



The biomechanical function of the foot pump in venous return from the lower extremity during the human gait cycle: An expansion of the gait model of the foot pump

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ARTICLE INFO

Keywords:

Foot pump
 Venous return
 Foot-gait model
 Foot compliance
 Pronation

ABSTRACT

The gait cycle has been modelled for energetics and musculoskeletal health and disease, but little has been published in relation to the function of gait as a mechanism in maintaining haemodynamic homeostasis through the lower limb. Blood returns from the lower limb drawn to the heart by the low pressure at the vena cava through hydrodynamic forces. Resisting these hydrodynamics forces are gravitational and frictional forces. The deficit between the forces acting for venous return from the lower limb by hydrodynamics and those acting against antegrade flow, is filled by extrinsic mechanical mechanisms including the respiratory pump, skeletal muscle pumps and the foot pump. The efficiency of the lower limb skeletal muscle pump and foot pump are likely linked to gait kinetics and kinematics. A model is proposed that attempts to expand upon previous gait models of the foot pump as part of the kinetic and kinematic events that occur during gait, whilst also developing the argument that the foot pump needs to be divided into passive-pressure phases, and combined active-muscular/passive-pressure phases during gait. This model suggests that non-weightbearing arch profiles will have little influence on the combined active/passive-pressure phases of the foot pump, while the ability to develop compliance and stiffness within the foot at the requisite periods of the gait cycle is likely to influence foot pump efficiency.

Introduction

There are many ways of modelling blood flow to describe the various characteristics of cardiovascular response, together with the factors that influence it [1]. These models are classified by the features of the system they represent, their complexity, range of application, and their limitations/challenges [1,2]. It has been cautioned that the effects of valves in the veins, the surrounding skeletal muscle pump pressures in the lower extremity, and the changes in intra-luminal and extra-luminal pressure that aid in venous return, need special consideration in cardiovascular modelling [1]. The unique effects of the foot pump seem largely overlooked. This means the cyclical effects of the repetitive weightbearing and non-weightbearing phases that occurs in the foot during gait are, as yet, not being considered as part of modelling the cardiovascular system. This could be because chronic venous disease receives less attention than arterial disease [3] and the importance of the foot pump is being overlooked [4]. Although explanations of the

action of foot pump during gait exist [4,5], no link between the role of foot pump function and the demands of locomotion in gait seem to have been made.

Unlike arterial blood flow which is driven by the pumping action of the heart and the elastic recoil of the artery walls, veins are subjected to far lower blood pressures. Venous flow occurs from areas of high pressure to areas of low pressure through hydrodynamic forces. Blood pressure, between diastolic and systolic in normotensive populations, averages in the aorta at around 95 mmHg [6], but as low as 0 mmHg in the inferior vena cava [7]. Blood flows along this pressure gradient from the aorta back to the right side of the heart. This hydrostatic force is opposed by friction forces and gravitation forces from the anatomy below the heart. For the lower limbs in normal bipedal posture, this is a particular problem overcome largely by using skeletal muscle activity.

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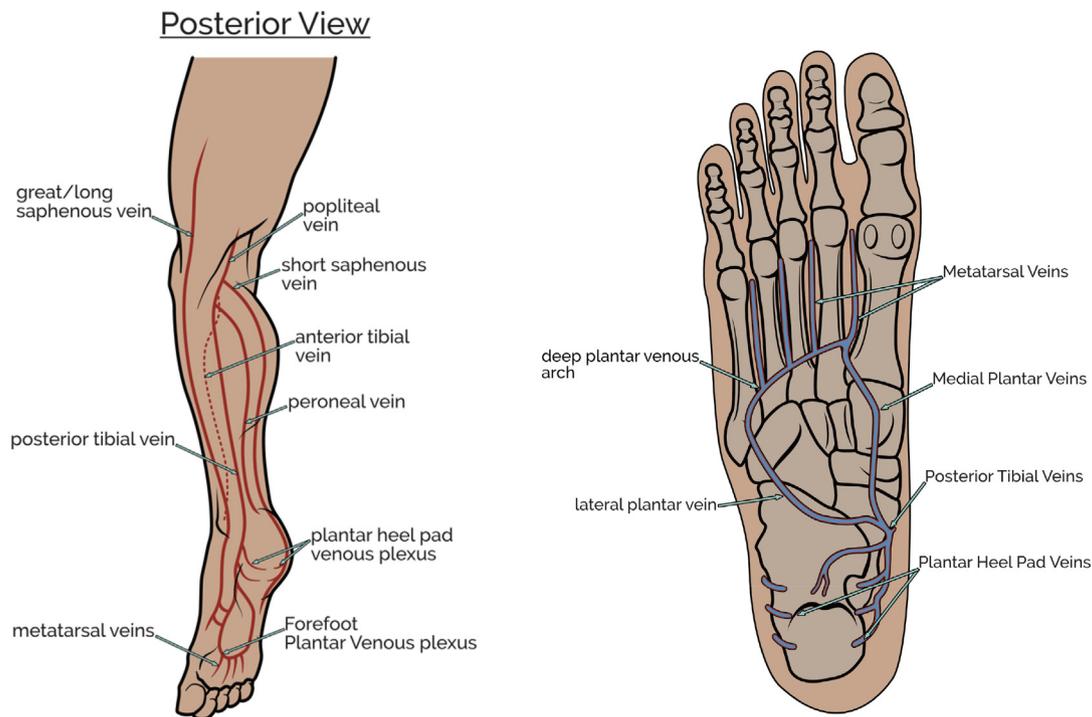


Fig. 1. The primary veins of the leg and foot. Veins are known to be highly variable in the human population, and are usually doubled, but for simplicity are shown singly here. Graphics used with permission of HealthyStep Ltd & A. Horwood.

Venous anatomy of the lower limb

The venous anatomy of the lower limbs is substantially more variable and complicated than the corresponding arterial anatomy [3] (Fig. 1). In comparison with arteries, the smooth-muscle layer in veins is weaker and the wall less elastic but contains proportionately more collagen thus making veins stiffer [3]. The high volumes of blood that veins can readily accommodate in the lower limb venous system is largely due to the elliptical cross section, which allows an increase in volume without increasing circumference pressure; an ability that is essential for muscle pump action [3]. Veins also contain bicuspid valves that play an essential role in returning blood to a superiorly positioned human heart, sited well above the majority of the body's anatomy in erect bipedal posture. Valves are most numerous in the lower leg decreasing in number towards the hip [3], and open passively and unidirectionally towards the heart maintaining antegrade flow by preventing reflux under gravity [3].

Three groups of veins are present in the lower limb. Superficial veins include the reticular veins, which communicate with the great and small saphenous veins. The superficial veins run above the deep fascia, while the great saphenous vein runs in the saphenous sub-compartment lying directly on the muscular deep fascia [3]. The deep veins exist in pairs [4,5] (termed doubled or *venae comitantes*) associated with arteries, which can aide venous return from their compressive pulsation, and from which they usually take their name. There is considerable variation from classical anatomical descriptions [3]. The intra-muscular venous sinuses in the deep calf veins are the principle collecting system for the calf muscle pump [3]. Perforating veins link the superficial and deep veins together, with on average 64 of them found between the ankle and the groin [3]. The superficial veins drain through the perforating veins towards the deep veins through valves, orientated to prevent reflux towards the superficial veins [3–5]. The exception is within the foot where valves if present, orientate for flow from perforating veins towards superficial veins [3,4,8]. In the heel the veins mainly run transversely across the heel so that on weightbearing the blood is squeezed towards the margins and not anteriorly [8].

The foot veins have been described as having five systems [4,5]; (i) superficial veins of the plantar foot; (ii) deep veins of the plantar foot; (iii) the superficial dorsal plexus; (iv) the marginal veins running along the foot's borders; and (v) the perforating veins. The superficial veins of the plantar aspect are known as Lejars' venous plexus, a network of tiny veins thought to be of little clinical interest [4,5]. The deep veins consist of a doubled deep plantar venous arch running from the proximal end of the first intermetatarsal space to the base of the fifth metatarsal (accompanying the deep plantar arterial arch) with a consistent proximally oriented valve [4,5]. This receives the deep metatarsal veins and surrounding muscular veins [4,5]. The medial plantar vein is thin and short containing a few proximally orientated valves running along the medial border of the longitudinal foot 'arch', receiving blood from the adjacent abductor hallucis, flexor digitorum brevis and plantar quadratus muscles [4].

The lateral plantar vein is more significant, constantly doubled, and is large at 2 mm width with fusiform swellings resembling venous sinuses [5]. Proximally directed valves are present, and the vein is continuous with the lateral part of the deep venous arch joining the medial plantar vein at the calcaneal confluence to form the posterior tibial vein [5]. It receives blood from the calcaneal veins, the adjacent plantar muscles and the lateral marginal vein. A secondary deep venous arch is also present deep in the quadratus plantae or within the complex local connections, which may act as a blood reservoir [5].

The superficial dorsal plexus presents a continuity of the superficial veins of the leg and ankle, separated from the marginal veins and dorsal arch by fascia similar to that found supporting the great and small saphenous veins [5]. The medial and lateral marginal veins are continuous with the great and small saphenous veins respectively [5]. The dorsal arch lies above the proximal ends of the metatarsal bases, receiving blood from the dorsal veins and perforating veins from the deep plantar veins [5]. The perforating veins are either valve-less or have deep to superficial orientated valves [3,4,5,8]. The calcaneal and cuboidal veins are consistent perforators that join the lateral marginal vein at the perimalleolar plexus [5].

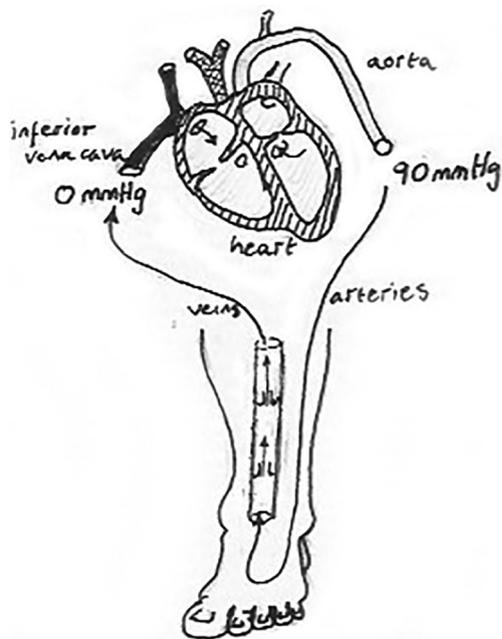


Fig. 2. A simple schematic of blood flow via hydrodynamic forces. The pressure difference draws blood through the veins and out of the lower limb assisted by unidirectional valves, but this force is insufficient in maintaining haemodynamics in gait and stance on its own.

Mechanics of venous valves and pumps

The venous bicuspid valves work by antegrade transvalvular pressure gradients, closing on reversal of the pressure gradient above and below the valve, so as the pressure becomes lower below the valves, there is a short reflux period of retrograde flow until the flow becomes sufficient to close the valve [3]. Decreasing hydrostatic forces generated by decreasing blood pressure towards the vena cava draws blood towards the right side of the heart through the valves, which prevent backflow [3] (Fig. 2). Supplementary to hydrostatic force are the actions of the respiratory pump [9,10] and skeletal muscle or venomuscular pumps [4,5,10]. The respiratory pump works as we breathe. During inspiration, the diaphragm moves downward causing decreased pressure in the thoracic cavity, while increasing pressure in the abdomen. This allows the blood to move from the higher pressure of the abdominal cavity towards the lower pressured thoracic cavity. On expiration, the pressures reverse but the valves prevent backflow [9,10].

The stiff venous walls, and valvular action through venomuscular pumps created by myofascial compartments, prevents blood accumulation in the lower extremities [3–5]. The muscular pumps of the abdomen, thigh, calf and foot are dependent on constraining deep fascial connective tissue laminae creating high pressures within muscular compartments to produce antegrade flow through bicuspid valves [3–5]. When muscle contracts, it tightens the connective tissue compartment, raising pressures on veins passing through the muscle or their compartment. In the posterior compartment of the calf, intermuscular veins are numerous and are arranged in spirals through which blood is expelled during longitudinal muscle excursion, known as volumetric pumps [4,5]. Extra-muscular venous tracts found in the deep posterior muscle compartment (tibial and peroneal veins) and anterior compartment (tibial vein) are subjected to compression against stiff structures like bone or tensioned aponeuroses creating peristaltic pumps, which while less efficient are still effective [4,5]. Pressures can exceed 200 mm Hg in the calf, making it the most efficient lower limb muscle pump, causing pressure rises in all lower limb veins [3,5]. The proximal valves open under the pressure created in the veins, and blood is ejected into the next muscle compartmental section of vein. On muscle

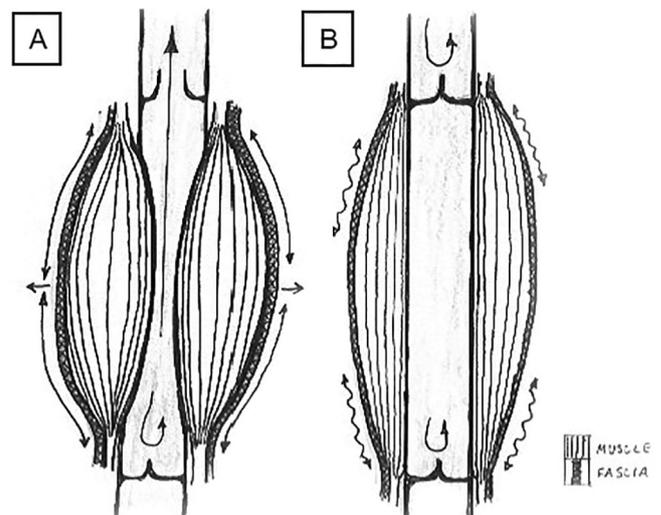


Fig. 3. The principles of the skeletal muscle pump. On contraction veins passing through muscles or muscular compartments are squeezed by a combination of muscle contraction and fascial compartment pressure increase (A) driving flow through valves superiorly and closing veins inferior to the muscle/compartment. This occurs regardless of the type of contraction. On relaxation of the muscles, fascial pressures decrease as fascia and muscle return to resting length. The valves preventing retrograde flow under gravity (B).

relaxation, the venous pressures fall in the compartment with decreasing fascial tension, causing the valves to close under reflux flow, thus preventing retrograde flow [3,4,5,10] (Fig. 3). The superficial veins are then able to flow via aspiration through the perforating veins towards the deep veins [5].

The foot pump

The foot was once thought to have little influence on the physiological return of blood from the leg, and venous return from the foot was thought to occur under muscle pump activity only [11–14]. The work of Gardner and Fox [11,15] reported on the role of the foot in venous return, describing and proposing a compression hydraulic pump. The pump requires compliance of the soft tissues surrounding the foot veins, which are embedded in dense fibro-fatty tissue [11,13] within deep fascia accompanied by arteries [4,5]. Deep fascia has a significant role in venous return from constraining the muscles and vessels [4,5,16]. Fox and Gardner reported that squeezing the foot in a supine position increased venous flow in the femoral vein (a test used for patency of femoral veins); that venous pressure rose on standing; and described extensive pooling of blood in two medial deep plantar veins in non-weightbearing vertical posture which emptied into the calf when weightbearing on the heel and metatarsal heads [11]. Binns and Pho hypothesised that the venous foot pump was a result of intermuscular compression of the lateral plantar vein due to muscular activity only [12]. Although the lateral plantar vein is suggested to be particularly important as it is larger with fusiform sinuses, it is also more likely to weightbear than medial veins [5]. Recent studies have confirmed that venous throughput is increased through the tibial, peroneal and popliteal veins on weightbearing when compared to lying supine with the foot loaded with force [13]. Both intrinsic muscle activity and weightbearing compression of the foot have been reported as being equally significant in expelling blood into the deep veins of the calf [13].

Gardner and Fox reported that standing on a narrow transverse pad under the arch only empties the veins in the immediate arch area, not from the entire plantar plexus of veins as occurred on loading the plantar aspect of the heel and forefoot [11]. This suggests that the primary weightbearing sites of heel and forefoot are used as a

compression pump with ground reaction forces (GRF) acting on the deep plantar veins at each step [11–13] with proximally and superficially orientated deep and perforating vein valves respectively preventing backflow from compressed deep foot veins [3–5,8]. Each compression of the foot on weightbearing has since been recorded to produce 33 ml of blood passing into the popliteal vein [13]. As expected from the description of the valves opening toward the superficial veins in the foot, a greater volume is recorded as being expelled into the superficial veins than the deep veins [13].

Ankle kinematics has a significant role in calf venous return and works in synergy with the foot pump [5,11]. Ankle dorsiflexion during weightbearing, controlled by triceps surae eccentric contraction, is reported to propel blood upwards through the deep veins of the calf, emptying them [5,11], and is referred to as the distal calf or piston pump [5]. Ankle plantarflexion under triceps surae concentric contraction is reported to draw blood out of the superficial veins of the calf via the perforating veins penetrating the deep fascial calf envelope, delivering the blood to the deep calf veins, which then propels the blood upwards [5,11] producing a proximal calf pump [5]. It is likely that activity in the deep muscle compartment of the calf is also involved in these pumps [4,5].

The plantar intrinsic muscles of the foot play a significant role in the foot pump equal to the compressive element [13], maintaining a significant muscle pump output. It appears that the combined action of stance phase weightbearing pressures on the foot, assisted by the plantar intrinsic muscle activity during the late midstance and heel lift, act as a combined active–passive foot pump driving venous flow towards the calf. Ankle kinematics under calf muscle activity acts in unison with the foot pump, significantly increase the efficiency of venous drainage of the lower limb [11–13]. The principles of the foot pump have been used clinically in reducing deep vein thrombosis post-operatively in the lower limb [17–19]. However, it appears such artificial foot compression devices are not as effective in producing venous flow as the natural foot pump, possibly from an inability to create adequate changes in foot profile necessary to stress the foot vein plexuses [14], and do not involve active muscle pumps [20].

Implications to Gait Energetics and Foot Function

Human plantigrade gait makes humans energetically efficient walkers, although digitigrade human foot posture in gait does not increase energetic costs [21]. Compared to digitigrade walking, plantigrade foot posture of human gait is advantageous in generating more pendular exchange of kinetic and potential energy as well as reducing average angular momentum forces around the ankle [21]. Although there are many competing models of foot function, most have concentrated on mechanisms that present and contrast ‘normal’ and ‘abnormal’ motion to explain musculoskeletal pathologies [22,23] and gait energetics [24,25]. Kinematic data has confirmed that the pain-free foot is a compliant multi-articular structure that relies on variable ranges of motion in all three-body planes [26]. Foot function fulfilling a haemodynamic role as part of locomotion remains largely overlooked in locomotion models, despite evidence that peripheral arterial disease reduces vertical and anterior-posterior components of GRF [27]. Haemodynamic function may be an important factor in the evolution of foot anatomy, even if not as significant a determinant as locomotion. Consideration of the haemodynamic role of the foot being dependent on a compliant structure to permit the action of a hydraulic pump at contact-to-midstance phase, followed by muscle activity for generating a muscle pump when stiffening the foot, may help to explain some foot anatomy and kinematic variation that still permit these events to take place. Clarification of terms like ‘hyperpronation’ become necessary [28], as the amount of deformation on weightbearing as part of foot pronation may be essential to maximise the foot pump function; thereby perhaps redefining what is meant by ‘normal healthy foot motion’.

Expanding the gait model for the venous foot pump

Previous attempts to describe the foot pump’s activity in gait [5] have simplified and slightly erroneously described the kinematics. The foot pump appears to be a mixed passive-active mechanism [13] that functions in unison with the venomuscular calf pumps. The passive elements involve plantar foot loading associated with compliance of the anatomy to allow the venous plexuses within the foot to become pressurised under compression of soft tissue against the ground and stiffer anatomy like fascia and bone. This creates a pressure-driven hydraulic pump. The active mechanism of the foot pump involves activation of the plantar intrinsic muscles during the period of foot stiffening in the later stages of stance phase. From consideration of the usual human gait cycle events, it is possible to construct a hypothetical model of preferred action for both the active and passive elements of the foot pump, operating at times separately and concurrently, to achieve a theoretically desirable function and reflect these events to locomotive needs.

Ricci’s model of foot pump activity in gait, starts during the swing phase with activation of the anterior muscle group compressing the anterior tibial vein as the foot dorsiflexes throughout the swing phase [5]. The foot does not normally adopt a dorsiflexed position in swing phase [29,30], although it becomes less plantarflexed to aid ground clearance and makes heel contact at 3–5 degrees plantarflexed [30]. Muscle activation of the primary ankle dorsiflexor, tibialis anterior, occurs concentrically just before toe off and during initial swing, but decreases in swing phase to activate just prior to heel strike and continues contacting eccentrically until forefoot loading [29,30]. It unlikely that significant tibial vein muscle pump action takes place in swing phase, but in the period from just before heel strike to just after forefoot loading when tibialis anterior activity peaks [29,30]. However, variations in tibialis anterior activity have been recorded during mid-stance, suggesting anterior tibial muscle action venous drainage is not predictable [30]. It is noted that the anterior crural fascia is stiffer than the posterior fascia, and this may be significant to the anterior pump [31]. During swing phase, it seems likely that venous blood will pool in the plantar venous plexuses, as described in feet in vertical non-weightbearing [11,13]; a process possibly enhanced by gravity interacting with increasing centrifugal forces during knee extension that occurs during the swing phase (Fig. 4).

The foot pump is likely initiated at the beginning of the stance phase, which usually starts with a heel strike. The heel pad has a rich supply of blood vessels, with small and larger vein plexuses found within the chambers of the heel fat pad [8], which should fill like the

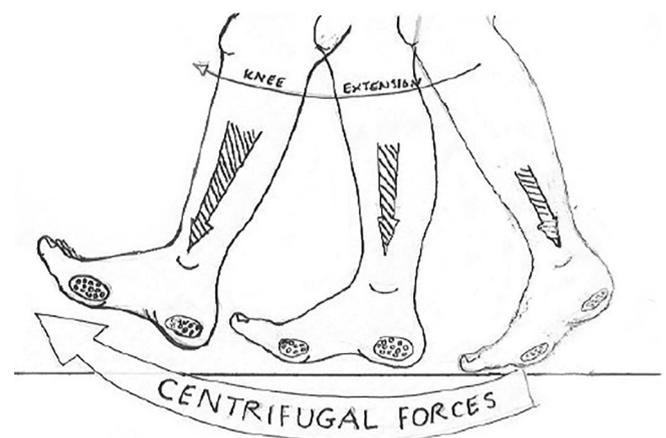


Fig. 4. During swing phase, gravity is able to freely act on venous flow resisted only by hydrostatic forces and the bicuspid valves. Blood pools in the foot under the influence of gravity (hashed arrows) and increasing centrifugal forces as the knee extends during late swing phase.

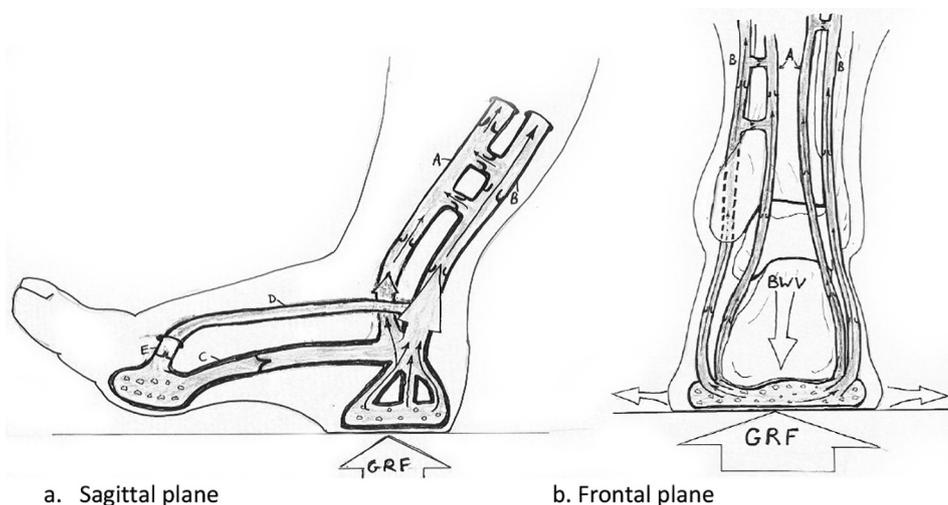


Fig. 5. Simplified diagrammatic representation of the foot and calf pumps in gait. At heel strike (4a & b), compression of the heel fat pad veins under the influence of forces from the body weight vector (BWV) producing ground reaction forces (GRF) that passively eject blood from the heel towards the deep (A) and superficial (B) calf veins. The midfoot and forefoot veins (C,D,E) are not pumped at this gait phase. The veins of the heel fat pad are orientated to run medially and laterally to the heel margins where they run to the superficial veins of the calf assisted by shear forces in the heel soft tissue spread than occurs under compression from GRF(4b).

rest of the foot during swing phase. On heel strike, the heel fat pad will undergo pressure deformation progressively from posterior lateral to anterior medial creating heel compression and shear forces. These forces will propel blood from the deep vein plexuses of the heel fat pad medially and laterally through the transversely aligned veins to the heel margins [5], and from there primarily through perforating veins to the superficial veins and the confluence of the lateral plantar vein as it becomes the posterior tibial vein [5]. The transverse vein orientation prevents blood from flowing anteriorly reinforced by the valve alignment towards the superficial veins [8] (Fig. 5). Heel compression is part of the passive phase foot pump driving the blood from the foot, abruptly raising the venous hydrostatic pressure in the deep and superficial veins of the leg with each step. The presence of venous pumping action within the heel fat pad has been proposed as an explanation, in part due to the high hysteresis found during heel pad deformation found in *in vivo* compared to *in vitro* studies [32–34].

During forefoot loading, compression of the plantar forefoot soft tissues occurs in conjunction with changes in the foot shape/profile during ambulatory loading with, usually, a reduction in medial longitudinal arch height [35–37], foot length, and width [37,38]. These changes in foot profile, associated with increased foot pronation, may allow more extensive soft tissue deformation under load than a rigid structure, which would only result in superficial soft tissue deformation. Compressive loading forces acting on the forefoot initiate profile changes that create vein pressures directing flow towards the superficial and marginal foot veins through the perforating veins, and proximally through the deep veins towards the deep and superficial veins of the leg [4,5]. After forefoot loading is achieved, the foot enters the early midstance period and therefore will be in single limb support. During this period, pressure starts to decrease gradually on the heel and continues to do so throughout the midstance phase as the body's centre of mass moves anteriorly over the foot [39,40]. The passive hydraulic pump phase will have peaked but continues in isolation until the centre of body mass lies above the centre of the foot, and the body's centre of mass has risen to its highest point during the gait cycle [21,24]. This moment of 'absolute midstance' is the point completing the isolated passive compression stage of the foot pump (Fig. 6).

The plantar intrinsic muscles are inactive in contact and early midstance phases [41], with only tibialis posterior of the deep posterior muscle compartment of the calf demonstrating significant but variable peak activity during early midstance [42]. Just before absolute midstance, the triceps surae start to activate, becoming increasingly active eccentrically throughout late midstance phase [43,44], initiating the calf muscle pump as the ankle dorsiflexes. Just after the absolute midstance moment of gait, the plantar intrinsic muscles start to become active, stiffening the foot for the propulsive phase of gait [41] and

engaging the active foot muscle pump. The eccentric calf muscle pump activity accompanying ankle dorsiflexion as the body proceeds over the foot, drives venous blood up the calf's deep veins, which is then re-supplied via the foot pump from below (Fig. 7). From absolute midstance, the foot pump becomes a combined passive-active pump of plantar pressure and intrinsic muscle activity. It is possible that the ankle dorsiflexion combined with the stiff crural fascia is responsible for compressing the anterior tibial vein against the bone during this phase of gait.

Foot stiffness increases via intrinsic muscle action [41], and foot profile deformation slows as it approaches its peak ambulatory range of deformation [35], being most changed just prior to heel lift [45]. The foot transforms from a compliant structure to a stiff propulsion platform during the period from absolute midstance to heel lift; changes that are necessary for ideal gait energetics [21,24].

At terminal stance, heel lift ends compression on the heel. The heel fat pad venous plexus is now free to fill with blood again, resetting the mechanical properties of the heel for the next step. Forefoot pressures, which have been increasing during the midstance phase as the body's centre of mass proceeds towards the forefoot, will peak [39,40] in conjunction with peak intrinsic foot muscles activity [41], assisted by concentric plantarflexion action of the calf muscles [43,44] and the high GRF this generates [40]. The combined pump mechanisms continue to drive blood superficially and proximally towards the tibial, peroneal and saphenous veins. Shortly after heel lift, the compressive forces start to reduce as forefoot GRF and surface contact areas diminishes [39,40] (Fig. 8). The venous foot pump's activity will cease prior to toe-off. The cycle then re-starts, with blood pooling in the foot's venous plexuses during swing phase.

If this proposed gait model of the foot pump is correct, then increased venous pressures should be initiated in the proximal veins of the lower limb and peak during periods of high GRF experienced at heel contact, forefoot loading, and at heel lift [24,39,40]. Steady but lower venous discharge should occur throughout the early midstance phase, increasing after absolute midstance until heel lift. Heel veins should only discharge until heel lift, but forefoot veins should demonstrate peak emptying during the late midstance/early terminal stances of gait as intrinsic muscle activity [41,44], foot stiffness [41] and forefoot plantar pressures peak [40]. The model suggests distinct passive and active-passive phases of the foot pump with predictable peaks in venous flow at heel strike, forefoot loading, and just prior to and during heel lift, concurrent with vertical and anterior/posterior GRF peaks. Venous refilling of the foot should occur during swing phase, and possibly also within the heel pad veins after heel lift. The venous flows predicted in the model are outlined in Fig. 9.

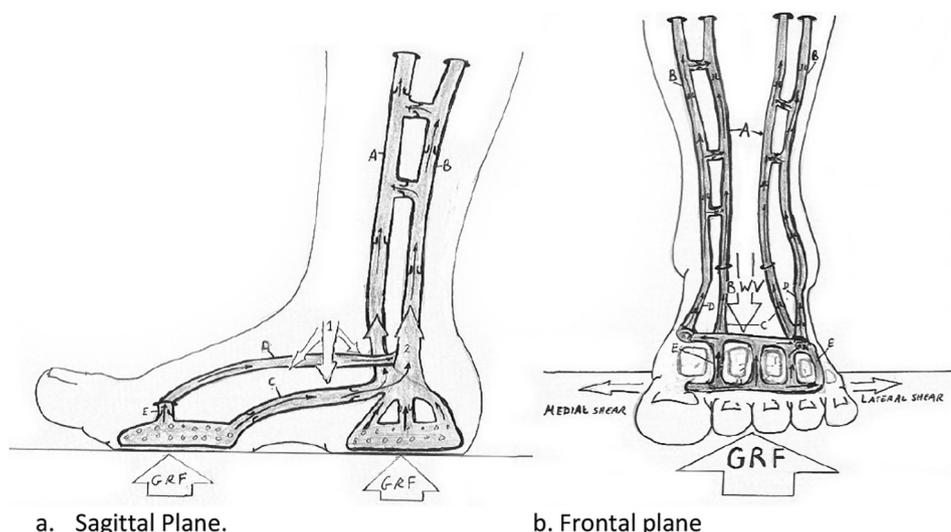


Fig. 6. At forefoot loading both the heel and forefoot venous plexuses are compression by GRF, driving blood towards the superficial/marginal veins of the foot and calf (B,D) through perforating veins (E) and proximally towards the deep calf veins (A) from and through the deep veins of the foot (C). The foot pump remains a passive mechanism due to foot compliance under load with arch drop and foot splay (represented by arrows at 1) which drive the foot pump (arrow 2) alone until around true midstance.

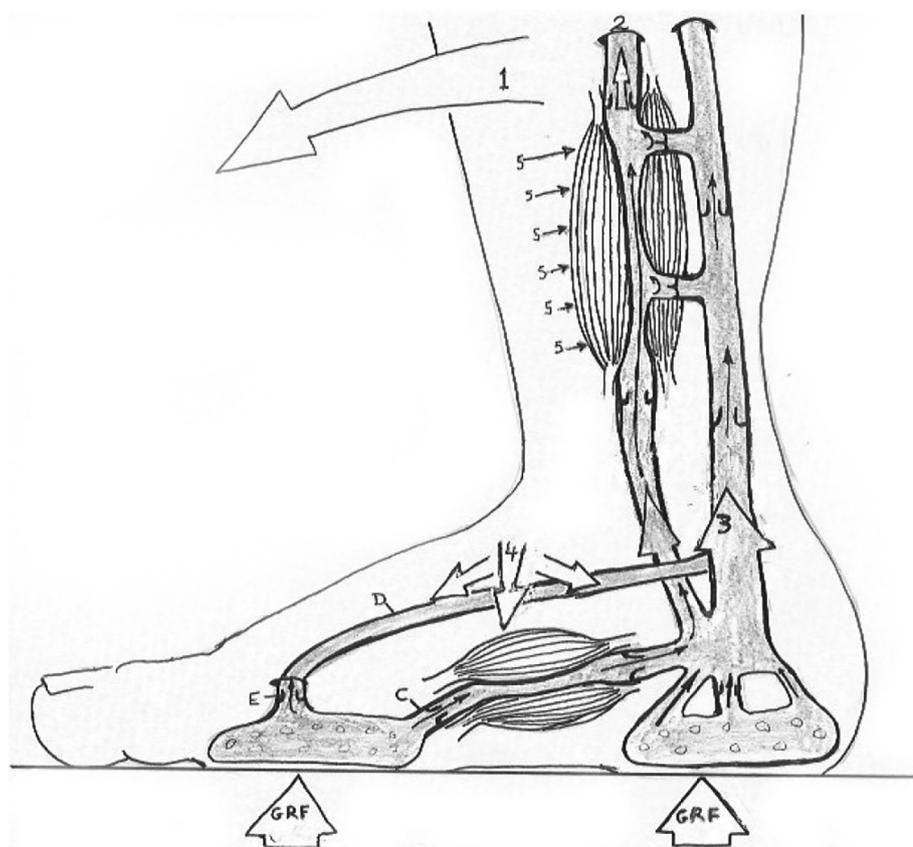


Fig. 7. After absolute midstance calf and intrinsic foot muscles become active, initiating the active calf and foot muscle pumps respectively. With eccentric calf dorsiflexion (arrow 1) blood is driven from the deep veins (A) up the leg (arrow 2) assisted by compression against the osseous structures (arrows 5). The foot pump becomes a combination of active and passive mechanisms driving blood into the calf (arrow 3). The foot continues to ‘flatten’ and spread until just prior to heel lift (arrows 4) although the rate of compliance decreases. Venous flow is increased proximally through perforating veins (E) towards marginal and superficial veins (D), and through deep forefoot veins (C) proximally.

Consequences and discussion of the gait model of the venous foot pump

This model raises the possibility that the foot plays an important concurrent haemodynamic role outside the locomotive roles of elastic energy absorption from initial compliance followed by stiffening to develop a stable platform from which to propel. This may have implications as to how we approach evolution and function of the foot beyond locomotive considerations, as it presents another reason why plantigrade foot posture is an advantage. Variation in foot motion during gait has been identified, and discussion has been made that research has struggled to identify those variations in foot profile that predictably lead to injury [26,46]. If the foot’s initial loading

compliance and later active muscle stiffening is essential for the venous foot pump to operate efficiently, then it might be that initial compliance accompanied by passive profile changes to around absolute midstance, followed by active muscle and foot profile stiffening might also be a prerequisite for energetic locomotion and injury avoidance. It is possible that the foot’s non-weightbearing profile, or degree to which it pronates is irrelevant, and only its capacity for compliance and stiffening should be considered. Achieving the process of compliance and stiffening is likely a variable process within the population, as would be expected in the circumstances of known high variation in both joint motion [26] and muscle activity [29,42–44] seen in humans; let alone the large range of anatomical variations reported in lower limb anatomy. Therefore, recording the profile of the foot in stance and gait

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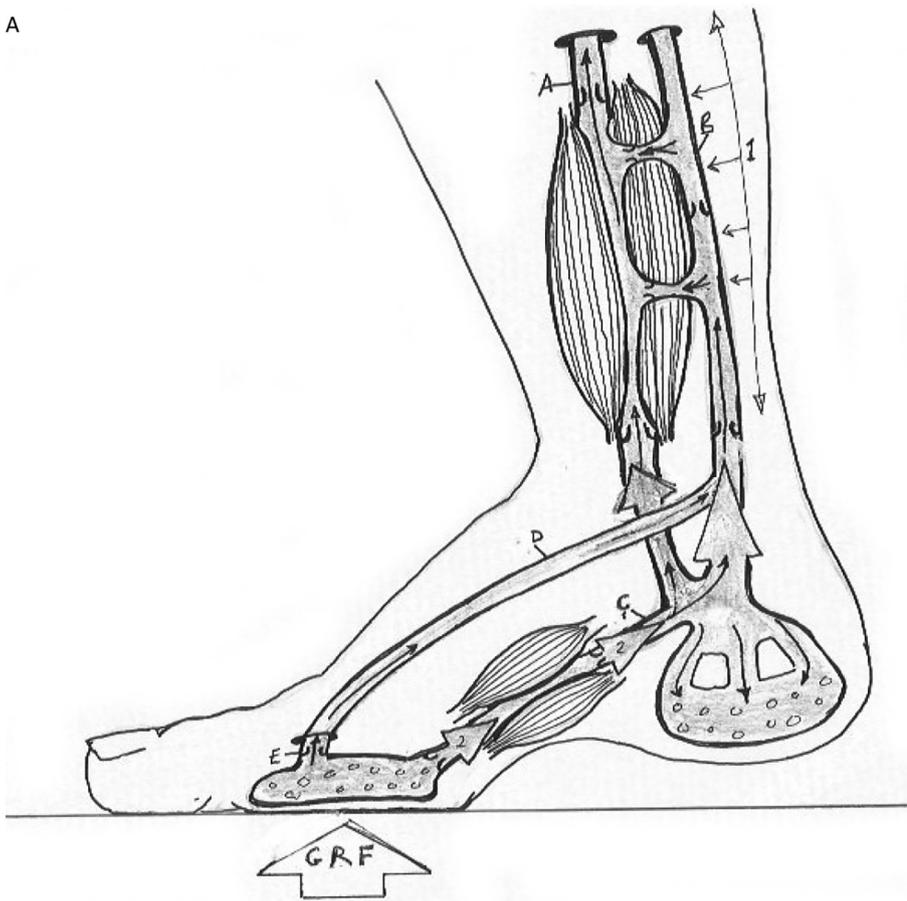


Fig. 8. At heel lift into early terminal stance, the forefoot is at its stiffest acting as a solid platform to achieve effective propulsion. The calf muscle pump undergoes concentric contraction driving blood from the superficial veins into the deep, possibly assisted by compressive tensions set up in the fascia from calf muscle volume expansion (fine line arrows 1). The passive element of foot pump is now reliant of fore-foot compression from GRF of the ankle plantar-flexion moment, while the active foot muscle pump is highly active driving blood through the foot veins (C,D,E) to refill the calf veins (A,B) (arrows 2).

might only be relevant if it can be linked to compliance and stiffening dysfunction in energetics of locomotion, haemodynamic efficiency, and the development of pathology.

Gait models of the foot pump suggests that different gait styles and/or perturbations in gait may change foot pump efficiency [5]. Such changes could result from; [i] the effects of alteration of contact forces or impact points from stride length variations; [ii] the effects of an

initial forefoot contact compared to heel contact; [iii] foot/calf muscle weakness; [iv] loss of ankle range of motion; [v] frontal plane excursions changing plantar pressure loading patterns; and [vi] changes in the compliance and stiffening periods within the foot.

Gait models and previous studies suggest several interventions that could be used to improve venous flow in the healthy as well as venous-insufficient populations. The model and existing data suggests that

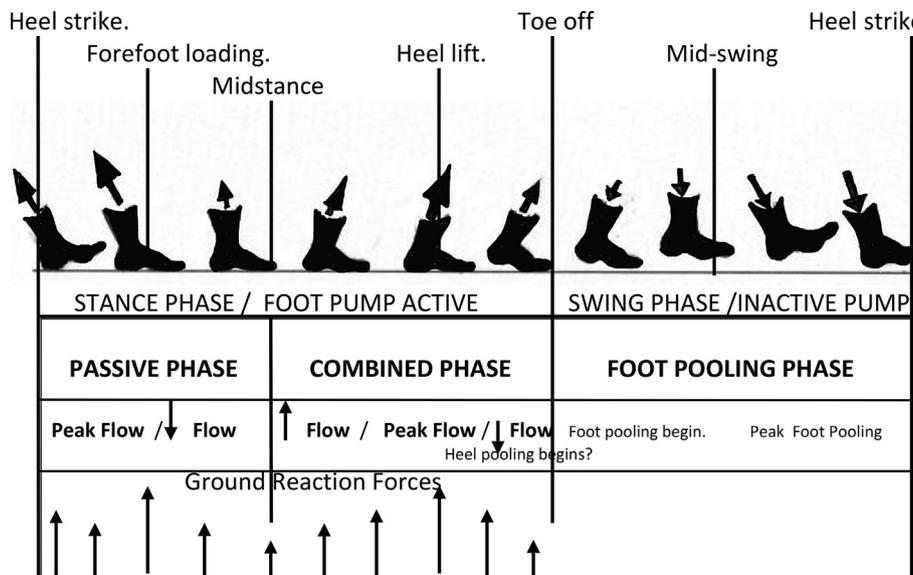


Fig. 9. Graphic representation of the proposed phases of action of the foot pump and venous flow rates in relation to ground reaction forces and gait phases in an expanded gait model of the venous foot pump.

regular weightbearing exercise will keep the foot and calf muscle pump active [13,47], supplemented with intrinsic foot and calf exercises [13,48,49]. A role for calf exercises has already been reported in venous insufficiency management [49]. Gait parameters, ankle and foot kinematics, and muscle strength might need to be considered and assessed clinically to enhance the effectiveness of the foot pump on venous return, presently something not routinely performed in cases of venous insufficiency.

Declaration of Competing Interest

The author declares that he is funded by Healthy Step (sensograph): id on a consultative basis. Healthy Step Ltd have been designing a range of compression hosiery in conjunction with AMH to aid venous return in the foot.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.mehy.2019.05.006>.

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