The Management

of

GRAVITATIONAL ULCER.

by

ROBERT B. WRIGHT.

CONTENTS

PART I.

INTRODUCTION.

In all the field of medical practice there can be few conditions more perplexing to the physician and more troublesome to the patient than that recurring chronic ulceration of the lower third of the leg variously termed varicose ulcer, non specific ulcer or gravitational ulcer. Such ulcers are characterised by the site of their occurrence - the gaiter area - and the fact that they usually heal with rest in bed but tend to recur when the sufferer becomes ambulant. There are great variations in size and severity of symptoms, some are painful, some are not; some are associated with obvious and gross varicose veins, some are not; some are apparently related to previous deep venous thrombosis and some are not.

The incidence of the condition is difficult to assess: some sufferers live a hermit existence and never come to hospital for treatment and many others spend a life-time and much of their income trying a multitude of treatments and "cures" and, like most sufferers from peptic ulcer, live at terms of more or less uneasy peace with their ulcer. At a recent discussion on varicose ulceration in the Section of Surgery of the British Medical Association, Mr. J.B. Oldham declared that it had been estimated that five in every thousand of the working population of this country were suffering from this affliction and A.M. Boyd suggested that there/

there were probably 50,000 cases within a 50 mile radius of Manchester. In Denmark the annual cost to the government of people disabled by varicose ulcer is in excess of two million crowns (Roholm 1941) and in Sweden, between three and four thousand people receive annuities every year on this account (Birger 1941). In the past year in the Varicose Vein Clinic at the Western Infirmary 74 new cases of gravitational ulcer have been seen. As the majority of these patients are married women with families, a great deal of responsibility and little time for rest, their management presents a very important and considerable social and medical problem.

Despite the prevalence of the condition treatment remains difficult and far from specific. While healing can usually be achieved the maintenance of that healing can never be guaranteed. A tremendous amount of time and energy has been expended on the problem yet few practitioners, even now, approach these cases with any feeling of confidence or enthusiasm. This attitude has been well described by Sir Benjamin Brodie (1846) thus - "....it is probable that many of you may pass by the bedside of such a patient without thinking it worthy of attention..... although the patient will not probably die of this disease, yet, without great care it may render her miserable. The disease may be much relieved by art."

HISTORY.

Hippocrates (2000 B.C.) noted the response of ulcers of the leg to the elimination of the effects of gravity. He taught that "...those who have an ulcer of the leg ought neither to sit nor to stand but to lie on a bed". He also recognised the value of binding up the legs in such cases. Marcus Aurelius Severinus (1580) stated that he had seen ulcers of two, four, seven and even ten years standing heal up completely after the surrounding varicose veins had been successfully removed by operation -"....the ulcers disappear just like plants which are drying out when the brook is diverted."

Ambroise Paré (1678) comments in his Treatise on Surgery "...ulcers whereinto a varix or swollen vein continually pours its matter are hard to be cured." He advocated ligation and division of the prominent varicose vein above the ulcer, but the discovery of the circulation of the blood by Harvey appeared to show that Paré's hypothesis was wrong and the method of treatment was abandoned, only to be reintroduced two centuries later by Trendelenburg (1881). Benjamin Brodie (1848) advocated supportive dressings and described in detail his method of binding the affected leg from toes to knee with strips of adhesive plaster, of containing the discharge from the ulcer and of keeping the patient ambulant. This method, somewhat refined, was popularised by Dickson Wright (1931) and/

and remains probably the best basis of treatment. John Gay (1868) in his excellent Lettsomian lectures stated that he had noted no constant relationship between the extent, severity or even the presence of varicose veins and pigmentation and ulceration. From clinical obser: -vations and post mortem dissections he concluded that "....ulceration is not a direct consequence of varicosity but of other conditions of the venous system with which varicosity is not infrequently a complication, but without which neither one of the allied skin affections is met with". He noted that in many cases, but not all, post mortem dissection revealed abnormal conditions of the deep veins.

Following the writings of Trendelenburg (1881) operative treatment of varicose veins directed at preventing retrograde flow in the affected veins became widely practised and the attention of surgeons was again focussed on the problem of the ulceration of the lower leg so commonly seen in association with varicose veins. But despite the most thorough and varied surgical assaults recurrences of both varices and ulcers continued. Further, it had long been known that in some cases of typical ulceration no varices were demonstrable.

Homans (1916) introduced the term postphlebitic ulcer which he applied to these non-specific ulcers of the lower leg which occurred in limbs that had previously been/

been the seat of phlebitis, superficial or deep. He, like Paré, Gay and others noted that the ulcer often rode on a vein - the so-called feeder vein, whose interruption Paré had advocated; he also noted that the ulcer might sit on a perforating vein. He, too, noted that often there was no apparent abnormality of the superficial veins and thus many could not justifiably be called varicose ulcers. Homans, at that time, considered that postphlebitic ulceration and induration followed only a deep phlebitis and might have associated superficial varicose veins or a straight incompetent internal saphenous vein which had been the seat of phlebitis. A typical history was that, three months to twenty years after a white leg a patch of induration or oedema appeared on the lower medial aspect of the leg and soon, following some minor trauma, broke down to form an ulcer which could be healed by rest and elevation of the limb. He stated "....it has never seemed that the veins were greatly concerned with the pathology of the condition; the most notable pathological feature is the thickening and hardening of the tissues which is most marked on the surface of the aponeurosis: the site becomes ischaemic from strangling of the blood vessels by fibrosis and this vicious cycle perpetuates the lesion." Homans believed that individuals first seen over the age of fifty who had had the lesion for years were incurable, and thatamputation of/

of the limb was the correct treatment: the early lesion would heal with rest and elevation and be controlled by support: for the average ulcer resistant to simpler measures he advocated wide local excision of the ulcer bearing area down to and through the deep fascia and the application of a thick split skin graft to the exposed muscle or periosteum - this he claimed gave permanent cures. He recommended nerve section and lumbar sympathectomy to alleviate pain and increase the peripheral circulation.

Mme. Bellocq (1925) showed that in the gaiter area the arterial plexus of the skin has a very wide mesh and the vessels are of a smaller calibre on the medial than on the lateral aspect; she thought this might explain the fact that ulcers most commonly occur on the inner aspect of the lower third of the leg.

In 1931 Dickson Wright introduced the term gravitational ulcer and advocated the adoption of measures to obviate venous stagnation in the lower limb which he believed to be the fundamental underlying causative factor. His method of treatment by elastic compression bandaging was a revival of the principles of Hippocrates and Brodie and had the great merits of being cheap and successful and of avoiding hospitalisation. In addition to compression bandaging he advised sclerosing of superficial veins by injection, and grafting unresponsive ulcers with emulsions of epidermis. v. Meisen (1932) furthered the view that

venous/

venous stasis in the lower limb was the decisive factor in the causation of gravitational ulcer. Birger (1941) and Bauer (1942) stressed the importance of deep vein thrombosis as a causative factor. Bauer stated that "...varices have almost nothing to do with the formation of ulcers - deep seated thrombosis everything" and advocated deep vein interruption as the method of choice in treatment. Bauer practised popliteal vein ligation. Linton and Hardy (1947) and Buxton and Coller (1945) favour interruption at the level of the superficial femoral vein to prevent reflux in the deep veins, but all also practised compression bandaging in addition.

PART II

GENERAL CONSIDERATION OF THE PROBLEM.

It is generally accepted that gravitational ulcer occurs in the lower portion of a lower limb in which there is venous stasis, but it is a notable feature that the ulcer rarely bleeds and is always set in a zone of increased fibrosis affecting the layer between skin and deep fascia. If the stasis is alleviated by rest or support, in the majority of cases the oedema resolves and the ulcer heals more or less soundly. When, by any method, a sound fibrous scar results recurrence of the ulcer is always at another site in the indurated zone. It would seem that the alleviation of stasis permits restoration of local vascularisation and thus of healing. To obtain permanent healing the aim must be first to heal the ulcer and then to prevent recurrence, i.e.

(a) to restore local vascularisation and

(b) to prevent venous stasis or control it.

The causes of venous stasis in the lower limb may be of local or central origin. Local stasis may be due to arterial or venous insufficiency. Arterial insufficiency alone may, rarely, result in ulceration indistinguishable from gravitational ulceration, but peripheral gangrene is more common. In the material to be discussed gross arterial deficiency was seldom a feature but when present it made treatment extremely difficult/

difficult. It would seem that the underlying causative factor in producing venous stasis in these cases must usually be venous.

The factors which influence the venous return from the lower limb are

1. Vis a tergo..... the arterial inflow.
2. The pump action of the limb musculature, which, to be most effective demands
3. Soft compressible veins, and
4. Competent vein valves.
5. Heart action producing a suction effect in the cavity veins.
6. Obesity.
7. Gravity.

In the normal limb venous blood flow is always centripetal no matter the attitude or activity of the subject and this is ensured by valve competence. Any factor which destroys or impairs valvular competence allows of venous reflux and stasis, i.e. a state of venous insufficiency. It is my belief that deep venous insufficiency is the fundamental cause of gravitational ulceration and that its elimination or compensation will result in control of that condition.

NORMAL CIRCULATION

Anatomy.

The venous drainage of the lower limb is made up of two main systems, the superficial and the deep veins separated by the deep fascia and interconnected by a variable number of communicating veins. The superficial veins on the dorsum of the foot drain into two main trunks, the internal saphenous vein in front of the medial malleolus and the external saphenous vein behind the external malleolus. These main superficial trunks are single, relatively straight and provided with numerous bicuspid valves guarding the junctions with communicating veins and their entry into the femoral and popliteal veins. They have very constant relationships to the deep fascia: the internal saphenous vein, throughout its length, lies on the deep fascia and is bound to it by a sheath from that investing layer in contrast to its tributaries which tend to be tortuous and ramify in the coarse subcutaneous fat as a wide interlacing plexus. The external saphenous vein, constant in origin, varies considerably in its mode of termination; in the majority it pierces the deep fascia in the mid line about mid-calf level. The variations have been well described by Barrow (1949).

The principal deep veins below the knee are the venae comites of the main arterial trunks, the anterior tibial, posterior tibial and peroneal arteries. These veins/

veins are frequently double trunks and into them drain the intra muscular veins. They contain numerous bicuspid valves giving them a beaded appearance in venograms and drain into the large single, or double, popliteal vein which has a varying number of valves, usually two or three. The popliteal vein is continued upwards as the femoral vein which contains few valves and ultimately as the iliac vein in which there is only one valve guarding the entry to the body cavity. Cavity veins have no valves and no muscle cuff pump, the deep veins of the limb have both and on their function depends the adequacy of the deep venous return.

All these features have been beautifully demonstrated by dissection and illustrated by John Gay (1868). In particular he demonstrated experimentally how effective the tenuous vein valves were in preventing retrograde flow of fluid in the leg veins.

Haemodynamics

To obtain a normal deep venous return the deep vein valves must be competent, the vein wall must be soft and elastic and surrounded by skeletal muscles capable of exercising a massaging pump action. Anything which interferes with valve action or pump action may produce stasis. Valve incompetence permits venous reflux and this will throw a strain on successively lower valves, if progressive and ultimately on the valves of the communicating veins/

veins which inter-connect the deep and superficial systems. If these valves in turn become incompetent the strain will be transferred to the superficial system and this is, I believe, the essential cause of most varicosities of the superficial veins, i.e. the primary defect is in the deep venous system (Wright, 1950) - a deep incompetence. If the compensation of the muscle cuff is insufficient to overcome the deficiency, gravitational effects ensue, e.g. oedema, induration and ulceration. This is the state of deep vein insufficiency.

Once valves have become incompetent from damage to the cusps they are irreparable and reflux inevitable. It can only be prevented by vein section. This, in turn, diverts the strain towards other potentially weak points where reflux may later appear, the condition being one of disordered haemo-dynamics. External support, elastic or semi-rigid, may aid the muscle cuff enough to maintain a balance in cases where the muscle cuff itself is insufficient.

Under what conditions will stasis occur?

(a) Superficial varicose veins with a positive Trendelenburg sign, always an indication of some degree of deep vein incompetence.

(b) Deep vein incompetence with communicating vein incompetence producing superficial varicosities with a negative Trendelenburg sign.

(c)/

(c) Deep vein incompetence from valve deficiency, vein wall rigidity and narrowing of the lumen resulting from previous deep phlebitis. In this group there may or may not be associated superficial varices.

Gay, in post mortem dissection of ten legs the seat of gravitational ulcer, found the deep veins partially obstructed by thrombus in six cases and dilated and tortuous in three.

It seems clear that the deep veins are the most important element in the venous system of the leg and that in gravitational ulcer one must treat the deep vein insufficiency while accepting that some degree of underlying deep vein valve incompetence is permanent.

Plan of Management.

If this reasoning were correct a plan for the treatment of gravitational ulcer would be,

i. To demonstrate reflux in and into superficial veins and to prevent it, and to eliminate stasis in these veins.

ii. To demonstrate and locate communicating vein incompetence and take steps to eliminate it.

iii. To disperse oedema and encourage venous return through the deep veins.

iv. To restore local vascularity and promote healing.

v. To support the skeletal muscle cuff and maintain its activity.

vi. To reduce the effects of gravity and obesity.

PART III.

PRACTICAL INVESTIGATION, TREATMENT & RESULTS.

In the earlier phases of the study cases of uncomplicated varicose veins were used to evolve methods of demonstrating and locating the sites of superficial and communicating vein reflux and these were applied in the assessment of the cases with ulceration.

Demonstration of reflux.

Demonstration of reflux into the superficial veins is usually easy. The method of Trendelenburg was employed and this test was positive in the majority of cases of varicose veins when first seen (Wright, 1950). On performing the test it is striking to note how the reflux down the saphenous vein occurs immediately and rapidly as soon as the controlling finger is released - much more blood flows back than one would expect from a mere spill over from the femoral venous flow. Further, in cases with a positive Trendelenburg sign, if a watching finger is placed on the line of the internal saphenous trunk and the patient instructed to cough an impulse is felt - in severe cases this impulse can be felt, and even seen, at ankle level. This clearly indicates that increases in intra-abdominal pressure are being transmitted to the superficial veins of the limb and

therefore/

therefore that at least the iliac valve and first saphenous valve must be incompetent, i.e. a positive Trendelenburg sign with a cough impulse is a clear indication of what might be termed first degree deep vein imcompetence.

The regularity with which varicose veins of the internal saphenous vein will recur, from above, after high ligation operations unless the internal saphenous vein is tied flush with the femoral vein above all branches is striking. In 60 consecutive cases of recurrence the Trendelenburg test was positive in 55 (Wright, 1949). Abdominal reflux into the internal saphenous vein can only be prevented by ligation at the sapheno-femoral junction. If superficial varices recur after adequate high ligation, reflux through incompetent communicating veins will be demonstrable and can be prevented by division of such communicating veins. This, again, confirms that the primary fault is in the deep veins. When superficial varices recur after treatment they recanalise from above downwards and this is so after inadequate ligation and after sclerosing injection. All these facts stress the point that the primary defect is in the deep veins and that superficial varices are a reflection of deep vein incompetence.

Prevention of Reflux.

Reflux into the main superficial veins can be prevented by division of the veins surgically at their points of entry into the deep veins (Homans, 1916), and to be effective must be exactly at these points (Wright, 1949), and by section of incompetent communicating veins. Stasis in the superficial plexus can, thereafter, be reduced by obliterating these veins by sclerosant injection or excising them. In this study almost all cases were treated as out-patients and sclerosants were therefore used.

Sclerosant Injection.

It was appreciated that sclerosing fluid injected with the intention of producing a chemical phlebitis carried the danger of damage to the deep veins and communicating veins and their valves. Experience shows this to be a theoretical risk provided the sclerosant is viscid, injected high and in small volume, and the patient's muscle tone is not reduced by general anaesthesia. The solutions used were Sodium Morrhuate 5 cc., Ethamolin 5 cc. in 1947-8, but, in view of the occasional severe systemic reactions and the commonly severe and painful peri-phlebitis so induced, were abandoned in favour of PGGG solution (Phenol 8 gms. Glucose (50%) 240 cc., Glycerin 120cc, Gelatin 2 gms.), prepared in the hospital pharmacy. This solution produces better

obliteration/

obliteration more rapidly, more extensively and without any noteworthy periphlebitis or systemic upset. Only one case of probable chemical pulmonary embolus has been noted in over two thousand cases who have received an injection of, at most, 5 cc. of PGGG.

Experimental injection of a radio-opaque sclerosing fluid similar to that used by Boyd (1948) and radiography showed that the fluid passed very rapidly into the deep veins through competent communicating veins in the upper thigh. In the deep veins this spill is rapidly diluted and dispersed and damage to valves is likely to be minimal. If the fluid is injected at a high level escape through communicating veins will only tend to damage the upper femoral or iliac valves, which in almost all cases are already demonstrably incompetent. This, of course, is not so if the fluid is injected below the knee, as advised by Boyd and Robertson (1947) when many deep vein valves may trap the sclerosant and be damaged. Early in the series a few cases of transient deep vein blockage followed by oedema due to this cause were seen and thereafter the method of below knee injection was abandoned. For the same reason retrograde injection through a ureteric catheter or Stevenson's needle (Dodd 1940, Foote, 1944) was not employed. In all cases the saphenous vein was ligated flush with the femoral vein, divided and a retrograde injection of 5 cc. PGGG, using a fine needle, performed at the highest level

of/

of the distal end of the divided vein. By this means thrombosis of the whole saphenous system to calf or ankle level is frequently noted before the patient leaves the operating table and no case suggestive of deep vein block has been encountered.

The external saphenous vein usually pierces the deep fascia about mid-calf in the mid-line and therefore the deep fascia must be incised to locate the termination of the vein. A vertical incision in the mid-line was found most useful and the deep fascia was always sutured over the divided stump.

Location of Communicating Veins.

Communicating veins are of two types (Warwick, 1931), direct communicating veins which are usually relat: -ively short and straight, frequently double, and join the main internal or external saphenous vein to the deep veins (Fig. I),

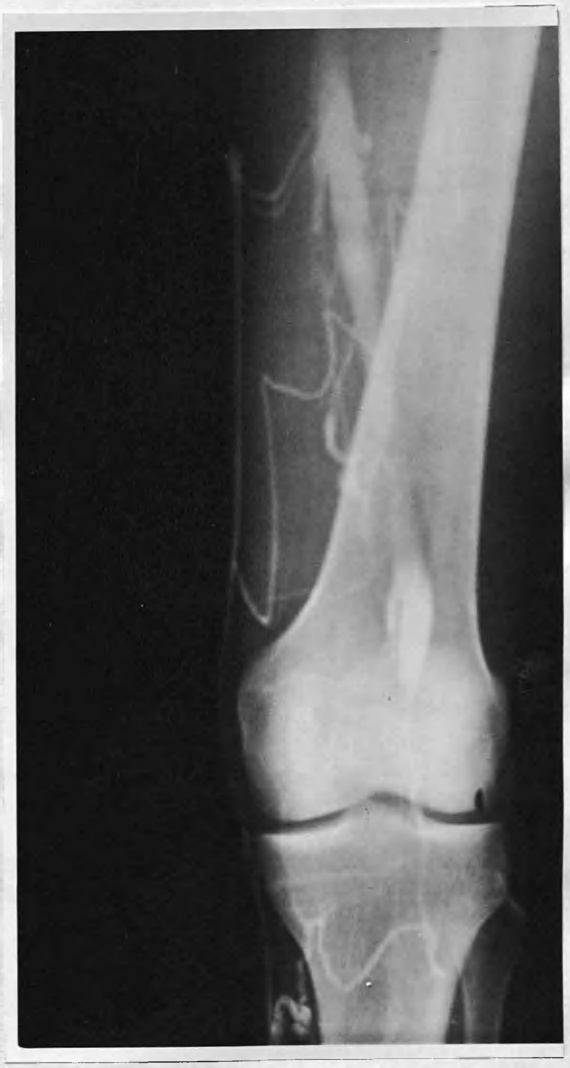


Fig. Ia. Fig. Ib.

Ascending venograms showing (a) Direct communicating veins some transverse and in pairs, some oblique, and (b) long oblique communicating veins in thigh.

and indirect communicating veins which tend to be longer, tortuous and often multiple and connect the plexus of superficial veins to the deep veins via the intramuscular veins (Fig. 2).

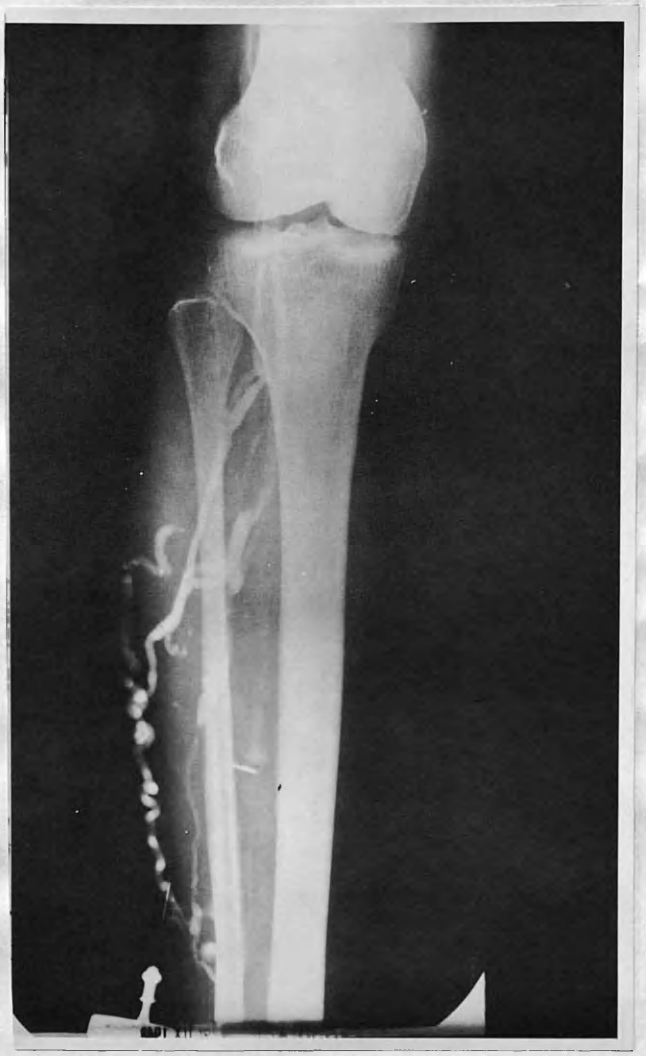


Fig. 2.
Ascending Venogram showing Indirect communicating veins.

The direct communicating veins vary greatly in number and distribution but are fairly constant at certain levels, viz. upper third of thigh, mid-thigh, through the adductor canal/

canal and about one inch below the medial tibial condyle and at the junction of the lower and middle thirds of the leg. The enormous variety can only be clearly demonstrated by venography (Fig. 3).

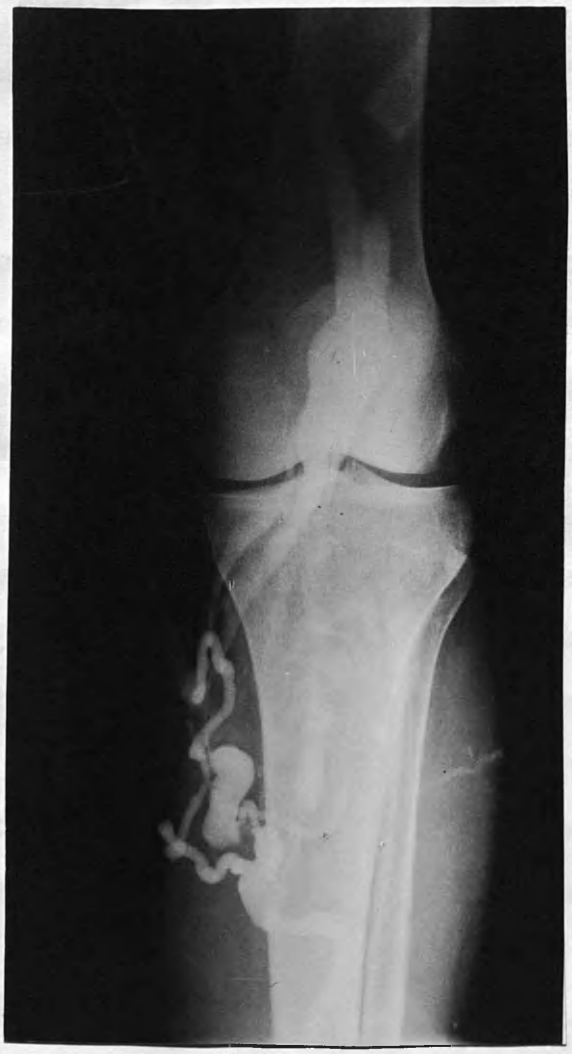


Fig. 3.

Ascending venogram showing multiple indirect communicating veins.

The indirect communicating veins are quite inconstant in site and number.

Numerous tests, all theoretically sound, have been/

been described for the location of incompetent communicating veins. All regard the venous system of the lower limb as if it were a large U tube with a varying number of inter-communications between the two limbs, (i.e. the saphenous vein and the femoral vein) running more or less horizontally. But this is not so. The course of communicating veins of both types may be long and tortuous. Oedema, the coarse subcutaneous fat above the knee, induration below the knee, the high tension in the incompetent saphenous vein on straining, and the presence of two bones in the deep compartment of the leg make adequate compression of the superficial veins by a tourniquet difficult or impossible without seriously impeding the flow in the deep vessels. The tourniquet tests of Ochsner and Mahorner (1936), Perthes (1895) and Slavin (1948) were all tried but found lacking for the reasons detailed. In more than one patient with gross superficial varices a pneumatic tourniquet was placed about mid-thigh and pumped up to a pressure of 140 mm. Hg. and then a small volume of 25 per cent Pyelosil was injected into the saphenous vein above the tourniquet which was slowly deflated while the limb was watched on the X-ray screen and, in many cases, at a pressure of 100 to 120 mm. of mercury the pyelosil was seen falling down inside the tourniquet - the patient was standing erect but not straining. This provides a clear demonstration of the fallacy of the tourniquet being thought "tight enough

to/

to occlude only the superficial veins without impeding the flow in the deep vessels."

It became clear that the most accurate method of locating communicating veins was by venography, but this is expensive, time-consuming, and requires a special and not easily acquired technique to be of value (Dow, 1951). While venography can demarcate accurately the anatomical distribution of communicating veins, it is by no means easy to demonstrate convincingly which are incompetent.

Location of Communicating Vein Incompetence.

In co-operation with Dr. J.D. Dow comparisons between clinical, venographic and operative findings were made in a large series of cases of varicose veins and the following practical clinical method of locating communicating vein incompetence was developed. The Trendelenburg test is first performed to demonstrate the presence or absence of abdominal reflux. The test is then repeated and the pressure at the fossa ovalis maintained for a full minute while the superficial veins are observed. If there are no grossly incompetent communicating veins the superficial veins fill slowly and uniformly from below; if superficial varices appear immediately or rapidly the leg segment is noted and also whether there is obvious reflux from above knee level.

The/

The patient is then instructed to exercise the calf muscles actively, if the varices become more full and tense the communicating vein valves are incompetent. If it is noted that superficial varices fill from above the knee the level of digital compression is lowered to the level of the adductor tubercle, and its adequacy tested by applying the cough impulse test with a watching finger on the line of the main trunk distally, and the test repeated. The actual site of emergence of the communicating vein through the deep fascia can often be felt with a finger tip, especially in the case of indirect communicating veins. If such a fascial defect is felt the test is repeated with one finger occluding the main trunk at the fossa ovalis and another the defect in the deep fascia: if this lower finger is at the site of emergence of the incompetent communicating vein the varices will not appear until the finger is removed. In cases where the Trendelenburg test is negative the second test is still applicable and accurate.

By these methods most grossly incompetent communicating veins can be located. If any are missed and not dealt with surgically re-appearance of superficial varices after operation will point to their location and permit of their subsequent section. Similarly, small and previously competent communicating veins may become incompetent later from the continued strain of deep vein incompetence emphasising again that the condition is dynamic and not static.

Assessment of the state of the deep veins.

The usual sequence of events in the development of gravitational ulcer is venous stasis - pigmentation of skin - induration - oedema - subcutaneous fibrosis - diminished local vascularity - minor trauma - indolent ulcer. The induration which precedes the ulceration varies considerably in extent, from a patch on the antero-medial aspect of the leg at the junction of the lower and middle thirds (Fig. 4),

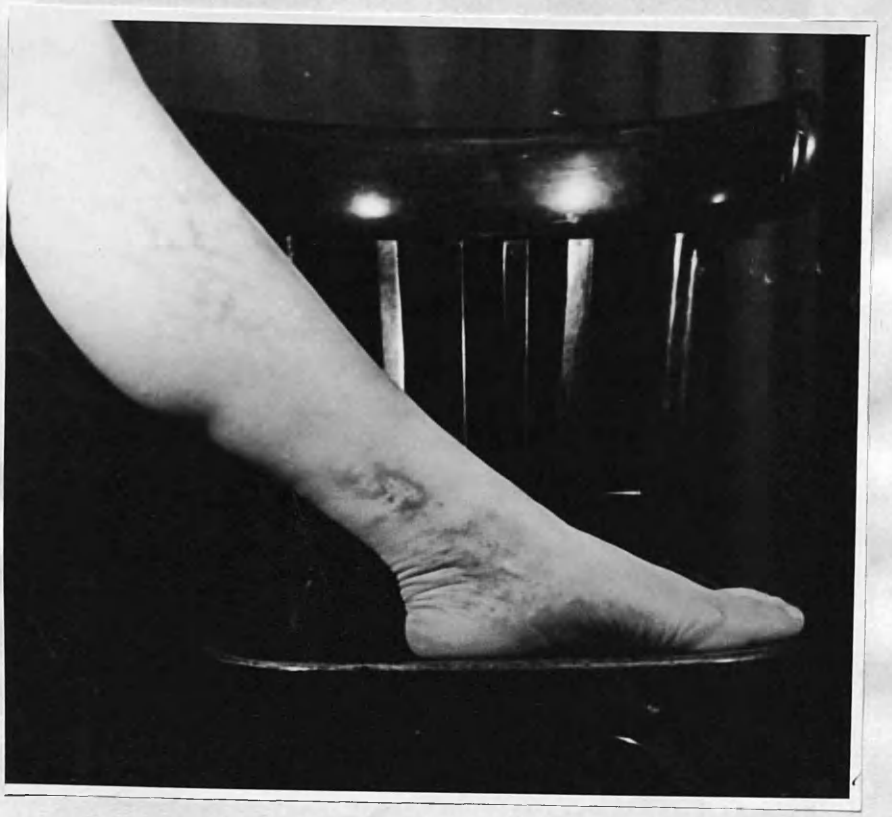


Fig. 4.

Patch of induration and "feeder vein" - pre-ulcer stage.

to an encircling gaiter which may be a narrow cuff or involve the lower two thirds of the leg - the "bottle leg" (Fig.5.).

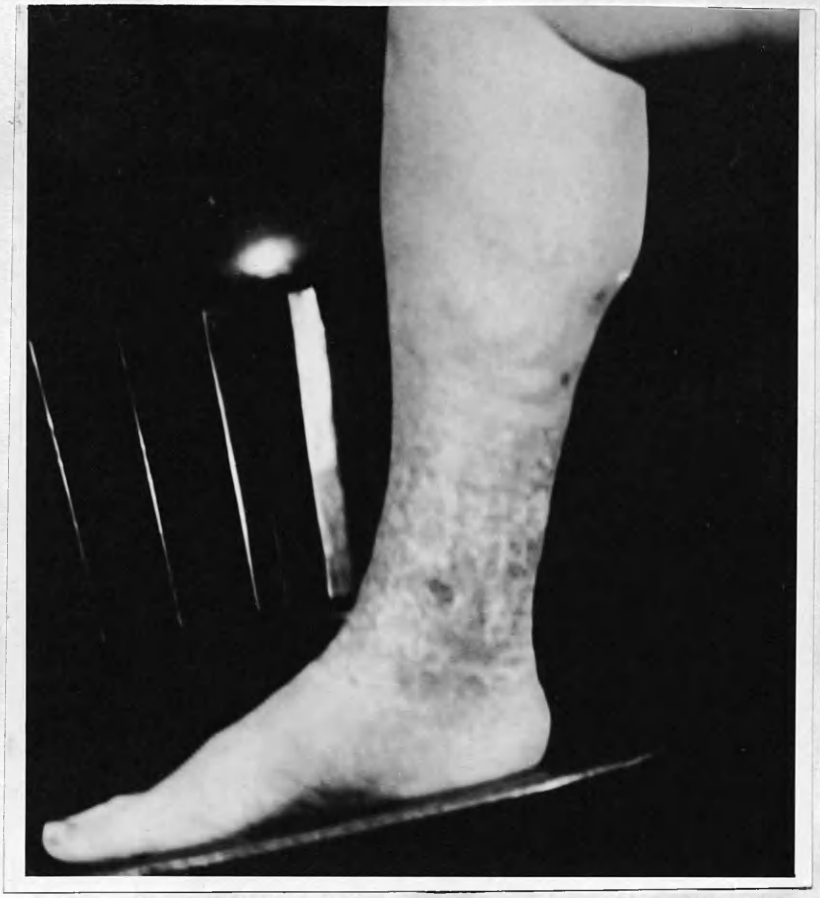


Fig. 5.

"Bottle Leg": high gaiter induration. This patient had a previous deep vein Thrombosis.

In the first type the patch of induration, and later the ulcer, often appears to sit astride a prominent superficial varicose vein (Fig. 6) - the feeder-vein of Paré: this is the typical varicose ulcer of Brodie (1846).

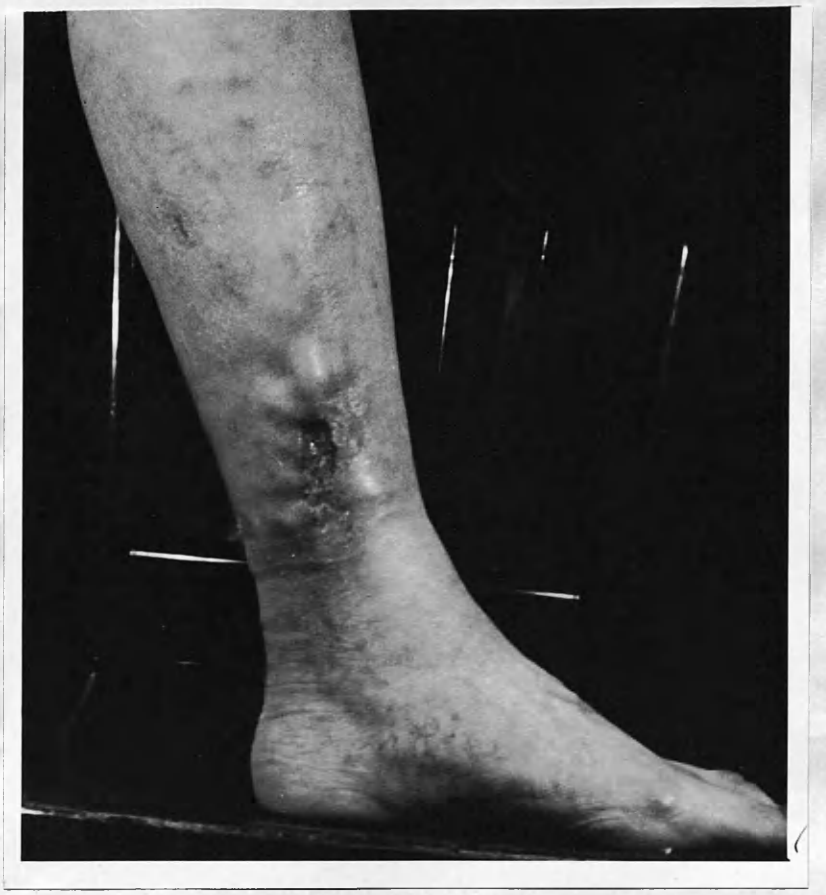


Fig. 6.

Varicose ulcer with "feeder vein".

In the second type the ulcer may be single or multiple (Fig. 7)

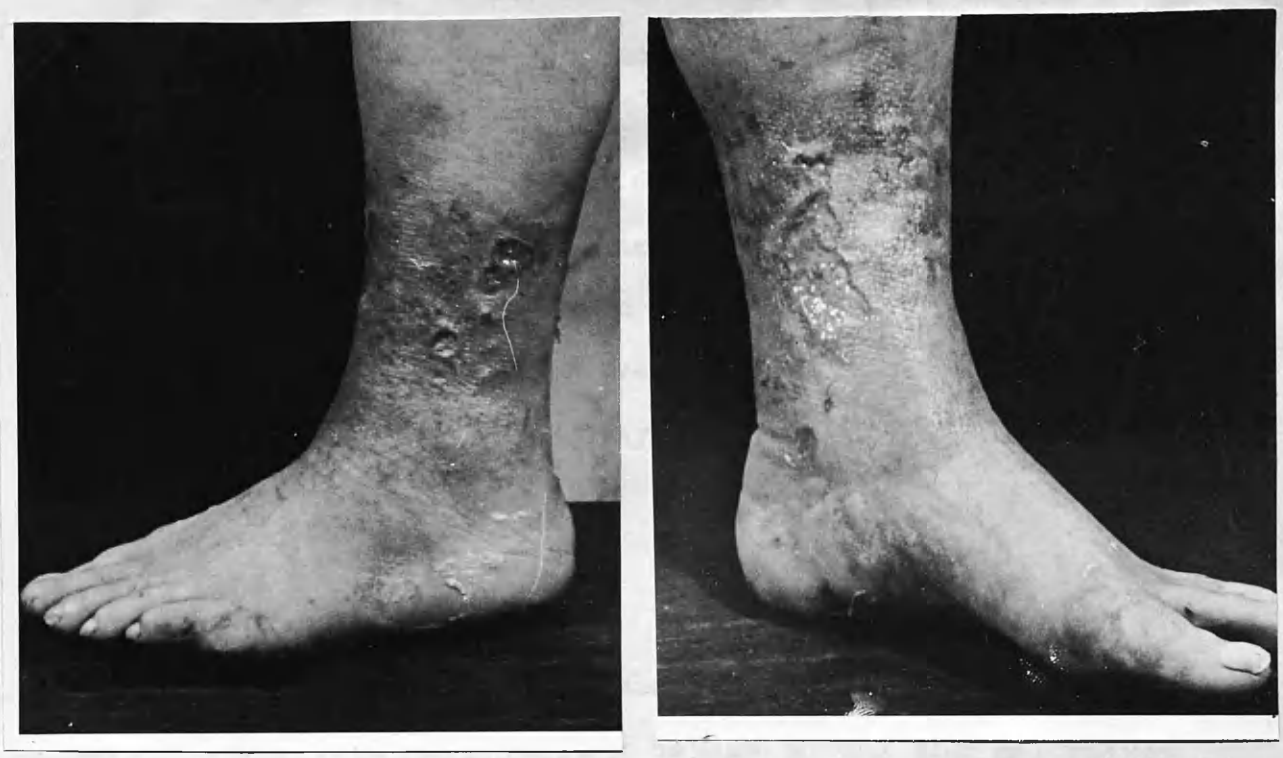


Fig. 7.

Post phlebitic leg - multiple ulcers on both aspects of the indurated gaiter zone.

and at the same site, but there are often no evident associated varicose veins nor any history of such. In a proportion of these cases there is a clear history of antecedent deep vein thrombosis or superficial phlebitis, and this type has been termed the post-phlebitic ulcer by Homans (1916) and others. Some authorities recognise two types of gravitational ulcer,

(1) varicose ulcer, where there is an ulcer associated with obvious varices and no gross oedema.

(2) post-phlebitic ulcer, in which oedema is always evident and varices may not be obvious.

John/

John Gay (1868) on the basis of post mortem dissection believed that "ulceration when it exists with varicosity but without other complication is a coincidence and not a consequence of vein disease". He also noted that "after deep thrombosis the channels are to a great extent restored by contraction of the clot and eventually organic combination with the vein wall" - a simple fact often lost sight of to this day.

Effects of Thrombosis.

The effects of thrombophlebitis in any part of the venous system are, varying damage to the intima, valves and vein walls with, in the acute phase, complete blockage of a varying extent of the lumen by thrombus. Later, as resolution takes place, fibrosis of the valves, walls and thrombus produces valve incompetence, loss of elasticity and recanalisation. The end result is a varying extent of vein incompetence and narrowing of the lumen - a state of venous insufficiency, compensation for which must be obtained by adequate collateral circulation if a normal venous return is to be achieved. Whether this last can be achieved or not depends largely on the extent of the thrombophlebitis and the previous competence of the affected veins.

Deep vein thrombosis may occur in patients with previously/

previously normal veins or in patients with superficial varices. From clinical observation it seems that the presence of superficial varicose veins, itself an indication of some degree of deep vein incompetence, may well indicate a predisposition to the development of deep vein thrombosis. In this series of 284 cases of gravitational ulcer there was a history of definite or probable deep vein thrombosis in 37, and, of these, 27 had a clear history of previous varicose veins. In only 7 did the history indicate that the varices had followed the thrombotic incident.

The damage to the deep veins inflicted by thrombosis is essentially threefold, first, loss of valves allowing venous reflux, second, loss of calibre causing impediment to blood flow - and this will affect both onward flow and reflux, and, third, loss of elasticity diminishing the efficiency of the massaging effect of the muscle cuff pump.

Investigation of results of White Leg.
Clinical Assessment.

Before attempting to evolve a method of assessing the degree of deep vein insufficiency it was considered profitable to try and obtain fuller information on the possible aetiological relationship of previous deep vein thrombosis to the subsequent development of gravitational/

gravitational ulcer. From the Medical Officer of Health of Glasgow a nominal roll of all cases of puerperal white leg occurring in the Corporation Maternity Hospitals during the period 1935-45 was sought. The total available roll was 110 cases - a fire in the records office had destroyed a large number of records. All these cases were circularised and later a questionnaire was sent, but unfortunately, despite repeated efforts and probably largely because of post-war changes of address, only 35 cases were traced and, of these, only 29 reported for clinical examination. The results of this investigation are summarised in Table I.

Varices Present BEFORE Thrombosis	Varices Present AFTER Thrombosis	Total with Varices	No Varices	No. with Ulcer	Interval in Years from thrombosis to Ulcer							
					2	4	6	8	10	12	14	16
6	6	12	17	5			1		1	1	1	1
r 0	4	4	2	0								

These numbers are too small for statistical analysis, but coupled with similar data for the series of cases treated (Table II) they confirm certain clinical impressions viz.

I. varicose veins following phlegmasia alba dolens are not common.

II. the commonest disability after phlegmasia alba dolens is oedema of the leg and ulceration is relatively less common.

III. bursting pain is not a constant feature of the post-phlebitic leg.

Table II

	No. of Ulcer Cases	No. of Cases with Varices	No. of Cases with History of Deep Thrombosis	No. of these with Previous Varices	No. where Varices followed Thrombosis	No. with previous Superficial Phlebitis.	No. where Varices followed Superficial Phlebitis
Males	98	95	10	5	2	17	2
Females	186	181	27	22	5	31	1

Many regard permanent deep vein blockage to be the usual late result of deep thrombosis and regard associated varicosities of the superficial veins as an over distended but necessary collateral circulation (Franklin, 1937, Ochsner and Mahorner, 1939, Rogers, 1939, Coombs, 1940, Foote, 1944, Anning 1950, Rivlin 1951). But, if the femoral vein in a normal leg is divided between ligatures oedema is present only for about 48 hours when a collateral circulation of deep veins opens adequately, and superficial congestion and varices do not result. I have had the opportunity to observe two cases in which the femoral vein had been divided and injected in retrograde manner with a sclerosant. In neither case did new varices appear and the resulting oedema in one has been improved by extirpation of previously present superficial varices. The other case, a young adult male, has gradually and spontaneously developed an adequate collateral circulation and the oedema has disappeared. If superficial varices were the usual collateral circulation to

a/

a deep vein block this would entail destruction of the communicating vein valves over a considerable extent to allow reflux into the superficial veins: such multiple incompetent communicating veins are not commonly seen. Further, if the superficial veins were essential, one would expect their removal to embarrass the venous circulation and the oedema to worsen: practical experience shows the reverse to be true.

After superficial phlebitis occurring naturally or chemically induced, as in the injection treatment of varicose veins, recanalisation of the thrombus is the rule and, in cases of varicose veins, can be observed to occur from above downwards. Such thrombus is much more adherent to the vein wall and associated with more severe toxic or chemical damage to the intima than the "quiet" thrombus of deep thrombosis and would be expected to produce more permanent obliteration of the lumen. Experience shows that to obliterate permanently the lumen of a vein subject to stress without destroying the entire vein wall is virtually impossible.

For these various reasons recanalisation of deep veins is to be expected after deep thrombosis. While a complete persistent block is not to be expected, a reduction in deep vein efficiency is certain and it would seem helpful in attempting to formulate rational treatment to try to assess the degree of this deep vein insufficiency.

The popular test employed for this purpose - and it is usually regarded as a test for deep vein blockage (Rivlin 1951) - is Perthes test: "a venous tourniquet is applied below the level of the lowest incompetent perforating vein, so as to occlude the superficial but not the deep veins. The patient then walks briskly about for three or four minutes. If the deep veins are thrombosed, then the occlusion of the superficial veins by the venous tourniquet has shut off the only route by which the limb blood can return to the heart, and as the patient walks, the veins below the tourniquet do not decrease in size and may even swell and cause pain." This test is quite impractical for many reasons; the lowest incompetent perforating vein may be at or below ankle level, the induration of the lower leg may make compression of the superficial veins by a tourniquet impossible without so impeding both arterial inflow and deep venous return as to cause pain and distension of varices without exercise, also, venous reflux in incompetent varicose veins on exercise can cause distension below an inefficient tourniquet. As already described, a modification of this test was employed in the location of incompetent communicating veins, themselves an indication of deep incompetence: but it could not be used to assess the degree of deep vein insufficiency of other nature.

Two other methods of investigating the state of the/

the deep veins were tried, viz. venography and pressure tests.

In a post-phlebitic limb with varices, if the main varicose trunk is occluded at the sapheno-femoral junction and the limb elevated the veins empty rapidly. Similarly, if a varicose segment is isolated between two superficial tourniquets (Warwick, 1931) and the limb elevated the loop rapidly empties. Both these tests demonstrate that a deep venous circulation is patent, this may be either a recanalisation of the previously thrombosed vein or a collateral circulation. At operations for varicose veins in post-phlebitic cases opportunity was taken to explore the femoral vein: while in many cases it appeared normal, in others it was thickened and matted to the femoral sheath, but even in such cases blood flow in the vein could always be demonstrated by digital stripping showing that at least partial recanalisation of the main vein was usual.

Venography of deep veins.

In conjunction with Dr. J.D. Dow a series of cases was studied by venography. By ascending venography, provided a proper technique was employed to compress the superficial veins (Dow 1951) a patient main deep trunk was invariably demonstrable (Fig. 8).

Fig. 8a

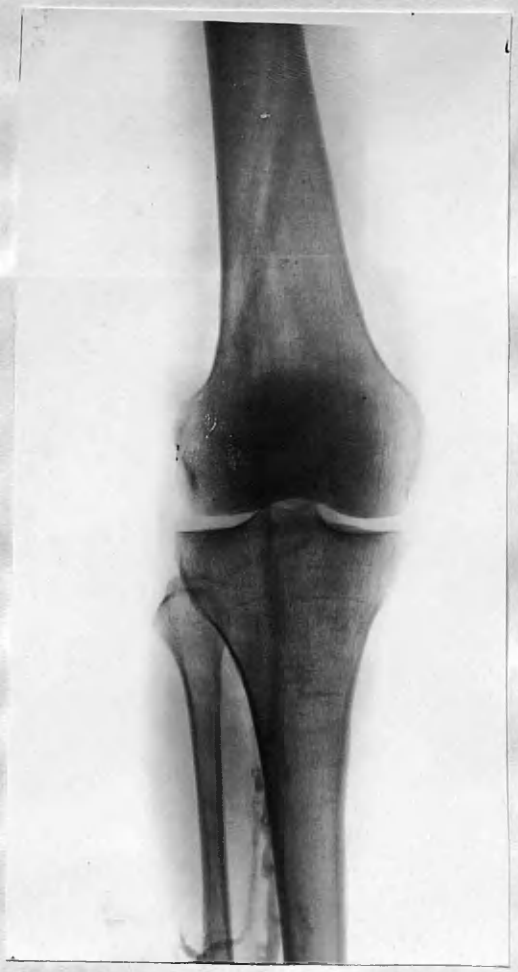


Fig. 8b

(a) Ascending Venogram (Case No. 19) in patient who had deep vein thrombosis 17 years previously. Patent main deep vein visible.

(b) Ascending venogram in patient who had puerperal white leg 42 years previously and ulceration of 10 years duration. Reflux into an incompetent communicating vein from a patent deep trunk can be seen. Popliteal and femoral veins look normal.

It was not possible to comment on the efficiency of this deep venous trunk. A further series of cases was examined by retrograde venography using a technique similar to that of Bauer (1948). As recorded by Dow, in 26 cases of known previous/

previous deep thrombosis the structure of the deep veins appeared normal in 15 (Fig. 9) in the others variations from the normal were noted.

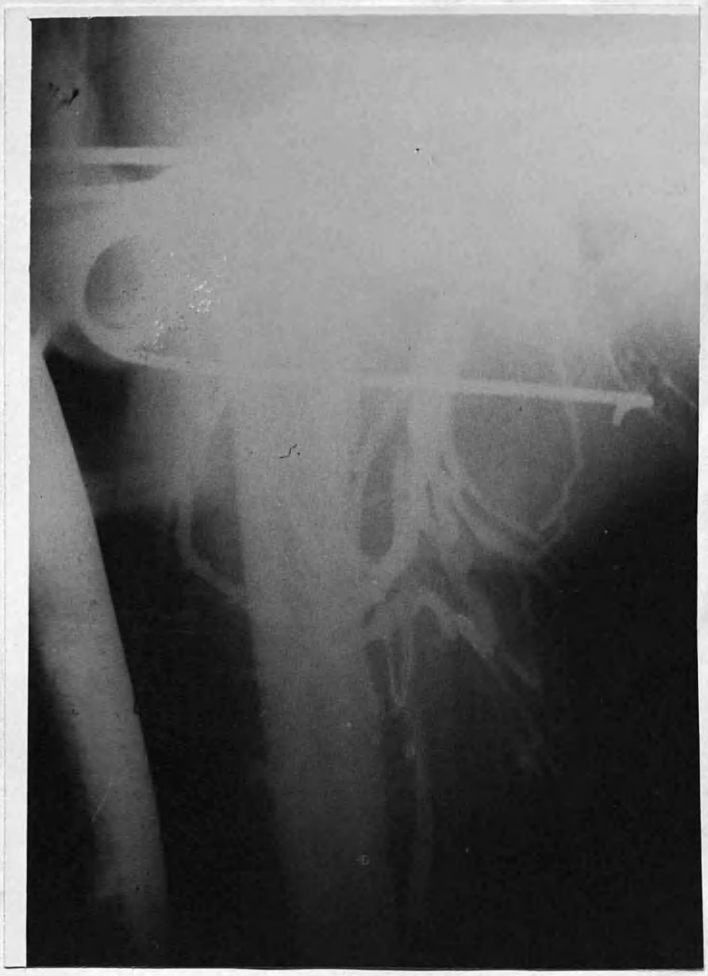


Fig. 9.

Retrograde venogram in patient who had puerperal white leg 12 years previously. Extensive filling of the main deep trunk and clearly demarcated valve formations are indistinguishable from normal.

The abnormalities noted were (a) the replacement of the main femoral trunk in part by a leash of vessels of varying/

varying calibre (Fig. 10) or (b) increased tortuosity of the deep trunk (Fig. 11).

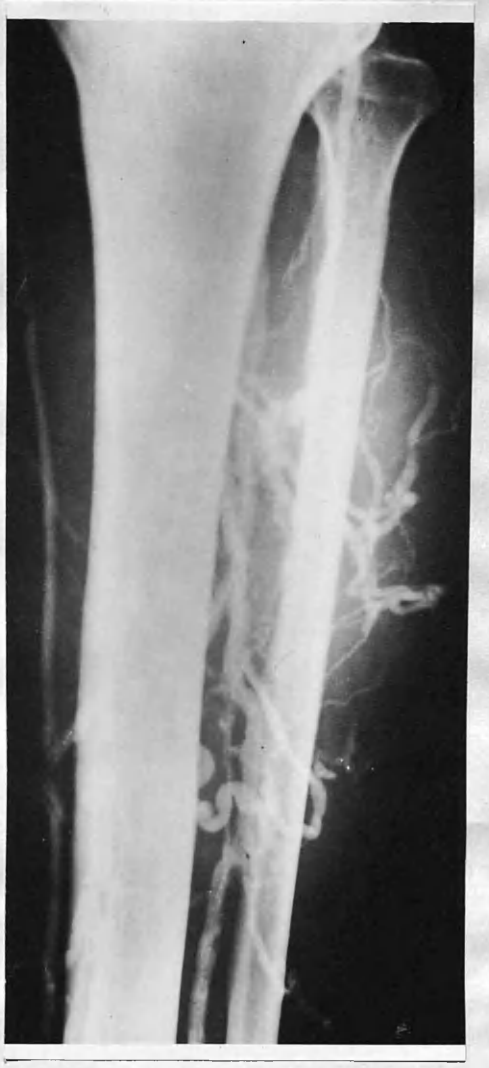


Fig. 10a

Fig. 10b

Ascending venogram in a male patient who had deep Thrombosis in the course of typhoid fever 26 years previously - to show the deep veins which are represented by numerous tortuous leashes and no usual deep trunk can be seen.

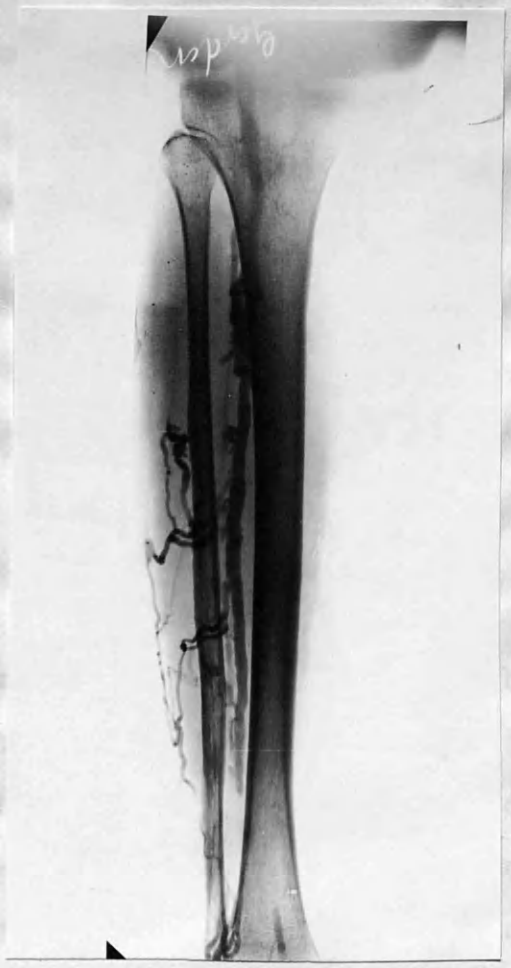


Fig. 11

Ascending venogram in a female patient who had puerperal white leg 15 years previously. Deep veins of leg dilated and varicose with multiple incompetent communicating veins.

While these abnormalities could be recognised they could not be related to any variations in appearance or severity of the cases and were of no special value in planning treatment. The technique is so specialised and time consuming that it is considered valuable only as a means of confirming impressions of pathology and adds nothing to the problem of treatment. Venography was not, therefore, applied/

applied routinely in diagnosis.

Pressure Tests.

The pressure in the veins of a normal lower limb at rest corresponds to a column of blood from the level concerned to heart level, (Smirk, 1936, Pollack and Wood 1949, Beecher 1937, Walker and Longland 1950). Thus, the pressure in a vein of the foot in a man of average height standing at rest is about 90 mm. of mercury. During exercise the muscle cuff pump lifts the blood from the foot towards the heart and in normal subjects the pressure in a foot vein falls to about 20-30 mm. of mercury. In varicose veins, on exercise, varying abdominal pressure continues to force blood down the saphenous vein by reflux and the pressure fall on exercise may be small, nil, or there may be a fluctuating rise. If a tourniquet is applied below the knee (Walker and Longland 1950) to prevent such reflux the exercise fall is near normal, but very variable results.are obtained on account of the usual venous tourniquet difficulties. Theoretically, in deep vein insufficiency, if the superficial reflux could be eliminated without affecting the deep return, the height of the pressure reading on exercise above normal would be a guide to the degree of stasis in the deep circulation, i.e. of deep insufficiency. Unfortunately, in the cases where this test might be of most value the presence of high tension varices,/

varices, and especially the gross induration of the lower limb and reflux through incompetent communicating veins below the knee render the test invalid. The procedure, involving intubation of a superficial vein on the dorsum of the foot with a polythene tube filled with citrate solution and connected to a manometer, is clearly not a simple bedside or out-patient procedure; and being subject to so many fallacies was abandoned after a brief trial as it did not provide any information not already known.

It became clear that there was no test of deep vein insufficiency of real practical value, other than the clinical classification of cases of gravitational ulcer into three groups of ascending difficulty in management. A patch of induration followed by ulceration indicates a less severe deep vein insufficiency than a high gaiter induration followed by ulceration - as judged by response to treatment. The three groups are

(a) Cases with superficial varicose veins and no history of deep thrombosis (Fig. 12)

(b) Cases with superficial varicose veins and a history of deep thrombosis (Fig. 13)

(c) Cases with no varices and a history of deep thrombosis (Fig. 14).

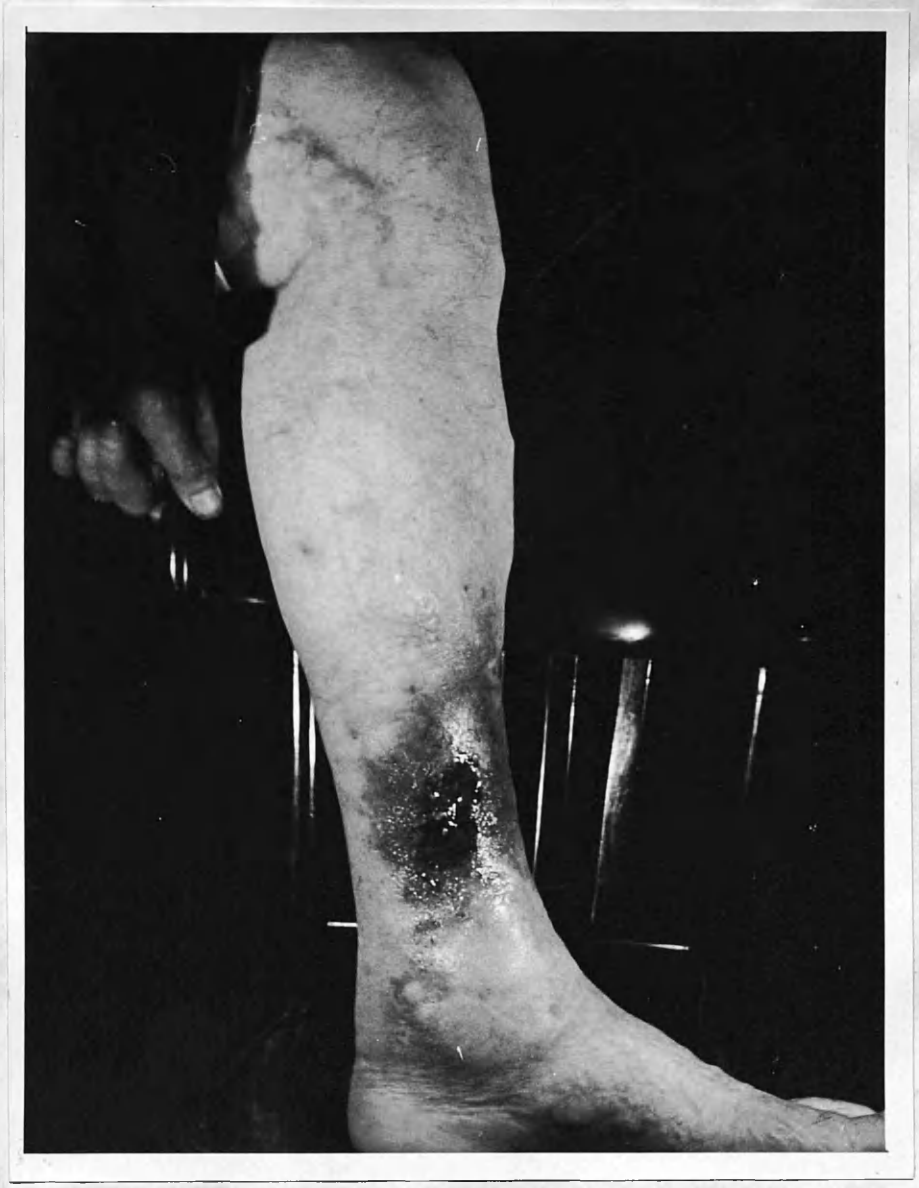
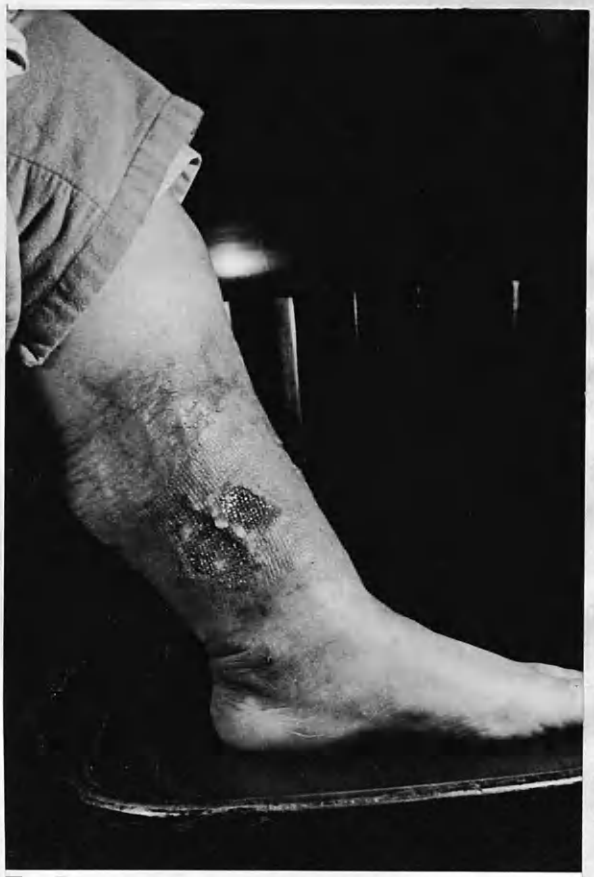


Fig. 12.

Gravitational ulcer in patient with obvious gross varicose veins and no history of deep thrombosis.



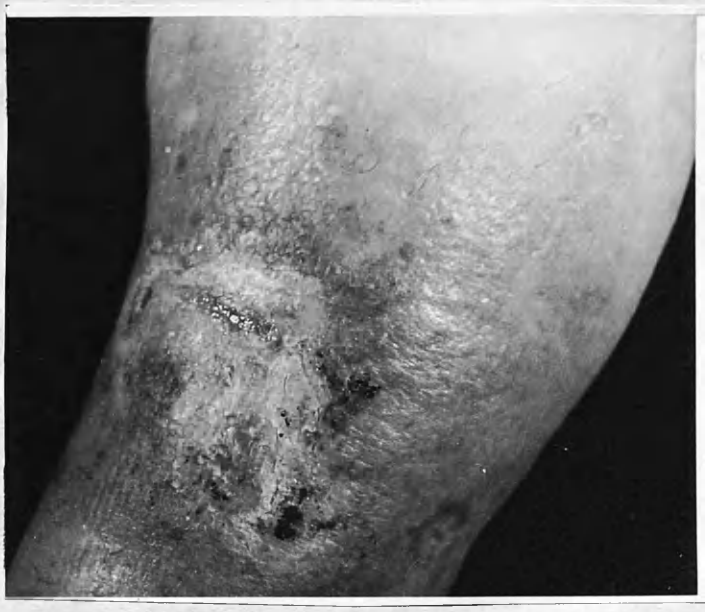


Fig. 13a Fig. 13b.

(a) Gravitational ulcer in patient with superficial varices and a history of previous deep thrombosis.

(b) Same leg soundly healed after 6 weeks viscopaste support.

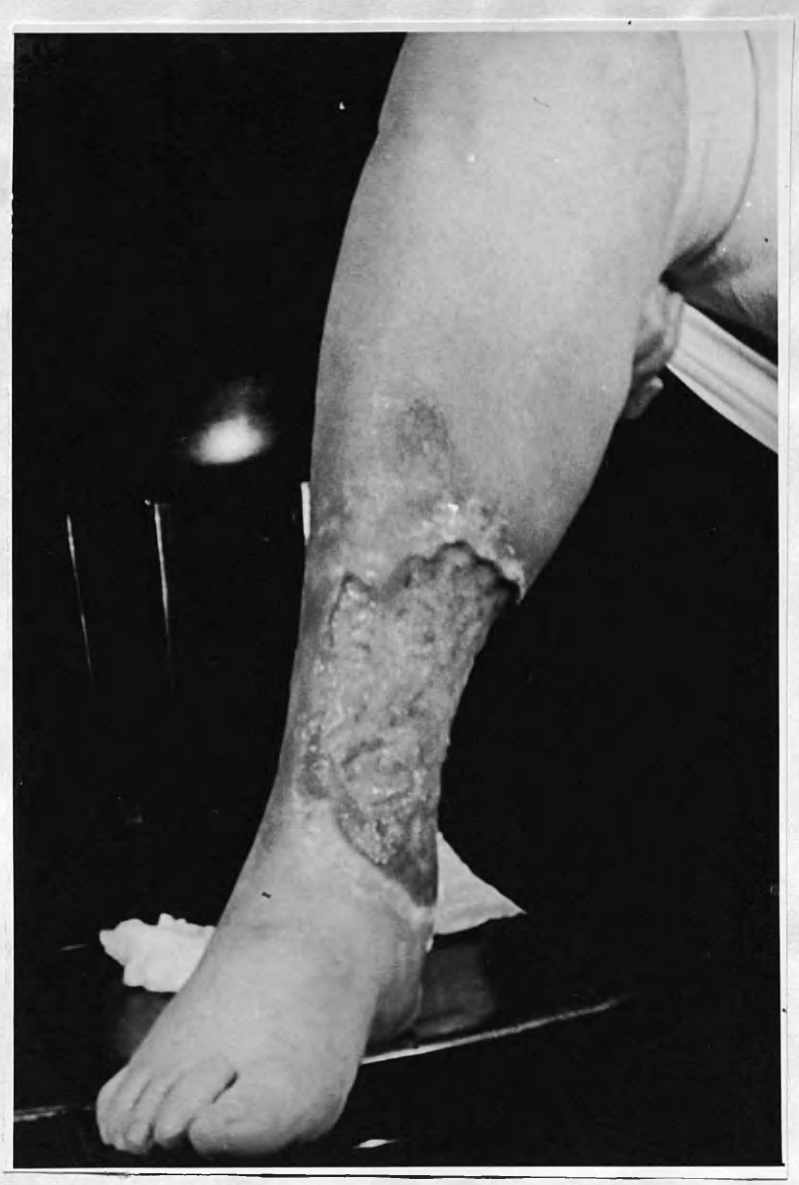


Fig. 14.

Gravitational ulcer in patient with no superficial varices and a history of deep vein thrombosis.

or another vlassification might be

(a) Deep vein incompetence with no impediment to deep return other than venous reflux.

(b) Deep vein incompetence with impediment to deep return in addition to reflux.

SCHEME OF TREATMENT.

On the basis of the foregoing conclusions the following standard method of management of gravitational ulcers was adopted.

1. The ulcer was encouraged to heal by applying a protective non-adhesive dressing of tulle gras, over this was applied a pad of orthopaedic felt to exert local pressure, disperse oedema and so help to restore the local vascularity, and the limb firmly encased in a semi-rigid boot of viscopaste from base of toes to just below knee. Such dressings were applied weekly until oedema was dispersed and then less frequently until the ulcer healed or became static. No antiseptics were used, nor did they seem necessary. Viscopaste was preferred to elastoplast because it is easier to apply and less prone to cause distressing skin reactions. If skin reaction did occur the limb was swathed in gauze soaked in 5 per cent ichthyol in calamine lotion and the viscopaste applied on top. The patient was kept ambulant to encourage deep venous return.

2. Superficial varicose veins were treated by appropriate ligation and retrograde injection of sclerosant solution. In almost all cases this was an out-patient procedure performed under local anaesthesia.

3. Demonstrably incompetent communicating veins were divided beneath the deep fascia which was then sutured.

4./

4. Deep venous return was encouraged by the provision of semi-rigid viscopaste support and keeping the patient ambulant, by encouraging reduction of obesity and by providing elastic stockings to be worn after healing had been achieved.

As will be recounted later, further measures were only applied if the standard treatment failed or was found inapplicable.

MATERIAL

The material studied consisted of all patients seen at the Varicose Vein Clinic in the Western Infirmary in the period 1947-50 inclusive: of these 284 had gravitational ulceration of one or both legs. The age and sex distribution is shown in Table III.

Table III.

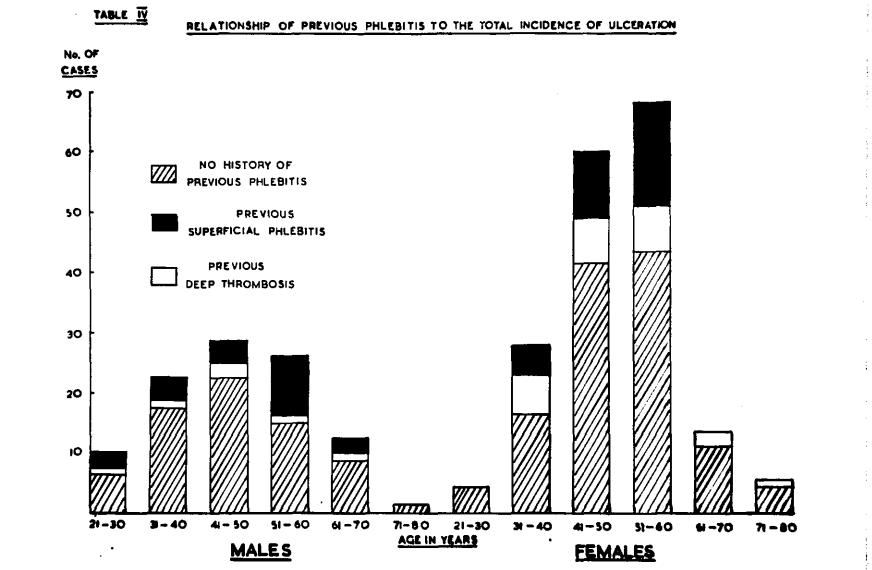
Age and Sex Incidence

Age in Years	21-30	31-40	41-50	51-60	61-70	71-80	Total
Male	9	22	28	26	12	1	98
Female	3	37	59	68	15	4	186

It is evident that no adult age group is exempt and that the proportion of females to males is 2 : 1. In the corresponding period the proportion of females to males attending the Varicose Vein Clinic was 3 : 1. Between the ages of 30 and 60 where the incidence in both sexes is highest the proportion of cases with a clear history of antecedent deep thrombosis is higher in females than in males, Table IV; presumably this is related to the peculiar incidence of phlegmasia alba dolens in females.

In Table IV is shown the relationship of previous phlebitis or deep thrombosis to the incidence of ulceration in each age group.

TABLE IV.



It will be seen that in only 37 cases was there evidence of previous deep thrombosis and in 48 a history of superficial phlebitis. Thus, in this series, 85 out of 284 cases (30 per cent) could, on the grounds of clinical evidence, be called post-phlebitic ulcers and this applies equally to both sexes and in all age groups.

Table V. Interval in years between Phlebitis and onset of Ulceration.

		- 1 yr.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	+ 15 yrs.
Male	D	3	1	1	2	-	-	1	-	-	1	-	-	-	-	-	-	1
	S	-	3	2	3	1	1	3	-	-	-	1	2	-	-	-	-	1
Female	D	2	4	4	-	1	-	1	1	-	1	2	2	2	1	-	-	6
	S	4	2	2	5	-	5	-	3	1	1	4	1	1	-	1	-	1

D = Deep Phlebitis
S = Superficial, Phlebitis

In Table V the interval between phlebitis and the onset of ulceration is shown. In 59 cases (69.4 per cent) the interval was less than 5 years and in 19 (22 per cent) the ulcer appeared before the second anniversary of the phlebitis. The longest interval was 30 years and in two cases the interval was 19 years.

Table VI. shows the chronological relationship of phlebitis to varicose veins.

TABLE VI.

	Age in Years	21-30	31-40	41-50	51-60	61-70	71-80	Total
Male	Varicose Veins before Superficial Phlebitis	1	3	3	6	1	-	14
	Varicose Veins before Deep Phlebitis	2	1	-	2	-	-	5
	Varicose Veins after Superficial Phlebitis	-	-	-	2	-	-	2
	Varicose Veins after Deep Phlebitis	-	-	1	-	1	-	2
	No Varicose Veins Superficial Phlebitis	-	-	-	1	-	-	1
	No Varicose Veins Deep Phlebitis	-	-	2	-	1	-	1
Female	Varicose Veins before Superficial Phlebitis	-	3	11	16	-	-	30
	Varicose Veins before Deep Phlebitis	-	8	5	7	1	1	22
	Varicose Veins after Superficial Phlebitis	-	1	-	-	-	-	1
	Varicose Veins after Deep Phlebitis	-	-	3	2	-	-	5

It is very clear that varices precede phlebitis, both superficial and deep, in the great majority of cases: indeed, in only 14 did the phlebitis precede the varices, according to the patient's history. It seems likely that in some of these 14 cases varices may have been present, but unnoted because they were not associated with symptoms before the phlebitis directed the patient's attention to his or her leg veins. This is in accord with the findings in the group of white leg cases investigated.

Table VII summarises the treatment applied to the 284 cases: in some cases multiple methods were employed.

Table VII Treatment Applied.

	Number of Cases		
	Male	Female	Total
High Ligation and Retrograde Injection	35	62	97
High Ligation and Retrograde Injection plus Communicating Vein Ligation	49	102	151
Communicating Vein Ligation only	4	4	8
Support only	9	14	23
Skin grafting	-	4	4
Deep Vein Ligation	1	4	5

RESULTS.

(a) General.

The results, as determined by a follow-up study of all cases, are shown in Table VIII.

Table VIII. Results.

	No. Treated	Failures	Recurrence	Successful
Male	98	1	5	92 (94%)
Female	186	4	9	173 (93%)

The shortest period of follow-up is 12 months, the longest 54 months.

Believing the underlying aetiological factor to be a disorder of haemodynamics which can only be, at best, partially corrected or compensated, it seems more profitable to consider the causes of success and failure than to attempt comparison with other reported series.

Table IX. Comparative Results.

Author.	No. of Cases	% Remaining Healed	Length of Follow-up
Buxton & Coller (1945)	26	87	3 - 15 months
Linton & Hardy (1948)	84	81	1 - 16 "
Glasser (1949)	40	72.5	6 - 48 "
* Bauer (1950)	196	84	6 - 36 "
Present Series	284	93.5	12 - 54 "

* "of the 16% of cases where recurrence had occurred, in almost two thirds this had seemed to be due to persistent incompetent veins in the superficial system"....This is Bauer's comment on his series treated by popliteal vein ligation.

But, in passing, it is fair to comment that the success rate is gratifyingly high and that possible recurrence can be anticipated and appropriate measures instituted. No case has come to amputation and only twelve have required hospitalisation at any stage in treatment. Cases are considered successful when the ulcer has remained healed for at least a year, the scar is soft and pliable and whether the patient is continuing to wear elastic support or not.

(b) Immediate Ligation.

It is well known that gravitational ulcers, in the nature of things, tend to heal with rest in bed or support of the limb and tend to recur if these measures are withdrawn. In order to try and evaluate the contribution of vein ligation a series of cases was treated by "immediate ligation" and no local support provided. 12 cases were so treated, all were successful, the average healing time being 6 weeks.

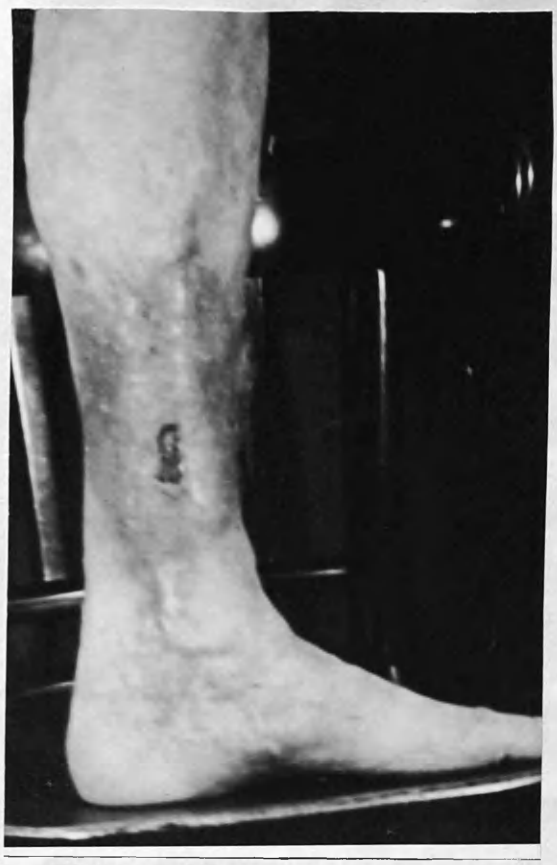


Fig. 15a

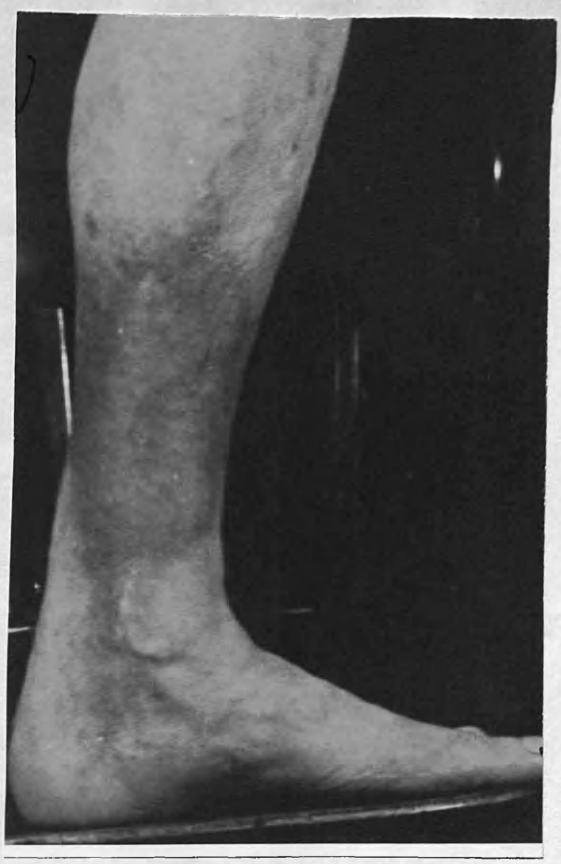


Fig. 15b

(a) Gravitational ulcer treated by immediate saphenous ligation.

(b) Same leg soundly healed 7 weeks after ligation.

In none of these cases was there any history of phlebitis. More cases were not so treated because of administrative difficulties and because out-patient operation under local anaesthesia was not considered wise where ulcers were dirty and there was inguinal lymphadenitis as a result.

(c) Cases with negative Trendelenburg test.

Further corroboration of the value of eliminating the effects of venous reflux is found in the study of six cases treated by division of incompetent communicating veins only - in these cases the Trendelenburg test was negative.

Case 1. Male aged 69. Varicose veins 30 years. Ulcer 10 years. Phlebitis 8 years ago.

12.4.49 Large incompetent perforating vein in calf excised.

2.5.49. Healed.

15.8.51. Ulcer remains healed - despite fairly severe intermittent claudication.

Case 2. Male aged 31. Bilateral high saphenous ligation and injection 5 years previously. Ulcer right ankle five weeks duration.

29.6.48. Large perforating vein excised. Ulcer healed in 4 weeks.

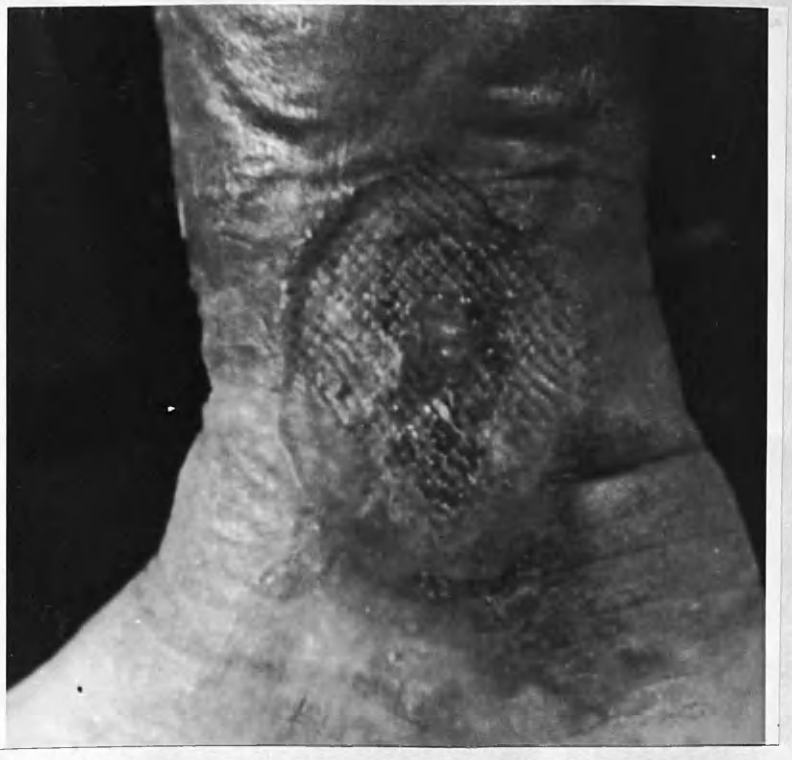
18.6.51. Remains soundly healed.

Case 3. Female aged 69. Varicose veins for 40 years. Ulcer right ankle for 4 months. No phlebitis. Fig. 16a

8.8.50. Excision of perforating vein.

20.8.50. Healed. Fig. 16b.

5.8. 51 Remains soundly healed.



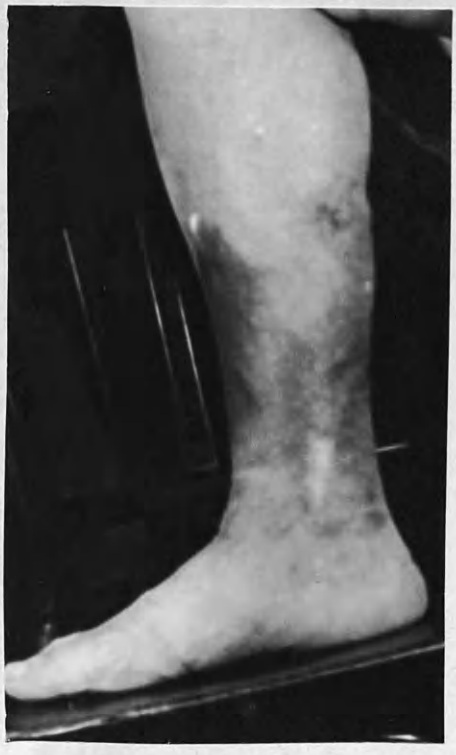


Fig. 16a. Fig. 16b.

(a) Gravitational ulcer: Trendelenburg negative: treated by excision of incompetent communicating vein.

(b) Healed: scar of excision on postero-medial aspect of calf.

Case 4. Female aged 56. Varicose veins for many years.

Ulcer 3 years duration. No,phlebitis.

2.5.50. Perforating vein excised.

14.6.50. Healed.

22.8.51. Remains soundly healed.

Case 5./

Case 5. Female aged 52. White leg, left, three times: 4 months in bed on each occasion. 28 yrs., 24 yrs., and 20 yrs. ago. Persistent oedema and recurrent ulceration of 24 years duration. No varices of internal saphenous vein: gross reflux into external saphenous vein.

30.4.48	External saphenous vein ligation.
11.5.48	Ulcer healed.
23.5.51.	Remains soundly healed, scar soft, white and mobile.

Case 6. Female aged 58. Varicose veins for 28 years since first pregnancy: no phlebitis; ulcer right ankle, two weeks duration.

5.4.49.	Perforating vein excised.
26.4.49.	Healed.
20.5.51.	Remains healed.

In all these cases removal of the local effects of venous reflux has enabled healing to take place.

(d) Recurrences.

In the period covered by the follow-up there have, so far, been fourteen cases in which recurrence of the ulcer has occurred after healing had been achieved. There follows a resume of the case history of these cases.

Case 7./

Case 7. Male aged 49. Varicose veins for 30 years. Recurring ulcer 10 years duration. Superficial phlebitis 3 years ago. Gross varices with incompetent perforating vein in upper calf.

27.6.50. High ligation and excision of perforating vein. Ulcer healed for ten months.

2.6.51. Ulcer recurred. Incompetent perforating vein at level of adductor tubercle demonstrated. Awaiting suitable ligation.

Case 8. Male aged 51. Varicose veins for 20 years. No phlebitis. Ulcer 2 years duration. Gross varices with incompetent communicating vein in lower third of thigh.

2.12.47. Ligation of incompetent veins. Healed in three weeks; remained healed for 29 months. Now has recurrence of ulcer and demonstrably incompetent communicating vein in lower third of leg.

Case 9. Male aged 54. Varicose veins for 7 years. Phlebitis 3 years ago. Ulcer 3 years' duration. High ligation 22.1.49. Ulcer healed 14.2.49. 3.5.49. Ulcer recurred. This patient has evidence of gross deep vein insufficiency - oedema and multiple incompetent communicating veins below the knee.

Case 10. Male aged 59. Varicose veins for 30 years. Ulcer right lower leg 7 years duration. Grossly incompetent varices and an incompetent perforating vein in the upper third of the leg. Following vein ligation the ulcer healed rapidly but recurred 14 months later when another incompetent perforating/

perforating vein was demonstrable: this latter was excised 15.8.50 and the limb has remained soundly healed since.

Case 11. Male aged 55. Developed deep thrombosis of left leg while in bed with pneumonia 1924: three months recumbency. Within 1 year developed a gravitational ulcer which has never completely healed. August, 1949, superficial femoral vein ligation. Ulcer healed and remained so for one year, despite persisting oedema. September 1950, ulcer recurred and has healed again with support - chronic alcoholic with poor arterial circulation.

Case 12. Female aged 49. Varicose veins for many years. No phlebitis. Ulcer for 3 years. 27.12.48. High ligation. Satisfactory till 5.1.51 when ulcer recurred and two inches above it there was a demonstrably incompetent communicating vein. 3.4.51. communicating vein excised. 13.6.51. Soundly healed and remains so.

Case 13. Female aged 46. Varicose veins for 10 years Ulcer 5 years. Phlebitis two months before ulcer appeared. 17.8.48 High Ligation. 23.11.49 Communicating vein in thigh demonstrable and ulcer recurred. 29.11.49 Communicating vein divided: healed in five weeks. 19.2.51 remains healed.

Case 14. Female aged 43. Varicose veins 23 years duration. 1942 White leg, left: 1946 ulcer left leg. 12.9.50 High Ligation, ulcer remained static. 21.11.50 Incompetent communicating vein excised. 5.12.50 Healed.

Case 15./

Case 15. Female aged 38. Varicose veins for 2 years. No phlebitis. Ulcer six months. 9.7.48 High ligation and communicating vein excised. 7.9.48 Healed. 30.4.51 Ulcer recurred. 8.5.51 Incompetent communicating vein in lower third of leg excised. 10.9.51 Healed.

Case 16. Female aged 39. Varicose veins for many years. Recurrent phlebitis for 3 years. Ulcer 3 weeks duration. 23.8.49 High ligation, ulcer healed. 25.1.51 Ulcer recurred. 23.4.51 Incompetent communicating vein excised. 18.6.51 Healed.

Case 17. Female aged 50. White leg 20 years. No varices. Ulcer 14 years. 13.10.47 High Ligation (elsewhere) ulcer healed. 9.8.48 Ulcer recurred: no varices. Heals with elastic support but recurs when this is discontinued.

Case 18. Female aged 57. Varicose veins for many years. Recurring ulcer 7 years. 10.2.48 High ligation: healed. 18.10.50 Ulcer recurred, incompetent communicating veins demonstrable in thigh and leg. 8.1.51 Communicating veins excised. 23.4.51 Soundly healed.

Case 19. Female aged 35. Deep thrombosis left, leg 17 years ago during bed rest for "Splenomegaly". Congenital syphilitic. W.R. persistently positive despite prolonged treatment. Bursting pain, oedema and ulceration lower leg continuously for 17 years. June, 1950, Popliteal vein ligation. Pain relieved: oedema unaltered: ulcer healed but broke down in three weeks: November, 1950 ulcer excised/

excised and grafted - healed for 8 weeks; August, 1951 further grafting; December 1951 original ulcer healed; fresh ulcer on inner aspect following slight trauma.

Case 20. Female aged 40. Obese, Para 8. Bilateral white leg 11 years ago. Recurrent ulceration right ankle since. December 1948, High ligation: no improvement. Femoral vein explored at this time appeared normal and was not disturbed. June, 1949, Ulcer grafted. Remained healed 1 year. Recurred following prolonged standing.

It can be seen from the histories of these cases that evidence of continuing deep vein insufficiency has always been present when the ulcer recurred. Further, in these cases where, to date, suitable further surgical treatment has been applied healing has been obtained. These cases illustrate the necessity of continuing after care, especially elastic support, and demonstrate that, despite such support, further blow-outs can occur. This emphasises that the underlying defect is in the deep veins. In 11 of these cases there was a history of proven or possible phlebitis (2) or deep vein thrombosis (9). But in the remaining 4 cases there was nothing to indicate such a possible preceding pathology of the veins. It is noteworthy that further treatment has produced healing with a soft pliable mobile scar in all these 4 cases, whereas in several of the other 11 healing remains unstable or has not been achieved.

(e) Failures.

Five cases treated have failed to heal. A resume of their histories and clinical features follows.

Case 21. Male aged 64. Varicose veins for 40 years: no phlebitis: extensive calcification of femoral arteries: recurrent gravitational ulcer (L) 14 years. 31.1.50 High Ligation and injection. Ulcer healed in 6 weeks. 20.3.50 Ulcer recurred. Incompetent cammunicating vein demonstrated in lower third of leg. 22.8.50 Communicating vein excised. Despite prolonged support and six weeks bed rest the ulcer failed to heal. 20.8.51 Oscillometry and arteriography showed popliteal arterial thrombosis. Ulcer healed after 8 weeks' bed rest but broke down within a week of becoming ambulant. 10.10.51 Developed patch of gangrene on pad of the great toe. In this case gross arterial insufficiency appears to be the cause of failure to heal.

Case 22. Female aged 57. 11.1.50 Varicose veins for 20 years. No phlebitis. Ulcer R. ankle 2 years' duration. Leg markedly indurated. 18.9.50 Induration resolving with elastic support. Developed erysipelas of the leg and despite continued support the ulcer persists. The lymphatics of the skin and subcutaneous tissues of this leg have been extensively damaged by the erysipelas and this may be the reason for failure. Ligation has not been attempted because of the latent infection.

Case 23./

Case 23. Female aged 75. 11.1.50 Varicose veins for 40 years. Gravitational ulcer 1 year's duration. Arteriosclerotic and frail. In view of the patient's age elastic support was prescribed: ulcer healed in five months but following an injury on 30.5.51 recurred and has persisted since but is again improving with elastic support.

Case 24. Female aged 44. 1928 Right White leg - puerperal, no marked varices at that time. 21.2.49 Ulcer for three years, moderate varices. 24.5.49 Right high ligation. Ulcer never fully healed: 23.8.49 Two incompetent communicating veins excised. Despite continuing support failed to heal. 20.6.51 Ulcer excised, split skin graft. Healing remains incomplete. W.R. negative. No gross arterial deficiency.

Case 25. Female aged 70. 12.4.50 Varicose veins for 20 years. Recurring gravitational ulcer of right ankle 4 years' duration. No history of phlebitis. Moderate oedema to mid leg. In view of the patient's age and poor general condition elastic support only was prescribed. Arterio-sclerotic. The ulcer remains static with elastic support but patient is not sufficiently incapacitated to justify considering amputation.

In three of these cases age, arterio-sclerosis and defective arterial circulation appear to me to be sufficient explanation of failure to heal. In one of the other cases chronic lymphoedema and skin infection seems to explain the chronicity. The fifth case cannot be "explained" on either/

either the grounds of arterial insufficiency or lymphatic obstruction and is a true failure.

(f) Cases treated by support only.

The cases treated by support only were those in which the patient refused operation, or where operation was contra-indicated because of the patient's general condition, or where there was associated gross arterial disease, or where no varices were apparent. Most were elderly and obese, though age and obesity were not, of themselves, regarded as contra-indications to surgical treatment.

PART IV.

DISCUSSION.

Homans (1916) described post-phlebitic ulceration as "the sort of ulceration which is associated with post-phlebitic varices of the small vessel type." Later (1938) he refers to post-phlebitic ulceration as ulceration occurring in a limb which has previously been the seat of deep thrombo-phlebitis. He recognised two types of superficial varices in association, (a) incompetent soft-walled varices, which he believed to be secondary to deep vein obstruction, or (b) straight thickened saphenous veins which have previously been the seat of phlebitis. He found the most notable pathological feature to be thickening and fibrosis of the tissues in the gaiter area and superficial to the deep fascia: the site becomes ischaemic from strangling of the vessels and this perpetuates the process. Homans advocated rest and elevation for the early case, rest and later, elastic support for the moderate case, but he regarded cases over fifty years of age in whom the ulcer had been present for many years as incurable except by amputation of the limb. He did not advocate ligation of communicating veins in the region of the ulcer in view of the danger of infection and difficulty of dissection.

Bauer (1940) believes that after deep thrombosis the deep veins remain obliterated or blocked in most cases and that the superficial system represents the usual collateral/

collateral return. He believes that varices of the superficial saphenous system are commonly a sequel of deep thrombosis. Despite these views Bauer (1948) advocates ligation and injection or stripping of incompetent superficial varices if such are demonstrable in ulcerated legs which have previously been the seat of deep thrombosis. He describes a stasis syndrome as a sequel to deep thrombosis the features being, induration, oedema and pigmentation or ulceration in the gaiter zone associated with bursting pain in the limb. As a result of venographic studies he describes two aetiological factors, (a) previous thrombosis leading to rigidity and loss of valves and variable narrowing of the vein lumen, and (b) phlebosclerosis, an idiopathic valve sclerosis leading to incompetence. He advocates ligating the large avalvular venous trunk at its lower end, i.e. in the popliteal fossa, to prevent venous reflux. He claimed to show by venography that, after popliteal vein division, the blood from the calf muscles was returned to the thigh muscle veins by numerous fine calibre channels and that no back flow occurred. But he did not trace the return into the main deep trunk above the level of operative division, where reflux must again operate. It would seem that multiple section of the deep incompetent trunk would be more likely to produce a lasting block to reflux. Such a procedure has been advocated by Leriche (1938).

Bauer's/

Bauer's claim (1946) that recanalisation hardly ever occurs after deep thrombosis and that "for the rest of life drainage is from deep to internal saphenous vein and upwards" is based on venographic studies. But his assumption that the deep veins were blocked because they did not show by his technique is invalid because he did not compress the superficial veins enough to force the radio-opaque medium into the deep veins (Dow, 1950). For the same reason he obtained pictures of normal variants of the superficial saphenous systems (Barrow, 1949) and not as he states, of collateral systems. He further claimed to be able to demonstrate deep vein incompetence by retrograde venography (1950) and Boyd (1948) and Lockhart Mummery (1951) have made similar claims. Bauer considered that in performing retrograde venography "it is absolutely necessary to make sure that the valves in the superficial femoral vein, if any, are closed at the moment when the dye runs into the vein." This, he stated, can best be done, and the most uniform results obtained, if the foot end of the operating table is suddenly lowered to 45 degrees or more and the injection then made. It seems unreasonable to assume that a sudden change of position should cause the blood flow in the femoral vein to cease. Indeed, if the femoral vein is opened at operation and the proximal end occluded profuse haemorrhage leaves the witness in no doubt of the continuing upward flow of blood from the limb. Further, the retrograde venogram is not a picture of a flowing stream but of a dense

radio/

radio-opaque fluid percolating downwards through an upward flowing stream of blood. Dow (1950) has shown clearly that the appearances described by Bauer as indicative of deep vein valve incompetence may be obtained in normal limbs when Bauer's technique is used.

Edwards and Edwards (1937) studied experimentally produced phlebitis in dogs and found that recanalisation proceeded pari passu with thrombosis and that the end result was the production of a valveless lumen which might be quite large or only microscopically demonstrable. If the lumen is inadequate the blood must flow by normal collaterals and if these are few they will dilate. Dilation to more than twice the normal diameter renders the valves incompetent because the length of each cusp in contact is between 0.2 and 0.5 of the diameter of the vein and the cusps are set in opposing pairs.

Homans (1928) observed that many patients with pigmentation, oedema, induration and ulceration of the gaiter zone had no varices and that such cases were a source of much pain and disability in the working classes. He described three types of phlebitis (a) deep phlebitis, deep to the investing fascia (b) superficial thrombo-phlebitis, which he thought was often followed by ulceration, and (c) thrombosis in varicose veins which he noted to be very common and to cause little disturbance as it rarely extends beyond the sapheno-femoral junction, usually recanalises and rarely /

rarely gives rise to embolism. He observed that, in phlegmasia alba dolens, there is usually no evidence of venous congestion in the limb because the collateral circulation via the intramuscular veins is adequate to obviate congestion and he believed the oedema to be due to associated lymphatic obstruction. Homans correlated this with his finding that if he tied a normal femoral vein at the groin the resulting blueness of the limb disappeared within forty-eight hours after which neither venous congestion nor swelling was visible. I have had the opportunity to observe this same train of events in a case of varicose veins in which the femoral vein was ligatured in error.

Reicharts (1936) showed that in animals most of the superficial lymphatics of the foot and leg as well as some of the deep lymphatics which drain the muscles of the leg terminate in the popliteal lymph gland and from thence large trunks run along the femoral vessels to the aorta. The deep lymphatics from the thigh muscles also drain into these main peri-vascular channels. Homans believed the oedema of deep phlebitis to be due to occlusion of these deep lymph channels by peri-phlebitis and that only in the very earliest stage could it be relieved by elevation and elastic bandaging. He advocated division of the internal saphenous nerve as a form of local sympathectomy to relieve pain, increase vascularity and secure a dry skin. He noted at operation that there was usually considerable inflammatory exudate/

exudate around the whole neuro-vascular bundle. Cruvelhier (1890) never found inflammatory changes in the intima, only in the perivenous tissues.

Leriche (1938) thought that if deep thrombo-phlebitis was treated by extraction of clot or vein excision annoying sequelae would be avoided. In cases of phlegmasia alba dolens he explored the common femoral vein and iliac veins and found them flat and empty or almost so - he concluded that the thrombosis was elsewhere. He resected one such flat vein, the patient was immediately improved and remained well after thirteen years. Leriche suggested that the initiation of deep thrombosis is silent and that extension produces three effects (a) venospasm, (b) growth of clot and (c) arteriospasm. Venospasm he thought to be constant, causing pain and encouraging extension of clot, arteriospasm to be frequent and perhaps constant. Because of his belief in these vasospastic effects he practised lumbar sympathetic block and claimed dramatic improvement. He found extension of the clot to be variable and noted that in cases where it was marked oedema was very persistent and the patients became invalids in later life. While believing spasm to be an essential element of the pathological change Leriche considered the extent of thrombus formation to be the commanding factor which determined the severity and extent of the late effects. Attention must therefore be directed mainly to the clot. He observed that in some cases there/

there were no late evidences of previous phlegmasia alba dolens and he suggested that in such cases spasm predominated. Often, however, late effects appeared - permanent oedema, cutaneous atrophy in the gaiter zone and ulcers - which he regarded as trophic and incurable.

Jung and Leriche (1928) showed experimentally that complete obliteration of all main veins at the root of a limb produced no visible change in the circulation and concluded that oedema resulted from something more than venous obstruction. Dos Santos (1929) showed by venography that in such experimental animals a collateral circulation quickly establishes itself. Leriche maintained that in phlegmasia alba dolens the main vein was not only blocked but periphlebitis set up reflex vaso-constriction in the available collateral vessels thus adding to the burden and producing oedema. Resection of a portion of the obliterated vein resulted in disappearance of the oedema and Leriche advocated this as a method of treatment and suggested that high lumbar sympathectomy be performed as an additional procedure to eliminate vasospasm. He did allow that many of the sufferers from gravitational ulcer were elderly and obese women and unfit for such a major operative procedure, for these he recommended intravenous injection of hypertonic saline. Leriche considered post-phlebitic ulceration to be quite different from varicose ulceration and that both occurred in the lower inner third of the leg because the intrinsic/

intrinsic vascularity of this region was poor.

In the past decade Bauer has investigated the late effects of deep vein thrombosis by venography and considers oedema to be a constant sequel which never completely disappears. This oedema, considered by Homans to be lymphatic in origin and by Leriche to be largely due to vasospasm, Bauer (1942) believes to be a result of venous stasis. In a study of 145 cases of phlegmasia alba dolens 81 per cent showed induration in the gaiter zone and 61 per cent developed ulceration. Induration was present for an average of 2.7 years before ulceration ensued. He claimed to demonstrate that practically all his cases had persisting complete obliteration of the principal deep venous trunks, and that the venous return in these cases was for all time relegated entirely to what he calls compensatory superficial venous systems. Accordingly he stated that the surgical interruption of obviously varicose superficial veins in vases of old standing phlegmasia alba dolens should never be performed. (It is interresting to note that the case he quotes as developing gross oedema when such ligation was performed was injected with sclerosant below the knee: it was probably this error and not the vein interruption which produced fresh evidence of deep vein blockage).

Bauer studied 44 cases of gravitational ulcer, seen in a period of three years at the Mariestad Hospital and regarded their aetiology as being:

6 cases ..... tuberculosis arteriosclerosis.

5 cases ..... Varices with no known thrombosis.

33 cases ..... Definite history of deep thrombosis.

i.e. there were 38 cases of non-specific leg ulcer and 87 per cent of these he regarded as being due to the effects of previous deep thrombosis. He does not state how many of these cases had varicose veins before the incidence of deep thrombosis. He investigated another series of 42 cases of varicose veins. Only 5 of these had ulcers and in all 5 there was a patch of induration around the ulcer: in the remainder there were no skin changes. These ulcers were situated in the middle of a segment of superficial thrombo-phlebitis and had a softer edge than post-thrombotic ulcers, relapsing thrombo-phlebitis was the only complaint other than cosmetic, he states.

As a result of his venographic studies he considered recanalisation after deep vein thrombosis to be a rare phenomenon and the circulation to be carried entirely by compensatory collateral systems of which he recognises three standard patterns. His first order is the normal internal saphenous vein, his second is the normal external saphenous vein, and his third an unusual, but normal, alternative termination of the external saphenous vein.

With/

With Dr. J.D. Dow (1950) I studied 34 cases with a definite history of deep leg vein thrombosis. In every case we were able to demonstrate a patent deep venous system and, indeed, if we failed to do so we blamed not a previous deep thrombosis but our own technique. If the superficial veins are compressed by firm crepe bandaging from ankle to groin the deep veins can be shown by the simple procedure of injecting any available vein on the dorsum of the foot distal to a firm venous tourniquet. If this is not done the radio-opaque solution rapidly escapes into the superficial circulation through communicating veins and a true picture of the deep veins is not obtained. This investigation was carried out because, like Turner Warwick (1930) I have not yet seen a case of the post-phlebitic state in which the clinical findings suggested a permanent complete deep vein blockage.

In ascending phlebograms, while it can be demonstrated that a vein lumen is present, it is not possible to state whether the vein wall and valves are normal or not and, if so, to what extent. A further series of cases was investigated by retrograde venography. No constant evidence of known previous deep thrombosis could be demonstrated - and this is what one would expect, for the extent of vein wall and valve involvement must vary considerably from case to case. In some cases valve formation appeared to be normal and the femoral vein could

be/

be visualised in its whole length. Such an appearance was regarded as normal, for the heavy dye percolates downwards through the upward flowing steam of blood and a complete hold up at a valve is not to be expected. In this type of radiography as in intravenous pyelography, apparent anatomical variations should only be regarded as significant if constant. The time between injection and photograph may coincide with a hold up as the valve cusps are closed by the down flowing dye but later photographs will show that the dye has gone more distally. Bauer (1948) regards an apparent sharp hold up as evidence of normal valve competence and the presence of radio-opaque fluid at knee level after retrograde injection as evidence of valve incompetence. Lockhart Mummery (1951) holds similar views. We recognised evidence of deep vein damage of two types.

(a) those where the vein channel was replaced by a leash of vessels of varying calibre.....(Fig.17)

(b) those where the deep veins appeared varicose (Fig.11)

But there was no constant relationship of either of these appearances to the presence of gravitational ulcer.

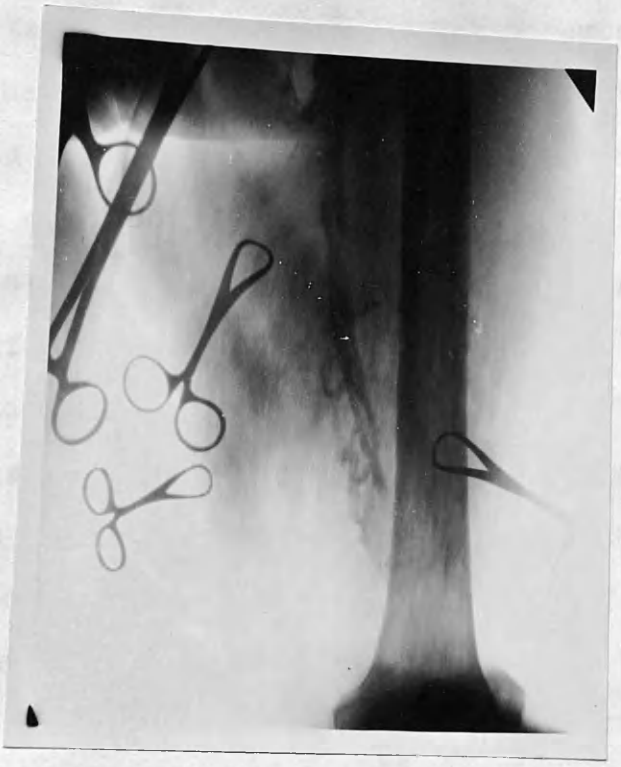


Fig. 17.

Retrograde venogram in patient with history of deep thrombosis 12 years previously: main trunk replaced by a tortuous leash of vessels with no clearly demarcated valves.

When the deep veins are exposed in cases of previous deep thrombosis the whole vascular bundle is seen to be matted together as described by Leriche (1938) and the vein wall thickened; but blood can be demonstrated to flow in such veins by digital stripping even though the lumen is a mere slit. The changes in the deep veins are increased rigidity, varying valve destruction and perivenous fibrosis and the lumen is reduced and may be tortuous/

tortuous or replaced by multiple small calibre channels. All these factors tend to produce venous insufficiency and stasis. In the acute phase of deep thrombosis when the lumen of the main trunk is blocked, a collateral venous circulation in the intra-muscular veins opens up. When the thrombosed segment recanalises it becomes incompetent to some degree allowing retrograde flow which will, to a varying extent, defeat the efforts of the competent collateral channels to conduct the blood centrally. The result is venous stasis inside the deep fascial envelope. Goetz (1949) has shown that Poiseuille's law is applicable to human blood vessels and that a reduction of the lumen of a vessel by half reduces its carying capacity to one sixteenth. The recanalised lumen, therefore, presents a barrier to effective venous return and adds to the stasis. For the same reasons sudden reflux is less likely to be effective in such veins and the strain will fall on the less rigid and poorly supported superficial veins. This, I believe, is the mechanism of production of superficial varicose veins as a sequel to deep thrombosis, and of the well recognised phenomenon of worsening of pre-existing superficial varices. During exercise in such limbs a build up of pressure inside the deep fascial envelope will occur and in the presence of incompetent superficial varices, be accompanied by a similar build up of pressure in the superficial veins. In cases where the iliac valve remains intact/

intact the development of superficial varices of the main internal saphenous system is not to be expected, although in such cases superficial varices may result from reflux through communicating veins whose valves give way under pressure.

Inside the fascial envelope which is semi-rigid the deep veins are enclosed in a muscle cuff which, above the level of the lower third of the leg, is entirely fleshy; below this level the cuff is musculotendinous and, as a result, less efficient as a pump. Thus the effects of stasis are most evident in the lower third of the leg and not in the foot which is the most dependent part. They are never seen in the upper third of the leg. The results of anoxaemia, impaired nutrition, oedema and induration are not evident under the deep fascia - as is clearly demonstrable at operation - but between the aponeurosis and the skin. In varicose ulcer the preceding patch of induration usually occurs at the site of a communicating vein which is demonstrably incompetent in the early stages but which disappears in the process of induration by becoming thrombosed. Later, following trivial trauma, the indurated zone breaks down to form an ulcer. The extent of this process varies from a patch to a gaiter and both may be seen in cases with no history of previous deep thrombosis or long illness but with evident varicose veins. This would seem to indicate that the extent of the induration is merely an index of the extent of deep vein insufficiency of whatever

aetiology/

aetiology and that the difference between varicose ulcer and post-phlebitic ulcer is one of degree. The more comprehensive descriptive term gravitational ulcer is therefore preferable.

What, then, is the significance of obvious varicose veins in association with gravitational ulcer? John Gay (1868) stated that "given an ulcer and a varicose vein, the ligature is the answer, at first sight, but follow-up shows that both ulcer and varices recur." He believed the chief benefit of treatment was bestowed by the rest in bed at the time of operation. When varicose veins are evident in a limb which has previously been the seat of deep thrombosis it is widely accepted that these varices represent a secondary distension of the collateral circulation and that any interference with these superficial veins will make matters worse. Many authorities (Foote, Anning, Rogers, Boyd) state categorically that the superficial varices must never be interrupted or obliterated in cases of previous deep thrombosis. From the clinical view point this seems an entirely wrong attitude. If the superficial veins are varicose and permit retrograde flow they must add to the total venous stasis in the affected limb, and prevention of reflux by vein section must benefit the extremity provided these superficial channels are not essential for the return of blood from the deep compartment of the limb. As has already been stated it can easily

be/

easily be demonstrated that there is a patent deep circulation and that, even in the most extensive deep thrombosis, multiple communicating vein incompetence is not demonstrable. This would be essential to allow the blood to escape from the deep compartment of the leg. In this series of cases the presence of demonstrably incompetent superficial varices has always been sought and these have been treated, as in the non-ulcerated limb, by appropriate ligation and sclerosant injection. No limb has been made worse and most markedly benefited. Indeed, the elimination of reflux into and stasis in the superficial veins alone, or aided by external support, may so diminish local oedema and anoxaemia that re-vascularisation and healing occurs (Fig. 18).

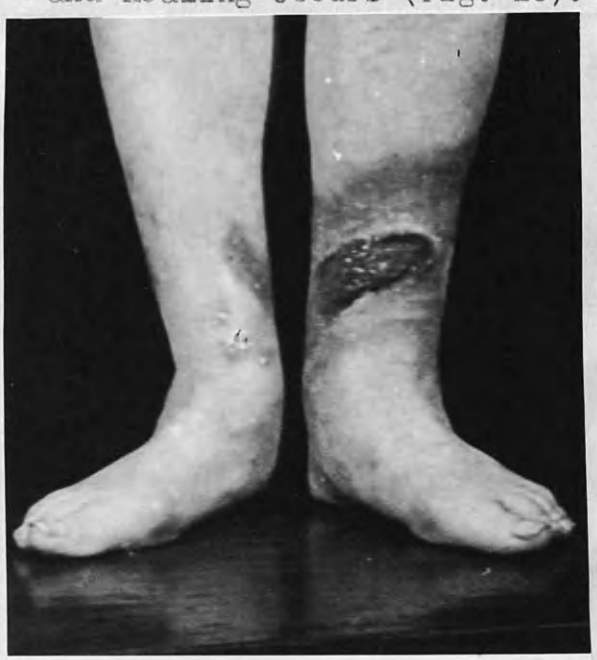


Fig. 18a

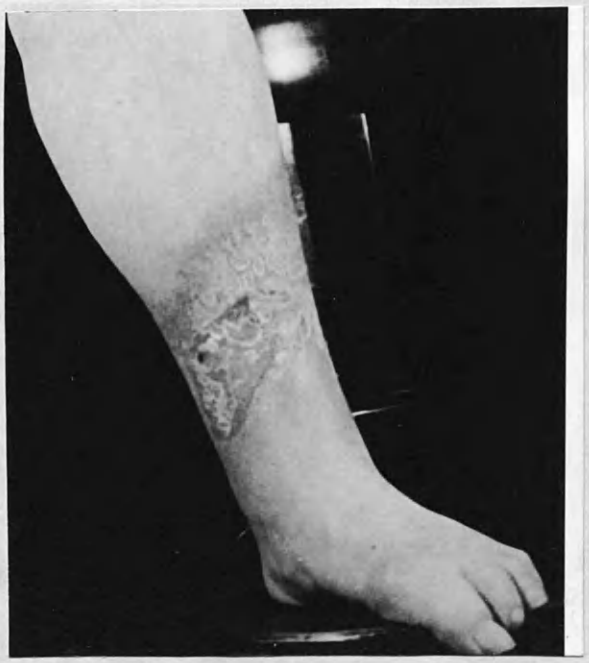


Fig. 18b.

Healing of gravitational ulcer and reduction of oedema following superficial vein ligation.

The deep vein defects remain. Only if elastic or semi-rigid support failed to control the ulceration was any further procedure practised. The first such procedure was to attempt to increase local vascularity by wide excision of the ulcer and a margin of the surrounding indurated zone down to and through the deep fascia into the deep compartment where there is little or no fibrosis, and applying split skin grafts to the exposed muscle or periosteum. This method was advocated by Homans (1916, 1938) and in this series was employed in five cases. All of these had previous deep vein thrombosis. Healing was secured in all cases but in three of the cases deep vein ligation was also performed (Fig. 19) before this healing became securely established.

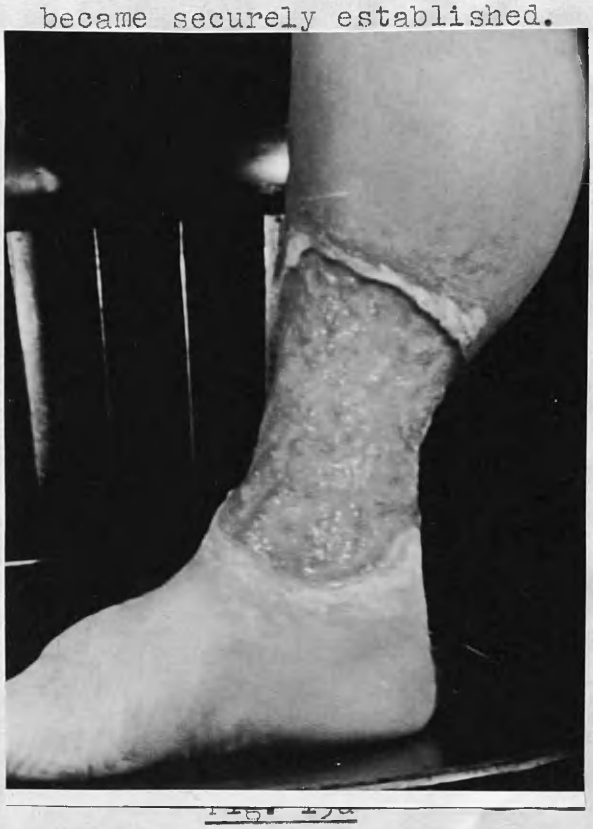


Fig. 19a

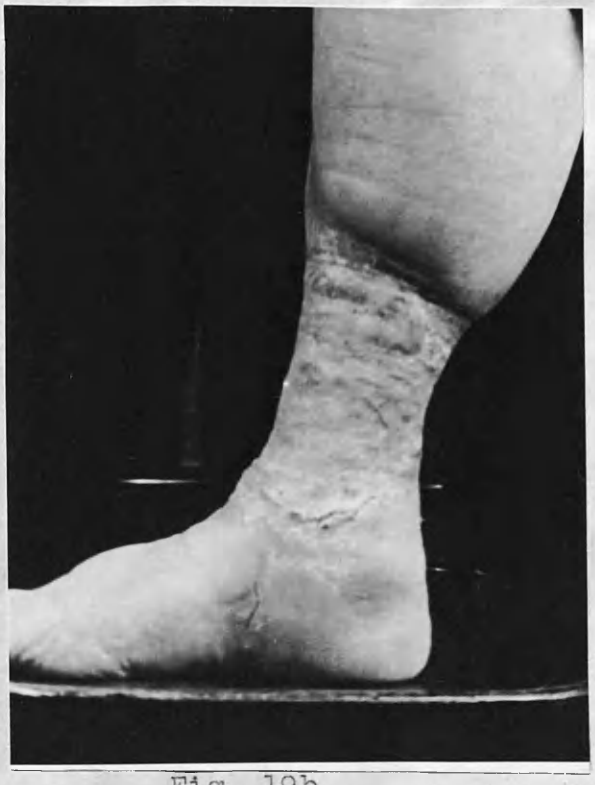


Fig. 19b

(a) Gravitational ulcer (Case No. 27) of 14 years' duration: no varices.
(b) Soundly healed after popliteal vein ligation and skin grafting.

Two further surgical procedures have been suggested, viz. lumbar sympathectomy (Leriche, 1938, Homans 1939, Mahorner 1944, Linton and Hardy 1947, Glasser 1949) and deep vein interruption (Parona 1894, de Takats 1929, Leriche 1926, Buxton and Coller 1945, Linton and Hardy 1948, Bauer 1948, and Glasser 1949).

Lumbar sympathectomy is a major operative procedure necessitating admission to hospital and a general anaesthetic. As no convincing evidence of its value in gravitational ulceration has been produced, it has not been employed in this series. It seems a very major procedure to obtain a warm dry limb. Sympathetic release will effect arteriolar relaxation and also venous relaxation and would therefore be expected to increase rather than decrease congestion and stasis.

Linton and Hardy (1948), combined high saphenous ligation and stripping with ligation of the superficial femoral vein in order to prevent reflux down the main vein. In their series 20 of 43 ulcers were still present the longest follow up being 16 months. Buxton and Coller, (1945), claimed greater success from ligation of the common femoral vein. Leriche, (1938), advocated multiple deep vein interruption but believed that any beneficial effect was due to sympathetic nerve interruption. Bauer, (1948), believed that high ligation of the superficial femoral vein would leave a deep vein column below the ligature into

which collaterals would pour blood, which would then flow distally in the incompetent trunk and advocated ligation of the popliteal vein.

While there is no doubt that recanalisation is the rule after deep thrombosis, the resulting lumen is narrowed, tortuous and valveless to a varying extent, the vein wall is thickened and rigid and matted to the artery and often to the accompanying nerves, all of which share in the inflammatory process. Thus nerve irritation, artery irritation, lymphatic involvement and sympathetic nerve involvement may all play some part in the production of signs and symptoms. It is, consequently, difficult to assess the results of the various procedures advocated for the relief of venous insufficiency. Single interruption at any level will only prevent reflux at that level and reflux via collaterals may still occur. The dissection of clearing the vein from the artery may result in some degree of sympathectomy. In the few cases in which I have performed deep vein ligation I have noted some resultant flushing of the foot and ankle suggesting sympathetic release. The most constant effect of deep vein ligation in my cases has been relief of bursting pain and this, too, is suggestive of a nerve section effect, presumably sympathetic. Oedema has never completely subsided after deep ligation and, in the immediate post-operative period, has usually been increased and has only regressed after prolonged elastic support/

support, when a collateral circulation has established itself. It seems likely that this new circulation may itself become incompetent from the continuing effects of venous reflux. In this series deep vein ligation, which has not commended itself as being thoroughly sound theoretically, has been performed only in a few cases which have resisted other methods. A study of these appears to show that the procedure may have some value but its application in treatment can not be wide until more is known of the long term results of deliberate interruption of diseased deep veins. I have treated five cases thus.

Case No. 26. Female aged 60. 1938. High Saphenous ligation for varicose veins of 14 years duration; following this developed bilateral deep vein thrombosis. 1939, developed gravitational ulcer of L. ankle. Healed but recurred despite continuing elastic support. 12.1.49 L. Superficial femoral vein ligation: oedema increased but gradually dispersed with continuing support and after 1 year ulcer soundly healed and has remained so. 14.2.50 R Superficial femoral vein ligation - oedema increased but after six months support was well controlled. This patient weighs over 16 stones.

Case No. 27. Female aged 64. 1930 fracture of Right ankle, six weeks in bed, ever since has had persistent brawny oedema of the gaiter zone. 7.8.50 (Fig. 19a) chronic ulcer of 14 years duration steadily progressive and associated with intense bursting pain of 16 years duration. Despite

two/

two periods of 1 year in bed five and seven years ago, the ulcer did not heal. 12.9.50 - no varices apparent: popliteal vein ligation and split skin graft. Immediate and lasting relief of bursting pain, graft took and leg remains healed with elastic bandage support (Fig. 19b.)

Case No. 29. Female aged 59. Para 10. 1926 White Leg (R) after birth of 10th child. Preceding varices became worse and 1928 developed ulcer of ankle which had recurred at varying intervals. 1947 Ligation and injection of varices, ulcer healed. 1950 ulcer recurred: no recurrence of varices. 8.1.51. Popliteal vein ligation and split skin graft to ulcer - popliteal vein double and both trunks extensively thrombosed. 28.2.51 Soundly healed and free of pain. Dec. 1951, remains soundly healed.

Case No. 30. Male aged 56. 1924 Pneumonia - developed L. deep venous thrombosis. 1932 developed ulcer in ankle region with bursting pain and oedema. Recurrent. Longest healing 16 months. No gross superficial varices. Jan. 1948 Internal Saphenous and Superficial Femoral vein ligation. Femoral vein thickened and tortuous. Pain relieved, oedema increased. Ulcer healed for 2 years but Jan. 1951 developed superficial ulceration in the gaiter zone which is controlled by elastic support.

Case No. 19. Female aged 35. Deem thrombosis left leg 17 years ago during bed rest for "splenomegaly". Congenital syphilitic. W.R. persistently positive despite prolonged treatment. Bursting pain, oedema and ulceration lower leg continuously/

continuously for 17 years. June, 1950, Popliteal vein ligation. Pain relieved: oedema unaltered: ulcer healed but broke down in three weeks; November 1950 ulcer excised and grafted - healed for 8 weeks; August, 1951 further grafting; December 1951 original ulcer healed; fresh ulcer on inner aspect leg following slight trauma.

It is difficult at this stage to evaluate the procedure of deep vein interruption. The one striking and constant benefit has been immediate and lasting relief from bursting pain where such was present. This suggests a nerve section effect. Oedema has, in all my cases, been immediately increased but tended to resolve or at least be reduced and controlled by continued elastic support. Two of the cases might be regarded as entirely successful Nos. 27 and 29, but in both the ulcer was excised and skin grafted as an additional procedure so that the contribution of deep vein interruption to the achievement or maintenance of healing can not be assessed. In the remaining three cases sound healing has not been achieved, despite repeated grafting in one case - (No. 19) which is further complicated by a persistently positive Wasserman reaction. All of these cases were post-phlebitic and all showed extensive deep vein damage. None were made worse by interruption of the deep veins but the benefit seemed to be relief of pain. The temporary increase in oedema responding to continued support suggested that, as a collateral circulation/

circulation opened up, back pressure effects were relieved.

If reflux were the only deep vein defect, deep vein ligation would seem to be a sound procedure although it would usually require to be done at several levels to defeat the effect of by-passing. Deep venous reflux as a primary defect may lead to the development of superficial varicose veins but many cases with extensive superficial varices never develop ulceration, and those that do always show evidence of some preceding thrombosis of at least a perforating vein. Deep vein thrombosis may be followed by gravitational ulceration in limbs where there are no superficial varices. Thus it would seem that the determining factor in the production of ulceration is something more than deep or superficial vein incompetence, viz. deep vein insufficiency the result of thrombosis. When there is superficial as well as deep vein insufficiency, the removal of the effects of the former proves beneficial to the limb and may so relieve stasis as to permit sound healing of gravitational ulceration and reduction in oedema and induration. Varicose veins are not a cause of ulceration. All gravitational ulcers are, I think, postphlebitic although there may be no history of characteristic deep vein thrombosis and the extent of the deep phlebitis may be quite localised. Figure 20 and Figure 21 show diagrammatically the various types of venous insufficiency encountered.

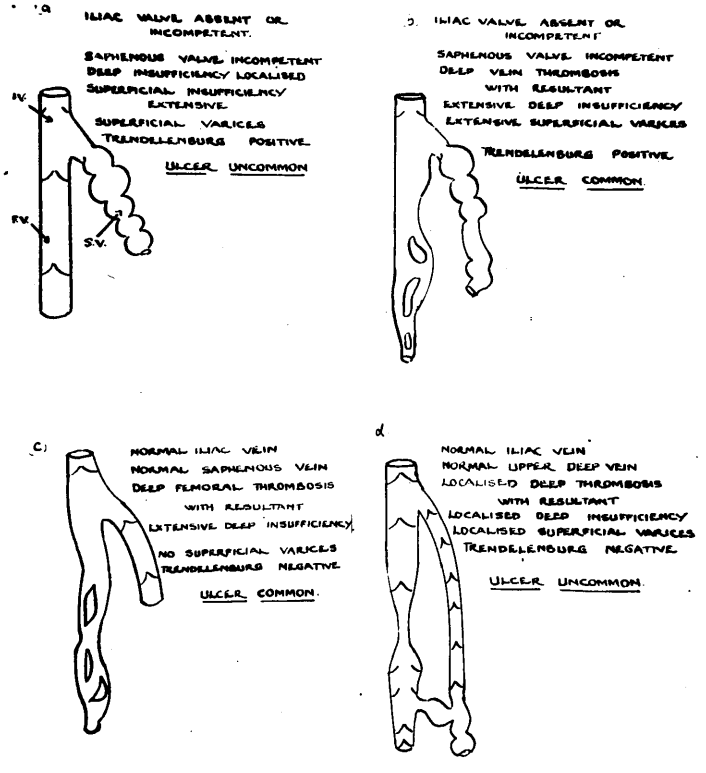


Figure 20.

Diagrammatic representation of leg veins in normal and pathological states.

I.V. - Iliac Vein
F.V. - Femoral vein
S.V. - Internal saphenous vein.

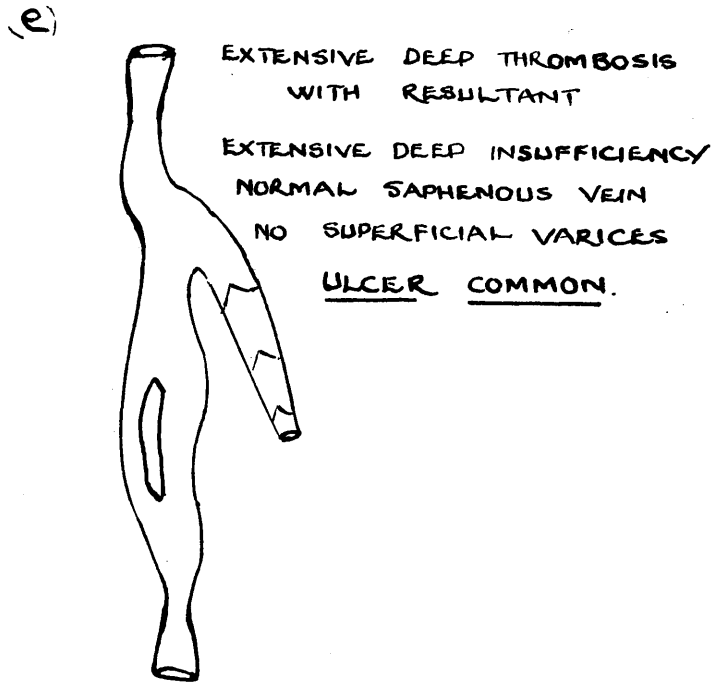


Fig. 21.

Diagrammatic representation of the effects of extensive deep vein thrombosis involving iliac and limb veins.

The nature and degree of deep venous insufficiency cannot be accurately assessed or located clinically or by ancillary methods. Reflux in superficial and communicating veins can be prevented by vein section, but to apply this procedure to deep veins not only produces a block to deep vein reflux but also to the deep venous return. The collateral circulation established to circumvent such a block may in turn be submitted to the strain of femoral or iliac valve incompetence and itself become incompetent. Fibrosis/

Fibrosis and loss of calibre, the result of thrombosis, will stimulate the opening up of a collateral circulation and it would seem that this can best be aided by supporting the muscle cuff and eliminating the effects of gravity. Bed rest is economically impossible for many of these patients and in any case tends to encourage stagnation. Ambulatory treatment seems the most reasonable prophylactic against deep vein stagnation just as continued elastic support seems to be the fundamental requirement of treatment.

If the iliac valve is absent(Eger and Casper,1938) or destroyed, reflux will be a permanent feature and thus treatment must be a continuing process and permanent cure can not be guaranteed. But, with careful assessment of the individual case and elimination of reflux as far as this is practicable together with measures to increase the local vascularity in the indurated zone, the great majority of cases with gravitational ulceration can be kept skin whole. Amputation should never be necessary.

General Summary.

1. The magnitude of the problem of gravitational ulcer is indicated.

2. A brief historical review of treatment methods is presented.

3. The normal venous circulation of the lower limb is described and the conditions which may lead to venous stasis reviewed.

4. A scheme of management based on attempts to restore the disordered haemodynamics is described and the technical considerations discussed.

5. A practical method of locating incompetent communicating veins is described.

6. Methods designed to assess the state of efficiency of the deep veins are described: no clinical or venographic test is accurate enough for practical use.

7. A follow up study of cases of puerperal white leg is presented.

8. A scheme of treatment is outlined and the results of its application to a series of 284 consecutive cases of gravitational ulcer presented.

9. A discussion of the views of others on various aspects of the problem is presented and the following conclusions noted:-

(1) Gravitational ulcer does not result from the presence of varicose veins alone, a phlebitic factor is a necessary precursor.

(2)/

(2) Gravitational ulcer may occur in the absence of superficial varices.

(3) Superficial varicose veins never represent a useful collateral circulation after deep thrombosis. Indeed, in the majority of cases the varices precede the deep thrombosis. The elimination of reflux into and stasis in such superficial varices is always beneficial to the total venous economy of the limb.

(4) Continued elastic support is essential to reduce the risk of recurrence from the continuing effects of deep vein insufficiency.

(5) Skin grafting directly on to muscle or periosteum is a useful procedure in resistant cases.

10. The procedure of deep vein ligation is discussed and five cases briefly presented.

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