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Editors
M. ALLGÖWER, Basel – S.-E. BERGENTZ, Malmö – R. Y. CALNE, Cambridge (GB) – U. F. GRUBER, Basel

Contributors
M. ALLGÖWER, Basel – C. BURRI, Ulm – W. G. FEGAN, Dublin – H. HENKEMEYER, Ulm – S. ISACSON, Malmö – W. E. MEIER, Zürich – I. M. NILSSON, Malmö – H. H. PÄSSLER, Ulm – Å. SENNING, Zürich – P. WAIBEL, Basel

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P. WAIBEL

Department of Surgery, Division of Vascular Surgery, University of Basle, Basle

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Introduction

In a recent study varicose veins have been found in 30.2% of working men and 33.6% of working women [95]. Other reports found a prevalence of varicose veins in women. This [10] difference could be explained by the fact that less married women with children and varicose veins may be found in an industrial population. On the other hand, for aesthetic reasons more women may seek advice for their cosmetic concern. Varicose veins increase in frequency with age [95]. Simple varicosities without signs of chronic venous insufficiency are seen in 23% of a working population. One third of these

show only spider veins, one third reticular varicosities and one third insufficiency of the long, short or both saphenous trunks. Insufficiency of the ostial valve was recorded in the long saphenous vein in 97, in the small in 13 and in both in 4 cases. These figures correspond well with those of PEARCE [73], LARSON and SMITH [51] and CARROLL [12]. 114 of 1,678 men examined showed signs of chronic venous insufficiency, 55% with pigmentation, 27% with induration also and 18% with ulcers or scars [95]. These figures show that varicose vein disease is common. In 8% of working men there is ostial insufficiency of a main superficial trunk and in 7% of industrial workers there are signs of chronic venous insufficiency.

The aetiology of *simple or primary varicosis* is unknown. By definition, deep veins in the leg in these cases are normal and primary varicosis rarely is followed by chronic venous insufficiency (CVI). Most of the patients begin with unilateral disease first, more often on the left side, bilateral disease following later. The long saphenous vein is affected in most cases, less often the long or even both. In the main trunks valvular insufficiency is common at the saphenofemoral or saphenopopliteal junction or in communicating veins. Primary or idiopathic varicose veins are said to be related to factors such as the upright position of man, standing or sitting during work and heavy weight bearing. These factors, though favourable to varicose vein development, are promoting rather than causative conditions.

The fact that episodes of hormonal activities like menarche, menopause or pregnancy are coincident with the beginning of varicosities has led to the idea that hormonal factors are important in the origin and development of varicose veins. Likewise, use of contraceptive drugs increases the extent of varicosities in some females. Two theories may explain the origin of varicose veins. Weakness of the vein wall with increased distensibility or primary valvular insufficiency is said to be important. As a matter of fact, ZSOTER and CRONIN [99] observed in individuals with varicose veins a significantly higher degree of venous distensibility even in veins of the forearm. SVEJCAR *et al*, [87] proved the collagen content in saphenous veins of such patients to be decreased even in segments without varicosity and GOODRICH and WOOD [34] showed increased venous distensibility during pregnancy or oral contraceptive therapy. However, more arguments are in favour of another theory which, furthermore, would explain the occurrence of both primary and secondary varicose vein disease. The common factor here is an elevation of the venous blood pressure in a low pressure system not prepared for such a charge. This could occur as a consequence of valvular insufficiency of communicating veins, which normally prevent elevated pressures of the deep

vein during muscular strain to be transmitted in the superficial system, or by abnormal function of arteriovenous communications, transmitting arterial pressure to the venous side.

According to LUDERBROOK [56] the valves of the iliac veins protect the ostial saphenous valves and prevent pressure elevations by straining, coughing and Valsalva manoeuvre to be transmitted to the saphenous trunk. Congenital valvular agenesia would, therefore, lead to dilatation of the saphenous trunk and to varicosities. The mechanical theory of venous pressure elevation is supported by the findings of REAGAN and FOLSE [76]. Using a Doppler ultrasonic flowmeter they found iliac valvular insufficiency to be significantly more frequent in children of individuals with varicose veins than in healthy persons. However, COTTON [15] has shown that the weakest part of the vein wall is situated centrally of vein valves and not distally, where varicosities normally occur. If the elevated pressure would act in a retrograde direction, these weakest parts of the vein wall should dilate and not those actually being the site of varicosities. Furthermore, BJORDAL [9] and HOJENSGARD and STÜRUP [42] have shown the influence of coughing and straining to be present in the deep veins of the leg but to be minimal on the pressure in the saphenous trunk.

On the other hand, the fact that varicosities occur peripheral to the valves could support the view that pressure elevations acting in a central direction could be more important. This could be explained by arteriovenous communications transmitting a higher pressure from the arterial side. Inasmuch as varicosities often occur in a reticular form long before insufficiency of the trunk and its valves appears, valvular insufficiency could be a secondary phenomenon and have nothing to do with the primary aetiology of the disease. KING [50] reports RICHARD LOWER to have been the first to describe arterialized veins. Some remarkable observations are in favour of the arteriovenous origin of varicose veins [19, 33, 35, 74, 85, 92]. In individuals with varicose veins the dye injected in arteriography appears earlier than in controls and frank communication between arteries and veins can be seen below the knee [92]. In healthy persons skin temperature on the medial aspect of the leg decreases from the groin to the ankle. In varicose vein disease this normal decrease is interrupted by a peak of temperature elevation just below the knee [35]. This behaviour is found even in the opposite leg without varicose veins in these individuals and according to HAEGER and BERGMAN [35] could only be explained by warmer blood being transmitted to the skin by arteriovenous communications. It should, however, be remembered in this place that 'hot spots' could be explained also by insufficient communi-

cating veins [79]. DE REUS and VINK [19] by operative dissection and GIUS [33], furthermore, by using an operation microscope, found arteriovenous communications in varicose vein disease chiefly in the lower leg, but also in the thigh. SCHROTH [85] recorded not only a significant shorter circulation time in individuals with varicosities, but also an elevated peripheral vein pressure in prone position and most important a significant elevation of the O_2-saturation in the saphenous trunk.

KING [50] explains the abnormal function of arteriovenous communications by hormonal influence especially in pregnancy. That the appearance of varicose veins in pregnancy is at least partially not due to local pressure on caval and iliac veins has been proved by FRIED *et al.* [31] and GOODRICH and WOOD [34], though IKARD and FOLSE [44] have recorded by Doppler measurements a compression effect in later pregnancy during standing position. Finally TANYOL [89] has drawn attention to the high incidence of varicosis in patients with portal cirrhosis. He has advanced the thesis that alcohol would be a possible factor by an angiotoxic effect, but he also considers hyperestrogenism in portal cirrhosis with increased cardiac output and appearance of arteriovenous connections a possible cause of varicosis.

Symptomatic or secondary varicosis with chronic venous insufficiency shows the same pattern of distribution as idiopathic varicose vein disease. It differs in complications and aetiology. Contrary to the primary form it often leads to oedema, pigmentation, subcutaneous induration and ankle ulcer. Secondary varicosis is a complication of deep vein thrombosis, malformation or agenesis of deep veins or their valves or secondary to congenital or acquired arteriovenous fistula. It is often combined with *chronic venous insufficiency (CVI)* which can be defined as the consequence of disproportion between arterial inflow and venous outflow from the diseased leg. The most important factor in post-thrombotic varicosis is insufficiency of the communicating veins of the leg leading to some venous hypertension during walking in the superficial veins normally protected by sufficient valves [94]. By post-thrombotic damage of the valves or by increased deep vein pressure following deep venous occlusion, valvular insufficiency occurs and the superficial venous network acts as a collateral circulation. Unsupported outside the fascial compartment superficial veins dilate, secondary superficial vein valve insufficiency occurs, the pressure in these veins during walking does not decrease and insufficiency of the collateral network ensues. At that time the saphenous system no longer acts as an efficient venous outflow but by reversal of flow increases venous insufficiency. Oedema appears first in the evening, later the whole day and finally it persists leading

to subcutaneous induration, scarring, and ulcer formation. Diapedesis of erythrocytes and transformation of haemoglobin is responsible for the typical brownish pigmentation aspect of the leg with venous insufficiency. The most important factor leading to secondary varicosis, chronic venous insufficiency and ulcer is deep vein thrombosis. BAUER [8] followed 41 patients with deep vein thrombosis and saw ankle skin induration in 75% and ulcers in two out of three within 10 years. Phlebography demonstrated sequelae of deep vein thrombosis in 23 out of 25 extremities examined. According to LINTON and HARDY [54] chronic venous insufficiency after deep leg vein thrombosis appears in 74% within 10 and in 90% within 20 years. The figures throughout the literature are similar. CRANLEY *et al.* [16], however, considers 30% of ulcers as not being post-thrombotic though he did not prove it by phlebography and considered only the history of the patient. The same was true for HALLIDAY [36]. According to HJELMSTEDT and BERGVALL [40] and KAKKAR *et al.* [46] only 1 out of 4 thromboses of the deep leg veins found by phlebography or ^{125}I-fibrinogen was symptomatic. Faced with these facts the figures of BAUER [8] and LINTON and HARDY [54] seem to be important.

Clinical Significance of Varicosis

Symptoms in primary varicosis are the same recorded in women without detectable varicose veins especially in summer and the days before menses start. The complaints do not differ much from those in chronic venous insufficiency and include heaviness, restless legs, leg fatigue in the evening, swelling in the evening and even some strange pain (table I). On the contrary, there are patients with extensive varicosities and trunk insufficiency without any complaints. This is true more often for men. In females, symptoms are more marked during the days preceding menses. However, symptoms are rarely severe and more people seek advice for cosmetic reasons or because of concern about the future of the leg than for real actual trouble. It is not surprising, therefore, that in many patients slight symptoms persist even after radical surgery. They may disappear, however, after additional efforts such as increased physical activities by walking and swimming and after taking the habit of leg elevation in the early afternoon. The complaints in primary varicosis seem more to be due to the cause of the disease than to the varicosity itself. This causative factor, however, is unknown and is not affected by elimination of venous trunks.

Table I. Sclerotherapy preceding operation and symptoms of primary varicosis, and chronic venous insufficiency with and without ulceration

	Primary varicosis (n = 100)	Chronic venous insufficiency	
		without ulceration (n = 100)	with ulceration (n = 50)
Sclerotherapy in history	26	71	43
Thrombosis in history	0	31	16
Fatigue in the evening	53	74	50
Restless legs	59	34	15
Oedema reversible	29	51	7
Oedema irreversible	0	21	43
Pain in the evening	41	49	21
Pain the whole day	1	24	24

Symptoms in secondary varicosis with chronic venous insufficiency are frequent and more pronounced. Pain appears erlier in the day and heaviness is often troublesome. Patients more often seek advice because of troubles and less often for merely cosmetic reasons. After operation symptoms seem to improve. However, it is not quite clear if the improvement is due to the operation itself. Many authors admit that supporting measures such as leg elevation and compression bandages or stockings are important in post-operative management. Most publications do not differentiate between improvement due to operation and due to upportive measures. It is also important to remember that patients are more closely followed after operations than after conservative treatment alone.

Patients with varicose veins, especially males and older individuals seek advice more often because of concern about the future of their legs and about possible complications. The incidence of varicose vein complications is difficult to estimate, because in most cases natural history of the disease is modified by therapy and, therefore, unknown. However, complications seem to be a rare event. Progression to chronic venous insufficiency and ankle ulcers is a rule in secondary, but an exception in primary varicosis. In the latter, chronic venous insufficiency may occur secondarily after deep vein thrombosis because patients with varicose veins in the younger age group have been shown to be more prone to postoperative and probably also postpartal vein thrombosis. KAKKAR *et al.* [47] observed in young individuals with varicosis an incidence of postoperative deep vein thrombosis of

56.6 % compared with 19.2 % in a group without this risk factor. However, nothing has been said about the extent of varicosis. Therefore, we still do not know at what time of the natural history of varicosity and with which extent of varicosity the risk starts. In the older age group there is no higher risk any more. If the risk of thrombosis is eliminated by removal of all varicose veins is not known. It has been shown that compression stockings which practically eliminate the effect of varicosis and lead to increased deep vein flow [80, 81] are not effective in preventing deep vein thrombosis. Furthermore, we do not know if the cause or the existence of varicosis is the true risk factor in these patients.

Other complications such as bleeding or varicophlebitis are extremely rare events. In the history of our patients bleeding occurred in 0.5 % just as in the patients of LOFGREN *et al.* [55]. The incidence of varicophlebitis is somewhat higher, 9 % in LOFGREN'S report and 3 % in our cases. It is more often seen in extensive varicosities. Superficial varicophlebitis is painful, but otherwise without great clinical significance. It rarely if ever leads to deep vein thrombosis. Lung embolism has been reported to occur after superficial phlebitis [97, 98], but the therapy of bed rest in these cases is probably the real cause of the complication. In no case of superficial phlebitis bed rest should be chosen as the treatment. Compression bandage and ambulation is to be preferred. Furthermore, GALLOWAY *et al.* [32] questioned superficial phlebitis to be the source of lung embolism. In their cases they could show the source to be iliac vein thrombosis without connection with superficial inflammation.

Most of the younger patients with varicose veins come to treatment for aesthetic reasons. This is especially true for women and the reason for the prevalence of female patients. In these patients it is extremely important to avoid any measures in treatment which could affect the function of normal deep veins or worsen the aesthetic problem of the patient. Any complication in these patients must be considered a disaster.

Clinical significance of secondary varicosis is due to the chronic venous insufficiency present and its complications. Treatment is not based on cosmetic considerations but aimed to improve work capacity and welfare of the patient. It is known that patients with CVI have recurrent bouts of deep vein thrombosis. By asymptomatic deep vein thrombosis ambulatory silent pulmonary embolism can occur and lead with time to pulmonary hypertension. This should always been kept in mind in these patients. Moreover, chronic venous insufficiency tends to progress to induration of the skin in the ankle area and finally lead to frank ulceration. Social consequences are

important by recurrent need for treatment of ulceration and dermatitis. Therefore treatment in these patients is, unlike that in primary varicosis, a surgical and medical problem and not a matter of cosmetic considerations. Since treatment in these cases is often difficult, prevention of deep vein thrombosis is the most important task.

Haemodynamics of Varicose Veins

In healthy individuals venous blood from the superficial system through communicating veins and finally the saphenofemoral or saphenopopliteal junction empties into the deep vein system. Backflow is prevented by segmental valves in the main stem and in the communications. The vein valves guarantee the physiological flow direction and protect the superficial vein system from pressure bouts during straining, cough and other situations which elevate the venous pressure. In the upright position the pressure in the superficial veins corresponds nearly to the hydrostatic pressure. It amounts to about 97 cm H_2O. During muscular exercise and walking the pressure drops to about 28 cm H_2O. After walking, the original pressure is restored after about 30 sec. Pressure in the still upright position is nearly constant. Coughing and straining have no effect.

Pressure in the popliteal vein in the still upright position is somewhat less (83 cm H_2O). During muscular exercise it does not fall and during straining and coughing the pressure is elevated. Pressure in the posterior tibial vein is about 112 cm H_2O and decreases during walking [39, 42, 75].

In primary varicosis during rest in the upright position the pressures in the superficial system are not modified. During walking, the decrease is less – half the normal decrease – and resting pressures after walking are restored after 2–4 sec. Compression of the insufficient long saphenous vein or compression stockings leads to normal behaviour of the pressure during walking [93, 94]. Resting superficial venous pressure is even normal in chronic venous insufficiency with secondary varicosis. During walking, however, the pressure drop is minimal, absent or even the pressure increases. Compression of the saphenous vein has no effect on the walking pressure. Resting pressure is restored immediately after walking. In the popliteal vein the pressure during walking may increase [94]. In varicosis, electromagnetic flowmeter studies have shown that during rest in the upright position there is no detectable flow in the long saphenous vein. During coughing, straining, laughing and even speaking there are some more or less important bouts of

retrograde flow. During walking there is retrograde flow in the long saphenous vein, which is the higher, the lower the pressure is and vice versa. Retrograde flow is abolished in varicosis by compression of the long saphenous vein in the thigh. Insufficient communicating veins show no detectable flow during rest; during walking, however, the flow is bidirectional. These findings of BJORDAL [9] support the clinical experience during the Trendelenburg-Brodie test. Finally, in varicosis MAATZ [58] found more often retrograde than anterograde flow in the superficial veins.

The most important haemodynamic deficiency in primary and secondary varicosis is insufficient decrease in superficial vein pressure, immediate return of resting pressures after walking and retrograde flow in the long saphenous vein during exercise. In primary varicosis the pressure abnormality can be abolished by compression of the long saphenous vein or by operation [93, 94]. In secondary varicosis compression or operation have no effect and popliteal vein pressure is elevated during walking [94].

Pre-operative Examination of the Varicose Vein Patient

The discrimination of varicose veins in three types is useful for practical reasons. *Spider veins* are frequent among women and above all of cosmetic concern. If this form has something to do with varicose vein disease at all is not proved. Prospective epidemiologic studies have to clear this problem. Another third of individuals or 7% of a population shows *reticular varicosities*. In these patients cosmetic considerations are also of primary importance. The long and short saphenous vein in these cases does not show any valvular insufficiency, however, there may be valvular insufficiency of communicating veins. Most often the anterolateral branch of the long saphenous trunk is involved. Finally 7% of a population belongs to the group with *valvular insufficiency* of the long and or short saphenous vein. It is this kind of varicosis which is of surgical importance. *Insufficiency of communicating veins* is also common here.

More important is the discrimination between primary and secondary varicosis (table II). Accurate diagnosis may be possible by questioning the patient, more often by clinical examination of the leg and sometimes only by phlebography. Pressure measurements may be useful in difficult cases. The history of deep vein thrombosis or pulmonary embolism is a good argument for the existence of a symptomatic form. However, it has to be kept in mind that one third of patients negate such a history and that in one

Table II. Discrimination between primary and secondary varicosis

	Primary varicosis	Secondary varicosis
History		
Phlebothrombosis	−	++
Embolism	−	++
Operations, childbirth, trauma	+	++
Early varicosis	+	++
Arteriovenous injury	−	++
Symptoms	+	++
Examination		
Signs of CVI	+	++
Phlebography	−	++
Peripheral venous pressure	−	++

third of cases only thrombosis proved by ^{125}I-fibrinogen is found by clinical examination [36, 46, 80].

Deep vein thrombosis is common after operations, childbirth and trauma and, considering the difficulty of detecting the existence of thrombosis by clinical means, suspicion must arise if the history of a patient reveals such promoting factors. Trauma may be responsible also for secondary varicosis following arteriovenous fistula, especially if history shows an injury in a part of the body where arteries and veins may have been hit. Congenital arteriovenous fistula and those acquired before bone growth ceases show increased growth of the extremity involved. Ankle oedema occurring every day in the evening may indicate a secondary form of varicosis. However, it has to be remembered that oedema also occurs in primary forms and even in individuals without varicosis. This is especially true for women.

More important than history is inspection of the leg. As a rule signs of chronic venous insufficiency with one or more of the following symptoms speak in favour of symptomatic varicosis: dilated small veins below the inner malleolus, pigmentation in the surroundings, induration of the skin in this malleolar area, oedema, scars, dermatitis or even frank ulceration (table II). Auscultation of arteries and veins may reveal a bruit indicative of arteriovenous communication and hence symptomatic varicosis. Increased growth of the extremity likewise is indicative of a secondary form.

Discrimination by phlebography is crucial and should be considered in every case of questionable symptomatic varicosis. There may be occlusion,

recanalization of deep veins, rigidity of the vein walls or signs of collateral pathways. Agenesis of valves or even parts of the deep vein system may occasionally be found. By retrograde phlebography extensive backflow of dye may be seen indicative of congenital or more often acquired valvular insufficiency of deep veins. If the existence of arteriovenous fistula is suspected arteriography is decisive. The site of the fistula can be detected and marked for correction.

Pressure measurements in the veins of the foot with the same cannula used for phlebography shows lacking pressure drop during walking and immediate return to resting values thereafter contrary to the behaviour in primary varicosis where compression of the saphenous vein is, moreover, followed by normal pressure drop. Valvular insufficiency thereafter has to be detected by clinical examination. The Brodie-Trendelenburg test is useful for this purpose, demonstrating the existence of retrograde flow in superficial veins. This examination can be improved by means of a Doppler flow detector. Its use is necessary only in questionable cases. It is useful to check first the existence of communicating vein insufficiency by having the patient elevate the leg, apply a pressure bandage to the thigh and examine the leg in upright position. Immediate filling of superficial veins below the pressure bandage within 30 sec is indicative of communicating vein insufficiency. If only one valvular insufficiency is present the leaking point can be marked. By removing the pressure bandage retrograde filling of the main trunk is indicative of ostial valvular insufficiency. In most cases further communicating vein insufficiency must be detected by segmental compression of the leg in a distal direction. The leaking points are marked (fig. 1).

Insufficient communicating veins may also be marked by palpating the leg and looking for fascial defects. However, this is difficult in oedema and in obese individuals. Moreover, fascial defects alone have been shown to be indicative of communicating vein insufficiency in only 16.8% of cases. The accuracy of the examination can be improved by thermography [72]. The patient is examined in a cool room. Thermography then shows 'hot spots' with temperature elevation of about 2°C compared with the surrounding skin. 'Hot spots' alone are indicative of valvular insufficiency in 34.8% of cases and if combined with fascial defect detection in 51.5% of cases [79]. Most revealing however is again phlebography, showing insufficient communicating veins by a blow out of superficial veins (fig. 2). In oedema or obese patients the Doppler ultrasound method is able to detect insufficient communications [29, 84]. Finally, in cases of severe induration, oedema and ulceration operative preparation of communicating veins by wide exposition is the