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THE VENOUS RETURN FROM THE LOWER LEG IN HEALTH AND IN CHRONIC VENOUS INSUFFICIENCY

A Synthesis

by

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Contents

The venous return from the lower extremity, against the hydrostatic forces, still presents a number of problems awaiting their solution. We cannot truthfully say that the mechanism of the healthy venous pump is wholely understood, and it is evident to any student of venous pathology that the difficulties increase, when it comes to the evaluation of the venous pump in patients with chronic venous insufficiency.

During the last few decades some important investigations have, however, been published, which throw some light on the venous circulation in the lower extremity in health as well as where the venous return is impaired. These reports appear scattered and often unrelated in the literature.

The *first object* of the present paper will therefore be to collect and review some of the pertinent reports, which have a bearing on the understanding of the conditions for the venous return.

The function of the venous pump in the lower leg has been studied phlebographically by the author by means of a standardized functional method (dynamic intraosseous phlebography (Arnoldi & Bauer 1960 a, 1960 b, Bauer & Arnoldi 1960) and an analysis of the findings was published by Arnoldi (1961 a, 1961 b). The *second object* of the present study will be to compare the phlebographic findings — and the resulting conception of the venous pump — with the information found in the literature regarding the physiological conditions for the venous return, in the hope that such a synthesis may increase our knowledge and understanding of the problems of chronic venous insufficiency.

Sound therapeutic principles must be based upon a definite conception of pathology. This is as true in venous insufficiency as in other branches of medicine. The author's conception of the pathology of chronic venous insufficiency is the basis for the *third object* of this

paper, namely a discussion of the therapeutic consequences of these ideas.

As is so often the case in medicine, the present analysis gives rise to more problems than it set out to solve and the *fourth object* has thus been to show some of the unsolved questions in need of further investigation.

PART I.
OBSERVATIONS ON ANATOMIC STRUCTURES AND PHYSIOLOGICAL CONDITIONS IN THE LOWER LEG.

CHAPTER I.
THE VEINS OF THE LOWER EXTREMITY.

When considering the veins of the lower extremity it is practical to distinguish between three systems: the subcutaneous, the subfascial and the communicating veins.

The subcutaneous veins: The saphenous veins and their tributaries drain most of the skin and subcutis of the lower extremity and part of the abdominal wall. They are as a rule found near the deep fascia, but in adipose extremities the veins lose their close relation to the fascia and are found embedded in loose fatty tissues.

It is generally assumed that about ten per cent of the venous return from the lower extremity take place through the subcutaneous veins. The blood-flow through the saphenous system is partly upwards, partly central through the communicating veins into the deep veins. The subcutaneous veins are equipped with valves, which under normal conditions prevent a distal flow of blood. The number of valves vary within wide limits.

The skin and subcutaneous tissues in the malleolar regions are as a rule drained by the tributaries of the ankle-perforating veins, which may or may not anastomose with the saphenous trunks. Where the ankle-perforating veins form a separate drainage system it may be regarded as a "third saphenous system" *(Arnoldi* 1958).

The subfascial veins: The deep veins of the lower extremity drain the structures under the deep fascia. The numerous tributaries from bones, tendons and muscles drain into a system of deep venous trunks. In the lower leg each musculo-fascial compartment contains a set of deep trunks, placed as vv. comites alongside the arteries. *(Fig. 1)*. The anterior and posterior tibial and the peroneal veins coalesce in the upper part of the leg to form the popliteal vein which at a somewhat higher level is joined by the gastrocnemial veins.

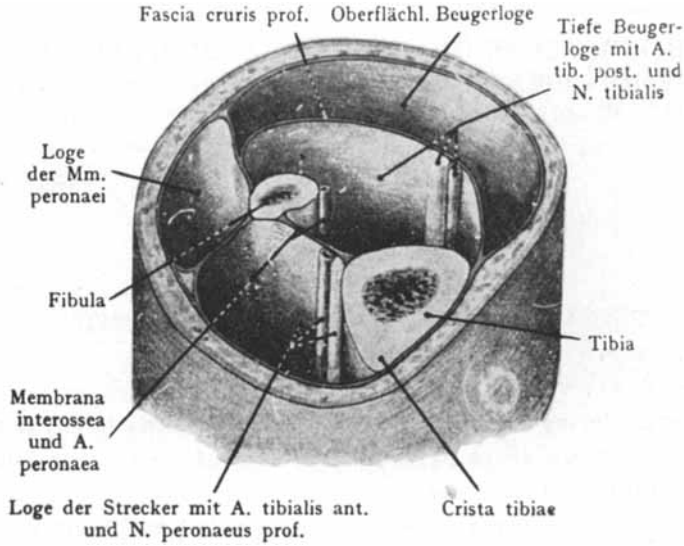

Fig. 1. (From Corning: Lehrb. top. Anatomie). Schematic cross-section through the lower leg. The four musculo-fascial compartments are shown, together with nerves and arteries. The deep veins are placed as vv. comites close to the arteries.

The central stem continues upwards in the adductor canal as the superficial femoral vein and is joined by the deep femoral vein in the upper part of the thigh. The common femoral vein continues above the inguinal ligament as the external iliac vein. The deep femoral vein drains a set of musculo-fascial compartments in the thigh.

The relations of the deep veins will be considered in Chapter II.

The communicating veins: It is customary to distinguish between two types of communicating veins: indirect and direct. The indirect communicating veins are small, but very numerous and are called indirect as they pass from a subcutaneous vein to a muscular vein in the leg or thigh. The direct communicating veins are fewer and relatively constant in their position. They are somewhat larger than the indirect communicating veins, and they connect the subcutaneous veins directly with the deep trunks.

The most important groups of direct communicating veins are fairly constant in their position. On the thigh they connect the internal saphenous vein or its tributaries with the femoral vein in Hunter's canal. In the lower leg a medial group is found near a line connecting

a point on the medial border of the tibia at the level of the middle of this bone with a point below and slightly posterior to the internal malleolus. A lateral group is generally found in front of the lateral border of the Achilles tendon. The communicating veins in the lower two thirds of the leg are generally called ankle-perforating veins.

While the anatomy of the communicating veins of the lower leg has been known for more than a hundred years, it is especially through the work of Cockett (1953) that the importance of the medial and lateral ankle-perforating veins in the pathology of the venous leg ulcer has been established.

The communicating veins are equipped with valves at the entrance into the deep veins, which under normal conditions only allow a central flow from the subcutaneous into the deep veins.

CHAPTER II.
THE MUSCULO-VENOUS RELATIONS IN THE LEG AND LOWER THIGH.

The musculo-venous pump with which we are concerned at present consists of the popliteal vein, the anterior and posterior tibial veins and the peronal vein together with the muscular tributaries which emerge into these veins. The muscles of the leg which are drained by these vessels are arranged in three compartments (Fig. 1); the anterior, which contains the tibialis anterior, extensor hallucis longus and the extensor digitorum longus. These muscles are drained by the anterior tibial veins; the lateral compartment, containing the peroneus longus and brevis muscles, which are drained by the peroneal veins. Finally, the posterior compartment, which lodges the tibialis posterior, flexor digitorum longus, flexor hallucis longus and the soleus muscles, is drained by the posterior tibial veins. These compartments are invested by very strong sheaths of the fascia cruris (Fig. 1).

The gastrocnemius muscle is placed behind the soleus in a separate fascial sheath, less well developed than those enveloping the other muscles of the leg. The gastrocnemial bellies are drained by a separate pair of veins, which join the popliteal vein above the confluence formed by the veins from the other three compartments.

In the upright position the vis-a-tergo is not sufficient to push the blood upwards fast enough to keep the venous outflow equal to the arterial inflow. The additional forces necessary are supplied by the action of the surrounding tissues upon veins with competent valves.

The surrounding tissues may act upon the veins in two principal ways:

At rest a "passive support" will restrict the inherent tendency of the veins to dilate under pressure. The passive support offered by the tissues is equal to the tissue tension, which varies from tissue to tissue and at different levels of the same organ (Chapter 4). Other things equal, a widening of the diameter of the total venous bed would cause a slowing down of the proximal flow, while a constriction would have the opposite effect. Even at complete rest the veins of the lower leg are supported by a positive pressure from without, and the strength of this pressure is determined by the tonus of the surrounding tissue.

During muscular activity the pressure on the veins increases enormously (Chapter 4). Under normal conditions the pressure increases and decreases rhythmically. During contraction the blood in the segment of vein under increased pressure will be displaced, and the presence of competent valves makes only the proximal route possible.

The relations of the veins of the lower extremity change very much. Bones, tendons, fasciae, muscles and fatty tissue have different tissue tension, and consequently the character of the passive support may vary considerably at different levels. Muscular activity may act very forcibly at places where a close contact is established between muscle and vein, and may be entirely absent a few inches away, where the vein may be related to bone or fat.

The author has been unable to find any references in the literature dealing with the influence of different environment upon the venous flow in the lower extremity, and no hints of a possible difference of function between different segments of the deep venous trunks. A perusal of four major textboks of physiology has given a uniform impression of the conception of the structure of the musculo-venous pump mechanism, which may be illustrated as done in *Fig. 2*.

The deep crural veins and the popliteal vein show a striking difference in muscular and fascial relations. While the tibial and peroneal veins lie deeply embedded among strong muscles, ensheathed by the septa of the crural fascia (Fig. 1), the popliteal vein looses its close relation to muscles and fascia as soon as it emerges from the soleus. Until it reaches the lower exit of the adductor canal it lies surrounded

by loose fatty tissue behind the popliteal plane of the femur *(Fig. 3)*. Of the strong fascial planes, so prominent in the leg, only a fine layer of connective tissue remains in the poples, easily torn by the dissecting finger during operation.

Thus for a distance of 6—10 cms no muscular force is found working directly upon the vein wall.

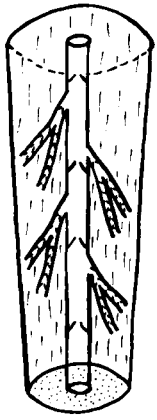

Fig. 2. A conception of the structure of the musculo-venous pump in the lower extremity, based upon the description prevalent in modern textbooks of physiology. Variations in musculo-venous relations are not considered. (Compare Fig. 26).

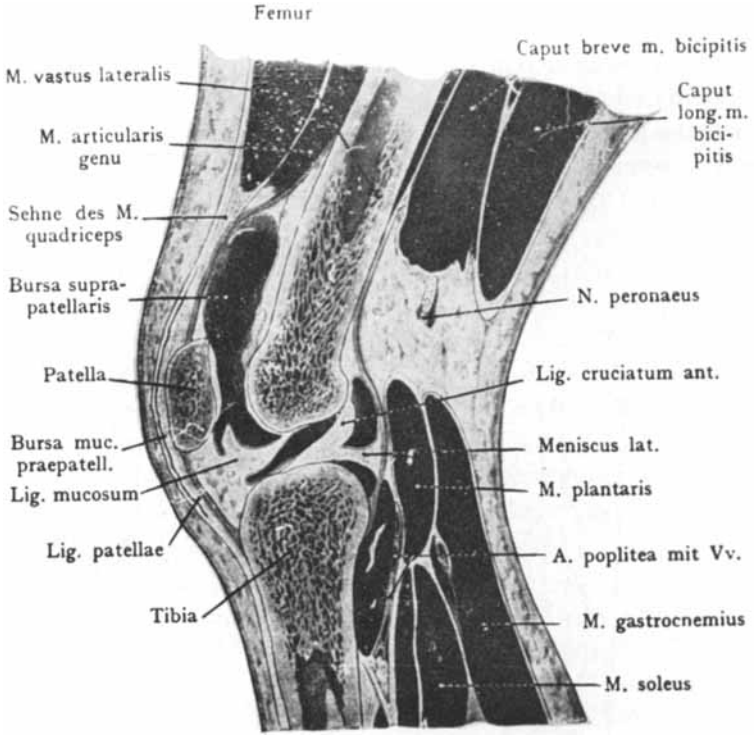

Fig. 3. (From Corning: Lehrb. top. Anatomie). Sagittal view of the knee and the popliteal region.

CHAPTER III.
THE VALVES OF THE DEEP VEINS.

The distribution of valves in the deep veins of the lower extremity presents another important difference between the crural and the popliteal (and femoral) segments. This subject has been studied in recent years by *Eger & Caspar* (1943), *Powell & Lynn* (1951), *Basmajian* (1952) and *Cockett* (1953).

While the deep crural veins show an abundance of closely spaced valves, the valves in the large trunks of the poples and thigh are much

fewer. The actual number of valves may vary considerably, but never attain the number found in equal segments of intramuscular veins.

The following analysis of the distribution of valves in the popliteal, femoral and external iliac veins is taken from *Cockett* (1953): *(Fig. 4)*.

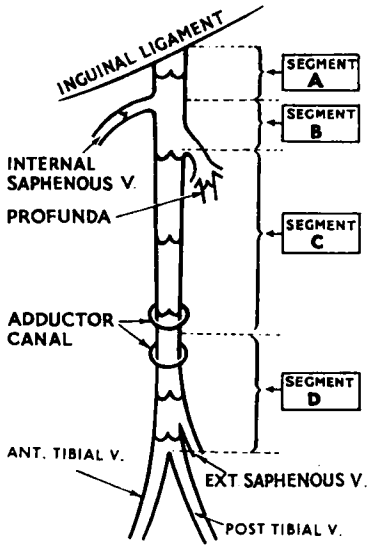

Fig. 4. (From Dodd and Cockett: Pathology and Surgery of the veins of the lower Limb). Compare analysis in text.

Segment A: Sixteen out of twenty-two cases had one valve in this segment, the rest were valveless.

Segment B: All cases valveless.

Segment C: Eight cases had four or more valves.
Twelve cases had three valves.
Three cases had two valves.
One case had one valve.

Segment D: Four cases had three or more valves.
Eight cases had two valves.
Eleven cases had one valve.

The greatest number of valves seen in the entire length was nine; the average number was five. In one case, only two valves were present in the whole vein from the inguinal ligament to the popliteal fossa. The most constant valve was that below the point where the profunda femoris vein joins the superficial femoral vein to become the common femoral vein.

That a complete congenital absence of valves in the entire deep system may occasionally occur was shown by *Lodin, Lindvall & Gentele* (1961) and *Lindvall & Lodin* (1961). These findings were based on phlebographic examinations.

The profunda femoris vein shows closely spaced valves, is intermuscular in its relations and emerges into a sparsely valved "collecting vein", the femoral. Thus, in these respects it is closely similar to the deep crural veins.

It is an often mentioned fact that intramuscular veins — with the possible exception of the soleus sinuses (*Dodd & Cockett* 1956) — are closely valved, and that the number of valves decreases sharply as soon as the intimate relation to muscle is lost. This is true in the upper as well as in the lower extremity and is found in animals as well as in man.

In this connection the findings by *Williams* (1954) who dissected the fore-limbs and hind-limbs of cats, dogs and monkeys, are interesting. The monkey (resus macaque) not infrequently adopts the upright position and thus the conditions for the venous return in the lower leg may in these periods be considered similar to those met with in man.

Williams found, that valves were much less common in the hind-limb of the monkey, than in the dog and cat. In all three species valves were about equally distributed in fore and hind-limbs. This was a reasonable expectation in the dog and cat, but if the valves are to be considered an antigravity mechanism they might be expected to be more numerous in the hind-limb of the monkey than in the fore-limb.

Williams is led to support *Jäger's* (1936) theory, "that the function of the valves is to protect the capillaries and venules from sudden excessive rises of blood pressure during muscular exercise".

At this point we may conclude from Chapter II and III, that the anatomic relations of the deep crural and the popliteal veins are fundamentally different, and that the valvular equipment in these segments show marked quantitative differences.

CHAPTER IV.
SUBCUTANEOUS AND INTRAMUSCULAR TISSUE TENSIONS.

The cells and the intercellular structures are surrounded by the interstitial extracellular fluid, which may be imagined as an anastomosing network of very fine channels. The hydrostatic tension of the interstitial fluid can be measured by means of simple manometric methods. In the literature several investigations are recorded, in which the tissue tensions have been estimated by indirect methods. The results of these investigations vary rather widely. I have preferred only to report the outcome of experiments in which direct measurements have been performed.

The concept of the interstitial spaces as a continuous anastomosing network of minute channels admits the possibility of pressure gradients in this system. *Burch & Sodeman* (1937) define the tissue pressure as the pressure with which the tissue structures resist any changes in their anatomical relations.

a) The subcutaneous tissue pressure.

The first to perform direct measurements of the tissue tensions was *Landerer* (1884). He found a subcutaneous pressure in animals (rabbits and dogs) varying between 25 and 65 mm H_2O. Only one experiment was performed on man. The subcutaneous pressure in the thigh, measured in the upright sitting position was found to be 550 mm H_2O.

Landerer found that the subcutaneous tissue pressure in rabbits and dogs varied proportionally with the arterial blood pressure. This correlation has not been investigated by later authors. The highest tissue pressure measured by Landerer (700 mm H_2O) was found in the Achilles tendon of a rabbit.

Meyer & Holland (1932) determined the intracutaneous and subcutaneous tissue tension in normal subjects by means of a direct manometrical method, using a fine cannula connected to a manometer. They found the intracutaneous pressure to fluctuate about a mean

value of 70 mm H_2O. The intracutaneous tension was found to be unrelated to the distance from the heart, and it was not affected by changes in capillary pressure.

The same authors found a mean value of subcutaneous pressure on the fore-arm of 30 mm H_2O (40—20 mm H_2O). This tension was not affected by changes in capillary pressure or by variations in the distance from the heart level.

Burch & Sodeman (1937 a, 1937 b) using a similar method, found slight variations in the subcutaneous pressure at heart level in different regions. Thus, in normal individuals the mean value at the dorsum of the hand was 17.9 mm H_2O, in the pretibial area 37.1 mm H_2O. In the standing position the subcutaneous pressure at the dorsum of the foot increased from a mean value of 30.8 mm H_2O to 80.5 mm H_2O.

The same authors determined the subcutaneous pressure in the pretibial area in patients with congestive heart failure, during the period when oedema was increasing. The pressure varied between 58 and 267 mm H_2O, compared to a normal mean value of 37.1 mm H_2O.

Burch & Sodeman (1937) agree with *Meyer & Holland* (1932) that increase in venous blood pressure over short intervals of time has only a very slight effect on the subcutaneous tissue pressure. *Holland & Meyer* (1932) found, however, an unaltered subcutaneous pressure in patients with cardiac oedema, compared with normal values. They do not state whether the oedema was increasing during the time of investigation.

Wells, Youmans & Miller (1938) measured the subcutaneous pressure in normal subjects at rest and in the upright position, as well as in patients with slight oedema, and in arms and legs of normal individuals following prolonged venous congestion produced by inflation of a blood pressure cuff to pressures from 50 to 100 cms H_2O, and found that nearly half the values ranged from 20 to 60 mm H_2O (Fig. 5). The subcutaneous pressure was found to rise somewhat, but not much and not regularly after prolonged congestion (Fig. 5). The rise was usually not more than 10 to 30 mm H_2O. Values of 110 or 150 mm H_2O were exceptional and were measured in the subcutis of the lower leg after nearly three hours of quiet standing.

Thus the various authors agree that the pressure in the subcutis ranges between 10 and 70 mm H_2O in normal subjects. There are indications that prolonged venous congestion may raise the pressure moderately, and that distension of the subcutis by a developing oedema may have the same effect. It seems as if the subcutaneous pressure in

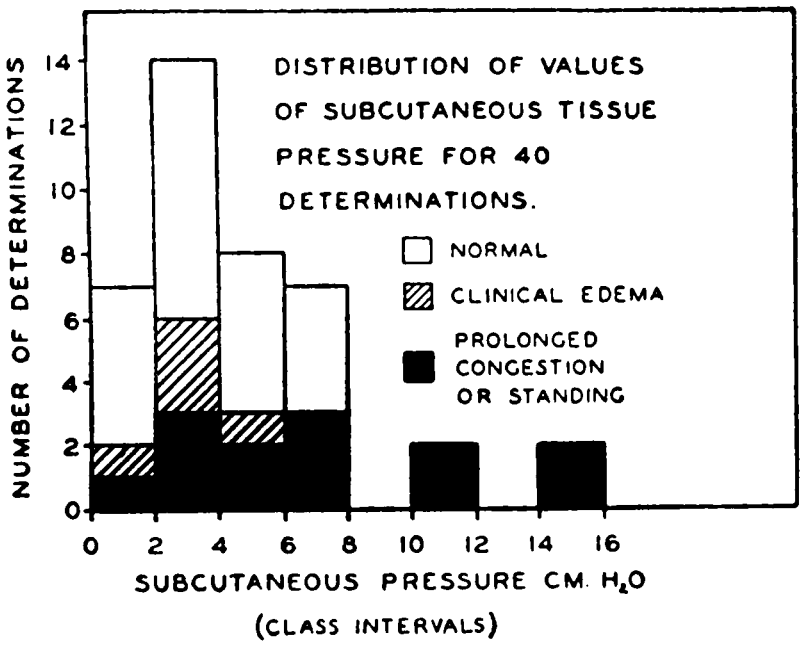

Fig. 5. Histogram showing distribution of values of subcutaneous pressure. (From Wells, Youmans and Miller, 1938).

normal individuals is independent of the venous pressure within wide limits.

It would be interesting to compare the subcutaneous pressure in normal subjects and in patients with chronic venous insufficiency. The indurative changes which are seen in the severe forms of venous disturbances would probably affect the tension in the subcutis. Such measurements have not been published as yet.

b) The intramuscular tissue pressure.

Henderson, Oughterson, Greenberg & Searle (1935) made some interesting studies of the intramuscular pressure in the relaxed biceps brachii in normal individuals and in patients confined to bed. They used a direct manometrical method very much like all the other authors mentioned in this chapter. In healthy young men the average value was 74 mm H_2O, while the average value in patients in bed was found to be 47 mm H_2O. A significant decrease in intramuscular pressure was found to take place after operation, especially after major

abdominal surgery performed under general anaestesia. Thus the intramuscular pressure measured twelve hours before operation averaged 73 mm H_2O, while the average value after operation was 48 mm H_2O.

Increase in intramuscular tension in the relaxed biceps brachii was found after administration of strychnine sulfate and after inhalation of CO_2.

The same authors determined the pressure in the gastrocnemius muscle in one patient standing at rest to 143 mm H_2O. Maximal contraction of the gastrocnemius (standing on one toe) increased the pressue to 312 mm H_2O.

Wells, Youmans & Miller (1937) showed in a series of experiments that in the normal relaxed subject in the recumbent posture, the intramuscular pressure values varied in different individuals and in the same person on different days from 20 to 110 mm H_2O. Values below 50 mm H_2O were found in most studies on muscles such as the biceps brachii, and the gastrocnemius, which have a thin or loose fascial covering. The higher values, usually from 50 to 100 mm H_2O were found in measurements on the anterior tibial and soleus, which are invested by a tight fascial sheath.

The same authors found that intramuscular pressure responded immediately to changes in venous pressure in the soleus and anterior tibial muscles, while the pressure changes in the gastrocnemius were minimal.

The authors conclude that intramuscular pressure in the relaxed subject appears to be determined by a variety of factors of which the most important are the tightness of the overlying fascia, the amount of extravascular fluid present in the muscle, and the degree of filling of its blood vessels.

The maximal values of intramuscular pressure during voluntary contractions were measured by the same authors. Here again the muscles with a tight fascial covering showed values of contraction pressure, which were much higher than those found in muscles with a poorly developed fascia (Fig. 6). It is of interest to note, that the highest values were obtained from the soleus and anterior tibial muscles, which form part of the section of the venous pump, with which this paper is concerned, and that the highest values were obtained in the region, where the ankle-perforating veins join the posterior tibial veins.

Measurements of intramuscular pressure have only been performed on normal subjects. It would be interesting to see, if patients with

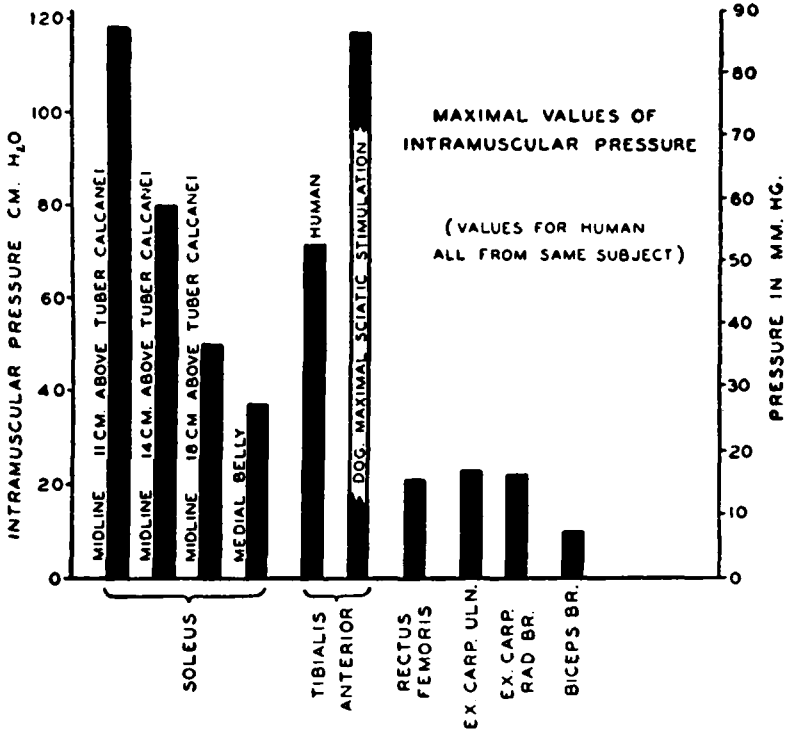

Fig. 6. Maximal values of intramuscular pressure during voluntary contractions in man and during maximal sciatic stimulation in the dog. (From Wells, Youmans and Miller, 1938).

chronic venous insufficiency show changes in intramuscular pressure at rest and during maximal muscular contraction. This is especially true in the deep venous incompetence of the phlebographic Group III (Chapter XI). As it is, we do not know for certain, whether the phlebosclerotic changes in the veins affect the pressure inside the fascial compartment, or if changes in muscular pressure occur, which might have a deleterious effect on the venous pump.

A comparison between the subcutaneous and intramuscular pressures in the lower leg in normal subjects shows, that while the subcutaneous pressure at rest ranges between 10 and 70 mm H_2O, the intramuscular pressure is always somewhat higher, from 50 to 110 mm H_2O, in the muscles belonging to the section of the venous pump with which we are concerned.

The intramuscular pressure was found to rise with the venous pressure, while the subcutaneous pressure was only slightly affected by changes in pressure in the adjacent veins.

Finally, contraction of the muscles of the section of the venous pump under discussion raises the intramuscular pressure to levels many times higher than the highest values obtained from the subcutis.

CHAPTER V.
VENOUS PRESSURE MEASUREMENTS.

Investigations on the pressure in the veins of the lower extremity at rest and under dynamic conditions have been of the greatest value for our understanding of venous physiology and pathophysiology. It is not the intention here to review the extensive literature on venous pressure measurements, but only to refer to the results of these investigations in so far as they seem pertinent to the present study. At present most authors are in agreement as regards the changes in venous pressure during rest and activity, and the present author has chosen the works of *Højensgård & Stürup* (1949, 1952) and *Stürup & Højensgård* (1950 a, 1950 b) as the basis for the following summary. Both as regards technique and critical evaluation of the results, the work of these authors is as yet unsurpassed.

It will be practical to consider the venous pressure measurements in the various parts of the venous systems of the lower extremity separately (saphenous veins, deep veins of the thigh, deep crural veins and ankle-perforating veins) and to report the findings (or lack of investigations) in normal subjects and in patients with chronic venous insufficiency in each venous system.

A. Subcutaneous veins (saphenous system).

1) *Normal subjects:* Normal subjects in the motionless standing position show a pressure in the saphenous veins, which at each point is equal to the hydrostatic pressure of a column of blood reaching to the level of the heart.

Muscular work (walking) causes the pressure in the veins to fall gradually until it reaches a stable level, which in the lower leg is 600—900 mm H_2O below the pressure found in the motionless standing position.

2) *Patients with idiopathic varicose veins:* In the motionless standing position the pressure in the varicose saphenous veins is at all levels equal to the hydrostatic pressure of a column of blood, reaching to the level of the heart. During walking there is none or only a negligible fall in the mean pressure in the varicose veins. A fall in pressure occurs gradually when the incompetent saphenous vein is compressed above the point of measurement. This is observed clinically as "emptying of the veins" in the Perthe's test.

3) *Patients with total postthrombotic destruction of the valves in the deep veins of the lower extremity and varicose superficial veins.*

In the motionless standing position the pressure in the subcutaneous veins is — at all levels — equal to the hydrostatic pressure of a column of blood reaching to the level of the heart. Walking fails to produce a decrease in pressure in the saphenous veins.

Compression of the incompetent saphenous veins by a tourniquet does not effect a fall in pressure. Clinically this is demonstrated as "no emptying of the veins" in the Perthe's test.

4) *Patients with partial postthrombotic destruction of the deep veins.*

In some patients in whom the thrombotic process had left a few deep veins intact, a demonstrable decrease in pressure could be observed during walking with tourniquet. (Postthrombotic *B. Højensgård & Stürup* (1949).

B. The deep veins of the thigh.

Højensgård & Stürup (1952) made the very interesting observation, that the pressure in the popliteal vein (the tip of the catheter was placed in the upper popliteal or lower femoral vein) during the motionless standing position was equal to the calculated hydrostatic pressure of a column of blood reaching to the heart, and that walking produced no decrease in mean pressure; the pressure fluctuated about the hydrostatic pressure at rest with very modest excursions. *These findings were identical in normal subjects and in all forms of venous insufficiency.*

C. The deep veins of the leg.

The pressure in the deep crural veins has never been measured.

D. The ankle-perforating veins.

The pressure in the deep crural veins has never been investigated under physiological conditions.

As regards the significance of the "idiopathic incompetence of the femoral vein" (Bauer (1947, 1948), *Højensgård & Stürup* (1952) make the following comment:

"While the venous return in the normal lower leg is an active process which lowers the mean pressure, the fact that the pressure in the popliteal and femoral veins does not fall during walking indicates that in these veins the venous return is more passive. Since no decrease occurs in the pressure in the popliteal vein, there is no tendency to retrograde flow in the femoral vein during the resting phase of the muscular action. Consequently absence of valves in the femoral vein can hardly be expected to be of anywhere near the same pathological significance as absence of valves in the deep lower leg veins (if the assumption holds true that a sustained high pressure in the lower leg veins is the essential factor in the production of the oedema which causes the symptoms and signs in extremities with venous diseases). The fact that normally the femoral vein is provided with only a few valves, while the deep lower leg veins possess numerous, densely arranged valves, points in the same direction.

The difference in the function of the deep veins apparent between normal limbs and limbs with primary varicosities on the one hand and limbs with postthrombotic syndrome on the other hand, is therefore presumably due, not to the difference in the valvular equipment in the popliteal and femoral veins, but to the difference in the valvular equipment in the deep lower leg veins — — —. If only for this reason, it seems appropriate to display some scepticism towards the recently advanced theory of the deleterious effect of an idiopathic valvular incompetence of the femoral vein in certain cases of varicosities — — —."

As will be seen from Chapter IX the author's phlebographic investigations demonstrated the great importance of the valves in the popliteal vein for the function of the venous pump, while postthrombotic destruction of the valves in the deep crural veins did not — *per se* — seem to have any additional deleterious effect on a pump mechanism, which was already impaired by the presence of incompetent ankle — perforating veins.

PART II.
OWN INVESTIGATIONS.

CHAPTER VI.
THE STANDARDIZED DYNAMIC INTRAOSSEOUS TYPE OF PHLEBOGRAPHY.

A. Method. (Arnoldi & Bauer (1960).

The patient is placed on the operating table in the horizontal position (Fig. 7). (A tiltable x-ray table will serve the same purpose). The skin at the lateral aspect of the os calcis is thoroughly rinsed and painted with a solution of iodine. A few mililiters of Xylocaine ® or novocain 1 per cent is applied to the skin and periosteum.

One minute later a wide-calibered needle mounted on a handle is introduced through the compacta into the substantia spongiosa of the os calcis. Care must be taken at this point not to widen the canal through the compacta unnecessarily, as this may result in a backflow of the contrast medium into the subcutaneous tissues.

When the needle is in situ the handle is removed and a syringe is applied to the needle. Gentle suction will produce a few drops of blood, a sign that the needle is in position. Five mililiters of Xylocain ® 0.5 per cent are then injected. At this point the patient generally feels a slight stab of pain. This passes quickly, however, and is the only pain felt throughout the procedure.

The patient is tilted into a semi-erect position (Fig. 8). The extremity to be examined rests lightly on a pedal and is rotated inwards 30 degrees, while the whole weight of the patient rests firmly on the other leg.

Twenty mililiters of Umbradil ® 35 per cent are injected after three minutes of complete rest. The injection, which now is painless, is performed as quickly as possible (45 to 60 seconds), and the first exposure is made as soon as the injection is finished.

The pedal is balanced to a considerable weight (12 kgs) (Fig. 8) so that moving it requires a powerful contraction of the muscles of the calf. After the first exposure the patient (who has been instructed pre-

viously) is asked to tramp the pedal as far down as possible. This very powerful contraction is repeated three times (the three contractions being timed to last fifteen seconds) and the second film is exposed immediately after the end of the third contraction, as soon as the leg has returned to the resting position.

The patient is then returned to the horizontal position with the needle *in situ* while the films are being developed. Only antero — posterior exposures are generally necessary, as the subcutaneous veins very rarely disturb the picture of the deep veins and the ankle-perforating veins.

B. Phlebographic demonstration of the function of the venous pump.

The intention, when using a dynamic type of phlebography, is to get an idea of the function of the venous pump. In order to obtain comparable results the apparatus must be calibrated by means of a normal material. The lowest number of tramps needed to empty the normal veins is chosen as standard procedure.

Fourteen normal subjects were examined as a control material. In these phlebograms most, or all of the contrast medium had disappeared from the veins of the leg and lower thigh in the second exposure; that is from the musculo-fascial compartments of the leg and their corresponding "collecting vein", the vena poplitea. This was taken as a phlebographic demonstration of a normal function of the musculo-venous pump in the leg.

On the other hand it was found, that in a great many phlebograms from patients with various forms of chronic venous insufficiency, the second exposure showed a marked retention of contrast medium in the deep veins of the leg. This was regarded as a phlebographic demonstration of an impaired function of the musculo-venous pump mechanism in the leg.

C. Material.

The material on which the following chapters are based consists of 337 phlebograms from patients with chronic venous insufficiency. Apart from the first period, when the method was tried out, only pa-

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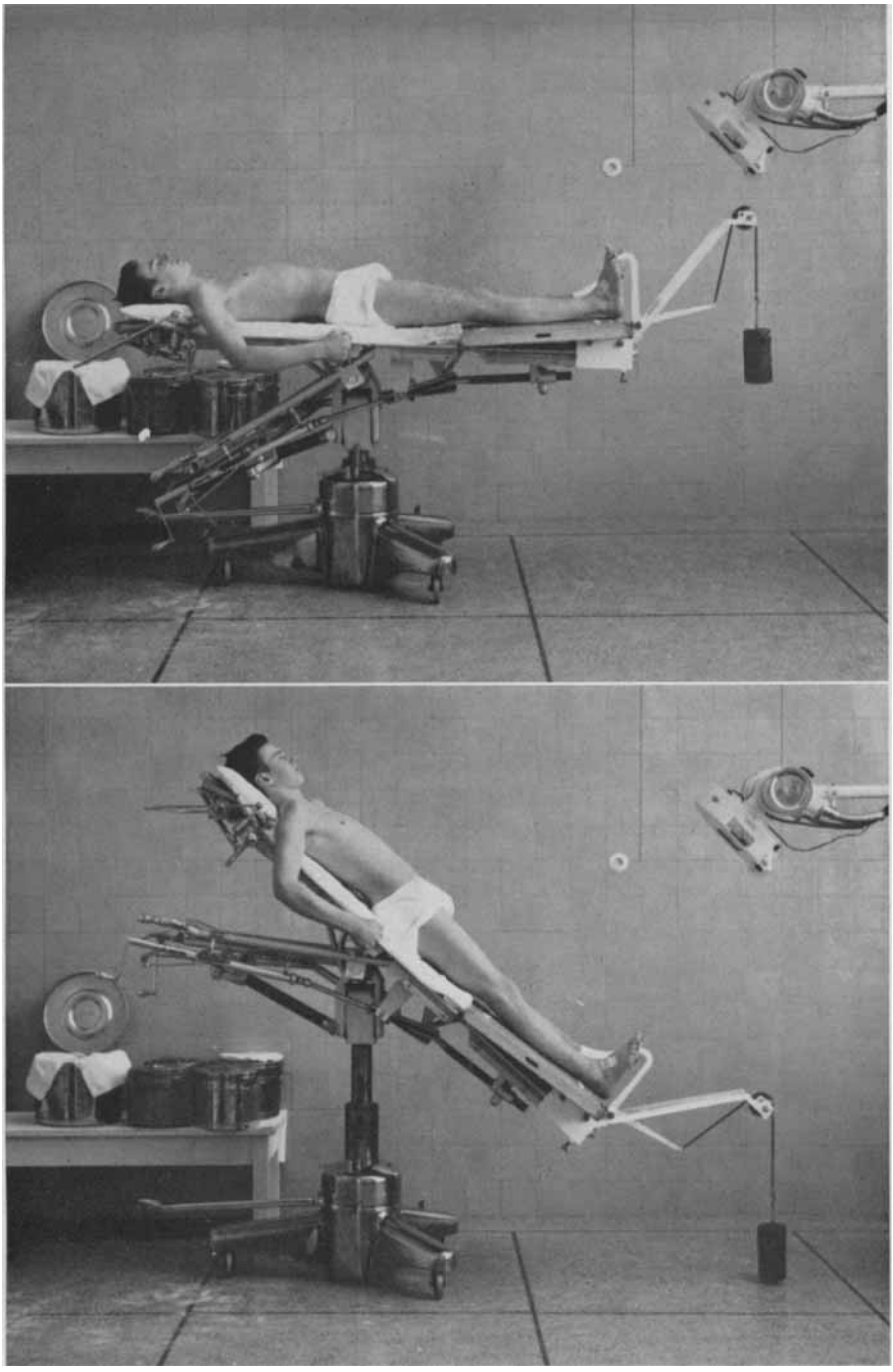
Fig. 7. Patient in the horizontal position prior to phlebografic examination.
Fig. 8. The table has been tilted into an angle of 45 degrees from the horizontal plane. The extremity to be examined rests on a spring pedal to which a considerable weight is attached (12 kgs).



tients with the more severe forms of venous insufficiency were examined (41 per cent of the extremities in the material were ulcerated). The figures and percentages presented in the following chapters are therefore not representative as regards chronic venous insufficiency as a whole, but only of the present material.

The number of patients in the phlebographic groups and the ratio men/women are recorded in Table I.

TABLE I

Records the number of phlebograms in the different phlebographic groups, and the ratio men/women.

	Men	Women	Total	Women %
Group I type A	13	36	49	74
Group I type B	3	9	12	75
Group II type A	31	108	139	78
Group II type B	19	43	62	69
Group III	13	63	76	83

D. The phlebographic groups.

An analysis of the phlebograms from extremities with chronic venous insufficiency showed, that the phlebographic material could be divided into five characteristic groups (Arnoldi 1961 a).

In the phlebographical analysis a set of criteria were employed, encompassing functional as well as morphological characteristics.

First, the phlebograms were divided into two main divisions: those with a normal function of the venous pump (good emptying in the second exposure), and those where the venous pump was impaired (retention of contrast in the second exposure). In the first division (normal function of the venous pump Group I) the contrast-filled deep veins appeared normal in the vast majority of the phlebograms and the valves in the communicating veins seemed to be competent. These patients had varicose saphenous veins. The division contained, however, a small number of phlebograms with signs of a previous attack of deep thrombophlebitis, confirmed by the patients' history. It was therefore found necessary to divide Group I into two types A and B. Group I type A (Fig. 14) contains the patients with primary saphenous varicosities of the simple type, while Group I type B contains patients with postthrombotic changes in the deep veins, with normal function of the venous pump. Superficial varicosities were always present.

In the second division of the material (impaired or destroyed func-

tion of the venous pump) the presence of large incompetent ankle-perforating veins was a dominant characteristic in a large number of cases, and this characteristic was employed to distinguish between Group II (incompetent ankle-perforating veins) and Group III (impaired function of the venous pump, but competent ankle-perforating veins).

In Group II it was possible to distinguish between two subgroups, according to the degree of impairment of the venous pump.

In Group II type A (Fig. 16, 17, 18) the venous pump was deficient owing to a leak through the incompetent ankle-perforators. Some degree of function remained, however, as the contrast medium was seen — in the second exposure — to have been sucked back from the subcutaneous system, into and upwards through the deep veins.

In Group II type B (Fig. 20, 21, 22) the venous pump was completely destroyed and the contrast-mixed blood was seen to move slowly upwards through the superficial as well as the deep veins, with a speed corresponding to the pressure exerted by the vis-a-tergo, quite unaffected by muscular activity (serial phlebography).

Group II type A contained patients with normal deep veins as well as patients with deep crural veins showing signs of an earlier attack of deep thrombophlebitis.

Group II type B only contained extremities with postthrombotic valvular destruction of all the deep veins in the phlebogram.

A further analysis of the material (Arnoldi (1961 b) showed that phlebographically normal popliteal valves were found in the large majority of the postthrombotic extremities in Group II type A, while the large majority of the patients in Group II type B had no visible popliteal valves (Table II).

TABLE II

A comparison between the postthrombotic cases in Group II type A and Group II type B. The state of the popliteal valves, the type of anticoagulants given during the acute thrombotic attack and the severity of the chronic stage are recorded.

	Group II type A	Group II type B
Number of cases	27 (100.0 %)	62 (100.0 %)
Popliteal valves normal	22 (81.5 %)	1 (1.7 %)
Popl. valv. not present	2 (7.4 %)	53 (85.4 %)
Popl. valv. not visible	3 (11.1 %)	8 (12.9 %)
Heparin treatment during acute attack	18 (66.7 %)	5 (8.0 %)
Dicoumarol alone during acute attack		10 (16.0 %)
Embolus in connection with acute attack		7 (11.3 %)
Number of leg ulcers	14 (52 %)	50 (81 %)

Fig. 14. Phlebogram from patient with primary varicose veins. (Group I type A).
Fig 14 a. shows the location of contrast medium immediately after injection in the quiet standing position (Diastole II). The contrast filled deep crural and popliteal veins have normal contour and valves. Traces of contrast medium outside the fascia cruris.
Fig 14 b. Exposed immediately after cessation of third muscular contraction (Diastole I). The deep crural and popliteal veins are empty apart from slight traces of contrast medium at the valvular pocketts. Normal function of the venous pump.
Fig. 16. Phlebogram from patient with varicose veins, incompetent ankle-perforating veins and intact popliteal valves.
(Group II type A).
Fig. 16 a. shows the location of contrast medium immediately after injection in the quiet standing position (Diastole II). The contrast is seen in the deep crural veins in the lower two thirds of the leg. Some contrast has been shunted out through two incompetent medial ankle-perforators and has filled a section of the subcutaneous system.
Fig. 16 b. is exposed immediately after the end of third muscular contraction (Diastole I). As a result of muscular activity the contrast medium has moved upwards in the deep veins. The popliteal vein is now visible and is seen to have normal valves, as have the visible deep crural veins. The lower fourth of the leg is free from contrast. The contrast medium in the incompetent ankle-perforating veins and subcutaneous veins has been sucked back into the deep system.
Fig. 17. Phlebogram from patient with varicose veins, incompetent ankle-perforating veins and intact popliteal valves. (Group II type A).
Fig. 17 a. shows the location of contrast medium during Diastole II. Contrast has been shunted out through an incompetent medial ankle-perforator and a short segment of subcutaneous vein has become visible.
Fig. 17 b. exposed during Diastole I. As the result of muscular activity the lower part of the deep crural veins are by now empty. The contrast has been sucked back into the deep veins from the subcutaneous and ankle-perforating veins and the column of contrast has reached the popliteal vein.
Fig. 18. Phlebogram from patient with varicose veins, incompetent ankle-perforating veins, earlier attack of deep thrombophlebitis, but intact popliteal valves. (Group II type A).
Fig. 18 a. Diastole II. The contrast medium is seen in the lower part of the deep crural veins, some of which have normal valves and contours. Some contrast medium has been shunted into the subcutaneous system by way of an incompetent lateral ankle-perforator. A section of the subcutaneous system has become visible.
Fig. 18 b. Diastole I. The contrast has disappeared from the lower fourth of the deep veins and from the ankle-perforator and subcutaneous veins. The proximal parts of the deep veins and the popliteal vein have become visible. Some deep veins show a woolly, irregular contour, and in sections the deep valves seem to have been destroyed. The popliteal valves are intact.

Fig. 14

Fig. 16

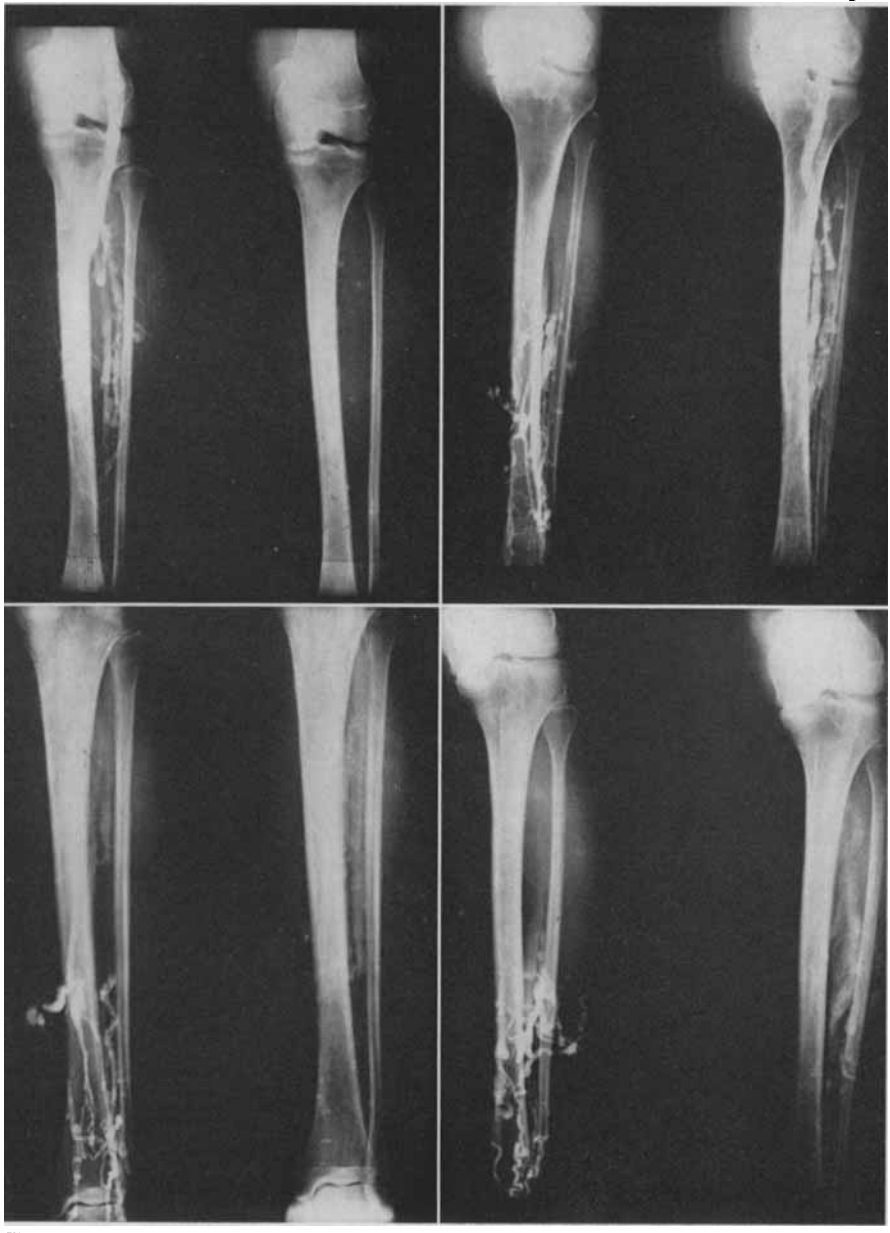


Fig. 17

Fig. 18

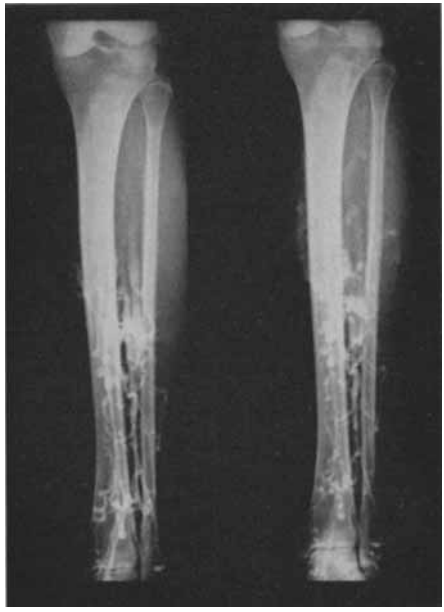


Fig. 20. Phlebogram from patient with varicose veins, incompetent communicating veins and previous deep thrombophlebitis, which has involved the popliteal vein. (Group II type B).

Fig 20 a. Diastole II. Immediately after injection in the quiet standing position the contrast medium has filled the distal two thirds of the deep crural veins. Some contrast has been shunted out into the subcutaneous system via incompetent lateral and medial ankle-perforating veins.

Fig 20 b. Diastole I. Immediately after the end of muscular activity contrast is seen in the deep crural veins from the ankle up till the popliteal level. The deep veins are irregular, somewhat woolly in contour. A few valves are seen in the anterior tibial veins, but the rest of the deep system is irregular and valveless, including the popliteal vein.

The contrast remains in the communicating veins and has progressed further upwards in the subcutaneous veins. Contrast is still seen in the lower fourth of the leg (Compare Figg. 16, 17 and 18).

There remained a group of patients, whose phlebograms showed an impaired or completely destroyed function of the venous pump, but where the ankle-perforating veins were competent and the valves of the deep veins apparently normal. In the large majority of these patients (Group III proper) (Fig. 24, 25) the diameter of one or several of the deep crural veins was abnormally wide. (When grouping the phlebograms the author used the width of the fibula, measured ten cms below

Fig. 21 *Fig. 22*

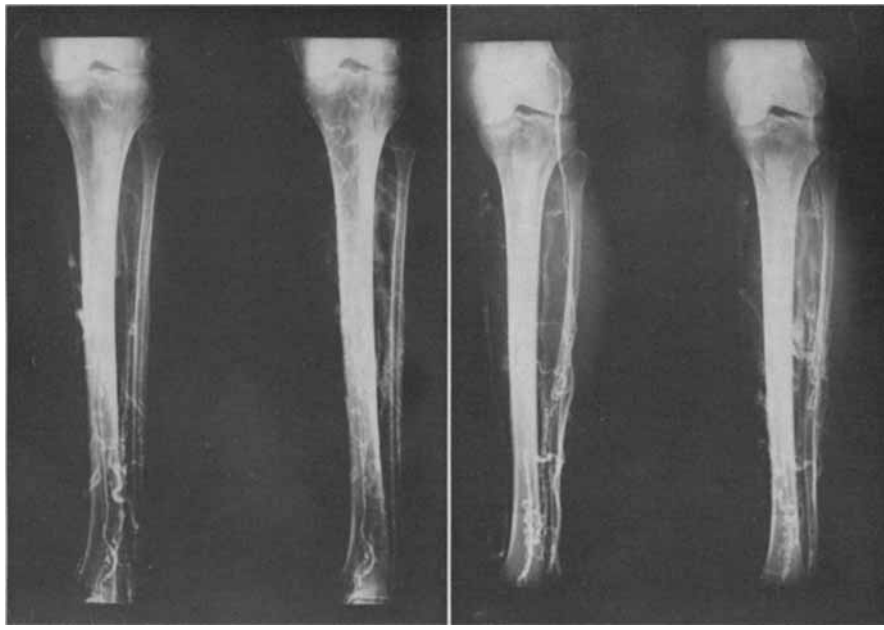


Fig. 21. Phlebogram from patient with varicose veins, incompetent communicating veins and widespread postthrombotic valvular destruction, including the popliteal vein. (Group II type B).

Fig. 21 a. Diastole II. The contrast medium is located in the severely deranged deep veins without demonstrable valves. Contrast has reached the subcutaneous veins by way of three medial and two lateral incompetent communicating veins.

Fig. 21 b. Diastole I. Muscular activity has failed to empty any section of the deep system. The contrast has not been sucked back from the subcutaneous veins, instead a further filling has taken place.

Fig. 22. Phlebogram from patient with varicose veins, incompetent communicating veins and widespread postthrombotic valvular destruction, including the popliteal vein (Group II type B).

Fig. 22 a. Diastole II. The contrast medium is seen in irregular, valveless deep channels as far as the popliteal vein. The subcutaneous system has received contrast via four medial and two—three lateral incompetent perforating veins.

Fig. 22 b. Diastole I. No emptying after muscular activity. Further filling of the superficial veins.

the tip of the capitulum for comparison). The phlebograms of Group III proper all showed a diameter equal to or wider than the fibula at this level. A small number of phlebograms were referred to as Group IV. In these cases the diameter fell short of the diameter of the fibula, but otherwise the characteristics were identical with those of Group III.

In the following evaluation of the function of the venous pump the phlebograms of Group IV are included in Group III.

In most of the phlebograms in Group III the popliteal vein seemed wider than usual, but no attempt was made to determine the normal caliber of this vein.

The author is aware that the grouping of the phlebograms according to the criteria mentioned above is open to critisism and that the grouping is not quite "logical", as different sets of characteristics are employed (the phlebographic appearance of the valves, the presence or absence of incompetent communicating veins, the diameter of the deep veins and the function of the venous pump). The groups cut across the established division of chronic venous insufficiency into primary idiopathic varicosis and venous insufficiency due to postthrombotic sequelae. Unfortunately, this cannot be helped, and one important discovery by means of dynamic intraosseous phlebography is, that the function of the venous pump may be normal, impaired or completely destroyed in idiopathic venous incompetence (Group I type A, Group II type A and Group III) as well as in postthrombotic venous insufficiency (Group I type B, Group II type A (postthrombotic cases) and Group II type B).

Intermediate forms occur. The wide diameter seen in Group III is sometimes found together with the idiopathic form of incompetence of the ankle-perforating veins (Group II type A). The author has preferred to use the presence of incompetent ankle-perforating veins as the dominant characteristic in these cases.

Group I type B contained so few phlebograms that an evaluation of the venous pump in this group is deemed futile until a larger material has been collected.

Fig. 24 *Fig. 25*

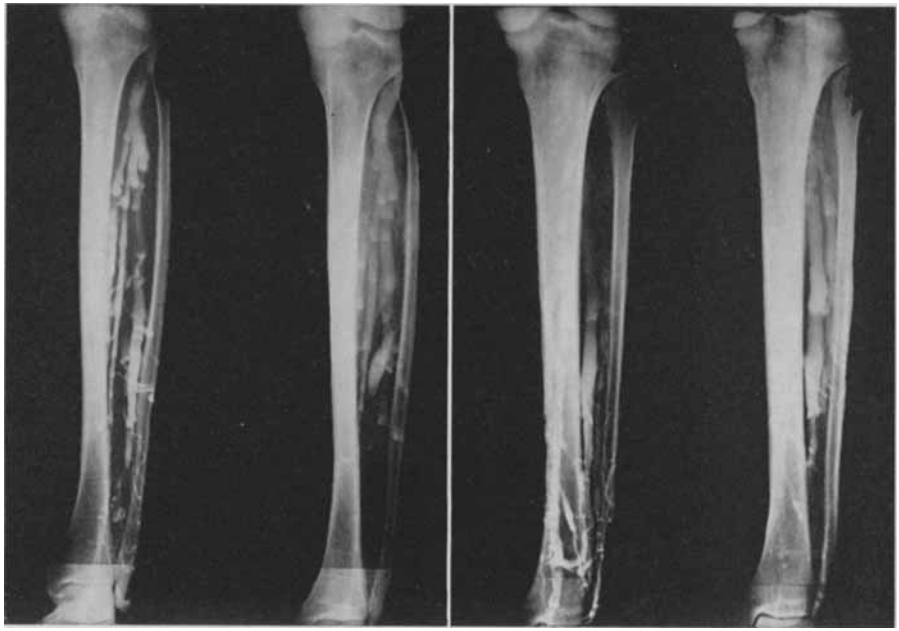


Fig. 24. Phlebogram from patient with primary varicose veins and »idiopatic deep incompetence«). Group III.

Fig. 24 a. Diastole II. Contrast medium is seen in the deep crural veins throughout the lower leg. The contrast filled veins have normal contour and abundant valves. The diameter in this exposure practically normal.

Fig. 24 b. Diastole I. The distal fourth of the lower leg is empty, but the rest of the deep veins have retained the contrast. The diameter of the deep trunks is enlarged compared with Fig 24 a, and exceeds that of the fibula ten cms below the proximal tip on several places.

Fig. 25. Phlebogram from patient with primary varicose veins and »idiopatic deep incompetence«. (Group III).

Fig. 25 a. Diastole II. Contrast medium is found in the normal looking deep veins to the lower two thirds of the leg. The diameter in this exposure is within normal limits.

Fig. 25 b. Diastole I. The deep veins in the lower fourth of the leg are practically empty, but several large trunks in the upper three fourths of the leg have retained the contrast. The diameter in these trunks exceeds that of the fibula ten cms below the proximal tip. The valves seem normal.

CHAPTER VII.
THE VENOUS RETURN FROM THE LOWER LEG IN THE NORMAL EXTREMITY.

The section of the lower extremity with which we are concerned at present may be represented by the schematic drawing shown as Fig. 9. The deep crural veins with their abundancy of valves are closely surrounded by muscles, which are ensheathed by the septa of the fascia cruris, while the collecting popliteal vein — much poorer in valves — is represented as being entirely free of muscular relations. The ankle-perforating veins and the subcutaneous venous system are represented schematically.

In the following paragraphs the author intends to describe the function of the venous pump in the normal extremity, using the information obtained from the literature regarding tissue tension and venous pressure (Chapter IV and V) in combination with the findings which emerged from the analysis of the phlebographic material.

Diastole II, Systole and Diastole I.

In order to facilitate the evaluation of the venous return, the author distinguishes between three phases in the cyclus contraction/relaxation of the muscles of the pump sections, namely Diastole II, Systole and Diastole I.

The first exposure in the standardized dynamic method of phlebography was made at a time, when the extremity to be examined had been completely at rest for several minutes. The extremity was not weight-bearing. Serial phlebography has shown that the blood flow during rest is slow (about 1—2 cms per seconds) and that this rate of flow is the same in normal legs and in extremities with chronic venous insufficiency, that is, whether the valves in the deep veins are functioning or not. Although direct measurements of the pressure in the deep crural veins have not been performed, it may be assumed that the pressure in the deep veins at rest at any point is equal to the hydrostatic

pressure of a column of blood reaching to the level of the heart, and that the valves are open.

This period of slow steady flow is called *Diastole II.*

Systole. The period from the onset of muscular contraction until relaxation sets in is called systole.

Diastole I. At cessation of muscular contraction the musculo-fascial compartment contains less blood than when the contraction began. The sudden fall in muscular tension (1200 to 50 mm H_2O in the lower third of the soleus) and the diminished contents of the musculo-fascial compartment is in the following assumed to cause a fall in venous pressure in the deep crural veins, below that found in the collecting (popliteal) vein outside the section of the muscle pump.

The inflow from the arterial system and from the veins outside the musculo-fascial compartment will fill the veins of the pump section. Diastole I is the phase which begins at the cessation of muscular contraction and lasts until the pressure in the deep crural veins has risen above the pressure in the popliteal vein. When this happens, the popliteal valves must open and the state of slow steady flow is reestablished (Diastole II).

The second exposure in the standardized method of dynamic phlebography is made immediately after the leg has returned to the resting

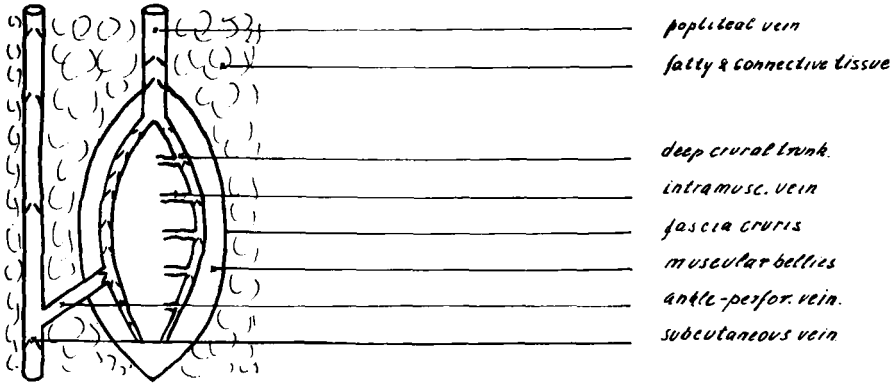


Fig. 9. Schematic drawing of a musculo-venous pump section in the lower leg. The deep crural veins with their abundancy of valves are closely surrounded by muscles, which are ensheathed by the strong crural fascia. The collecting popliteal vein is shown in its relation to the loose fat in the popliteal space. A superficial vein is connected with the deep veins through an ankle-perforating vein.

position and may thus be expected to record the localisation of the contrast medium in Diastole I.

The venous return from the lower leg in the normal extremity.

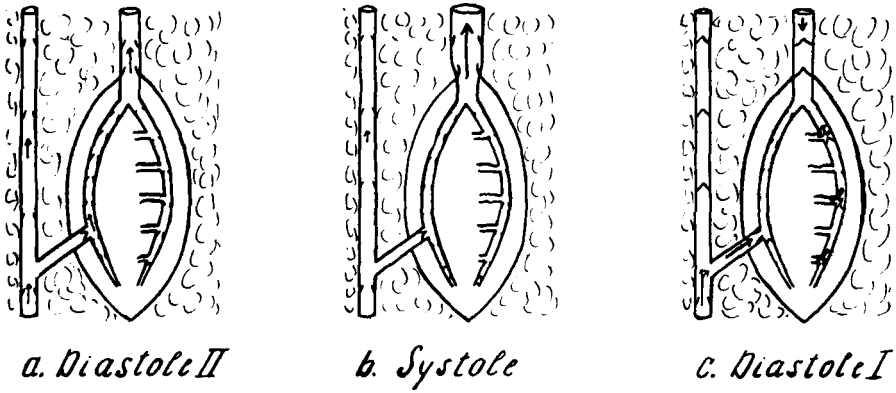
A. Diastole II.

In the quiet standing position the pressure in the great saphenous vein, measured at ankle level, corresponds to the hydrostatic pressure of a column of blood reaching to the level of the heart. In the average person this is equal to about 1000 mm H_2O. At the level of the knee the pressure in the great saphenous vein and in the popliteal vein is approximately equal and corresponds to the hydrostatic pressure at this level. The pressure in the deep veins inside the musculo-fascial compartments of the leg has never been measured, nor has the pressure in the communicating veins and the ankle-perforating veins.

By means of serial phlebography the contrast medium could be observed to pass slowly upwards at a speed varying between 1 and 2 cms per second in the quiet semierect position. As we cannot assume that the blood will pass from a point with low pressure against a higher pressure, we may presume that the pressure in the deep crural veins is higher than in the popliteal vein. Probably the pressure is somewhere near the expected hydrostatic level (all valves presumably open) and the slow progress of the contrast-mixed blood must be due to the vis-a-tergo.

The amount of contrast medium seen outside the fascia cruris was always negligible and — when present — it could be traced back to leaks from the calcaneus into the subcutaneous veins below the malleolar region. Certain cases of retrograde filling via the ankle-perforating veins were not found in the control material. This would seem to mean that the valves at the entrance of the ankle-perforating veins into the deep veins are closed in the quiet standing position; otherwise it would be improbable that the contrast medium, which is heavier than blood should not find its way out, even against the bloodstream *(Greitz* (1955). This again must mean that the pressure in the deep veins is higher than in the subcutaneous veins at the same level in the quiet standing position. How much higher we do not know, as no measurements are available as yet.

One point to consider is, whether the phlebographic appearance is an artefact, caused by an elevated deep pressure transmitted through the osseous sinuses from the injection pressure. The question cannot be



Figg. 10 a, b and c. represent in schematic form the function of the venous pump in the lower leg during Diastole II, Systole and Diastole I in a normal subject.

Fig. 10 a. Diastole II. This is the state of slow steady flow maintained by the vis-a-tergo. The valves in the subcutaneous, deep crural and popliteal veins are open, allowing the hydrostatic forces full effect. The difference in environmental support raises the effective pressure in the deep crural veins to a slightly higher level than in the subcutaneous veins. Hence the closed valves in the communicating vein.

Fig. 10 b. Systole. As the result of muscular contraction the pressure in the deep crural veins rises above that in the popliteal vein and the blood is pressed out of the musculo-fascial compartment. Normal valves prevent an outflow distally and through the communicating veins.

Fig. 10 c. Diastole I. At the cessation of Systole the pressure in the deep crural veins falls abruptly. When the pressure falls below the popliteal values the popliteal valves will close and seal the upper outlet from the pump section.

The low pressure inside the fascia cruris makes a central flow possible from the subcutaneous into the deep system via the communicating veins. The pressure in the adjacent segment of the subcutaneous veins will begin to fall.

The low pressure in the deep trunks will facilitate the outflow from the muscular tributaries.

Filling of the central trunks from the muscular tributaries and from the superficial veins causes a rise in pressure eventually inverting the pressure gradient at the popliteal valves. When this has been effected the popliteal valves will open and the state of steady flow (Diastole II) has become reestablished. The cycle is complete.

answered, but the pressure absorbing qualities of the spongiosa and the slow proximal progress of the contrast make it improbable that this factor is of any great importance. Until further investigation proves different we will assume, that the difference in deep and subcutaneous pressure is real.

Another factor which may go a long way in explaining the difference in pressure in the deep and subcutaneous veins is the pressure in the surrounding tissues. The tissue pressure in the subcutis (10—70 mm H_2O) was found to be somewhat lower than the intramuscular pressure at rest (50—110 mm H_2O). The difference is probably even more pronounced as complete relaxation is well nigh impossible to achieve in a standing conscious person. Assuming that the elastic properties of the vein wall are equal in the deep and superficial veins, the difference in tissue support would tend to diminish the effective pressure in the subcutaneous veins, compared with the deep veins.

B. Systole.

In the normal extremity muscular exercise causes the pressure in the great saphenous vein — measured at the ankle — to fall from about 1000—1200 mm H_2O to about 300—400 mm H_2O. This fall in pressure is gradual and is within certain limits dependent upon the muscular work involved. The pressure in the popliteal vein fluctuates about a mean value af 400—600 mm H_2O, that is, muscular exercise leaves the mean pressure in this vein practically unaltered and the result at knee level is, that the pressure in the saphenous system eventually falls below that in the politeal vein.

The pressure inside the fascia cruris has not been measured, and the pressure in the ankle-perforating veins during muscular contraction is also unknown.

Muscular contraction caused a steep rise in pressure in the muscles of the leg, highest in the soleus (1200—480 mm H_2O) and the tibialis anterior (700 mm H_2O), while the gastrocnemius only showed a maximal contraction pressure of 310 mm H_2O.

Barcroft & Dornhorst (1948) demonstrated by means of plethysmographic investigations that the "pump-out" mechanism of the leg in a normal person could overcome a constricting force of 1200 mm H_2O.

Thus, although direct measurements of the venous pressure in the deep crural veins are still missing, we may assume from the circumstantial evidence at hand, that the pressure of the surrounding contracting muscles raises the venous pressure high enough to overcome

the hydrostatic pressure at the outlet of the musculo-fascial compartments by a comfortable margin.

The muscular contraction raises the pressure in the deep veins of the leg far above the subcutaneous venous pressure. As a consequence the valves at the juncture of the communicating veins and the deep veins must remain closed during systole.

The pressure in the popliteal vein also rises during muscular contraction, but the rise is modest. This fact is more easily understood, when one realises that no appreciable muscular action is brought to bear upon the popliteal vein, and that the loose fatty tissue embedding the vein may act as a buffer by allowing the vein to expand.

All investigations show that the fall in pressure in the subcutaneous veins during exercise is gradual, and that a single contraction only results in a moderate fall in mean pressure.

The present method of dynamic intraosseous phlebography does not show the expulsion of the contrast-mixed blood during systole, but only what has happened to the contrast after three cycles of contraction and relaxation.

C. Diastole I.

The pressure in the great saphenous vein at ankle level falls gradually during rhythmic muscular contractions from a level corresponding to the hydrostatic pressure of a column of blood reaching to the right auricle to a minimum level of about 300—400 mm H_2O. This fall in pressure is due to two factors, namely the presence of competent valves in the subcutaneous veins and the central flow of blood through the communicating veins into the deep veins.

We have seen that the phlebographic investigations by means of the dynamic intraosseous method make it highly improbable that a central flow through the communicating veins takes place during the phase of prolonged quiet standing (Diastole II), and that the steep rise in intramuscular pressure during Systole makes such a flow even less feasible in this phase.

We know, however, from clinical experience and from the Perthes test, that a series of rhythmic muscular contractions will empty the subcutaneous veins by means of an inflow of blood through the communicating veins into the deep crural veins. To understand how this inflow is made possible, the hydromechanics in the leg in the period following immediately after the cessation of muscular contraction are of special interest.

When relaxation sets in, the very high intramuscular tension which was found during Systole falls. To what levels it will fall is still unknown, as the measurements available only tell us about the tension in the relaxed muscle after prolonged rest, that is in Diastole II. This resting value was found to range from 20—50 mm H_2O in the gastrocnemius and from 50—110 mm H_2O in the soleus and tibialis anterior. The intramuscular tissue tension varies with the tension in the adjacent veins. As the veins in the pump section contain less blood at the end of Systole, than after a period of rest, it is not inconceivable, that a lower muscular tension exists in Diastole I than in Diastole II.

If we assume, that the venous pressure in the by now partially empty deep crural veins falls to somewhere near the level of the resting intramuscular pressure, a very considerable difference in pressure will suddenly exist between the superficial veins (1200—600 mm H_2O) and the deep crual veins (110—20 mm H_2O). Now, for the first time in the entire cyclus contraction/relaxation a central flow of blood through the communicating veins is made possible. The pressure in the subcutaneous veins will begin to fall.

Diastole I, the period during which this central flow is possible, is probably of short duration. The fall in pressure in the subcutaneous veins is gradual and the minimal values are not reached until a number of muscular contractions have been performed. This must mean that only a fraction of the blood contained in the crural parts of the subcutaneous venous system reaches the deep crural veins in each Diastole I.

The deep trunks receive blood, not only from the communicating veins, but also from the numerous muscular tributaries. The low pressure in the deep trunks in Diastole I facilitates the outflow from the tributaries. The inflow from the communicating veins and the increased flow from the subfascial tributaries tend to fill the partially empty deep trunks rapidly and the rigid fascial sheaths have the effect, that an increase in volume is followed by a rapid increase in pressure inside the musculo-fascial compartment.

When the pressure in the deep veins below the popliteal valves rises above that found proximal to the valves (and this pressure has not changed appreciably during the whole cyclus) the valves will open. We have come to the end of Diastole I, and the phase of slow steady flow has been reached (Diastole II).

Jäger (1936) observed directly, that relaxation following contraction of the leg muscles in the frog caused the valves in the veins proximal

Fig. 11 *Fig. 12*

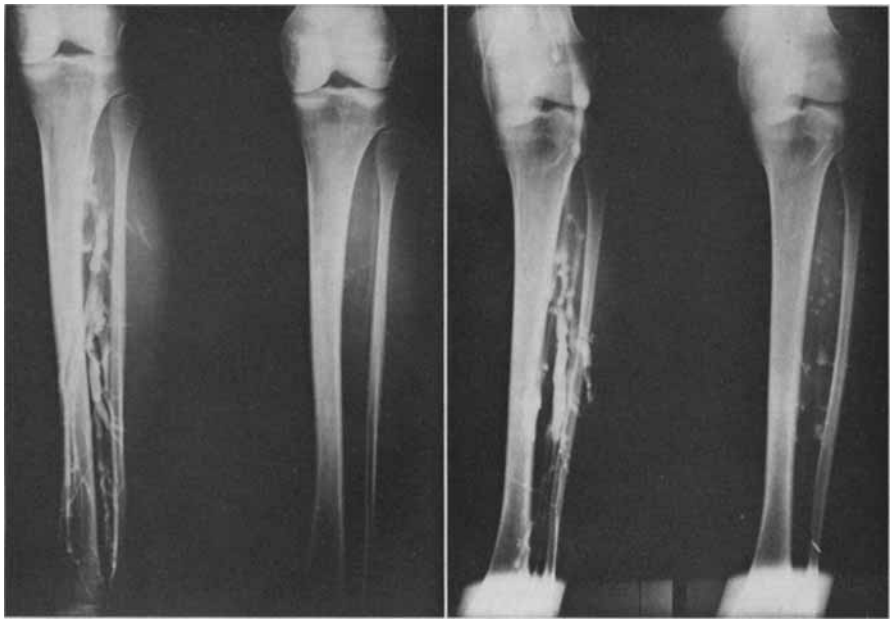


Fig. 11. Phlebogram from normal subject.
Fig 11 a.: shows the location of contrast medium immediately after injection during the quiet standing position (Diastole II). The contrast filled deep crural and popliteal veins have normal valves. The subcutaneous veins contain no contrast.
Fig. 11 b.: exposed immediately after the end of muscular contraction (Diastole I). Very fine traces of contrast medium left in the upper half of the leg.
Fig. 12. Phlebogram from normal subject.
Fig. 12 a.: shows the location of contrast medium in the deep crural and popliteal veins during Diastole II.
Fig. 12 b.: shows good emptying after three muscular contractions. (Diastole I). Retention of contrast is seen proximal to the valves. This is considered within the normal limits.

to the muscle to close for a few moments, whereupon the valves opened again and the proximal flow began.

Fig. 10 a, b and c describe the three phases in the contraction/relaxation cyclus in the normal leg.

Fig. 11 and 12 are examples of phlebograms from a normal leg.

CHAPTER VIII.
THE VENOUS RETURN FROM THE LOWER LEG IN PATIENTS WITH PRIMARY VARICOSE VEINS.

Among the patients with chronic venous insufficiency those suffering from simple varicose veins form the large majority. That is not the case in the present material (Table I). Apart from the early period of investigation these patients were as a rule not examined unless they suffered from oedema, induration or ulceration.

As a whole, this group of patients had slighter symptoms than the rest of the material, and leg ulcers were only found in fourteen per cent (Table III).

The subcutaneous varicosities are not visible as long as the valves in the communicating veins are competent, and phlebograms from these patients are therefore in all essentials exactly like those obtained from normal subjects. They are listed as Group I type A.

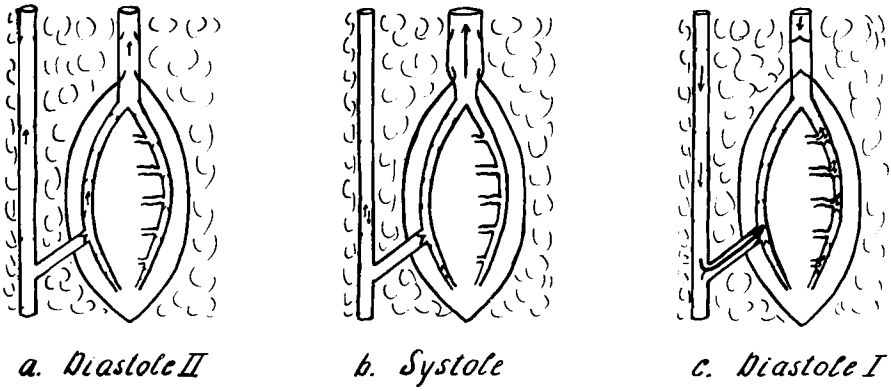
Subcutaneous and intramuscular tissue tension.

It is here assumed that the subcutaneous and intramuscular tissue tensions at rest and during muscular activity are identical in patients with simple varicose veins and in normal extremities. This assumption may be wrong, but no measurements of this sort have been performed in patients with chronic venous insufficiency.

Venous pressure measurements. The gradual fall in venous pressure, measured at ankle-level to minimal values of 300—400 mm H_2O, which is observed in the saphenous veins in normal extremities during muscular activity does not take place in patients with varicose veins. The pressure in the popliteal vein fluctuates about a mean value which corresponds to the hydrostatic pressure at that level. The pressure in the deep crural veins and the ankle-perforating veins has never been measured.

A. Diastole II.

Serial intraosseous phlebography has shown that the contrast medium proceeds upwards with a speed of 1—2 cms per second in the



Figg. 13 a, b and c. represent in schematic form the function of the venous pump in the lower leg during Diastole II, Systole and Diastole I in an extremity with primary varicose veins. (*Group I type A*).

Fig. 13 a. Diastole II. During the phase of slow steady flow the valves in the deep crural and popliteal veins are open and the driving force is effected by the vis-a-tergo. The hydrostatic forces are fully effective in the deep as well as in the varicose subcutaneous veins. Owing to the slightly higher pressure inside the fascia cruris the valves in the communicating veins are closed during Diastole II.

Fig. 13 b. Systole. The rising pressure inside the fascia cruris expells the blood from the musculo-fascial compartment. The route through the popliteal vein is the only feasible, owing to the normal valves in the deep crural and the communicating veins.

Fig 13 c. Diastole I. The sudden fall in pressure inside the fascia cruris below that found in the popliteal vein will close the popliteal valves. A central flow through the communicating vein is now possible. This has a tendency to lower the pressure in the varicose subcutaneous veins, but owing to the lack of functioning valves here a retrograde flow from above will restore the high venous pressure and make up for the loss of blood to the deep veins.

motionless semierect position. In the phase of steady flow the valves in the deep veins must be open and the hydrostatic pressure must be effective at all levels. This has been shown to be the case in the superficial veins and the popliteal vein by direct measurements of pressure.

As in the normal leg, the pressure in the deep veins may be assumed to be somewhat higher than in the subcutaneous veins at the same level (owing to the difference in tension in the surrounding tissues). Consequently the valves in the communicating veins stay closed in this phase.

Diastole II is the phase demonstrated by the first phlebogram. At the moment of exposure the contrast medium is confined to the deep trunks and nothing is seen in the communicating veins or in the saphenous varicosities.

B. Systole.

Maximal contraction of the soleus and the anterior tibial muscles causes a steep rise in intramuscular pressure, from 50—110 mm H_2O at rest to 700—1200 mm H_2O during contraction. The contraction pressure will easily overcome the pressure in the popliteal vein, and is higher than the maximal hydrostatic pressure found in the lowest part of the leg in the semierect position adopted for dynamic phlebography. The sudden steep rise in pressure inside the musculo-fascial compartment will squeeze the blood into the popliteal vein. This is the only way possible owing to the valvular competency, and the fact that the pressure in the lower part of the soleus rises to much greater heights than in the upper part (1200 mm H_2O against 480 mm H_2O).

The high pressure in the deep veins makes a central flow through the communicating veins impossible during Systole.

The standardized form of dynamic intraosseous phlebography gives no information as to what happens to the contrast medium during Systole. The second exposure is made after the completion of three cycles in Diastole I.

C. Diastole I.

At the cessation of contraction we assume that the intramuscular tension returns to somewhere near the resting level (50—110 mm H_2O). The pressure in the popliteal vein reaches its minimum, but the fall is modest. The pressure in the deep veins of the leg falls below that in the popliteal vein. The resulting pressure gradient will cause the popliteal valves to close. This conception is supported by the direct observations by Jäger (1936) on the conditions in the veins of the lower extremity of the frog.

The low pressure in the deep crural veins in Diastole I will favour a central flow through the communicating veins from the subcutaneous system, where the mean pressure until now has remained practically unchanged. The central flow is possible as long as the pressure in the saphenous veins is higher than in the deep veins. As in normal subjects, the low pressure inside the fascia cruris must be supposed to rise

quickly, as a result of the inflow from the subcutaneous veins and from the arteries and owing to the rigid character of the fascial sheaths.

The inrush of blood from the saphenous veins into the deep veins will tend to diminish the volume of blood and the pressure in these veins. This tendency to a fall in pressure is compensated by a retrograde flow of blood through the incompetent sapheno-femoral (popliteal) junctions during Diastole I. If this backflow is hindered by means of a tourniquet, the flow into the deep veins will empty the superficial varicosities (Perthes test).

Diastole I is phlebographically demonstrated by the second exposure in dynamic intraosseous phlebography. In the phlebograms of Group I type A the musculo-venous pump in the leg had a normal function. Most or all of the contrast medium had disappeared from the veins of the lower leg in the second exposure.

Fig. 13 a, b and c represent the author's conception of the venous pump in patients with primary varicose veins.

Fig. 14 a and b are phlebograms of Group I type A.

TABLE III

Records the subjective symptoms noted on the day of admittance in the various phlebographic groups, together with the number of leg ulcers.

Symptoms	Group I a 48	Group I b 12	Group II a 130	Groub II b 49	Group III 68
Without	21 (42 %)	3 (25 %)	28 (22 %)	2 (4 %)	1 (1.5 %)
Heaviness & tiredness	24 (48 %)	8 (67 %)	57 (43 %)	34 (70 %)	37 (54 %)
Slight pains	12 (24 %)	5 (42 %)	43 (33 %)	18 (38 %)	22 (32 %)
Bursting pains	—	1 (8 %)	18 (14 %)*	13 (27 %)	31 (46 %)
Restles legs & nightly cramps	1 (2 %)	3 (25 %)	18 (14 %)	6 (13 %)	13 (29 %)
Leg ulcers	7 (14 %)	4 (33 %)	69 (50 %)	50 (81 %)	9 (12 %)

*) Of the eighteen patients, thirteen had deep veins reminiscent of Group III.

CHAPTER IX.

THE VENOUS RETURN FROM THE LOWER LEG IN PATIENTS WITH PRIMARY VARICOSE VEINS AND INCOMPETENT COMMUNICATING VEINS OF THE LOWER LEG. (GROUP II TYPE A).

Isolated incompetence of the communicating veins in the lower leg was demonstrated clinically in four cases. In all the rest of the patients in this group incompetence of the communicating veins was accompanied by saphenous varicosities. The incompetent communicating veins practically always belonged to the group of ankle-perforating veins.

The clinical symptoms were graver than met with in Group I type A (simple varicose veins) and the frequency of leg ulcers much higher (Table III).

Group II type A contained patients with "idiopathic" incompetence of the ankle-perforating veins as well as a number of cases with phlebographic signs of an earlier attack of deep thrombophlebitis. The thrombotic process had practically always left the popliteal valves intact (Table II). In the evaluation of the function of the venous pump in Group II type A it will be of interest to see, whether the incompetent valves in the "postthrombotic" deep crural veins have any additional detrimental effect on the pump function, when compared with the cases where the deep veins were normal and no history of deep thrombophlebitis could be elicited.

Subcutaneous and intramuscular tissue tensions. In the following evaluation it is assumed that the subcutaneous and intramuscular tissue tensions are approximately the same as the values found in normal extremities. Measurements from patients with chronic venous insufficiency are not available. A priori one would assume that the subcutaneous tissue tension is altered by induration and ulceration.

Venous pressure measurements. In the quiet standing position the pressure in the saphenous veins was found to correspond to the expected hydrostatic pressure at each level. In the presence of saphe-

nous incompetence the normal gradual fall in venous pressure during rhythmic muscular activity did not take place.

The pressure in the popliteal vein fluctuated about a mean level which corresponded to the hydrostatic pressure at this point. The excursions were modest.

The pressure in the deep crural and in the ankle-perforating veins has never been measured.

A. Diastole II.

As in the normal leg and in the leg with simple varicose veins, serial phlebography performed with the patient in the motionless semierect position showed a slow proximal flow of the contrast-mixed blood in the deep veins. The speed varied between 1 and 2 cms per second. In the phase of steady flow, the valves in the deep crural veins and the popliteal vein must be assumed to be open, and the pressure at any given level in the deep veins must be somewhere near the calculated hydrostatic pressure of a column of blood reaching to the level of the heart. The difference in tension in the surrounding tissues would see to it, that the pressure is slightly higher in the deep veins than in the subcutaneous veins.

When the valves in the ankle-perforating veins are incompetent, the blood would thus be expected to flow from the deep veins into the saphenous varicosities in Diastole II.

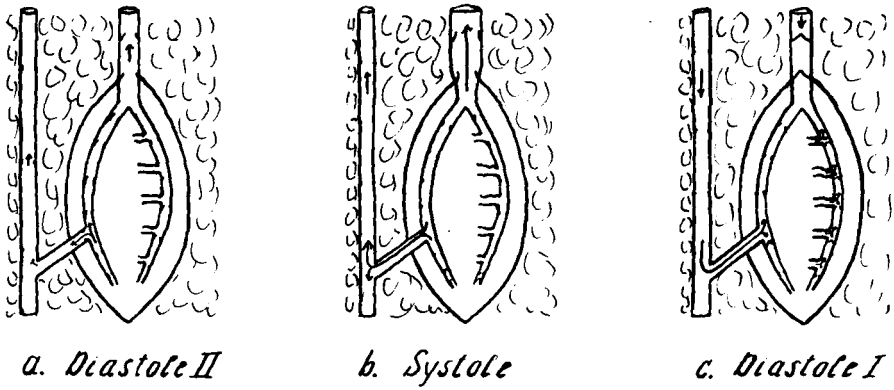
The first exposure in Group II type A confirms this assumption. When the contrast-mixed blood reaches the level of the incompetent ankle-perforating vein, some of the contrast medium is seen to seek its way towards the periphery, and a section of the subcutaneous network becomes visible (Figg. 16, 17, 18).

The contrast medium is slightly heavier than blood. It is thus conceivable that a peripheral flow of contrast can be found together with a central flow of blood (Greitz (1955). That the direction of the bloodstream through the incompetent ankle-perforating veins is in fact towards the periphery, can be directly observed by means of the back-flow test during operation. When an incompetent ankle-perforating vein is cut at the fascial level, the central segment will bleed towards the periphery. This is seen even when the extremity is raised above the level of the heart. A back-flow is not observed when the valves in the communicating veins are competent. (The phlebographic competence or incompetence of the ankle-perforating veins was controlled by means

TABLE IV

Incidence of positive or negative findings with phlebography and clinical examination in 175 cases of incompetent ankleperforators verified at operation.

No. of operated cases	Phlebo-graphy	Clinical exam.	Percentage
122	+	+	69.7
45	+	—	25.7
5	—	+	2.8
3	—	—	1.7
175			99,9



Figg. 15 a, b and c. represent in schematic form the function of the venous pump in the lower leg during Diastole II, Systole and Diastole I in an extremity with varicose veins, incompetent ankle-perforating veins, but intact popliteal valves. (*Group II type A*).

Fig. 15 a. Diastole II. State of slow steady flow. Driving force: the vis-a-tergo. All valves open in the deep system. Full effect of the hydrostatic forces both in the deep and the subcutaneous veins. Owing to the slightly higher effective pressure inside the fascia cruris and to the incompetence of the valves in the communicating veins, some of the blood is shunted towards the subcutaneous veins. The driving force is small.

Fig. 15 b. Systole. The steeply rising pressure inside the fascial compartment drives the blood upwards towards the thigh and outwards through the incompetent communicating veins.

Fig. 15 c. Diastole I. The sudden fall in pressure inside the fascia would tend to cause a backward flow from the popliteal vein into the crural veins. This is prevented by the normal popliteal valves.

The pressure in the subcutaneous veins is by now higher than in the deep veins. A central flow through the communicating veins has now become possible. Blood pressure and volume are restored by a retrograde flow through the varicose subcutaneous veins.

As soon as the pressure inside the fascia cruris exceeds that in the popliteal and subcutaneous veins the phase of slow steady flow has become reestablished.

of the back-flow test during operation in a large part of the present material. A satisfactory correlation was found (Table IV).

Thus, the evidence at hand is in favour of the assumption that the direction of the blood stream through the incompetent ankle-perforating veins is towards the periphery during Diastole II. The rate of flow must be imagined to be rather slow and the driving force modest.

B. Systole.

At the onset of muscular contraction the tension in the subfascial compartment rises steeply towards the maximum values of 700—1200 *mm* H_2O. The highest values are found at the level of the ankle-perforating veins. The blood is squeezed upwards into the popliteal vein and outwards through the incompetent communicating veins into the saphenous veins. The outward expulsion of blood is very strikingly demonstrated during operation. Even when the leg is elevated, muscular contraction will effect a spurt of blood from the cut central end of the incompetent ankle-perforating vein.

The standard technique in dynamic intraosseous phlebography gives no direct information as to what happens to the contrast medium during Systole, but the "back-flow test" demonstrates how the very high systolic subfascial pressure is directly transmitted into the subcutaneous network in the ulcer area through the pressure leak formed by the incompetent ankle-perforating vein.

C. Diastole I.

At the cessation of Systole the intramuscular tension presumably falls to at least the same low level as has been measured in the normal extremity at rest (50—110 mm H_2O). As the supporting external pressure falls away, the pressure in the by now partially empty deep trunks falls below the hydrostatic pressure in the popliteal vein, and the popliteal valves close. The pressure in the subcutaneous veins is now higher than in the deep veins and a central flow through all the communicating veins — competent as well as incompetent — becomes possible.

The central flow from the periphery and the increasing inflow from the muscles fill the deep trunks, where the pressure immediately will begin to rise again.

The second exposure in dynamic intraosseous phlebography shows that the contrast medium, which formerly was found in the incompetent ankle-perforating veins and the adjacent parts of the subcutaneous

system, has been "sucked" into the deep veins, often leaving both the subcutaneous and the communicating veins free of contrast. As this is seen in Diastole I after the third muscular contraction, one must imagine that, during the three cycles which separates the two exposures, a continuous to-and-fro flow has taken place between the deep and subcutaneous systems via the ankle-perforating veins.

The second exposure shows retention of contrast in the deep trunks, but the segments below the level of the incompetent ankle-perforating veins are as a rule empty (Figg. 16, 17, 18). The column of contrast medium in the deep veins has moved upwards into the most proximal parts and into the popliteal vein. Thus, while the function of the venous pump is seen to be impaired, muscular activity still seems to have some effect on the transport of blood.

The importance of the deep crural valves for the pump mechanism in the lower leg.

It is very interesting to notice, that postthrombotic valvular destruction in the deep veins of the leg does not seem to add to the degree of impairment of the venous pump in Group II type A, as long as the popliteal valves are normal.

Group II type A contained 27 patients with phlebographical signs of postthrombotic valvular destruction in the deep crural veins and a history that confirmed the x-ray diagnosis. Clinically these patients were of the same type as the majority of the group, which had an "idiopathic" incompetence of the subcutaneous and communicating veins. The percentage of ulcers was identical and the subjective complaints did not differ in character (Table II and III).

Phlebographically the degree of impairment of the venous pump was identical. (Fig. 18).

In this connection it is of interest, that the few patients in Group I type B, who all had a history of deep thrombophlebitis, which was confined to the deep crural veins, had a phlebographically normal function of the venous pump. The communicating veins were competent.

The vast majority of the phlebograms in Group II type A with sequels of deep crural thrombophlebitis had normal popliteal valves. (Table II).

Thus it seems as if the function of the venous pump in the lower leg is dependent upon competent valves at the outlets from the musculo-fascial compartment (the communicating veins and the popliteal veins). Incompetence of the ankle-perforating veins will effect an impairment of the function of the venous pump. Valvular destruction in-

side the musculo-fascial compartment does not add to the degree of impairment, as long as the valves in the popliteal vein are competent. What will happen when not only the crural valves, but also the popliteal valves are destroyed will be discussed when dealing with the Group II type B.

Figg. 15 a, b and c represent the cyclus Diastole II, Systole and Diastole I in Group II type A.

Figg. 16, 17 and 18 are typical phlebograms from Group II type A.

CHAPTER X.

THE VENOUS RETURN FROM THE LOWER LEG IN PATIENTS WITH VARICOSE VEINS, INCOMPETENT COMMUNICATING VEINS AND TOTAL VALVULAR DESTRUCTION IN THE DEEP CRURAL AND THE POPLITEAL VEINS.

(Group II type B)

All the patients in Group II type B had roentgenological signs of previous deep thrombophlebitis. In the overwhelming majority the thrombotic process had involved the popliteal as well as the deep crural trunks (Table II). These patients had, as a group, severe symptoms of chronic venous insufficiency and the percentage of leg ulcers was very high (81 per cent) (Table III).

Dynamic intraosseous phlebography failed to show any signs of an active pump function after muscular contractions, and in the few patients investigated by means of serial phlebography, the contrast medium was seen to flow slowly upwards through all the veins of the leg. The subcutaneous veins became filled via the incompetent ankle-perforating veins (which always were present) and the rate of flow was quite unaffected by muscular activity.

Intramuscular and subcutaneous tissue tension. Until further evidence is at hand it will be assumed, that the intramuscular and subcutaneous tissue tensions in patients with venous insufficiency in Group II type B are identical with those measured in normal subjects. A priori one would assume that the induration and ulceration, so common in this group, would change the subcutaneous tissue pressure.

Venous pressure measurements. The pressure in the saphenous veins corresponds at all levels to the calculated hydrostatic pressure of a column of blood reaching to the level of the heart. Where varicose veins are present (and practically all the patients in Group II type B had varicose subcutaneous veins) the pressure in the saphenous veins is left unchanged by muscular activity. The pressure in the popliteal vein fluctuates about a mean value corresponding to the hydrostatic pressure at that level. The deviations from the mean value are modest.

The pressure in the deep veins of the lower leg and in the ankle-perforating veins has never been measured.

A. Diastole II.

The slow upward flow of contrast medium which was seen in serial phlebography takes place with a speed of 1—2 cms per second. At the level of the incompetent ankle-perforating veins some contrast is shunted out into the subcutaneous veins, where the proximal flow is seen to continue at the same speed as in the deep veins.

In the group under discussion here, all valves were destroyed in the contrast-filled deep veins of the leg (except in the lower fifth of the leg, where normal valves as a rule could be seen in the slender deep trunks) and in the popliteal vein. The hydrostatic pressure must therefore be assumed to be fully effective at all levels during Diastole II.

In the first phlebographic exposure the contrast medium is seen to fill the subfascial veins, the ankle-perforating veins and the subcutaneous network and the contrast has reached approximately the same level in the deep and subcutaneous veins.

The difference in tissue support would account for the outward flow through the incompetent communicating veins and the peripheral direction of the blood flow can be verified by the back-flow test.

B. Systole.

At the onset of muscular contraction, the tension in the musculofascial compartment rises steeply, in the lower third of the soleus to about 1200 mm H_2O. This pressure is higher than the hydrostatic pressure at the same level in the semierect position adopted in dynamic intraosseous phlebography. The result is an expulsion of blood from the deep crural veins through the popliteal and the incompetent communicating veins. This is easily demonstrated by the back-flow test as re-

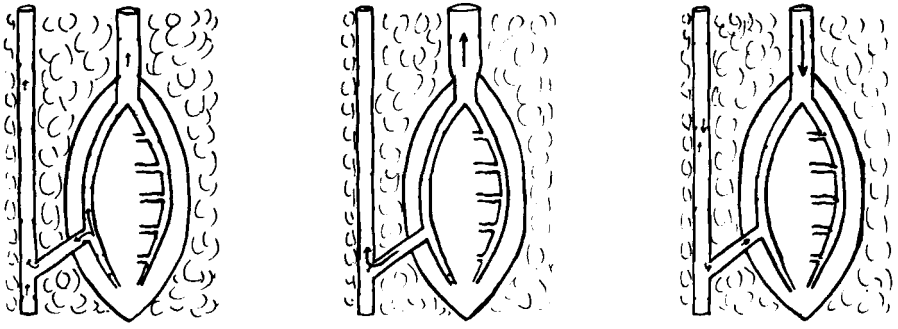


Fig. 19 a, b and c. represent in schematic form the function of the venous pump in the lower leg during Diastole II, Systole and Diastole I in an extremity with varicose veins, incompetent communicating veins and total deep valvular destruction, including the popliteal valves. (*Group II type B*).

Fig. 19 a. Diastole II. During the phase of slow steady flow the blood passes upwards driven by the vis-a-tergo. Owing to the difference in pressure blood is driven outwards from the deep veins via the incompetent communicating veins into the subcutaneous system. The pressure gradient is modest and the rate of flow slow. Hydrostatic forces are unhindered in all systems.

Fig. 19 b. Systole. The steep increase in pressure caused by muscular contraction drives the blood in the deep trunks out of the fascial compartment into the popliteal veins, and — via incompetent communicating veins — out into the subcutaneous system.

Fig. 19 c. Diastole I. Immediately after muscular activity the pressure inside the fascia cruris falls abruptly. This would in a normal extremity cause the popliteal valves to close. When the popliteal valves (together with the deep crural valves) are incompetent, the inversed pressure gradient will cause a rush of blood backwards into the deep crural veins. At the same time the by now higher pressure in the subcutaneous veins will effect a central flow into the deep veins through the communicating veins.

Thus the initial low pressure inside the fascia will rise very rapidly until the pressure exceeds that of the popliteal and subcutaneous veins. Diastole II is quickly reestablished and Diastole I must be of very short duration. The amount of blood transferred from the subcutaneous into the deep veins must consequently be very small.

gards the incompetent ankle-perforating veins, and the slight rise in pressure in the popliteal vein at this moment is in all probability caused by the inrush of blood from the crural veins.

The standardized dynamic intraosseous method of phlebography does not show what happens to the contrast medium during systole.

As in all other forms of venous incompetence hitherto investigated, a central flow of blood through the communicating veins does not seem possible during Diastole II and Systole in Group II type B.

C. Diastole I.

At the cessation of contraction the deep veins inside the musculofascial compartments in the lower leg are imagined to contain much less blood than before. The high intramuscular tension falls abruptly to at least the resting values of 50—110 mm H_2O. The immediate result must be a sharp fall in venous pressure. At this moment normal popliteal valves would close, but in these extremities the popliteal valves are absent (Table II) as are the valves in the deep veins of the lower leg.

If we imagine the deep crural veins to be empty or nearly empty and the pressure much lower than the pressure in the popliteal vein, the unhindered hydrostatic force will cause an immediate rush of blood backwards from the deep veins in the thigh and at the moment that an unbroken column of blood is established throughout the deep system, the venous pressure must become equal to the calculated hydrostatic pressure at all levels. The only imaginable hindrance for the establishment of an unbroken column of blood where the valves are absent is a blockage caused by intimal contact in an absolutely empty segment of vein. In theory this may occur, but cannot be expected to last for more than an exceedingly short time.

The evidence at hand thus indicates that Diastole I is exceedingly short in Group II type B, and that the state of steady flow with full hydrostatic pressure at all levels (Diastole II) is established almost at once following muscular relaxation.

In normal subjects and in Group I type A and Group II type A, Diastole I was found to be the only phase in which a central flow was possible through the communicating veins into the deep veins. If it exists at all in Group II type B, Diastole I must be so short that the volume of blood exchanged between the subcutaneous and deep veins of the leg is almost negligible. In these patients the saphenous varicosities will not empty during the Perthes test.

In normal subjects and in other types of chronic venous insufficiency discussed above, Diastole I was seen to be favorable for the outflow from the muscular veins into the deep trunks. In Group II type B the period of low pressure in the deep trunks must be extremely short and the flow in the minute muscular veins and venules must be imagined to take place against a practically constant high pressure.

The second exposure in dynamic intraosseous phlebography shows what has happened to the contrast medium after three cycles of contraction and relaxation. Nothing much seems to have happened. The contrast is found in the deep veins, the communicating veins and the subcutaneous veins and has reached a higher level than in the first exposure, but not higher than could be expected where the vis-a-tergo is the sole effective driving force (Figg. 21, 22).

Thus a total destruction of the valves in the communicating veins and the deep veins of the leg and thigh (at the outlets from the musculo-fascial compartment) causes a total destruction af the venous pump.

Figg. 19 a, b and c represent the cyclus Diastole II, Systole and Diastole I in Group II type B.

Figg. 21 and 22 are phlebograms of Group II type B.

CHAPTER XI.

THE VENOUS RETURN FROM THE LOWER LEG IN PATIENTS WITH VARICOSE SAPHENOUS VEINS AND IDIOPHATHIC INCOMPETENCE OF THE DEEP VEINS. COMPETENT COMMUNICATING VEINS.

(Group III).

All the patients with phlebograms of this pattern had varicose saphenous veins. The number of leg ulcers in the group was low (Table III) and the percentage almost identical with that found in Group I type A (simple varicose veins). There was, however, a striking difference in the character of the subjective complaints. A very common symptom in Group III was the severe "bursting pain" in the leg, which was never met with in patients with simple varicose veins in the present material (Table III).

The phlebograms of Group III showed normal deep veins of the leg, in so far as the contours of the veins and the valvular structure did not show any signs of postthrombotic changes. The diameter of one or several of the deep veins was, however, much wider than seen in the normal control material. The diameter of the fibula, measured ten cms below the tip of the capitulum was taken as a means of comparison. In the normal control material the deep veins generally had a diameter

between one third and half that of the fibula, while the majority of the phlebograms in Group III showed deep veins which at some points had diameters equal to or wider than the fibula.

The communicating veins in the leg were phlebographically competent. Incompetent communicating veins were found together with enlarged deep veins, but this mixed group was listed under Group II type A.

In most cases the diameter of the popliteal veins was "rather wide", but no attempt was made — in the present material — to determine the normal caliber of this vein.

In the second exposure, contrast was seen in one, several or all deep veins, but sometimes only in an apparently especially wide segment of a vein. As this phenomenon never was seen in the normal control material it was regarded as a sign of incompetence of the venous pump mechanism. The degree of incompetence varied from complete destruction to slight impairment.

Subcutaneous and intramuscular tissue tension. Until further evidence is at hand the subcutaneous and intramuscular tissue tensions will be considered identical with the tensions measured in normal subjects.

Venous pressure measurements. Venous pressure in the varicose subcutaneous channels is equal to the hydrostatic pressure of a column of blood reaching to the level of the heart. It may be assumed, that the pressure in the popliteal vein behaves as in normal individuals, patients with simple varicose veins and patients with total valvular destruction, that is, it fluctuates about a mean value roughly equal to the hydrostatic pressure at this level.

The pressure in the communicating veins and in the veins inside the musculo-fascial compartment has never been measured.

A. Diastole II.

The first exposure shows that the contrast medium has filled the deep veins of the leg and sometimes the popliteal vein. Contrast is not seen outside the fascia cruris.

Diastole II is the phase of slow steady flow. The valves are open in the subcutaneous as well as in the deep veins and consequently the pressure at any given level must be equal to the weight of a column of blood reaching to the level of the heart. The difference in tension in the surrounding tissues tends to make the effective pressure in the deep veins higher than in the subcutaneous varicosities, with the result that

the competent valves in the communicating veins stay closed. A central flow through these veins is therefore not feasible during Diastole II.

B. Systole.

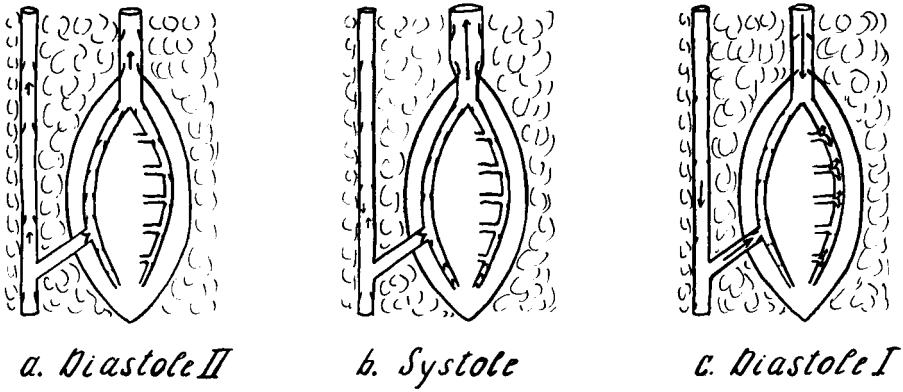
At the onset of muscular contraction the tension in the soleus and anterior tibial muscles was observed to rise steeply from the resting level of 50—100 mm H_2O to a maximal figure of 1200 mm H_2O in the lower third of the soleus. These measurements came from normal extremities.

Wells, Youmans & Miller (1938) who investigated the maximal contraction pressure in different muscles, found that the level of tension reached during maximal contraction depended upon the structure of the fascial sheath. The soleus and the tibialis anterior muscles have strong fascias, while the gastrocnemius and the rectus femoris, where the rise in intramuscular tension was much more modest (Fig. 6), have less well developed fascial coverings.

The reason why some deep veins of otherwise normal appearance seem to be dilated and empty poorly after muscular activity, is still unknown. Several possible explanations offer themselves: One possibility is, that the tone of the vein wall is lowered in the same manner as seen in the early stages of primary saphenous varicosities *(Arnoldi* 1957 and 1961 a). The dilation might also be the result of a shrinking of the muscular tissue inside the fascial sheaths, the loss of support giving rise to a secondary widening of the veins and a less effective expulsive power in the pump section. — A relaxation of the fascial structures would, according to the investigations mentioned above, bring about a fall in maximum tension during contraction and possibly give room for a secondary widening of the deep veins. Finally, a severe phlebosclerotic process may lead to a state with wide rigid veins which cannot be compressed by the surrounding muscles. A phlebosclerotic degeneration is always preceeded by a loss of elastic tissue.

Research in this field is non-existing. The theory of a hormonally induced loss of tone is perhaps supported by the fact that many of the patients in Group III gave a history of increasing complaints at the time of menstruation. Visible muscular atrophy was never demonstrated in the extremities with phlebograms of this type.

The theory of fascial hypotrophy or relaxation as a factor in venous insufficiency has recently led to the development of a new surgical approach in chronic venous insufficiency, in which the fascia over the



Figg. 23 a, b and c. represent in schematic form the function of the venous pump in the lower leg during Diastole II, Systole and Diastole I in an extremity with varicose veins and »primary deep incompetence«. Competent communicating veins. (*Group III*).

Fig. 23 a. Diastole II. In the phase of steady flow the valves are open and the hydrostatic forces have full effect in the deep as well as in the varicose superficial veins. The driving force = the vis-a-tergo. The slightly higher effective pressure in the deep system keeps the valves in the communicating veins closed.

Fig. 23 b. Systole. The high pressure inside the fascia cruris expells the blood via the popliteal vein. The distal route and the route through the communicating veins are blocked by competent valves.

Fig. 23 c. Diastole I. (could be explained in the following way): At the end of muscular contraction the pressure inside the fascial compartment falls steeply. This makes a filling from the varicose subcutaneous veins possible. The resulting refilling af the deep trunks reaches a point where the distension of the wide deep veins overcomes the competence of valves, including the popliteal valves. As soon as an unbroken column of blood is established the hydrostatic forces have full effect throughout the system.

Should the wide deep veins be the seat of phlebosclerotic changes, the shortening of Diastole I would seem to become even more pronounced.

gastrocnemius muscles is tightened by duplication *(Askar* (1961). The idea was first proposed by *Gullmo* (1959). One of the beneficial effects of the time honoured treatment with pressure bandages could be the establishment of an extra "fascial" sheath over the muscles of the lower leg.

The deep veins in the lower fifth of the leg were as a rule of normal caliber in Group III, and the valves in the communicating veins were competent. The result of muscular contraction must therefore be that the blood is driven upwards into the popliteal vein. Standard dynamic

intraosseous phlebography does not show, what actually happens to the contrast during Systole.

C. Diastole I.

As the muscles relax the high intramuscular pressure falls to about the same level as measured during Diastole II, and the pressure in the deep veins of the leg presumably falls parallel with the intramuscular tension. The pressure in the popliteal vein falls slightly, but the result will be a lower pressure below the popliteal valves than above. If the popliteal valves are normal they will close immediately.

The apparent dilation of the deep veins in Group III may involve a segment of a vein or all the deep veins visible in the phlebogram. Should the popliteal vein be involved together with the deep crural veins, one may imagine a state in which the valves no longer are competent. When an unbroken column of blood is established the pressure in the deep veins will become equal to the full hydrostatic pressure of a column of blood reaching to the level of the heart.

Diastole I will probably never become as short as in Group II type B, as the valves must be supposed to be competent during the first part of the period of central flow through the communicating veins and from the muscular tributaries, until a certain degree of dilation has been reached and contact between the valvular rims no longer is possible.

In an extremity of this type the period most favorable for the central flow from the smaller muscular veins into the deep crural trunks is shortened, as was found to be the case in Group II type B.

Extremities with total postthrombotic valvular destruction (Group II type B) differed considerably from those with primary deep incompetence (Group III). While 81 per cent of the postthrombotic patients had leg ulcers, this was only seen in 14 per cent in Group III (Table III). All the postthrombotic legs in Group II type B had incompetent ankle-perforating veins, but in Group III the communicating veins were competent.

A look at Table III will show that the most serious subjective complaints, the severe bursting pains in the lower leg, are almost exclusively found together with total valvular destruction (Group II type B) or deep idiopathic insufficiency (Group III). Most of the patients in Group II type A with this symptom had deep veins reminiscent of Group III.

The theory does not seem too far fetched that the bursting pains in

the leg are caused by the practically constant high pressure in the deep veins of the leg (shortening or disappearance of Diastole I).

The severe subjective complaints in the leg not infrequently persist after radical surgical treatment of varicose veins and incompetent communicating veins. In the author's experience these symptoms are as a rule relieved after resection of the popliteal vein.

Isolated retention of contrast in a dilated section of vein is difficult to explain. A small "reservoir" may be a variation of normal physiology, just as faint traces of contrast confined to the valvular sinuses are considered normal. Retention of contrast in a larger segment of wide caliber is, however, definitely abnormal, and was never met with in the normal control material. The cause of this local retention of contrast medium is unknown, and an attempt at explanation is deemed futile, as long as information about the venous and intramuscular pressure in the musculo-fascial compartment are wanting in this type of chronic venous insufficiency.

Figg. 23 a, b and c represent the cyclus Diastole II, Systole and Diastole I in the typical cases of primary deep insufficiency.

Figg. 24 and 25 are phlebograms from Group III.

CHAPTER XII.
THE AUTHOR'S CONCEPTION OF THE STRUCTURE OF THE VENOUS PUMP.

Fig. 2 represents the conception of the structure of the venous pump which is prevalent in the physiological literature at present.

The evaluation of the conditions for the venous return described in the present paper, which is based upon the phlebographic examination of the function of the three segments of the pump inside the deep crural fascia, has together with the results of measurements of venous pressure and tissue tension, given rise to the conception of the structure of the venous pump in the lower extremity, which is illustrated by Fig. 26.

The central stem (popliteal, femoral and external iliac veins) which is sparsely valved and for the most part embedded in fat or in loose

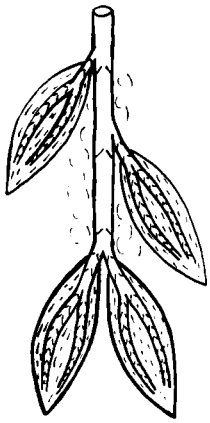


Fig. 26. Schematic presentation of the author's conception of the structure of the venous pump in the lower extremity.

The musculo-venous pump in the lower extremity is represented as a set of pump sections attached to the central collecting vein (popliteal-femoral). Each pump section consists of a muscle or set of muscles, ensheathed by fascia together with the vessels. Four sections are found in the lower leg (Fig. 1). The upper pump section in Fig. 26 might represent the section drained by the deep femoral vein (see text).

connective tissue, has a passive collecting function, while the densely valved veins inside the musculo-fascial compartments are more actively engaged by the pump mechanism. The practically unaltered mean pressure in the central stem during muscular activity and the great variations in pressure which take place in the veins of the pump sections, are in favour of this conception.

The loose support of the environment of the central stem and the elasticity of the vein wall enable these veins to act as a buffer for the great variations in pressure, which are found where the pump sections emerge into the collecting veins.

The function of the venous pump of the lower extremity as a whole is facilitated by the alternating action of the pump sections. The few valves in the central stem are usually placed below the entrance of the veins from the pump sections.

It seems as if the fascial structure, and thus the force of the expulsive power of the pump segment, is finely adjusted to the hydrostatic pressure at each level under normal conditions (Fig. 6), and that further investigations on the musculo-fascial structures would be worth while in patients with chronic venous insufficiency.

The phlebographic examination of the function of the pump sections in the lower leg demonstrated the importance of the valves at the outlets from the musculo-fascial compartments. The pump function might become deficient from a leak of blood through the incompetent communicating veins out into the subcutaneous veins. Lack of function

of the valves at the upper outlet in the popliteal vein (together with incompetence of the crural valves) results in a very grave impairment of the pump function.

On the other hand it did not seem as if the valves inside the musculofascial unit are of importance for the function of the pump section as a whole. The main function of these valves is probably — as *Jäger* (1936) suggested — to act as a protection for the venules and capillaries against too violent variations in venous pressure.

The vein valves are only one of the factors determining the function of the pump mechanism, but at present it is the only factor which has been studied to any extent. The impaired function in Group III suggests that a disturbance in other factors necessary for the perfect function of the venous pump may be of importance.

PART III.
THERAPEUTIC CONCLUSIONS.

The analysis of the phlebographic material (Arnoldi 1961 a and b) and the evaluation of the function of the venous pump in the various forms of chronic venous insufficiency presented above, has necessarily influenced the author's conception of the rational treatment of venous disorders in the lower extremity. As was to be expected the conclusions arrived at, have confirmed established ideas to a large extent, but they have also emphasized the importance of an exact diagnosis of the type of insufficiency in the individual case. The phlebographic investigation has illuminated the importance of the extent of the initial attack of deep thrombophlebitis for the frequency and severity of the late postthrombotic sequelae; and it has perhaps increased our understanding of the origin of the severer subjective complaints in chronic venous insufficiency.

CHAPTER XIII.
THE ACUTE STAGE OF DEEP THROMBOPHLEBITIS.

Signs of postthrombotic changes in the deep veins were found in Group I type B, Group II type A and Group II type B.

In Group I type B the venous pump was seen to function in a normal way (definition) and the popliteal valves were as a rule intact.

In the postthrombotic cases in Group II type A the signs of postthrombotic changes were confined to the deep crural veins, while the popliteal valves were normal in the large majority of cases (Table II).

The impairment of the function of the venous pump was due to incompetent communicating veins, and the postthrombotic valvular destruction did not seem to add to the degree of incompetence. The percentage of leg ulcers in the "idiopathic" and postthrombotic material in Group II type A was identical.

In Group II type B the thrombotic process had involved the popliteal as well as the crural valves. The phlebograms showed a total destruction of the function of the venous pump, and the percentage of ulcers was very high (81 per cent). (Table III).

Thus it seems that a thrombotic process confined to the deep veins of the lower leg has a better long term prognosis than a process, which also involves the veins at a higher level. Our treatment of acute deep thrombophlebitis should recognize this fact.

The diagnosis of acute deep thrombophlebitis should be established at the earliest possible stage, and the classical symptoms of phlegmasia should not be allowed to develop. The therapeutic procedures should be applied with an eye to a speedy prevention of the proximal progress of the thrombus.

Table II is of interest as it shows the history of the treatment in the acute phase of thrombophlebitis in the patients in the postthrombotic material in Group II types A and B. While two thirds of the patients in Group II type A had been treated with heparin, only eight per cent of the patients in Group II type B had had this treatment. Dicoumarol did not seem to have influenced the course of the disease in any favorable direction when employed as the only anticoagulant.

It is of course realised that the material presented in Table II is small. The evidence of the table, such as it is, does, however, stress the importance of the choice of anticoagulants. With a slow acting anticoagulant of the dicoumarol type, there is a definite risk that the ascending thrombotic process may reach the important popliteal valves before a full effect on the coagulability of the blood has been obtained, with the result that the risk of grave chronic sequelae will be considerably increased.

It is felt necessary to stress this point in view of the tendency in some clinics to rely on preparations of the dicoumarol type as the sole anticoagulant in cases of manifest deep thrombophlebitis. It may well be that no significant difference can be found in the rate of embolic incidents during the acute phase, whether heparin or dicoumarol is used. Fatal embolism is a comparatively rare occurrence in the days of early mobilisation and prophylactic anticoagulants. Grave postthrombotic

sequelae have, however, been found in the majority of the extremities which have been followed through the years after the acute attack.

The evidence of the present investigation indicates that the severity and frequency of the postthrombotic sequelae may be reduced, when the thrombotic process is confined to the veins inside the musculofascial compartment. Only heparin acts quickly enough to stop the process before it reaches the popliteal valves.

CHAPTER XIV.
SURGICAL THERAPY IN CHRONIC VENOUS INSUFFICIENCY.

When considering the therapeutic conclusions drawn from the present evaluation of the function of the venous pump it is practical to distinguish between the subcutaneous manifestations (oedema, induration and ulceration) and the subjective complaints, especially the graver forms (the "bursting" crural pains).

A. Ulcus cruris venosum.

According to the prevailing conception gravitational oedema is the first step in the development of the sequence: oedema — induration — ulceration. A constant high pressure in the venules and the venous ends of the capillaries is responsible for a filtration oedema, which when persistent, causes a proliferation of fibrous tissue and a diminution of the arterial supply in the subcutis. The increasing fibrosis leads to induration and devitalisation and finally even slight traumatisation will cause a breakdown in the skin and subcutis — ulceration. Healing will not occur as long as the venous pressure remains high in the ulcer area and the oedema persists.

It is strikingly demonstrated during operation that the pathologic processes present in the tissue in the ulcer area are confined to the skin and subcutis. Under the deep fascia the muscles look completely normal to the naked eye. It has been shown that the increase in leg volume seen when oedema is present is restricted to the subcutis (Arnoldi 1959).

The high venous pressure — hydrostatic or contraction pressure (Systole) is transmitted to the capillaries in the ulcer area by way of the varicose saphenous veins and the incompetent ankle-perforating veins.

Table II shows that incompetent communicating veins (practically always of the ankle-perforating type) were present together with saphenous varicosities in 119 extremities with ulcers. Only 20 cases were found without phlebographically demonstrable incompetence of the communicating veins. These cases all had varicose saphenous veins. Incompetence of the ankle-perforating veins was found to embarrass the function of the venous pump. Simple varicose veins did not have this effect.

The rational surgical treatment of oedema, induration and ulceration seems to be a radical elimination of the pressure leaks formed by the incompetent saphenous and communicating veins. In the author's experience this is best done by radical exstirpation of the varicose saphenous veins, combined with operative closure of the incompetent ankle-perforating veins a. m. Cockett.

B. Surgical treatment of severe pains in chronic venous insufficiency.

It has been the author's priviledge, while working with Professor Gunnar Bauer in Mariestad, to observe the effect of popliteal resection on a considerable number of patients with the more severe forms of chronic venous insufficiency. *Bauer* (1959) has reported on the results of popliteal resection as a treatment for leg ulcers. According to the author's much more limited experience with this type of treatment the most striking effect of popliteal resection is the often prompt disappearance of the sometimes invalidating bursting pains felt diffusely in the leg, pains that sometimes have led the patients to ask for amputation.

The effect and the rationale of the popliteal resection is but imperfectly understood, and the various explanations offered do not seem entirely satisfactory.

As seen from Table III bursting pains were as a rule found together with deep venous incompetence, and in the evaluation of the function of the venous pump it was proposed (Chapter XI) that these pains might be due to the disappearance or shortening of Diastole I, that is to a practically constant high pressure in the deep crural veins and their muscular tributaries.

Phlebographic examinations by means of the dynamic intraosseous method are as yet comparatively few, as regards the result of popliteal resection upon the function of the venous pump. While some phlebographic examinations have indicated a better function of the venous pump after operation, others have shown an unchanged picture. No correlation between the effect of the operation on the clinical signs and symptoms and changes in the phlebographic pattern has yet been demonstrable.

When the popliteal vein is resected the blood must find its way upwards through the collaterals which are supposed to have normal valves and are of a smaller caliber. (That an extensive potentiel collateral system exists is evident from the fact that radical saphenektomy, ligation of incompetent communicating veins and popliteal resection may be performed as a one-stage operation without noticeable postoperative oedema).

If these premises are correct, the relief of crural pain could be explained in this way: After popliteal resection the blood will be squeezed upwards through the "para-popliteal" collaterals (and outwards through the incompetent ankle-perforating veins, unless ligation is done at the same time). At the cessation of Systole the pressure in the musculo-fascial compartments in the lower leg will fall below the pressure in the femoral veins, and the competent valves in the collaterals will prevent a backward rush of blood. The elimination of the pressure gradient between the femoral and crural veins will be retarded. The effect of popliteal resection would thus be the reestablishment of a Diastole I.

A thorough phlebographic evaluation of the effect of popliteal resection on the function of the venous pump will not be possible, until a material is at hand in which popliteal resection has been performed upon patients belonging to Group II type B, who have had a Cockett's operation for incompetent ankle-perforating veins done at an earlier stage, and in whom the result of this operation has been controlled phlebographically before the popliteal vein is resected.

The author would reserve popliteal resection to cases with severe pains in the leg, caused by chronic venous insufficiency, and preferably when the patient describes the pain as being of a diffuse bursting character. In the author's experience ulceration is better treated by elimination of the subcutaneous pressure leaks: saphenektomy and ligation of incompetent communicating veins.

CHAPTER XV.
INDICATIONS FOR DYNAMIC INTRAOSSEOUS PHLEBOGRAPHY.

The author's experience with dynamic intraosseous phlebography encompasses more than 600 examinations. In two of these cases a necrotic breakdown of the skin occurred over the lateral malleolus, which was used as site of injection in the first phlebograms. No other complications have been met with. At this point it should be emphasized that intraosseous injections must be performed under scrupulously aseptic conditions. Cases of osteitis and ostemyelitis have been reported following all forms of intraosseous injections *(Søndergaard* (1946), *Heinild, Søndergaard & Tudvad* (1947). Our experience shows, however, that if performed under aseptic conditions, the intraosseous method has a very low percentage of untoward complications.

The clinical evaluation of the extremity with chronic venous insufficiency is often not at all easy, when the patient is seen for the first time.

In the large majority of patients with simple varicose veins the diagnosis is simple enough, and the presence of typical incompetent ankle-perforating veins should be demonstrated without too much difficulty in a great many cases.

The presence and localisation of incompetent ankle-perforating veins and atypical communicating veins may, however, be very difficult to demonstrate in patients with severe subcutaneous manifestations (oedema, widespread induration or large ulcers). If the surgeon wishes to limit the extent of his exploratory incisions, a preoperative phlebographic demonstration of the incompetent communicating veins is desirable.

A history of deep thrombophlebitis may be elicited in most patients with postthrombotic changes in the deep veins, but both the history and the clinical examination are often unable to decide how far the

thrombotic process has proceeded upwards. Apart from the academic interest the question is important to the surgeon, who may consider a popliteal resection as a remedy for the patients severe pains, or who may wish to treat the venous ulceration in this way. A popliteal resection is, in the author's opinion, not indicated where the popliteal valves have a normal function, but may be the only effective therapy in cases with total valvular destruction. In such cases a phlebographic evaluation is essential.

Legs with simple varicose veins, an occasional ulcer and moderate oedema, but with severe and often bursting pains, are quite often the seat of a primary idiopathic incompetence of the deep veins. The only possible way to come to a correct diagnosis is by means of a functional type of phlebography.

In general it may be said, that to the surgeons who treat chronic venous insufficiency schematically, the need for phlebography is really non-existent, while it is essential to those who wish to make a proper diagnosis and to treat their patients accordingly.

What type of phlebography should be preferred? This question cannot be answered definitely as all depends upon the wishes of the examinator. If a total filling of all the veins of the lower extremity is desired, the intraosseous method should not be used, as it will only show the deep veins of the leg (and not invariably all the deep veins) and the incompetent communicating veins of the lower leg. The concentration of contrast is generally too weak to allow any clear-cut morphological diagnosis above the popliteal vein.

Finally, the author has become convinced that the intraosseous technique, as performed in the present dynamic form of phlebography is not suitable for the demonstration of acute thrombotic affections in the lower leg. For this purpose a method should be preferred, which can be depended upon to show all the subfascial veins.

The limited area of contrast filling and the possibility of a shift of contrast medium from one venous system to another makes the intraosseous method ideally suited for a functional evaluation. If this type of examination is preferred, dynamic intraosseous phlebography is — in my experience — superior to any other known method.

FUTURE PROBLEMS

The assessment of the structure and the function of the musculovenous pump in the lower leg which has been outlined in this paper is founded upon facts collected from the literature, from the results of the author's own investigations by means of the dynamic intraosseous method of phlebography and upon the undefineable fund of clinical information which comes from ten years of active interest in chronic venous insufficiency. Even so, it is only too clear that further research is needed in order to fill the many gaps in our knowledge regarding the physiology and pathophysiology of the venous return from the lower extremity.

The last paragraphs of the present paper will therefore be concerned with some of the problems which arose in connection with this attempt to formulate a new conception of the structure and function of the venous pump, problems which must be solved before this theory is either accepted or refused.

Subcutaneous tissue tensions: In this paper it was assumed that the values of subcutaneous tissue tensions, and consequently the environmental support of the superficial veins, were identical in normal subjects (the reported findings) and in patients with various types of chronic venous insufficiency. This is, however, by no means certain. There are indications that the subcutaneous pressure may rise with developing oedema, and it would seem quite probable that the grave changes in the skin and subcutis, which are seen in the severer forms of venous incompetence, will influence the subcutaneous tissue tension considerably.

Investigations into tissue tensions in patients with venous diseases are as yet nonexistant.

Intramuscular tissue tensions: It has been assumed that the intramuscular tissue tensions are identical in normal subjects (reported

findings) and in patients with chronic venous insufficiency, both at rest and during muscular contraction. This may be true, but information in this respect in nonexistent.

It seems of interest that this question should be solved, especially with reference to the form of venous incompetence described in the study as "Group III" or idiopathic deep incompetence. The underlying pathological disturbance in this form of pump deficiency is as yet wholly unknown. The short discussion in Chapter XI indicates the need for further investigations, not only as regards the venous component of the pump, but also into the muscular physiology and the fascial quality in these extremities.

Venous pressure measurements: The essential unit in the new conception of the structure of the venous pump is the muscle (or group of muscles), ensheathed by a common fascia and drained by a set of closely valved intramuscular veins into a sparsely valved extramuscular collecting vein.

This "segmental" conception of the structure is based on anatomical relations, upon the results of an analysis of functional intraosseous phlebography and upon the reported values of venous pressure measurements in the subcutaneous and femoral veins.

The description of the pressure gradients between the various parts of the venous drainage system during Diastole II, Systole and Diastole I depends, however, too much on circumstantial evidence, as long as the pressure variations inside the fascial compartment are as completely unknown as at present. Furthermore the influence of the incompetent ankle-perforating veins upon the venous pressure in the subcutaneous veins in the lower leg has never been investigated. Exact information not only regarding the absolute values of pressure in each component part of the venous system in the lower leg, but also — and especially — regarding the varying pressure gradients between these parts during the contraction/relaxation cyclus, are needed before the present structural and functional conception can be accepted as more than a hypothesis.

The results of such investigations will shortly be published in this journal.

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