The Hoof Mechanism Revisited

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Introduction

It is a basic principle of farriery that to fasten the shoe to the hoof, nails must NOT be palmar to the widest part of the hoof (quarters). Presumably, this preserves the hoof mechanism as palmar nailing would prevent the operation of the mechanism and be deleterious. After centuries of farriery, and the application of what must be millions of shoes, no direct pathological consequences result from correct, conventional shoeing. The question remains - how important is the hoof mechanism; at what level of the circulation does it operate; does it operate regardless of the type of shoe applied; what would happen if it were abolished?

Background

There was a period in the history of farriery when compression of the frog and the digital cushion were thought to be the vascular pump of the foot[1]. Accordingly, the sole was thinned and the heels and bars trimmed short to ensure the frog bore weight and made ground contact. This was the 'College System' advocated by the head of the London Veterinary College, Professor Edward Coleman, and widely adopted in the early 20th Century. However, Coleman had his detractors. Frederick Smith stated[2] "Perhaps in nothing was his ignorant self-confidence more evident than in the subject of shoeing. He was not long installed as Professor before he discovered things in his study of the foot which did not exist, and at a time when his knowledge was rudimentary he produced shoes intended to combat evils that were only imaginary."

Decades of debate and experimentation, dating back centuries, were required to 'prove' the existence of the hoof mechanism (HM). In 1891, both Lungwitz[3] and Smith[4] devised an apparatus for measuring hoof expansion under load and came to the same conclusions, unaware of each other's work[1]. Their diagrams are still used to describe the hoof mechanism (Fig. 1).

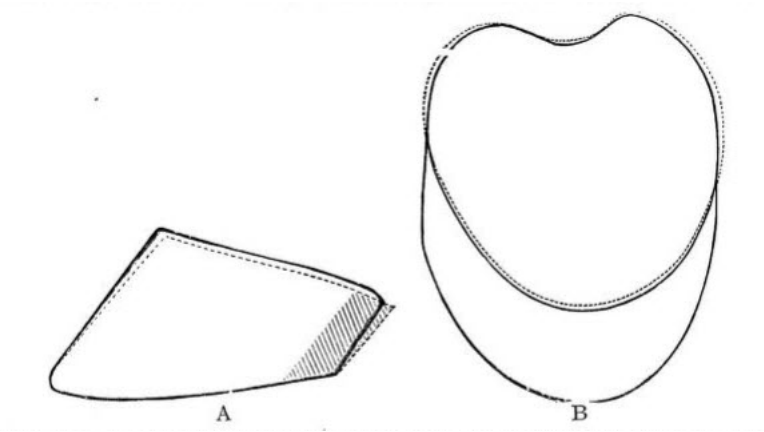

Fig. 1. Lungwitz 1891 diagram showing the areas of the hoof that expand, 'retreat' and sink during the load cycle. The shaded area shows where the heels expand.

The hoof mechanism defined

The hoof capsule deforms during cyclic limb loading; the proximal hoof wall moves inwards and downwards, the quarters expand outwards and the sole flattens (Fig. 2).

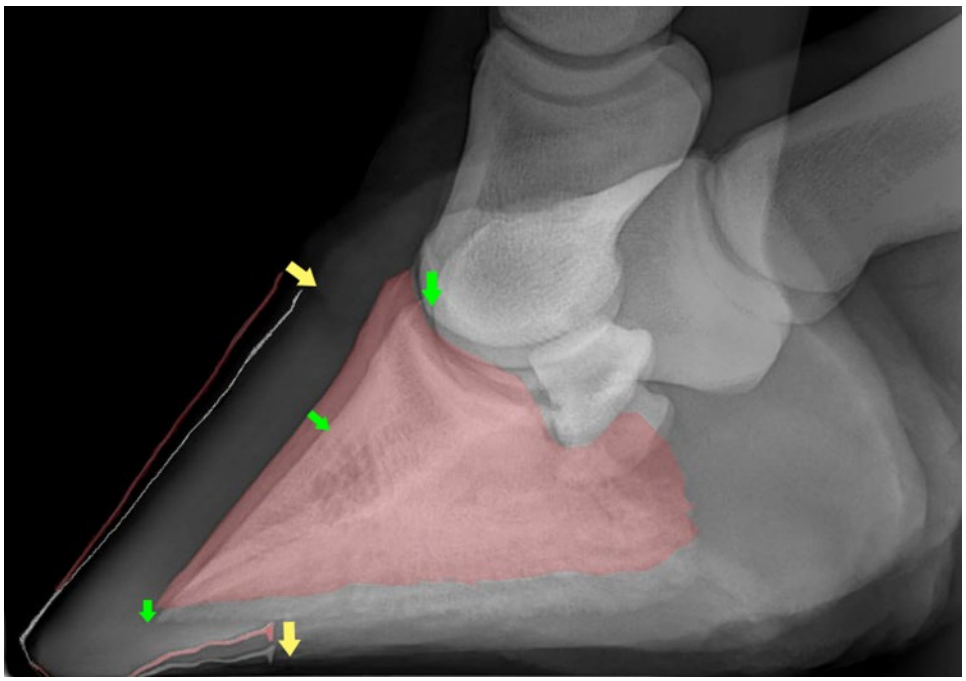

Fig. 2. Non-loaded (pink outline) and loaded (white outline) paired radiographs of a normal horse foot. Barium sulphate paste outlines the surface of the midline, dorsal hoof wall and the sole. Loading to twice the horse's body weight flexes the distal interphalangeal joint (DIPJ) and moves the distal phalanx inwards and downwards (green arrows). Commensurate with this, the proximal hoof wall moves inwards and the sole flattens (yellow arrows).

The hoof mechanism proposes that expansion and contraction of the hoof capsule is entrained with the limb load cycle, and that this is of physiological importance particularly to the blood vascular system. Certainly venous return is readily demonstrated in a cannulated, perfused cadaver limb undergoing cyclic loading (Fig. 3).

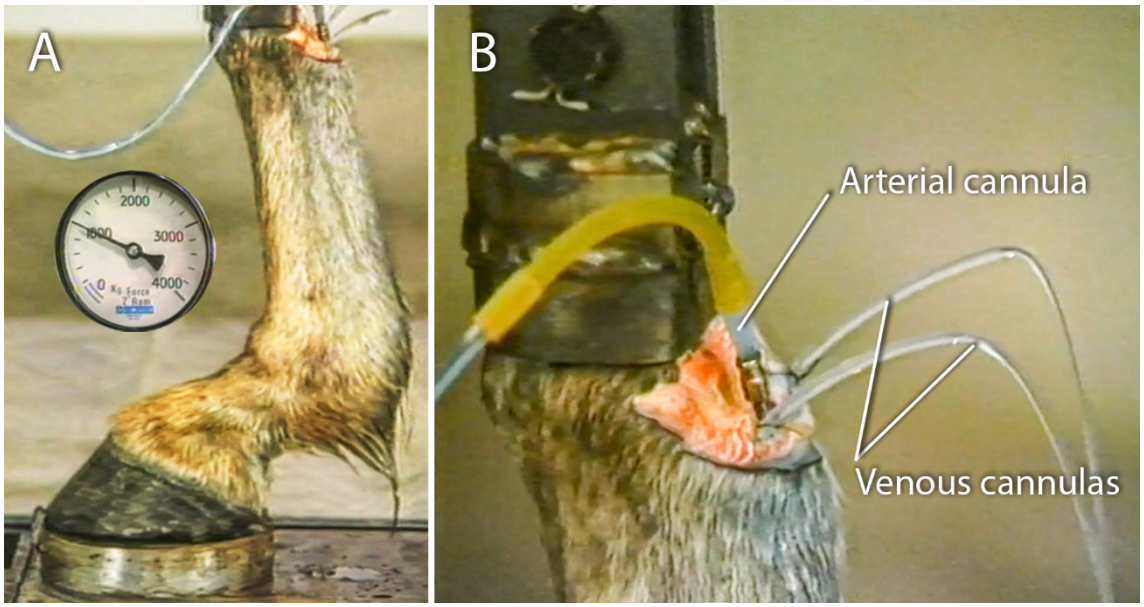

Fig. 3. Cadaver limb with common digital artery and medial and lateral digital veins cannulated. The artery is perfused with normal saline and the limb is cyclically loaded to 1000 kg (A) and then unloaded. At peak load the perfusate spurts from the venous cannulas thus demonstrating how blood is pumped up the leg and returned to the heart to complete the circulation. (See full video at https://vimeo.com/339479479)

The foot, situated a considerable distance from the heart, is subjected to large fluctuations in hydraulic pressure and centrifugal force, especially during the stance phase of the high-speed gallop. Several physical problems are overcome for blood to circulate to the foot and return to the heart. The French veterinary scientist Bouley made insightful observations in 1851 describing the horse's foot as an additional heart, working as ''a pushing and sucking pump''.[5] The ''pushing'' phase (venous return) comes from descent of bones, tendons, and cartilage into the semi-flexible hoof capsule, compressing the dermis and the blood vessels contained within. The low-resistance veins and lymphatic vessels are more affected by the pressure than the thick-walled arteries, and conduct blood and lymph away from the foot. During the support phase of the stride, venous pressure increases to a peak in the digital veins. At the trot this parallels the peak vertical force of the load.[6] Likewise, in a cadaver limb, pressure measured in the sublamellar dermis peaks in conjunction with the load cycle[7] (See video at https://vimeo.com/339479479).

The absence of valves in the extensive anastomosing venous network of the foot ensures an unencumbered, rapid return of blood to the heart (and coincidentally facilitates retrograde venography). For a pump to operate effectively, no back flow must occur. Bouley assumed that systolic blood pressure in the completely valveless arterial system was the mechanism preventing retrograde arterial flow. However, the horse foot experiences loading forces during the stance phase equivalent to 3 times the body weight at the gallop, inducing internal forces far exceeding systolic pressure. Hence, without the presence of other protective mechanisms to safeguard the digital circulation, turbulent arterial backflow would occur. When the loading (pressure) phase ends, veins are refilled (Bouley's ''sucking'' phase) ready for the next cycle. This phase is easily demonstrated on cannulated, cadaver distal limb specimens that have the distal check ligament intact.[8] See full video at https://vimeo.com/339479479).

Angiography

A more complete understanding of foot circulation came from the pioneering, angiographic studies of van Kraayenburg and colleagues in 1982,[9,10] using contrast agent injected into the palmar digital arteries of conscious, standing horses. With the foot relaxed and unloaded, contrast medium injected into the digital arteries completely filled the terminal arch within the distal phalanx (DP). From the terminal arch, arterial branches exited via numerous foramina in the dorsal parietal surface of the DP to supply the dorsal, lamellar region. However, when one forelimb was lifted and then contrast was injected into the palmar artery of the now supporting limb, arterial constrictions were evident that prevented blood flow into the terminal arch and the dorsal lamellae. The arterial termination points were adjacent to the medial and lateral extremities of the distal sesamoid (navicular) bone and within the terminal arch itself. Although technical difficulties prevented this being demonstrated in vivo, it was readily replicated in cadaver limbs subjected to calibrated loads in a hydraulic press. Each increment of load further flexed the distal interphalangeal joint (DIPJ) until flow in the palmar digital arteries was terminated. The load required was less than the forequarter mass of the horses (262 ± 52 kg).[9,10]

Thus, it was established that for each limb there was a mechanical load that could occlude arterial blood flow to the foot. Arterial closure was considered essential and physiologic in normal horses, and was the likely mechanism preventing arterial back flow during the compressive load phase of the stride. Thus, approximately 130 years after the Parisian veterinarian Bouley proposed that blood pressure prevented arterial back flow during the stance phase, it was the South African farrier van Kraayenburg that completed the explanation by discovering that, complementary to arterial blood pressure, there was a valve-like, closing mechanism in the digital arteries of the loaded distal limb.

van Kraayenburg's angiography of the cadaver distal limb under load has been replicated in the authors' laboratory; first using digital subtraction angiography in 1992,[8] and recently using computed tomography (CT) and Mimics modelling software (Materialise NV, Leuven, Belgium), with barium sulphate as a vascular contrast medium[11]. Barium sulphate is a suspension of particles too large to enter capillaries, so either arteriograms (Fig. 04) or venograms are obtained by infusing the appropriate vessels.

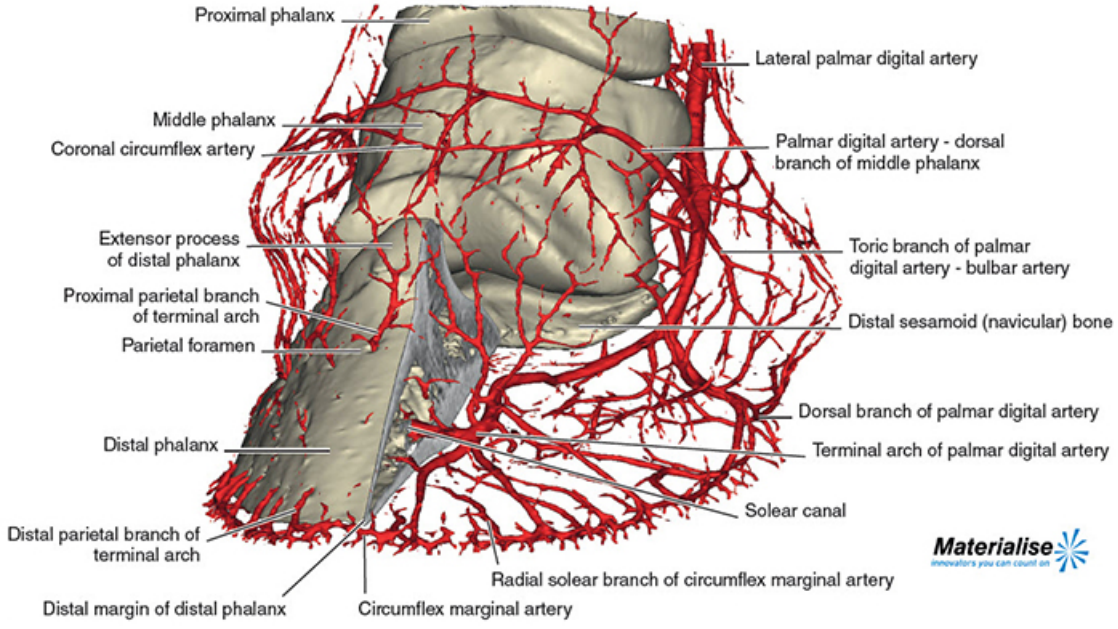

Fig. 4. Arteriogram from Mimics modelled computed tomography (CT) data of arteries and bones after infusion of the median artery of the left forelimb with barium sulphate (normal cadaver specimen). The distal phalanx is in sagittal section without its lateral half. The terminal arch of the medial and lateral palmar digital arteries is visible, deep within the distal phalanx. The arterial branches that radiate from the terminal arch exit through foramina in the parietal cortex of the distal phalanx. The distal parietal branches of the terminal arch exit the distal phalanx and unite to form the circumflex marginal artery. From the circumflex marginal artery, radial solear branches curl inward under the distal margin of the distal phalanx and supply the solear dermis. Copyright "The Illustrated Horse's Foot" by Christopher C. Pollitt, Elsevier, St Louis, Missouri. ISBN 9780702046551.

Barium arteriograms of normal, nonloaded Standardbred feet showed the expected vascular pattern. Contrast medium in the medial and lateral digital arteries, entering the solear canal on the palmar aspect of the DP, showed no evidence of constrictions (Fig. 5).

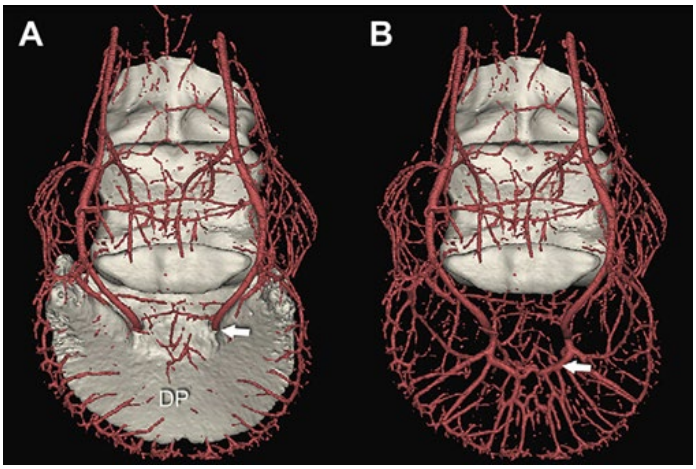

Fig. 5. Mimics models of computed tomography data of normal Standardbred cadaver foot with barium sulphate infused into the common digital artery. The medial and lateral branches of the artery enter the solear canal of the distal phalanx via foramina (arrow in A). Within the distal phalanx the arteries anastomose and form the terminal arch (arrow in B).

When the distal limb was moderately loaded (Fig. 6), Mimics models of the CT data showed occlusion of the medial and lateral digital arteries adjacent to the extremities of the navicular bone (see white arrows in Fig. 06) thus confirming the results of van Kraayenburg and colleagues.

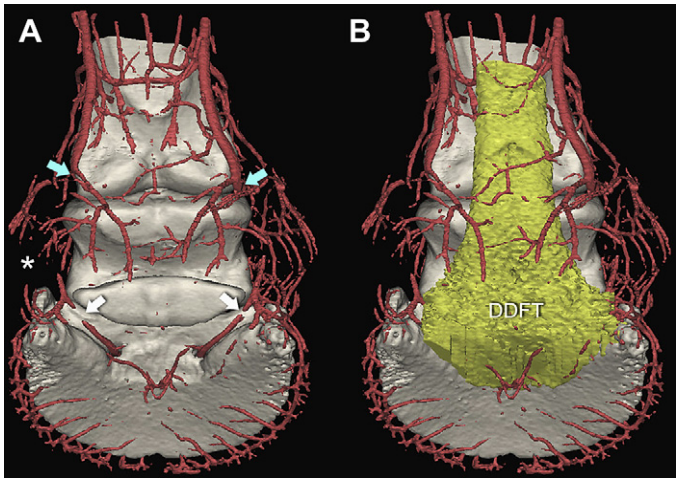

Fig. 06. Palmar views of Mimics models of a moderately loaded distal limb with the common digital artery infused with barium sulphate. There is occlusion of the medial and lateral digital arteries adjacent to the extremities of the navicular bone (white arrows in A). A large segment of both the medial and lateral digital arteries is also void of contrast (distal to the blue arrows in A). There is reduced perfusion of the heels and quarters of the foot (asterisk in A). The deep digital flexor tendon (DDFT) has been added to the Mimics model in B. The medial and lateral margins of the insertion of the DDFT closely overlay the points of arterial occlusion.

However, proximal to the navicular bone, a large segment of both the medial and lateral digital arteries was also void of contrast (see blue arrows in Fig. 6A). There was reduced arterial contrast in the heels and quarters of the foot (see asterisk in Fig. 6A). Addition of the deep digital flexor tendon (DDFT) to the Mimics model (see Fig. 6B) showed that the medial and lateral margins of the insertion of the DDFT closely overlaid the points of arterial occlusion. During limb loading, tension in the DDFT may compress the arteries and thus participate in the closing mechanism.

When the distal limb was heavily loaded (Fig. 7) and the common digital artery infused with barium sulphate, there was no filling of any artery below the coronary band.

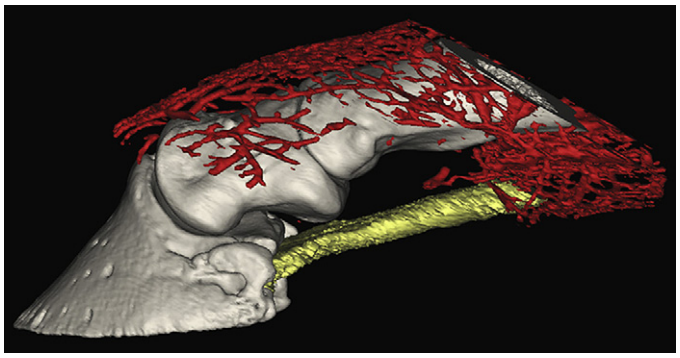

Fig. 07. Lateral view of a Mimics model of a heavily loaded distal limb with the common digital artery infused with barium sulphate. There is zero arterial contrast fill below the coronary band and no blood has entered foot.

Increased tension within the flexor tendons and the digital annular ligaments, as well as global compression of the soft tissues between bone and hoof, prevents arterial blood entering the foot. This degree of DIPJ flexion occurs during locomotion at even moderate speeds. Thus, the horse's foot appears to experience a short period of total arterial occlusion during each limb cycle that coincides with venous and lymphatic outflow from the foot. During the non-loaded, swing phase of

the stride, arterial flow to the foot is restored and veins and lymphatic vessels are refilled. These filling and emptying events alternate in the heart-like manner proposed in 1851 by Bouley.[5]

The over-riding priority of the vascular system of the foot is to deliver energy to the lamellar interface of the suspensory apparatus of the distal phalanx (SADP). Without a fully functional, pain-free, SADP the horse is disabled and from an evolutionary point of view is non-existent and out of the equine gene pool. The horse is the ultimate ungulate with, for each of its 4 limbs, a single distal phalanx attached to the inside, lamellar surface of each hoof capsule (Fig 08).

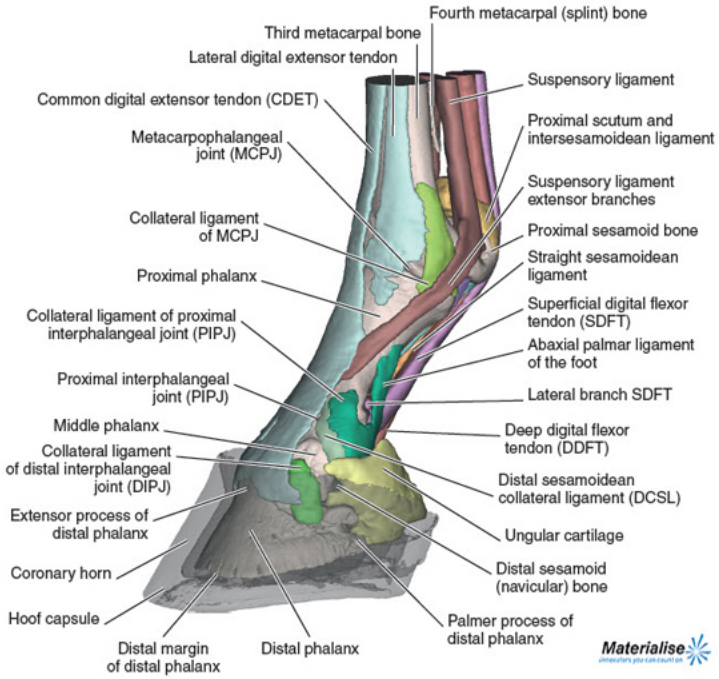

Fig.8. Mimics model of a normal distal limb with a transparent hoof capsule. The single distal phalanx is attached to the inside surface of the hoof capsule. Copyright "The Illustrated Horse's Foot" by Christopher C. Pollitt, Elsevier, St Louis, Missouri. ISBN 9780702046551.

Attachment of surfaces in biological systems requires energy and the source of that energy is the glucose delivered by the arteries of the blood vascular system (Fig. 9).

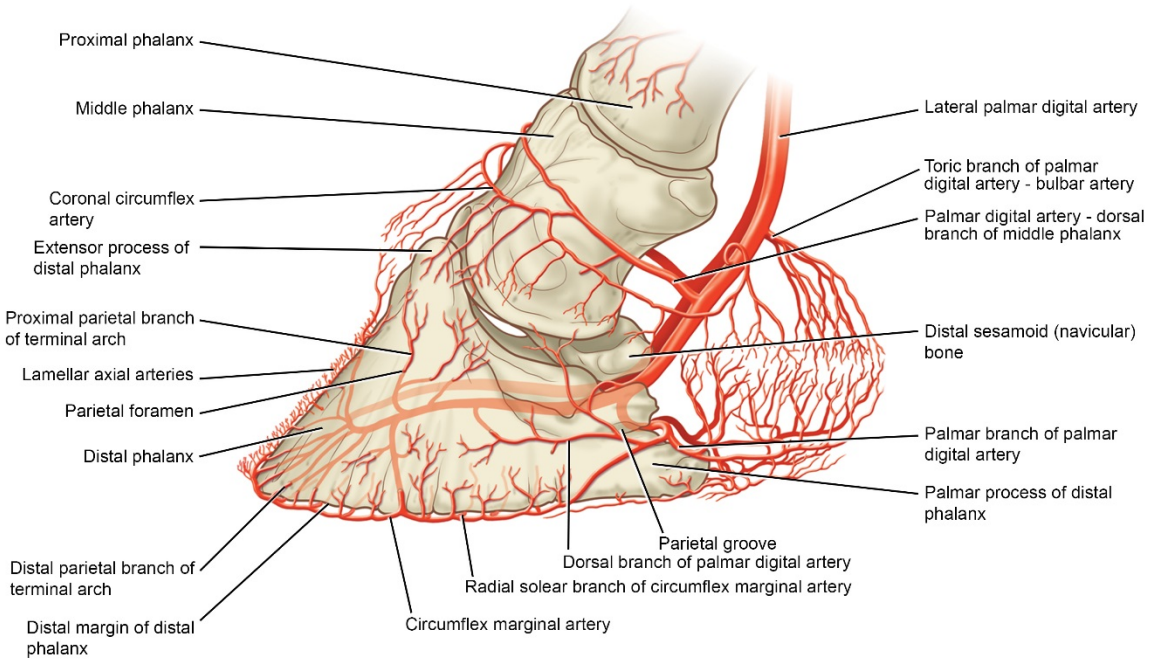

Fig. 9. Diagram of the arterial system of the horse's foot showing the three major branches arising from the medial and lateral palmar digital arteries (the toric, dorsal artery of middle phalanx and the dorsal branch of the palmar digital artery) before they enter the foramina of the distal phalanx solear canal to form the terminal arch. The arterial branches of the terminal arch exit through foramina in the parietal cortex of the distal phalanx. The proximal and distal parietal branches of the terminal arch cross the sublamellar dermis branching to form multiple lamellar axial arteries that ultimately supply the capillaries of the secondary dermal lamellae. The distal parietal branches of the terminal arch unite to form the circumflex marginal artery from which radial solear branches curl inward under the distal margin of the distal phalanx to supply the solear dermis.

The lamellar microcirculation

The lamellar epidermis itself is avascular and relies on adjacent dermal capillaries to deliver the nutrients required to maintain attachment at the lamellar dermal/epidermal interface. Significantly, lamellar capillaries are located only in secondary dermal lamellae (SDLs) and are absent in the primary dermal lamellae and sublamellar dermis. This means that the capillaries are extremely close to the target of energy delivery; the basal cells of the secondary epidermal lamellae (SELs). Delivery of glucose and exchange of water and other materials occurs exclusively across these capillaries (Fig. 10). Most gases and inorganic ions pass through the capillary wall in less than 2 milliseconds[12].Arteries and veins serve to get blood to and away from the capillary bed, but it is the capillaries that deliver the essence of lamellar integrity: glucose. Without a supple of blood sugar (glucose) the lamellar interface disintegrates[13].

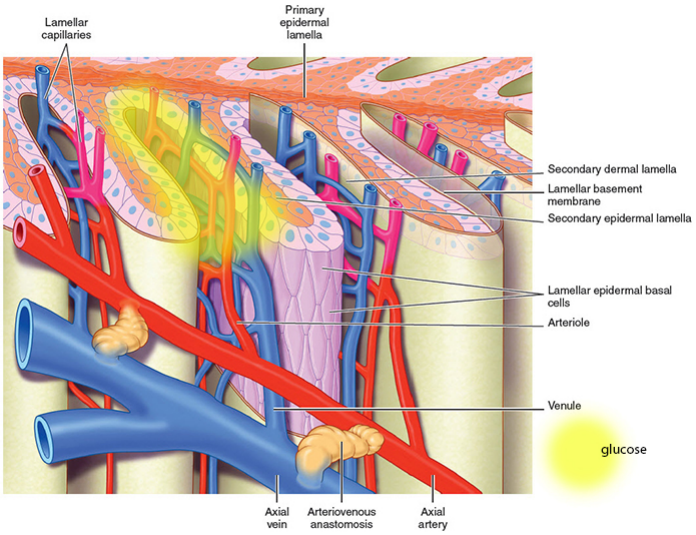

Fig. 10. Lamellar microcirculation diagram. The epidermal lamellae are dependent on their capillary circulation; every secondary dermal lamella (SDL) has capillaries delivering essential nutrients (mainly glucose shaded yellow) to the target organ, the adjacent epidermal basal cells. Lamellar capillaries are within 5 to 7 µm (the diameter of one red blood cell) of all faces of the secondary epidermal lamellae (SELs) and are virtually absent from the primary dermal lamellae and the sublamellar dermis. Copyright "The Illustrated Horse's Foot" by Christopher C. Pollitt, Elsevier, St Louis, Missouri. ISBN 9780702046551.

Thus, the effects of the hoof mechanism in arterial supply and venous return is now well established. The inflow of blood via arteries and outflow via veins is a physiological fact and, as described above, is easy to demonstrate. However, this is only half the full story. Neither of these two (albeit major) components of the vascular system are involved in the critical task of maintaining the viability of the lamellar attachment apparatus. This is the task of the lamellar capillaries and we know virtually nothing of how they operate. Are they subject to the same stop/start blood circulation that cyclic loading and the hoof mechanism imposes on the major vessels of the foot? Bouley, in 1851, surmised that blood pressure alone was sufficient to drive arterial blood through the vascular bed of the foot. Could this be true of the capillary circulation?

Recent research

Our recent lamellar microdialyis studies show that cyclic loading is very important in lamellar perfusion. Microdialysis probes placed directly in the lamellar zone measured real-time glucose concentrations and blood flow. Increases in limb load cycling frequency (particularly walking) caused an increase in lamellar blood flow and a profound increase in lamellar glucose availability.[14] (Figs. 11 and 12).

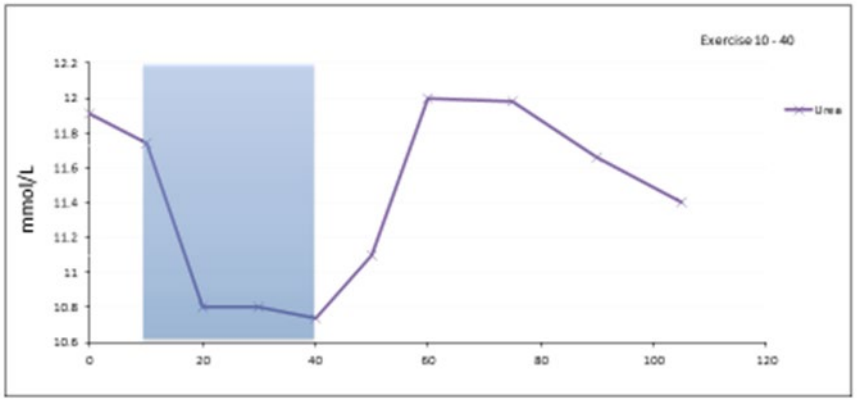

Fig. 11. Lamellar microdialysis measurements of urea (purple line) during 30 minutes (shaded blue) of walking. There is a profound decrease in urea clearance, as is expected during increased blood flow.[14]

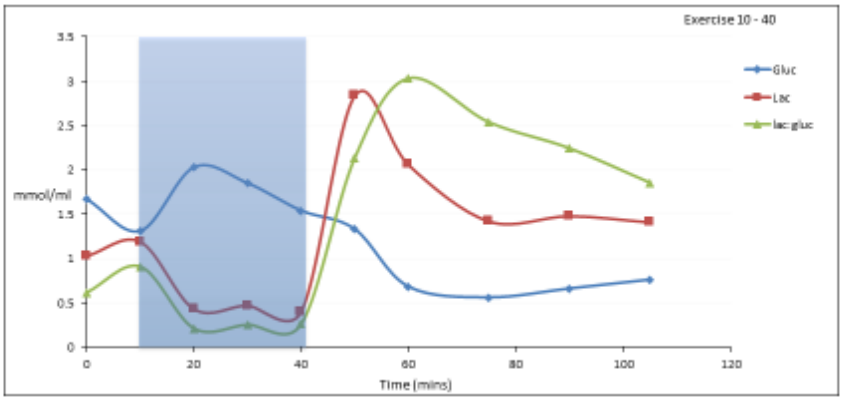

Fig. 12. Lamellar microdialysis measurements of glucose (blue line) during 30 minutes (shaded blue) of walking. There is a profound increase in lamellar glucose delivery.[14]

What if the hoof mechanism was totally abolished – would there be pathological consequences? To abolish the hoof mechanism we immobilised the distal phalanx relative to the hoof wall. Under local or general anaesthesia a locking compression plate T plate was accurately orientated on the midline of the dorsal hoof wall and screwed into the body of the DP phalanx just distal to the extensor process. The distal tip the screws engaged the DP palmar cortex (Fig. 13).

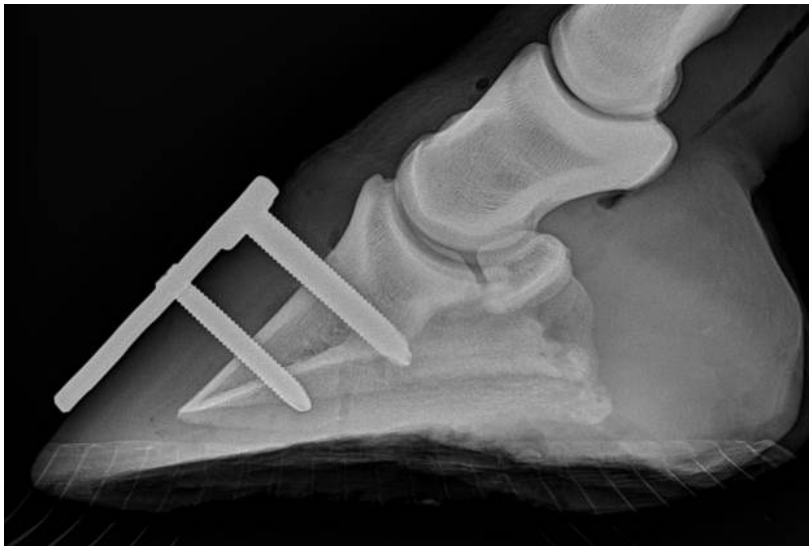

Fig.13. Radiograph of a normal horse foot with the distal phalanx fixed to the dorsal hoof wall with screws and a locking compression plate T plate thus abolishing the hoof mechanism.

On recovery from anaesthesia, the 6 horses received pain relief and were monitored for 7 days. After day 2 there was no lameness at the walk and no further pain relief was required. The horses were humanely euthanized on day 7 and lamellar samples collected, fixed and processed for histopathology.

All the lamellar samples from all 7 horses had lesions of moderate to severe histopathological laminitis (Fig 14). Apart from one unique feature the histopathological lesions were indistinguishable from those induced by 48 hours of experimental hyperinsuliaemia or alimentary carbohydrate overload.[15] There was extensive elongation and thinning of secondary epidermal lamellae (SELs) with universal detachment of lamellar basement membrane. Recognizable capillaries and BM were absent between SELs adjacent to the keratinised axis of the primary epidermal lamella (PEL). Uniquely, in this region, there were numerous dead epidermal basal cells as evidenced by their pyknotic nuclei (arrowheads).

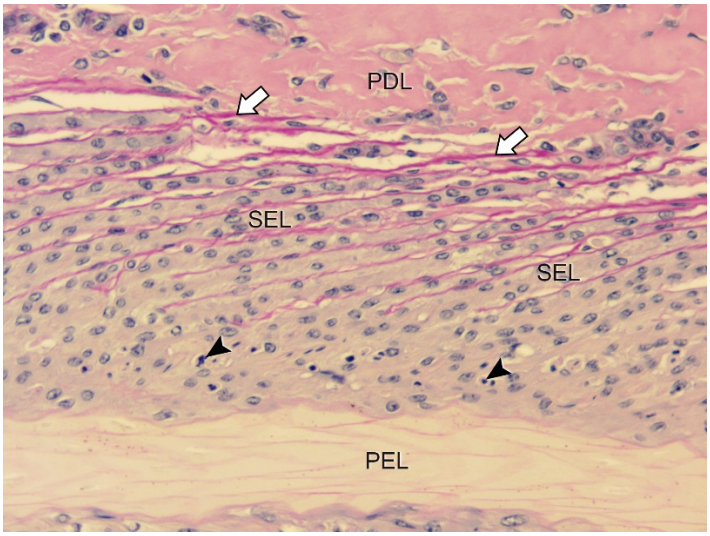

Fig. 14. Photomicrograph of lamellae from a horse seven days after abolition of the hoof mechanism by fixation of the distal phalanx. There is universal elongation and thinning of secondary epidermal

lamellae (SELs). SEL tips are stretched and pointed with universal detachment of magenta coloured basement membrane (arrows). Recognizable capillaries and BM are absent between SELs adjacent to the keratinised axis of the primary epidermal lamella (PEL). Notably, in this region, there are numerous dead (apoptotic) epidermal basal cells as evidenced by their pyknotic nuclei (arrowheads). X20; PAS stain.

Cell death (apoptosis) to this extent is absent in other experimental models of laminitis and occurred only when the hoof mechanism was abolished for 7 days by fixation of the distal phalanx. At some time point during the 7 day experimental time frame, lamellar basal cells, starved of their essential nutrients (probably glucose), became profoundly dysregulated and either died or detached from each other and their underlying basement membrane in a laminitis-like manner. Interestingly, despite extensive laminitis histopathology that would otherwise be associated with significant lameness, the horses were pain free. Presumably, the fixed position of the distal phalanx within the hoof capsule prevented its dislocation downwards and the resultant painful crushing of the solear dermis; the usual scenario with chronic laminitis.

Conclusion

The hoof mechanism, as proposed by the great French veterinary scientist Henri Marie Bouley in 1851[16], now has fresh evidence to support its profound physiological significance. The hoof mechanism operates beyond arteries and veins down to smallest vascular element, the capillaries of the microcirculation. Even the finest thin-walled capillaries appear to rely on cyclic loading to deliver their essential glucose to the lamellar basal cells. Arteries and veins may exist to supply and return blood from the foot, but it is the plethora of lamellar capillaries, constantly supplying vital energy, that maintain the integrity of the lamellar interface. *Par ung seul clou perd on ung bon cheval* could be paraphrased to "For want of a hoof mechanism,[7] the foot was lost".

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