THE TREATMENT OF VARICOSE ULCERS AND VEINS*

BY GRANT P. PENNOYER, M.D.

OF NEW YORK, N. Y.

Varicose ulcers and other late results of varicose veins of the lower extremity cause prolonged, severe pain and disability, and frequently do not receive the attention which they deserve from the medical profession. Patients with much less disabling lesions of the head, chest or abdomen receive the most careful study by the senior members of the hospital staff, while the unfortunate, who has suffered for years with a chronic ulcer of his leg, is sent to the out-patient department. Here the extensive dressing required is sometimes regarded as a nuisance, and the case is assigned to a nurse or clinical assistant to apply an ointment and supporting bandage, while the doctor spends his time considering lesions more dramatic but much less important. The patients become discouraged with the inefficiency of the treatment, and often apply bandages and local applications as best they can for themselves. The result is that chronic leg ulcers are much more numerous than is our general impression. Those who are under treatment are patients in our hospital dispensaries where they do not usually see the members of this society. They frequently have visited their local doctors, but have either failed to receive relief or have been unable to meet the expense of prolonged treatment. The long, tedious series of dressings may even make the small clinic fees an economic luxury for them. The extreme gratitude of these patients for a cure, and the way they bring in their similarly afflicted friends are a source of real satisfaction to the doctor who works with them.

The indolent leg ulcer is always a result of some underlying pathology, and it is the proper recognition and treatment of this which is requisite for success. The ulcer is merely a symptom and sign of the real disease like the glycosuria of diabetes or the cough of tuberculosis. Success in healing does not depend upon the virtues of some local application, but upon the understanding and control of the underlying circulatory difficulty. These patients suffer infinitely more and longer than many for whom we advise major operations. The doctors in the out-patient departments should carefully study each case. An ulcer which fails to heal after a reasonable time under dispensary treatment should receive the attention of the experienced surgeon, and not be allowed to become discouraged and drift away.

The ulcers to which I refer have as their basis mechanical difficulties in the venous and lymphatic circulation. I am not discussing those originating from syphilis, diabetes, etc., but the underlying causes of the ulcer may be multiple. A so-called diabetic or syphilitic ulcer may be greatly benefited by the elimination of some varicose veins which are aggravating it.

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A review of the normal physiology of the venous return from the lower extremities is helpful in understanding the pathology in these cases. In the erect position, the propulsive action of the heart-beat and the arterial pressure have almost nothing to do with the venous circulation in the lower extremity. The enormous area of the capillary bed compared to that of the arterial slows the blood-current in the capillaries down to the point where there is scarcely any pressure behind it. Krogh\(^1\) points out that a single capillary may have a larger cross-section than the tiny arteriole which supplies a large group. Also, each time an artery divides, the cross-section of its branches is greater than that of the original trunk. This results in a rapid fall of blood-pressure as we reach the terminal arteriole. The normal capillary pressure is only four to five centimetres of water at the level of the heart, a pressure insignificant when compared with the pressure required to lift the venous blood from the lower leg back to the heart when we are in the erect position. The well-known pumping action of the muscles as they press on the veins, which are equipped with valves to prevent back flow, is the propulsive force behind the venous return from the dependent extremities. The vein, with its frequent valves, is really a series of chambers, each one of which as it is compressed by the muscles empties into the one above. The normal muscular activity, being as it is alternate contraction and relaxation, is ideal for this pumping action in the veins. It is evident why we have such discomfort when we are compelled to stand still for long. Our constant shifting positions from one foot to the other, etc., as we attempt it, are really reflexes to stimulate the venous circulation. Even when we deliberately dangle our legs, relaxed as much as we voluntarily can, the venous blood-pressure in the foot is far below the theoretical hydrostatic pressure which would result from its distance below the heart. The muscle tone, which is really rapid rhythmic contractions, is keeping this venous pump at work. Stand still, and the dorsal veins of the feet distend; exercise the leg muscles, and they collapse. During vigorous activity, the blood-pressure in the normal foot veins approaches zero, due to the rapid removal of blood from them by this pumping action of the muscles. Anything which cripples this venous pump, such as incompetence of the venous valves, immobilization, injury to the muscles, etc., cripples the venous return from the extremity, unless the extremity is elevated approximately to the level of the heart.

The superficial veins are deprived of this muscular support. They are provided with numerous communicating veins with their valves so placed that blood can flow from the superficial to the deep veins. As soon as the venous pressure in the saphenous veins rises above that in the deep veins, blood can flow freely into the deep system where the muscular activity pumps it upward. This provides a sort of safety valve against excessive pressure in the saphenous systems as long as the valves are competent. The pressure in the normal saphenous vein is much less than the hydrostatic pressure which would result from its distance below the heart and which one would expect to be required to keep the blood flowing upward.
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Other factors in the venous circulation are the varying negative pressure in the thorax during respiration and the positive pressure in the abdomen. The effect of normal respiration on the venous return of the lower extremity while standing is insignificant, as evidenced by study of the venous pressure during respiration. Increased abdominal pressure is necessarily transmitted to the abdominal veins and the lower extremity veins as they enter the abdomen. The extraordinary height to which this abdominal pressure can rise during violent effort has recently been measured by Murphy and Mengert. The abdominal veins have no valves, and this pressure can not only pump the blood on into the thorax, but also force it back against the valves in the veins of the thigh. During violent abdominal straining, our thigh muscles are also in strong contraction, which protects the deep veins but the saphenous system valves are subjected to the full pressure. The normal saphenous valves have been shown to give way at pressures of about 180 millimetres of mercury, which is not unusually high for the abdominal pressure during violent muscular work. Add to this pressure the constant strain of the hydrostatic pressure, and it is evident why varicose veins are so numerous. As the upper valves give way, more pressure is transmitted to those remaining below. As the succeeding valves give way, the whole superficial system of veins becomes wide-open channels with nothing to hinder the free regurgitation of blood downward. I do not say this mechanical back pressure is the only explanation of varicose veins but it is certainly present in every case of the common surface varices. Congenital and familial weakness of the veins certainly plays a part, and perhaps many other factors which we do not recognize and cannot control.

The source of all the difficulties resulting from simple surface varicose veins is this free downward regurgitation of venous blood towards the periphery, and the resulting capillary stagnation under pressure. The measured venous pressure in varices approaches the theoretical hydrostatic pressure which would result from the position of the vein in relation to the heart. This is still further increased by abdominal straining. This pressure is of necessity transmitted to the capillaries, as otherwise the blood current would be completely reversed in the capillaries. The blood in these veins is by analysis much higher than normal venous blood in carbon dioxide and non-protein-nitrogen content, and much lower in oxygen. The capillary permeability has been shown to be very sensitive to just these conditions, increased pressure, increase of carbon dioxide and lack of oxygen. The effect of this is a profound change in the nutrition of the subcutaneous tissue. As the deep veins are intact, the disease is entirely confined to the subcutaneous tissue, which is a constant and striking clinical fact even in the most advanced stages of oedema and ulceration.

The condition of the valves in the veins communicating between the deep and superficial systems is important. When these are competent, these perforating veins are constantly removing the blood from the surface veins, and the varices, being rapidly emptied, and the pressure in them thereby kept low,
may do no more harm than greatly add to the burden of the deep veins. This is the situation when we find large varices without clinical symptoms. The extra burden on the deep circulation is undesirable, and the patient usually has fatigue and weight sensations which he does not connect with his veins, or which he has had so long that he accepts them as natural. If the communicating veins do not have competent valves, they cannot so effectively decompress the surface varices. Furthermore, blood part way up the deep veins can escape into the superficial group, where it again falls back towards the foot to reenter the deep veins, thus completing a vicious cycle. If the varices are extensive, the volume of this regurgitated blood may be more than the total volume of blood returned to the heart to receive oxygen and nutrition. This vicious cycle of old blood has been demonstrated under the fluoroscope by lipiodol injections. This situation results in nutritional changes of the entire subcutaneous tissue out of proportion to the size of visible varices.

The pathology which results from this venous back pressure on the capillary circulation is very extensive. An early and almost constant clinical sign is the common pigmentation which results from the increased capillary permeability and diapedesis of red blood-cells. I consider this pigmentation as diagnostic of impaired nutrition from varicose veins, even when they are not visible. When ulceration has occurred, the element of infection and cellulitis becomes important. These ulcers are always surrounded by a zone of acute inflammation, and frequently there are exacerbations of this infection element when large areas of the leg become very hard, swollen and painful. It is the addition to the already existing venous stasis of the element of repeated and chronic infection which results in the lymphatic blockage and elephantiasis aspect of these cases. There is a local obstruction of the lymphatics in areas of cellulitis and infection. Subcutaneous injections of India ink, which is normally rapidly removed by the lymphatic circulation, remain relatively fixed in relation to the lymphatics for a long time. This has recently been shown by Kuhns to be true also in joints. The absorption of substances injected into joints, which are normally quickly removed by the lymphatics, is greatly delayed if the joint is acutely inflamed before the injection.

This lymphatic obstruction within the tissues, resulting from the prolonged chronic and repeated infection in the ulcer cases, added to the existing venous difficulties, gives all the necessary elements for the gradual formation of a true elephantiasis, as described by Matas, i.e., venous and lymphatic obstruction with repeated attacks of non-pus-forming cellulitis. Halsted, in studying the arm cases following breast amputations, made the same observations. Homans has always emphasized the lymphatic element in these cases. The progressive fibrosis of the subcutaneous tissue still further destroys the lymphatic circulation, and finally the whole subcutaneous tissue and skin of the leg is deprived of lymphatic as well as the normal venous drainage.

The above conditions are the late results of simple surface varicose veins,
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but there is also a group of cases where the deep veins have been damaged by a previous severe deep phlebitis or phlegmasia alba dolens. The late difficulty of the deep veins in these cases is usually not a diminution of the capacity of the veins as commonly supposed, but incompetence of the valves. These valves are just as important in maintaining the circulation of the extremities as the heart valves themselves. As soon as they are crippled, either mechanically or by infection, there is nothing to prevent the backward flow of blood in the dependent leg until the venous pressure is high enough to lift the whole column of blood up to the heart, a pressure way above the normal working pressure of the capillaries. These cases have the same difficulties as the simple surface varices, i.e., oedema, ulceration, chronic infection, elephantiasis, etc., but the disease is not confined to the subcutaneous tissue. Some of these cases have large surface varices, which are sometimes thought to be compensatory dilation. I believe that they are usually not compensatory but true varices which are the result of the high venous pressure. If they have a positive Trendelenburg, it is safe enough to obliterate them, but the patient receives little benefit. As a group, these cases are most discouraging to treat, as it is impossible to restore the deep venous circulation. Most of the failures of the Kondoleon operation for elephantiasis, which is an attempt to give the superficial tissues lymphatic and venous drainage by anastomosis with an intact deep circulation, are undoubtedly cases which fall into this class. The excision of ulcers and placing skin grafts on the deep tissues in also doomed to failure without an intact deep circulation, as well as the obliteration of the surface varices. Pressure by bandages, etc., is usually of little help. Reduction of the capillary pressure and restoring the venous circulation by elevation is the only way to heal the ulcers in this group. These cases are usually easy to recognize by their characteristic history of severe oedema which involves the superficial and deep tissues dating directly from the attack of milk leg.

With these points in mind, it is much easier to develop a rational therapy for the ulcer cases. If the patient has large surface varices, and has not progressed to the stage of lymphatic block, chronic brawny oedema, and elephantiasis, remarkably rapid and permanent cure can be obtained by sclerosing the offending veins by chemical means. The ulcer in these cases usually has obvious and intimate contact with the varices. There is little or no oedema. Large veins lie just above the ulceration and very frequently under it, in which it is easy to demonstrate a positive Trendelenburg sign. The majority of the cases fall into this group. The chemical sclerosis of the offending veins by injecting them will often immediately relieve the patient of all pain and initiate healing in the ulcer. We have in our series an old lady of eighty who had had an ulcer of this type unhealed for forty years, which healed in a month after obliteration of a few veins. The larger the veins, and the more obvious their relation to the ulcer, the more satisfactory is the result of this treatment. The local application to the ulcer is immaterial.

If the ulcer patient has progressed to the point of hard, brawny oedema
or early elephantiasis, the problem is much more difficult. The varicose veins are frequently lost in the dense subcutaneous tissue. They often have no obvious relation to the ulcer, and the pathology may seem to be out of all proportion to the size of the veins. These patients are often fat, and it is frequently impossible to do a Trendelenburg test. The whole subcutaneous tissue has lost its normal venous and lymphatic drainage. Obliteration of the varicose veins in these cases should be done but it is sometimes of little benefit. If it fails, these cases with large chronic or repeatedly recurrent ulcers should be admitted to the hospital and operated upon according to the method of Homans.¹⁴ The results are very satisfactory in the twelve cases we have done. I have already shown three of them before this society. The ulcer is excised along with the underlying deep fascia and scar tissue, and Thiersch grafts are laid right on the deep tissues, whether it is periosteum, tendon or muscle. Thiersch grafts are used as the large areas involved can be covered more quickly by this method, and the results are good. The grafted area receives a new source of lymph and venous drainage from the deep circulation.²⁰, ²¹ The hard brawny œdema disappears from this area, and the new skin, now properly nourished, is soft and normal. The principle is similar to that of the Kondoleon¹⁷ operation for elephantiasis, in which large areas of the deep fascia are excised to allow free anastomosis between the deep and subcutaneous circulation. If ulcers recur, they occur on the edge of the grafted area, or in an entirely new area, where the deep fascia is still in situ.

Unsuitable for this treatment is the occasional case with damaged deep veins from a severe phlebitis. It is sometimes difficult to be certain that the case falls into this group, as many of the post-phlebitis cases respond well to treatment. Extensive œdema of all the tissues, superficial and deep, is the early and ever predominant feature of these discouraging cases. DeTakats⁴ has demonstrated how the X-ray is valuable in determining if the disease is confined to the subcutaneous tissue. In a properly exposed plate, the layers of the soft parts can be made out especially when the subcutaneous tissue is very much thickened.

Trout¹⁸ published a series of cases successfully operated upon essentially according to the method of Homans, but added a point to the technic. He excised a strip of fascia above the ulcer. This combines the Kondoleon operation with Homans¹⁹ excision of the ulcer. This involves raising flaps of skin, which in the poorly nourished diseased tissue is precarious. The only case I tried this on recently sloughed a large area of one flap.

As to technic, I believe that the skeptics of the injection treatment for varicose veins have either not tried the treatment or are unable to overcome the doctor’s innate fear of intravenous medication, embolism, etc. Homans, long a skeptic, now acknowledges it is an excellent way to heal ulcers but believes the injected veins all recanalize. This is not true in our experience. Our series now numbers over a thousand cases, about a hundred of which I have been able to follow over three years. I admit that some veins do
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recanalize. The exact percentage is difficult to state, as it is often hard to be sure whether a vein is really the old vein or a new one. Many of the cases do, in time, form new varices. The treatment does not pretend to rid the patient of the fundamental causes for his veins, which are naturally always present, but simply to destroy the veins present at the time. We have many cases in the clinic who have been previously operated upon by distinguished surgeons who have developed large new varices, frequently right in the scars, where all the veins have been removed. Veins, on the laboratory shelf, hardened in formalin did not recanalize. This tendency to form new veins can be diminished by paying particular attention to the trunks in the thighs. I can see no necessity for ligation, as they can be easily chemically obliterated. The internal saphenous vein may be varicose, i.e., its valves be incompetent, and yet not be visibly dilated or tortuous. To make it still more difficult, it frequently cannot be palpated in the thigh fat. In these cases it can usually be found by the impulse of a fluid wave transmitted from a percussion tap on the vein below the knee, where it is dilated and tortuous. Often there are several trunks in the thigh besides the internal saphenous. If the recurrence of new veins in the leg is rapid, one should strongly suspect he has missed one or more varices in the thighs, the back pressure from which is stretching out the leg veins. The occasional recanalizing of a treated vein and the usual tendency in time to form new veins is not a serious objection to the injection treatment. It is such a simple matter to give one or two more injections as it proves necessary. A simple treatment given to an ambulatory patient is such a contrast to repeating the old operative procedure. The recurrences, taken in time, are usually much smaller than the original veins. I always explain to my patient that I am unable to eradicate the fundamental causes of his varicose veins, and am simply removing the veins which he has at the present time.

Recanalizing of injected veins can be largely prevented by good technic. It is perfectly true that the characteristic hard thrombophlebitis which follows the injection does not necessarily mean a permanent sclerosis. Too weak injection fluid, excessive dilution by blood in a large vein, too rapid circulation in the vein may cause a chemical phlebitis of insufficient severity to destroy the vein permanently.

Another limitation of the treatment is the occasional occurrence of ulcers. The solutions are all caustic. The essence of the treatment is a chemical destruction of the intima of the vein, and a drug with sufficient irritating power to do it will destroy the poorly nourished subcutaneous tissue of these cases, statements of drug manufacturers notwithstanding. I have seen these sluggish chemical burns occur after all the commonly used solutions. If the leakage has been slight and rather deep, the actual necrosis of the skin may be delayed for several weeks, the area meanwhile looking like a localized chronic infection. If the leakage has been extensive, the sloughing will be very prompt, and discouragingly slow to heal. With good technic, this accident should be rare, certainly not in over 2 per cent. of cases. I always
explaining this possibility and probability to intelligent patients to protect the treatment and myself from unjust criticism. Minor points in technic diminish the possibility of slough in addition to skill in venous punctures. A very easily sliding syringe and not too small a needle help, so when the needle is properly placed, the reflux of blood into the syringe is very free and the injection can be made almost without resistance. Also, if there is the slightest doubt as to the position of the needle, or if it has slipped in and out of the vein repeatedly, one should be very quick to acknowledge failure, and either try a new vein or abandon the treatment till a later date. I have never seen serious complications from these leakage sloughs.

We have had no emboli in our series. They have been reported in the literature, and can usually be traced to cases where the injections have been given in the presence of a pre-existing phlebitis. Injections should never be given if there is any possibility of infection being already present in the veins, as the chemical irritation seems to aggravate it.

Our method of injecting veins is very simple. We have used sodium salicylate, quinine and sodium morrhuate solutions. For extensive and permanent sclerosis, I believe that sodium salicylate is the best, but the severe cramp which follows its injection frightens many patients away to clinics who are using the other two solutions, the injection of which is painless. For the ordinary varices of the lower leg, I have the patient sit on an examining table with the leg in my lap as I sit in front of him. I do the lower veins first. As the blood-current is reversed, it is much easier to obtain good sclerosis in the thighs and larger veins above, if the smaller veins below are occluded first. It is easier to identify and inject all the lower leg veins if they are treated first. I do not use tourniquets unless the veins are very large or the injection is made in the upper thigh. In large sacculations, etc., where there would be excessive dilution, I attempt to inject the vein collapsed and hold the solution in the treated segment by double tourniquets. This is easily accomplished by raising the extremity and applying the tourniquets after the needle is placed in the vein. If all the veins below are blocked, good sclerosis can be obtained even in the internal saphenous in the thigh by injecting with the patient standing. I have never seen a thrombosis of the deep femoral result from an injection. The blood-current in it is so much more rapid than in the varix that the sclerosing agent is too rapidly diluted and swept on to do any harm.

For the comfort of an ambulatory patient, I recommend only one injection be given at a time and not repeated until the acute inflammation of the previous injection has started to subside. This usually means three to seven days' intervals. Under this plan the patient can usually continue with all his regular activities throughout the treatment.

If the injected vein is large, it is a distinct help to keep the vein collapsed by a firm pressure bandage or stocking during the sclerosing process. The thrombus which forms and organizes in the vein is smaller, and the hard tender swelling is much less noticeable. I have done many without compres-
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sion bandages, and it seems to me those who feel that they do not help in the large veins, have not applied them so as to keep the veins really collapsed.

The technic we have used in excision of ulcers is easy. The patient is kept in bed with the leg elevated until the oedema has been reduced as much as possible. This usually requires seven to ten days. If there is much purulent discharge and active infection, Dakin wet dressings may be used. The whole ulcer area is then excised, taking particular pains to remove all the underlying scar tissue and deep fascia. Success depends upon placing the grafts on healthy deep tissues. Any sort of graft probably could be used, but we have used Thiersch grafts as the large areas exposed can be covered rapidly this way, and the thigh from which they are taken heals rapidly without scar. The individual grafts are laid so they overlap each other and the edges of the wound, so they completely cover the raw area. They seem to take well right on periosteum, tendon and muscle. Even if take has not been 100 per cent., the region has a more normal venous and lymphatic circulation, and when it does heal, the skin will be pliable, soft and healthy. Perforated rubber tissue is placed right on the fresh graft and stuck around the edges of the wound with chloroform so it cannot shift. An even layer of gauze dressing is placed over this, and then a thick flat rubber sponge is strapped firmly over this whole area with wide adhesive tape. Further pressure is obtained by a firm circular bandage. This pressure is a very important point in getting the graft to take. The first dressing is done in five to seven days according to the odor. The patient is kept in bed with the leg elevated until the wound is healed and the grafts firm, which is usually about three weeks. When the patient first gets up, the grafts are apt to become very cyanotic, and their nutrition must be watched carefully the first few weeks in order not to lose them. Supporting pressure with sponges or absorbent cotton bandaged firmly over the area, and frequent periods of elevation help. As the new circulation establishes itself, the grafts become thicker, firmer and more like the normal skin.

To summarize, varicose ulcers, if they receive the study they warrant, can be made to heal. This can usually be accomplished by obliterating the varicose veins. In legs with chronic, brawny oedema and elephantiasis, where the lymphatic and venous circulations of the subcutaneous tissue are diseased beyond repair, permanent cures can be obtained by excising the ulcers, together with the underlying scar tissue and deep fascia, and placing skin grafts directly on the deep tissues. If the deep circulation is also diseased, this case is discouraging and elevation is the only cure.

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GRANT P. PENNOYER