The Heel

Anatomy, Blood Supply, and the Pathophysiology of Pressure Ulcers

Adam Cichowitz, MBBS (Hons), BMedSc,† Wei Ren Pan, MD,*† and Mark Ashton, FRACS*†*

Abstract: There remains much confusion regarding the pathophysiology of pressure ulcers. Data indicate that the prevalence of pressure ulcers is increasing. The heel is unique in structure and well adapted to the task of shock absorption. However, it is often subject to prolonged pressure, which predisposes it to tissue breakdown, with attempts at reconstruction prone to failure.

Four dissections were carried out of the heel region, which included removing each heel pad en bloc for histology. Seventeen arterial injection studies, 12 venous studies, and a combined arterial and venous study of the foot were performed. The results were correlated with clinical cases and previous research.

The heel was found to be richly vascularized by a subdermal plexus and periosteal plexus with vessels traveling between the 2 within fibrous septa that connect the reticular dermis and periosteum of the calcaneus. These septa effectively create isolated compartments containing relatively avascular fat. A layer of panniculus carnosus muscle was observed in the subcutaneous tissue.

It is likely that the metabolically active panniculus carnosus muscle is involved early in the course of pressure ulcers. Extensive pressure damage can be concealed by intact skin. Friction and shear are additional factors important in skin breakdown.

Key Words: panniculus carnosus, septa, ischemia, pressure, friction, shear, top-to-bottom, bottom-to-top, middle model, compartment syndrome

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In the context of an aging population, the problem of pressure ulcers continues to grow. Despite the significant humanitarian, social, and economic cost to the patient and community, the issue fails to receive the attention it deserves. There remains a lack of understanding of the basic pathophysiology of pressure ulcers due largely to limited research, which has led to inadequate prevention and treatment.

An American study reported that 1.6 million pressure ulcers develop annually in hospitals in the US with an estimated yearly cost of $2.2 to $3.6 billion. Each stage III or stage IV pressure ulcers adds between $14,000 and $23,000 to the cost of care.[1] Other research has calculated that the development of a pressure ulcer adds $10,845 to the cost of care, prolongs the hospital stay by 3.98 days and increases mortality by 7.23%.[2] The prevalence of heel pressure ulcers varies across settings but in hospitalized patients, it ranges between 10% and 18%[3] with the sacrum and heel being the most common sites.[4] The predominant age group of patients is 71 to 80 years. Seventy-four percent of pressure ulcers are superficial (ie, stages I and II). However, the prevalence of pressure ulcers is increasing despite many recent changes in health care.[3]

The most commonly used staging system for pressure ulcers classifies tissue damage according to the anatomic depth of involvement.[5,6] It implies a linear progression of damage that begins at the skin surface and progresses to involve deeper tissues. This top-to-bottom view of pressure ulcer evolution has been challenged by an alternative bottom-to-top model which suggests that damage begins at the underlying bony prominence and extents up toward the skin (inverted cone configuration).[7] However, studies have consistently demonstrated the sensitivity of muscle to pressure and ischemia due to its high metabolic activity.[8,9] This would imply a "middle model" of pressure ulcer development with injury first manifesting at any level where muscle is interposed between skin and bone. The problem with this model is that cadaver and clinical dissections have shown that muscle is seldom if ever interposed between bone and skin over bony prominences in normal weight-bearing positions.[10] Furthermore, many experimental models have failed to account for friction and shear in the causation of pressure ulcers. Dinsdale[11] found that the combined effect of pressure and friction was an experimental pressure ulcer that was quite superficial with minimal extension below the dermis.

Amid the confusion surrounding the pathophysiology of pressure ulcers, a study was undertaken to analyze the anatomy and blood supply of the heel to better understand the development of pressure ulcers in general but also the specific mechanism of tissue breakdown and necrosis in the heel.

MATERIALS AND METHODS

Four dissections were performed of the heel region in 3 separate cadavers (1 embalmed, 2 fresh) which included histologic analysis of the heel pad following en bloc removal from the underlying calcaneus. This involved fixing the tissue in 10% buffered formalin and dividing each heel into 16 blocks, which were individually embedded in paraffin and cut into 7-μm sections. The sections were then stained with standard hematoxylin and eosin. Masson's trichrome is a staining technique that highlights collagen, and was used to demonstrate the connective tissue architecture of the heel. The slides were carefully analyzed and photographed, and measurements of the various tissue layers and structures were performed with a digital Vernier caliper.

Arterial studies were performed on a total of 17 lower limbs from fresh cadavers that showed no obvious evidence of peripheral vascular disease or scarring using a technique previously described by Taylor and Pan.[12,13] The arterial injection was carried out by either total body or isolated limb perfusion using a mixture containing radiopaque lead oxide. The contribution of different arteries to the blood supply of the foot and heel was defined by dissection, metal clip tagging of vessels, mapping branches with colored pins, and radiography. The integument was analyzed using a subtraction technique whereby the bones of the foot were replaced subperiosteally with radiolucent balloons to obtain unobscured images of the vasculature.

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From the *Jack Brockhoff Reconstructive Plastic Surgery Research Unit, The Royal Melbourne Hospital, Melbourne, Australia; and †Department of Anatomy and Cell Biology, The University of Melbourne, Melbourne, Australia.

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Reprints: Adam Cichowitz, MBBS (Hons), BMedSc, 3 Rochelle Court, Wheelers Hill, Victoria 3150, Australia. E-mail: agcich@hotmail.com.

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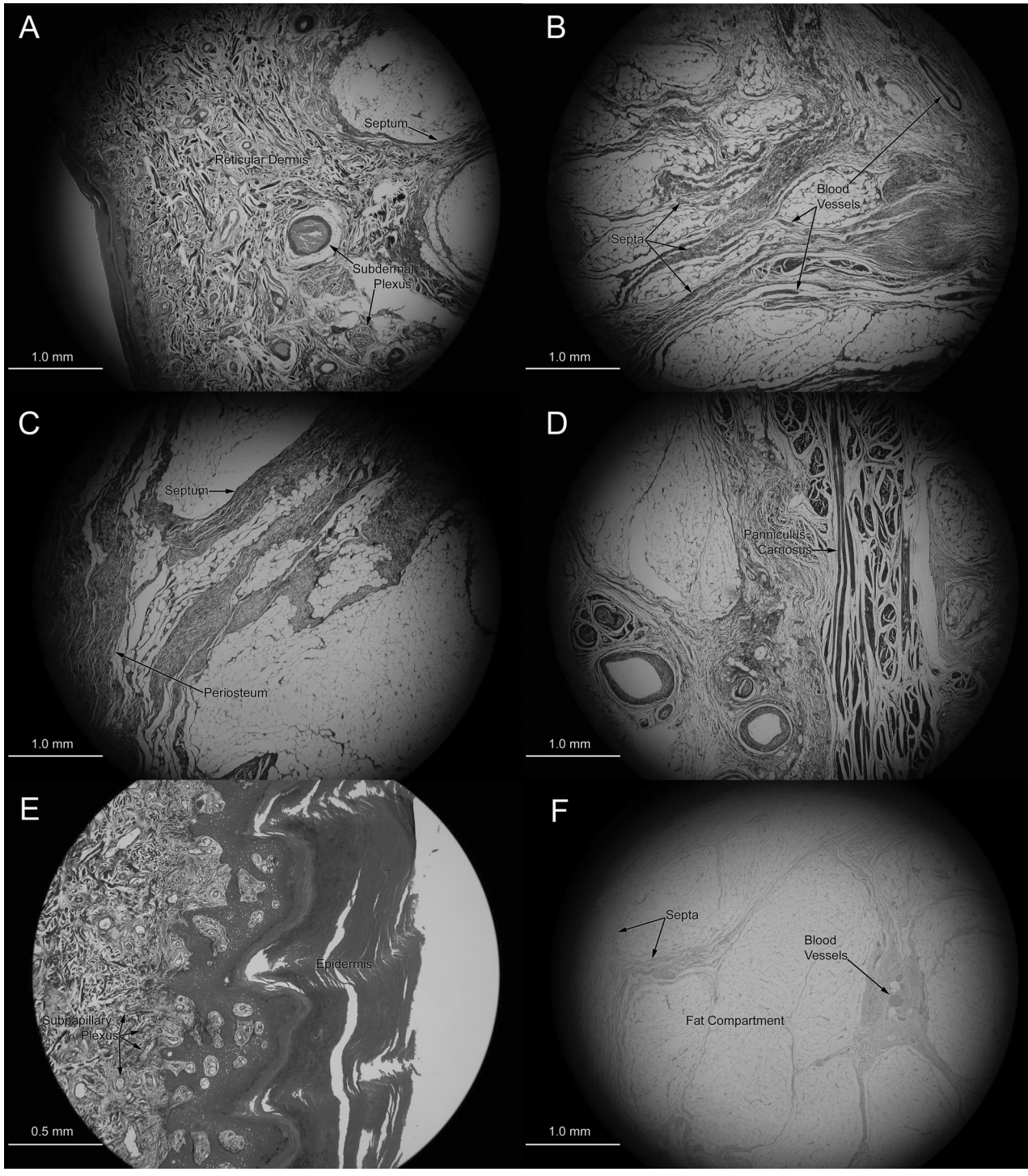

FIGURE 1. Histologic studies of the heel. A–C, Collagenous septa run between the reticular dermis and periosteum of the calcaneus and transmit blood vessels between a subdermal plexus and periosteal plexus. D, A layer of panniculus carnosus muscle lies within the subcutaneous tissue. E, The upper dermis is well vascularized by a subpapillary plexus and is protected from external trauma by a thick layer of epidermis. F, The fibrous septa create relatively avascular compartments of fat within the subcutaneous tissue. A–E, Masson's trichrome stain. F, Hematoxylin and eosin stain.

Twelve venous studies of the lower limb were performed and compared with the arterial studies to obtain a detailed view of the blood supply of the heel. The technique used has been previously described[14] and involved total body venous infusions of lead oxide, gelatin and the preservative chlorocresol in 6 fresh cadavers, following which perforators were tagged and the integument removed

and radiographed. Furthermore, a combined arterial and venous injection study of the foot was performed by cannulating the arteries and veins of an isolated foot and then slowly perfusing the vessels with a tourniquet applied proximally. This technique yielded highly detailed images of the arterial and venous anatomy of the heel.

RESULTS

Histologic analysis revealed the heel pad to form an almost fully contained cup-like structure consisting of skin overlying a shell of connective tissue within which fibrous septa were seen to ramify throughout the heel connecting the underlying periosteum of the calcaneus to the overlying reticular dermis, thereby anchoring skin to bone (Fig. 1A–C). Evidence for the shock-absorbing function of the heel was provided by the observation of loculi or islands of fat between the septa. During the dissections, the fat was found to be rather fluid in nature and under pressure so that the tissue bulged when cut. An abundance of sweat glands was observed and a layer of panniculus carnosus muscle was present in the subcutaneous tissue within the cup-like shell of connective tissue (Fig. 1D). As expected, no hair follicles or sebaceous glands were found.

As calculated from measurements obtained from the histologic specimens, the heel had an average thickness of 18.5 mm (n = 64, standard deviation = 3.1 mm). The average total thickness of the skin was 3.75 mm (n = 64, standard deviation [SD] = 0.97 mm) which is comparable with other areas of the body, while the epidermis was relatively thick at 0.46 mm (n = 64, SD = 0.17 mm), especially the stratum corneum (Fig. 1E). The average thickness of the panniculus carnosus muscle was 1.52 mm (n = 64, SD = 0.40 mm), representing only 8.2% of the thickness of the heel but 41% of the total skin thickness. The fat compartments were spherical to ellipsoidal in shape and varied from approximately 0.6 to 10 mm in diameter with the majority between 1 and 5 mm (Fig. 1F).

The dissections and analysis of the arterial studies revealed the heel to be supplied posteriorly by the medial calcaneal branch of the posterior tibial artery with a variable but small contribution from the lateral calcaneal branch of the peroneal artery. The anterior part of the heel was supplied primarily by the lateral plantar artery with a reliable contribution from the medial plantar artery (Fig. 2). There was a rich anastomosis between these vessels at 2 levels in the heel-subdermal and periosteal (Fig. 3). The subdermal plexus was more densely vascular than the periosteal plexus although there were significant anastomoses between the 2 via vessels traveling in the fibrous septa as confirmed by the histologic studies (Fig. 1A, B, F). In contrast, the compartments of fat confined between the fibrous septa were almost avascular. High magnification views of the dermis confirmed the presence of a subpapillary plexus forming capillary loops in the dermal papillae, supplying the upper dermis and anastomosing with the deeper subdermal plexus (Fig. 1E).

The venous drainage of the heel into the deep venous system essentially mirrored the arterial supply with the venae comitantes of the lateral plantar artery largely responsible for draining the deeper tissues of the heel and periosteal plexus. There was also a small amount of venous drainage into the venae comitantes of the medial plantar artery and the medial and lateral calcaneal branches of the posterior tibial and peroneal arteries respectively (Fig. 4). However, the superficial venous drainage of the heel differed from the arterial supply with drainage of the subdermal plexus and subpapillary plexus into veins which ultimately joined the great saphenous and small saphenous veins (Fig. 5). Like the arterial supply, the superficial and deep

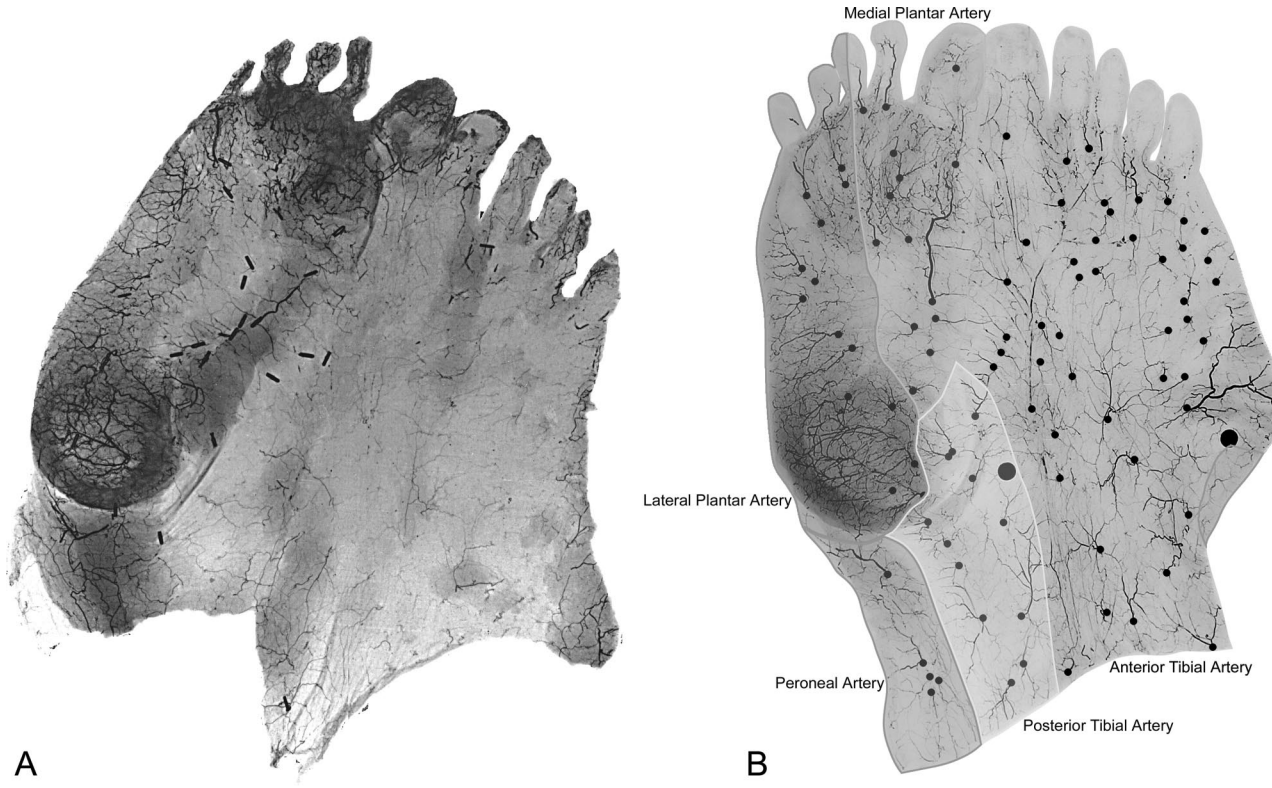

FIGURE 2. A, B, Radiographs of the arterial supply of the skin of the foot. B, The vascular territories have been defined in terms of the source artery. Dots mark the cutaneous perforators.

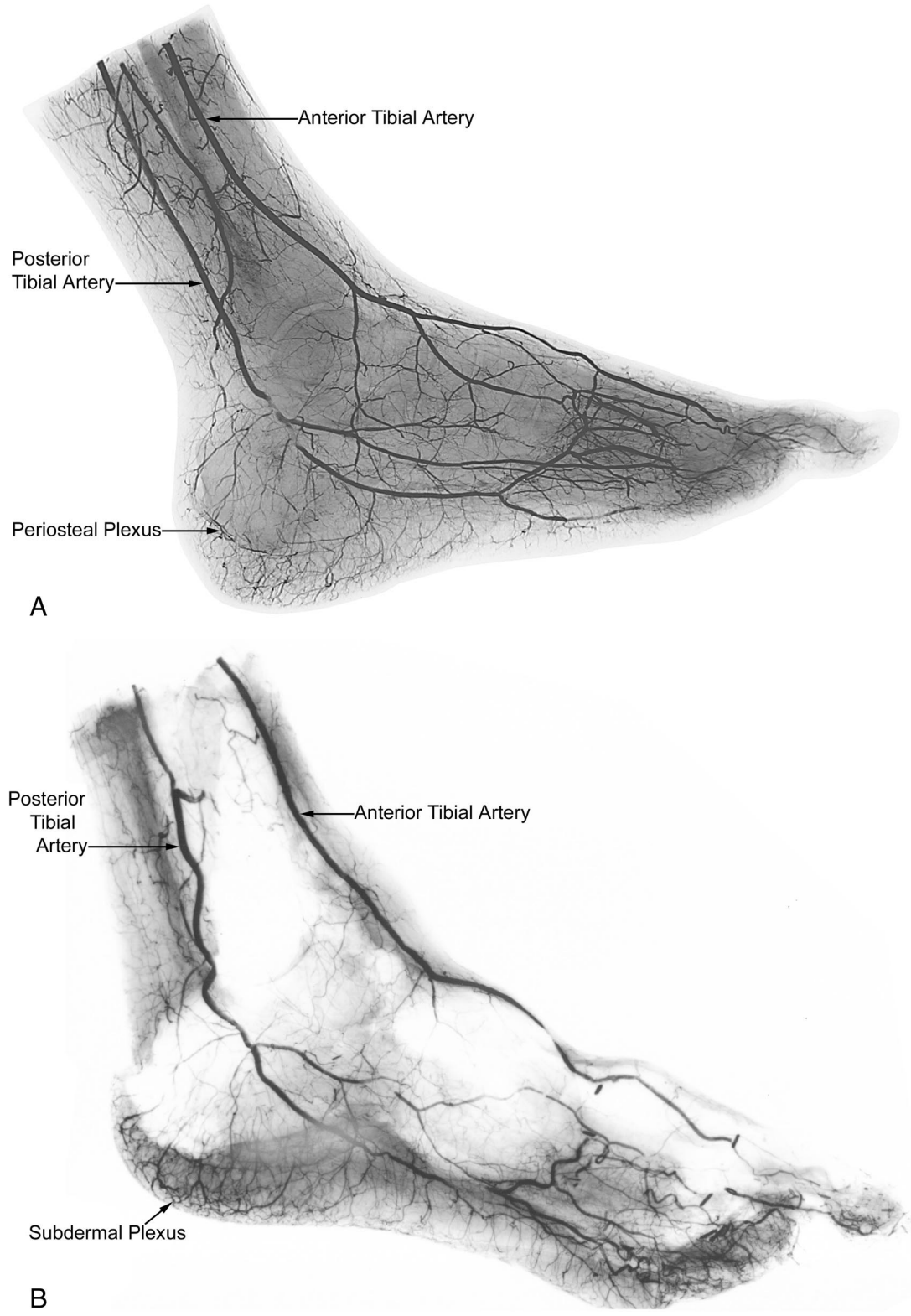

FIGURE 3. A, B, Radiographs of the arterial supply of the foot demonstrating the periosteal plexus and subdermal plexus. B, Following removal of the underlying bones.

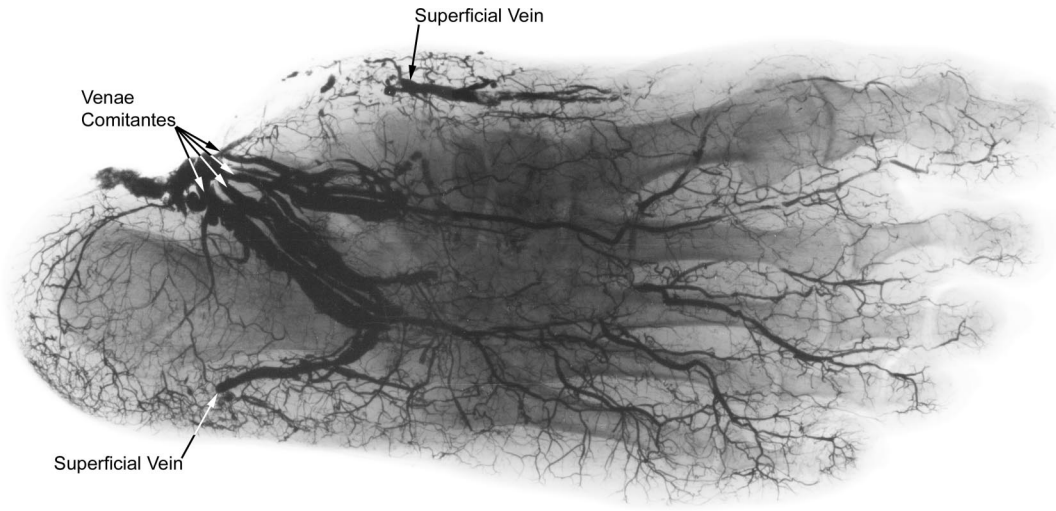

FIGURE 4. Radiograph of the arterial and venous anatomy of the foot following perfusion an isolated foot. Veins can been seen to the level of the midfoot where valves have halted distal progression of the lead oxide mixture.

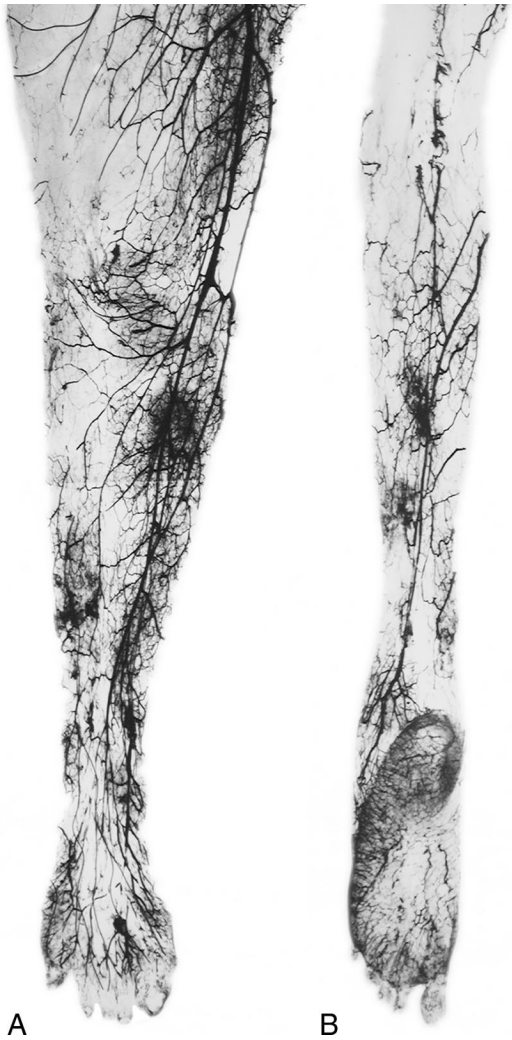

FIGURE 5. Radiographs of the venous drainage of the skin of the dorsum (A) and plantar surface (B) of the foot.

venous networks communicated via vessels traveling in the fibrous septa of the heel.

DISCUSSION

Theoretical and experimental models indicate that pressure is 3 to 5 times higher internally near a bony prominence than it is at the skin surface. Authors have thus asserted that pressure ulcers must begin internally at bone and progress outward.[7] This supposition fails to take into account the vascular supply and metabolic demands of different tissues which together determine their susceptibility to pressure and ischemia.

The periosteum of the calcaneus has a rich vascular supply from the periosteal plexus supplemented by anastomosing vessels from the subdermal plexus traveling via the fibrous septa. This arrangement of septa transmitting vessels between periosteum and skin is remarkably similar to that in the fingertips which are also intermittently subject to intense pressure and friction. Although periosteum is subject to high pressures, it is normally relatively quiescent in terms of metabolic activity. It is therefore difficult to argue that periosteum is the most sensitive tissue to pressure-related ischemia and the initial focus of injury in pressure ulcer development. On the other hand, muscle has a high metabolic activity and is inherently susceptible to ischemia.[8,9] The only muscle located in the heel is the panniculus carnosus layer in the subcutaneous tissue which nevertheless has a rich vascular supply from the adjacent subdermal plexus and vessels running up in the fibrous septa from the periosteal plexus. The panniculus carnosus may be the primary site of injury in heel pressure ulcers. This notion is supported by 1 study that demonstrated early necrosis of the panniculus carnosus muscle in experimentally induced pressure ulcers over the trochanteric region of the fuzzy rat.[15]

The heel is a unique and complex structure specifically adapted to its function of shock absorption. This is part of the reason why it is difficult to reconstruct and why such efforts are prone to failure unless the mechanical demands and blood supply are taken into account when designing a reconstructive effort. The tissue in the heel with the most marginal vascular supply is the relatively avascular fat located in loculi or compartments between the fibrous septa. Early necrosis of subcutaneous fat has been demonstrated in a previous study involving biopsies of pressure ulcers in humans.[16] The subcutaneous fat in the heel may be especially vulnerable to

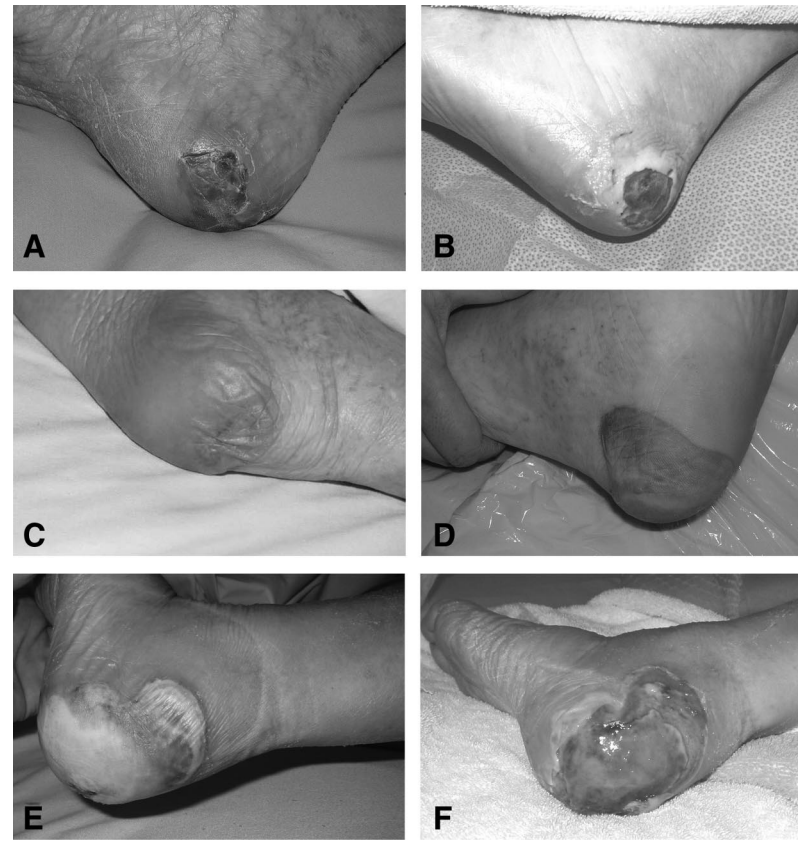

FIGURE 6. Photographs demonstrating various forms of pressure damage to the heel. A, B, Superficial ulceration. C, D, Deep injury concealed by intact skin. E, F, The true extent of tissue damage in a patient was revealed only after breakdown of the overlying skin.

ischemia because the fibrous septa essentially form sealed compartments that inhibit the dissipation of external pressure and create a situation analogous to compartment syndrome. Pressure within the fat compartments leads to ischemia with resultant inflammation and edema, which in turn lead to increased pressure and further ischemia, thereby establishing a vicious cycle of tissue destruction. The heel may also undergo tissue breakdown as a consequence of its limited surface area of contact and small subcutaneous tissue volume with pressure exerted directly on the bone.[17] Furthermore, the ability of the heel pad to absorb shock declines with age.[18] Heel skin in the elderly is also less resilient than in the young.[19]

Skin is extremely resistant to ischemia and can withstand 12 hours of normothermic ischemia without necrosis.[20] Daniel et al[8] observed skin damage as early as 8 hours but hypothesized that skin destruction was due to "primary mechanical damage." Histopathology of pressure ulcers confirms that underlying tissue damage can be severe before ulceration of the skin appears.[15,16] However, Dinsdale[11] produced superficial ulcers when friction was combined with pressure. Friction is the adherent force resisting shearing movement of the skin but may result in denuded areas of the dermis through repeated epidermal shedding or avulsion of sheets of epidermis. Additionally, prolonged exposure of skin to moisture from perspiration, urinary or fecal incontinence, or wound exudate can further weaken intercellular bonds in the epidermal layer and lead to maceration and ulceration.[21,22] The epidermis of the heel is relatively thick although it may be involved early in the pathogenesis of pressure ulcers depending on circumstances such as the presence of friction, shearing forces, maceration, and mechanical damage.

Clinically, 2 types of heel pressure ulcers are observed-superficial (stages I and II) and deep (stages III and IV). The stage of an ulcer does not seem to reflect the degree of pressure applied. Deep lesions are not necessarily caused by more pressure than superficial lesions. It is likely that the mechanism of injury is important. Superficial lesions seem to have friction and shear as significant contributing factors whereas prolonged pressure as a relatively isolated factor tends to result in deep lesions. Isolated pressure damage in the heel probably progresses according to a "middle model" with the initial injury involving the panniculus carnosus muscle and subcutaneous fat and eventually spreading throughout the heel. This explains how the extensive tissue damage described by stage IV may be concealed by intact skin, which would traditionally be categorized as a stage I lesion (Fig. 6).

CONCLUSIONS

The heel is the second most common site for pressure ulcers after the sacrum. Its structure is unique and complex. It is well adapted to the task of shock absorption but it is often subject to prolonged pressure which if unrelieved can result in tissue breakdown and necrosis. The heel consists of skin overlying a cup-like shell of connective tissue within which interconnecting septa join the reticular dermis and periosteum of the calcaneus. Four arteries supply the heel and form a rich anastomosis with vessels traveling in the fibrous septa between a periosteal and subdermal plexus. However, the septa create relatively avascular compartments of fat which may be vulnerable to ischemia in a scenario akin to compartment syndrome. The heel also has a small surface area of contact and little subcutaneous tissue volume with pressure exerted directly on bone. Metabolically active tissue in the form of panniculus carnosus muscle is found in the subcutaneous layer and it is likely that this tissue and the subcutaneous fat are involved early in the course of pressure ulcers.

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