stated that visible changes at the disk in the acute stage are rare. In seventy cases referred to here definite changes were noted by the surgeon in twenty-one. In most instances the report was: disk blurred, distinctly blurred, pinker and less distinct, cloudy, or slight swelling ; in two cases the disk was described as swollen and oedematous, and in three the terms used were papillitis or optic neuritis. The incidence of slight changes is probably higher than these figures indicate, as special attention has not been paid to this point in my records. Certainly the observer should be prepared to meet them frequently. Intense swelling, the typical choked disk, if it occurs at all, is very rare in the unilateral type. It is a common feature in bilateral cases. It is suggested that intense swelling in one or both eyes or bilateral incidence of even mild visible changes with severe visual failure indicates a different aetiology.

Frequency of Retrobulbar Neuritis in Disseminated Sclerosis

Discussion is invited on this point and particularly on the following statements: (1) Disseminated sclerosis is the only known common cause of acute unilateral retrobulbar neuritis as here defined. (2) Apart from certain rare allied acute inflammatory diseases of the nervous system it is the only cause. These statements cannot be proved or disproved ; but if they are accepted as approximating to the truth much will be gained. Clear ideas can then be substituted for the muddled mental state that must exist so long as the incidence of disseminated sclerosis as the cause is variously estimated as " from about 20 per cent. to about 80 per cent.," and we may at last begin to hear less of hypothetical sinus disease and dental or tonsillar sepsis as possible causes. Nothing more can be offered here in support of these statements than evidence that disposes at once of the lower estimate (20 per cent.), but is for the rest indirect. Seventy cases of acute retrobulbar neuritis were examined within a week or two of onset. In 31.3 per cent. the patients were already suffering from disseminated sclerosis ; in 41.8 per cent. the diagnosis " probably disseminated sclerosis " was made ; in the remaining 26.8 per cent. there were no other suspicious symptoms and no definite organic signs.

Where so much depends on intimate knowledge of the numerous early symptoms and on personal interpretations of physical signs, agreement by different observers on the number of cases that fall into these groups cannot be expected. My own opinion is that conclusive historical or physical evidence of the presence of disseminated sclerosis can be obtained in over 30 per cent. of cases of unilateral retrobulbar neuritis in the acute stage, and that the diagnosis can be made with a close approach to certainty in about 50 per cent. What is the fate of the cases in which the cause is uncertain? The answer could be given if we were able to keep in touch with our patients sufficiently long, but this is hardly possible, because, as we have seen, ten, twenty, or more years may pass before other signs or symptoms appear. Fleischer followed. up some of his cases and found that 66 per cent. of them developed disseminated sclerosis. Further than this direct evidence does not take us at present.

Significant indirect evidence is obtained by comparing the age of onset and sex incidence in acute uncomplicated cases and in cases of patients with established disseminated sclerosis who give a history of an acute attack of visual failure in one eye as their first symptom. In the first group in 85.8 per cent. and in the second in 86.9 per cent. the acute attack occurred between the ages 21 and 40 ; in the majority in both groups, between 21 and 30. The average age of onset in

Traquair's 139 cases of " acute unilateral type " was thirty-two years. Misgivings about the value of these figures arising from a consideration of the proportion of the population living at these ages may be set at rest by a comparison of the sex incidence in the groups just named. The facts may be set forth as follows: acute unilateral retrobulbar neuritis, uncomplicated, 64.7 per cent. females ; history of unilateral retrobulbar neuritis as first symptom in patients with disseminated sclerosis, 63.7 per cent. females ; all cases of established disseminated sclerosis, 58 per cent. females.

Traquair found that 65 per cent. of his cases of the " acute unilateral type " of retrobulbar neuritis were females. This agreement in age at onset and sex preponderance in females in the two groups is remarkable, and it is suggested that it indicates a common cause. As 65 per cent. of the patients with established disseminated sclerosis who gave a history of acute retrobulbar neuritis at any stage in the course of the disease were females, another conclusion seems to be justified—namely, that females are more susceptible to it than males, and that in them it is more often the first manifestation of the disease. It is to be hoped that the ensuing discussion will lead to some advance in our knowledge of the aetiology of acute retrobulbar neuritis ; the subject is one wherein the profession is in great need of guidance.

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ADAMANTINOMA OF THE TIBIA:
AETIOLOGY AND PATHOGENESIS *

BY

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(With Special Plate)

Extra-alveolar adamantinomata have been infrequently reported. Those occurring at the base of the skull and in relation to the pituitary gland are well recognized, and their origin is satisfactorily explained on embryological grounds.[1] So far as I know the only other extra-alveolar adamantinomata are the very infrequently described adamantinomata of the tibia. Besides the case to be described I know of only two recorded cases—that of Fischer[2] and that of Baker and Hawksley.[3]

These cases of adamantinoma of tibia are so similar as to indicate that we are dealing with a rather clear-cut, if rare, clinical and pathological entity. In each case there is a history of trauma ; in each case the trauma is slight so far as surface injury is concerned ; in each case this trauma is followed by a tumour after a latent period ; in the two recent cases at least there is evidence suggesting that during the latent period some local lesion persisted ; in each case the tumour is one of the bone and periosteum and not of the soft parts. The histology of the tumour is the same in all cases, being that of certain types of adamantinoma, basal-celled carcinoma, or rodent ulcer, a structure which possibly justifies the diagnosis of adamantinoma. To this list of similarities one may add the tendency to local recurrence. While the interest of these cases lies partly in their clinical significance and their morphological diagnosis, their chief interest to the pathologist lies in the realm of aetiology and pathogenesis, and in the hope that the study of a rare lesion may throw light on wider problems.

THREE REPORTED CASES

In 1913 Fischer[2] reported a primary epithelial tumour of the tibia. He outlined the clinical history and gave an elaborate account of the morphology, diagnosing the tumour, apparently with some trepidation, as an adamantinoma of the tibia. The diagnosis in his case, as in the other cases, seems to be justified perhaps just in so far as tumour diagnosis is adequately based in morphology. His case was that of a male, aged 37, who slipped and knocked his leg on a stair. The injured part was injected and painful, but the description does not suggest a very gross injury. Five months later a tumour was observed at the site of the injury, and nine months after trauma the tumour area was resected. On this resected bone Fischer's diagnosis was made. His description leaves no doubt but that the tumour was one of bone and periosteum and not of the superficial soft parts.

Baker and Hawksley's case[3] was reported in 1931, and I am greatly indebted to these authors for the opportunity to examine their very beautiful preparations, and to compare them with my own material. Their case was that of an engine driver, aged 46 at the time of examination. He struck his left shin with an iron bar. The injury appears not to have been a gross one, and there was no skin wound. Slight injuries to the same region seven and eight and a half months later were associated with much pain, pointing to some persistence of a lesion at the site, and ten months after the first trauma a subperiosteal resection of the tumour then present supplied the material for diagnosis. Again the tumour was in the bone.

Very similar is the clinical history of my own case. It differs from the others only in the peculiarly long latent period. For the opportunity to study the case I am indebted to Dr. C. C. Elliott of Capetown, who has taken a lively interest in its clinical and pathological aspects. In 1915 the patient, then aged 36, was kicked on the shin. Only superficial bruising resulted. The whole thing healed up promptly so that there was nothing to show, although pain or tenderness was never entirely absent thereafter. In 1923, eight years later, a swelling developed fairly rapidly. This was apparently looked on as a bone sarcoma, and amputation was advised. The patient refused amputation, and nothing was done until he again sought medical help in 1931, sixteen years after the injury. A very definite tumour was then present. Following x-ray examination the tumour area was scraped out, and on the histological examination of the material so obtained the diagnosis of adamantinoma of tibia was made. Six months later, in October, 1931, the tumour recurred and grew rapidly, causing so much pain that amputation was done at the patient's request. Fig. 1 shows the x-ray picture of the original tumour; Fig. 2 the gross section of the bone. The evidence is of a slowly growing tumour originating in periosteum or bone. Even in the recurrence the tumour appeared to be confined to bone, and examination of many sections of the skin and superficial soft tissues failed to reveal any tumour. Histological examination of the recurrent tumour raised points of some interest. The differen-

tiation of the tumour towards an adamantinoma-like structure was less complete. In large areas the cells were chiefly spindle-shaped ; there were very numerous mitotic figures. Examination of these small parts of the tumour might very well have resulted in a diagnosis of spindle-celled sarcoma or myxosarcoma. After the trauma of operation there seems to have been a shift towards a structure more suggestive of malignancy, but still the soft parts did not become invaded. Figs. 3 and 4 illustrate the appearances, and leave no doubt of the identity of the tumour with those previously described and illustrated.

We may accept it that these are primary epithelial tumours of the skin—adamantinomata if we like. We have a uniform type of tumour associated with a very characteristic and uniform type of clinical history, of which the outstanding features are trauma in the shin region, persistence of some local reaction or lesion of slight degree, and then, after a period, the emergence of a tumour of characteristic morphological and biological features.

AETIOLOGY—FOETAL CELL REST OR TRAUMA?

Obviously the first question is " Whence came the epithelium in which the tumour originated? " Was it a foetal cell rest or was it an acquired implant dating from the trauma? Fischer definitely accepted the first view. He considered that the whole surface epithelium had originally multiple potentialities, including the potentiality of tooth-germ formation. This potentiality, however, was limited to that period of foetal life during which the tooth germs were forming. Only epithelial germs or rests isolated at this period would have the potentiality of tooth-germ formation or, in tumour terms, adamantinoma formation. With further differentiation of the body this potentiality of the epithelium was lost, and later sequestrations or cell rests would not have this potentiality. Accepting this, it follows that the germ rest from which our tumour arose must have dated from foetal life, from the time of tooth-germ formation indeed, and could not have been an implant of adult life. To quote Fischer: " The normal epithelium of the adult skin bears no other actual developmental potentiality than that of forming horny pavement epithelium. If the tumour arose as the result of the deposit of an epithelial germ (that is, adult deposit or implantation) then of necessity the tumour should illustrate the actual structure of pavement epithelium." To Fischer, then, the adamantinoma structure of the tumour indicated its origin in a cell rest dating from that period of foetal life during which the normal tooth germs were forming. After discarding ideas of metastatic and teratomatous origin he says:

" Hence there remains only the assumption that this germ was formed at the site during the intrauterine period, and to this assumption I would give preference above all other views which may be advanced as an explanation of our tumour."

KEY TO ILLUSTRATIONS ON SPECIAL PLATE

FIG. 1.—Radiograph of adamantinoma of tibia—a well-defined cystic tumour in the shaft of the bone ; it is of slow growth, with considerable sclerosis.

FIG. 2.—Gross specimen ; a cystic adamantinoma of tibia.

FIG. 3.—Histological structure of the growth before recurrence. The similarity to the developing tooth germ, to the previously published adamantinoma of tibia, and to a rodent ulcer is obvious.

FIG. 4.—Sarcoma-like structure of part of the recurrent growth.

[Figs. 5 to 15 are taken from the traumatic rodent described in the text.]

FIG. 5.—Implantation dermoid in the injured area.

FIG. 6.—Giant-cell absorption of injured tissue in the area. Below, note part of the tumour quite similar to the adamantinoma.

FIG. 7.—Area similar to Fig. 6, but showing a bud of proliferating epithelium like the tumour.

FIG. 8.—More massive tumour-like proliferation from a piece of isolated epithelial tissue. In serial sections this could be traced for a considerable distance, and was definitely tumour.

[Figs. 9 to 11 are selected sections taken in order from a group of serial sections of the tissue.]

FIG. 9.—Above is surface skin epithelium, and below an apparently isolated epithelial mass. In both parts there is some budding of epithelium.

FIG. 10.—Shows that this deep mass is linked up with the surface by proliferated epithelium, apparently along the line of the original hair follicle. On either side of this proliferated tissue note buds of tumour.

FIG. 11.—Shows part of the epithelial mass entirely embedded in such proliferated cells forming tumour.

[Figs. 12 to 15 are similarly selected sections in order from similar sections of the area.]

FIG. 12.—Below, isolated hair follicle tissue ; above, tumour.

FIG. 13.—Later section showing these approximating.

FIG. 14.—Later section showing that the apparently isolated epithelium is linked up and closely related to the tumour.

FIG. 15.—Still later section showing this linking up, producing a picture like that seen in Fig. 10.

Fischer goes with great fullness into this problem of aetiology, and it is very interesting in reading his paper to-day to see how strongly he was influenced by what was perhaps an imperfect interpretation of Cohnheim's theory, and also by the then current ideas of cell specificity. He practically ignored the history of trauma as of aetiological interest, saying only " perhaps also the trauma which was advanced in the clinical history contributed towards more rapid growth of the epithelial germ." But since Fischer wrote in 1913 our views of tumour growth have widened considerably. Cohnheim's cell rests have a smaller if better defined place in our aetiology, and we are less sure about cell specificity. In the light—or shade —of such theories, and with only one case available for study, it was perhaps reasonable to take Fischer's view of the trauma ; but I do not think that to-day we can lightly put aside as of no aetiological significance a fact so prominent and constant in all the cases as this one of trauma.

Drs. Baker and Hawksley deal chiefly with the clinical and histological aspects, and do not attempt to discuss at length the aetiology and pathogenesis. They follow Fischer in postulating a foetal epithelial rest, but lay a little more stress on the trauma as stimulating this rest into growth. They say: " The epithelium must almost certainly have been subperiosteal from embryonic life. . . . The epithelium presumably remained of primitive basal-celled type, stimulated into tumour growth by the first injury." One must confess to a dislike of hypothetical foetal cell rests in positions where they are not adequately explained on embryological grounds, but if we are to discard this relatively simple explanation of the tumours we must replace it with an explanation which takes account of the constant clinical history, the peculiar site and constant structure of the tumours, and an explanation which is in accord with present ideas of the relation of neoplasia to other pathological processes.

POSSIBILITIES OF EPITHELIAL IMPLANT

The alternative view would seem to be that the tumour originated from epithelium implanted at the time of the trauma. When we recall the relative slightness of the trauma without breach of skin surface it is perhaps difficult to imagine any traumatic implantation of epithelium, least of all an implantation into the bone. That would seem to require a very considerable trauma, but how else can we explain the localization of the tumour in the bone in each case? Perhaps one might imagine that, apart from surface laceration, epithelial cells could become sequestrated by a traumatic breaking up of the deep germinal layers of epithelium, the surface remaining intact. I have taken opportunities to study bruises in human and animal tissues, and have not seen any such breaking up of the Malpighian layer or isolation of its cells. One must admit that if such could occur the shin region would be one of the most likely sites, but even so, the known fate of traumatic or experimental epithelial implants scarcely favours the view that such isolated cells could give rise to this very specific type of tumour. That such sequestrated cells should remain latent in the tissues for eight years as in our case presents us with a problem more perplexing than the problem of our tumour. I think, however, that we can so modify this conception of the traumatic origin of the tumour that it will take into account the clinical history of trauma and subsequent persistent pain, explain the special situation and structure of the tumours, and give as reasonable an account of these as we can of most tumours.

The outstanding anatomical feature of the part under consideration is the superficial position of its hard, heavy bone, and especially of its sharp anterior edge. This gives to injuries in this region their peculiar character, and the persistence of hard swelling and pain in this region long after injuries which have not broken the skin is common experience. The conditions are such as to favour laceration of the periosteum and haematoma formation, with subsequent ossification. Another feature of interest is that the skin, freely movable in the lateral direction across the tibia, is not so freely movable over the bone in the longitudinal direction. The type of injury in our

cases—a kick, or a fall on a stair—is a shearing injury in this long axis of the limb tending to drag the soft tissues over the superficial bone, tearing the tissues and their anchorage to the periosteum. There can therefore be considerable laceration of the subcutaneous tissues, skin appendages, and periosteum without breach of the skin surface, and it is this dragging, with consequent deep laceration, rather than the surface injury, that is the peculiar feature of trauma in this region, entailing as it does subcutaneous and periosteal haemorrhage, a long process of organization and ossification, and therefore prolonged tenderness and swelling.

" THWARTED REPAIR " THEORY

Can epithelial cells or tissues become sequestrated in such injury, and, if so, what will their fate be in this special environment of prolonged growth and ossification? The injury described will involve tearing not only the connective tissues, but the deep skin appendages such as the hair follicles, and it is in the subsequent fate of these special epithelial tissues in this special environment of tissue repair that we must look for the explanation of our tumours. Their fate will depend on many factors, such as the nature and extent of the injury, the extent of interference with blood supply and lymphatic drainage, the degree of disorganization of the isolated epithelial tissue, and the environment in which the now isolated epithelium is placed. Such hair follicle tissue may simply be absorbed, or again it may become the basis of regenerative growth and repair. In the superficial soft tissues either absorption or repair is likely to be completed, but in the deeper part—in the region of lacerated periosteum and of the organizing and ossifying haematoma related to it—the conditions are peculiar. There, with the longer evolution of inflammatory and reparative processes, we can picture conditions which constantly thwart complete repair, and yet, from tissue activity, tissue destruction, or tissue growth, as constantly supply the chemical or biological environment for continued attempted reparative growth.

We may have difficulty in understanding how thwarted reparative growth at the edge of a chronic ulcer passes over into neoplastic growth, but we know that it does, and it has at least that degree of apparent reasonableness that comes from familiarity. We may have difficulty in understanding how thwarted hyperplasia in liver cirrhosis passes over into neoplasia, but we appreciate the significance of this in the aetiology and pathogenesis of carcinoma of the liver. Similarly, the reasonable explanation of adamantinoma of tibia appears to be that in the region of the ossifying haematoma thwarted repair ultimately passes over into tumour growth. That the exact reaction or environment necessary to maintain a growth stimulus and yet prevent repair will rarely arise, and then only by the fortuitous coincidence of many factors, fits in with the rarity of these tumours. The special features of the tibia region, which we have referred to, account for the fact that the few recorded cases have occurred there. The theory takes into account the constant clinical features of trauma and subsequent pain or tenderness. It does away with the necessity for postulating foetal cell rests in unlikely places or of picturing implanted cells remaining latent and inactive in the tissues for years. It removes the tumour from the position of a very mysterious and isolated entity into the great group of tumours which have chronic irritation as a big factor in their aetiology.

This view also accounts up to a point for the other peculiarity of these tumours—namely, their position only in the bone, or rather in the invaded bone and the old ossified haematoma, now incorporated with the bone. But once neoplasia was established and the tumour, which had so originated in the bone, had gained the power of recurring in the bone and invading new bone, why did it not invade the soft parts? Was the malignancy of the tumour in some way conditioned by its environment? All tumours of this basal-cell type have a limited kind of malignancy, with little ability to invade new types of tissue, and our tumour is not exceptional. What is it that limits the direct extension of such tumours? Is the

malignancy an endowment of the tumour cell itself, or is it a potentiality of the cell, only to be manifested in a certain environment? May it not be that the isolated cell does not contain the whole elusive secret of malignancy?' These questions arise whatever view we take of the aetiology of the adamantinoma of tibia.

ITS FURTHER EXAMINATION

There is one other possible view of these tumours which should perhaps be referred to. Should we look on these rare tumours as arising certainly from trauma, but as a peculiar response to trauma in persons congenitally predisposed to such reactions? One thinks of individual and racial peculiarities of response seen in traumatic keloids or in the occasional response to trauma by production of lipomata. This would not alter the general view of the origin of the tumours in aberrant repair, but would account for that aberrance by a congenital and general peculiarity of tissue reactivity instead of by an acquired and local one. It seems unnecessary to assume such a view, although it is, of course, very difficult to exclude such intangible factors in aetiology.

If our theory be correct it raises interesting points about the relation of trauma and tumour, and suggests that some other cases of epithelioma following trauma may be explained similarly. The following case of post-traumatic epithelioma gives interesting confirmation of the views put forward.

A European male, aged 59, presented himself at hospital with an ulcerated area the size of a threepenny piece on the left side of the neck over the upper end of the sterno-mastoid, about an inch below and behind the angle of the mandible. The area was excised and the tissue showed the histological structure of rodent ulcer. His story was that in January, 1932, five months previously, he had cut his neck while shaving, and the cut failed to heal. At the site there developed a scab which frequently broke off, and the ulcer gradually increased in size. Prior to the cut there was, he says, no

sore there. Further study of serial sections of the tumour showed that it was growing in an environment of persisting tissue absorption and irregular repair. Sections showed a small sequestration dermoid as one manifestation of aberrant repair, areas of giant-cell absorption, of hair follicles and hairs, and also tumour growth indistinguishable in parts from regenerative growth of hair follicles. Figs. 5 to 15 illustrate these points and make description unnecessary. It is demonstrable that this tumour arose on a basis of aberrant regeneration of hair follicles, and the study of this tumour goes to support the view of aetiology and pathogenesis put forward for the adamantinoma. The picture is exceedingly like the adamantinoma, and had this tumour been growing in the tibia we should have been strongly tempted to call it adamantinoma.

This raises the question of whether or not the diagnosis of adamantinoma of tibia is justified. The gross characteristics of adamantinoma of the jaw appear in our tumour but not in Fischer's. Perhaps the limitation of the tumour to the bone is a feature justifying the diagnosis. In any case both tumours are best considered of basal-cell carcinoma of traumatic origin, having their basis not in congenital cell rests, but in the passage of a constantly frustrated reparative hyperplasia into neoplasia, in a special environment of tissue reaction. This view cf the aetiology and pathogenesis of the adamantinomata of tibia is further satisfying in that the neoplasia is brought into relationship with other types of pathological growth in inflammation and repair. The most promising feature of the cancer problem to-day is the growing tendency to bring the pathology of tumour growth back into relationship with the rest of general pathology, from which it was too long isolated as something peculiar and apart in a distorting mist of theories.

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DISPLACEMENT OF THE UPPER FEMORAL EPIPHYSIS

REPORT ON TWENTY-THREE CASES

BY

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LIVERPOOL

(With Special Plate)

This paper is based upon the study of twenty-three cases of displacement of the upper femoral epiphysis. Of these, thirteen were under observation long enough to make it possible to consider the results obtained by treatment, while ten were recent. No cases were recorded in which the history and radiographs did not definitely establish the diagnosis. An instance of the difficulty of diagnosis is that of W. R. (Figs. 1 and 2 on Plate), in which the history and the second radiograph appear to be typical of "slipped epiphysis," but which the earlier radiograph proves to have been a fracture of the neck of the femur—a rare injury in adolescence. In persons over the age of 25 it is often impossible to distinguish radiographically between old cases of "slipped epiphysis" and old cases of Perthes's disease. Such cases were excluded.

Both from the history and from the radiographic appearances it was possible to differentiate two types of displacement. First, the type due to a single gross trauma, in which the condition is probably one of fracture through the juxta-epiphyseal line, though it is held by some[1] that the separation takes place through the actual epiphyseal cartilage. In this type there is definite, complete loss of continuity between diaphysis and epiphysis. The second type is that in which the deformity develops gradually, and there appears to be a bending of the neck

of the femur, somewhat similar to that seen in the softened long bones of the rachitic child.

These two types, though giving rise to identical endresults if not treated, are different in aetiology and pathological anatomy, and manifestly cannot be treated in the same way. In this series a number of different methods of treatment were observed, and the final results recorded.

DIAGNOSIS

The history in the traumatic type is of a definite injury, following which the patient has pain and stiffness in the hip. In spite of the considerable displacement which is present, as evidenced by the radiographs, these patients are not completely disabled, but can limp along, and the injured limb is able to bear a little weight. In the gradual-slip type the patient usually gives a history of pain and increasing stiffness in the hip for a period of about twelve months ; the condition is almost invariably diagnosed at first as " rheumatism."

The physical signs in the two types are similar, but are more pronounced in the traumatic variety. There is limitation of abduction and internal rotation, resulting in severe cases in adduction and external rotation deformity ; there is often limitation of hyperextension, and sometimes flexion deformity. There is from one-half to one inch of real, and a varying amount of apparent, shortening. In one case there was marked abduction and external rotation, with fullness in Scarpa's triangle ; this led to a provisional diagnosis of anterior dislocation of the hip being made until a radiograph showed the true nature of the injury.

ANATOMICAL CONSIDERATIONS

Up to the age of 4 years the head, neck, and trochanters of the femur are represented by a common cartilaginous mass, but later on, the neck, growing up from the dia-
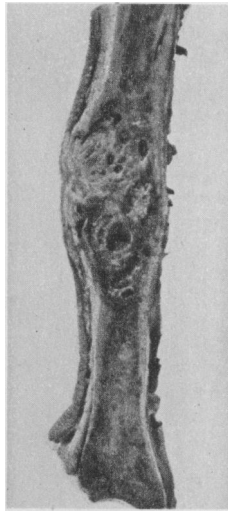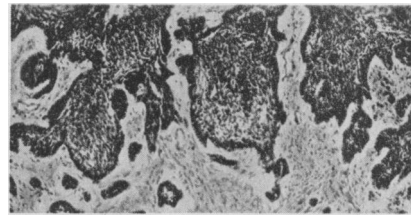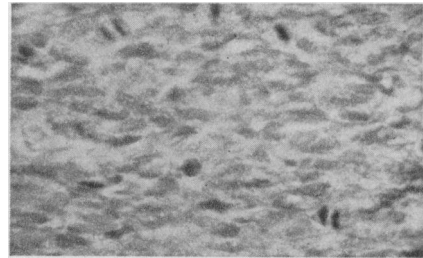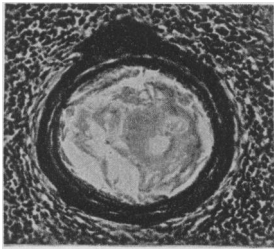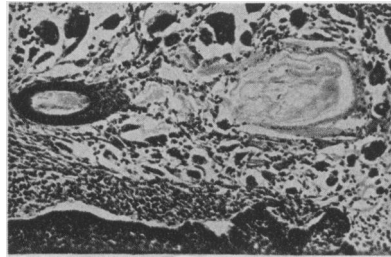
B. J. RYRIE: ADAMANTINOMA OF THE TIBIA
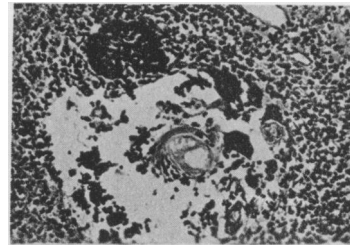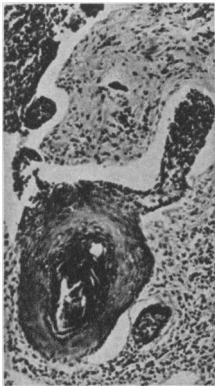

FIG. 1.

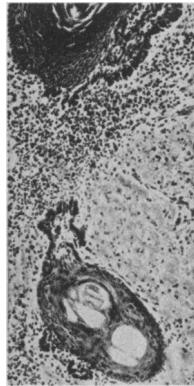FIG. 2.

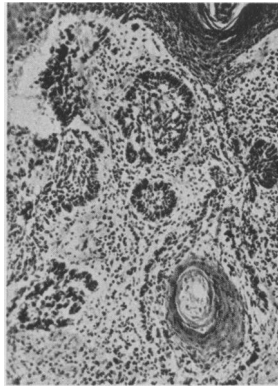FIG. 3.

FIG. 4.

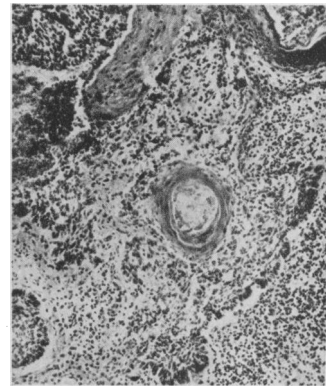FIG. 5.

FIG. 6.

FIG. 7.

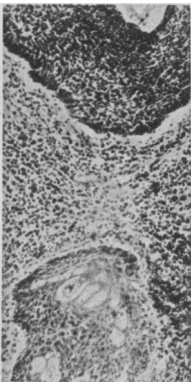FIG. 8.

FIG. 9.

FIG. 10.
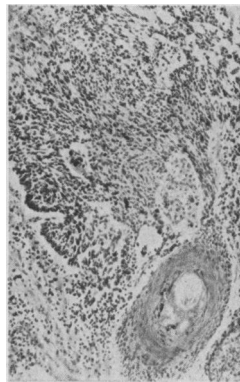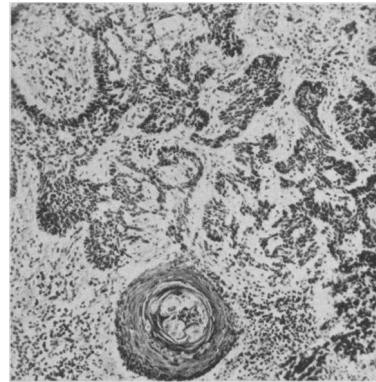

FIG. 11.

FIG. 12.

FIG. 13.

FIG. 14.

FIG. 15.

For description of figures, see key in text of article.