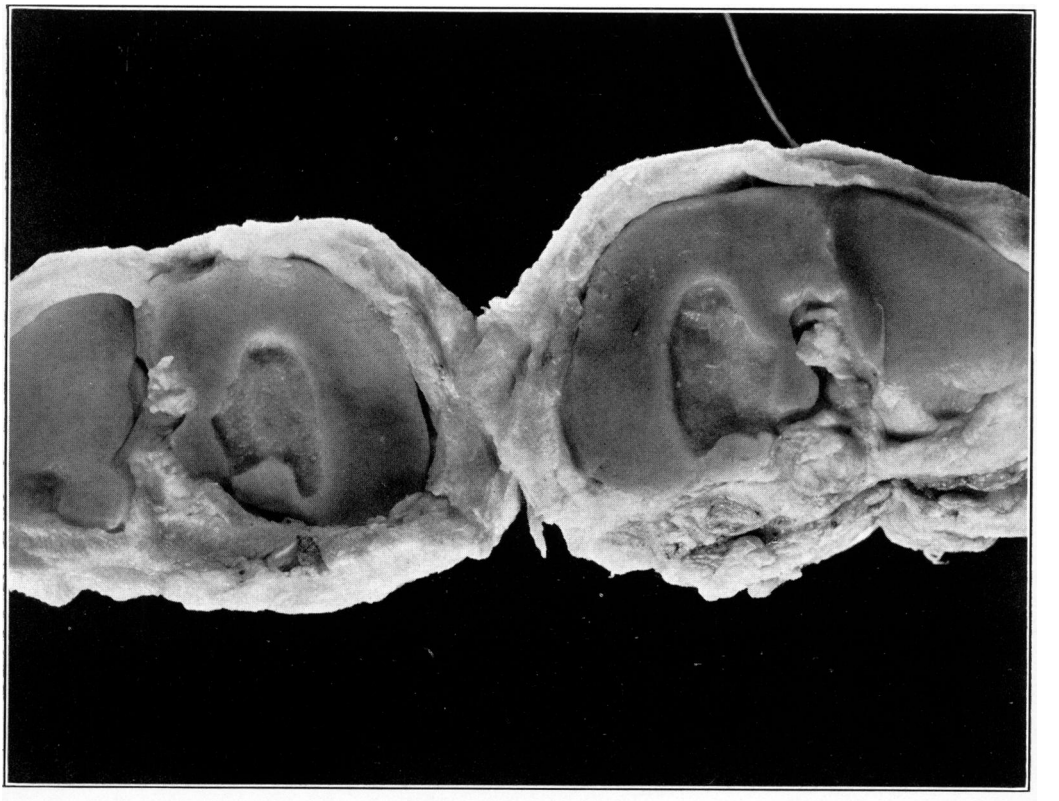
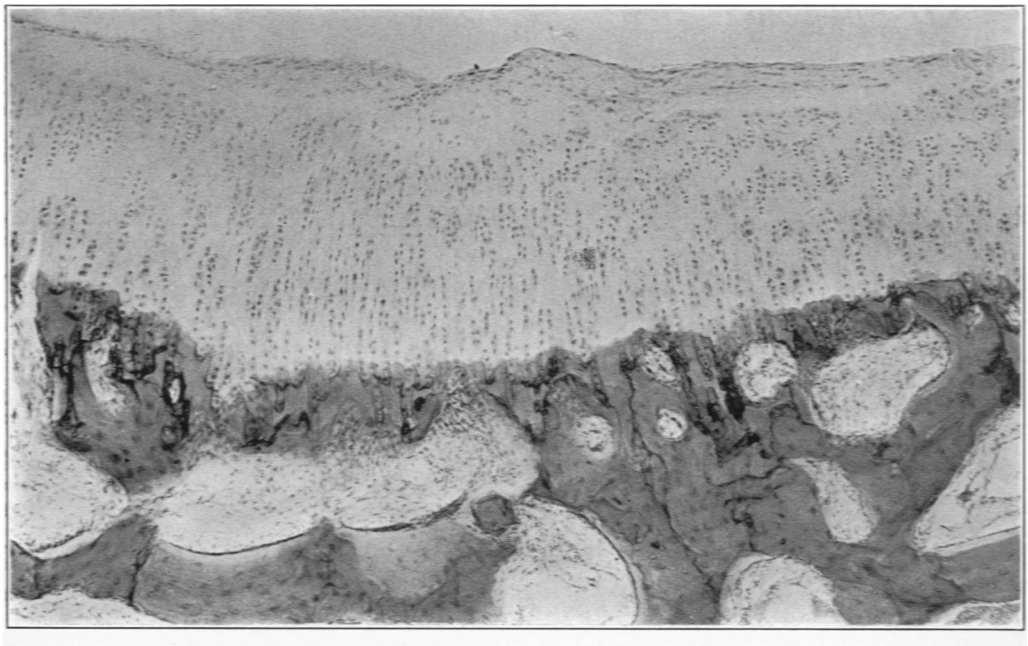


14

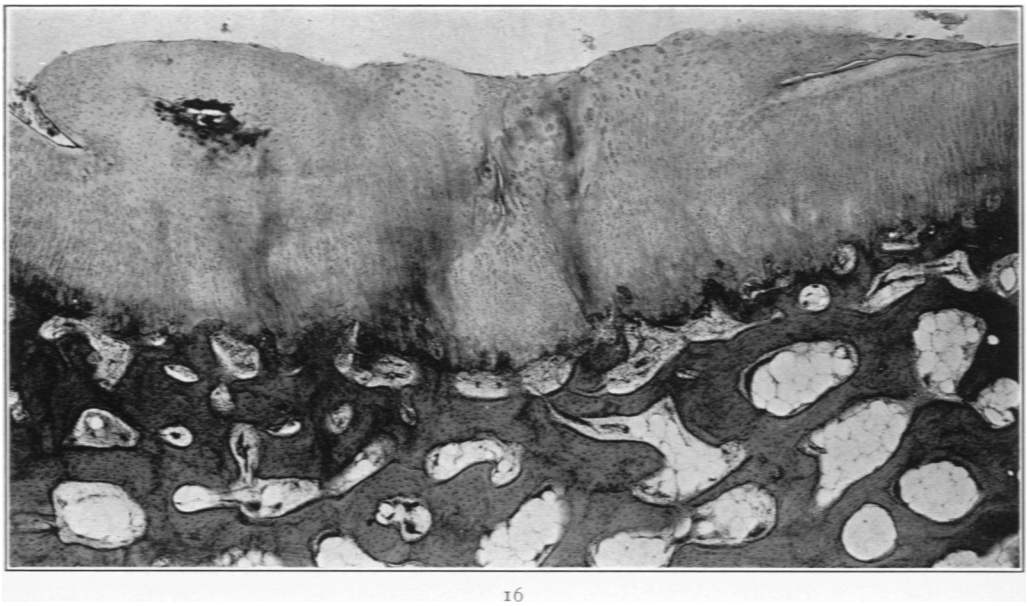
PLATE 76

FIG. 15. Early changes in articular cartilage are illustrated in this photomicrograph. The layer of calcified cartilage is thinned out, slightly depressed and completely broken in many places. Beginning distortion of cartilage cells and a surface proliferation of cartilage is illustrated. × 91.

FIG. 16. A very low power photomicrograph showing more extensive distortion of cartilage and proliferation of surface cells. There is much fibrillation and splitting of the articular cartilage matrix. One area of calcification has occurred. Note the depression, thinning and disruption of the calcified layer of cartilage. × 33.



15

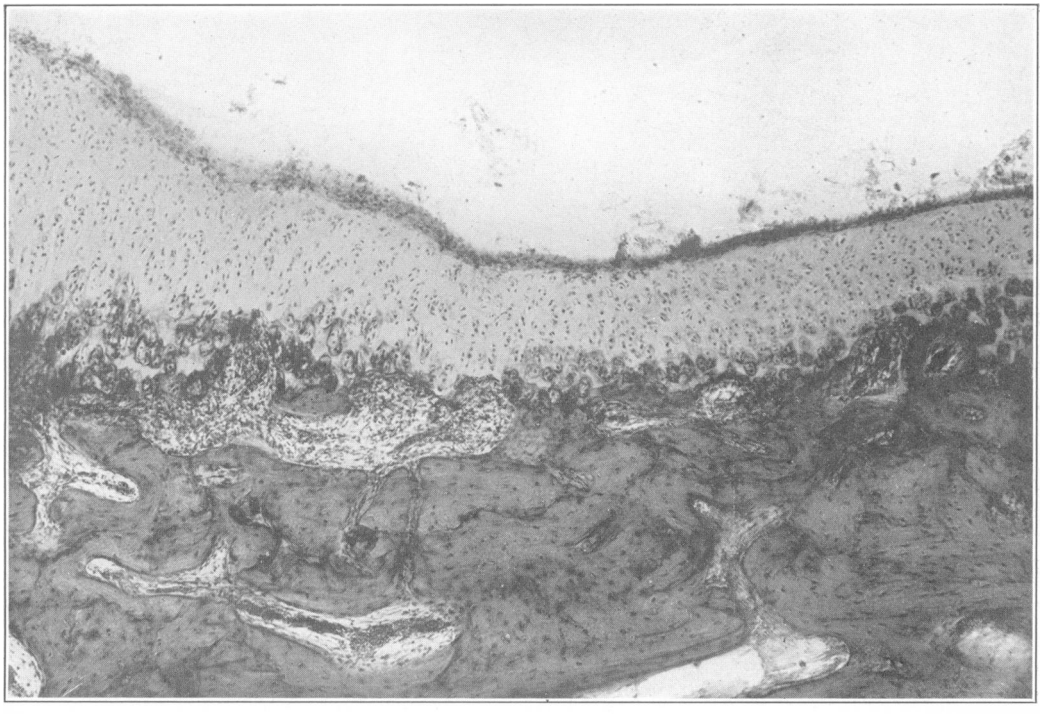


16

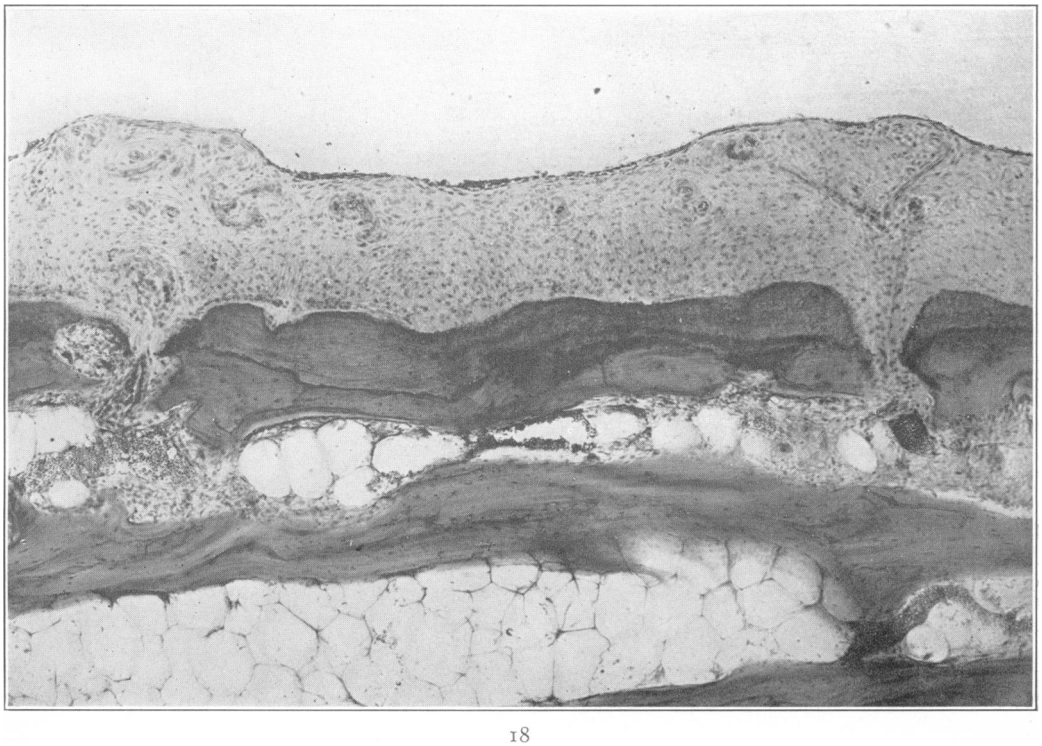
PLATE 77

FIG. 17. Beginning depression of articular cartilage and nearly complete destruction of the layer of calcified cartilage is illustrated in this photomicrograph. Note the alteration of cartilage and its resemblance to fibrocartilage. × 91.

FIG. 18. A more advanced metaplasia of cartilage into fibrocartilage. Note the complete absence of the calcified layer and the invasion of cartilage by blood vessels from the subchondral bone. × 91.



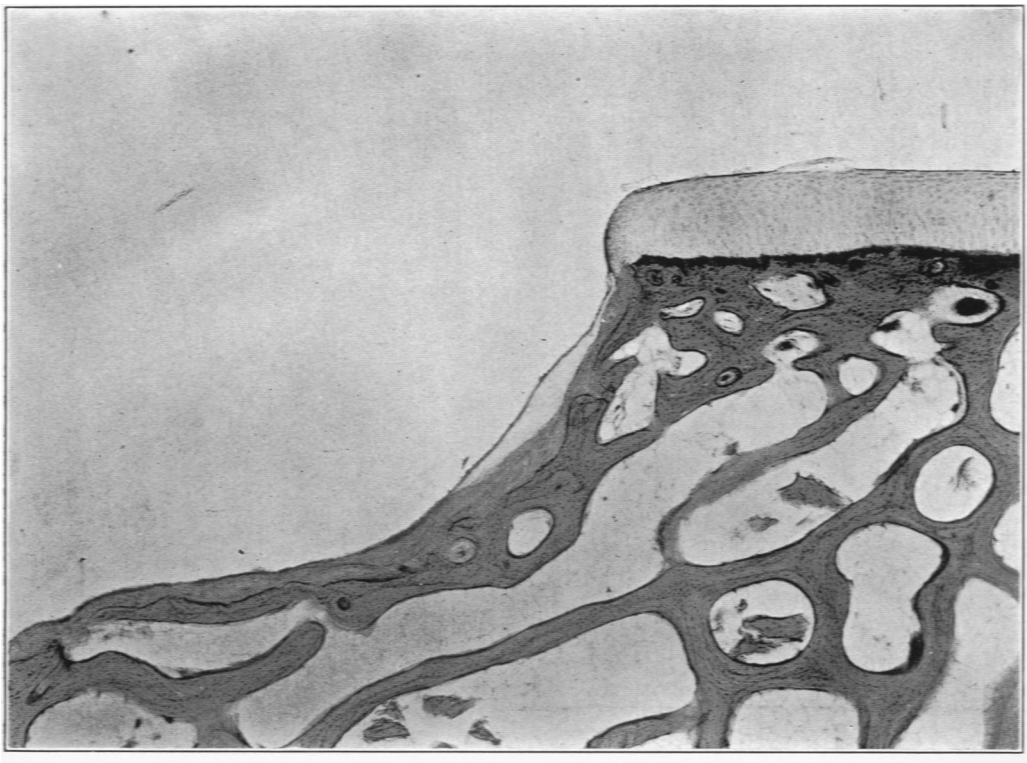
17



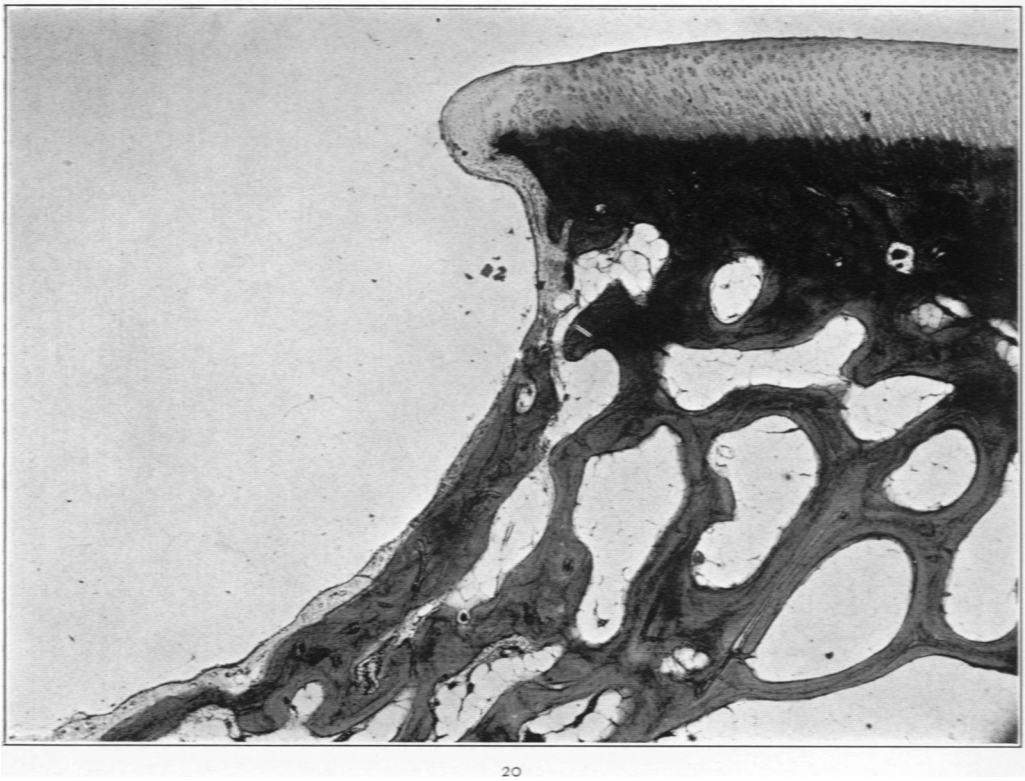
18

PLATE 78

FIGS. 19 and 20. Low power photomicrographs which include one-half of each of the larger lesions from two specimens. Note the abrupt break in articular cartilage, the deep extension of the lesions into subchondral bone and the synovial membrane-like tissue lining the depressions. There is practically no inflammatory cell infiltration in any part of the sections. × 36.



19

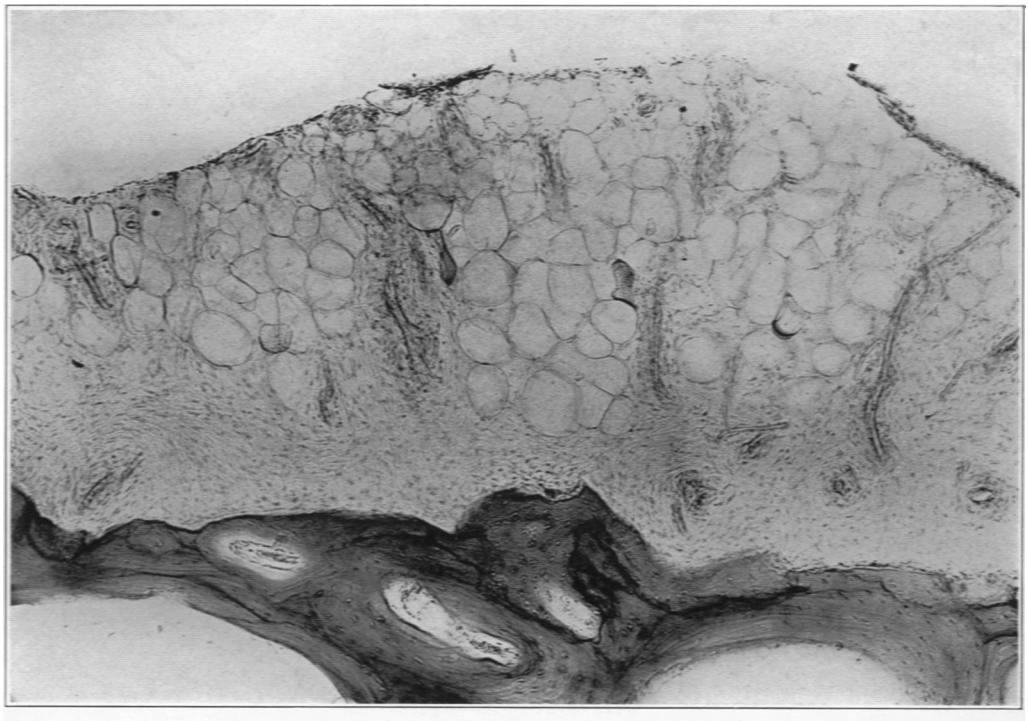


20

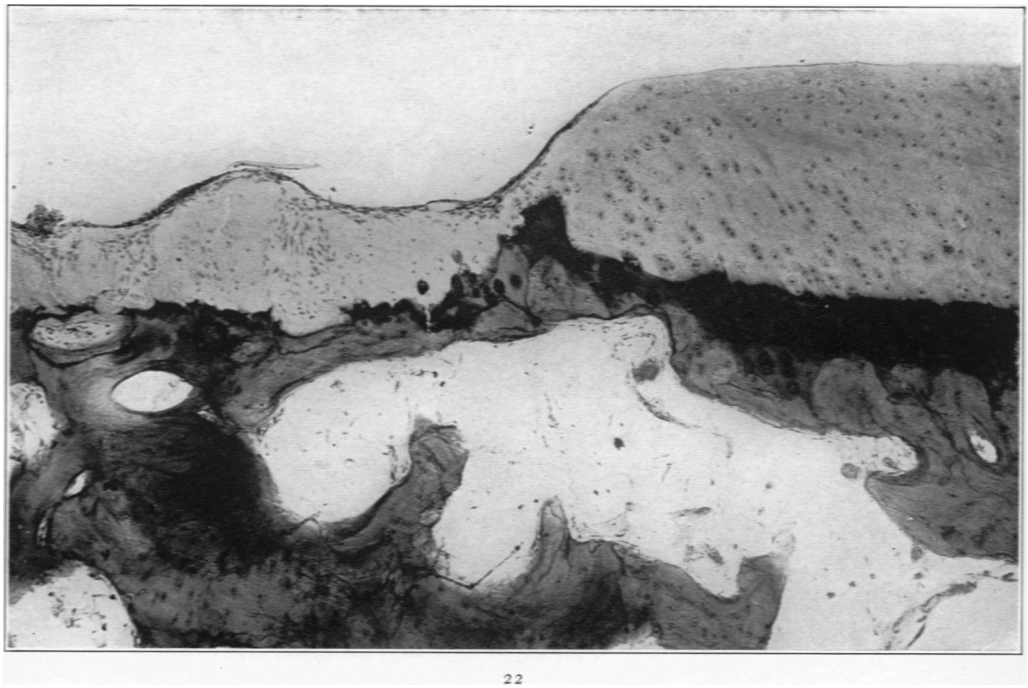
PLATE 79

FIG. 21. Fat replacement of the connective tissue and fibrocartilage which lined the larger and more extensive defects of articular cartilage is illustrated in this photomicrograph. × 91.

FIG. 22. One margin of a degenerative lesion in the articular cartilage of an old milch cow. Note the decrease in number of cartilage cells in the more intact cartilage and the intense calcification of the deepest layer of cartilage. The connective tissue lining the degenerated area is largely hyalinized. × 91.



21



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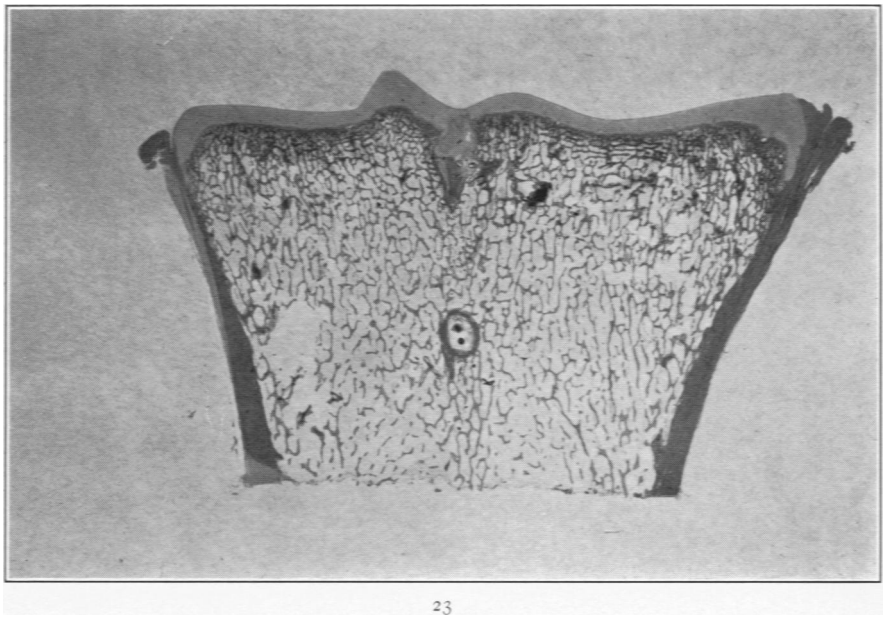
Bennett and Bauer

Degeneration of Cartilage in Bovine Joints

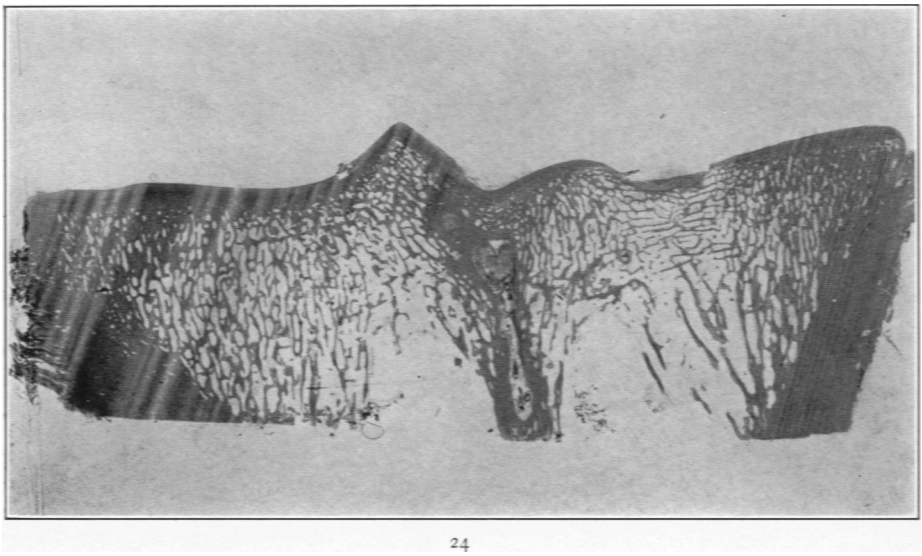
PLATE 80

FIG. 23. A photograph of an entire transverse section (celloidin) showing the evenly distributed subchondral bone trabeculae and thin cortex of the metacarpal bone of a calf. × 2.

FIG. 24. A photograph of an entire transverse section (celloidin) of the metacarpal articular cartilage and subchondral bone from a young steer with an average sized lesion of articular cartilage. The difference in the arrangement of the bone trabeculae beneath the medial and lateral articular surfaces and the cartilage defect is clearly shown. × 2.



23



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