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Periosteal Circulation and its Contribution to the Nutrition of Compact Bone

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PERIOSTEAL CIRCULATION AND ITS CONTRIBUTION TO THE NUTRITION OF COMPACT BONE

by

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Each person whose signature appears below certifies that he has read this thesis and that in his opinion it is adequate, in scope and quality, as a thesis for the degree of Master of Arts.

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The author wishes to express appreciation to Dr. Kenneth Arendt for the untiring service he has rendered to me as chairman of my graduate committee, to the other men who have served on this committee for their assistance and their inspiration, and to all members of the Department of Physiology and Biophysics for their continuing moral support.

To my parents, whose encouragement and unselfish generosity have made my education possible, I give my heartfelt love and gratitude.

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SECTION I

INTRODUCTION

Literature Review

The relationship between the periosteum and the compact bone it covers has been a source of controversy in the biological investigations of several centuries. Some have argued that it is merely a limiting membrane as its name suggests (Macewen, 1912; Moore and Corbett, 1914), while others have proposed that the periosteum has nutritional and/or osteogenic properties.

Although French naturalist Duhamel de Monceau (1739) advanced the idea that bone grows from the periosteum "like an exogenous stem rises from the inner layer of the bark," surgeons of the day generally ignored this tissue (Baillie, 1795; Cooper, 1826; Miller, 1845).

In 1763, von Haller suggested that the "origin of the bone is the artery carrying the blood and in it the mineral elements." Although this theory found influential supporters (Hunter, 1835), it fell into disrepute and was subsequently abandoned because of the opposition of Syme (1840), Flourens (1842) and Ollier (1867), who neglected a consideration of the vessels while stressing the osteogenic importance of periosteal tissue cells. It was not until the early twentieth century, and the work of Leriche and Policard (1926), that the importance of the blood was reintroduced.

Keith (1927) suggested that the cells which assume a bone-forming role are derived from endothelium of the capillary system.

Infarction experiments in Foster's lab (1951) supported this hypothesis. A series of studies by Trueta (1950, 1952, 1953a, 1953b, 1953c, 1953d) utilizing microradiographic and photographic techniques, established this concept.

The osteogenic argument appears fairly well resolved in current literature. There is general agreement that the periosteum is composed of two layers: an outer fibrous layer and inner osteogenic layer (Weinmann and Sicher, 1955; Hollinshead, 1960; Gray, 1966). The fact that the inner layer is so adherent to bone has contributed to the historical confusion, since stripping of the periosteum during surgical procedures usually removed only the outermost layer. More precise experiments (Foster et al, 1951; Trueta and Cavadias, 1955) resulted in bone infarctions, emphasizing the determinant role of the periosteum.

The osteogenic cells of the inner osteoblastic layer are direct descendants of cells of the inner layer of bone perichondrium that develop in cartilage (Ham, 1965). The organizer is the "osteogenetic vessel from which springs the syncytial frame of cells" on which bone is established. The sequence of cellular phylogeny consists of the endothelial cell, intermediate cell, osteoblast, osteocyte and osteoclast (Trueta, 1963).

Although certain aspects of periosteal function remain to be elucidated, its osteogenic importance in times of injury is well documented. Following suppression of periosteal supply there is a distinct paucity of new osseous tissue (Kistler, 1934a, 1934b) and a definite alteration of diaphyseal structure (Marneffe, 1952).

A series of radiographic studies conducted by Brookes (1957, 1960a, 1960b, 1963) confirmed the establishment of centripetal blood flow from the periosteum following injury and suggested that this flow might be a primary factor in the survival of ischemic bone. Even though some have questioned this phenomenon (Laing, 1953), most investigators agree that disturbance of marrow blood flow results in a "take over" by the periosteal circulation (Nelson et al, 1960; Larsen et al, 1961). Mtsumoto (1965) found that the periosteum is capable of full centripetal supply of cortical bone from the surface to the marrow cavity when marrow flow is disturbed.

Still unresolved is the question of periosteal contribution to the nutrition of intact compact bone. In 1691, Clopton Havers wrote of "a few nutrient arteries" entering the bone in localized areas, and then giving rise to "vast numbers of veins" leaving the bone surface. By 1743, Hunter had specifically described periosteal arteries as anastomosing to form two circular vessel series around the bone. Von Haller (1763) confirmed that "without any doubt" vessels were seen in the periosteum "by the injection of coloured fluids."

The first detailed account of cortical blood supply was presented in 1904, when Lexer and coworkers made radiographic records of post-mortem specimens injected with a mercury-turpentine emulsion. These studies suggested four sources of arterial blood in long bones: periosteum, diaphysis, metaphysis, and epiphysis. Other observers disagreed. Foote (1921) detected only two sources: periosteal and medullary. Johnson (1927) and Anseroff (1934) found evidence of three systems of nutrient arteries: periosteal, nutrient and epiphyseal. The classification

of three sources persists in contemporary literature (Laing, 1953; Peterson and Kelly, 1961; Abramson, 1962; and Lanzi, 1965). Brookes (1965) recently reaffirmed this by micro-radiographic preparations. It is, then, "taken for granted" that normal compact bone possesses "in some degree" a periosteal arterial blood supply (Brookes, 1960b). Opinions vary on the degree of supply.

Periosteal vessels actually penetrating cortical bone were described by Langer in 1876. Other early observers were Testut (1880), Foote (1921) and Kolodny (1923). A systematic study of bone circulation conducted by Lexer (1903, 1904, 1915, 1922) led to the conclusion that the periosteal vasculature is indeed important to the normal nutrition of compact bone. Anatomical findings established by Lexer were accepted by colleagues as unchallenged facts. Even today, his illustrations of the major vessels are considered excellent (Morgan, 1959).

India ink injections in post-mortem dogs examined by Johnson (1927) and Kistler's (1934a, 1934b) charcoal infarction studies in the rabbit femur supported Lexer's findings. This classical concept of periosteal supply to the cortex was diagrammed by Testut and Latarjet in 1948. Experimental variations utilizing the stripping of periosteum or suppression of other nutrient sources supported this concept of periosteal importance (Caiero and Mainetti, 1932; Rholich, 1939; Foster et al, 1951). These authors similarly concluded that the outer third of the cortex is nourished primarily by periosteal circulation.

Marneffe (1951) believed that the entire compact bone of the lower diaphysis is supplied primarily by periosteal blood, a theory later endorsed by Tilling (1958) and Peterson and Kelly (1961).

Haliburton and associates (1958) proposed that the talus vascular supply is of periosteal origin. Laing (1956) and Lewis (1956) also stressed the importance of periosteal supply to compactum integrity.

Investigations of osseous circulation have been pursued for some time at Nuffield Orthopaedic Centre, Oxford, under the leadership of Professor Joseph Trueta. After perfusion of over 2000 mammalian bones, Trueta (1953) concluded that periosteal circulation is difficult to estimate correctly since most of the superficial cortical vessels remain empty of perfusion medium in the majority of specimens. This leaves any judgment based solely on this method open to question (i.e., Hyrtl, 1864; Johnson, 1927; Macnab, 1958; Brookes and Harrison, 1957; Brookes et al, 1961). Other methods of observation utilized in his laboratory have included ultra-violet examinations, electron microscopic photographs and celloidin section. All methods have shown vessels entering the cortex from the periosteum, indicating a periosteal arterial supply. In some preparations two vessels were seen in haversian canals, supporting the historical view that blood can flow in both directions and not only centrifugally.

Conflicting evidence has appeared repeatedly in the literature The belief that periosteal vessels are merely capillary connections between cortex and periosteum, capable of collateral centripetal reinforcement to the compactum vascular net upon injury, but serving simply as centrifugal venous drainage for normal tissue, is supported by several authors.

As early as 1922, Drinker and his coworkers raised a dissenting voice, stating that periosteal circulation was of no importance to the

integrity of the compactum. Gregoire and Carriere (1921) incorrectly denied the very existence of anastomoses between periosteum and medullary vessels. The theory of periosteal unimportance is best summarized by Murray Brookes of Guy's Hospital Medical School, London:

> "Investigations into the problems of vascular organization and haemodynamics of long bones (Brookes, 1958a, 1958b; McAuley, 1958) lend support to the concept formulated by Brookes and Harrison (1957) of a medullary arterial system responsible for the supply of blood to both the sinusoids of the marrow and the capillaries of compact bone. In normal conditions it is held that the periosteal blood vessels play no part in the arterialization of underlying bone cortex, but for blood which has flowed through the vascular canals of the compactum a venous drainage pathway is available in the capillaries of the periosteum." (Brookes, 1960b)

> "In other words there is a single circulatory system in compact bone and the direction of the blood flow is centrifugal." (Brookes, 1963)

The conclusions expressed above were the result of numerous gross dissections, micro-radiological preparations and histological examinations. Animals were injected intravascularly immediately post-mortem with Micropaque, a radiopaque suspension of barium sulphate, and microradiographed on maximum resolution plates. Following fixation, individual bones were examined either whole or in hand-cut slices. Twenty-micron sections were studied histologically. It was discovered that the barium sulphate suspension failed to enter the cortex from the periosteum, but found easy access from the medulla. This led to the assumption that the sole function of periosteal vessels is the nourishment of the osteogenic membrane (Brookes, 1958a).

Brookes (1963) suggests that Trueta's experiments of 1963 indicate a centripetal flow only because experimental medullary suppression

had established a pathological basis for collateral activation. He contends that the capillary nature of both periosteal and cortical vasculature, and the singular vascular net of the cortex support the premise of centrifugal flow.

Objective

Although the writer agrees with Claude Bernard's dictum (1855) that anatomy is part of the basic knowledge which the physiologist must master, one must recognize the inadequacy of histological sections as the sole basis for interpretation of the dynamic physiology of living blood vessels.

To date, most studies have been conducted on post-mortem tissues. The procedures employed in some investigations have limited the interpretation of results since they alter the normal physiological pattern of blood flow (e.g., charcoal embolism, Kistler, 1934a, 1934b; marrow destruction, Marneffe, 1951; periosteal stripping, Foster et al, 1951). In addition, technical difficulties encountered in contemporary anatomical studies have necessitated the representation of photographic subjects by drawings or diagrams.

In this investigation, all data were derived from observations and perfusion of living periosteal and cortical vasculature. By maintaining physiological conditions, it was the author's intent to answer questions raised by post-mortem studies concerning the role of periosteal circulation and its possible contribution to the nutrition of compact bone.

SECTION II

MATERIALS AND METHODS

Vascular patterns of the antero-medial surface of young adult rabbit tibiofibulae were studied. Animals of both sexes, with an average weight of 1.8 kg, were used. Surgical anesthesia was induced by injecting pentobarbital sodium (Nembutal, 75 mg/kg body weight) into the marginal vein of an ear previously dilated by application of moist heat.

Respiration was maintained by tracheal cannulation in conjunction with a Phipps and Bird positive pressure respirator. Rarely encountered anesthetic cardiac arrest was reversed in most instances by external massage and 0.5 cc of a 3:1 solution of Metrazol (0.1 gm pentylenetratrazol) and epinephrine (1:1000 Adrenalin Chloride) administered intravenously.

Blood loss in surgical procedures was minimized by the use of a Wappler Cold Cautery Scalpel, model C-450. The tibialis anterior muscle was retracted and wrapped in a gauze pad moistened by Ringer's mammalian solution to eliminate twitching produced by dehydration. The periosteal circulation was protected from dessication by topical application of mineral oil.

The area chosen for study was bound cephalically by a point approximately 1.0 cm below the knee and caudally by a point 1.5 cm above the ankle. The leg was supported by an ankle clamp and the body rotated so that the relatively flat anterior surface of the tibiofibula was in

FIGURE 1

Anterior exposure of rabbit tibiofibula.

FIGURE 2

Perfusion apparatus.
C: perfusion cannula entering anterior tibial artery
T: transducer employed to measure pressure

FIGURE 1

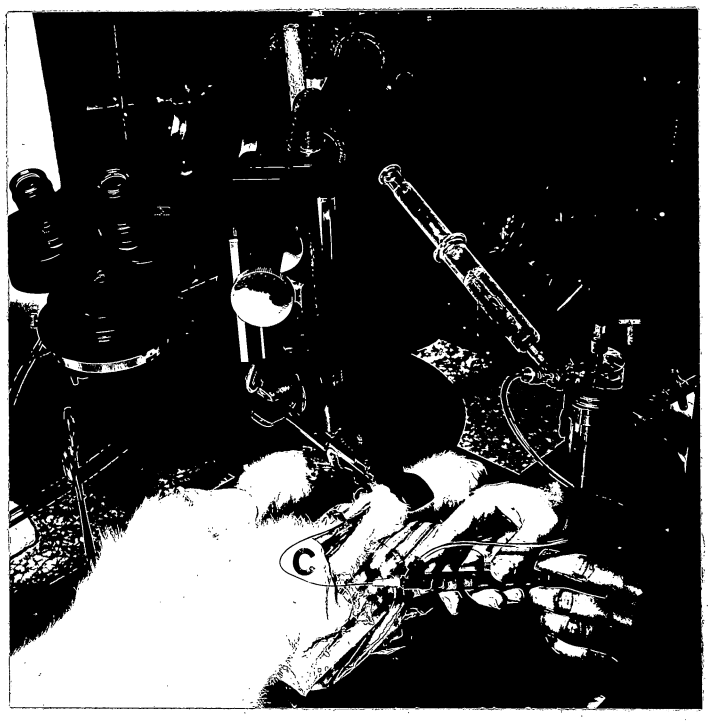


FIGURE 2

a horizontal plane (Fig. 1). Vascular phenomena were observed with a stereoscopic microscope.

In all preparations, general vascular distribution was noted, with special attention to characteristic vascular units designated as triads. A triad consists of a single distributing arteriole extending over the periosteal surface from either the lateral or medial periphery and a flanking pair of veins. Subperiosteal branches of the distributing arterioles were identified by their curving descent into the longitudinal canals of the cortical bone surface, while branches directly penetrating the compactum formed a right angle turn and disappeared from view. The periosteal distributing arterioles and collecting venules were selectively cauterized with a tungsten electrode of 305 μ diameter, and the effects on adjacent vascular beds noted. Subperiosteal flow was observed following isolation of a triad by three-sided cauterization, the arteriolar supply remaining intact.

In one series of examinations the periosteum was stripped to permit study of density and orientation of the surface cortical vasculature. Special care was exercised to fully remove the adherent inner osteogenic layer. Oozing of blood from the bone surface was regarded as indicative of centrifugal flow of medullary origin.

Presence of vascular smooth muscle was determined indirectly by topical application of epinephrine (1:1,000 or 1:10,000 Adrenalin Chloride) from a modified 25 gauge needle blunted and beveled to emit a 0.002 cc drop. It was possible with this technique to identify the distributing arterioles and surface branches as well as subperiosteal metarterioles which were terminal endings of long extensions from surface

arteriolar branches. A given vessel was classified as an arteriole only when constriction was initiated at the site of epinephrine application. Diameter changes of representative vessels were measured with a calibrated vernier ocular micrometer.

Ramifications of the periosteal vasculature were studied directly by localized perfusion of the anterior tibial artery in regions proximal to periosteal triads. The perfusion medium used was Batson's #17 Anatomical Corrosion Compound (Polysciences, Inc., Rydal, Pa.). Adequate reaction time was afforded by a ratio of 5 cc monomer base solution to 0.5 cc catalyst to 1 drop promoter. Sufficient Batson's blue dye was added to produce a distinct color. Injections were made with a 2 cc syringe-25 gauge needle unit connected to PE 10 Intramedic polyethylene tubing. The cannula was inserted following central ligation of the artery to prevent retrograde perfusion. The artery was also ligated at the lower metaphysis in selected specimens to prevent perfusion of the talus and thus permit observation of restricted periosteal distribution. Perfusion pressures were recorded by means of a Grass model 5 polygraph equipped with a Statham P23AC strain gauge pressure transducer and maintained within the physiological limits of the anterior tibial artery (mean pressure of 35 mm Hg), (Fig. 2). Intact and sagitally sectioned perfused bone specimens were microscopically examined and photographed.

Variation in quantitative data is reported as $\pm$ 1 standard deviation.

SECTION III

RESULTS

Fifty-eight tibiofibulae were examined in this study. Classification and enumeration data recorded from twelve specimens appear representative of the pattern noted in all bones. The central diaphysis consistently evidenced the preponderance of periosteal vessels, while the proximal third of the exposed area exhibited a sparse vascular distribution. By individual count, the average exposed periosteal surface area of 2.25 cm^2 revealed a profuse blood supply. Mean values for distributing arterioles were 8 $\pm$ 2 triads, several of which were ladder-like shunts across the breadth of the bone; 11 $\pm$ 4 arterioles not of a triad pattern; 35 $\pm$ 14 branches within the surface plane; 80 $\pm$ 12 branches dipping into the longitudinally-oriented vessels; and 17 $\pm$ 4 branches directly penetrating the compactum.

Two distinct planes of vasculature were evident. The distributing arterioles and collecting venules were on a plane superior to the longitudinal subperiosteal vessels. Sagittal sections showed membranous tissue approximately 0.5 mm separating the two horizontal planes. Upon retraction of the periosteum, subperiosteal vessels were seen to lie in grooves within the surface compactum. Retraction precipitated broadcast stasis in these surface channels. Fourteen bones were examined for evidence of medullary blood supply as demonstrated by active bleeding from the cortex following periosteal stripping. Oozing sites within the 2.0

cm^2 stripped area were scant, ranging in number from 0 to 19 immediately upon retraction, and 0 to 15 within 60 seconds.

Selective cautery was utilized to determine the effects of induced stasis on general periosteal blood flow. Twenty-nine pair of arterioles and venules were individually cauterized. Results were consistent in each series. Initially, a distributing arteriole was cauterized at its origin. Immediate stasis resulted in over half the adjacent vasculature. Venous return became sluggish. Subsequent cauterization of the collecting venules of the same triad resulted in cessation of flow in approximately 90% of contiguous longitudinal vessels. Cauterization of a single venule evinced change in no vessel except that cauterized. The final series of cauterizations produced isolation of 35 triads. Tissue surrounding each triad was cauterized on all sides except that with the supplying arteriole. In each case the flow of anastomosing and subperiosteal vessels within the isolated area continued essentially unabated. Perfusion of a single triad resulted in a localized cast, providing supportive evidence of segmental distribution.

Ligation of the anterior tibial artery resulted in broadcast stasis of the longitudinal vessels. An occasional short vessel, appearing to be a venous segment arising from and returning to the compactum, continued to conduct flowing blood.

In 14 bones treated, topically applied epinephrine consistently produced constriction of all triad arterioles, single distributing arterioles and surface metarteriolar branches. Adjoining longitudinal vessels went into stasis, while blood flow in adjacent venules became sluggish. Distinct constriction of smaller branches descending directly to the

subperiosteal surface or deep compactum was difficult to assess, but in nine specimens, epinephrine initiated definite constriction in 22 branches to the subperiosteum and six branches to the deep compactum. Measured diameters of 18 representative vessels were between 20 and 55 μ, all exceeding Ruch and Patton's (1965) 5 to 20 μ range for capillary diameter. The mean diameter of 8 branches representative of those extending to subperiosteum and deep compactum was 25.8 ± 3.5 μ. Distributing arterioles had a mean diameter of 40.9 ± 10.7 μ. Vaso-constriction resulted in diameter decrements ranging from 3.75 u to 37.5 μ. Mean values were 18.1 ± 3.4 μ diameter loss in distributing vessels and 5.3 ± 1.8 μ loss in descending branches.

Because the molecular components of Batson's #17 corrosion compound are well within the diameter limits for capillary transport, maximum perfusion of the vascular net presented minimal problems. Arrival of perfusate at the origin of a triad arteriole was immediately followed by rapid filling of surface and subtending vasculature, and subsequent perfusion of the flanking venules (Fig. 3). Microscopic observations of the periosteal vascular network during this perfusion revealed movement of the solution directly into the longitudinal channels of the surface compactum (Figs. 4 and 5). Disappearance of the medium into vessels perpendicular to the bone surface implied direct descent into the deep compactum. Sagittal sections showing perfusate-filled transcortical vascular elements corroborated this observation. When the perfusion was restricted to a given area, only the compactum subtending the perfused periosteum revealed dye-filled vessels (Fig. 6). In all cases the cortex opposite the perfused area was free of perfusate (Fig. 7). Perfusate-

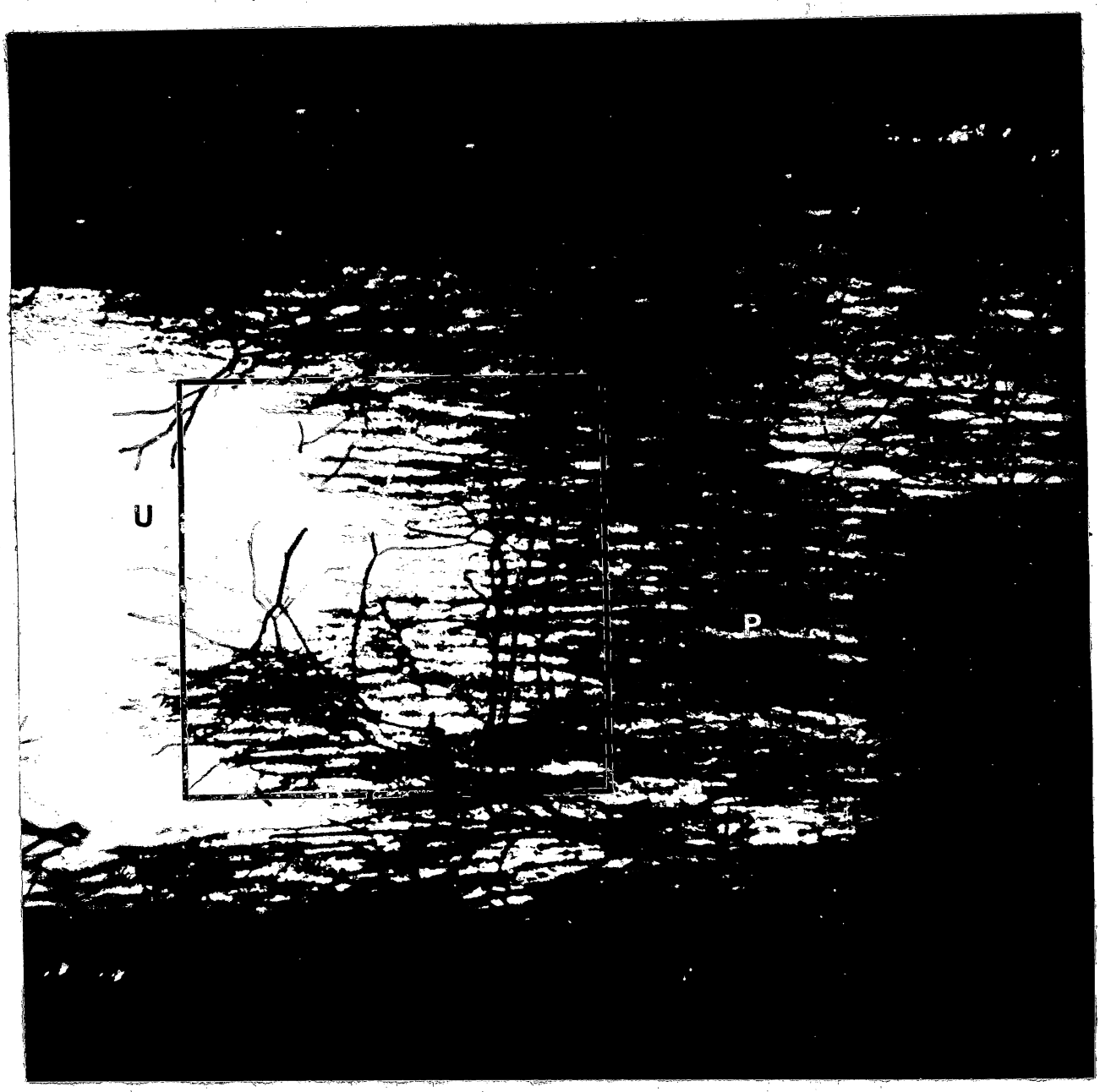


FIGURE 3 (A)

Periosteal triad and attendant vasculature.
P: perfused subperiosteal vasculature
U: unperfused subperiosteal vasculature

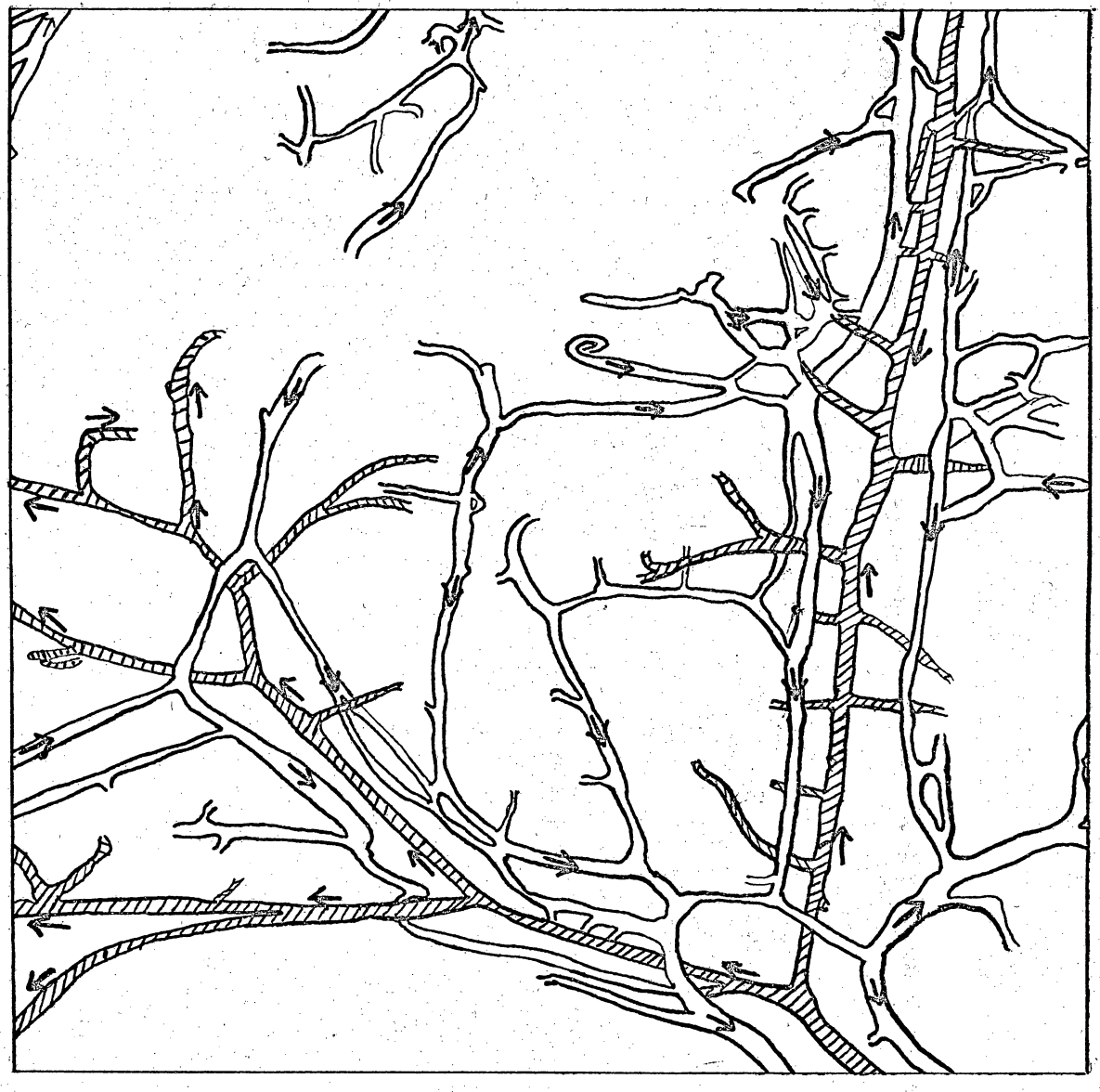
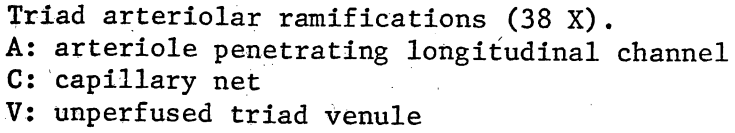


FIGURE 3 (B)

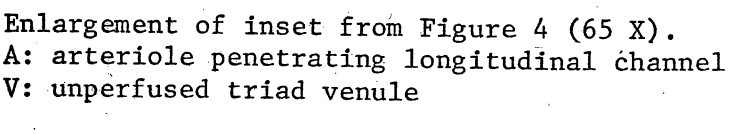
Diagrammatic flow pattern of periosteal triad and attendant vasculature.

FIGURE 4



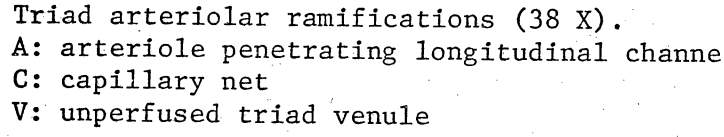
Triad arteriolar ramifications (38 X).
A: arteriole penetrating longitudinal channel
C: capillary net
V: unperfused triad venule

FIGURE 5



Enlargement of inset from Figure 4 (65 X).
A: arteriole penetrating longitudinal channel
V: unperfused triad venule

FIGURE 4



Triad arteriolar ramifications (38 X).
A: arteriole penetrating longitudinal channel
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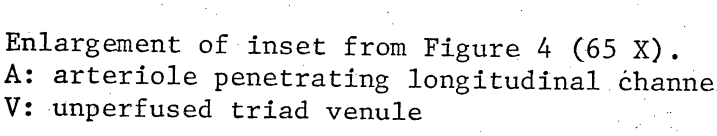
FIGURE 5



Enlargement of inset from Figure 4 (65 X).
A: arteriole penetrating longitudinal channel
V: unperfused triad venule

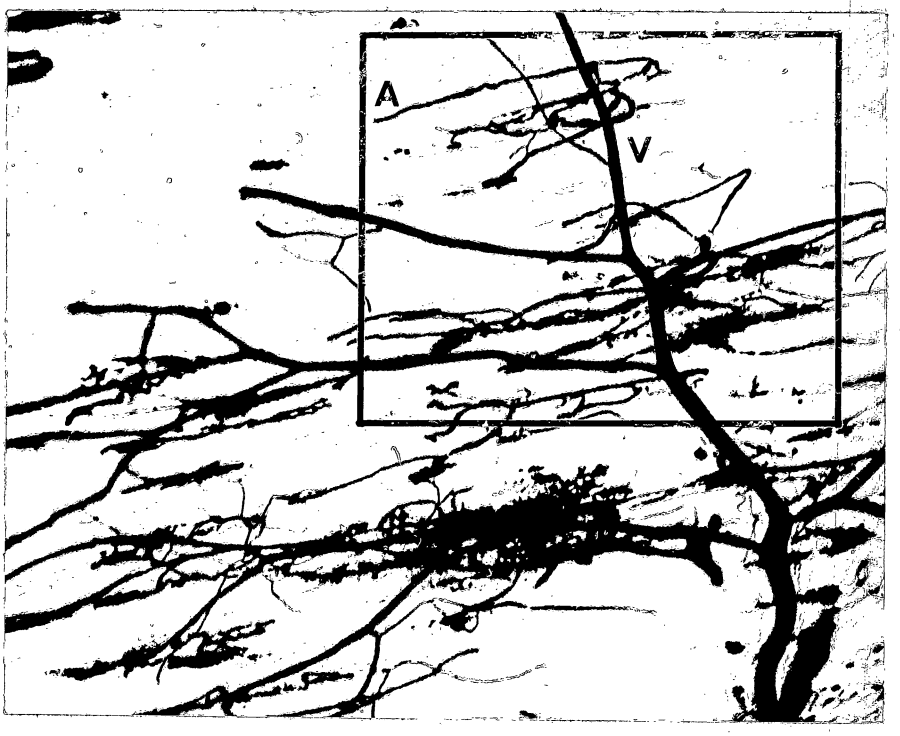


FIGURE 4

FIGURE 5

FIGURE 6

Sagittal view of perfused transcortical vessels (62 X).
M: medullary canal
P: periosteal perfusion surface
V: perfused venule

FIGURE 7

Sagittal section of perfused bone shown in Figure 6 (13 X).
C: unperfused cortex
M: medullary canal
P: periosteal perfusion site

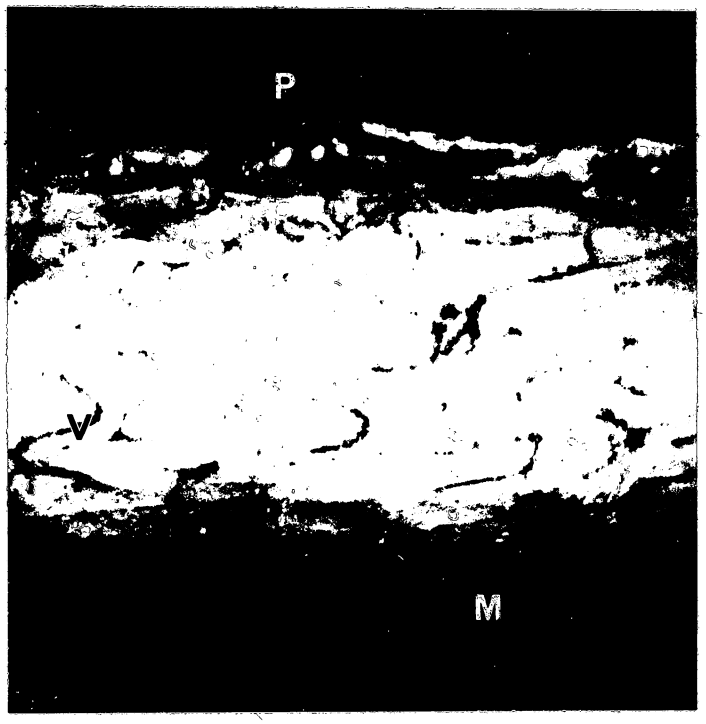


FIGURE 6

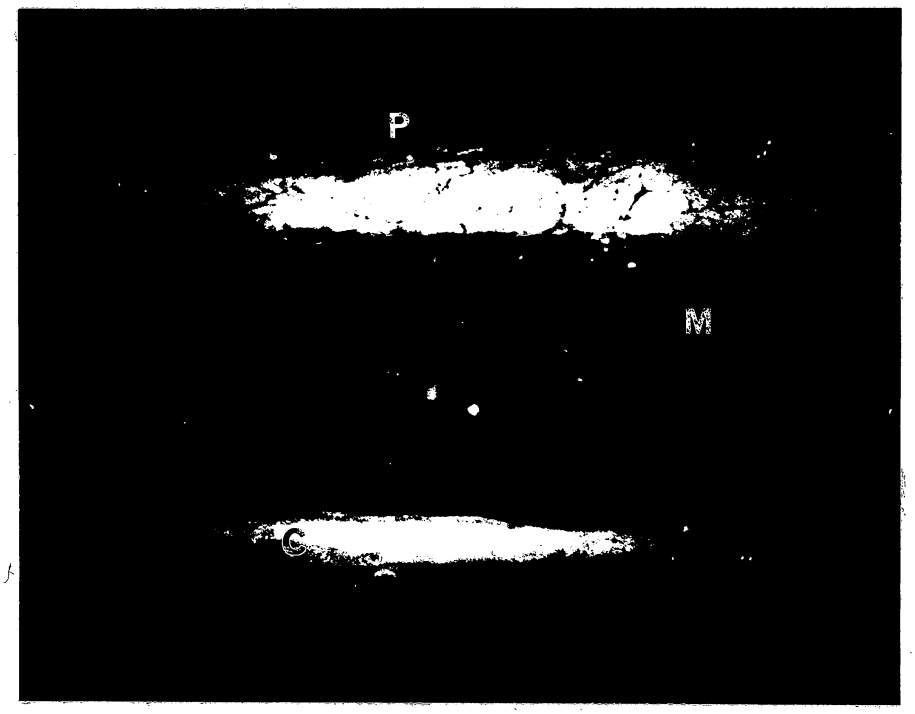


FIGURE 7

FIGURE 8

External view of perfused surface compactum, as seen following periosteal stripping (6 X).
C: unperfused cortex
D: dense perfusion of subperiosteal vessels

FIGURE 9

Endosteal view of transcortical perfusion.
Same bone as Figure 8 (6 X).
C: unperfused cortex
M: medullary canal showing endosteal projections of periosteal-injected perfusate

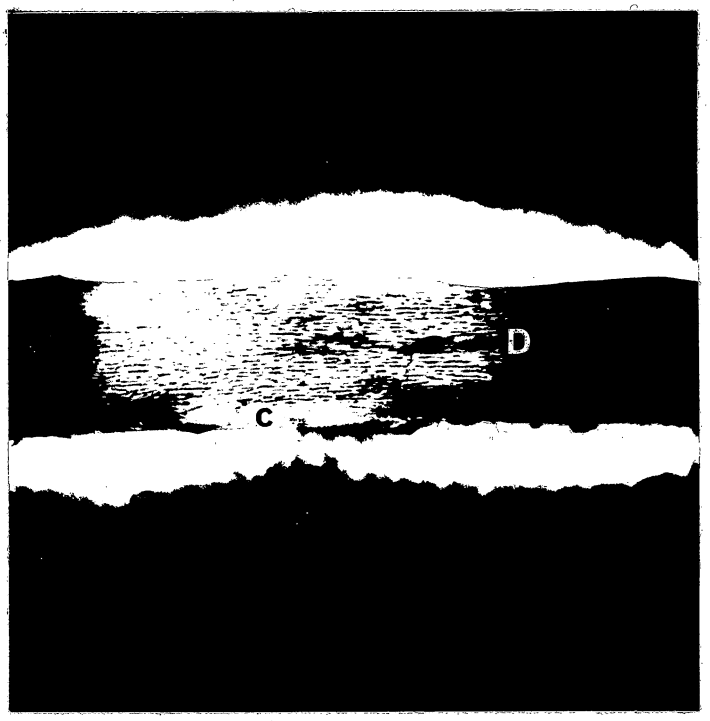


FIGURE 8

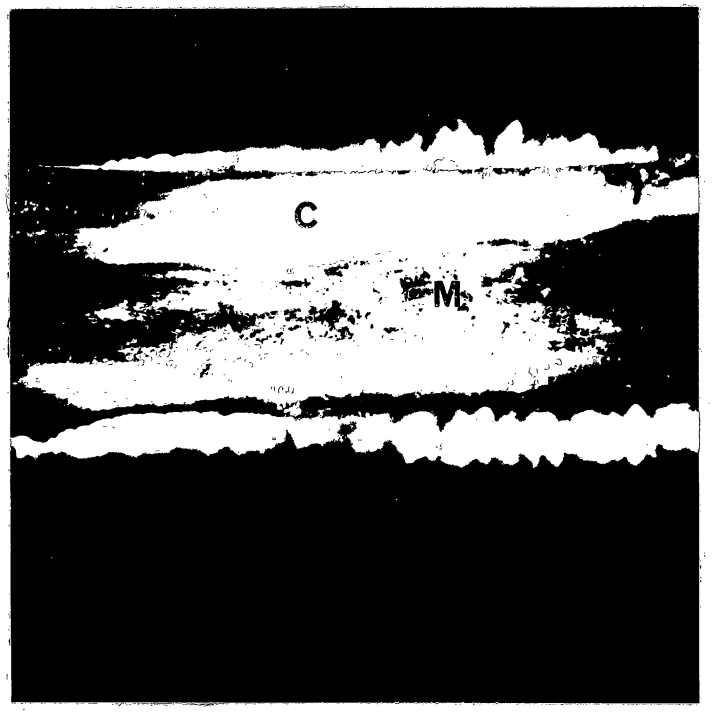


FIGURE 9

filled subperiosteal vessels photographed following removal of the periosteum are shown in Figure 8. Endosteal projections of these transcortical vessels are viewed from the medullary canal in Figure 9.

Visual observations of normal blood flow verified the preceding experimental results. Introduction of dilute solutions of India ink permitted observation of movements of individual dye particles. Some were seen to flow down the perpendicular vessels and disappear from view, while others were carried by a normal flow pattern into the subperiosteal vessels.

In two specimens, the anterior tibial artery was perfused just superior to the ankle. Sectioning of the bones revealed distribution of the dye throughout both cortical and medullary vasculature of the entire talus region.

SECTION IV

DISCUSSION

The importance of periosteal circulation as a collateral source to the compactum of long bone is well established, but conflicting data have been presented concerning its normal contribution to intact bone. Harmony is also lacking concerning the abundance and general function of periosteal vessels. A copious vascular pattern was suggested by Foote in 1921, and has been supported by Tilling (1958) and Nelson (1960). In contrast, Lamas et al (1946) spoke of a diaphyseal periosteum as having "almost no blood vessels." Francassi (1941) and Rhinelander (1965) agree that the mature animal possesses a "meager periosteal blood supply."

Counts from living animals, reported herein, suggest a consistently ample vascularization in both periosteum and superficial cortex, with an average of 150 $\pm$ 20 arteriolar vessels per a 2.5 cm^2 area. Periosteal perfusions show dense capillary and venous networks. Sagittal sections of perfused specimens reveal profuse vascularization of the compactum.

Classification of periosteal circulation as a capillary network has been supported by many authors (Macnab, 1958; Morgan, 1959; Peterson and Kelly, 1961). Brookes and coworkers (1961) contend that no periosteal arteries penetrate the cortex. Nelson (1960) concurred in finding that "by and large," periosteal and cortical vessels lack musculature. Hert (1960) and Brookes (1963) found continuous connections

among cortex, marrow sinusoids and periosteum, but only at a capillary level. In contrast, Arendt et al (1961) and Rhinelander (1965) found that vessels in compact bone consist of more than simple capillary ramifications.

In the series of topical epinephrine applications reported here, readily quantified constriction ensued in numerous periosteal branches. Changes in diameter ranged from 6.4 to 18.1 μ. Original diameters of all vessels measured exceeded the known limits of capillary classification. These studies lead to the conclusion that the periosteal circulation is not an entirely passive capillary net, but includes muscularized vascular components capable of initiating directional flow.

Selective cauterization of periosteal arterial supply resulted in cessation of subtending surface compactum flow and marked retardation of venous return. Longitudinal flow continued when venous return was diminished, but the arterial supply left intact. Results of these procedures indicate that a considerable portion of the surface cortical network receives periosteal blood. Lack of change in flow pattern following cauterization of a single venule infers a shift of subperiosteal drainage to another venous segment. The rapid abatement of oozing from the bone surface following removal of the periosteum suggests a similar shunting of venous blood. Hemostasis in the affected transcortical vessels produces a change in the pressure gradient which is probably sufficient to reroute venous drainage through medullary channels. The profuse venous vasculature of the periosteum, but minimal oozing of blood from membrane-stripped bone suggests that periosteal venous vasculature is not the principal route for drainage of blood of medullary origin,

but rather that it serves primarily to collect periosteal blood. This theory is supported by the experiments of Branemark (1961), which showed that vessels of medullary origin penetrate the endosteal portion of the cortex and, after a short excursion, reenter the marrow cavity to drain into medullary venous segments.

Modified flow in response to variations in periosteal supply evinces a cortical distribution of periosteal origin. The presence of such supply is substantiated by the results of perfusion studies. Perfusions of single triads at physiological pressures disclose periosteally derived channels penetrating the cortex. Localized perfusion of the membrane produce cortical vascular casts confined to the area immediately subtending the filled periosteal area. Absence of perfusion medium in the compactum on the opposite side of the bone confirms its penetration from the periosteum as opposed to derivation from a medullary source. This implication of a segmental distribution from periosteum to compactum is supported by results of cautery-isolation experiments. When a triad was isolated on all sides but that of the distributing arteriole, blood flow within the area was essentially unimpaired in both vascular planes.

Heavy perfusion of the talus supports Haliburton et al (1958), who postulated that the periosteum is the major source of ankle nutrition. Dye found in the medullary canal near the ankle implicates medullary vessels as a probable route for venous drainage of the ankle.

The results of this study suggest that circulation in bone is not solely centrifugal. Physiological experiments demonstrating centripetal flow from abundant periosteal arterioles indicate a periosteal contribution to the nutrition of compact bone.

SECTION V

SUMMARY

The nature and ramifications of periosteal circulation were observed in living rabbit tibiofibulae. Profuse periosteal vascularization was qualitatively and quantitatively analyzed. Presence of smooth muscle components in vessel walls was demonstrated by marked vaso-constriction in response to topically applied epinephrine. Vessels exhibiting this response were identified as arterioles or metarterioles. Persistent flow in cautery-isolated triad units elucidated the segmental nature of periosteal vascular distribution. This segmental distribution was viewed in cortex sagittal sections following perfusion of a single triad.

Removal of the periosteum resulted in broadcast stasis within the longitudinal vessels of the surface compactum. This was interpreted as evidence of the necessity of periosteal supply for the integrity of cortical blood flow. The resultant and transitory bleeding from damaged vessels was indicative of a probable low pressure centrifugal flow of medullary origin presently redistributed to other drainage routes. Perfusions at physiological pressures demonstrated a cortical supply derived from periosteum, as evidenced by photomicrographs of intact and sagittally sectioned bone. Vessels entering subperiosteal longitudinal channels and penetrating the compactum were readily identified in perfused vasculature. Absence of perfusion medium from the opposite cortical wall substantiated the perfusate's entrance from the periosteum.

It is concluded that the evidence presented in this study strongly suggests a positive involvement of the periosteal arterial blood supply in the nutrition of compact bone under normal physiological conditions.

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