THE SURGICAL MANAGEMENT OF ULCERATIVE (STASIS) DISEASE OF THE LOWER EXTREMITIES*

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There is a great deal of confusion about varicose veins and varicose ulceration of the lower extremities. The purpose of this paper is twofold: firstly to attempt to clarify the present-day concept of varicose ulceration of the lower extremities, and secondly to outline a surgical treatment of varicose ulceration especially for the ulcerative stasis disease of the lower limbs. This stasis disease is also referred to as gravitational ulceration and as ulceration of the post-phlebitic syndrome. It is proposed to describe briefly in tabulated form the clinical features of the different pathological states that are associated with varicosities. Clinically varicose veins can be distinguished as (1) primary and (2) secondary. The clinical features are classified under these headings as follows:

Primary	*Secondary*
Etiology	
Heredity. Pregnancy. Occupational stresses, of standing.	Usually secondary to deep-vein thrombophlebitis, especially where anticoagulant therapy not or inadequately carried out. Occasionally idiopathic.[1]
Oedema	
Minimal; swelling confined mainly to bulging of varicosities.	Most prominent feature, especially at the end of a day's work requiring standing.
Ulcers	
Malleolar in site, surrounded by darkish pigmentation and often eczema; usually single, and indurated cellulitis not a usual feature.	Not necessarily malleolar. Irregularly placed, multiple, often situated more proximally and associated with a greater amount of induration, eczema and cellulitis. Indurated cellulitis a prominent feature. Fungus infection of skin common.
Symptoms	
An ache on standing relieved by activity and walking. Nocturnal cramps. Signs and symptoms of complications, e.g. superficial phlebitis, pain of the ulceration and secondary infection. Distribution of varicosities conforms usually to the anatomical pattern of the long and lesser saphenous veins.	Bursting feeling and ache on standing. Severer symptoms generally. Intractable and recurrent painful ulceration. Repeated attacks of cellulitis in indurated areas, apart from ulceration of skin. Nocturnal cramps usually more severe and more frequent. Varicosities conform to irregular pattern with the presence of 'blow-outs' and feeder veins from ulcerative and eczematous skin.

PATHOLOGICAL CHANGES

Both primary and secondary varicose veins are complicated by venous stasis disease, resulting in severe skin changes of the distal parts of the lower extremity. It is generally accepted that the underlying cause of varicose veins in the absence of any deep-vein disease is an intrinsic defect in the vein wall of the saphenous system itself, in addition to the occupational factors which may keep a patient standing for long periods. As indicated, swelling is not a prominent feature in the primary varicosities, although ulceration and skin changes do occur. Nor is indurated cellulitis a prominent or regular feature. Radical and efficient removal of the offending varicosities, blow-outs and feeder veins from the affected skin areas results in satisfactory and lasting cures for many years in the great majority of cases.

The outstanding feature, however, of the ulcerative stasis disease of the lower extremities, which is usually secondary to deep-vein disease (from any cause), is the invariable tendency to swell, to produce incompetent superficial varicose veins, and to produce the severe crippling disabilities of varicose ulceration, eczema and indurated cellulitis[2]. These changes affecting the skin and subcutaneous tissues of the stasis area occur on the medial and lateral aspects of the distal third of the leg. The oedema is ascribable to lymphatic-vessel disease and to venous insufficiency, either or both factors being present in varying degrees in individual cases. Unless repeated episodes of inflammation have occurred, the lymphatic element ordinarily diminishes with time. It is severest in the early period following the acute thrombophlebitis to which it is an accessory. The venous element of the stasis arises either from permanent occlusion of the deep veins of the extremity or the tendency for reflux venous flow in a vein which has been rendered valveless by the original infection. The varicose veins which are characteristic of stasis disease appear in response to the general increase in the venous bed and pressure secondary to deep-vein disease of the legs. Venous stasis accompanied by an appreciably increased vein-pressure results in oedema. If one considers the end stage of deep-vein thrombosis in a deep principal vein as a stiff tube having thick walls of connective tissue and non-functioning valves, one can easily appreciate the considerable capillary pressure which occurs in the lowest part of the leg when the 'vein pump' is unable to counteract the hydrostatic pressure in the valveless principal veins. The secondary varicosities forming in such a limb are the subcutaneous component of the developing collateral venous system. Inasmuch as these veins are unsupported by fascia and muscle pressure they quickly dilate, rendering the valves incompetent, and they become themselves reflux venous channels. It is important to appreciate that these superficial varicosities secondary to deep vein disease are incompetent, and their presence merely aggravates venous stasis. It is therefore logical to ablate such varicosities, contrary to the generally-held

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view that no limb which has been the site of deep-vein thrombophlebitis should have such varicosities removed because this will lead to a greater degree of oedema. This erroneous belief is based on the concept that these secondary varicosities are the only channels for the return of venous blood from the lower extremities. These secondary veins serve no such function; they only help to perpetuate the pathological processes. There are other return channels through which venous return can efficiently take place, viz. muscular veins, recanalized diseased deep veins, and the capsular veins of the knee joint.

The skin changes which appear both in stasis disease and in primary varicose veins are characteristically located in the same areas, with minor difference in their situation. These areas are the medial aspect of the ankle above the malleolus in the course of the long saphenous vein system, and the lateral aspect at about the same level in relation to the lesser saphenous system. To explain this uniformity of the affected area it must be postulated that some peculiarity exists in the structural anatomy at these points or in the pressure relationships within the veins, which may be responsible for the changes in the skin and subcutaneous tissue. If such a peculiarity can be worked out a method might become available for the permanent healing of stasis ulceration. The probable etiology of the ulcers which appears in these areas is the presence here of the greatest number of communicating veins between the deep venous system and the superficial channels. This reasoning has been applied by Linton[3] in developing a method of individually ligating these branches. The presence of these imcompetent perforating veins has caused a number of workers to reduce venous stasis by means of uniform external pressure.

Besides the presence of the perforating veins in the area of ulceration, the character of the tissue changes which precede the breakdown of the skin must be considered. Changes advanced enough to make a return to normal unlikely must be recognized as a factor determining the need for total eradication of the diseased skin and subcutaneous tissues of the areas. In attempting to treat these conditions surgically one must take into consideration pathological processes that take place. This surgical therapy includes the removal of all the damaged skin and subcutaneous tissue together with the underlying fascia; the long and lesser saphenous systems, and the local perforating and feeder veins. The irreversible character of the tissue changes locally seems to warrant this radical approach.

The results obtained by this surgical treatment, and the observations on the secondary stasis changes and the anatomy of the perforator and feeder veins at operation, are described below.

CLINICAL MATERIAL

The surgical management of these cases of stasis ulceration of the lower limb is based on 1,000 cases of primary and secondary varicosities operated on over the past 10 years both in hospital and private practice. Of these 1,000 cases, 200 phlebectomies were performed on patients with a definite history of previous deep-vein involvement and showing signs of the typical post-phlebitic syndrome. In these 200 cases, 30 skin grafts were combined with the phlebectomy operation.

DETAILS OF TREATMENT

The preoperative management follows the conventional methods and is directed towards elimination of infection in and around the ulcerated area, complete control of the oedema of the foot and leg and the re-epithelization of the ulcerated area. Bed rest and elevation of the limb is absolutely necessary to achieve these aims. No ulceration will heal rapidly or completely without bed rest. The parenteral administration of antibiotics has reduced the morbidity considerably and relieved the pain of these skin conditions. Control of a commonly superadded fungus infection is absolutely essential. Repeated moist dressings of saline with and without antibiotic solutions are applied together with solutions of aluminium acetate. For the past year we have had most favourable results in the treatment of infected ulceration and skin lesions with a solution of streptomycin (1 g. to 100 c.c.) applied as a moist dressing and kept constantly moist with repeated applications or the addition of the solution to the dressing *in situ*. This moist dressing must be applied to the ulcer and infected area only, otherwise maceration of the healthy surrounding skin is likely to take place.

The severity and extent of the cutaneous and subcutaneous fibrosis and the extent of the varicosities is determined by palpation in the erect position after the oedema has subsided.

As already indicated, in the majority of cases of ulceration due to primary varicosities a complete and efficient eradication of the long and lesser saphenous systems, the secondary cutaneous branches, the perforators, and feeder veins, is all that is required. This eradication is really a phlebectomy of all the superficial varicosities.

It is not intended to describe in detail the surgical technique employed for the 1,000 cases over the past 10 years, but to discuss general principles. This procedure is a major one, not to be carried out as an out-patient procedure. The patient is carefully examined to exclude any other vascular problem such as obliterative arterial disease or arterio-venous fistula. We do not utilize any of the tests to detect perforator branches, because the technique is so radical that all these are extirpated.

Proper pre-operative preparation of the skin of the lower limb including the lower half of the abdomen is carried out.

General anaesthesia is employed. If a bilateral operation is required, one leg at a time is operated on with a week's interval. Careful marking of all superficial varicosities is carried out before anaesthesia. Bonney's blue is the best dye to use—others are inclined to run with cleaning and swabbing of the leg. All main trunks, secondary branches, and especially the feeder veins from ulcerated areas, are carefully marked in continuity.

High ligation of the long saphenous through a vertical groin incision is performed, exposing it with its tributaries at the saphenofemoral bulb. The tributaries are so often anomalous that they do not conform to the anatomical text-books; for example, a double saphenous is not uncommon and branches vary in number from 1 to 6. Extraluminar stripping of the main saphenous trunk is carried out with the aid of an extraluminar Mayo stripper, down to the ankle and even beyond to the foot. This will entail stripping

behind healed ulceration. We have found the intraluminar stripper quite inadequate for this purpose because of anatomical anomalies.

The radical excision of the secondary branches must be carried out by incisions—usually lengthy ones over these branches themselves, for we have found the use of intra- or extraluminar stripping techniques for these branches to be unsuccessful. This phlebectomy may be tedious, but the results justify the labour. These secondary branches invariably reveal a perforator communicating with the deep trunk. Multiple ligation along the course of these branches is not sufficient as such a technique could quite easily miss these perforators.

Excision of feeder veins from the ulcer-bearing area is absolutely necessary; otherwise recurrence of ulceration is sure to follow. These feeder veins are also attacked through a direct incision in the length of the limb and up to the edge of the healed ulcer.

Post-operative care includes supportive elastoplast type of bandage of the whole limb (over cotton bandaging; otherwise zinc-oxide sensitivity with severe results has been known to occur). Over the stripped skin area and below the adhesive elastic bandages special elongated dressings are placed over the length of the limb to prevent post-operative effusion of blood and haematoma. The patient is made to get out of bed the following day and walk. Repeated muscular activity is also encouraged while lying in bed.

For secondary varicosities combined with skin ulceration and other skin changes, the same pre-operative treatment is carried out and the same technique of varicose vein ablation is used, but in addition attention is directed locally towards the healed ulcer and surrounding skin. It is carried out either in one stage combining the phlebectomy and the skin grafting, or in two stages, when the phlebectomy is done initially and thereafter the skin grafting carried out at a secondary operation. The two-stage technique has the advantage that in some of the cases the original phlebectomy has been sufficient to maintain a good result with the ulcer and skin lesions. If grafting is continued with at the initial operation, the ulcer area is excised widely enough to reach good skin. The subcutaneous lesions, which are densely fibrosed and often contain small abscess-like areas filled with cloudy fluid, is removed in a single tissue-block, taking with it all the deep fascia adherent to its under-surface.

From 1 to 6 veins of major size have been found in cases perforating the fascia in this area to communicate between the subcutaneous and deep veins. These are clamped and ligated and a split-thickness skin graft, taken usually from the antero-lateral aspect of the opposite thigh, is used to cover the defect after complete haemostasis has been effected. The graft is meticulously sutured in place with absorbable gut to the edge of the healthy skin. The graft is applied directly on the muscle bed and if a hollow exists in the graft bed, sutures are applied to the middle of it to this muscle bed to obliterate dead spaces as far as possible. Proper supportive post-operative presure-dressings also help to achieve this. Post-operative antibiotics are always employed as well as ascorbic-acid tablets in massive doses. Bed rest is essential until the first dressing is removed, usually 7-8 days after grafting. As these sutures are absorbable there is no necessity to remove them. When the graft has healed completely, a progressive regime of graded ambulation is instituted. All the graft operations performed (30) have healed particularly well, with only one immediate failure. The success of these grafts is striking and one explanation offered is the efficient lymphatic bed present.[4]

PATHOLOGICAL CHANGES AND LOCAL ANATOMY DETECTED IN THE STASIS AREA

Complete removal of the stasis area in one skin-block together with its deep fascia has provided an opportunity to observe the pathological anatomy and tissue changes which are present. In all our cases 1-6 venous channels have been noted, passing through the deep fascia between the subcutaneous tissue and subfascial veins. Within the excised tissue itself these are large-walled tortuous venous channels, which tunnel the densely fibrous tissue. The number and size of the communicating veins diminish strikingly at the border of the stasis area so that, although there are many veins within the removed specimen, there are much fewer at the edge of the excision. The cut surface of the skin specimen is fibrous and wet with liquid fat and very often milky fluid. Cultures of this material are sterile.

POST-OPERATIVE CARE OF OPERATED LIMB

Recurrences of ulceration have been observed with this grafting technique, and one has noted that this has occurred in those patients who live too far away to return for constant and repeated follow-up examination, and in these subjects oedema has been allowed to recur. It is our experience in 200 cases already referred to that eradication of all superficial varicosities will not remove the pathological process that results in oedema. It is wrong therefore to promise any patient that swelling will be cured. If oedema still persists then it must be effectively and rigidly controlled. We advise elastic stockings, elastic bandages, or both. If elastic stockings are used they must be fitted to the other normal leg, or to the affected limb when all oedema has subsided, after a night's rest in bed with the foot elevated. The stockings must be made of heavy two-way stretch material and must cover the heel, leg and lower thigh. They can be made of either nylon or cotton. Where occasionally one stocking will not control the oedema then the patient should wear two. In exceptional cases an elastic bandage may have to be applied over the elastic stocking. Some may prefer the stocking over an elastic bandage.

Patients are taught by means of an instruction sheet the care of feet and legs, cleanliness, supportive bandaging to eliminate oedema, not to stand excessively, and to indulge in a 2-hour rest during each day if this is possible. The patient is also instructed to keep the foot of his bed permanently raised 6-9 inches.

Mention has been made of indurated cellulitis in stasis disease of the lower extremities. This is often a severely painful and recurring condition, characterized by acute cellutitis, severe local pain and a dense 'pancake' of stasis change in which all of the pathology of this condition exists without ulceration of the skin. For these conditions we have applied the same operative treatment as outlined for stasis ulceration, carrying the graft to normal skin.

CLINICAL RESULTS

Thirty grafts which have been applied by this method in 26 patients have been followed up for periods of 2 years. The results are summarised in Table I.

TABLE I. CLINICAL RESULTS OF GRAFTING

	No. of Grafts	*No. of Patients*
Number of failures	8	8
Number successful	22	18 *

* In 4 patients grafts were performed bilaterally.

TABLE II. POST-OPERATIVE OEDEMA IN RELATION TO GRAFTING

Grafts	*Post-operative Control of Oedema*	
	Good	*Poor*
Successful 22 in 18 patients ..	17	5
Failures 8 in 8 patients	1	7

COMMENT

It is hoped that this paper has indicated two definite groups of varicose veins, viz. primary and secondary, which both cause venous stasis disease. The secondary, however, is more severe and the accompanying stasis ulceration more marked. Stasis ulceration can be considerably relieved by a radical phlebectomy, with or without a wide excision of the ulcer-bearing area, to which a skin graft is applied. The incompetent communicating veins in the stasis area are likewise completely ablated. Failures do occur but only when the venous stasis is associated with secondary varicosities. These failures are most frequent in patients with marked and persistent oedema which is either uncontrolled by efficient supportive stockings or bandaging, or controlled with difficulty.

The techniques of ligation of the popliteal vein as recommended by Bauer,[5] or ligation of the superficial femoral vein as described by Linton,[6] are not included in the operative measures described here.

This work is based on 1,000 cases of primary and secondary varicosities operated on. Of these 200 were secondary varicosities for which a radical phlebectomy was carried out. In the latter group 30 cases were combined with grafting.

SUMMARY

The severity of stasis changes in the lower limbs leading to skin ulceration and other skin lesions is discussed on an etiological and pathological basis.

A radical phlebectomy of the long and lesser saphenous systems together with the excision of secondary branches, perforators and feeder veins is briefly described.

This operation together with excision of irreparably diseased ulcer-bearing skin is offered as a means of salvaging a great number of these severely crippled subjects.

The majority of ulcers resulting from primary varicose veins will heal with efficient eradication of these veins alone.

Failures with the more intractable ulceration associated with the group of secondary varicose veins do occur.

Operation to remove varicose veins secondary to deep-vein disease can be carried out without any further oedema or damage occurring in the affected limb. Two hundred such cases have had these secondary veins removed with satisfactory results. This dispels an old erroneous belief that it is unwise to remove such varicosities. It must be emphasied that this operation will not relieve the oedema but will relieve venous stasis.

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REFERENCES

1. Luke, J. C. (1941): Surg. Gynec. Obstet., **73,** 472.
2. Norwich, I. (1953): S. Afr. Med. J., **27,** 663.
3. Linton R. R. (1953): *The Surgical Treatment of Post-phlebitic Syndrome with Ulceration of the Lower Extremity*, 2nd. Congr. Int. Soc. Angiology, Boston, 20 September.
4. McGregor, A. L. Personal Communication.
5. Bauer, G. (1948): J. Int. Chir. **8,** 837.
6. Linton, R. R. and Hardy I. B. jr. (1948): Surgery, **23,** 458.