THE AETIOLOGY OF VENOUS ULCERATION AND ITS MANAGEMENT BY LIGATION OF THE ANKLE COMMUNICATING VEINS

A REPORT OF 50 PATIENTS TREATED BY THIS METHOD

J. C. ALLAN, CH.M. (RAND), F.R.C.S. (EDIN.)

Surgeon, Johannesburg, and Departments of Anatomy and Surgery, University of the Witwatersrand, Johannesburg

The management of venous ulceration of the leg has been a problem in medicine for centuries and the voluminous literature on the subject demonstrates that the results of treatment leave much to be desired.

HISTORICAL SURVEY

The understanding of the pathogenesis of venous ulceration has been slow. This is because the basic causative factor of valvular deficiency in the deep veins seems to have escaped the attention of early observers. In the absence of some definite aetiological agent, many causes were, therefore, ascribed to the condition. Before, and even after Harvey discovered the circulation of the blood in 1628, venous ulceration was thought to be due to humours. In the 14th century, Ambroise Paré considered that ulcers were due to the stagnation of menstrual blood in the legs during pregnancy, and even as late as the first part of the 19th century, Astley Cooper[1] and Critchett[2] maintained that these ulcers were related to amenorrhoea.

After having disappeared for nearly two centuries, the term 'varicose ulcer' was used again to describe the condition, when its association with varicose veins was emphasized by Cooper,[1] Brodie,[3] Chapman,[4] and others. The turning point in the understanding of the aetiology and pathogenesis of venous ulceration probably occurred in 1868, when John Gay[5] stated that venous thrombosis played a part in the aetiology and that the ulceration was not a direct consequence of varicosity, but of other conditions, of which venous and arterial obstruction were the most likely.

Homans[6,7] gave a renewed impetus to the study of the condition when he emphasized its relationship to previous deep venous thrombosis and proposed that obliteration of communicating veins was desirable in the management of the disease. Since then, Birger,[8] Bauer,[9] Anning,[10] and many others have shown conclusively that most cases of venous ulceration follow a previous episode of deep venous thrombosis. As a result of this finding, the sequelae of deep venous thrombosis in the leg, viz. swelling, induration, pigmentation, eczema and ulceration have become known as the *post-thrombotic syndrome*.

PATHOPHYSIOLOGY

The aetiology of venous ulceration is intimately associated with the upright posture. In the recumbent position (Fig. 1), the mean arterial pressure in the lower limbs is about

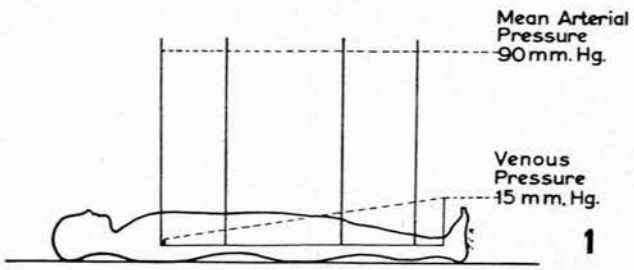

Fig. 1. Arterial and venous pressures in the recumbent position.

90 mm.Hg, and the average venous pressure is about 15 mm.Hg. In the erect position (Fig. 2), both arterial and venous pressures are increased by the height of the column of blood from the heart to the point at which the measurements are made. In a person of average height, in the standing position, the systolic arterial pressure at the ankle is about 170 mm.Hg, and the venous pressure is about 80 mm.Hg. When the subject passes from the recumbent

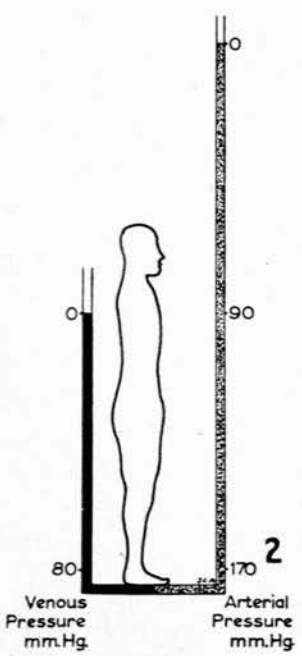

Fig. 2. Arterial and venous pressures in the standing position.

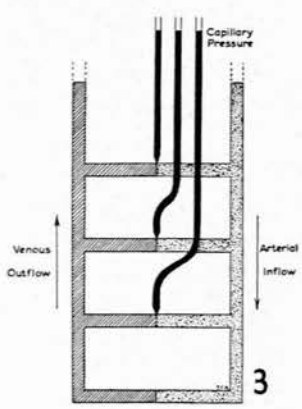

Fig. 3. Diagrammatic representation of the relationship of capillary pressure to the position of the capillary bed. The further inferior the capillaries the higher the pressure.

to the standing position, the effect of gravity is to pull downwards on both sides of the circulation. This means that the point of junction of the two sides will be subjected to greater pressure than either of the two sides themselves. This junction is the capillary bed, and the more inferiorly this lies, the greater will be the pressure within it (Fig. 3).

In the upright position, and while standing perfectly still, the blood in the veins of the lower limb flows in a proximal direction. This flow indicates that the distal capillary pressure exceeds the more proximal venous pressure. The capillary pressure also exceeds the colloid osmotic pressure of the blood and results in increased filtration of fluid into the tissues of the ankle and foot. This was clearly demonstrated by Atzler and Herbst in 1923.[11] Tissue fluid is normally removed by the lymphatics and by reabsorption into the capillaries. This reabsorption is brought about by the reduction of the filtration pressure to below the colloid osmotic pressure. This is achieved by the 'pumping' action of the muscles of the calf, which reduces the venous and capillary pressures.

Venous System of the Leg

The venous system of the leg is divisible into deep and superficial systems, these being connected by communicating veins (Fig. 4). The deep and superficial systems are endowed with valves arranged in such a way that blood flow is unidirectional from the periphery to the heart. The communicating veins are valved so that blood flows only from the superficial to the deep venous system (Fig. 5A).

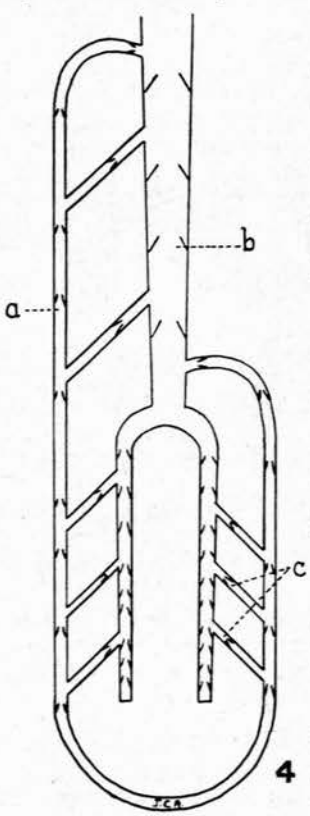

Fig. 4. The three venous systems of the lower extremity. (*a*) = the superficial system, (*b*) = the deep system, (*c*) = the communicating veins.

The deep veins, being situated between layers of muscle under the deep fascia of the leg, are compressed during muscular contraction and the blood is forced out in a proximal direction (Fig. 5B). When muscular relaxation occurs the pressure in the deep system is momentarily lower than in the superficial system and blood passes from the superficial to the deep system *via* the communicating veins. During muscular relaxation, reflux of blood down the deep veins is prevented by closure of the valves. The result of muscular contraction and relaxation is thus an alternating reduction of pressure in the deep and superficial veins. This, in turn, results in a reduction of filtration pressure in the capillaries, minimizing or preventing the accumulation of tissue fluid.

Any factor which increases capillary pressure or prevents its reduction will result in increased filtration of fluid and the production of oedema. One such factor is an arteriovenous fistula. The arterial pressure is transmitted directly to the vein and, if the capillary bed is close to the point of communication, the capillary pressure will also be increased. The commonest cause, however, of increased capillary pressure is increased venous pressure, following destruction of the valves

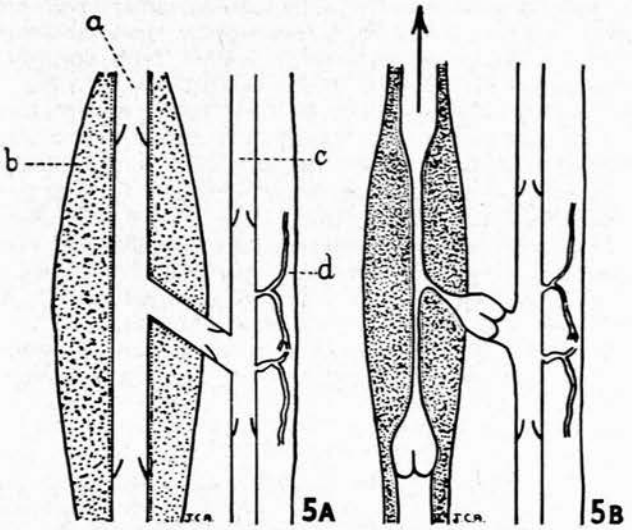

Fig. 5A. Diagrammatic representation of a deep vein in muscle and a superficial vein in the subcutaneous tissue, connected by a communicating vein. (*a*) = deep vein, (*b*) = muscle, (*c*) = superficial vein, (*d*) = skin.
Fig. 5B. Contraction of muscle causes valves to close and flow is uni-directional.

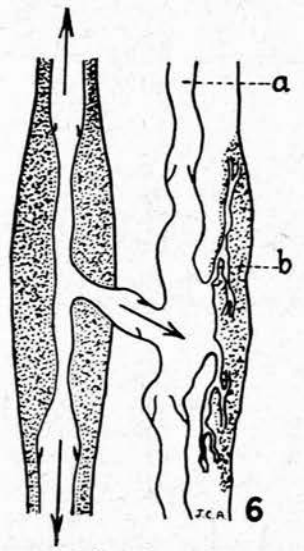

Fig. 6. Effect of muscle contraction after destruction of valves. Superficial veins dilate and skin and subcutaneous tissues become oedematous. (*a*) = dilated and tortuous vein, (*b*) = oedema of skin and subcutaneous tissues.

in the deep and communicating veins. Valvular destruction usually follows an episode of deep venous thrombosis, when, after recanalization, the veins are converted to irregular valveless conduits.

Compression of valveless channels during muscular contraction results in blood being forced backwards down the deep veins and along the communicating veins (Fig. 6). With this increased pressure the superficial veins dilate and ultimately form varicosities in the calf. The medially situated posterior arch vein[12] is particularly affected because the communicating veins connect with it. Varicosities owing to deep venous reflux also occur on the lateral aspect of the calf.

Many patients state that their varicose veins started in the calf and spread progressively upwards. This sequence may distinguish the varices caused by communicating-vein incompetence from those of the so-called idiopathic variety, which usually start in the thigh and spread downward.

The increased pressure in the superficial veins is also transmitted to the capillaries in the region. This results in increased filtration and the production not only of oedema of the skin and subcutaneous tissue, but of pigmentation owing to the escape of red blood cells into the tissues. This pigmentation may vary in colour from light-brown to dark slate-grey.

However, an extensive red area affecting the lower calf and ankle region is often more noticeable in patients with venous ulceration. This redness, which assumes a bluish hue on standing, does not disappear on elevation of the leg. It seems likely that the redness is due to an increased arterial flow through the part. A possible explanation for this is that the venous back-pressure, which may be of the order of 150 - 180 mm.Hg on muscular contraction, is forced backwards along the capillary loop and possibly into the neighbouring arteriole. If dilatation of the arteriole occurs, the peripheral resistance is lost and the arrangement now constitutes a type of arteriovenous fistula (Fig. 7). The unreduced arterial pressure transmitted to the capillary loop would increase the filtration pressure further and aggravate the oedema.

ANKLE AND CALF COMMUNICATING VEINS

The effects of retrograde venous pressure are most evident where the communicating veins link the deep and superficial venous systems directly. These points are on the medial and lateral aspects of the calf, as has been described by Cockett.[13] The lower portion of the calf and the ankle region are thus the parts most commonly involved in the oedema process. The presence of oedema in the skin and the subcutaneous tissue predisposes the region to eczema and ulceration. The slightest trauma to the part may result in the formation of a spreading ulcer which has tardy healing properties. Reduction of increased capillary pressure will reduce oedema and induce healing of an ulcer. This may be accomplished by bed-rest with elevation of the legs or by firm compression of the ulcer-bearing area. It follows that after an ulcer is healed it is necessary to maintain the reduction in capillary pressure, otherwise ulceration will recur.

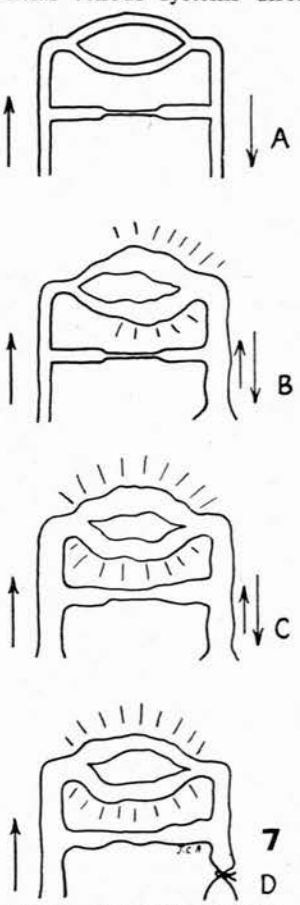

Fig. 7. Schematic representation of a capillary loop and arteriovenous anastomosis subjected to prolonged and excessive venous pressure. Ligation of the vein does not eliminate the hypertension. *A* = normal capillary loop and arteriovenous anastomosis, *B* = venous back-pressure, *C* = venous back-pressure extending into arteriole and arteriovenous anastomosis, *D* = ligation of vein without elimination of the increased pressure.

The communicating veins in the lower part of the leg link up with the posterior arch vein and the smaller tributaries of the saphenous veins, rather than directly with the saphenous veins themselves.[13,14] Dissections of 150 human legs (of African and European subjects) in the Department of Anatomy of the University of the Witwatersrand have confirmed this work. The communicating veins on the medial aspect of the leg link the superficial venous network with the venae comites of the posterior tibial artery. In the lower third of the leg, the space between soleus (and tendo calcaneus) and flexor digitorum longus and flexor hallucis longus is filled with loose fat. In addition, the posterior tibial venae comites are immediately deep to the fascia covering the flexor muscles.

In this region, the communicating veins are short and of relatively large calibre. In the middle and upper thirds of the calf, the communicating veins reach the posterior tibial venae comites by passing between soleus and flexor digitorum longus. As the venae comites are situated more deeply in these parts of the calf, the communicating veins are longer and of lesser calibre than those in the lower third. Measurement shows that the majority of the medial communicating veins are found 2 - 5 inches above the tip of the medial malleolus and that these veins are generally of large calibre (Fig. 8).

On the lateral aspect of the leg, anterior and posterior groups of communicating veins may be distinguished. These link the superficial venous network with the peroneal venae comites. The anterior group consists of a number of narrow channels passing between the bellies of the peroneus longus and brevis muscles. The posterior group consists of a few larger channels passing between soleus and peroneus longus. These channels lie between the layers of the fascia which separates soleus from peroneus longus and peroneus longus from peroneus brevis. The common positions of the lateral communicating veins are

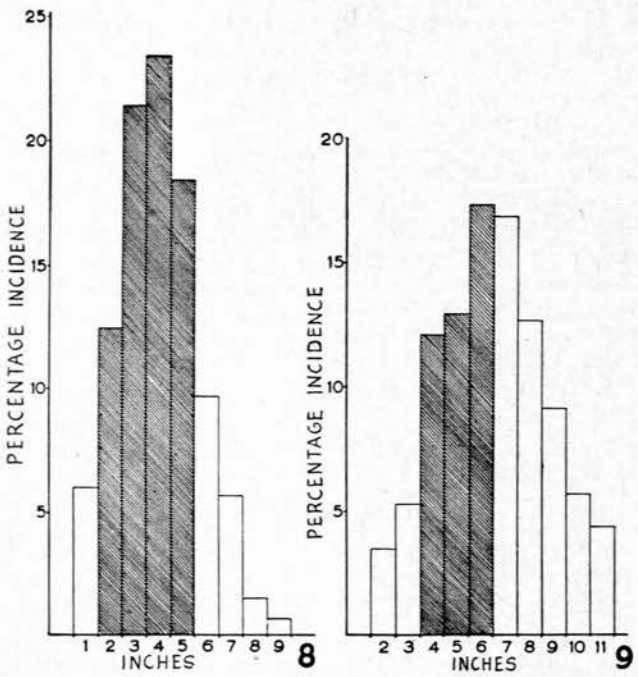

Fig. 8. Percentage incidence of communicating veins at one-inch (2·54 cm.) intervals above the medial malleolus. The cross-hatched area represents veins of relatively large calibre.

Fig. 9. Percentage incidence of communicating veins at one-inch (2·54 cm.) intervals above the lateral malleolus. The cross-hatched area represents veins of relatively large calibre.

between 4 and 9 inches from the tip of the lateral malleolus; those at the 4-, 5-, and 6-inch levels are generally somewhat greater in calibre than the others (Fig. 9).

It is clear that removal of the great and small saphenous veins will not destroy the channels along which the reflux of blood takes place from the deep veins. Experience, also, has shown that removal of the saphenous veins alone is an inadequate operation for the treatment of venous ulceration, because the main source of increased venous and capillary pressure remains after the operation. Conversely, since the communicating veins do not link with the saphenous veins, there would be little or no indication for removal of the saphenous veins unless they appeared grossly diseased. In general, no operation for the treatment of venous ulcer is satisfactory unless the 'blow-back' of blood under high pressure from the deep veins has been eliminated. This, in effect, means eradication of incompetent communicating veins between the deep and superficial venous systems.

MANAGEMENT

Rémy, in 1901,[15] was the first to advocate ligation of the channels connecting the deep and superficial venous systems. In 1917, Homans[7] repeated this advice on the basis that the valves in these veins had been destroyed in the thrombotic process. In 1929, Trout[16] not only ligated the communicating veins, but also excised the deep fascia and covered the defect with a skin graft. Since then, reports on the ligation of communicating veins have been published by Linton,[17,18] Sherman,[14] Cockett and Elgan Jones,[19] Cockett,[13] Dodd *et al.*[20] and Cranley *et al.*[21] All these authors have used medial and lateral incisions to approach the communicating veins. In 1955, however, Felder *et al.*[22] described a posterior subfascial approach through a 'stocking-seam-line' incision. This allowed both medial and lateral communicating veins to be ligated through one incision.

The standard operation recommended by Linton[17] is ligation of all communicating veins by the subfascial route, together with removal of the great and small saphenous veins and ligation of the superficial femoral vein in the thigh. Cockett and Elgan Jones[19] recommended ligation of the medial and lateral ankle communicating veins by the extrafascial route. This is a somewhat restricted approach compared with that of Linton.

PRESENT SERIES

In the 50 cases recorded in the present series, the operation was restricted to ligation of the medial and lateral ankle communicating veins. The subfascial approach was used in 6 cases and the extrafascial approach in 44.

Pre-operative Treatment

In this series all the ulcers were healed before operation, either by ambulant treatment or by bed-rest. Ambulant treatment was carried out in the outpatient department of the Johannesburg General Hospital and consisted in cleaning the ulcer with physiological saline and covering it with plain white 'vaseline' gauze. Over this was applied a thick pad of cellulose sponge and the whole leg from the metatarsal heads to just below the knee was enveloped in a firm compression bandage, either of the Unna's paste type or of adhesive bandage. At first the compression bandage was changed at weekly intervals and later at longer intervals.

Although this method was more protracted than the bed-rest method, it had the advantage that it allowed the patient to work and saved the cost of hospitalization. If the ambulant treatment was unsuccessful or if the ulcer was large and painful, the patient was treated in bed with the foot of the bed raised so that, in the recumbent position, the level of the ulcer was above the level of the heart. This usually resulted in rapid healing of the ulcerated area. When the ulcer had completely epithelized, the surrounding skin, if scaly, was rubbed daily with dilute hydrogen peroxide to remove the scales; if redness and oedema of the skin were

present, 'hydrocortone', 1% in lanoline or eucerin, was applied to the area twice daily.

For two days before the operation, all patients were kept in bed with the feet raised, to diminish oedema of the ankle.

Operative Technique

With the patient's legs elevated 10° from the horizontal, the medial communicating veins were approached through a 6- to 7-inch incision, parallel to the medial border of the tibia, and extending upwards from a point ¾-inch above the tip of the medial malleolus. The lateral communicating veins were approached through a 5-inch incision, extending upwards, just behind the line of the fibula, from a point 2 inches above the lateral malleolus. Figs. 10 and 11 indicate the extent of the incisions and explorations.

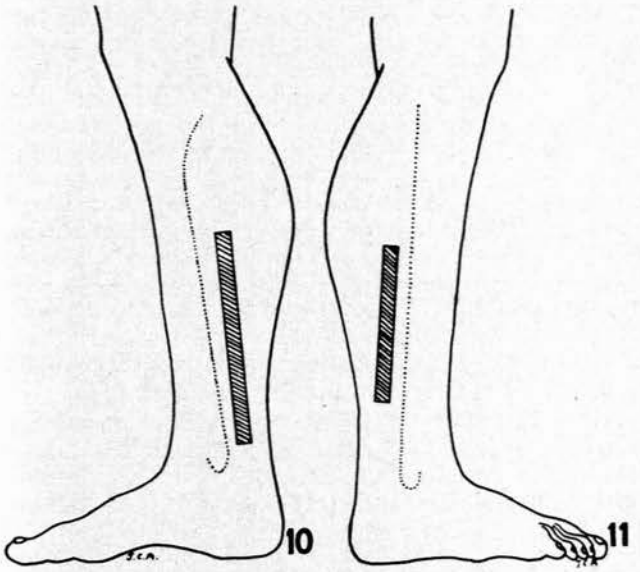

Fig. 10. The extent of the incision and exploration on the medial side of the calf.
Fig. 11. The extent of the incision and exploration on the lateral side of the calf.

The skin edges were not touched with forceps, retraction being obtained with blunt hooks. The subcutaneous fat was explored and any veins found were followed by dissection up and down the calf until the communicating veins were found. These veins were carefully isolated from the surrounding fat and from the edge of the aperture in the fascia through which they emerged. Slight traction was applied to the vein and it was then ligated with 00 plain catgut, flush with the vena comitans into which it entered. It was then divided distal to the ligature. Care was taken not to exert too much traction on the vein so that the deep vein would not be included in the ligature. In the search for the communicating veins, care was taken not to undercut the skin too far lest necrosis take place later.

When all the incompetent communicating veins in the region had been found and ligated, as much as possible of the posterior arch vein was resected and the wound closed with 00 single-strand non-absorbable suture. The stitches were applied close to the skin edge to minimize obstruction to blood vessels and subsequent skin necrosis. The wound was covered with gauze and cottonwool, and the leg from the metatarsal heads to the knee was enclosed in two firmly applied crêpe bandages.

In this series no difficulty was experienced in finding the communicating veins by either the extrafascial or subfascial route.

Postoperative Treatment

The patient remained in bed until the 5th postoperative day, with the foot of the bed raised on 6-inch blocks. On the 5th postoperative day, the patient was allowed out of bed, after adjustment of the leg bandages and the application of a strong elastic stocking. Stitches were removed on the 10th postoperative day and the patient was discharged from hospital wearing a firm, well-fitting, *heavy-duty* elastic stocking.

Results

All the patients in the series were White. Of the 50 cases, 9 were in the pre-ulcerous state. Prophylactic operations were performed on these patients and after 3 years no patient had developed an ulcer. Since it may be argued that these patients would not have developed ulcers, they have not been included in the results.

The general features of the cases are shown in Table I. It will be noted that there were twice as many females as males and the left leg was affected more than twice as often as the right. In this series 27% had previously had some form of operation for varicose veins or venous ulceration.

TABLE I. ANALYSIS OF PATIENTS WITH VENOUS ULCERS

	No.	%
Total patients	41	100
Males (average age 49 years)	13	32
Females (average age 51 years)	28	68
Right leg affected	11	27
Left leg affected	29	71
Both legs affected	1	2
Patients with previous surgery for varicose veins and/or ulcer	11	27

Twenty-eight patients (68%) had a definite history of deep venous thrombosis. In 7 patients (25% of those affected) the cause was obstetric, in 5 (18%) it followed injury, in 3 (11%) it followed operation, in 1 (3%) it followed injection for varicose veins, in 3 (11%) the cause was a medical condition, and in 9 (33%) the cause of the deep-vein thrombosis could not be ascertained.

The postoperative complications are shown in Table II.

The incidence of wound breakdown — and in none was this extensive — was 20%. The majority of these wounds had an area of breakdown of half-an-inch or less. In only 2 cases of septic disruption was the wound involved for more than 1 inch.

TABLE II. POSTOPERATIVE COMPLICATIONS

Complication	*No.*	%
Wound breakdown	10	20
Aseptic disruption	6	
Septic disruption from infection (*Staph. aureus*)	4	
Recurrent deep venous thrombosis	1	

Of the 41 patients treated for previous ulceration, 4 were untraced. The remaining 37 (90%) were carefully followed up for 3 years. During this period ulceration of varying extent recurred in 6 patients (15%).

The cause of recurrence in all cases was trauma to the ankle region. All 6 of the patients had discarded their elastic compression. On the other hand, 6 patients in the series had discarded their elastic compression and had not shown any signs of recurrence of ulceration.

DISCUSSION

As has already been stated, external compression of the superficial veins and capillaries will promote healing of a venous ulcer, and all but a very few of these ulcers

will heal with this therapy. The remainder will heal with bed-rest. Once the ulcer is healed, the problem is to *keep* it healed.

External Compression

It is probable that the type of compression which is applied to the leg for healing the ulcer, if maintained permanently, would keep the ulcer closed. However, the inconvenience of this type of bandaging is clearly apparent, because it requires to be changed at frequent intervals; it requires an expert to apply it properly; the patient is unable to bath the leg; it is not uncommon for skin sensitivity reactions to occur under the bandages. These factors are found irksome by most White patients, but the local Bantu-speaking African, on the other hand, tolerates this form of compression treatment very well.

In view of these disadvantages, the logical step to take after an ulcer is healed is to wear elastic stockings to provide continued compression of the leg. To be effective the stockings must necessarily be thick and strong and, therefore, cause excessive heating and perspiration of the leg. If the patient does not discard the stockings on this account, they are commonly worn until their elasticity is so diminished that they are valueless for compression. The inevitable result of this is recurrent ulceration.

Since superficial-vein hypertension is one of the main causes of venous ulceration, it is reasonable to suppose that an additional safety factor in maintaining healing would be introduced by reduction of this pressure. This is achieved by division of the communicating veins. However, even after this operation, most patients have a degree of swelling of the leg which requires control by external pressure. Crêpe bandages are completely unsuitable for this purpose. Elastic bandages having a loop at one end are preferred by some surgeons, but the most satisfactory means of compression is a strong well-fitting elastic stocking. In addition to providing compression, the elastic stocking affords a degree of protection for the area of healed and fibrotic skin, which was previously the site of the ulcer, and which may now be regarded as a *locus minoris resistentiae*.

Causes of Recurrent Ulceration

Perhaps the most important point in a discussion of this sort is a consideration of the causes of recurrent ulceration. Clearly, the basic factor is a persistence of oedema of the skin and subcutaneous tissue, which impairs the already reduced circulation of the healed ulcer-bearing area. On this basis, the slightest trauma will initiate a new ulcer. The factors responsible for recurrences are, therefore, those causing persistent oedema.

In a patient who has had an operation for eradication of communicating veins, the most likely cause of continued increased capillary pressure and oedema of the ankle region would be a persistent incompetent communicating vein which had escaped the attention of the surgeon. This would be an unusual cause if the surgeon were aware of the anatomical positions of the direct communicating veins. Since there are relatively few of these structures in the leg and since their positions are more or less constant, there is little or no problem in finding them when they are present. To omit to eradicate an incompetent communicating vein is to court recurrence; but is it necessary to ligate and divide competent communicating veins in case they should later become incompetent? Turner Warwick[23] showed that a communicating vein, when subjected to intraluminal pressure, would burst before its valve gave way. If this work is accepted, and Dodd[12] stated that it has been confirmed anatomically and by venography, there would appear to be no indication for ligation of any communicating vein in which the valves are competent. There would seem to be doubtful virtue in removing a normal communicating vein in the upper calf for an ulcer at the ankle.

Cranley *et al.*[21] stated that lymphatic obstruction in venous insufficiency of the leg does not seem to be of major significance. This statement is based on the fact that the clinical presentation of lymphatic disease of the leg is totally different from that of venous disease. However, in considering the physiological processes of fluid interchange, there is little doubt that the lymphatics play a part in the removal of fluid from the tissues. Admittedly, the grosser presentations of lymphatic obstruction are different from those of venous disease; but what of local lymphatic obstruction? In regions where venous thrombosis has occurred and chronic inflammation or ulceration has existed for some time, it is very likely that the delicate lymph channels have been destroyed. In this event, even after the amelioration of capillary hypertension by vein ligation, there may still remain a disturbance in fluid removal and thus persistence of local oedema.

It is a well-known physiological fact that capillary pressure increases with dilatation of the arteriole supplying the loop. Keele and Neil[24] have shown that dilatation of arterioles by heating leads to a rise of capillary pressure of about 20 mm.Hg on the arterial side and about 30 mm.Hg on the venous side. This rise is due to the more direct transmission of the general arterial pressure to the capillaries through the lessened resistance of the dilated arteriole. If a high pressure were applied for a sufficient length of time to the venous side of the capillary loop, the whole loop would ultimately become dilated and the feeding arteriole might also become dilated by this back-pressure. The effect of dilatation of the arteriole increases further the capillary pressure and now the arrangement constitutes, in effect, an arteriovenous fistula. Presumably, a normally occurring arteriovenous anastomosis could also be involved in this process. Arteriolar dilatation could well account for the presence of bright-red blood under high pressure seen at operations on these patients.

If the arteriole has become permanently dilated, and this is particularly likely in the older patient, whose arteriolar media is replaced by fibrous tissue, the high capillary pressure will persist even after vein ligation (Fig. 7). This would lead to persistent skin oedema. Perhaps this is the reason for the failure of lumbar sympathectomy to effect an improvement in cases of venous ulceration. It may also account for the fact that the condition is aggravated by hot weather or standing before a hot stove. It seems possible that, in certain cases, long-continued venous disease may result in complications from superimposition of arterial factors. Whatever the considerations, both practical and theoretical, it is true to say that the majority of patients have some residual oedema of the leg after an operation for venous ulceration.

In 1957, Dodd *et al.*[20] published the results of ligation of communicating veins by the subfascial route in 174 cases. Twelve per cent were fair results (in which the oedema and coarseness of the skin persisted) and 7% were unsatisfactory (in that the dominant symptom had persisted or ulceration had recurred). In 1961, Cranley *et al.*[21] published the results of stripping of the great and small saphenous veins and ligation of the communicating veins in the calf (Linton operation) in 122 cases. Ulceration recurred in 13%.

In the series reported here, there were 37 cases in which the ankle communicating veins were ligated by the extrafascial and subfascial routes. After 3 years ulceration had recurred in 15%. All these recurrences were in patients who had discarded their elastic stockings. In addition, 15% were able to discard their elastic stockings without recurrence of ulceration. Thus, the remainder (70%) wore elastic stockings continuously after the operation and suffered no recurrence.

It appears from these figures that whether an extensive or limited procedure is performed, the recurrence rate is significant. Since oedema of the ankle region is a constant finding in patients with recurrent ulceration, the cause of recurrence is almost certainly trauma applied to an area of residual oedema. The oedema of the integument, whether it be of lymphatic or arterial origin, may be controlled only by pressure from without and this is most satisfactorily achieved with elastic stockings.

It is interesting also to note in Cranley's paper[21] that 15% of patients treated conservatively developed recurrent ulceration and Wiley (in the discussion on this paper) stated that his results were similar. In view of the similarity between conservative and operative treatment, Wiley questioned the necessity for radical surgery in cases of venous ulceration.

Nevertheless, patients who have had ligation of the communicating veins are more comfortable than those who have not. Pain, in particular, is greatly relieved by the operation.

It appears that the best chance of maintaining healing of a venous ulcer is obtained by ligation of communicating veins followed by the *permanent* use of a well-fitting elastic stocking. Not only does the elastic stocking help to control the residual oedema, but it also acts as a mechanical protection against trauma, which in most cases is the precipitating factor in recurrent venous ulceration. More than that, the patient must be warned to avoid any form of trauma to the region of the healed ulcer, since the slightest injury may lead to a new ulcer.

SUMMARY

1. The pathophysiology of venous ulceration is considered. Oedema of the integument and superficial-vein hypertension are regarded as the predisposing factors. These are due to 'blow-back' of blood along communicating veins, from the deep veins in which the valves have been destroyed by previous thrombosis.

2. The possible causes of recurrence are examined. These are thought to be persistence of incompetent communicating veins, local lymphatic obstruction and local capillary hypertension of arterial origin.

3. The most satisfactory treatment is eradication of incompetent communicating veins followed by the permanent use of an elastic stocking. The need to avoid trauma to the ankle region is emphasized.

I wish to thank Prof. D. J. du Plessis for his kindness in providing the facilities for the management of these patients, and Dr. K. F. Mills for permission to publish the details of the cases. My thanks are also due to Profs. P. V. Tobias and R. A. Dart for placing at my disposal the facilities of the Department of Anatomy.

REFERENCES

1. Cooper, A. P. (1837): *Lectures on the Principles and Practice of Surgery*, 8th ed. London. (*Op. cit.*[16])
2. Critchett, G. (1848): Lancet, **2**, 416. (*Op. cit.*[19])
3. Brodie, B. C. (1846): *Lectures Illustrative of Various Subjects in Pathology and Surgery*. London: Longmans. (*Op. cit.*[18])
4. Chapman, H. T. (1853): *The Treatment of Obstinate Ulcers and Cutaneous Eruptions on the Leg without Confinement*, 2nd ed. London: Churchill. (*Op. cit.*[14])
5. Gay, J. (1868): *On Varicose Diseases of the Lower Extremities*. London: Churchill. (*Op. cit.*[17])
6. Homans, J. (1916): Surg. Gynec. Obstet., **22**, 143.
7. *Idem* (1917): *Ibid.*, **24**, 300.
8. Birger, I. (1947): Acta chir. scand., **95**, suppl. 129, 1.
9. Bauer, G. (1942): *Ibid.*, **86**, 1.
10. Anning, S. T. (1952): Lancet, **2**, 789.
11. Atzler, E. and Herbst, R. (1923): Z. ges. exp. Med., **38**, 137.
12. Dodd, H. and Cockett, F. B. (1956): *The Pathology and Surgery of the Veins of the Lower Limb*. London: Livingstone.
13. Cockett, F. B. (1955): Brit. J. Surg., **43**, 260.
14. Sherman, S. (1949): Ann. Surg., **130**, 218.
15. Rémy, C. (1901): *Op. cit.*[17]
16. Trout, H. H. (1929): Arch. Surg., **18**, 2281.
17. Linton, R. R. (1938): Ann. Surg., **107**, 582.
18. *Idem* (1953): *Ibid.*, **138**, 415.
19. Cockett, F. B. and Elgan Jones, D. E. (1953): Lancet, **1**, 17.
20. Dodd, H., Calo, A. R., Mistry, M. and Rushford, A. (1957): *Ibid.*, **2**, 1249.
21. Cranley, J. J., Krause, R. J. and Strasser, E. S. (1961): Surgery, **49**, 48.
22. Felder, D. A., Murphy, T. O. and Ring, D. M. (1955): Surg. Gynec. Obstet., **100**, 730.
23. Warwick, W. T. (1931): *The Rational Treatment of Varicose Veins and Varicocele*. London: Faber.
24. Keele, C. A. and Neil, E. (1961): *Samson Wright's Applied Physiology*, 10th ed. London: Oxford University Press.