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# The biomechanics of leg ulceration

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**Research performed in the late 1960s, using $^{24}Na$, suggested that the perfusion of skin and subcutaneous tissues is critically dependent on the relationship between capillary ($P_c$) and tissue pressures ($P_t$). Perfusion changes differed significantly between controls and patients with venous disease and the differences could be interpreted as evidence that $P_t$ remained high in venous diseased patients. From this starting point, a biomechanical theory for the aetiology of venous ulceration was developed and tested by measuring skin elasticity, limb cross-sectional area and laser Doppler flux. The results confirm that, modelled as a two-compartment system (vascular and interstitial fluid), forces can be demonstrated sufficient to cause intermittent capillary closure and subsequent reperfusion injury. These forces are maximal in the gaiter area, the site of most leg ulcers.**

In 1970, a great deal was known about leg ulcers, but there was no all-encompassing theory to account for even uncontroversial facts. Ulcers were often associated with venous disease; however, other dependent ulcers occurred. The gaiter area was the most prone area and perforating veins appeared important. Elevating the legs, treating venous disease, support hose and bandages were all accepted methods of treating the ulcers. During the 1980s, in an attempt to explain the phenomena of venous ulceration, two rival hypotheses were developed. The first was that of Browse and Burnard *(1)*, who demonstrated that fibrin cuffs were associated with venous hypertension and were identifiable in legs at the stage just before ulceration. Their theory suggested that fibrin acted as a diffusion barrier to oxygen and tissue necrosis occurred as a result. A critique by Michel *(2)* looking at this from a purely physiological viewpoint shed serious doubt on the theory, leaving the field free for Coleridge Smith *et al. (3)* to develop their 'White Cell Trapping Theory'. This group of workers elegantly pieced together the final common pathway of ulceration noting the margination of white cells, the release of various proteolytic enzymes, oxygen-free radicals and eventual cellular death. However, neither of these theories, despite their impressive sallies into the microscopic changes, could account for many of the established clinical observations listed above. Particularly, they could not explain why the foot was usually spared and the gaiter area not. Thus, as well as explaining well-known observations, any alternative theory of venous ulceration had also to account for these new biological findings.

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## Preliminary experiments

The inspiration that guided me into this difficult field came from two sources. The first was the late D A MacDonald. In the late 1960s he and Wormersley were at the peak of their achievements in understanding blood flow in arteries *(4)*. MacDonald firmly believed that, given the complexity of modelling and measuring arterial flow patterns, conventional methods would not be possible when investigating the venous system. The second source of inspiration came from working as a technician in King's College, London, using radio-labelled sodium in guinea-pigs *(5)*. This work, which was asthma related and mostly to do with capillary permeability, gave the necessary insight to circumvent MacDonald's worries. A novel approach was adopted using sodium-24 ($^{24}Na$) to measure the effect that the venous system had on the tissues, rather than measuring pressure and flow in the veins themselves. When a depot of $^{24}Na$ is injected into tissues, the washout curve of the isotope clearance takes an exponential form ($Q_t = Q_0\ e^{-kt}$).

The clearance constant *(k)* of this equation is the measure of how rapidly the clearance occurs. This technique in controls and patients with venous disease showed *(6)* (Fig. 1) that tissue clearance was inversely related to venous pressure, was influenced by impaired venous function and was a potential method of monitoring treatment.

More importantly it showed that by increasing total tissue pressure ($P_t$), $^{24}Na$ clearance increased until capillary pressure ($P_c$) was exceeded, at which point $^{24}Na$ clearance fell. Interestingly, by using a hydrostatic stocking, a device similar to a G-suit *(7)*, it was possible to maintain $^{24}Na$ clearance (a marker for the clearance of oedema/tissue fluid) constant in all positions. Although neither $P_t$ nor skin blood flow had been accurately measured (except indirectly by measuring skin temperature), the following hypothesis evolved *(8)*:

- Patients with deep venous thromboses (DVT) behaved as if the oedema in their legs worked as a hydrostatic stocking, ie $P_c$–$P_t$ remained constant. Therefore . . .
- $P_t$ must remain high in DVT patients much of their waking hours.
- The outer integument, the skin containing this pressure must always be stressed. Therefore . . . (Hooke's Law)
- The skin could eventually ulcerate, ie the elastic limit of skin may well eventually be exceeded.

In other words, venous ulceration had a primary mechanical cause.

### Laplace's and Hooke's laws combined

Unfortunately, the methodology necessary to test this hypothesis was either too early in its development or simply non-existent. However, by the late 1980s, new investigative techniques had become available and the first part of the hypothesis was proved correct by Pflug *et al. (9)*. This team showed that patients with venous disease demonstrated high compartmental pressures which were posturally related. At the same time, convincing physiological work by Lansman *et al. (10)* drew attention to the importance of the differential pressure between the tissue and capillaries. Using this new evidence, and by combining Laplace's formula with Hooke's, it was possible to produce a second version of the mechanical theory *(11)*. This attempt took the mechanical theory one stage further by demonstrating that the forces in the gaiter area differ substantially from other parts of the leg. It was proposed that stress (produced by oedema, venous disease, etc) could eventually cause sufficient strain on the skin to allow disruption (as per Hooke's Law). However, invoking the Laplace relationship, the anatomy and compliance of the structures in the lower limb made it more likely that high stress levels would be experienced in the inextensible tissues with small radius at ankle level. This version was immediately and correctly challenged by Scott and Coleridge Smith *(12)*, who argued that these forces were still not sufficient to break skin which, like leather, was extremely strong. However, by this stage the theory had been taken one stage further *(13)* and the importance in engineering terms of what are known as stress concentrations was pointed out. These are regions in non-uniform structures which in effect bear an unreasonably high proportion of the stresses acting on the structure.

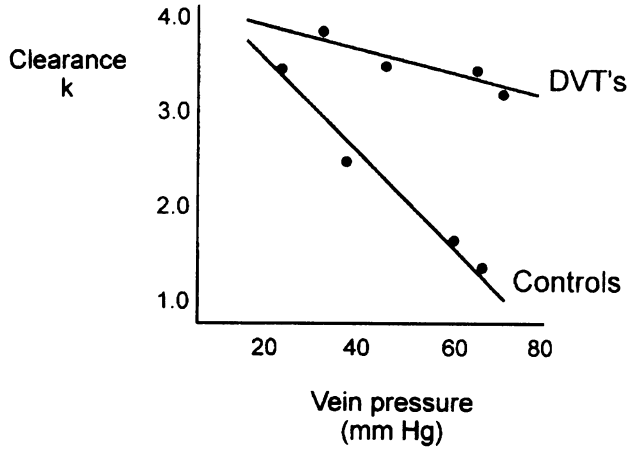


*Figure 1.* Clearance ($k$) of $^{24}Na$ as a measure of venous function with increasing venous pressure produced by postural change; clearance reduces dramatically in controls but not in patients with venous oedema.

## Stress testing skin

In order to test this second mechanical hypothesis, it was necessary firstly to measure *in vitro* skin elasticity in healthy and diseased states; secondly to determine whether the geometry of healthy and diseased limbs differed and, finally, to assess whether the stress changes produced by stretching had an effect on skin viability.

Starting with skin elasticity, using the technique of uniaxial skin extensometry of Mourad *et al. (14)*, large variations in biomechanical properties of skin were found between controls and patients *(15)*, the stiffest skin being found in patients with lipodermatosclerosis. Using CT scanning, significant differences in limb soft tissue area were shown between controls, venous, and lymphoedematous patients, ie limb geometry differed between patient groups. Previous conviction that skin stress would also cause different capillary responses in patient groups was illustrated semiquantitatively using thermography *(16)*. This finding was confirmed using the quantitative techniques of laser Doppler and Mourad's extensometer. Patients with venous disease had a significantly lower hyperaemic response after skin was stretched compared with controls *(17)*.

## Biomechanical synthesis

This second series of experiments had produced physical values which, using a well-known engineering technique 'Finite Element Method' (FEM) allowed us to reconstruct two-dimensional pictures of the stresses and strains encountered in the lower limb. Shutt *et al. (18)* added a

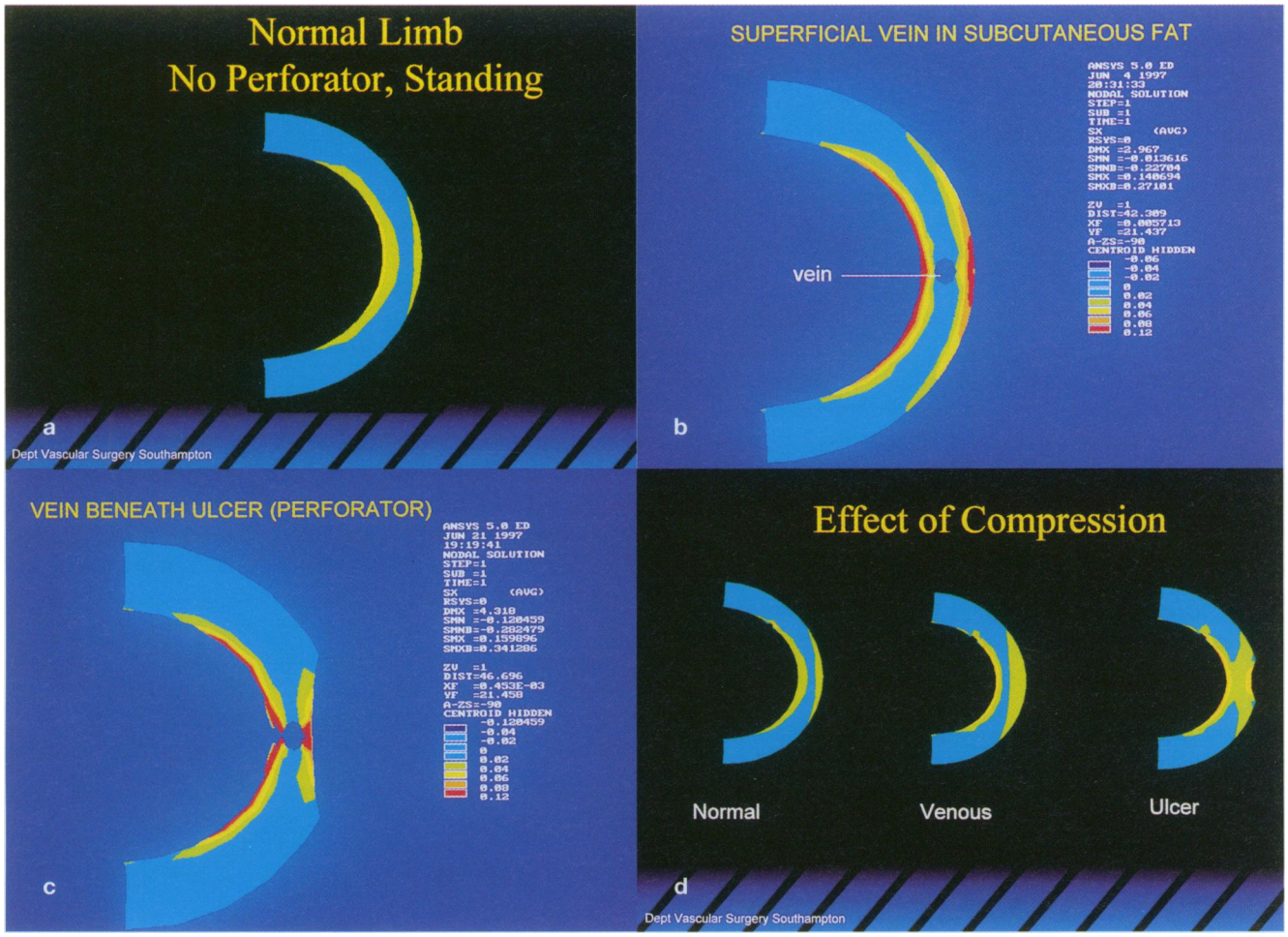


*Figure 2.* Finite element method. Reconstruction of strain in gaiter area (Note: red indicates high strain levels). *(a)* Control (standing). *(b)* Control with perforating vein (note high strains (red) over perforator). *(c)* Ulcerated leg (high strains in base of ulcer). *(d)* Ulcer elevated and compressed.

fascial defect and simulated perforator vein (the biological equivalent of producing a stress concentration as outlined above), and the kind of changes illustrated in Fig. 2 resulted. High tissue pressure was predicted to produce the greatest strain in the gaiter area over the site of perforating veins and it seemed momentarily that this second mechanical hypothesis held good. Not so, critical review by physicist and engineering colleagues revealed that sufficient forces to account for the pathological changes described by Browse, Burnard, Coleridge Smith and others had not been demonstrated. It was then realised that stress *per se* was not important, but that strain was, since skin is well adapted to stand stress, but *not* strain.

Realising this, it then became possible to link the findings into a coherent theory consistent with the known facts. Firstly, using some basic geometry (see Appendix), then computer modelling, a mechanical theory was developed which links together, in quite a simple way, many of the conflicting claims of the last 30 years. The leg can firstly be viewed in cross-section. In this plane, the Laplace relationship applies, provided that the outer shell is thin, and bending moments can be neglected which, of course, as critics pointed out, the skin is not. However, the problem of the thick outer wall can be overcome using the Lamé–Clapeyron solution

$$T = PR/h$$

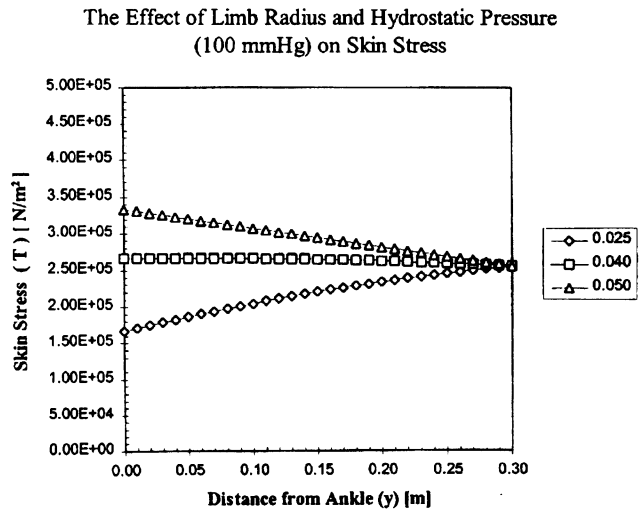


*Figure 3.* Using empirically derived values for elasticity *(E)*, relatively small changes in cross-sectional area produce large changes in stress at the ankle level (0.00).

where $h$ is the wall thickness, $P$ the distending pressure, $R$ the radius and $T$ the circumferential stress. By adapting this formula, Dodds has modelled a lower limb as a cylinder and, by using the known ranges of hydrostatic pressures, estimated the ankle skin stress and showed how this is affected by changes in radius (Fig. 3). In this simple model, assuming that the tissues (arteries, veins, tissue fluid, etc) are compartmentalised and all contained within skin, it can be shown that the raised tissue pressure may distort capillaries leading to their closure. This problem can be affected significantly by the overlying stiffness of the skin as in lipodermatosclerosis. This hypothesis underpins the empirical work which has been reported showing changed hyperaemic patterns on skin stretching in various patient groups *(17)*. It also gives a potential rationale for the capillary failure noted by such workers as Tanner *et al. (19)* and some of the phenomena associated with white cell trapping *(20)*. Importantly, this new model of the lower limb, which links the idea of tissue vessel compliance and tissue pressure directly, almost certainly explains the original $^{24}Na$ findings. It also raises the possibility that much of the circulation's response to

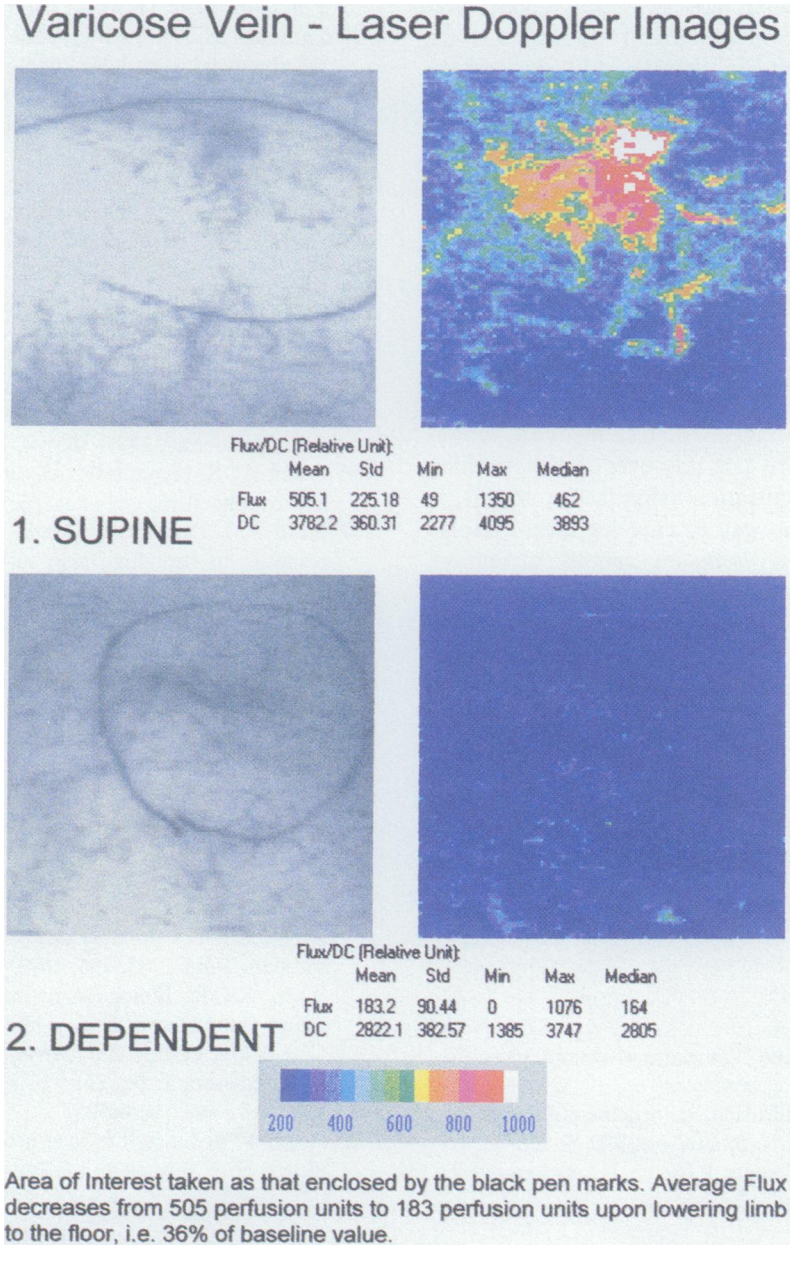


*Figure 4*. Note the paradoxical reduction of the laser Doppler flux over the site of the perforating veins marked by black pen. Average flux decreases from 505 perfusion units to 183 upon lowering limb to floor. *(Courtesy Moor Instruments Limited and the Editor of the British Journal of Surgery* 1998; **85**: 1721).

postural changes could be achieved biomechanically rather than through the axon reflexes suggested by Sir Thomas Lewis. These postural changes can be demonstrated (Fig. 4) by using a laser Doppler scanning device. If this mechanical view of postural control appears unorthodox, then sceptics, in the best Hunterian tradition, should read how nature can solve circulatory problems in other mammals with simple mechanical devices *(21)*.

The final hypothesis—(no longer solely mine) but nevertheless biomechanical—formulated with my colleagues Shutt and Dodds is, I believe, easy to understand and easily tested; it can be stated in simple terms.

The anatomy of the lower limb suggests that increased strains occur in the gaiter area. *A priori* reasoning, computer modelling and measurement confirm that, in the gaiter area, the relationship between capillary pressure and tissue pressure is such that these forces can cause capillary closure, reduced flow or shunting of skin perfusion. This intermittent closure is almost certainly associated with a reperfusion injury *(22)*, white cell trapping and (in a reparative way) fibrin cuffs. When the chronic injury outweighs the reparative capability, ulceration occurs.

This paradoxical under-perfusion of the skin overlying perforators in the upright position is clearly illustrated in Fig. 4.

The converse of this (elevation, treatment of venous disease, providing support, possibly even the destruction of the foci of stress concentration using fasciotomy) is, as the literature testifies, the way to cure leg ulcers. Holan *(23)*, reporting his own work on venous ulceration, quoted the Nobel Laureate Lederberg among others and warns against the dangers of reductionism in science. We have, I believe, over the last 20 years or so, become preoccupied with minutiae of the pathophysiological changes associated with venous ulceration and that we have lost sight of its causation.

I would like to acknowledge the great help given to me by Mrs L Mason in collating and presenting this work, which covers over 30 years.

## References


1 Browse NL, Burnard KG. The cause of venous ulceration. *Lancet* 1982; **2**: 243–5.

2 Michel CC. Oxygen diffusion in oedematous tissue and through pericapillary cuffs. *Phlebology* 1990; **5**: 223–30.

3 Coleridge Smith PD, Thomas P, Scurr JH, Dormandy JA. Causes of venous ulceration; a new hypothesis. *BMJ* 1988; **296**: 1726–7.

4 MacDonald DA. *Blood Flow in Arteries*. London: Edward Arnold Ltd, 1960.

5 Buxton R StJ, MacDonald RJS. The disappearance of radiosodium from the skin of sensitised guineapigs. Proceedings of the Physiology Society. *J Physiol* 1958; **146**: 11P.

6 Chant ADB. The effects of posture, exercise and bandage pressure on the clearance of $^{24}Na$ from the subcutaneous tissue of the foot. *Br J Surg* 1972; **59**: 552–5.

7 Wood JE. A hydrostatic pressure stocking for treatment of ulcers due to chronic venous insufficiency. *Circulation* 1959; **20**: 1043–8.

8 Chant ADB. The clearance of sodium-24 and its relevance to the venous system. MS thesis, University of London, 1972.

9 Pflug JJ, Zubac DP, Kersten DR, Alexander NDE. The resting interstitial fluid pressure in primary varicose veins. *J Vasc Surg* 1990; **11**: 411–17.

10 Lansman JB, Hallman TJ, Rink TJ. Single stretch-activated ion channels in vascular endothelial cells as mechanotransducers? *Nature* 1987; **325**: 811–13.

11 Chant ADB. Tissue pressure, posture and ulceration. *Lancet* 1990; **336**: 1050–51.

12 Scott HJ, Coleridge Smith PD. Tissue pressure, posture and venous ulceration (Letter, comment). *Lancet* 1990; **336**: 1585.

13 Chant ADB. Tissue pressure, posture and venous ulceration (Letter, comment). *Lancet* 1991; **337**: 378.

14 Mourad MM, Edwards C, Marks R. Skin extensibility in the gravitational syndrome. *Bioeng Skin* 1988; **4**: 199–215.

15 Shutt AM, Dodds SR, Cowan AR, Chant ADB. Single site skin extensibility in venous diseased and control lower limbs. In: Negus *et al.*, eds. *Phlebology* volume 1. London: Springer-Verlag, 1995: 182.

16 Chant ADB. Hypothesis: why venous oedema causes ulcers and lymphoedema does not. *Eur J Vasc Surg* 1992; **6**: 427–9.

17 Shutt AM, Dodds SR, Cowan AR, Chant ADB. Capillary response to stretching skin in venous disease and normal subjects. In: Negus *et al.*, eds. *Phlebology* volume 1. London: Springer-Verlag, 1995: 105–7.

18 Shutt AM, Dodds SR, Cowan AR, Chant ADB. 2D finite element models of the gaiter region reveal a mechanical cause for venous ulcers—the effects of perforators. In: Negus *et al.*, eds. *Phlebology*, volume 2. London: Springer-Verlag, 1995: 832–4.

19 Tanner R, Cherry GW, Hale C, Ryan TJ. Impaired hyperaemic response in the microcirculation in patients with leg ulcers. *Br J Dermatol* 1989; **121** (Suppl 34): 45.

20 Thomas PRS, Nash GB, Dormandy JA. White cell accumulation in dependent legs of patients with venous hypertension: a possible mechanism for trophic changes in the skin. *BMJ* 1988; **296**: 1693–5.

21 Wassersug R. Biological hydrostats. Wrapping the armadillo's penis (news). *Nature* 1997; **388** (6645): 826–7.

22 Chu-fa He, Cherry GW, Arnold F. Postural vasoregulation and mediators in reperfusion injury in venous ulceration. *J Vasc Surg* 1997; **4**: 647–53.

23 Holan V. Relation between aetiology and treatment of leg ulcers. *Br J Surg* 1996; **83**: 249–50.


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## Appendix

The biomechanical theory outlined above implies that the skin elasticity ($E_s$) vascular elasticity ($E_v$) capillary perfusion pressure ($P_c$) and the tissue fluid pressure ($P_t$) are interdependent and together are the primary determinants of tissue blood flow and skin nutrition. An approximate relationship between these parameters can be deduced from first principles using a simplified biomechanical model of the leg.

Laplace's law states that the surface tension $T$ (N/m), pressure $P$ (N/m$^2$) and radius $r$ (m) of a thin-walled cylinder are related by

$$T = Pr \tag{1}$$

However, Lamé and Clapeyron showed that Laplace's law only held true provided that the surface tension was evenly distributed and the surface was relatively thin, which clearly is not the case for the skin of the leg. They therefore included the surface thickness $h$ (m) in their calculations and deduced that the surface stress $\sigma$ (N/m$^2$) could be calculated from

$$\sigma = \frac{Pr}{h} \tag{2}$$

Young's modulus of elasticity $E$ (N/m$^2$) for a biological material can be determined empirically by measuring the deforming stress $\sigma$ (N/m$^2$) and the resulting strain ($\varepsilon$) using the formula

$$E = \frac{\sigma}{\varepsilon} \tag{3}$$

In a simple cylinder model of a compliant tube (ie the leg), the circumferential strain is related to the initial radius $r_0$ (m), the final radius $r_1$ (m) after stretching by

$$\varepsilon = \frac{r_1 - r_0}{r_0} \tag{4}$$

and from equations (2), (3) and (4) it follows that

$$E = \frac{Pr_1 r_0}{h(r_1 - r_0)} \tag{5}$$

This simple model can be further refined by considering the leg not as a cylinder but as part of an inverted, truncated, hollow cone (Fig. 5), whose volume $V$ (m$^3$) is given by the standard equation

$$V = \frac{1}{3}\pi L(r_b^2 + r_b r_u + r_u^2) \tag{6}$$

assuming that $r_b$ is the radius of the bottom of the cone, $r_u$ is the radius of the top and $L$ is the height. The radius $r_y$

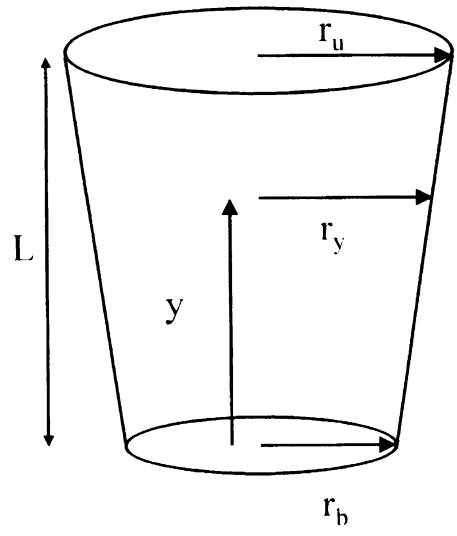


*Figure 5.* Geometry of an inverted, truncated, hollow cone.

at any height $y$ from the bottom is given by

$$r_y = r_b + \frac{(r_u - r_b)}{L} y \tag{7}$$

So if the cone is filled with a fluid of density $\rho$ (eg blood = 1060 kg/m$^3$) then the static pressure $P_y$ (N/m$^2$) at level $y$ is given by

$$P_y = P_0 - \rho g y \tag{8}$$

where $P_0$ = pressure (N/m$^2$) at the bottom of the cone (where $y = 0$) and $g$ is the acceleration due to gravity ($g = 9.81$ m/s$^2$).

Substituting equations (7) and (8) into Lamé's equation (2) gives the local skin stress $\sigma_y$ (N/m$^2$) at any level $y$ from the bottom of the fluid-filled, truncated, inverted cone

$$\sigma_y = \frac{(P_0 - \rho g y)}{h}\left(r_b + \frac{(r_u - r_b)}{L} y\right) \tag{9}$$

Empirically determined values of skin elasticity based on our own research and other published work then enables equation (9) to produce the theoretical predictions illustrated in Fig 3. Further development of this biomechanical model to include separate vascular and tissue compartments and the non-linear mechanical properties of real skin allows the relationships between vascular pressure, tissue pressure and skin elasticity to be predicted in more detail.

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