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The fine structure of the proximal growth plate of the avian tibia: vascular supply

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INTRODUCTION

The vascularity of the growth plate has an important role in the pathogenesis of certain diseases of long bones as well as a vital role in regulating their growth in length. Nairn (1975) emphasises the role of the metaphyseal blood vasculature in establishing avian osteomyelitis. Furthermore, he claims that direct vascular communication between metaphyseal and epiphyseal vessels at the proximal end of the tibia permits the rapid spread of osteomyelitis to the epiphysis, thence producing a subsequent suppurative arthritis. Controversy exists with regard to the presence of direct communication of metaphyseal and epiphyseal blood vessels across the growth plate in postnatal vertebrates. This issue is somewhat confounded by the fact that birds characteristically have cartilaginous rather than osseous epiphyses (Beaumont, 1967; Wise & Jennings, 1973), with the exception of the tibia and metatarsal bones (Wise & Jennings, 1973; Nairn & Watson, 1972). Beaumont's (1967) study by light microscopy of the intraosseous vasculature of the ulna is the only complete description of an avian intraosseous vasculature. Although little has been published on the vascular supply to the growth plate, Lutfi (1970), while examining the growth of the proximal tibial epiphysis in the bird, has not found any communication between epiphyseal and metaphyseal blood vessels coursing through the growth plate.

Broiler chickens often suffer tibial dyschondroplasia, which produces significant deformities in chickens of marketable age (7–9 weeks). Dyschondroplasia of this type is considered to be a direct consequence of an inadequate metaphyseal vascular supply (Wise & Jennings, 1972; Riddell, 1975). Therefore it was decided to examine the fine detail of the vascular supply of the normal proximal tibial growth plate, to provide a basis for studying possible vascular involvement in the pathogenesis of this form of dyschondroplasia.

MATERIALS AND METHODS

Tissues were collected from ten normal White Leghorn and ten normal commercial broiler chickens at seven weeks of age. The birds were given 2000 i.u. of heparin intravenously before anaesthesia some fifteen minutes later with intravenous sodium pentobarbitone. The ischiatic artery, erroneously called the femoral artery in previous papers (Howlett, 1979, 1980), was then cannulated from the medial aspect and perfused with Tyrode's solution to which sucrose and lignocaine had been

added to make final concentrations of 5% (w/v) and 0·1% (v/v) respectively. Perfusion was performed at a pressure of 180 mmHg, using at least 50 ml of modified Tyrode's solution or until the fluid returning in the ischiatic vein was clear. After this initial flushing of the vascular tree, perfusion was continued with various liquid markers: colloidal carbon; 'colorpaque' (Pilot Chemical Co., Ware, Herts, England) at full or half strength (dilution with isotonic saline); latex; or partially polymerised methyl methacrylate, either through the ischiatic artery or the nutrient artery of the tibia. Perfusion with the marker was continued either for about ten minutes or until the marker was apparent in the venous return. At this point all major veins and arteries were ligated and the limb, disarticulated through the hip, was immersed in 10% buffered formalin for ten days.

Methyl methacrylate casts

Following fixation, tissue perfused with methyl methacrylate was immersed in a water bath at 50 °C for four hours to ensure complete polymerisation of the resin. The medial aspect of the metaphysis and growth plate was then exposed by sectioning. The tissue was gently irrigated with 10 M hydrochloric acid followed by hot concentrated potassium hydroxide to the point where the vascular casts were clearly exposed. The tissue was then washed in running water for one hour and the preparation sliced from the bone. This slice was critical point dried, coated with gold palladium and examined by scanning electron microscopy.

Latex casts

The procedure for exposure of these specimens was similar to that for the resin casts. Preparations were examined using a Zeiss stereomicroscope with a maximum magnification of ×70.

Colloid carbon and colorpaque markers

Hand cut sections, ranging in thickness from about 100 to 150 μm, were cut from limbs that had been perfused by colloid carbon and colorpaque. Prior to examination by light microscopy these sections were cleared by immersion in a solution of glycerine for at least one month.

Two normal White Leghorn and two normal broiler chickens were perfused with Karnovsky's fixative and sections were prepared for transmission electron microscopy as described previously (Howlett, 1979, 1980). In addition, tissue fixed in buffered formal saline and embedded in paraffin was serially sectioned at a thickness of 4–5 μm both longitudinally and transversely, in order to observe any direct blood vascular communications across the growth plate.

RESULTS

The blood supply to the hind limb was derived from two arteries, the arteria ischiadica and the arteria iliaca externa, with the latter supplying only the anterior part of the crural region (Nishida, 1963). The arteria ischiadica gave rise to the arteria poplitea which in turn gave rise to the arteria genu suprema, arteria tibialis anterior and arteria tibialis medialis (Nishida, 1963). These three arteries and their branches surrounded the anterior portion of the tibia, sending many small nutrient arteries directly to the proximal tibial metaphysis and epiphysis.

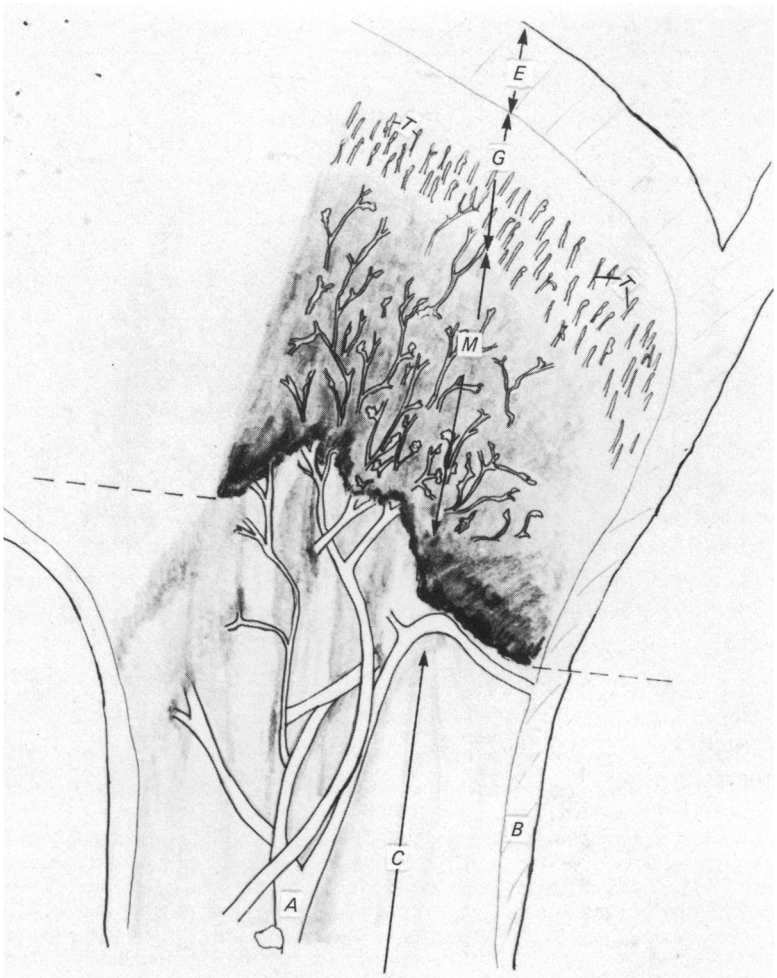

Fig. 1. A drawing of the proximal end of the tibia depicting the vasculature of the metaphysis. The vessels were outlined by latex perfusion of the tibial nutrient artery. The drawing is from the latex casts of these intraosseous vessels. Vessels below the line (- - -) are in the marrow cavity; while those above the line have been exposed by sectioning the metaphysis and growth plate longitudinally and eroding the surrounding tissue by sequential irrigation with concentrated solutions of potassium hydroxide and hydrochloric acid. *E*, epiphyseal cartilage; *G*, growth plate; *M*, metaphysis; *C*, marrow cavity; *A*, large ascending branch of the nutrient artery of the tibia; *T*, terminal metaphyseal arterioles; *B*, compact diaphyseal bone.

Metaphyseal vessels

The arteria tibialis anterior passed between the tibia and fibula; where it emerged onto the anterior surface of the tibia, it supplied the tibia with its main nutrient artery via the major nutrient foramen of the tibia. This foramen was situated at the junction of the proximal and middle thirds of the bone. The nutrient artery passed through the cortex at an oblique angle for some 10 mm and, upon entering the marrow cavity, continued in the same direction for a further 1·5–2·0 mm whereupon it divided into a proximal and distal branch.

The proximal branch immediately ascended and after 2 mm divided into two branches. These vessels coursed for a further 5 mm giving off occasional twigs which

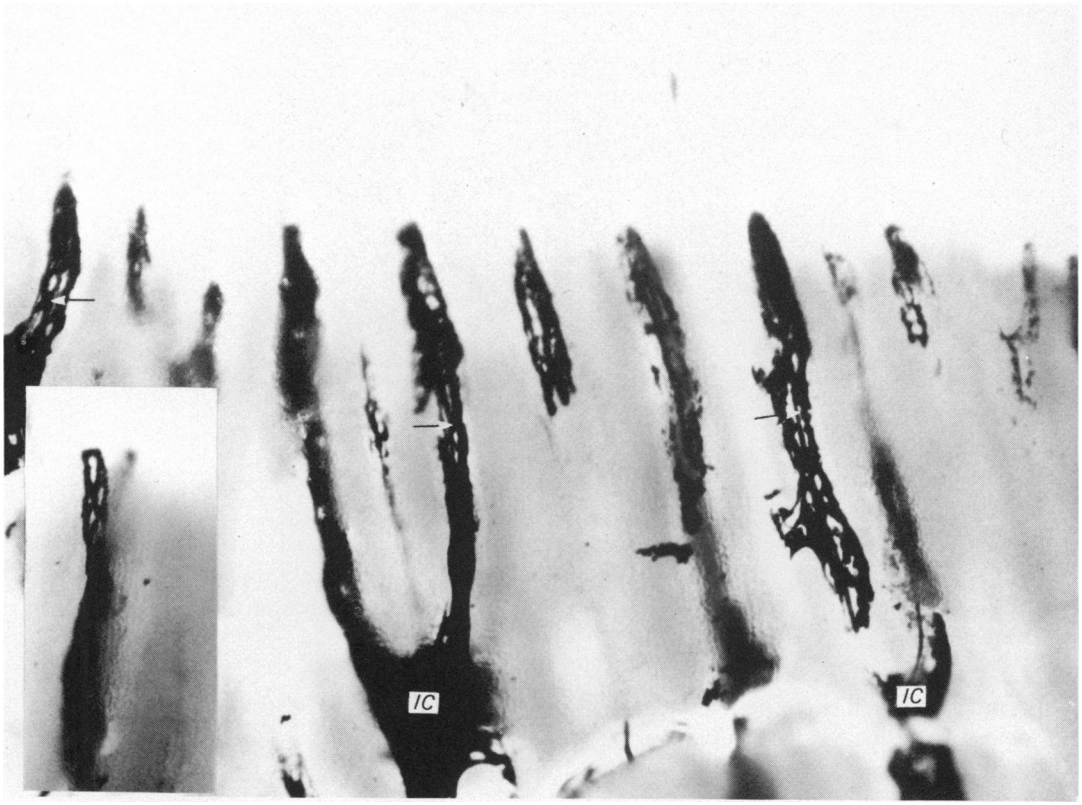

Fig. 2. Colloidal carbon outlines the metaphyseal vascular complexes. The centrally positioned arteriole (arrows) is enmeshed by a plexus of irregularly shaped venous sinusoids. Intercommunicating venous channels (*IC*) occur about 5 mm from the most centrifugal site of metaphyseal vascular penetration. Unstained. × 80. *Inset*. A tortuous 'basket weave' of venous sinusoids has been outlined by carbon perfusion. The nutrient artery of the tissue was isolated, cannulated and perfused with colloidal carbon. The sections were approximately 200 μm thick and cleared in glycerine for 4–6 weeks. Unstained. × 80.

flowed perpendicular to the endosteal surface. At the level of the nutrient foramen, the nutrient artery branched and subdivided until, at the level of the diaphyseal-metaphyseal junction, there was a spray of some 12–14 branches. During the passage through the marrow cavity some branches arose at an acute angle and coursed obliquely along the shaft for variable distances in the direction of the parent ascending artery before turning towards the diaphyseal cortex, branching as they approached the shaft wall. These vessels did not reach the metaphysis as a rule, since most vessels which penetrated the cortex anastomosed with periosteal vessels. However, some arteries continued within the cortical bone for considerable distances finally leaving it through either the periosteal or endosteal surfaces, to anastomose with either periosteal or metaphyseal vessels (the latter circumstance was observed only occasionally). From the medullary aspect of the metaphysis, further arterial subdivision occurred so that each osteochondral tunnel within the 'sponge-like' metaphyseal endochondral zone had at least one tributary (Fig. 1). At the proximal end of the metaphysis, there was a dense arborisation of the branches of the nutrient artery and each terminal vessel usually provided a cluster of four to six arteriolar

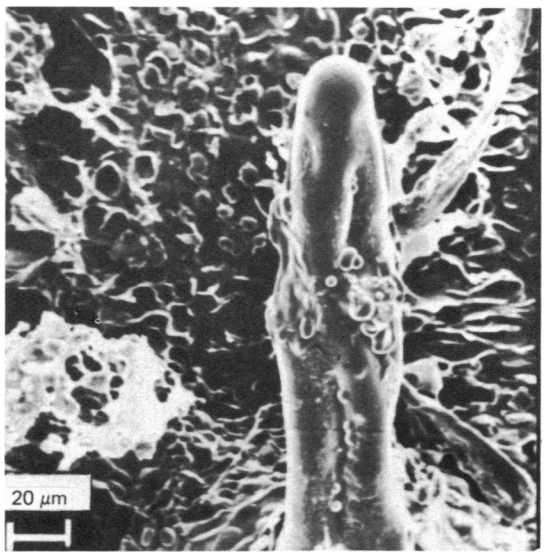

Fig. 3. A methyl methacrylate cast of a metaphyseal vascular complex highlighting the 'looping back' of these vessels and the increased capacity of the venous return. Sections were critical point dried and coated with gold palladium. × 350.

branches which ascended directly and perpendicularly, with virtually no anastomosis for the last 2–5 mm (Fig. 2). At its termination, each arteriolar vessel divided into two to four sprouts which looped back upon themselves (Fig. 2), and at the same time their calibre increased two to two and a half times. These venous sinusoids virtually ensheathed the ascending arteriole in a tortuous woven pattern (Fig. 2). At a distance of 5 mm distal to the tips of the invading metaphyseal vessels, there were large tortuous and irregularly contoured intercommunicating venous channels (Fig. 2). These channels became more and more frequent with anastomoses filling most of the interlacing tunnels which honeycombed the metaphysis. The venous channels had irregular shapes because their extremely thin walls moulded to the contour of each irregularly shaped tunnel within the metaphysis. Casts of the whole vascular complex at the most proximal point were approximately 30 μm enlarging to about 50 μm in diameter some 0·2 mm from the tip (Fig. 3). The metaphyseal vascular complexes were reasonably frequent and evenly spaced; the range was 0·18–0·25 mm between centres (Fig. 4). At the most proximal tip, the venous sinusoids measured 10–25 μm in diameter, enlarging to 60–75 μm within 0·5 mm from this point.

Numerous small metaphyseal arteries which arose from the periarticular plexus of vessels penetrated the metaphysis directly and anastomosed with the terminal twigs of the nutrient artery (medullary arteries) particularly those which were positioned circumferentially.

The venous channels enwrapping the terminal metaphyseal arteriole (Figs. 2, 5) did not appear to have a basement membrane (Fig. 6). These vessels were lined by extremely attenuated and fenestrated endothelial cells (Figs. 6, 7). Occasionally there were gaps between the cells, particularly at the advancing vascular edge. Frequently, endothelial cells abutted the cartilaginous matrix with no discernible intervening basement membrane and with few accompanying undifferentiated

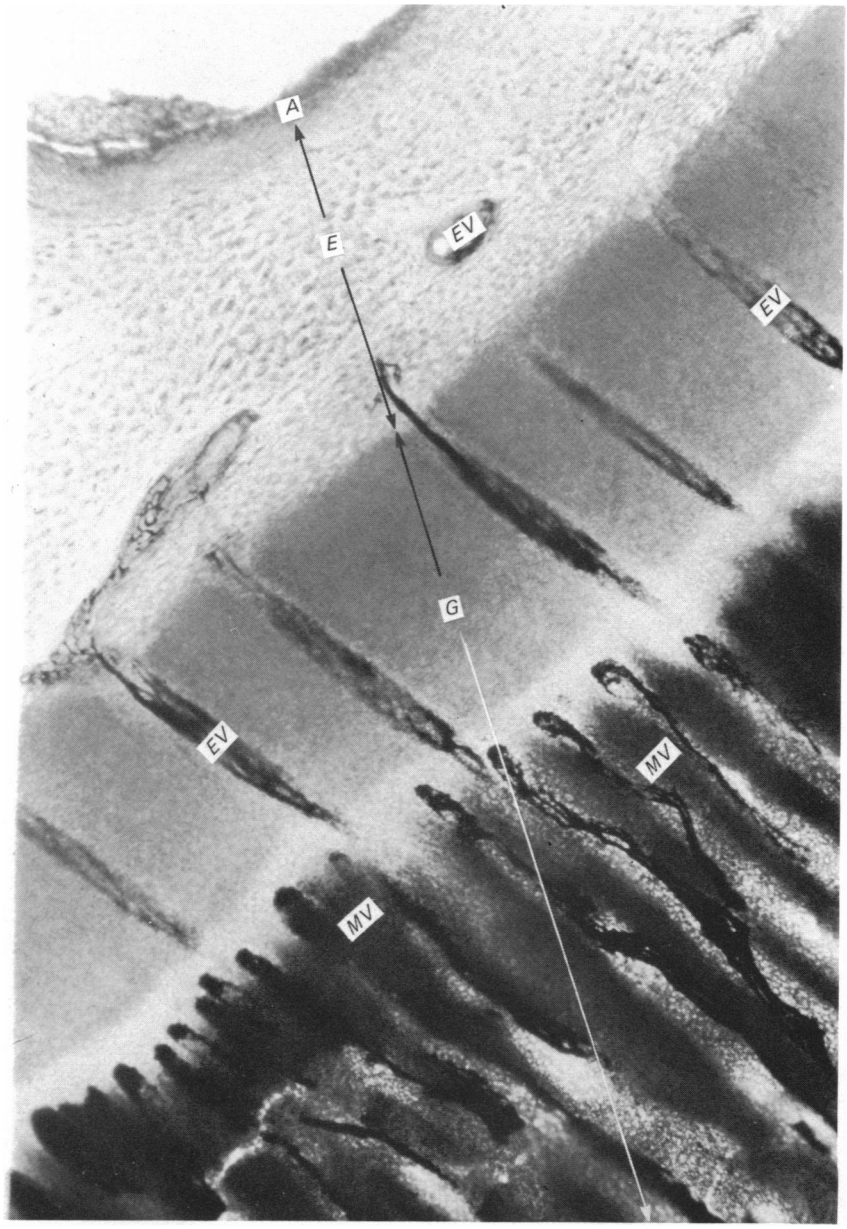

Fig. 4. A glycerine-cleared section of the proximal end of the tibia displaying epiphyseal and metaphyseal blood vessels perfused with carbon via the ischiatic artery. *A*, articular cartilage; *E*, epiphyseal cartilage; *G*, growth plate cartilage; *EV*, epiphyseal blood vessels; *MV*, metaphyseal blood vessels. Unstained approximately 100 μm thick section. × 50.

mesenchymal cells (Fig. 7). Desmosomal junctions as well as interdigitations occurred between endothelial cells situated some 0·6 mm distal to the most proximal portion of the ingrowing vessels (Fig. 8). These wide vessels, 45–90 μm in diameter, had intermittent accompanying pericytes and undifferentiated supporting mesenchymal cells (Fig. 7) as well as occasional endothelial gaps. Because of these features, they were best classified as venous sinusoids.

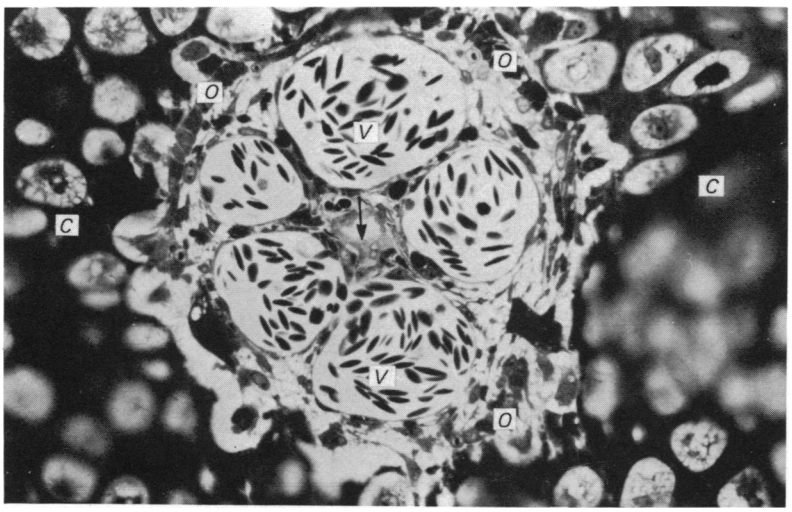

Fig. 5. A cross section of a plexus of metaphyseal vessels which has penetrated the growth plate. There is a centrally positioned small arteriole (arrow) encompassed by thin walled venules (*V*). The vessels exist in a cartilaginous tunnel and at this position between the calcified cartilage (*C*) and the blood vessels there is an intervening ring of preosteoblasts and osteoblasts (*O*). Methylene blue. × 300.

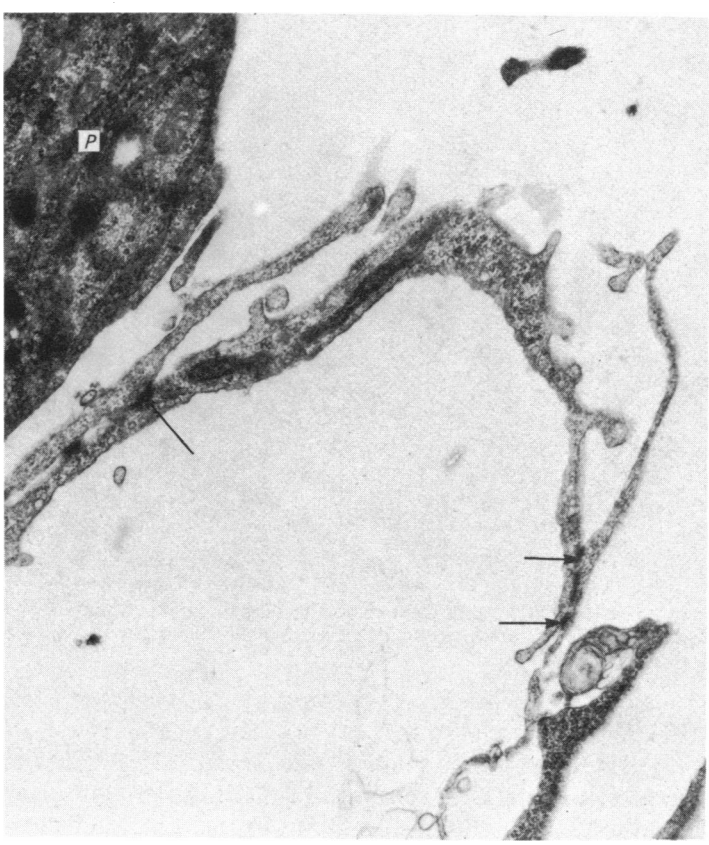

Fig. 6. An advancing edge of a metaphyseal sinusoid-like channel. The endothelial cells are attenuated and attached to each other by desmosome-like junctions (arrows). *P*, pericyte. Uranyl acetate and lead citrate. × 6600.

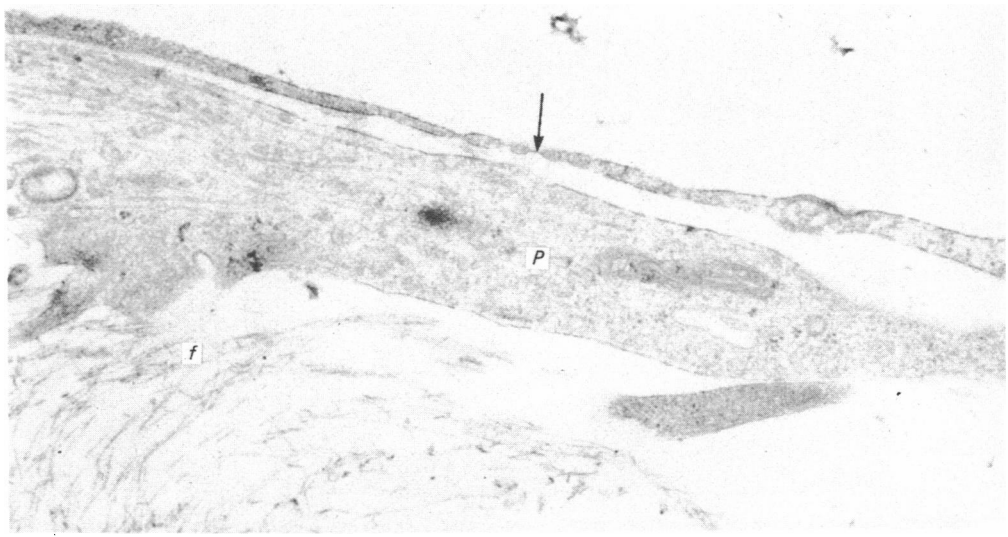

Fig. 7. Attenuated and fenestrated (arrow) endothelial cells line the advancing metaphyseal vascular channels. Pericyte (*P*) and collagen fibrils (*f*) occur in the adjacent cartilaginous matrix. Uranyl acetate and lead citrate. × 30000.

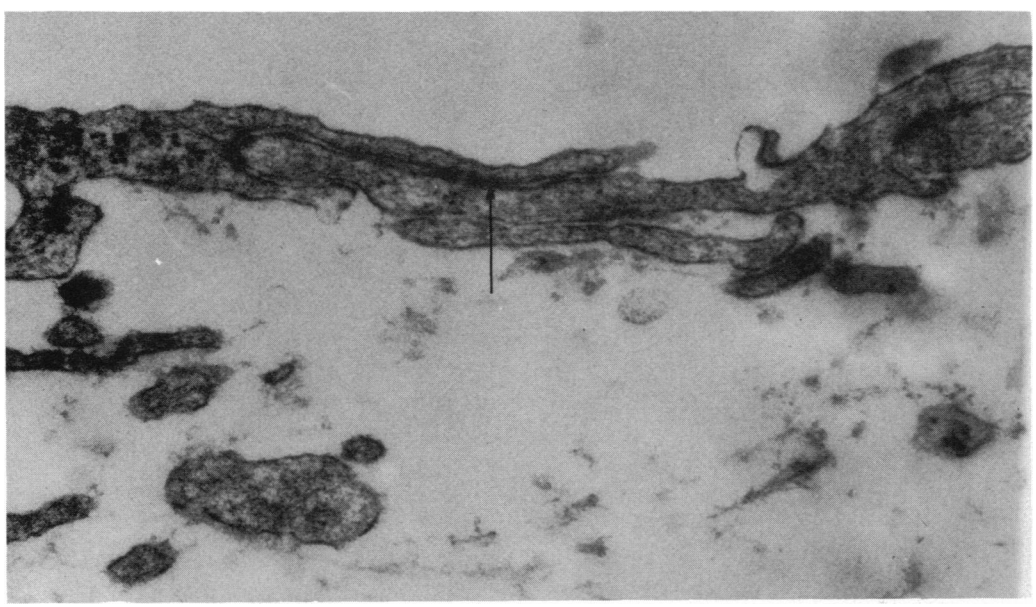

Fig. 8. Desmosome-like junctions (arrow) as well as interdigitations occur not infrequently in metaphyseal vessels 0·6–1·0 mm from the most proximal advancing vascular tip. Uranyl acetate and lead citrate. × 55000.

Epiphyseal vessels

The main arteries to the epiphysis penetrated anteriorly and posteriorly, arching parallel to the growth plate. The epiphyseal side of the growth plate was supplied by arcades of vessels lying in the epiphyseal hyaline cartilage adjacent to the growth plate (Fig. 4). Several terminal arterioles arose from each arcade (Fig. 4), entered

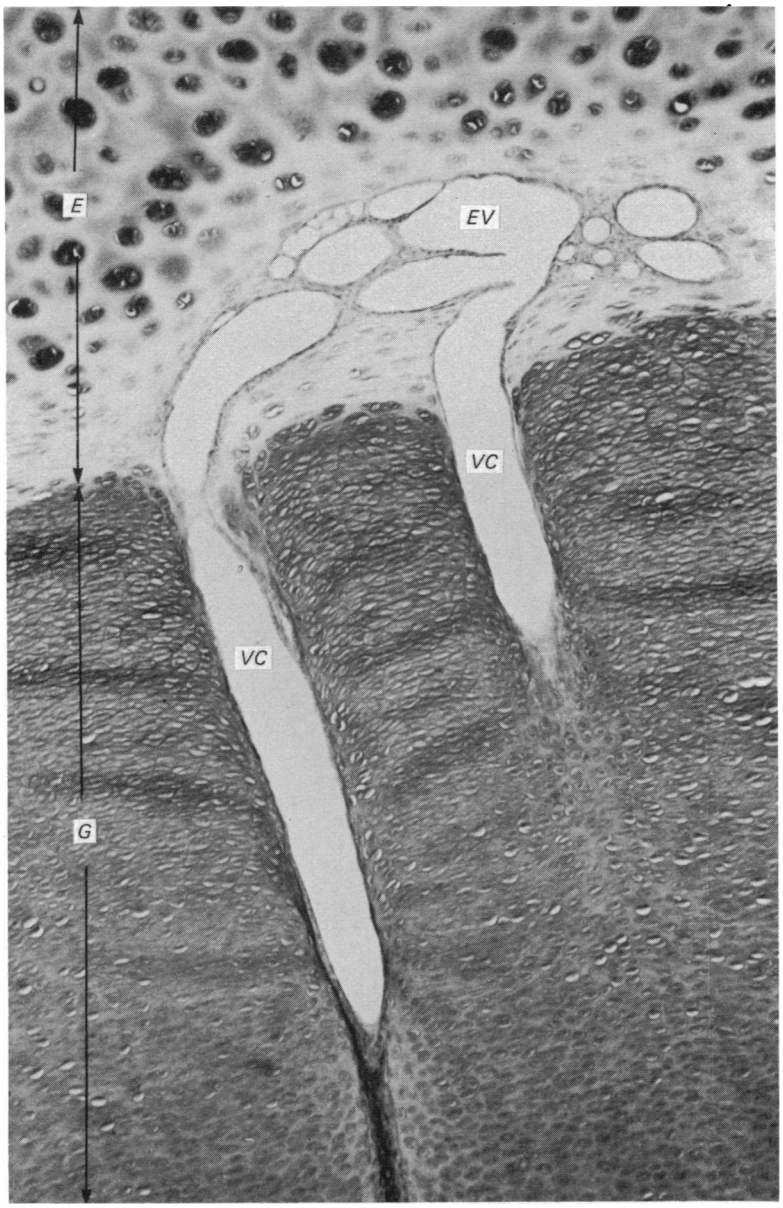

Fig. 9. An epiphyseal vascular complex (*EV*) situated in epiphyseal cartilage (*E*) has given off prominent wide thin walled vascular channels (*VC*) which penetrate the growth plate (*G*) cartilage to about the junction of the zones of proliferating and hypertrophic chondrocytes. Haematoxylin and eosin. × 150.

the proliferative zone of growth plate cartilage vertically and penetrated to about the junction of the proliferative and hypertrophic zones of chondrocytes (Fig. 9). Arterioles descended and terminated in a series of large capillary–venule channels (Fig. 10) which then ascended, ensheathing the arterioles in the growth plate (Figs. 10, 11). As they emerged into the hyaline epiphyseal cartilage, each venular complex formed one to three collecting venules (Fig. 12). These epiphyseal vascular plexuses were about 0·4–0·65 mm apart.

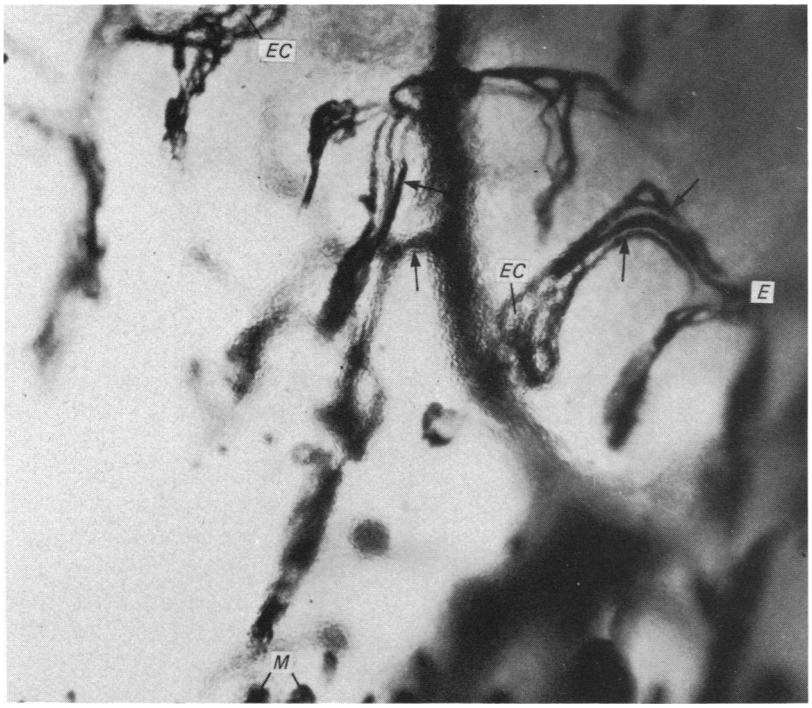

Fig. 10. Fractured preparation of growth plate and epiphyseal cartilage permits visualisation of the terminal ends of both epiphyseal (*E*) and metaphyseal (*M*) vascular complexes when outlined by colloidal carbon. Epiphyseal arteries appear to terminate as large arteriovenular complexes (*EC*). Arrows, collecting veins. Unstained 3 mm block of tissue which had been cleared in glycerine for 10 weeks. × 80.

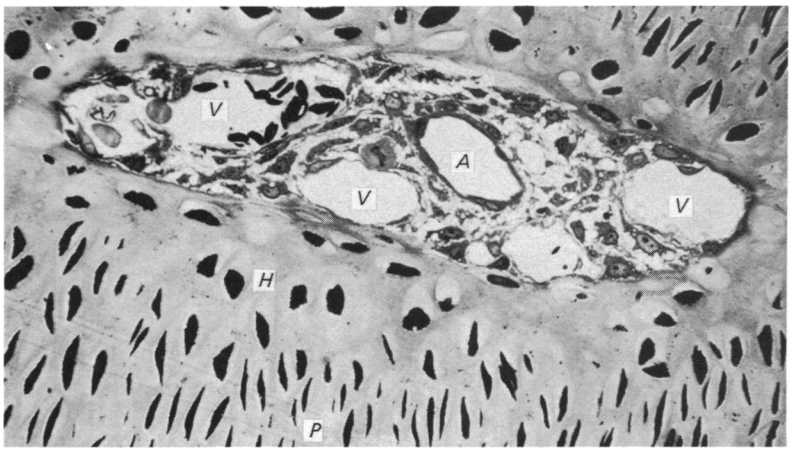

Fig. 11. The epiphyseal vascular complex consists of a centrally positioned arteriole (*A*) flanked by thin walled venular channels (*V*). Loosely packed undifferentiated mesenchymal cells, many with irregular contours and long intertwining cytoplasmic processes, support and enclose these vascular channels. Most of the venular channels have a part of their wall directly adjacent to the growth plate cartilage. There is minor differentiation of proliferating chondrocytes (*P*) to hypertrophic chondrocytes (*H*) adjacent to the vascular tunnels. Toluidine blue; section 1·5 μm thick. × 370.

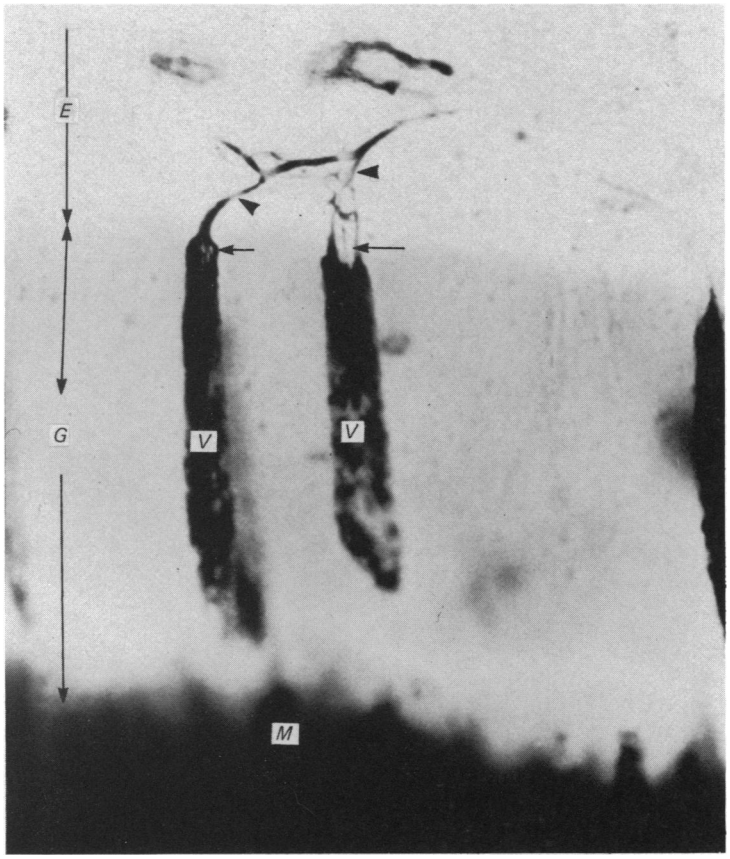

Fig. 12. Carbon perfusion has outlined two epiphyseal vascular complexes (*V*) within the growth plate cartilage (*G*). Collecting venules (arrows) emerge from the growth plate cartilage into epiphyseal cartilage (*E*) and fuse to form muscular venules (arrowheads). *M*, metaphyseal vascular complexes. The unstained section is approximately 200 μm thick and cleared in glycerine. × 80.

Fine structure

The epiphyseal arteriole was roughly at the centre of the vascular complex (Figs. 10, 11). Its luminal diameter was within the range of 15–50 μm, being narrowest distally. The wall consisted of a continuous layer of endothelium and a layer of smooth muscle cells. These two cellular layers were intimately associated (Fig. 13). The arteriole terminated in a capillary–venule complex, some channels having wide lumina (Figs. 9–11). At the distal end, the highly attenuated and fenestrated endothelium of portions of these epiphyseal vessels might directly abut cartilage, with no or minimal mesenchymal cellular accompaniment (Fig. 11). In some regions, endothelial cells interdigitated and had desmosome-like junctions. The venules positioned more proximally had more conspicuous basement membranes and increased numbers of pericytes and fibroblasts, but within the cartilage of the growth plate there was usually no continuous layer of accompanying cells. Diameters of the venular lumina ranged from 15–30 μm. The features of the vessel walls, together with their luminal diameter, indicated that these draining channels were post-capillary venules (Rhodin, 1968).

An occasional chondroclast occurred in epiphyseal cartilaginous tunnels. From

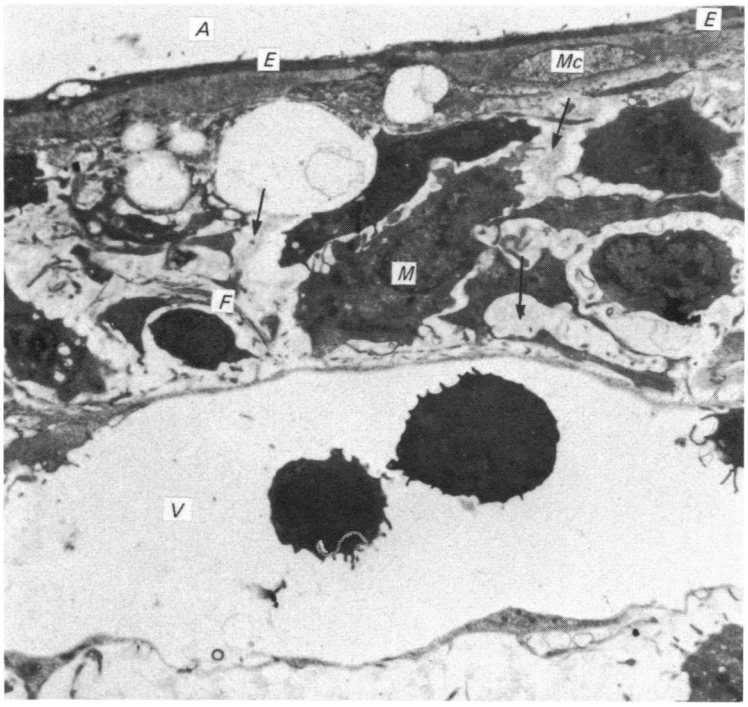

Fig. 13. A longitudinal section of an arteriole (*A*) and accompanying venule (*V*) from an epiphyseal vascular complex. These vessels are separated by undifferentiated mesenchymal cells (*M*) with irregular cytoplasmic borders and long intertwining cellular processes. Between these cells are clumps of floccular material (arrows) and occasional aggregates of collagen fibres (*F*). Endothelial cells (*E*) and myocytes (*Mc*) of the arteriole are in close apposition. Uranyl acetate and lead citrate. × 4400.

the junction of the epiphyseal and growth plate cartilage to roughly half way through the proliferative zone of the growth plate, the vascular complexes had a loose supporting stroma of undifferentiated mesenchymal cells. Thereafter, this supporting stroma diminished in amount and its patchy distribution allowed much of the cartilaginous tunnel to be filled by vascular channels (Fig. 9). Many of these undifferentiated mesenchymal cells had long intertwining processes as well as irregular plasmalemmal contours (Figs. 11, 13). Interspersed between the vascular channels and the undifferentiated mesenchymal cells, and within the epiphyseal cartilaginous tunnels, there was a loosely aggregated, lightly stained, floccular material (Fig. 13) scattered throughout which were occasional small aggregates of collagen fibres.

Some vascular plexuses tapered abruptly to a solitary channel (Figs. 4, 9) almost as if the thin walled vessels were being compressed, perhaps by increased matrix production and/or enlargement of the chondrocytes; both processes were noticeable in the hypertrophic zone.

In summary, epiphyseal vessel complexes were supported by loose undifferentiated mesenchymal tissue within the cartilaginous tunnels. Each complex consisted of an afferent descending arteriole, which terminated in a limited capillary plexus and returned via efferent postcapillary venules which drained into two to four collecting venules in the upper third of the growth plate. The vessels which finally entered the epiphyseal cartilage were muscular venules.

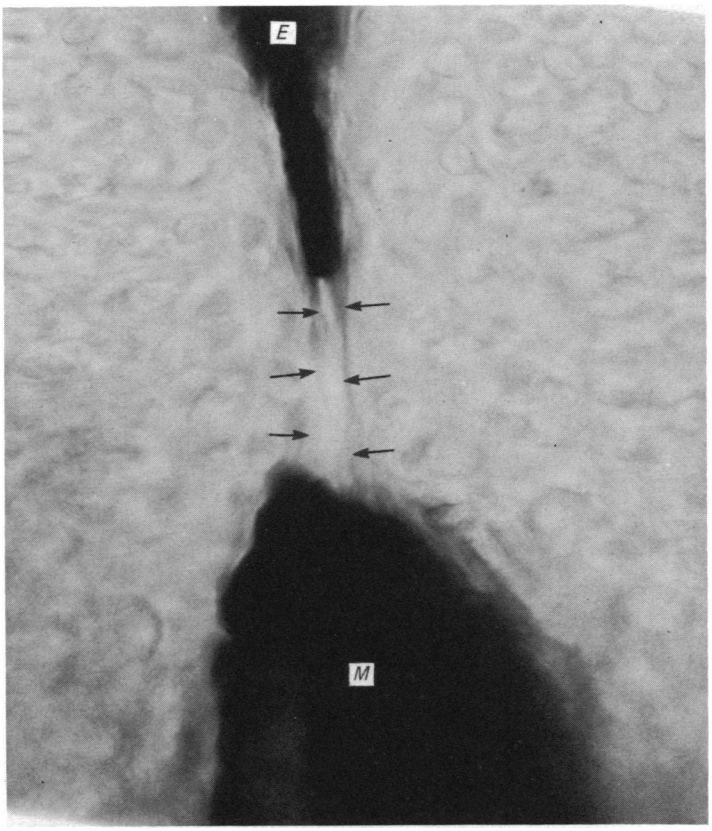

Fig. 14. Colloid carbon has outlined the metaphyseal (*M*) and epiphyseal (*E*) vascular complexes. There is an apparent channel interconnecting these vascular complexes (arrows) but it has not been labelled with carbon. This indicates that intraosseous vascular communication does not exist between these two vascular systems. Unstained 200 μm thick section cleared in glycerine. ×285.

Vessels in the growth plate

The growth plate was supplied by two independent intraosseous vascular channels between which there was a thin avascular zone composed of maturing and hypertrophic chondrocytes. Vessels from the epiphysis and metaphysis anastomosed extraosseously by perichondrial and periosteal communicating arcades.

Longitudinal sections of the growth plate displayed non-patent eosinophilic constricted connections which (having an 'hour-glass' appearance) occurred frequently between the metaphyseal and epiphyseal vascular plexuses particularly when they were close together (Fig. 15). Similarly when horizontal sections were cut serially, from the proliferating zone through to the calcified degenerating hypertrophic chondrocytic zone, the cellular pattern was punctuated by small localised eosinophilic sites surrounded by circumferentially arranged chondrocytes. Furthermore, when examined by transmission electron microscopy, these non-patent connections consisted of necrotic cellular remnants, fine fibrillar and moderately stained floccular material (Fig. 16).

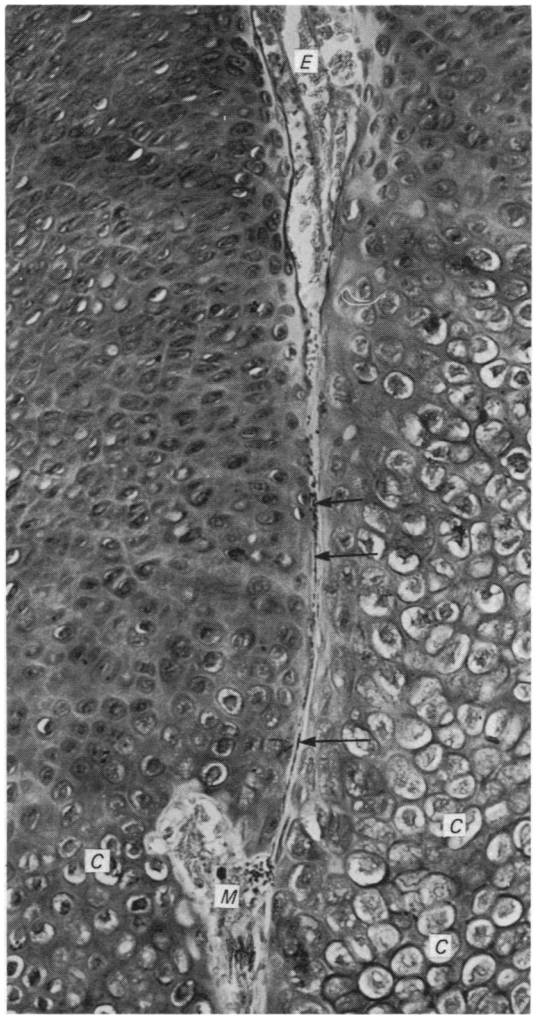

Fig. 15. This section is one of a number of serial sections cut through an apparent communication channel between the metaphyseal (*M*) and epiphyseal (*E*) vascular complexes. There does not appear to be any communication but there are pyknotic nuclear remnants (arrows) present within an eosinophilic streak. *C*, hypertrophic chondrocytes. Haematoxylin and eosin. × 130.

DISCUSSION

This study shows that the intraosseous blood vasculature of the proximal end of the tibia is essentially similar to that described in the ulna of the chicken (Beaumont, 1967) even though there is a zone of secondary ossification in the proximal tibial epiphysis of the bird, in contrast to the ulna. Indeed, Beaumont (1967) postulated that the lack of a secondary osteogenic zone in the epiphysis may be the reason for the differing osseous vascular pattern of the bird when compared to the mammal.

Since the work of Trueta & Morgan (1960) it is generally agreed that in the postnatal mammalian growth plate there is a dual blood supply which does not anastomose across the growth plate. However controversy still exists with regard to the avian growth plate. Wolbach & Hegsted (1952), Westmoreland & Hoekstra (1969) and Nairn (1975), among others, claim that these two sets of blood vessels completely

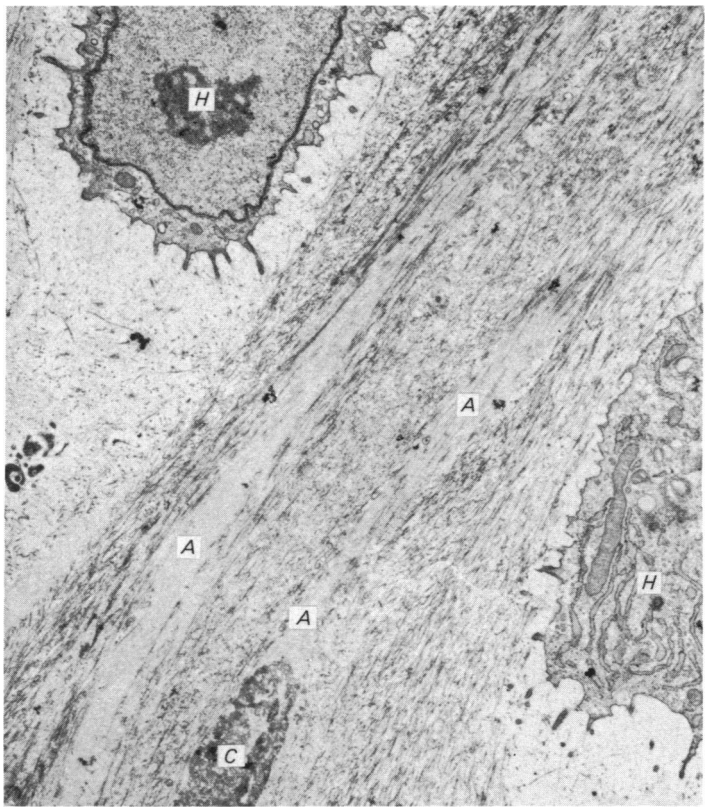

Fig. 16. This region is part of the apparent communication between the metaphyseal and epiphyseal vascular channels. Normal hypertrophic chondrocytes (*H*) flank this zone. The zone consists of amorphous material (*A*), which appears to correspond to the eosinophilic material seen in Fig. 15, not unlike basement membrane collagen. Cellular remnant (*C*) equivalent to the pyknotic cells seen in Fig. 15. Uranyl acetate and lead citrate. ×9500.

penetrate the growth plate and establish vascular communication between the marrow cavity and the epiphysis. On the other hand, Lutfi (1970) investigating maturation of the proximal tibial epiphysis of the bird and its accompanying vasculature states that, within the underlying growth plate, epiphyseal and metaphyseal vessels not only fail to anastomose with each other but do not completely penetrate this cartilaginous zone. Later similar observations were made in the turkey (Wise & Jennings, 1973; Poulos, 1978). Beaumont (1967) observes that although occasional vascular loops from the epiphyseal side pass through the entire thickness of the growth plate there are no actual anastomoses across the growth plate.

The present study, in the chicken, confirms observations by Lutfi (1970) in the chicken as well as by Wise & Jennings (1973) in the turkey. Furthermore it emphasises that the postembryonic bird is similar to the mammal in that it has a dual non-anastomosing blood supply to its growth plates.

The controversy regarding vascular anastomoses across the growth plate has probably arisen from the observation of constricted eosinophilic 'streaks' which connect some ascending and descending cartilaginous tunnels. While the tunnels contain plexuses of blood vessels, light and electron microscopy has definitively

demonstrated that there are no patent blood vessels within the 'communicating streaks', although the cellular debris, fibrillar and amorphous proteinaceous material are suggestive of vascular remnants, conceivably from receding epiphyseal vessels. Wise & Jennings (1973) suggest that some ascending metaphyseal blood vessels and their accompanying cells use this 'line of least resistance' established by the 'retreating' epiphyseal vessels as the bones lengthen. Indeed, although long bones lengthen by interstitial growth of chondrocytes resident within the growth plate, the depth of patent epiphyseal vascular penetration remains constant relative to the local morphology of the chondrocytes. In fact, the avascular zone of the growth plate is occupied by the hypertrophic zone where cells enlarge and cease to palisade. The increase in cellular volume was the reason advanced by Wise & Jennings (1973) for compression and collapse of epiphyseal blood vessels in this zone. However in the authors' opinion, the increase in cellular volume would be only a minor component in the forces causing vascular compression when compared to the effect of the substantial matrix production in this cartilaginous layer.

The measurements of the spacing of blood vessels which penetrate the growth plate confirm those previously recorded in 1952 by Wolbach & Hegsted. Furthermore, the observation that metaphyseal vascularity consists of an ascending medullary arteriole terminating in twigs looping back on themselves to form an intertwining plexus of venular sinusoids is in agreement with the speculation of Beaumont (1967) and Poulos (1978) rather than with the views of other workers (Wolbach & Hegsted, 1952; Wise & Jennings, 1973), who suggested that the latter vessels were capillaries.

The confused classification of blood vessels found entering the growth plate from both epiphysis and metaphysis has arisen partly because previous workers did not extend their observations with the electron microscope and perhaps partly because the vascularity of the growth plate is not of major interest. Confusion exists with regard to the number of blood vessels within cartilaginous tunnels. For example, Wise & Jennings (1973) maintain that some cartilaginous tunnels contain only one vessel while other authors (Poulos, 1978; Beaumont, 1967) claim that there are several vessels in each tunnel. However, Beaumont (1967) describes each arteriole branching into a number of capillaries which join a single vein running back along the length of the canal. In contrast, Poulos (1978) considers that the arteriole terminates in four to five branches with a sinusoidal venous return.

In the present study it has been established that venous sinusoids occur in the metaphyseal vessel complexes but not in the epiphyseal vessel complexes. In addition electron microscopical observations have permitted definitive classification (according to the system proposed by Rhodin, 1967, 1968) of the blood vessels in metaphyseal and epiphyseal tunnels. The chronological development of the proximal tibial epiphysis of the chicken including its vasculature has been investigated by Lutfi (1970) and the present observations confirm and extend his light microscopy findings.

In a recent paper Howlett (1980) describes the metaphyseal venous sinusoidal channels as having a very knobbly contour, a result of vascular sprouts filling recently opened and empty chondrocytic lacunae.

Both epiphyseal and metaphyseal efferent vessels have a very attenuated lining endothelium, a necessary feature in relation to the nutrition of the chondrocytes, some of which reside at considerable distances from blood vessels, particularly those in the avascular hypertrophic region of the growth plate. Alternatively, the mech-

anical protection afforded by the cartilage may allow vessels to develop with little need of mechanical strength.

In mammalian epiphyseal vessels, Kalayjian & Cooper (1972) observe frequent endothelial openings while in the present study the only openings are associated with the passage of leucocytes. However, Kalayjian & Cooper were examining cartilaginous ingrowth of vessels in close proximity to endochondral osteogenesis, a possible equivalent to metaphyseal endochandral osteogenesis, whereas the present study reports on vessels deep within the proliferative zone of the growth plate, a feature not seen in mammalian growth plates.

When considering the role played in the pathogenesis of avian osteomyelitis by metaphyseal vessels, it is now possible to state that although multiplying bacteria may use the 'eosinophilic streaks' as a tract to the epiphysis, there is no direct vascular route across the growth plate. In addition, when speculating where the initial clump of bacteria may lodge in the metaphysis to form the 'nidus' of osteomyelitis, it would seem probable that this would occur in the large sinusoidal vessels where there are profuse anastomoses, resulting in turbulence and quiet 'backwaters'. This would be some 3–5 mm distal to the maximum penetration of the metaphyseal vessels, rather than at the end of the arteriolar twig which bends back upon itself.

SUMMARY

The vascular supply to the proximal tibial growth plate of the 7 weeks old chicken is described using various vascular markers. In addition the ultrastructure of metaphyseal and epiphyseal vessels as well as their supporting tissue is reported.

The metaphyscal arterioles terminate in large calibre vessels which have occasional endothelial gaps and no basement membrane or supporting cells, and therefore could be classified as venous sinusoids. In contrast the epiphyseal arteriole terminates in a capillary–venule plexus lined by an attenuated and fenestrated but continuous endothelium.

This paper definitively establishes that communication of epiphyseal and metaphyseal vessels across the avian growth plate does not occur. The eosinophilic streaks which often join these two vascular supplies have been described ultrastructurally and would appear to be remnants of the 'retreating' epiphyseal vessels.

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