Clinical Study and Treatment of Varicose Veins

By Hugh Montgomery, M.D., and Harold A. Zintel, M.D.

In general, the common management of varicosities of the lower extremities often leaves much to be desired. There appears to be too little understanding of the basic physiologic disturbances, too much delay in initiating definitive therapy, and altogether too frequent use of inadequate and even harmful therapeutic measures. Perhaps the lack of interest in the patient with varicose veins is partially due to the fact that seldom do varicosities shorten the patient's life or cause death. It is possible that the many tests for varicosities and the multiple nomenclature associated with some of these tests have discouraged a better general understanding of this subject. The symptoms of varicose veins and their complications (vague discomfort, pain, edema, dermatitis, ulceration and hemorrhage) do produce considerable disability over many years and are real problems to the patient. The high incidence of varices in the general population demands that all general practitioners as well as the surgeon and vascular specialist have a basic understanding of these problems. We should have a sound idea of the underlying physiologic disturbances and the most useful therapeutic measures. The proper diagnosis should be made early, and the type of therapy which will give the patient the greatest relief and promise for the future should be promptly instituted.

From the Vascular Section of the Edward B. Robinette Foundation, Medical Clinic, Hospital of the University of Pennsylvania and the Department of Surgery, School of Medicine, University of Pennsylvania, Philadelphia, Pa.

It is the purpose of this paper to present briefly the normal and abnormal anatomy and physiology of the major veins of the lower extremity, to accent the importance of venous and opposing pressures in the problem, to outline a simple method of testing for the various types of varicosities, and to describe the types of therapeutic measures used.

A varicose vein by definition is an enlarged and tortuous vein. The present discussion is limited to the major venous channels of the lower extremity and includes neither the small, isolated, superficial cutaneous varices, the so-called spider web nevi, nor arteriovenous fistulas. The problem is with veins having incompetent valves ("incompetent veins"), regardless of whether or not the veins are as yet enlarged or tortuous. Because of valvular incompetence the veins will eventually become enlarged and tortuous, and other segments of the same vein and/or adjacent veins will also become involved.

Etiology

The three common causes of valvular incompetence and varices are as follows. (1) Hereditary: It is not unusual to find that several individuals in each of several generations of a family have marked varices. (2) Occupational: The exact role of an occupation requiring standing for prolonged periods of time is not known. It is assumed, however, that prolonged standing, with or without moving, produces varices more quickly in the individual prone to develop varices than in an individual not prone to the development of
varices. (3) Inflammatory: Inflammation as produced by thrombophlebitis may destroy the valves or render them incompetent. Thrombophlebitis occludes veins temporarily, and though the occlusion is in time relieved by recanalization, the valves remain incompetent. This also occurs following the injection of sclerosing agents. One must keep in mind the distinction between a vein having incompetent valves, which for practical purposes is synonymous with the term "incompetent vein," and the "occluded vein," a vein that has lost its lumen.

**Normal Physiology**

There are no valves of the vena cava and, more frequently than not, none of the common and external iliac veins. According to Bas-
majian only 1 to 7 per cent of patients have a valve in the common iliac vein and only 24 per cent in the external iliac vein. The majority of individuals (67 per cent) have their most proximal valve in the common femoral vein immediately above the entrance of the great saphenous vein. There is great variability of the number of valves found in the major venous channels (table 1). When a normal individual stands erect the effective hydrostatic pressure in the veins at any level in the leg is equal to a column of blood extending up to the right heart. In most individuals the proximal valve in the common femoral vein supports a column of blood extending up to the level of the right heart. There may be no other valve in the common femoral vein (table 1).

When a normal individual is in the supine position the venous pressure in any vein of the leg is roughly that of a column of water 15 cm. high. The effective osmotic pressure of the plasma is some 20 cm. of water. This increases somewhat on prolonged standing because of ultrafiltration into tissues. The hydrostatic pressure in the tissues in the leg in the supine position is something under 10 cm. of water. On prolonged standing this increases little in the subcutaneous areas, but may increase in muscle to some 50 cm. of water. In the average individual standing still, the venous pressure just above the ankle is equivalent to some 135 cm. of water (table 2). The pressures in the deep and superficial veins are about the same. Forced expiration with the epiglottis closed increases pressure by 50 cm. of water to make the total pressure some 185 cm. of water. Thus under the circumstances described or in an individual coughing, sneezing or straining, the venous pressure may be as high as the brachial artery pressure of 185 cm. of water (137 mm. Hg).

![FIG. 1. Schema of veins of leg.](http://circ.ahajournals.org/)

Asmussen, Christensen, and Neilsen have estimated that 500 cc. of blood may pool in the legs of normal persons standing quietly. On walking, the milking action of the muscles on the deep veins becomes effective, and the pressure in the veins at the level of the ankle is almost immediately reduced to some 70 cm. of water. Because the action of the muscles on normal veins with competent valves propels the blood toward the heart and tends to reduce venous pressure, it is referred to as the action of the "venous heart."

A simplified schema of the venous blood flow of the lower extremity is shown in figure 1.
The flow is normally upward and inward toward the common femoral vein, upward in the popliteal, superficial femoral, long and short saphenous veins and inward through the communicating veins. The blood flow of a cross section of the leg has been described as being similar to the cross section of a wheel with the blood flow inward through the spokes to the hub. Only below the ankle level can venous blood normally flow both inward toward the deep vein and outward toward the skin.

The superficial veins of the legs are poorly constructed to withstand marked increases in pressures for very long because they do not have strong fascia and muscle tissue support. This probably explains why primary varices, or varices of the superficial vessels are those most frequently encountered. Valvular incompetence of the deep veins with secondary varices of the lower superficial leg veins occurs most frequently following deep thrombophlebitis.

After elevation and then dependency of the leg, the normal rate of filling of its superficial veins is slow because refilling is by arterial blood flow through the capillaries. The normal filling time in the veins of the calf is usually considered to be 30 or more seconds. It may, of course, be lengthened by vasoconstriction or shortened by vasodilatation such as occurs in hot weather. When the valves are incompetent the filling time of the veins is usually between 2 and 10 seconds. Less than 30 seconds is considered to be a definitely abnormal filling time (fig. 2).

**Abnormal Physiology**

Primary varices, those of the long and short saphenous veins, represent the most frequently encountered type of varices in clinical practice. Their first appearance is often in the thigh or upper leg. Varices of the short saphenous vein are encountered less frequently than varices of the long saphenous system. In the development of primary varices of the long or short saphenous veins, continued excessive venous pressure and/or inherently weak valves and the lack of strong supportive perivascular tissue produces the incompetence of the proximal valve of the vein. With the development of incompetence of a valve the next most distal valve is subjected to a greater venous pressure, and it becomes more susceptible to becoming incompetent. Eventually the valves of the entire vein may become incompetent and the vein dilated and tortuous. As previously stated, occasionally the valves of a perforating or communicating vessels (usually in the region of the low thigh) become incompetent. When this is the case the first evidence of a varix may be a dilated isolated segment of the saphenous vein sometimes referred to as a "blowout." Occasionally, valves of the saphenous vein above and below the "blowout" may be competent.

![Veins Incompetent Diagram](image)

**Fig. 2.** Filling time of veins below a tourniquet level. N: normal filling time of 30 seconds or more. A: abnormal filling time of less than 30 seconds.
vein down to that point may have incompetent valves.

Postinflammatory or secondary varices usually follow old deep thrombophlebitis and usually appear as collateral channels first in the lower leg and ankle region and frequently are confined to the lower leg. Patients who develop such secondary varices usually give a history of having had old deep thrombophlebitis, or milk leg, three or more years prior to the development of visible varices although the varices may appear as early as one year and as late as 20 years after the phlebitis. The patient will usually, but not always, give a history of having had phlebitis following operation, delivery, trauma or severe infection. In a series of 246 cases reviewed by Ochsner and his co-workers, 24 per cent of the patients had none of the previously mentioned conditions, and 4 per cent of the patients were considered to have phlebitis resulting from varicosities.11 The valves of the deep venous system, superficial femoral and popliteal veins are destroyed as previously described. Since the hydrostatic pressure is greatest in the most dependent portion of the leg, it is the veins in that part that develop varices first. (It is, of course, possible for an individual to have primary varices and then develop secondary varices either by extension or by reason of further valvular incompetence resulting from deep or superficial phlebitis.)

The blood flow through varices may actually be reversed when the individual stands.12 This can be demonstrated by injecting radiopaque material proximally in a varix and then demonstrating a downward or peripheral migration of the radiopaque material by serial x-ray films. The lack of competent valves and probable reversal of blood flow indicate that the patient would be better off with these abnormal vessels eradicated, provided there is evidence of adequate collateral venous return.

In the patient with varices the venous pressure in the supine position and that on quiet standing can, for practical purposes, be taken to equal these pressures in the normal.13 Marked pressure differences between those having normal and those having abnormal veins are apparent only when the subjects walk. In the presence of varices of the primary type the venous pressure is slightly reduced, but is not reduced to a normal value3 on walking. When secondary varices exist there is no reduction in pressure on walking4, 10, 14, 18 (table 2). It is undoubtedly the abnormally high walking venous pressure that leads to sensations of tightness, pain, edema, dermatitis, and ulceration of the leg. Patients with primary varices and especially those with secondary varices have venous hypertension when walking. Treatment should, therefore, be directed toward reduction of the walking venous pressure. When the incompetent saphenous vein of an individual with primary varices is occluded by digital pressure or tourniquet, the venous pressure is reduced by walking to some 70 cm. of water, a normal reduction. One would therefore expect obliteration or ligation of the long saphenous vein to restore the normal pressure relationship in patients with primary varicosities of the long saphenous vein, and this is the case. As previously stated, it has been estimated that a

Table 2.—Pressures (Average, Various Authors) in Superficial Vein of Ankle

<table>
<thead>
<tr>
<th>Condition</th>
<th>cm. H₂O</th>
<th>mm. Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subject supine</td>
<td>15</td>
<td>11</td>
</tr>
<tr>
<td>Normal subject leg elevated</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Normal subject standing still</td>
<td>135</td>
<td>100</td>
</tr>
<tr>
<td>Normal subject standing still with forced expiration†</td>
<td>185</td>
<td>137</td>
</tr>
<tr>
<td>Normal subject walking with or without tourniquet</td>
<td>70</td>
<td>52</td>
</tr>
<tr>
<td>Primary varices, subject supine</td>
<td>15</td>
<td>11</td>
</tr>
<tr>
<td>Primary varices, subject standing still</td>
<td>135</td>
<td>100</td>
</tr>
<tr>
<td>Primary varices, subject walking</td>
<td>95</td>
<td>70</td>
</tr>
<tr>
<td>Primary varices, subject walking with tourniquet</td>
<td>70</td>
<td>52</td>
</tr>
<tr>
<td>Postphlebitic, subject supine</td>
<td>15+</td>
<td>11+</td>
</tr>
<tr>
<td>Postphlebitic, subject standing still</td>
<td>135+</td>
<td>100+</td>
</tr>
<tr>
<td>Postphlebitic, subject walking</td>
<td>135+</td>
<td>100+</td>
</tr>
<tr>
<td>Postphlebitic, subject walking with tourniquet</td>
<td>135+</td>
<td>100+</td>
</tr>
<tr>
<td>Effective osmotic pressure of plasma</td>
<td>20</td>
<td>15</td>
</tr>
</tbody>
</table>

* 120 cm. to right atrium.
† 50 cm. H₂O produced by forced expiration against closed glottis.
normal person standing still "pools" approximately 500 cc. of blood in the legs. The amount of pooling in patients with large varices must be considerably in excess of this amount.

Mayerson and associates\(^1\) believe that an increased blood volume explains the slight increase in supine resting venous pressures observed in some patients with varices (table 2). Chapman and Asmussen\(^6\) found that cardiac output and systolic pressure were decreased after ligation of varicosities. In the postphlebitic leg the valves of the deep veins are usually incompetent and the valves of the superficial veins may have also become incompetent. The standing pressures in such veins are moderately in excess of that of normal veins and of primary varices. Walking with and without tourniquets fails to decrease this pressure, and may increase it presumably because the tourniquet obliterates only the superficial veins and the deep veins are either incompetent or occluded.

**Examination of the Patient with Varices**

The physical examination of the patient with varices does not require a special knowledge of the complicated terminology associated with the many tests. The necessary equipment consists of (1) a place for the patient to lie down, (2) a watch with a second hand, (3) a pen or a marking pencil, and (4) a tourniquet of soft rubber tubing at least three-eighths of an inch in diameter. The clothing is arranged so that the entire lower extremities are exposed. The legs are examined for evidence of scars, erythema, edema, pigmentation, ulcers, any active phlebitis, arterial pulsations and any severe blanching on elevation. With the patient standing, the course of the more conspicuous veins is marked on the skin. The tension of these vessels is noted by simple palpation. Less conspicuous vessels may often be demonstrated after warming the patient, or by having the patient strain by forced expiration with the glottis closed in order to increase the venous pressure. The remainder of the examination consists of observing the rate of filling of the varices with and without the use of a tourniquet.

With the patient supine, one leg is raised and supported at a 45 degree angle for 30 seconds. The patient quickly assumes the standing position with his weight equally distributed on both feet, and the filling time of the varices is noted. A filling time (without tourniquet) of less than 30 seconds indicates abnormal filling (fig. 2). The maneuver is repeated, again starting in the supine position, with a tourniquet placed around the thigh at a level approximately 6 cm. below the inguinal fold in the midline and the patient quickly assumes the upright position. The tourniquet should prevent reflux blood flow through an incompetent long saphenous vein. Moderate tourniquet pressure will easily occlude the superficial veins. (The deep veins will not be occluded unless the tourniquet is very tight.) When the varix has filled to what is thought to be a tension equivalent to the tension observed when the patient was standing without a tourniquet, the vein is said to be filled and the filling time is noted. The maneuver is repeated, applying the tourniquet at the mid-thigh, lower thigh, upper leg and at levels down to the lowest varices.

In the case of the simplest, most frequently encountered type of varices, primary varices of the long saphenous vein, the tourniquet at high thigh will considerably delay the venous filling to 30 seconds or more, and one can assume that the communicating veins and the deep veins are competent (fig. 2). If, however, with the same use of the tourniquet, there is little or no delay in filling, an incompetent communicating vein is present below the level of the tourniquet. Application of the tourniquet at successively lower levels will demonstrate the site of this and perhaps other incompetent communicating veins (fig. 2). A tourniquet above the level of any incompetent communicating vein will not prevent the contribution of this incompetence to the filling of a varix of the lower leg, but a tourniquet applied just below it will. Frequently palpation of the thigh in the area of an incompetent communicating vein will reveal a "soft spot" in the tissues which indicates the point of emergence of the incompetent communicating varix into the saphenous vein in the subcutaneous fat.
This point of emergence is usually slightly tender. Digital pressure on the vein in this area will produce a conspicuous delay in the filling time of the varices if it is the only, or the lowest incompetent communicating vein. Of course, a tourniquet at no level will delay filling of the varices if there is incompetence of the valves of all three systems of veins, the superficial, deep and communicating (fig. 2). An incompetent short saphenous vein is distinguished from an incompetent long saphenous vein by the posterior position of the former in the calf and by the fact that firm pressure by the finger in the lower portion of the popliteal space will delay its filling.

In summarizing the information (fig. 2) obtained as previously described, one can determine whether (a) all the veins have competent valves, and, if there is incompetence, whether it is of (b) the long saphenous vein, (c) the communicating veins, (d) the deep superficial femoral and popliteal veins, (e) combinations of the former, and (f) the short saphenous vein.

Before surgery is considered for the treatment of varicosities it is necessary to demonstrate that the deep veins are not occluded. This is done by firm bandaging of the leg from the ankle to the groin with an elastic or rubber bandage and having the patient walk for at least five minutes.17 The bandage prevents blood flow in the superficial veins but not in the deep veins or arteries. If the deep veins are occluded by disease the patient will, with such bandaging, complain of the severe pain of ischemia while walking. If there is evidence of occlusion of the deep veins, then extensive surgical excision of the varices is contraindicated. A single tourniquet will not take the place of such bandaging because flow may take place around an occlusion of a deep vein by way of communicating and superficial veins.18

**Surgical Treatment**

In general the best treatment of primary varices of the long and short saphenous vein is by surgical means. In the past we tried (1) injections of sclerosing agents, (2) ligation of the long and/or short saphenous veins, (3) combinations of ligation and sclerosing agents, (4) ligation of incompetent communicating veins, and (5) multiple ligation. At the present time it appears that vein stripping is the most useful method. The method of vein stripping was used a number of years ago and then for some unknown reason fell into disrepute. It has been more effective, however, in the treatment of varicosities than have any of the previously mentioned methods or combinations of methods which are described above.

According to a recent review of the literature by Fenney,19 the injection treatment of varices is followed by recurrence of varices in 57 to 98 per cent of the individuals so treated, and combined ligation and injection in 15 to 96 per cent of the individuals. Apparently the longer the individuals are followed the higher is the recurrence rate.

Vein stripping accomplishes in one operation more than could be hoped for by any other method or combination of methods. The data reviewed by Fenney indicate that great saphenous ligation, ligation of the other superficial veins at the saphenofemoral junction, and stripping of the great saphenous vein are associated with recurrences in from 2 to 7 per cent of properly selected patients with primary varices. Stripping abolishes all of the communications to the veins stripped and does more than abolish the communicating vein or veins demonstrated by the examiner. It leaves no segments of superficial and communicating veins to become dilated and tortuous after a collateral circulation has been established. It does not leave an occluded vein which can later become recanalized. The disadvantages of vein stripping are that patients are asked to remain in the hospital until the fifth postoperative day and that some patients may have slight transient edema of the ankle region for several weeks following operation.

For superficial primary varices and varices of the long and short saphenous veins, stripping of the involved vein or veins is advocated provided the deep veins of the leg are not occluded, as demonstrated by the compression bandage test previously described. For stripping of the long saphenous vein the patient is
placed supine on the operating table with the lower abdomen and both extremities completely exposed. The involved leg is then completely prepared with an antiseptic agent. The foot and the toes are wrapped in a sterile towel, but the ankle region is left exposed. The saphenofemoral area is then exposed, as for a saphenofemoral ligation, through a vertical incision approximately 2.5 inches long. The long saphenous vein is identified and divided between Kelly clamps approximately an inch and a half below the saphenofemoral junction. A chromic no. 0 ligature with a half knot is placed loosely around the vein distal to the clamp on the distal end of the severed vein. After applying hemostats to the cut edges of the vein, the small end of a flexible shafted intraluminal stripper is easily introduced into the lumen as the clamp is released. With gentle probing of the vein the stripper is easily introduced to just below the level of the knee. Not infrequently it can be inserted to the level of the ankle. When the tip of the stripper has been advanced as far as possible, a half inch incision is made over the tip of the stripper and the vein is exposed and divided. A clamp is placed on the distal end of the vein. The hemostats are removed and the stripper is advanced until the large olive tip of the proximal end of the stripper engages the proximal end of the vein segment. The catgut ligature is then tightened and the knot completed around the vein and the shaft of the stripper. The vein segment is then removed by firm traction on the distal end of the stripper. The skin overlying the stripped segment is then compressed with the spread fingers of both hands over a folded towel. Usually two to five minutes of such compression will control the hemorrhage from the ruptured communicating and collateral vessels. If the entire saphenous vein has not been removed, the procedure is then repeated attempting to remove the remaining segment of vein to the ankle. Occasionally the long saphenous vein is exposed just anterior to the medial malleolus and the vein is stripped upward instead of downward. Sometimes this is the only possible means of stripping the veins. Finally the uppermost, remaining portion of the saphenous vein is excised together with its various branches, and the saphenous vein is ligated flush with the surface of the common femoral vein. The entire leg is moderately compressed with an elastic bandage extending from the toes to the groin. Ambulation is allowed on the evening of the day of operation. On the fifth day after operation the sutures are removed, the use of elastic bandages is discontinued and the patient is allowed to go home with no restriction of ordinary activities. In individuals having very tortuous varices it may be impossible to strip the veins and multiple ligations may be necessary. This difficulty favors the early treatment of varices.

For the patient with a single or with several demonstrated communicating veins producing a blowout of a very short segment of the saphenous vein, excision of the segment and of a portion of the communicating vein will frequently suffice. This assumes, however, that the patient probably does not have nondemonstrable incompetent communicating veins. For this reason we frequently advise stripping of the upper or even of the entire long saphenous vein even though only a short segment of the saphenous vein is involved. Varices of the short saphenous vein are treated in a similar manner. The initial incision is made transversely in the lower popliteal space.

In other hands good results have been obtained, without stripping, by multiple ligations and excisions of segments of incompetent superficial and communicating veins. When performed in numerous stages under local anesthesia the procedure has the advantage of keeping the patient ambulatory.\textsuperscript{20} It is doubtful, however, whether the multiple operations on an ambulatory basis actually save the patient much time or expense.

In general, it is best to inform patients preoperatively, especially those with primary varicosities, that operations cannot cure the underlying cause of varices. When varices develop because of an inherent vessel or valvular weakness, it is entirely possible that varices can develop in other vessels. The patient should know that operation will not eliminate all of the dilated veins of the legs when such dilated
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veins are tributaries of the long or short saphenous trunks. They should understand that the incompetent veins causing the dilatation of these tributaries must be eradicated by operation. Any few remaining dilated vessels are dealt with very satisfactorily after stripping by injection of 1 cc. of sodium morrhuate or similar sclerosing agents. Usually the very small or "intracutaneous" varices are not treated by excision, and can be lessened by individual injections if the patient objects to cosmetic defect. Patients should know that the elimination of these tiny "intracutaneous" varices is very time consuming. The patient can be told that intelligent surgery will prevent the more serious symptoms and complications of varices. With proper treatment he can live comfortably, but he is not actually cured of his tendencies to develop varices.

For the patient with incompetent deep veins, popliteal and superficial femoral, we have usually employed elastic support, rest, and occasional grafting of ulcers but have occasionally used the venous ligation of Linton.21 He has advocated ligation of the superficial femoral vein and stripping of the long and short saphenous veins if varicosities are demonstrated in them. For this operative procedure the entire leg is prepared as previously described. The saphenofemoral junction is exposed as well as the superficial femoral vein below the profunda vein. Number 8 soft rubber catheters or small pieces of rubber tubing of similar size are placed around the most proximal portion of the long saphenous vein and the superficial femoral vein just below the profunda branch to act as tourniquets. With the aid of a water manometer such as is used to measure spinal fluid pressures, a three way stopcock, saline solution containing 10 mg. of heparin per 100 cc. and an 18 gauge needle, the venous pressure is taken in the superficial femoral vein distal to the occluding catheter. The pressure reading is observed after the more proximal portion of the superficial femoral vein and the long saphenous veins have been temporarily occluded for several minutes with the rubber catheters. If the venous pressure is 30 cm. or more of water as measured from the level of this vein, only the superficial femoral is ligated and interrupted. If the long or short saphenous veins are varicose, their stripping is delayed for two or three months to allow time for venous collaterals to form. Unfortunately this operation and the popliteal vein ligation of Bauer22 do not reduce walking pressure in the veins of the leg,14, 23, 24 and the end results are far short of those obtained following surgery for primary varicosities. Rest, elevation and elastic support are frequently essential adjuncts to surgical therapy in secondary varices.

Summary

The widely different kinds of varices are easy to differentiate if the underlying anatomy and physiology are understood. Appropriate therapy is wholly dependent upon this understanding. Surgery is a reasonably definitive therapy of primary varices, but secondary varices usually require rest, elevation and elastic support.

REFERENCES

8 Wells, H. S., Youmans, J. B., and Miller, D. A., Jr.: Tissue pressure (intracutaneous, subcutaneous and intramuscular) as related to venous