DIAGNOSIS AND SURGERY OF HIGH-PRESSURE VENOUS LEAKS IN THE LEG

A NEW OVERALL CONCEPT OF SURGERY OF VARICOSE VEINS AND VENOUS ULCERS*

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Varicose veins have often been referred to as the Cinderella of surgery. This has been an apt simile, for she was apparently a drudge and few people took the trouble to take a really good look at her. When they did look at her, as the prince found out, she was found to be fascinating and attractive.

The key to the understanding of varicose veins and their effects lies first in knowing the physiology of venous return from the leg, and only then can we appreciate the disordered physiology which results in varicose veins and venous ulcers. Following on this, the key to proper diagnosis and surgical treatment is a complete knowledge of the venous anatomy. The application of certain new operative methods and new instruments, and an awareness of the supreme importance of post-operative care in these patients, have each added their quota towards Cinderella's transformation.

Physiology

The venous return from the legs of a man lying horizontal is by a completely different mechanism to that of a man

FIG. 1.—Diagram illustrating the physiology and anatomy of the venous drainage of the leg. Note that the main deep veins are enclosed within a musculo-fascial sheath. Piercing this sheath in the leg are two types of perforating veins: (1) small *indirect* (in the upper part of the leg over the muscle bellies), and (2) larger *direct*.

arrangement of a powerful muscle within a closely investing rigid fascial envelope forms a very efficient pump. When the calf contracts, blood in the vessels within the musculo-

in his more usual vertical position. With the legs horizontal at or above heart level, the normal arterial *vis a tergo* is all that is required to return the venous blood, and all veins in the limb, superficial and deep, play equal parts. With man erect, however, the venous return from the legs and feet depends almost entirely on muscular activity and in particular on that beautiful mechanism the calf pump.

The leg can be regarded as a mass of strong muscles, enveloped by a tight fascia, the fascia lata. The fascia is particularly strong and closely applied in the lower leg and calf region. This

fascial envelope normally leaves it only in an upwards direction, with considerable force, owing to the arrangement of the valves.

Fig. 1 illustrates this mechanism diagrammatically. It will be seen here that certain veins perforate the musculo-fascial sheath to drain the subcutaneous tissues and skin. There are two long collecting veins, the great and short saphenous, which perforate the sheath to join the deep veins, at the groin and at the popliteal fossa respectively.

In addition, there are a number of shorter perforating veins, which are of two sorts, as shown in Fig. 1. (a) There are a fairly large number of very small *indirect* perforating veins dotted about over the big muscle bellies—they are small and numerous and connect minor venous channels in the skin and subcutaneous tissues with small intramuscular veins. (b) There are also a small number of *direct* perforating veins. These open direct into the main deep veins of the limb, are few, are found in certain definite anatomical situations, particularly round the ankle, and are of great importance. (The great and short saphenous are really examples of specially large and constant direct perforating veins.)

Now, it is easy enough to understand how the blood inside the musculo-fascial sheath is returned against gravity, by the activity of the calf pump. How is venous drainage of the subcutaneous tissue and skin outside the musculo-fascial envelope of the ankle and leg effected in the erect position? This is the tissue which particularly concerns us in connexion with varicose veins and ulcers. In order to understand this fundamental point we must consider the measurement of venous pressures in the leg and foot.

When a man is standing in the upright position the pressure in a superficial vein of the leg or foot is equal to the height of a column of blood from that point up to the heart—that is, about 120 cm. of water or 80–100 mm. Hg (Fig. 2). This pressure is the same both inside the musculo-fascial sheath in the main deep veins and outside it in the superficial veins, at the same level in the limb. When the

FIG. 2.—The man on the left is standing still. The pressure in his deep veins and superficial veins is the same, and is equal to the pressure of a column of blood extending from that point to the left auricle. The man on the right is exercising his calf pump. The pressure in the deep veins remains high, but the pressure in the superficial veins has fallen almost to zero, as the blood in them has been aspirated into the calf via the perforating veins.

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limb is exercised, however, such as by marking time, a striking change takes place. At once the pressure in the superficial veins falls to nearly zero. The pressure in the main deep veins, however, remains high with violent fluctuations coinciding with muscular contractions. Thus, in some remarkable way (Höjensgard and Stürup, 1952a) the activity of the calf pump drains the subcutaneous tissues of venous blood very efficiently. The clue to how this occurs is given if the behaviour of some radio-opaque substance, such as pyelosil 35%, injected into the subcutaneous veins of the dorsum of the foot, is watched under the screen. At rest, with the patient upright, the contrast medium hangs about in the superficial veins, showing little tendency to move. As soon as the calf pump is put into action, however, the contrast medium disappears from the superficial veins, is seen to enter the deep veins of the calf, and within a few seconds is seen in the popliteal vein. None or very little ascends the limb by way of the great saphenous vein.

Thus, in the exercising limb the venous blood from the subcutaneous tissues is sucked inwards via the perforating veins, into the deep veins, and thence pumped upwards by the calf pump. In other words, the flow is "centripetal." Höjensgard and Stürup (1952b) have shown very nicely, by simultaneous pressure measurements of superficial and deep veins during exercise, that the venous blood is literally aspirated into the calf pump via the perforating veins, during the phase of diastole of the calf pump.

We can now appreciate the enormous importance of the strategic valves which guard the entrance of the perforating veins into the deep veins. They prevent reflux outwards into the subcutaneous tissues during the phase of muscular contraction. If one of these valves becomes incompetent or is destroyed, the affected perforating vein in effect becomes a *high-pressure leak*, and the normal high venous pressure, which occurs in the deep veins, is transmitted into the superficial venous network drained by the particular perforating vein whose valve has ceased to function.

Now, turning back to our basic diagram (Fig. 1) of the venous system of the leg, a leak at the top of the limb, such as might occur through an incompetent great saphenous valve, transmits only a pressure of round about 40–50 mm. Hg, which is the normal pressure in the deep vein at this level of the limb. Moreover, this pressure leak is transmitted into a long, fairly strong vein with numerous branches, and therefore the leak is, as it were, disseminated much more easily.

FIG. 3.—Progress of the great saphenous leak. A, Normal anatomy. 1, Antero-lateral branch. 2, Postero-medial branch. 3, Anterior branch at knee level. 4, Posterior arch vein. 5, Direct ankle-perforating veins. B, Early stage—great saphenous leak. C, Advanced great saphenous leak, but with competent ankle perforators (dilated veins round ankle but no ulcer). D, Advanced great saphenous leak with, in addition, an incompetent direct ankle perforator (severe eczema or varicose ulcer).

On the other hand, a leak at the bottom of the limb, such as might occur through an incompetent ankle-perforating vein, will transmit a pressure of 100 mm. Hg straight out on to the delicate venous mesh round the ankle, with explosive effect. This is a high-pressure leak indeed.

Thus, in the normal exercising leg the pressure in the deep veins (inside the deep fascia) is high, while the pressure in the superficial veins is low or almost zero. The basic pathology of varicose veins and venous ulcers, and other disorders, is the destruction of a valve or valves in one or more perforating veins, allowing this high internal pressure to be transmitted out to the superficial system, with its consequent steady gradual dilatation and degeneration.

The art of diagnosis and treatment of venous disorders lies in an exact knowledge of where these leaks are likely to occur, and what the effects of each leak are. The earlier a leak is diagnosed and stopped up by efficient surgery, the less will be the irreparable damage to the superficial veins and tissues.

Let us now turn to a more detailed consideration of the sites of high-pressure venous leaks from the deep veins. The common high-pressure leaks are the long saphenous leak, the short saphenous leak, and the perforator leaks.

Long Saphenous Leak

The normal pressure in the deep veins in the erect position at the level of the inguinal fold is about 40 mm. Hg, and therefore this is the extent of the pressure leak which occurs if the highest saphenous valve becomes incompetent. This force then steadily acts as a retrograde dilator of the whole great saphenous system. This retrograde dilatation may take many years to reach its maximum effect, and the slow but inexorable march of events is shown in Fig. 3. The retrograde dilating pressure of 40 mm. Hg is greatly enhanced by muscular activity or effort. For instance, a housewife bending over a sink washing is increasing this dilating force up to 70–80 mm. Hg or more at each effort she makes. Coughing causes a powerful retrograde pressure wave, which can be appreciated by the finger over the vein as a cough impulse. In the early years of the valvular incompetence, only the upper reaches of the great saphenous system may become dilated, but later the steady thrust of the retrograde dilating pressure affects the lower reaches round the ankles. There are several remarkable and little-appreciated facts about the progress of this retrograde dilatation. The anatomy of the great saphenous system, although variable in detail, follows a steady basic pattern, which is shown in Fig. 3 A. The great saphenous trunk itself is a strong muscular structure—it becomes *dilated* but almost *never tortuous*. The branches of the great saphenous trunk, however, are thin-walled and weak; they were never designed to stand up to high intravenous pressures. Thus, when the retrograde dilating pressure reaches them they become enormously *dilated and tortuous*, and eventually completely outstrip the parent trunk in size and prominence. These masses of dilated varices that one sees on the limb are therefore the dilated and tortuous branches of a trunk which remains straight albeit dilated, and in even moderately well covered limbs is situated so deep that it is often not visible at all. This is the reason why it is possible to strip the great saphenous vein even in the more advanced cases, because the stripper passes up the main straight trunk.

Another important fact to grasp is the anatomy of the venous drainage of the inner side of the leg. Reference to Fig. 3 A shows that there is a major branch of the great saphenous vein joining it at knee level, called the posterior arch vein (first illustrated by Leonardo da Vinci). This vein takes the brunt of the retrograde dilating force when it reaches knee level and, as it dilates, transmits this high pressure to the area of the ankle which is drained by the inner ankle-perforating veins (the ulcer-bearing area). If the ankle-perforator valves are competent, these veins steadily "drain off" this high pressure into the calf pump,

and little more than eczema or a small easily controlled ulcer may result (Fig. 3 C). If, however, in addition, the valve of one of the ankle perforators becomes incompetent, a summation of the pressure effects of the long saphenous leak and the higher pressure of the ankle-perforator leak occurs in this area and the result is serious ulceration and induration (Fig. 3 D) (the lesion classically known as a varicose ulcer).

Treatment of Long Saphenous Leak.—(1) Stop up the leak at its source. This means flush sapheno-femoral ligation. If one small branch is left between the ligature and the femoral vein, this will still transmit the high-pressure leak and the whole procedure will be invalidated. (2) Remove so far as is possible the effects of the leak. Strip the dilated great saphenous trunk. The combination of flush sapheno-femoral ligation, and stripping, gives results far in advance of anything which has hitherto been tried, even in advanced cases. (3) Look for and stop up any coexistent leaks. In particular an ankle perforator must be looked for and tied if there are dilated veins in the ankle area or an actual ulcer.

Short Saphenous Leak

The short saphenous vein penetrates the fascia of the popliteal fossa to join the popliteal vein. Its entrance into the popliteal vein is guarded by a valve. If this valve is destroyed (as by a thrombus) or becomes incompetent it will constitute another outward leak from the deep veins. The pressure in the popliteal vein, with the patient erect and exercising, is higher than that in the femoral vein at the groin by about 20–30 mm. Hg, so a leak at popliteal level will transmit a pressure of around 60–70 mm. Hg, especially during muscular exercise.

Reference to Fig. 4 will show how this high pressure is transmitted out to a short strong straight vein which runs down to the lateral aspect of the ankle, where it collects the venous drainage by a large number of smaller veins, mainly from the region over and behind the lateral malleolus. It communicates with the internal ankle-perforator system, however, by veins which pass medially round the limb behind the soleus and tendo Achillis.

The effect of this leak is strictly analogous to the long saphenous leak. The retrograde dilating force is greater, and its effect is to dilate the trunk of the short saphenous, which, however, remains straight. The branches, however,

FIG. 4.—Progress of short saphenous leak. A, Normal anatomy (there are many variations, but this is the common anatomy). There is usually one lateral direct ankle-perforating vein in the position shown. B, Uncomplicated short saphenous leak. Note great dilatation of terminal branches round lateral malleolus. C, Short saphenous leak, plus lateral perforator leak (usually oedema, and eczema or ulceration behind lateral malleolus).

especially those round the lateral malleolus, become enlarged and dilated. The effects of this leak are more serious than those of the great saphenous leak and make their presence felt more quickly. The patients complain of swelling of the ankle, particularly behind the lateral malleolus, aching, and eczema which is typically first seen behind the malleoli in the post-malleolar groove. If short saphenous incompetence is combined with lateral ankle-perforator incompetence (Fig. 4), then the effect may be a disabling retro-malleolar ulcer.

The short saphenous leak is much more common than is generally supposed and is one of the causes of so-called recurrent varicose veins, in that it is often overlooked when operation is performed on the great saphenous system.

Treatment of Short Saphenous Leak.—(1) Stop up the leak at its source. This means a flush sapheno-popliteal ligation in the popliteal space. Again, any tributary left between the ligature and the popliteal vein will still transmit the high-pressure leak, and will further cause the development of a crop of small varices round the scar, which are especially troublesome in the popliteal fossa. (2) Remove the effects of the leak. The short saphenous system is stripped from lateral malleolus to popliteal fossa. (3) Look out for and stop up any coexistent leaks—the most common one is the lateral ankle-perforating vein.

Perforator Leaks

We have seen (Fig. 1) that the perforating veins are of two types: *indirect* and *direct*.

The *indirect* ones are small, numerous, and inconstant, and are scattered over the big muscle bellies. A leak from one of these has no serious effects, except perhaps the development of a small crop of tiny dilated surface venules near the perforator in question.

The *direct* perforating veins, however, are few and relatively constant in position, and they open into the main deep venous trunks. A leak from one of these has more serious effects. It is therefore important to know their exact position.

FIG. 5.—Two venograms, showing the normal (A) and the incompetent direct perforating vein which is fairly constantly found at the lower third of the thigh (B). The enormous dilatation which such an incompetent perforating vein can undergo is well illustrated.

There is one constant perforator at about the lower third of the thigh (Fig. 5), which usually runs direct from the saphenous vein, or one of its small branches, into the main deep femoral vein, or one of its venal comites. The effect of a leak here (a relatively low-pressure leak) is comparable to the effects of the great saphenous leak. The position and the effects of a leak via this perforating vein are shown in Fig. 5.

The most important direct perforating veins, however, are found in the lower half of the leg. This part of the leg has relatively little in the way of muscle bellies, and thus

FIG. 6.—Positions of direct ankle-perforating veins of inner and outer sides of leg. They are the main venous drainage of the ankle skin (ulcer-bearing area) in the erect position. The numbers 1, 2, and 3 refer to main groups of branches of the great saphenous vein which join it at or about knee level. No. 3 is the important posterior arch vein leading down to the area of ankle drained by the ankle-perforating veins.

FIG. 7.—Effects of incompetence of ankle-perforating veins. The effects of a high-pressure leak at the bottom of the calf pump is a steady dilatation of the perforating vein and the whole venular bed which it drains.

the small indirect perforating veins are absent here, the major part of the venous drainage of the area being handled by four fairly constant large direct perforating veins. Their anatomy is shown in Fig. 6. There are usually three on the inside of the limb and one on the outside.

The lowest of the three perforating veins on the inner side of the limb, which occurs just behind the medial malleolus, is relatively small and unimportant, and becomes incompetent less often than the upper two. These veins are then the main venous drainage of the ulcer-bearing area of the ankle in the erect position. If the valve at the junction with the deep posterior tibial vein is destroyed, or becomes incompetent, these short wide veins constitute a high-pressure

leak at the bottom of the calf pump. When the calf pump is in action a pressure of about 100 mm. Hg—that is, about normal arterial pressure—is transmitted straight out into the fine venous mesh round the ankle (Fig. 7). This causes a considerable local venous hypertension in the subcutaneous tissues of the ankle. The immediate effect of this is some swelling, a tense uncomfortable feeling, and the appearance of dilated venules. These dilated venules appear particularly as a “flare” disappearing into the heel pad—the “ankle flare,” a most important sign of a venous lesion at the ankle (Fig. 8).

FIG. 8.—Ankle flare. This flare of dilated venules draining away into the heel pad is a typical sign of a venous ulcer or of ankle-perforator incompetence or of superficial gross venous incompetence.

FIG. 9.—A, Venogram of an ulcer. B, Arteriogram of an ulcer. These two pictures show how the subcutaneous tissues of the ankle under and around a venous ulcer are honeycombed with small dilated vascular channels. This haemangioma-like sponge can be filled equally well with contrast medium from either the venous or the arterial side of its circulation.

The next effect may be the appearance of either of two typical lesions: (1) In the thin leg without much fat a scaly, itchy, dry eczema appears over the ankle area, or (2) in the fat cold leg, which many women possess, a hard indurated painful plaque appears, which is red, hot, and tender in the early stages. This is an area of fat necrosis and is the initial lesion of the induration which is so typical of these legs. Pigmentation starts to appear at this stage as well.

The last stage in the process is the appearance of the ulcer, which may either break out on its own or be precipitated by trauma.

Thus, the venous ulcer, far from being an *ischaemic* lesion, is a *hyperaemic* lesion. It is sitting on a honeycomb of dilated venules which in fact is rather like a haemangioma. The longer the ulcer has been present the more profuse and sponge-like is the venous sump on which the ulcer sits. Moreover, as time goes on, this hyperaemic area acquires an increased arterial supply; and this venous "sump" under the ulcer can be filled equally well from the arterial or venous side of the circulation, and therefore can be shown equally well by an arteriogram or a venogram (see Fig. 9).

Finally, the cause of destruction of the strategic valve in the ankle-perforating veins, and therefore the cause of their incompetence, is deep thrombosis in the majority of cases. Post-thrombotic ankle-perforator incompetence is therefore the main immediate cause of the ulceration, induration, and swelling of the ankle, which is such a prominent feature of the post-thrombotic syndrome of the leg.

Treatment of Perforator Leaks

The treatment of high-pressure leaks at the ankle, perforator leaks, and their effects, is to relieve the unnatural venous hypertension in the subcutaneous tissues of the ankle region. This can be done by conservative methods or by operation.

Conservatively.—(1) By putting the patient flat, with the feet a little above heart level (raising the foot of the bed). (2) By applying an elastic compressing force from the outside (this is the basis of the elastic-stocking and elastic-bandage method of healing ulcers).

Operatively.—By finding and ligating the incompetent perforating veins. They must be ligated *at source*—that is, either below or as they emerge from their holes in the deep fascia. This simple measure will control and heal early cases of ankle ulceration. In the more widespread and long-standing cases, as much as possible of the haemangioma-like venous sump round the ankle must be removed as well, to get a good and lasting result. The exact technical methods of achieving this have been dealt with elsewhere (Cockett, 1955; Dodd and Cockett, 1956).

Conclusion

In this short paper I have attempted to deal only with the main mechanical principles underlying the pathology of varicose veins and venous ulcers. *En passant*, as it were, a very beautiful and intricate piece of physiology has been explored—the mechanism of venous drainage of the subcutaneous tissues of the leg and ankle in the erect position. Only if this physiology and the exact anatomy of the main perforating veins are mastered does the story of varicose veins and venous ulcers become intelligible. Once it is mastered the art of diagnosing the exact site of the venous leak or leaks in a patient who presents with varicose veins or an ulcer becomes an interesting clinical exercise.

Successful surgery then depends to a great extent on just how much time and care the surgeon is prepared to spend on his patient. The proper performance of the various flush ligations of the perforating veins at all levels in the limb needs time and care. The ancillary procedures, like stripping and skin grafting, need scrupulously careful operative technique and aftercare. Successful surgery of varicose veins cannot be done in ten minutes at the end of a long list.

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AETIOLOGICAL ASPECTS OF AMMON'S HORN SCLEROSIS ASSOCIATED WITH TEMPORAL LOBE EPILEPSY*

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The cornu Ammonis has in recent years received much attention in the discussion of the pathological processes associated with temporal lobe epilepsy. The problem and its historical development have been outlined in several publications coming from the Maudsley group—for example, Meyer (1954), Falconer *et al.* (1955), Meyer and Beck (1955)—and in order to avoid repetition the reader is referred to these publications. In the present paper it is intended to deal with certain aspects of the pathology and aetiology of this condition, which in previous papers were either omitted or reported upon in inadequate detail.

The present report is based upon 40 cases in which radical temporal lobectomy has been carried out by Mr. Murray Falconer.

Subdivision of the Pathological Findings

In order to maintain a proper perspective it would be best to outline briefly the various pathological changes that have been met with in this material.

Focal Lesions

Our material conforms with that of Earle, Baldwin, and Penfield (1953) in that about one-third of the cases (13 out of 40) showed a sharply circumscribed abnormality, sometimes visible to the naked eye. Table I lists these changes and defines their location. In two of the cases with tumours the lesion was of such an extent as to be of little interest with regard to the functional disturbances recorded. On the other hand, small well-defined tumours in the early stage of their development and vascular lesions were found restricted to very narrow limits. Interesting though these focal lesions may be, they are not, however, the subject of the present paper. It should be noted that in no case was definitive Ammon's horn sclerosis encountered in this group, although this region was encroached upon by an angiomatous malformation in one instance.

Diffuse and Disseminated Lesions

The pathological changes in the remaining two-thirds of the cases may be divided into two subgroups—namely, (1) those cases in which Ammon's horn sclerosis was present in addition to other diffuse lesions in the lobe, and (2) those cases in which little or no abnormality of importance was encountered. The changes and their distribution are shown in Tables II and III, where they are arranged in order of severity. In the Ammon's horn sclerosis group the changes encountered were diffuse laminar nerve-cell loss in the cortex involving chiefly layers II and III; partial or complete loss of nerve cells in the pyramidal layer of Ammon's horn; and gliosis of the white matter and of the subpial layer of the

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