Virchows Arch [Pathol Anat] (1981) 391:53–79

Virchows Archiv A

Development of Caries Sicca in a Dry Calvaria

C.J. Hackett*
19 Prince Edward Mansions, Moscow Road, London W2 4EN, England

Summary. This study of the development of the bone changes in caries sicca was based upon the microscopy of over a hundred serial sections from eleven undecalcified blocks from a dry calvaria. In the absence of cells much depended upon the distribution of the mineral, the pattern of the collagen fibre bundles, the size, shape and arrangement of the vascular and osteocyte spaces, and of canaliculi. Examination by polarized light and microradiography was used. Mineral redeposition was not seen [3].

The changes observed had all occurred as relapses of active disease in a previously inflammed and now sclerotic bone. It is possible, however, to propose the changes that occur and the sequence of their development.

The first change is an inflammatory osteoporosis which starts in the deeper part of the outer table of the calvaria (Fig. 13). This spreads in all directions in the diplöe to form the destructive focus, up to 30 mm in diameter. As the inflammatory reaction subsides, fibre-bone fills the holes in the middle of the focus and follows the outward spread of the osteoporosis. This is then remodelled through further osteoporosis and lamellar bone filling until the whole focus becomes sclerotic bone in which some fibre-bone may persist. The characteristic multi-nodulation arises from localized periosteal and diplöeal bony enlargements.

Key words: Pathology – Syphilis – Dry calvaria

Introduction

No description of the pathogenesis of the multi-nodular appearance of the outer surface of the calvaria, so characteristic of caries sicca, appears to have

* I am grateful to Dr. Paul Byers, Director, Department of Morbid Anatomy, Institute of Orthopaedics, Royal National Orthopaedic Hospital, England, for encouragement, advice and technical services. Histological assistance provided by Jean Revel, and photographic and graphic assistance by T.R. Davies

During the 16 years of these studies the late Professor E. Uehlinger of Zurich often gave me sound advice and strong support

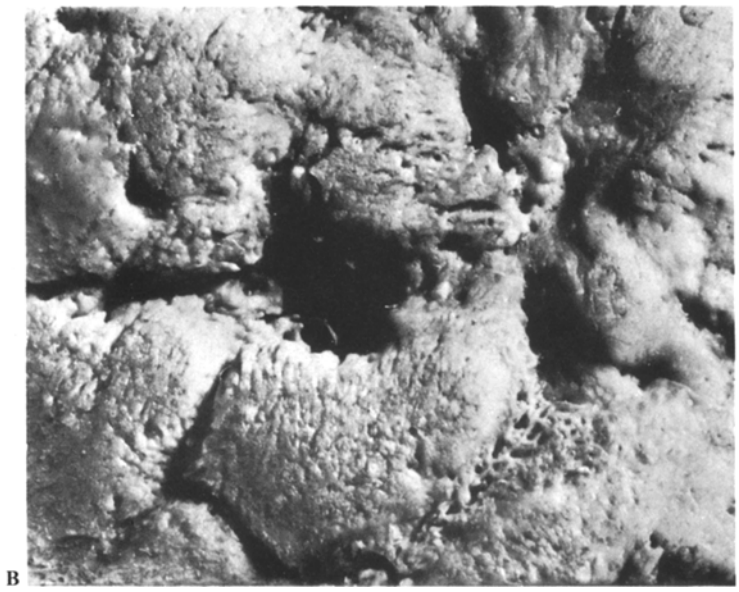
0340-1227/81/0391/0053/$05.40

Fig. 1. Calvaria $\frac{♀}{I}$ 372E with typical caries sicca of Virchow [5]. "A peculiar jagged, radiate, often, star-shaped depression –. Its borders are relatively smooth, round and not eroded –. The changes are grouped round the centre, radiate and join again but give the impression of a uniform pattern. This is decisive. It is unimportant whether the defect is deep or wide and flat". A circumvallate cavity is on the left frontal (*arrow*)

Fig. 2. Inner surface of frontals. The arabesque pattern of the early meningeal periosteal new bone is becoming smoother. The thickness of the changed calvaria is clearly seen. In radiographs the thickening is seen to be made up of many embedded nodules of sclerotic bone (× 2)

been published. Because of the historical and epidemiological importance of accurate recognition of treponemal diseases in the past, a study was made of criteria for their diagnosis in dry bone [2]. Virchow's opinion [5] that caries sicca occurred only in syphilis was substantiated, and proposed as the diagnostic criteria for that condition. This paper reports a microscopical study.

A



B

C

Fig. 3A–C. Surfaces of blocks 2A and B, 5 and 9 (×4)

Fig. 4. Lower surface of part of the slice from which the blocks were cut. Note nodular profile of the outer surface, and the smooth inner profile and surface. Areas of osteoporosis are darker because of more abundant holes to retain dust (×2)

Material

A 20 mm strip of thick heavy tissue was removed from the frontal and parietal bones of a dry calvaria ($\frac{9}{1}$ 372 E) (Fig. 1) labelled syphilis, in the pathology museum of St. Georges Hospital Medical School, London. This bone had typical gross caries sicca. I am indebted to Professor Sir Theo Crawford for allowing the removal of this strip, without which this study would not have been possible.

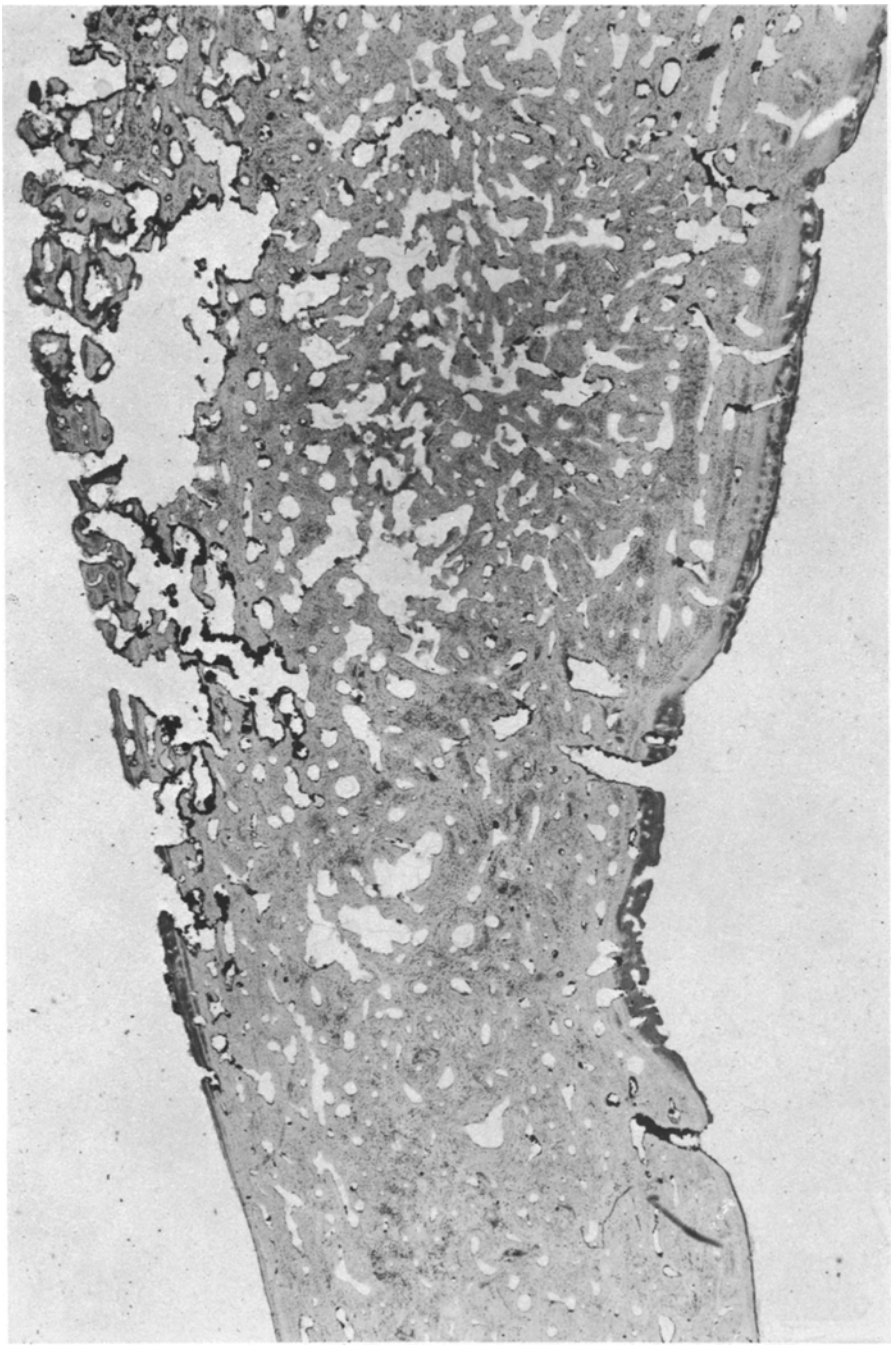
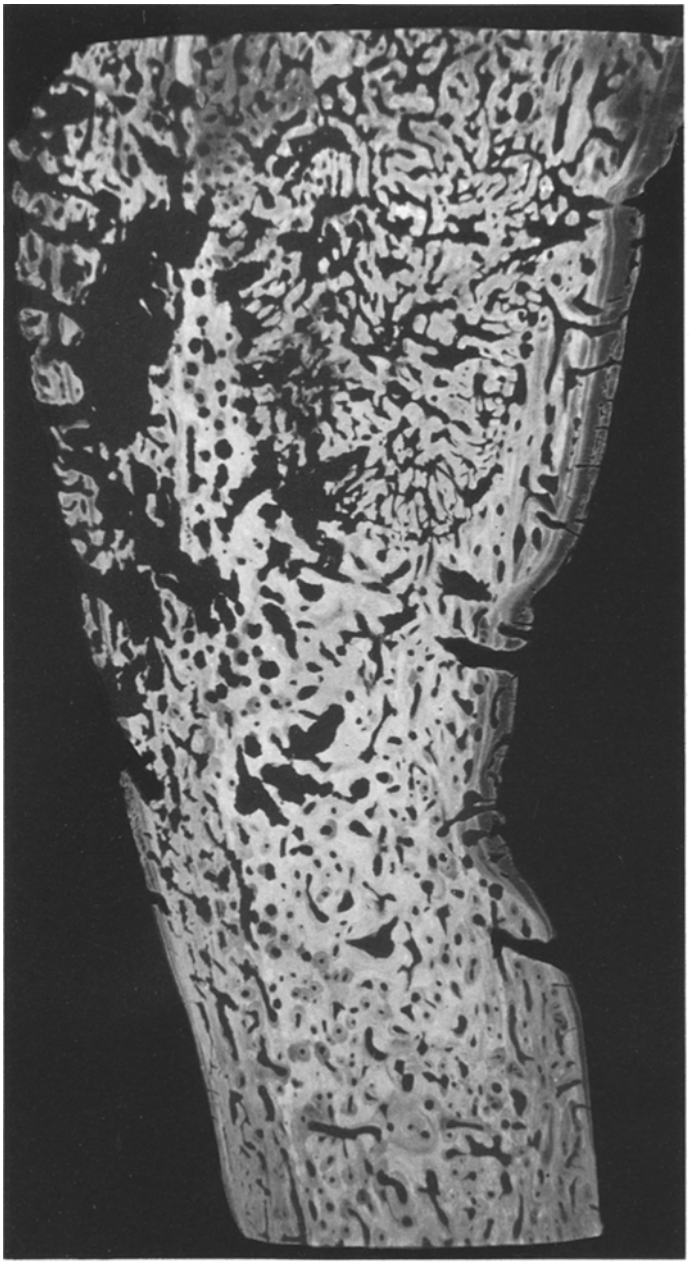


Fig. 5. Section through the early focus; inflammatory osteoporosis on right and on the left are of fibre-bone and sclerotic bone. All the active changes have occurred in a previously sclerotic calvaria. (2B-6/25 left, sections 30 and 70 μm; ×10) see Fig. 3A (2B-6/25 indicates that the section is the 6th of 25 from block 2B and the sections start from the left surface). Ordinary light and microradiograph

Methods

Serial sections (up to 26) were prepared from 11 of 14 blocks. These undecalcifid blocks were embedded in perspex; slices 300 μm thick were sawn from them, and many were then hand ground to 70 μm. A few were microradiagraphed at this thickness and some were taken down to about



30 μm before mounting. A satisfactory thickness for subsequent examination of unstained sections was 70 μm.

The absence of cells leaves only the mineralised tissue on which to base the study of the pathological changes and their progress. The tissue is best examined in undecalcified sections in ordinary and polarised light, and in microradiographs. The arrangement of the collagen fibres, the number, size, shape and arrangement of the vascular and osteocyte spaces, and mineral distribution are the features available for study.

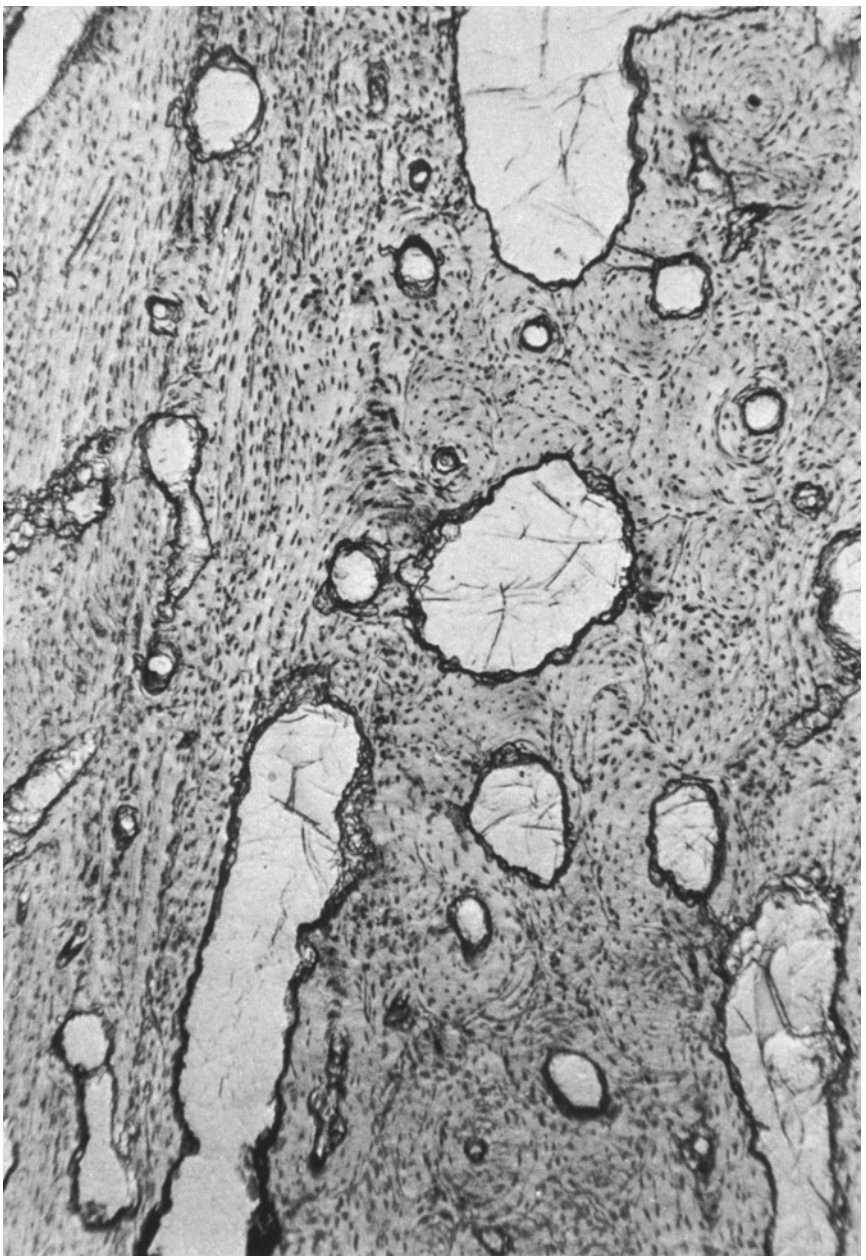


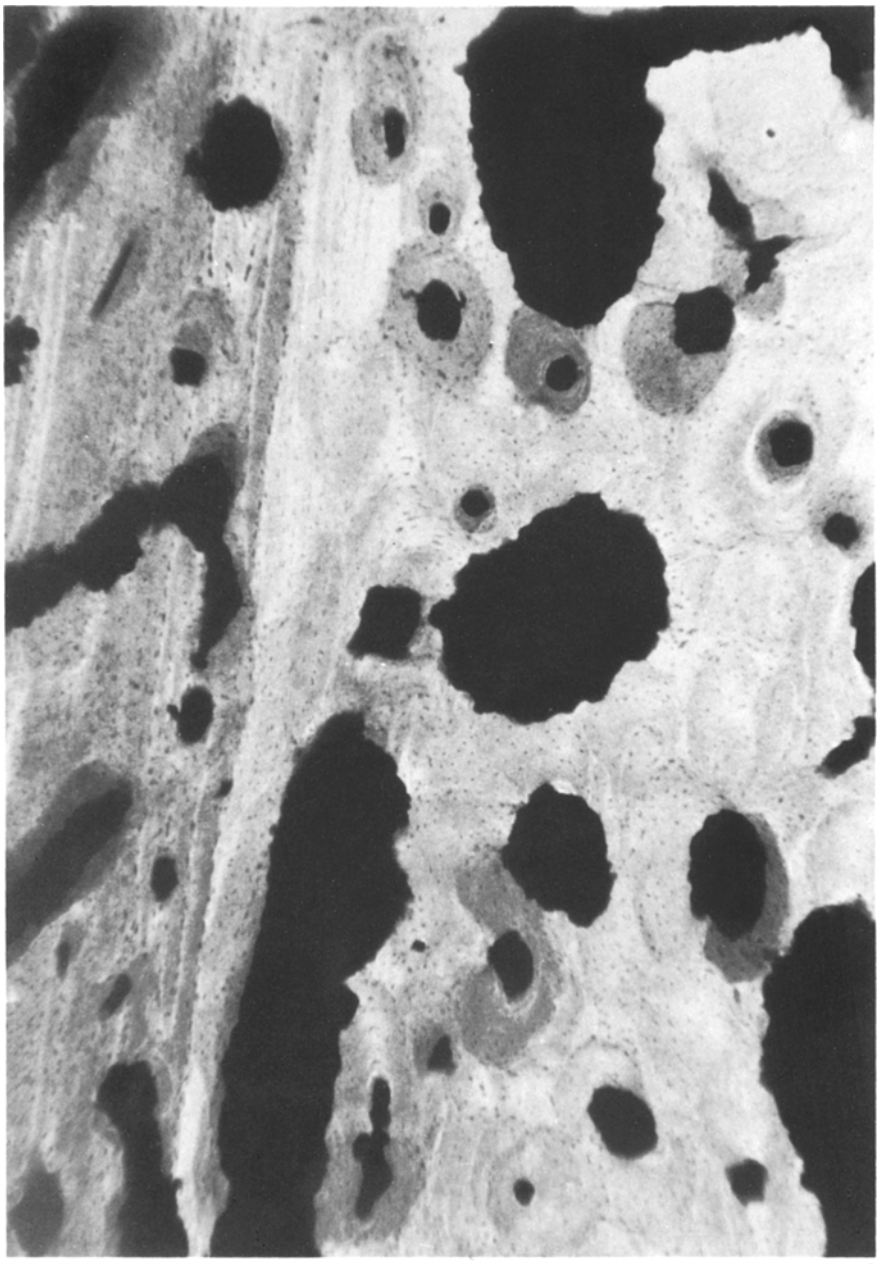
Fig. 6. Inflammatory osteoporosis in sclerosed bone. The osteocyte spaces in some areas are lenticular and regularly arranged. There are some small areas of fibre-bone. In the polarized light photomicrograph the holes are seen to cut across existing structures. The boundaries of the holes appear 'gnawed' in the microradiograph. Outer surface. (2A-25/26 lower. 70 μm. ×55). (See Fig. 3A). Ordinary and polarized light, and microradiograph

Fibre-bone (Figs. 7, 8) is recognised by the basket-work pattern of the collagen fibre bundles seen under polarised light, the more numerous, smaller, spherical and irregularly arranged osteocyte spaces, with shorter and more tangled canaliculi. Lamellar bone (Figs. 9, 10, 11) has an alternating orientation of successive collagen lamellae, less numerous lenticular more regularly shaped and arranged larger osteocyte spaces, with longer, thinner and straighter canaliculi. Fibre-bone is usually more mineralised and appears to be more rapidly deposited than lamellar bone. Sometimes the bundle bone type of Pritchard [4] may be seen.

6

Progress of Changes

Caries sicca is an inflammation of bone. As such one would expect hyperaemia and initial increase of vascular spaces. This reactive phenomenon would also be accompanied by fibre-blastic proliferation (granulation tissue), and an inflam-



matory cell infiltration. The effect of this tissue upon the bone is to modify the turnover or remodelling process. Normally this is a constant activity affecting 20 to 40% of bone surfaces with the two phenomena of resorption by osteoclasts and formation by osteoblasts. In normal individuals these are balanced leaving the mass and organisation of the bone relatively undisturbed. But when stimulated by trauma, infection or otherwise, resorption and formation are altered

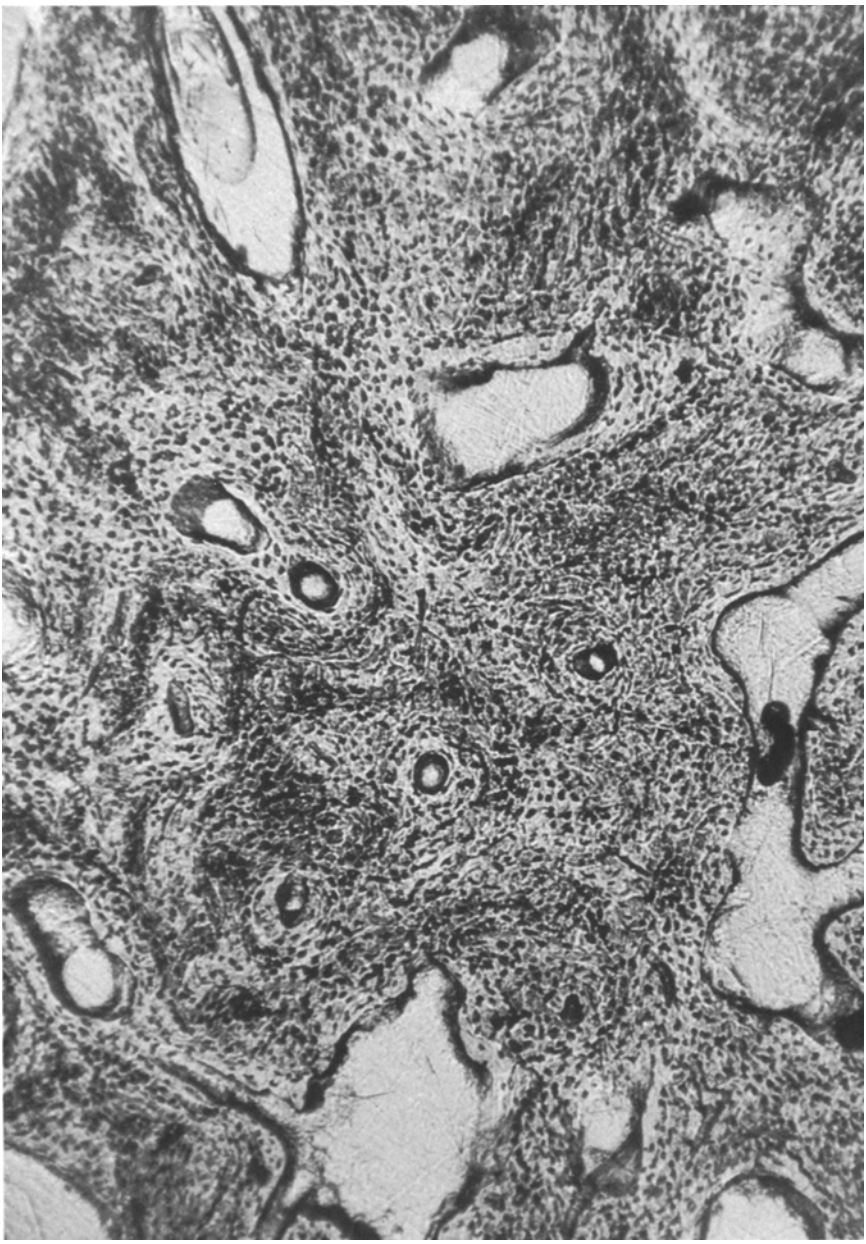
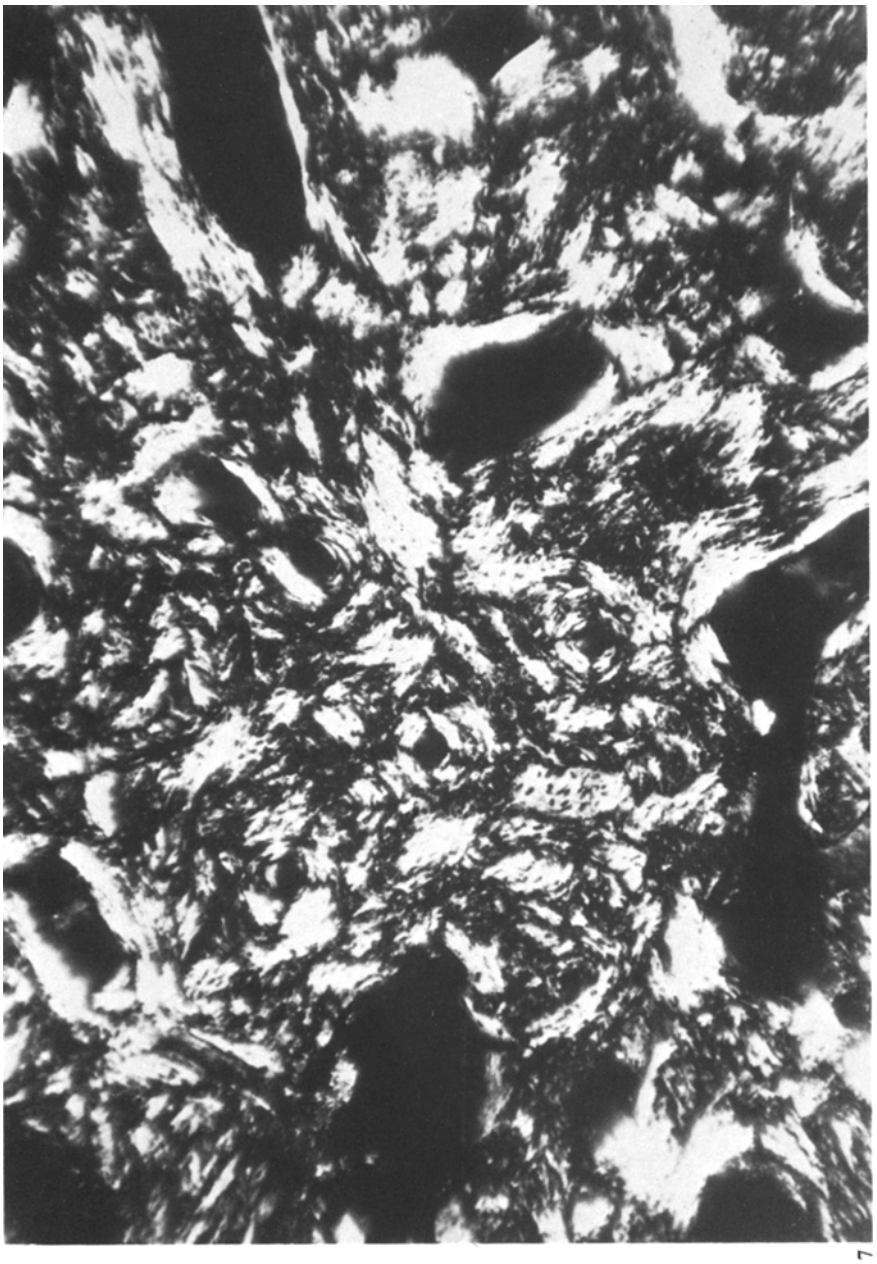


Fig. 7. Fibre-bone fills the spaces resulting from inflammatory osteoporosis. Small round osteocyte spaces are irregularly arranged. In the large canal on the left may be some remodelling osteoporosis. The denser (more mineralized) streams of fibre-bone are seen in the microradiograph. (5-15/25 left. 70 µm. ×40). (See Fig. 3B). Near outer surface. Ordinary and polarized light, and microradiograph

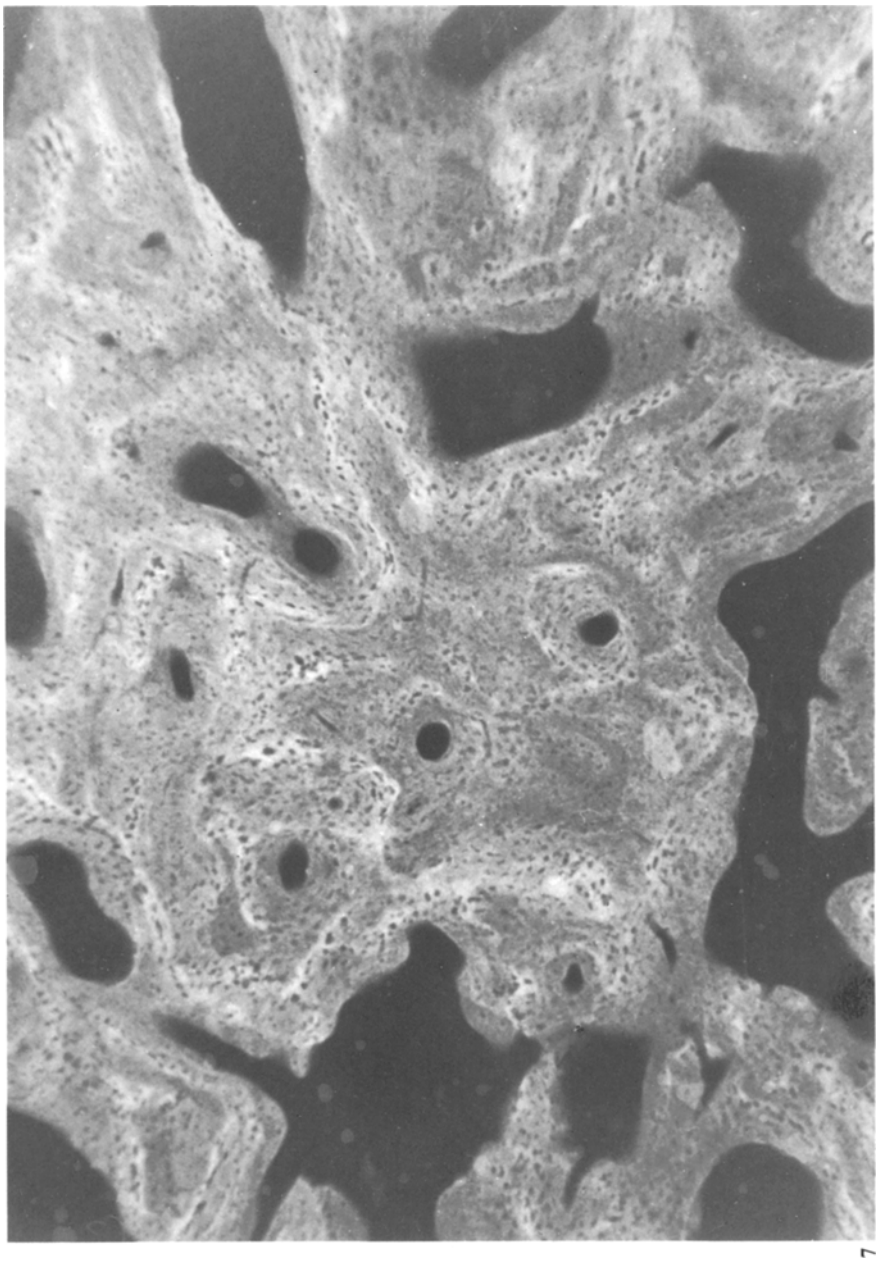
in rate and duration of activity, sometimes in opposing directions and sometimes in the same direction. The result can be considerable alteration in the architecture of the bone.

The following is a discription of these two processes which eventually lead to the multinodular appearance in the specimen.



Initial Inflammatory Osteoporosis

The earliest superficial change (Figs. 3A, 13-2) is a cluster or focus (about 30 mm) of pits less than a millimetre in diameter, opening on a slightly raised area on the outer surface. As the focus spreads the central pits become confluent



(Fig. 5). This is the 'confluent clustered pits' which develops from the earlier 'clustered pits'.

In this specimen these changes had occurred in sclerotic bone. In no section was normal bone seen.

In sections these pits are seen to lead into gaps (20–30 μm) in the mineralized

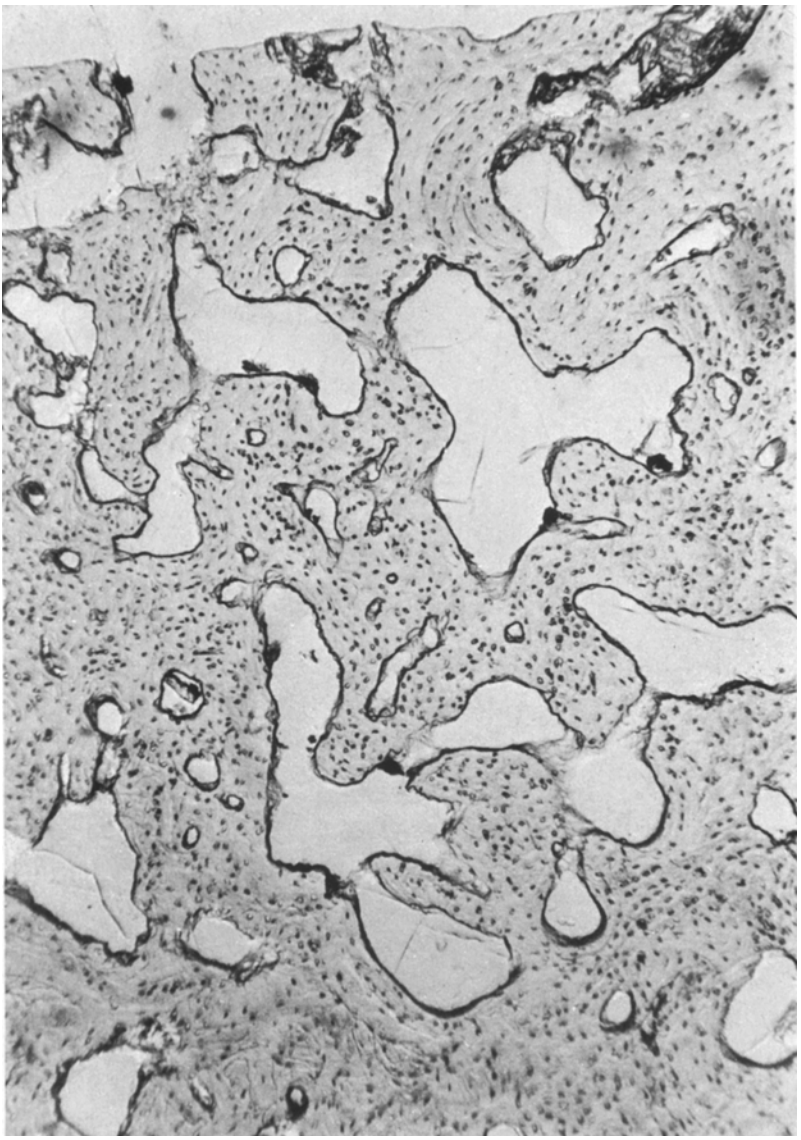
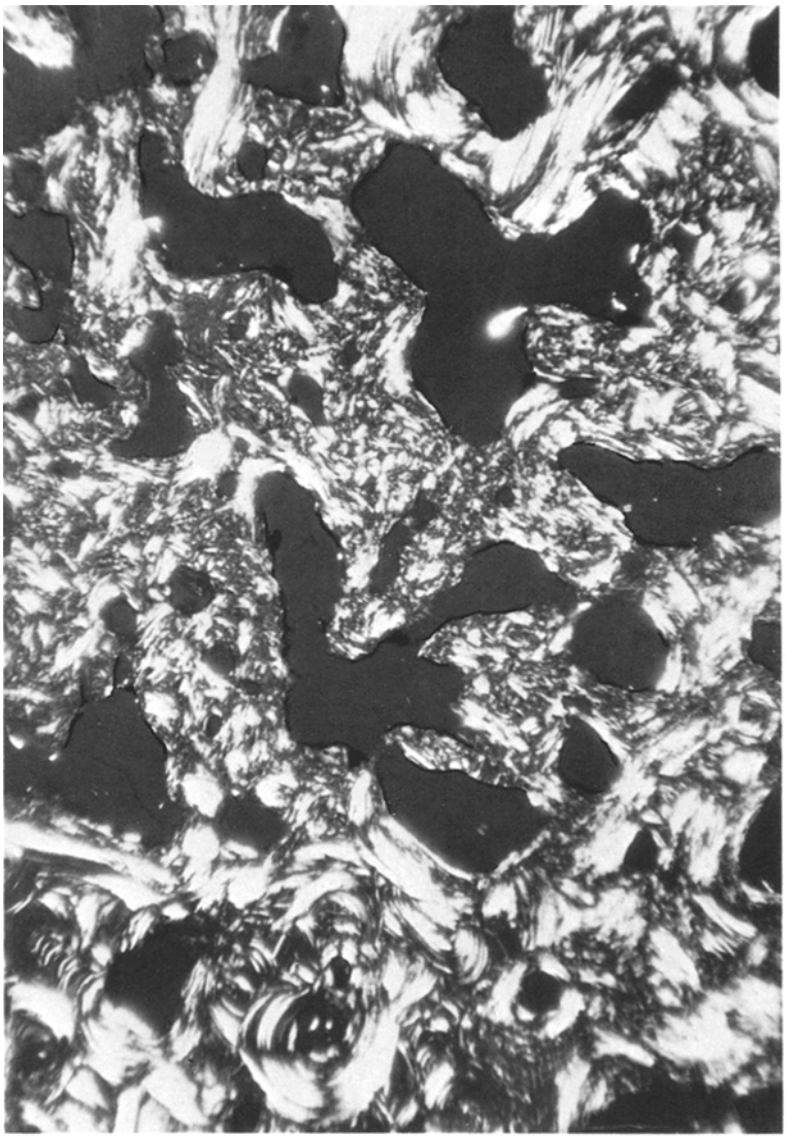


Fig. 8. Remodelling; stage i. Osteoposoris of fibre-bone. The margins of the holes are well-defined and only slightly denticulate. (2B-17/25 left. 70 μm. ×60). (See Fig. 3A). Ordinary and polarized light, and microradiograph

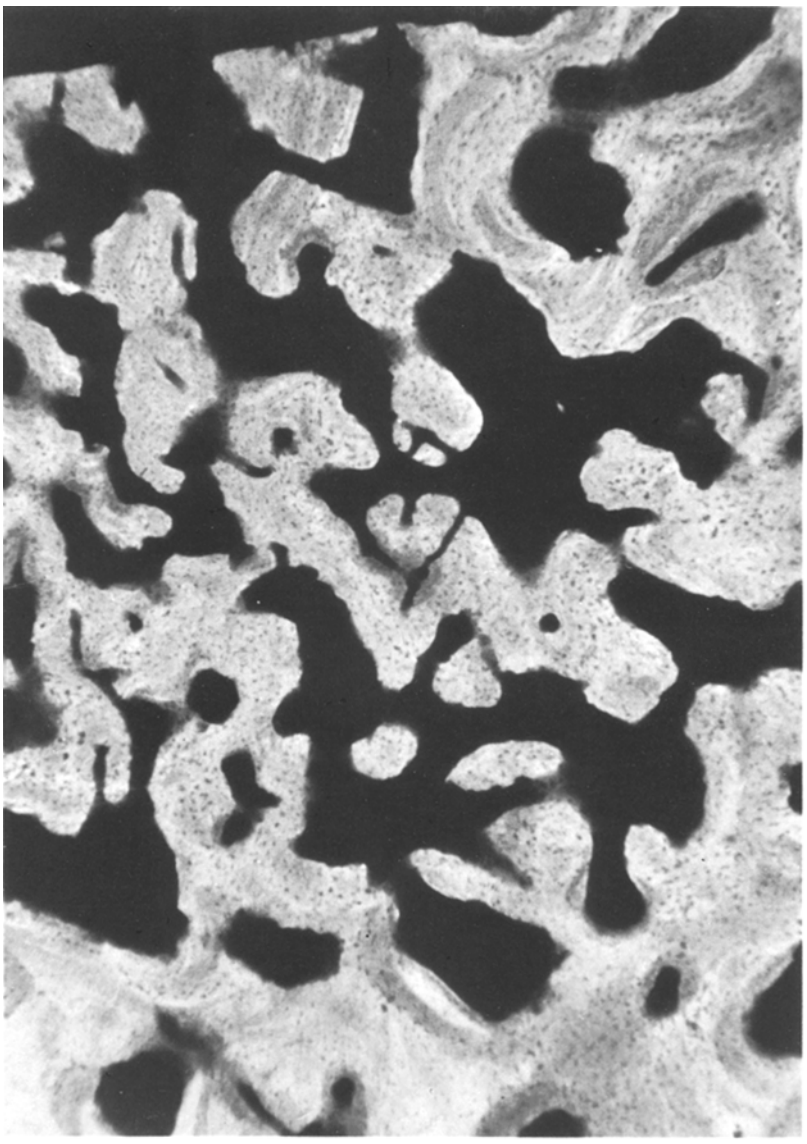
tissue (Fig. 13-2). These early inflammatory holes are rounded, but their shape depends upon the plane of the section and confluence with adjacent holes. Their walls are rather finely and irregularly dentate, or gnawed (Fig. 6). This absorption of the bone by osteoclasts comes from the subacute syphilitic inflammation, and probably commences at the junction of the inner surface of the outer table with the diplöe (Fig. 13-1). This is an initial inflammatory osteoporosis. These vascular channels are numerous and may reduce the tissue to a net-work of thin trabeculae.



Periosteal bone is deposited on both surfaces of the calvaria but perhaps is pitted over a smaller area than the underlying osteoporosis.

In the centre of the focus there is considerable loss of tissue, up to 10 mm, from confluence of the holes, so that the focus opens onto the outer surface (Fig. 13-3).

The inflammation extends in the diplöe, but similar large openings are not found on the meningeal surface. Unlike what occurs in tuberculosis, no sequestra are formed.



These superficial destructive discrete foci result in the shallow openings on the outer calvarial surface which, on healing pass through the stages of 'focal superificial cavitation', 'circumvallate cavitation' (Fig. 1) to 'radial scars' [2]. Discrete changes were scanty in this specimen. In the previous study [2] the intermediate changes of the contiguous forms that precede caries sicca, namely 'serpigenous cavitation' and 'nodular cavitation', were seen in many calvariae.

In periosteal new bone on the meningeal surface there are fewer pits leading into small (30 μm) vascular channels.

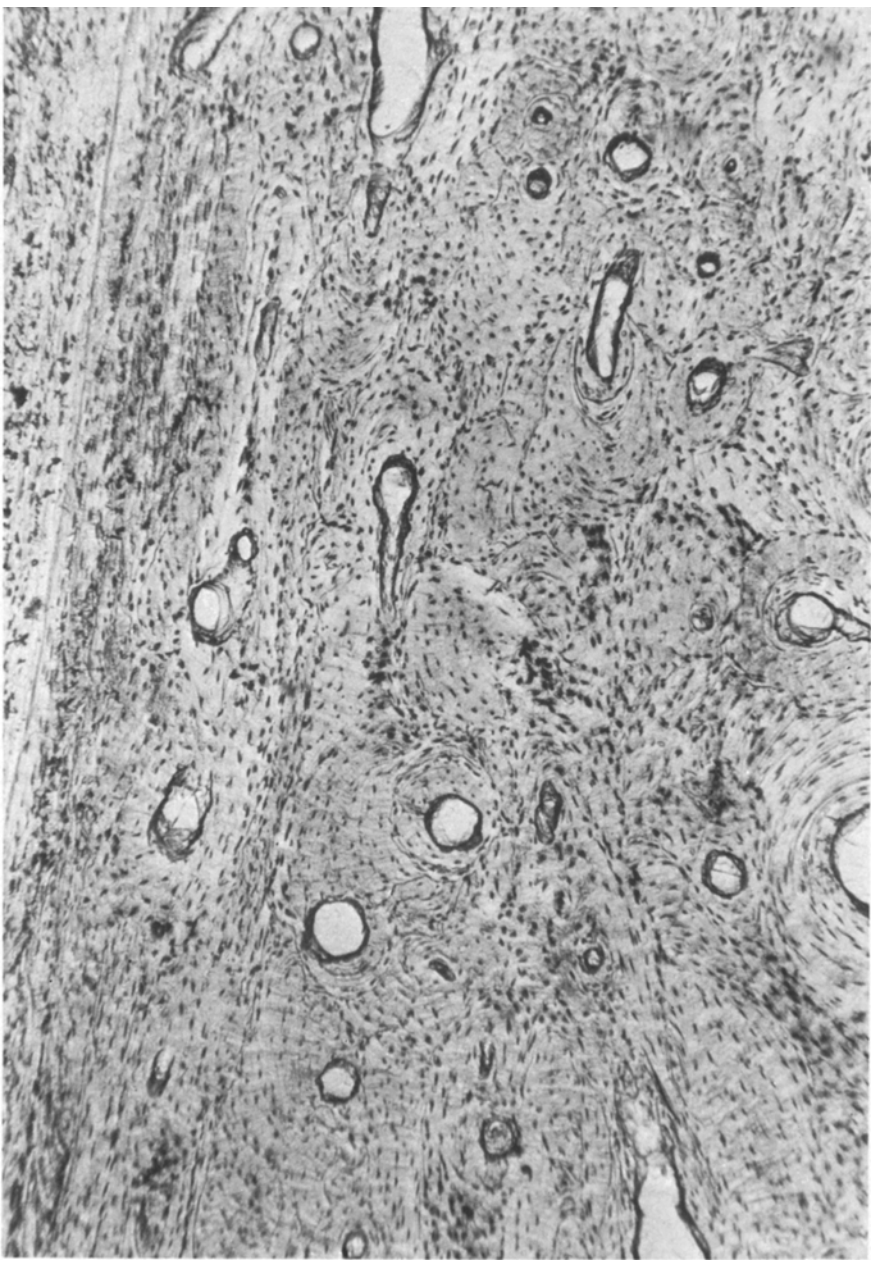
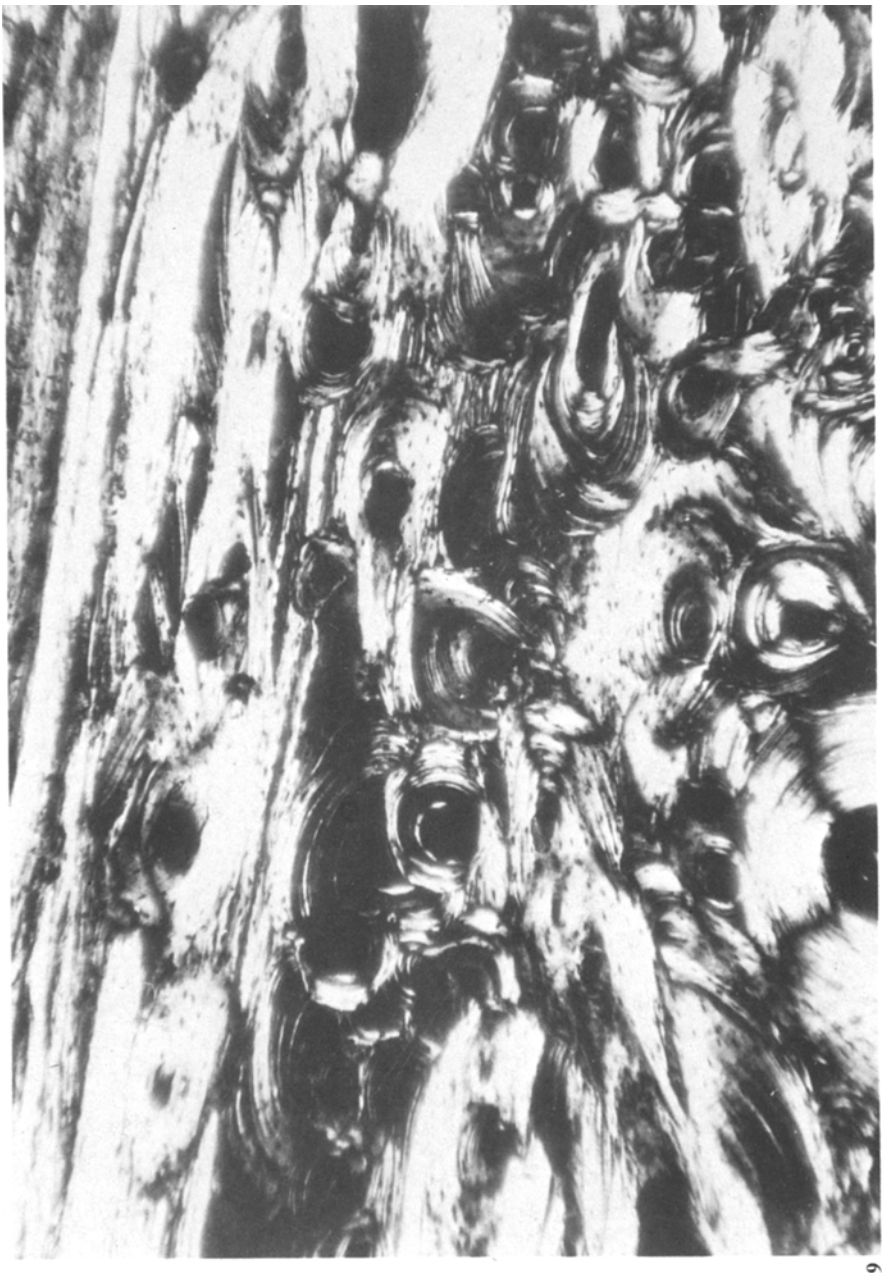


Fig. 9. Remodelling; stage ii. Lamellar bone filling. The larger osteons are filled by lamellar bone, but some fragments of fibre-bone persist in interosteon tissue. (2A-15/26 lower. 70 μm. ×60). (See Fig. 3A). Near outer surface. Ordinary and polarized light

At first the surface of this meningeal new bone has a distinctive finely arabesque pattern [2] from the imprint of the small blood vessels arising from the endosteal tissue and under the influence of the pulsating intracranial pressure. As healing proceeds this pattern is lost, the surface becomes shiny (Fig. 2), and the grooves of the normal meningeal vessels may be roofed over.

Periosteal penetrating (Sharpey's) fibres are often seen in the outer deposits [6].



Fibre-Bone Filling

As the inflammation subsides the holes are filled from their walls with rapidly mineralized fibre-bone (Figs. 13-3, 10). This change spreads centrifugally from the centre to the periphery of the focus. There is thus more mineralized tissue

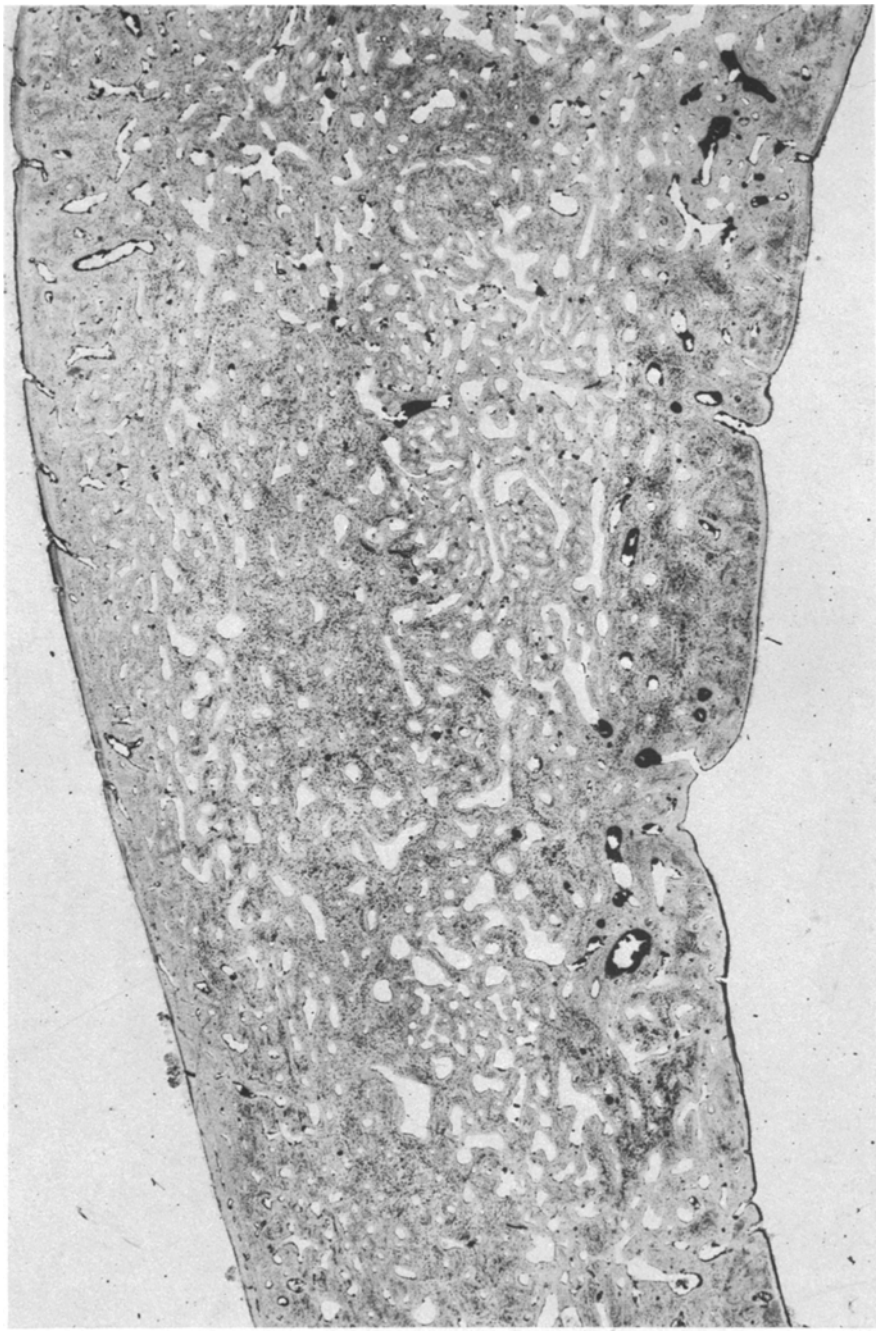
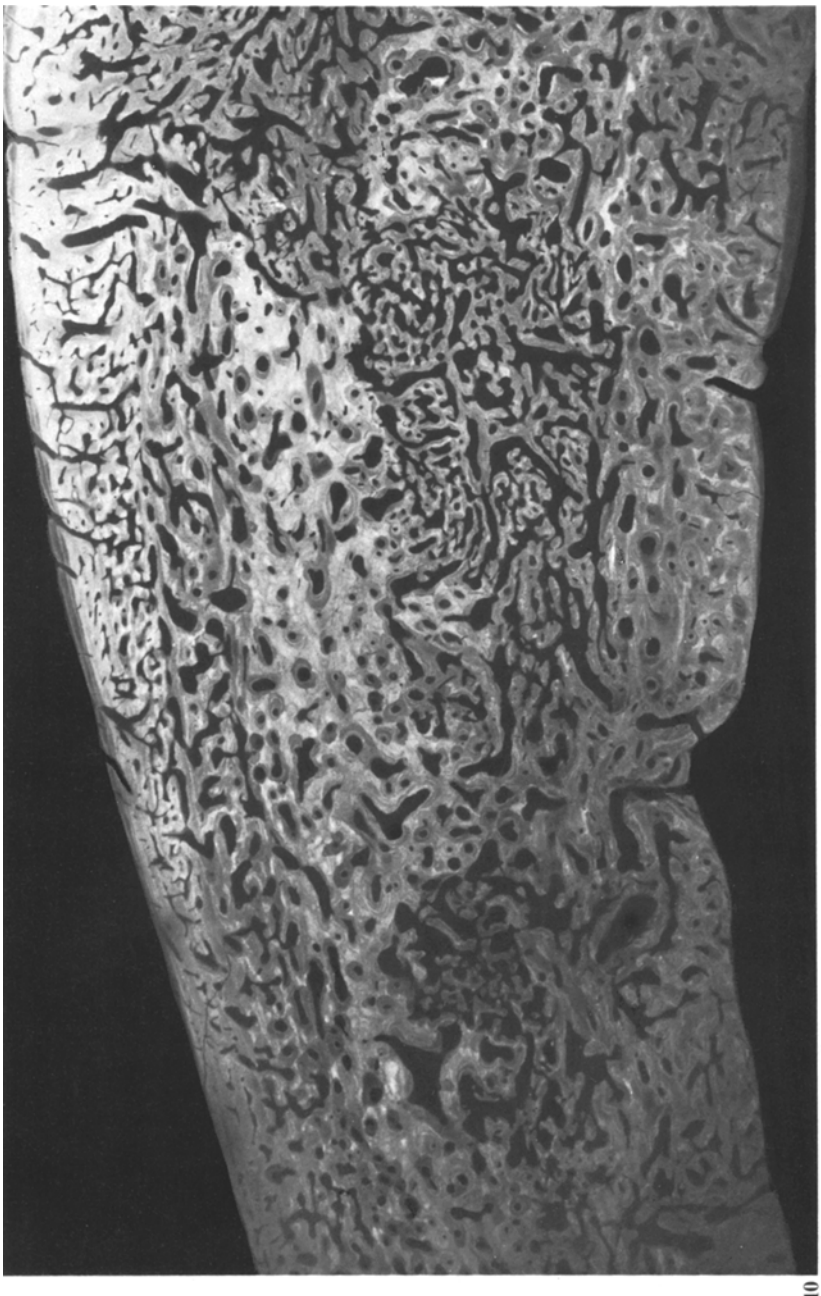


Fig. 10. Periphery of inflammatory focus in sclerosed bone. Osteoposoris in on the right. There is much remodelling of fibre-bone. The contrasts are marked in the microradiograph. (2B-17/25 left. 70 and 30 µm. ×10). (See Fig. 3A). Ordinary light and microradiograph



than holes, and the bone is more solid (Fig. 7). Residual fragments of lamellar bone may persist outside this bone.

To the unaided eye in reflected light fibre-bone in these unstained sections appears white, while under low-power microscopy in transmitted light it is dark from the concentration of the osteocyte spaces and their canaliculi.

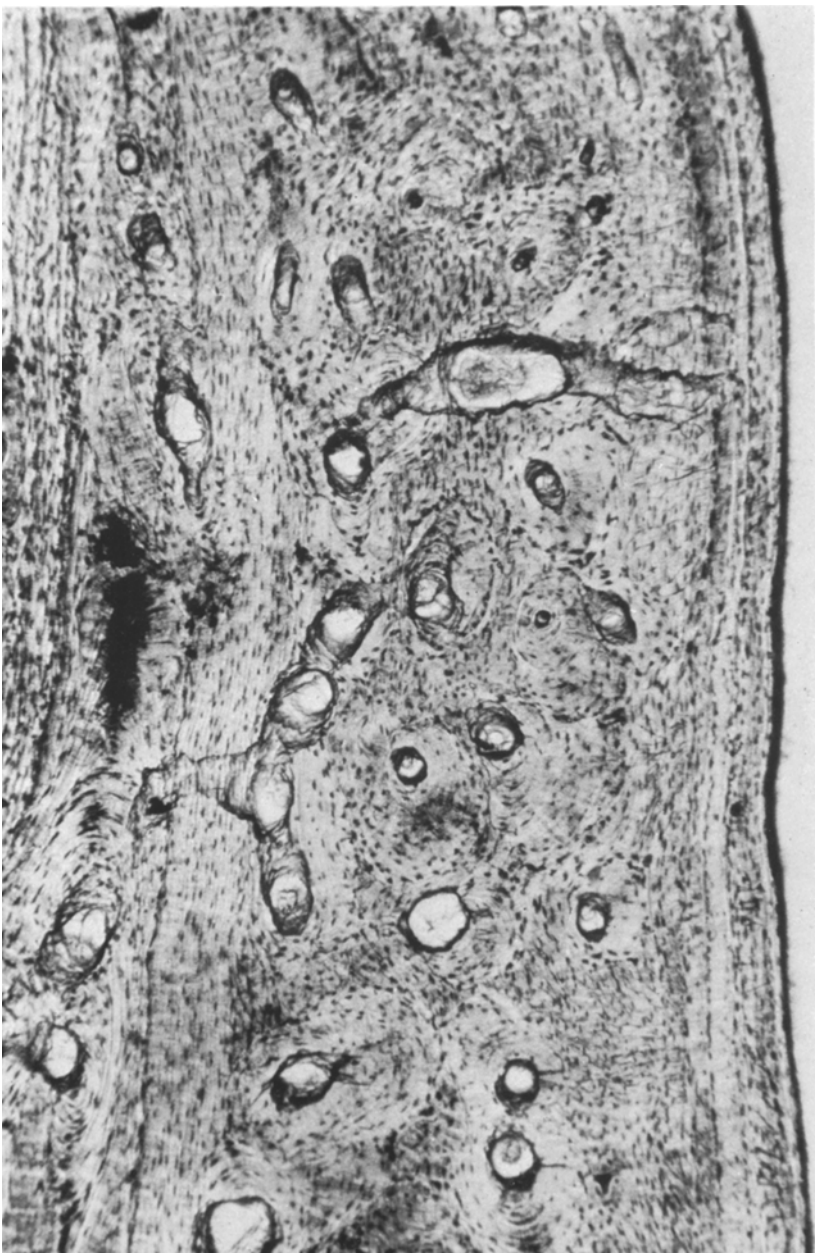
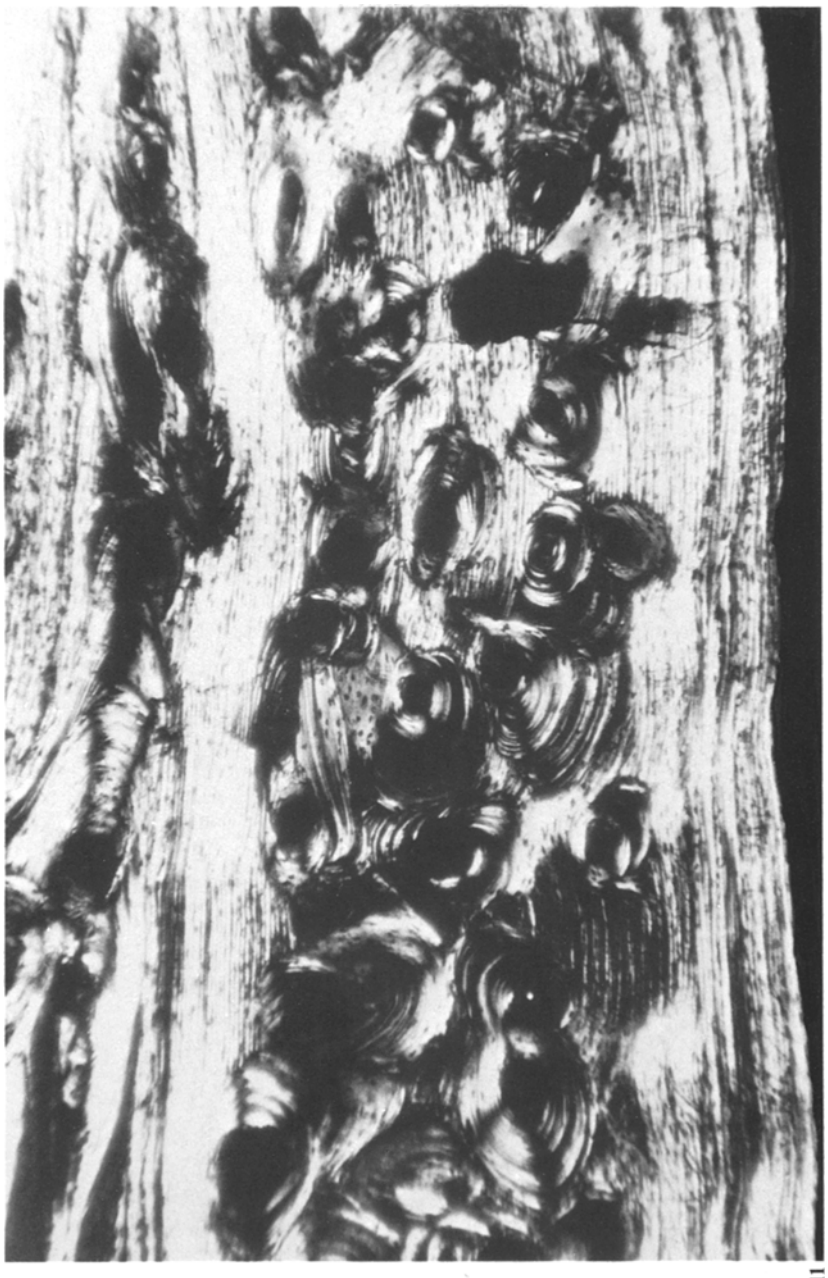


Fig. 11. Periosteal new bone. Straight parallel lamellae persist, and cross the upper half of the figure. This is a remnant of the original outer table, now buried in the sclerotic periosteal new bone. All the bone is sclerotic but some further inflammatory osteoporosis may be active in the larger gaps lying diagonally across the field. A zone of many small osteons beneath the surface is frequently seen; it may lie in the new bone or beneath it. (2A-19/26 lower. 70 μm. ×36). (See Fig. 3A). Meningeal surface. Ordinary and polarized light, and microradiograph

Remodelling

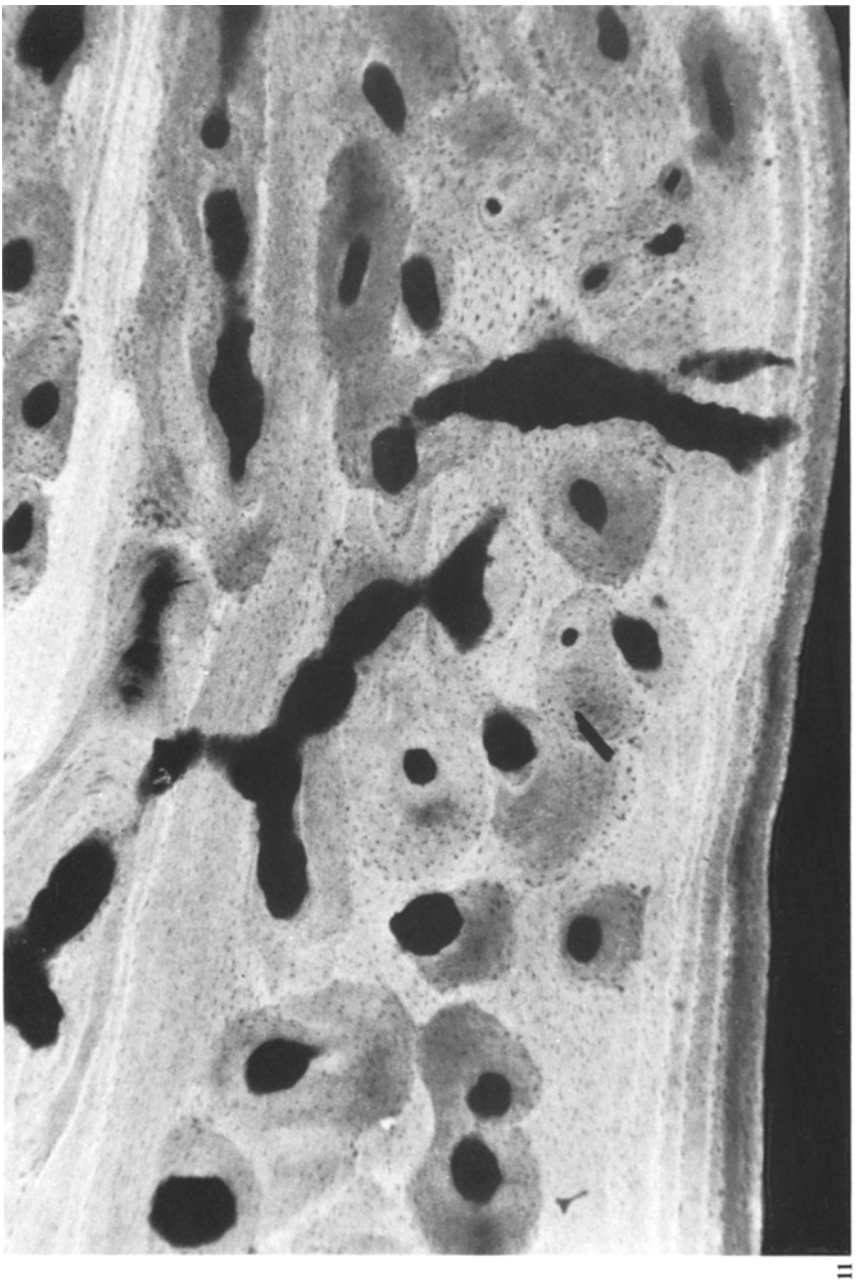
This fibre-bone is next remodelled (Fig. 13-4) into lamellar bone in two stages.

The first is the rapid removal of the fibre-bone by osteoclasts to produce a remodelling osteoporosis (Fig. 8) by holes with only slightly irregular boundaries. Weber's [6] findings in syphilitic changes in the shafts of long bones



help the understanding of the changes in caries sicca in the calvaria. A distinct boundary line may occasionally be seen between the new periosteal bone and the original cortex.

Next, on the walls of the holes in the remodelling osteoporosis, lamellar bone is less rapidly laid down until only small channels remain. These are



the remodelled osteons (Fig. 9) and their canals. Though much of the fibre-bone will be removed, remnants may persist in the new interosteon tissue.

The progress of the remodelling follows the previous centrifugal spread of the fibre-bone filling. In time mainly lamellar bone is present in osteons of many sizes. The bone is dense and the connecting (Volkmann's) canals are numerous and narrow. The bone is now sclerotic (Figs. 4, 5).

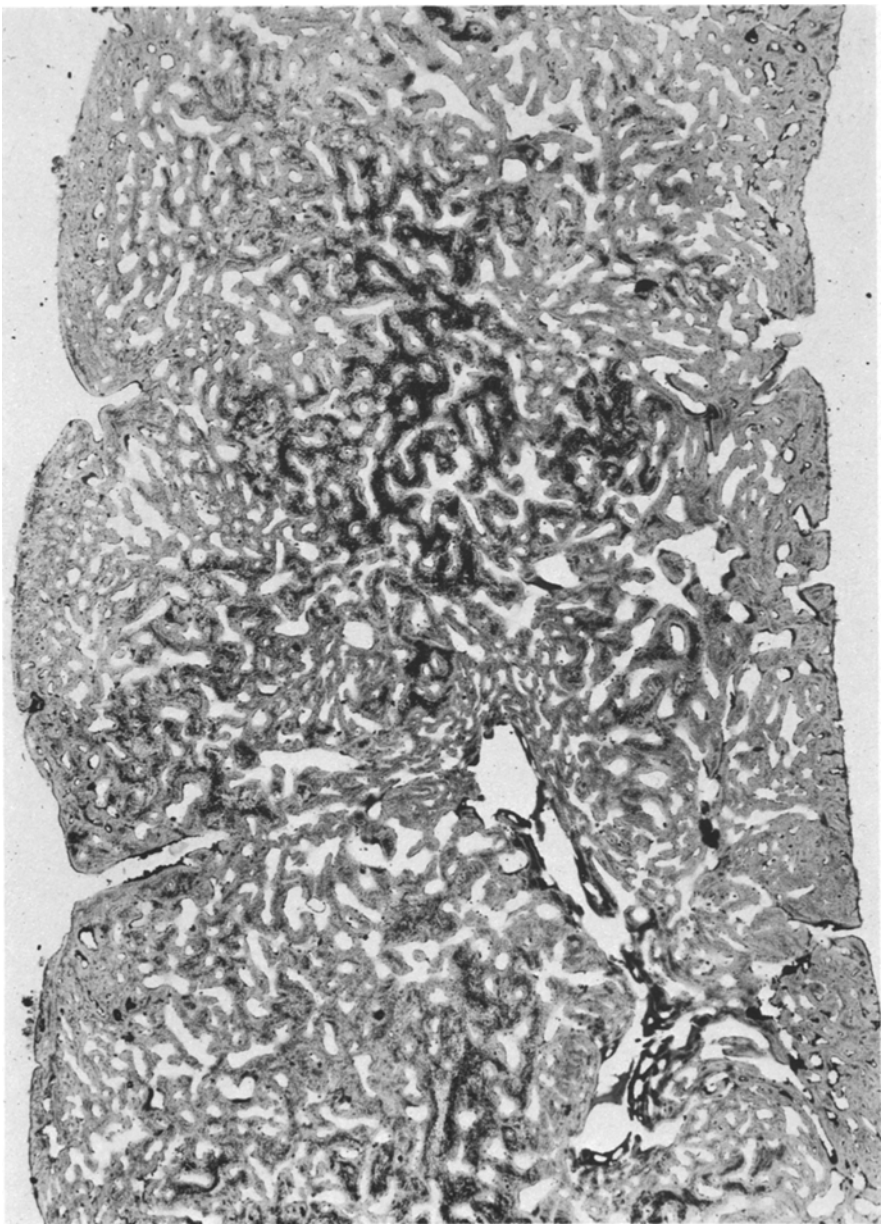
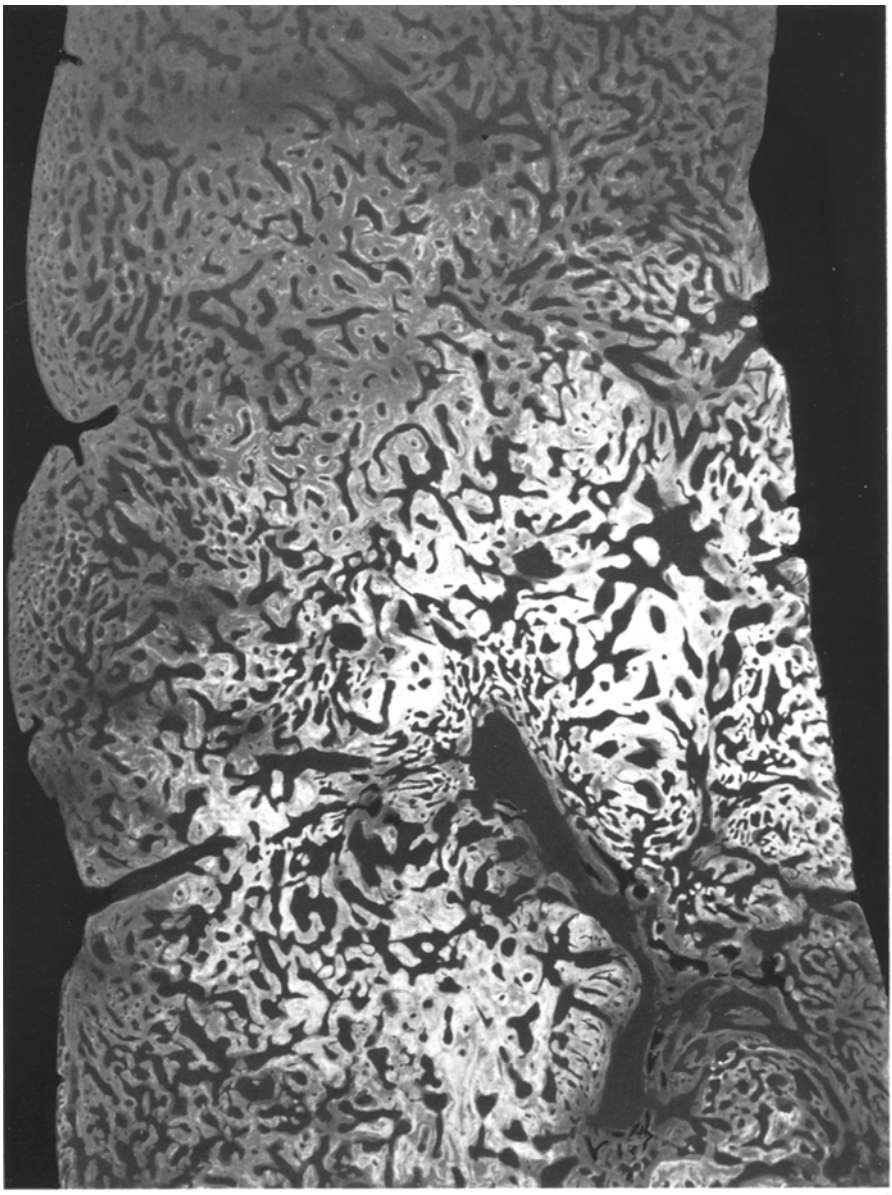


Fig. 12. Multi-nodulation; type i. Most of the tissue is fibrebone especially in the dark tracery. The vascular channels in the side nodules may point inwards, while those in the central nodule appear to come from the diplöe. (9-8/25 left. 70 µm. × 10). (See Fig. 3C). Ordinary light and microradiograph

The extent of the periosteal thickening can be assessed by the fragments of parallel lamellae in the remodelled bone lying away from the inner and outer surfaces of the calvaria (Fig. 11). Periosteal thickening on both surfaces may increase thickness of the calvaria to about three times.

The succession of inflammatory osteoporosis, fibre-bone deposition, and remodelling proceeds centrifugally until the periphery of the focus is reached



12

(Fig. 13-6). Often a difference could be recognised between the remodelling bone of the subsiding focus and the surrounding sclerosed bone which had replaced the normal bone. The pattern is sometimes further complicated by the appearance of more holes in the remodelling bone at the centre of the focus even before it is quiescent. This is a relapse of active disease so characteristic of the treponematoses.

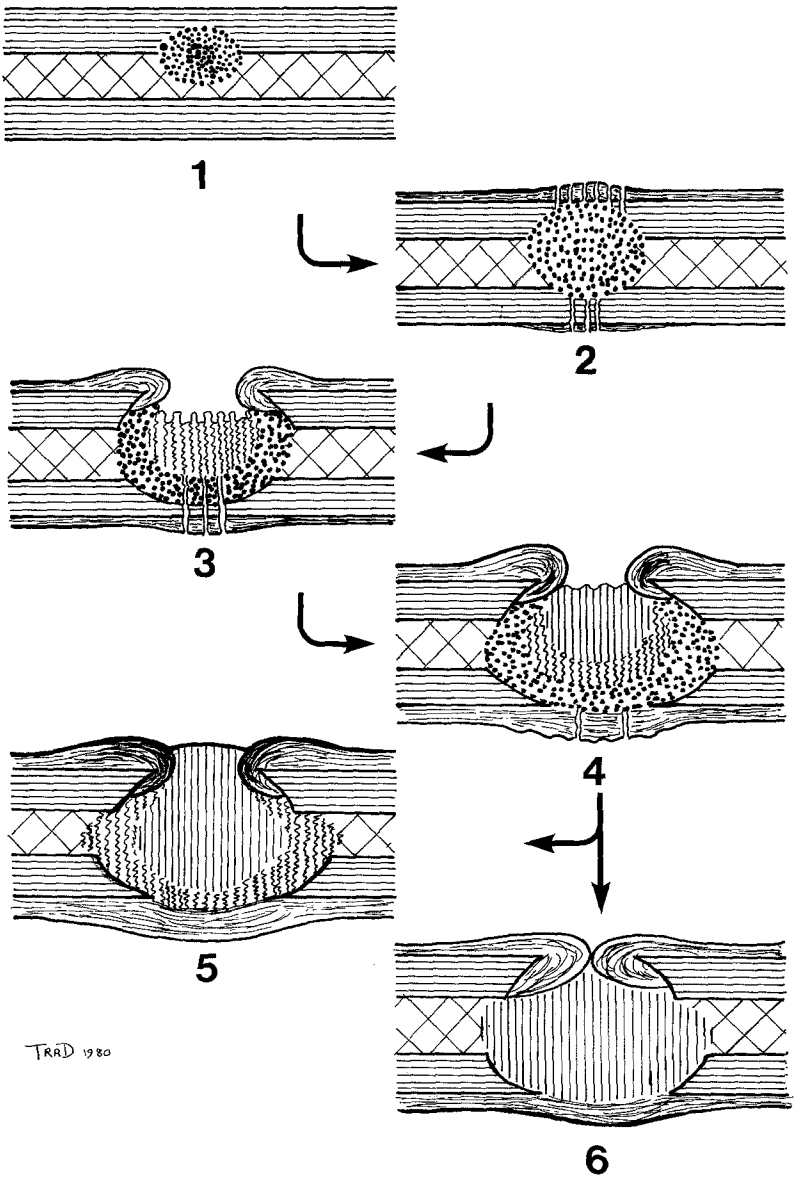


Fig. 13. Porposed sequence of caries sicca. The active pathological changes start as inflammatory osteopososis at the junction of the inner surface of the outer table and the diplöe (1), and extend in all directions (2). These holes are then filled by fibre-bone (3). This is next remodelled by osteoporosis followed by filling by lamellar bone (4, 5), which results in healing and sclerosis (6). Each of the two lower figures has a different pattern of multi-nodulation, types i and ii, but the internal patterns indicate the next to last and the last stages of remodelling. The periosteal new bone goes through a similar sequence, and ends in sclerosis. This is not shown in the above diagram

Multi-Nodular Surface changes

During this remodelling, the open foci on the surface of the outer table, may undergo two development.

a) The raised margins may flatten and meet the raised, and now smooth, central diplöeal nodule so that three almost equal nodes result, type i (Figs. 12, 13-5),

b) If the two raised margins are not far apart, less than 5 mm, they may approach and join each other to form a fissure beneath which small crevices remain each side of the submerged diplöeal nodule, type ii (Fig. 13-6).

The direction of the vascular channels in some nodes suggests these processes.

The above changes are those of discrete foci. In caries sicca, however, there are contiguous and confluent foci as well as further active changes in already changed bone, so that a complicated picture results (Fig. 1). As Virchow [5] (Fig. 10 in [2]) said the nodules and fissures between them in caries sicca form a pattern which is unmistakable, but difficult to describe. Multi-nodulation occurs only on the outer table.

The appearance of caries sicca may arise from the confluent focal destructive changes of syphilis which result from the focal endarteritis obstruction. Somewhat similar changes occur in treponemal changes in long bones, as "Nodes/expansions with superficial cavitation" [2]. Brookes [1] stresses the outward arterial flow from the meningeal vessels through the diplöeal sinuses and smaller vascular channels to the outer surface of the calvaria.

References

1. Brookes M (1971) The blood supply of bone. *An approach to bone biology.* Butterworth, London, pp 117–122
2. Hackett CJ (1976) Diagnostic criterial of syphilis, yaws and treponarid (treponematoses) and of some other diseases in dry bones. *Sitzungsber. Heidelb. Akad. Wiss.* 4. Figs. 3, C, H, 10
3. Hackett CJ (1981) Microscopical focal destruction (tunnels) in exhumed bones. (In Press)
4. Pritchard JJ (1972) General histology of bone. In: Bourne GH (ed) *The bio-chemistry and physiology of bone,* 2nd edn. 8. Academic Press, London
5. Virchow R (1896) Beitrag zur Geschichte der Lues. Derm Z 3:1–9. (Translation in the libraries of the Royal College of Physicians, Institute of Orthopaedics, and Royal Society of Medicine London, and Natural History Library, Smithsonian Instititution, Washington, D.C.
6. Weber M (1927) Schliffe von mazierten Röhrenknochen und ihre Bedeutung für die Unterscheidung der Syphilis und Osteomyelitis von der Osteodystrophia fibrosa sowie für die Untersuchung fraglicher syphilitischer prähistorischer Knochen. Beitr path Anat 78:441–511 (Translation in Libraries of the Royal College of Physicians, and Institute of Orthopaedic, London

Accepted November 3, 1980