THE PATHOLOGY OF GRAVITATIONAL SYNDROMES IN THE LEG AND THE PLACE OF STRIP GRAFTING IN TREATMENT

I. THE PHYSIOLOGY OF VENOUS RETURN

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This paper is the first of a series dealing with the problems and syndromes associated with the gravitational effects in the lower limb. In this connexion it is important to consider the normal mechanisms of venous return and the problems arising with alterations in posture.

Since Harvey's concept of a closed vascular system over 300 years ago, not very much has been added to our knowledge of this important subject.

The concept of valves was recognized by Pierre Dionis (c. 1668-1718), Surgeon to the Queen of France and to the Empress Maria Theresa. He stated that 'valves form steps which serve to help the ascension of the blood and so facilitate its return to its source'.[1]

Jean Louis Petit (1674-1750), the first director of the Academie de Chirurgie of Paris, stated that anything obstructing the rising of the blood in the veins was the principal cause of varicosities. He also noted that pregnancies were important, and prolonged standing and tight garters were both mentioned.

It was Sir Benjamin Collins Brodie (1783-1862) who first described and demonstrated the reverse flow of blood in a varicose saphenous vein; so the Trendelenburg test is really Brodie's test, for Trendelenburg followed almost 50 years later. Certain of Brodie's sound and fundamental observations are still followed today. He made tying of the saphenous vein a logical procedure; this vein was tied at gradually higher levels throughout history, starting at the time of Celsus (53 B.C.-A.D. 7) in Roman days. Brodie exposed the veins, divided and extracted them without ligature, tearing out as much vein as possible after grasping the remaining ends, and controlling bleeding by pressure and touching bleeders with a cautery.

In the upward trend of tying, one must mention Ambroise Pare (1510-1590), who did mid-thigh ligations. This French surgeon described the 'pathology' thus: 'The matter of them is usually melancholy blood for Varices often grow in men of a melancholy temper and which usually feed on gross meats or such as breed gross and melancholy humours. Also women with child are commonly troubled with them by reason of the heaping together of their suppressed menstrual evacuation. It is best not to meddle with such as are inveterate.' His lack of knowledge of physiology is certainly compensated for by his sound judgment and practical clinical sense.

Paul of Aegina (A.D. 607-690) also did mid-thigh ligation of the vein and ante-dated the present-day Trendelenburg ligation by some 1,200 years. It was Homans who eventually advised the ligation of the saphenous vein flush with the femoral vein and so completed the trend of upward interest away from the affected area about the ankle.

Perhaps the most significant factor is the alteration in posture from that of the quadruped to that of the biped. Varicose veins of the legs are virtually unknown in veterinary practice. The erect posture creates a problem of venous return in the standing position. The valves are not the only factors to be considered but also the problem of forward propulsion, which is affected, as we know, by the negative pressure in the thorax and also by the arterial propulsive action and further by the existence of a venous pump.

There are other factors which may act in a similar way to the valves, e.g. the venae comitantes, their site and significance, anatomical factors causing venous overlay or kinks, the popliteal fascia, subcutaneous tissue in differing normal states, venous tone, differences in the medial and lateral sides of the lower leg, and other factors influencing venous tone such as the higher centres, endocrines, etc.

ARTERIAL, CAPILLARY AND VENOUS PRESSURE

Landis [2] has shown that under normal conditions, with the capillary at heart level, the pressure is about 32 mm. Hg at the arteriolar end of the capillary and about 12 mm. Hg at the venular end. As the osmotic pressure of plasma proteins is 28 mm. Hg, the normal fluid exchange between the capillaries and the interstitial spaces is maintained.

However, the feet swell even in normal people when they stand for some time. Standing therefore modifies the factors influencing capillary permeability. Among other factors influencing capillary filtration is a rise in atmospheric temperature, e.g. an increase from 14°-44° C doubles the rate of filtration.[3] Increased filtration results in increased hydrostatic tissue-tension.

This tissue-tension reaches a maximum of 35 mm. Hg in man.

In the normal subject, the venous pressure will rise, on standing, to about 90 mm. Hg in about 30 seconds. Exercise of the calf muscles reduces this pressure to near 32 mm. Hg.[4] This is a strong indication of the existence of a muscle pump functioning apart from the propulsive arterial pressure.

Does the venous pressure of 90 mm. Hg, recorded in the legs on standing, result from back-flow of venous pressure due to antigravity failure in the erect position? Or does it follow from the purely physical and normal physiological process of propulsion forwards from the arterial tree?

The arterial blood pressure and its fluctuations can be determined in different ways. With an oscillometer cuff applied in the lying and the standing positions, the systolic blood pressure can be palpated at the level of the cuff. The application of a sphygmomanometer at a higher level, with further study of the oscillometer readings, gives the true value of the pulsations seen. The normal figures vary in the sexes (Table I). They are slightly higher in the male, especially for the maximum pulsations.

TABLE I. APPROXIMATE BLOOD PRESSURES (IN MM. HG) AT WHICH OSCILLOMETER READINGS ARE RECORDED IN THE SEXES UNDER VARYING CONDITIONS

	Female		
	On Elevation	*Supine*	*On Standing*
First flicker	20—24	20	50—70
Maximum pulsation	60—90	80—110	160—180
Last flicker	170	200	300
Blood pressure in arms (on palpation)	104	115	120
Blood pressure in legs (on palpation)	90	110	180
	Male		
	On Elevation	*Supine*	*On Standing*
First flicker	18—24	18—24	80
Maximum pulsation	100—120	120—140	200—220
Last flicker	200—215	220	Over 300
Blood pressure in arms (on palpation)	100	115	125
Blood pressure in legs (on palpation)	120	140	220

The blood-pressure level obtained by disappearance of the pulse does not correspond with the disappearance of the oscillometric pulsations. It is usually in the region of the upper limit of the maximum pulsations. (It is also noteworthy that exercise has been observed to reduce the amplitude of the pulsations. This is being investigated further.)

Samson Wright [5] and Duffield and Harris [6] have shown that increase of the venous pressure causes a corresponding increase in the capillary pressure, which can rise to the level of the systolic blood pressure.[6] This is important, in considering the effects of posture. If we assume that the venous pressure rises to 90 mm. Hg by reason of antigravity failure in the veins, then the arteriolar tree must dilate to allow an arteriolar pressure somewhere near 110 mm. Hg.

Landis [2] states that capillary pressure less than venous pressure is only possible for very brief periods. Duffield and Harris placed a cannula in the femoral artery of a dog in one leg and in the femoral vein of the opposite leg. By applying pressure above the latter point by a sphygmomanometer cuff, the venous pressure was raised by external compression to a height almost equal to that of the arterial pressure measured at the same time.

Landis wrote: 'In one instance the venous pressure was higher than the arterial but this was momentary. Almost immediately the arterial pressure rose to a higher pressure than in the veins. Incidentally with the raising of the venous pressure, the arterial pressure rises considerably, e.g. before the venous pressure was raised by compression, the arterial pressure was 44 mm. Hg, but after compression the arterial pressure rose as high as 109 mm. Hg. This rise in arterial pressure might be due to several causes, e.g. stimulation of pressor nerves, and if this were the case ordinary manometric readings of human blood pressures may be influenced in a similar way but there is no evidence in favour of this view. The rise may also be due to the administration of ether, where in deep anaesthesia the arterial pressure falls, and rises under light anaesthesia. We are convinced that the latter explanation is the explanation for the arterial rise in this case.'

It seems that Duffield and Harris did not appreciate the significance of their own experiments as Landis has so excellently shown. A rise in venous pressure is always associated with a rise of arterial pressure in excess of the venous pressure. This constitutes a normal physiological response.

The behaviour of the veins and the arteries (in terms of manometric pressures) is not as simple as that of fluid columns. Firstly there is narrowing of the arteriolar tree as it approaches the capillary. This narrowing is normal and reduces the blood pressure. An increased arteriolar contraction can act similarly, decreasing the blood pressure locally. Landis states that pressure changes differ in the same capillary from moment to moment and may differ widely in adjacent capillaries arising from the same arteriole. During a period of arteriolar spasm capillary blood pressure is habitually low, e.g. 7 mm. Hg, but on vasodilatation it rises for a brief period to exceed 40 mm. Hg. Spontaneous variations in peripheral blood flow and capillary blood pressure are, under proper conditions, equally large. An increased tone of arterioles, for example, reduces the capillary pressure.

Another factor is the state of the venous channels and, as is well known, there is a factor of venous and venular tone which is also dependent on weather conditions as well as on the higher centres. Anaesthetists know well the problem of contracted veins in patients who are nervous and who have been lying waiting for their operations under conditions conducive to nervousness. Just as arteriolar narrowing diminishes capillary pressure, so an increase in venous tone by reason of narrowing of the venous column will also prevent the full effects of the 90 mm. Hg column of venous blood upon the capillary bed.

Further, the so-called arterio-venous channels must be considered. They connect the arterioles and venules and are particularly numerous in the palms of the hands and soles of the feet and toes. They are predominantly under control of the higher centres and are liberally supplied with sympathetic fibres. They con-

tract strongly with nicotine, and dilate fully on heating the body and on exposure to excessive cold; but on cooling the body they contract. The behaviour of these arterio-venous shunts is extremely erratic and the results of skin-temperature testing may well depend on the activity of the arterio-venous shunts rather than on that of the capillaries. It is of interest that frequently, on warming the arms, the toes will warm considerably: yet the 'ulcer' or 'gravitational' area will not warm as much. It may actually become cooler, except in cases with gravitational phenomena, when the area mentioned warms rapidly and even more satisfactorily than do the toes.

Further, as Professor Boyd [7] has indicated in a personal communication, injection of arteries with Thorotrast usually results in a blushing of the toes and feet; yet there is quite a pallor in the medial supramalleolar area under discussion.

The experiments of Parrisius and Wintterlin [8] are of interest when we consider the reaction of the capillary and subpapillary venous plexuses of the skin to changes in posture.

Normally in the supine position a white background can be seen with numerous capillary vessels coursing over it. The veins of the subpapillary plexus being invisible, the background is white. On standing there is a rapid livid coloration of the background. This is due to the subpapillary venous plexus becoming dilated and showing up clearly, with more capillaries visible, noticeably wider in the venous limb. On exertion, e.g. 20 elevations on tiptoe, the background becomes pale but the capillaries are unaffected. We suggest that these movements empty the venus subpapillary plexus without affecting the capillaries. As the input from the arterial side is unaffected and the capillary flow is unchanged, and as there is a diminution of the subpapillary venous plexus and as the inflow is actually increased in exercise, the circulation under these conditions is being by-passed through the arterio-venous shunts of Hoyer.

However, the diminution of oscillometric pulsation on exercise needs an explanation. Is it that by emptying of the subpapillary venous plexuses, the venous back pressure is reduced and the arteriolar pressure reduced as well by reflex action, thus diminishing the pulsations? It is likely that the circulation is deflected to the muscles as well as the arterio-venous shunts and that although the flow through the leg is increased, the capillary bed is unaffected and the skin venules are relatively empty, whereas the venous return to the heart and the circulation through the muscles is increased.

There is also further evidence of increased arterio-venous flow under conditions of increased venous pressure. Blalock showed that normally the oxygen level in the femoral vein is highest when the patient is recumbent and decreases rapidly in the upright position:

He wrote: 'In unilateral varicose veins the venous oxygen level is higher in the femoral vein of the diseased side, and this is accentuated when there is ulceration. Changes in posture cause less alteration in the venous oxygen content of the diseased side.

'Almost the same remarks are made regarding the oxygen content of the blood of the saphenous vein and the femoral vein.

'When ulceration is not present, the venous oxygen content of varicose veins of the lower part of the extremity is lower than that of a similarly placed normal vein of the opposite extremity; but when ulceration or infection is superimposed on varicose veins the oxygen content is usually higher on the diseased side.

'In thrombosis of the inferior vena cava with dilated veins the data are essentially similar to those in other varicose veins. These observations suggest that the total flow through the leg in a case of varicose veins and ulceration is increased.' [9]

Thus it seems that the arterial side provides a large proportion of the pressure present at all times in the venous tree. A rise in arterial pressure, however, does not necessarily mean a corresponding rise in the venous pressure, as the flow may be deflected through the shunts into a venous tree competent to withstand the effects of gravity. But a rise in venous pressure from any cause is reflected in a corresponding rise in arterial pressure which is then responsible for forward propulsion, pump or no pump.

To summarize the possibilities of arterial propulsion: From the arguments discussed it is apparent that arterial pressures can always rise above venous pressures and allow a forward propulsion, even in the presence of obstruction, as long as the obstruction does not rise above the level of the systolic blood pressure.

The arterial pressure is therefore well able to cope with the venous pressure from a column of blood rising to the heart level and, in the normal, to respond to such venous pressures as may arise from alterations in posture. The flow from the arterial side is assisted towards the heart by other factors, which are mainly associated with the muscular pumps.

THE MUSCULAR PUMP

The existence of a mechanism which, for lack of a better term, we may call the muscular pump, is suggested by the fact that the pressure found in the veins of the leg, even in a normal subject after standing for 30 seconds, can be reduced by such exercise as elevation of the heel off the ground for 20-30 times. The studies of Parrisius and Wintterlin also support the existence of such a muscular pump. They observed that the livid background in the subpapillary venous plexuses (due to distension with blood) changed to a marked pallor after exercise.

The normal mechanism of this pump consists of muscles with dilated lakes (intramuscular veins) and veins lying between muscles (intermuscular veins or venous plexuses). The concept of the pump implies contraction and emptying of the intramuscular veins into the popliteal and the femoral veins, which are supplied with competent valves. At the same time the contraction of muscles within the fascial confines compresses the intermuscular veins, pushing the blood upwards into the receiving columns as the increase in pressure closes distal valves but opens proximal valves. Upon relaxation of the muscles a relatively negative pressure is created in the veins which, during this period, receive blood from distal areas and (through the communicating veins) from the superficial areas. The blood from the medial side of the lower leg passes through the communicating veins as well as up along the long saphenous vein. This hypothesis explains why reduction in venous pressure occurs only in the normal

veins below the knee. Measurements in the femoral vein show only fluctuations without true reduction in venous pressure.

The introduction of the terms systole and diastole of the pump seems justified. During systole contraction of the calf muscles forces blood into the venous tree with forward and upward propulsion by reason of competent valves. During diastole of the pump the intermuscular veins fill by aspiration from the superficial areas, the distal areas and from the subpapillary venous plexuses of the skin.

There are differences on the medial and lateral aspects of the normal leg. Injection of radio-opaque dye through the lesser saphenous behind the lateral malleolus demonstrates filling of the muscular lakes (better described as the soleus and gastrocnemius venous plexuses) and the intermuscular veins (Fig. 1). After a calf thrombosis, no filling of the lakes occurs (Fig. 3). When the dye is injected into the long saphenous vein anterior to the medial malleolus, the dye passes through the long saphenous tree and the superficial veins (Fig. 2).

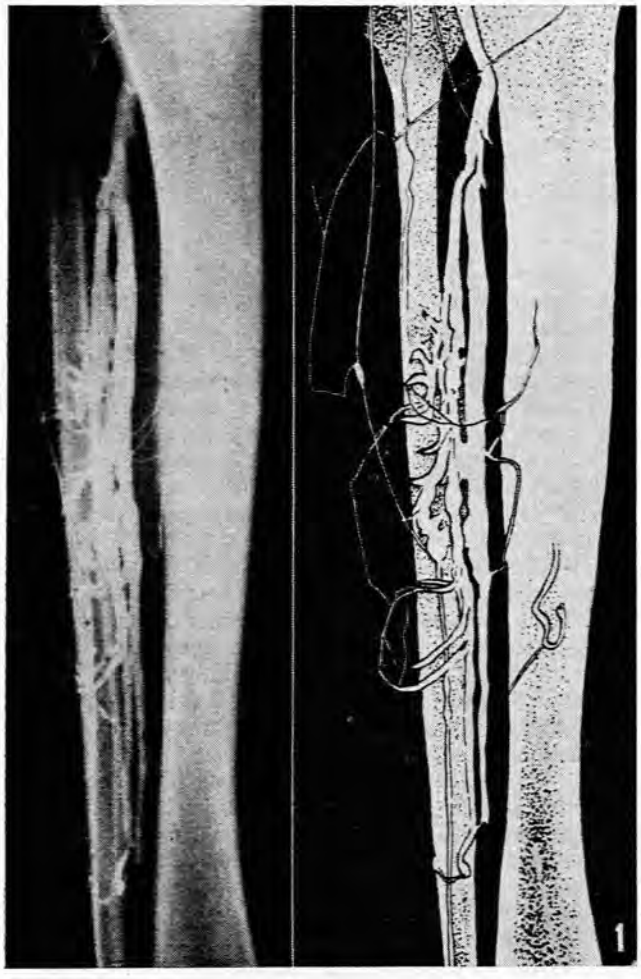

Fig. 1. Injection of radio-opaque dye into the small saphenous vein behind the lateral malleolus, demonstrating intramuscular lakes and intermuscular veins.

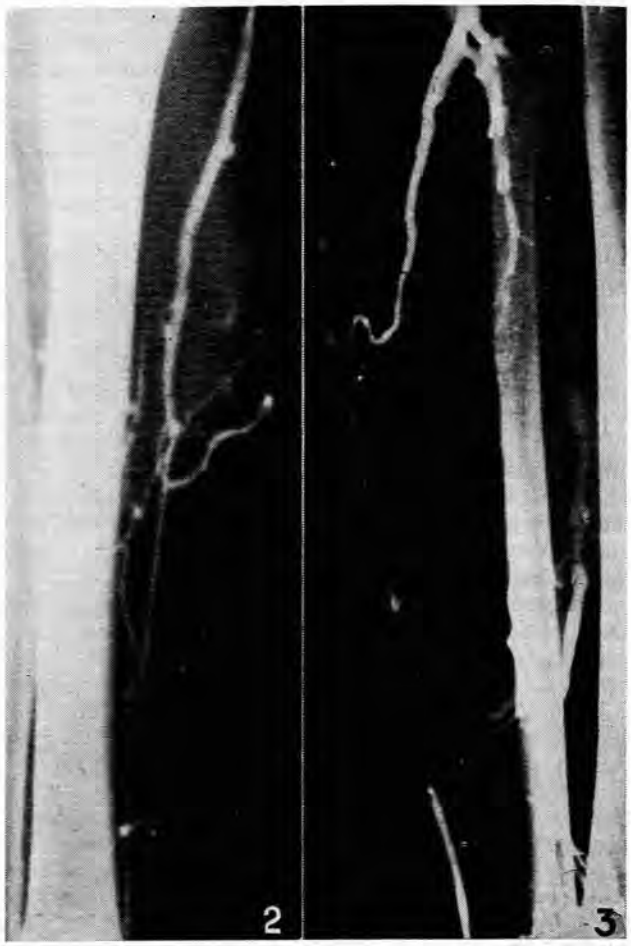

Fig. 2. Long saphenous tree as seen on injection of the vein at the medial malleolus.
Fig. 3. No filling of intramuscular lakes after calf thrombosis.

At rest in the lying position it is unusual to demonstrate the deep veins of the calf. The intramuscular lakes are never demonstrated. Serial radiography shows that the dye eventually passes into the deep veins above the knee. Some variations occur in the rapidity of removal of the dye from the leg veins into the deep channels and out of the system.

Because of the anatomical differences described, muscular action is constantly emptying the lateral aspect of the leg, where there is a more intimate transmuscular course; on the medial side, however, the course is through the superficial venous tree unless the muscular pump establishes an aspirating system to empty the medial veins through the deep channels.

During walking there is systole and diastole of the calf. The knee action is also supportive, because when the calf contracts the ankle is plantar flexed and the knee is extended. In this position the popliteal fascia is taut and aids compression of the contained venous columns, thus assisting the forward propulsion of the venous blood past the otherwise unsupported portion

of the popliteal fossa. At the same time there is relaxation in the anterior compartment of the leg. With the next step the ankle dorsiflexes and the knee flexes. Now there is relaxation of the popliteal fascia and the calf muscles, with filling of the intramuscular lakes and intermuscular veins. The muscles of the anterior compartment are now contracted and the blood from here together with that passing through the popliteal fascia drains, at this stage, into the popliteal vein which has been relatively emptied of blood during the previous stage by passage of blood through to the femoral vein.

Thus walking itself ensures efficient muscular pumping in the leg. Failure of reduction of pressure on exercise when these pressures are taken in the veins below the knee, must therefore be due to a defect in the mechanism described. In other words, if a relatively negative pressure is not established in the intermuscular and popliteal veins at the correct time of the pump cycle, then reduction of pressures can never occur. If valves are ineffective, a reverse flow occurs in the femoral or popliteal veins because of the high venous pressure, which may be due to obstruction or dilated incompetent deep veins and, consequently, the pumping mechanism will fail to reduce the pressure on exercise.

It is difficult to decide how much activity is necessary for a normally functioning pump mechanism. It has been shown that the normal pump activity from tonic contractions is quite considerable, but in older patients with deformed feet, who rarely do much walking, the muscular pump must be far from normal.

The medial side of the leg particularly takes the strain in all the pathological states affecting the venous system of the lower leg. In long-saphenous varicosities the highest pressure will occur in this area. In disturbances of cyclical pressure in the popliteal and intermuscular deep veins it is again the medial portion of the lower leg which will suffer by failure of aspiration of the blood from this area into and through the deep tree. Increases of deep pressure will also be reflected by increases in pressure in the veins behind the malleoli.

On the lateral side there is, however, some form of protection. The supramalleolar area is considerably protected from increased pressure-changes by the bulk of the calf muscles. It is only through the communications of the intermuscular veins with the retromalleolar area that the strain may be felt in these areas.

The investing fascia of the muscle groups supports the contained muscles. It also forms part of the relatively rigid walls of the posterior compartment of the leg. Thus when the calf muscles contract, they compress the intermuscular veins and so aid in emptying them.

For these reasons reconstructions of the popliteal fascia is important after operations, to prevent the gross mass-recurrences common in this area after ligations without reconstruction.

THE ISOLATED CUFF

On the medial side of the leg there is a thick, deep layer of fascia (Fig. 4). It lies over the large calf muscles attached to the tibia medially and, passing over the soleus and gastrocnemius muscles still more medially and then posteriorly, it is inserted laterally into the fibula. It is possible to pass the hand freely beneath this fascia over the contained muscles with little restriction by vertical septa. In contrast, on the lateral side of the leg the deep fascia is firmly attached to underlying muscles with numerous septa connecting the fascia to underlying bone.

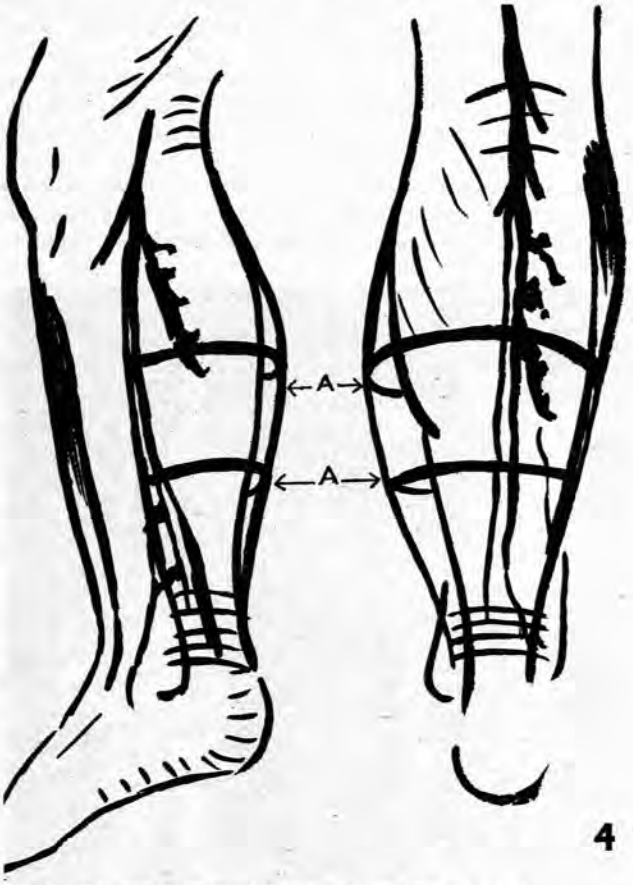

Fig. 4. Extent of the deep fascia cuff, indicated by 'A'.

On the medial side the communicating veins pass into this cuff through the relatively poorly supported subfascial space, whereas on the lateral side the perforators receive considerable support. Thus increases in venous pressure will more easily distend the perforators on the medial side and these changes will be reflected in the overlying medial supramalleolar region. This area has been called the 'isolated cuff area' because of the relatively isolated subcutaneous tissue area superficial to this extensive subfascial space, through

which relatively few perforators supply the subcutaneous tissues, once the longer vessels are obstructed.

THE VALVES OF THE VEINS

Even in the normal subject antigravity failure becomes evident in the lower legs on standing. The normal leg injected through the lesser saphenous vein at the ankle (Fig. 1) is compared with the leg in the dependent position (Fig. 5). With a light tourniquet around the leg great dilatation occurs in the venous tree (Fig. 6). This illustrates what normally occurs in man in the erect position, especially when tight garters and other forms of external pressure are applied to the legs. It also indicates how easily the venous tree can dilate on obstruction. Moreover, it has a tremendous capacity to form collaterals under pathological conditions (Figs. 3, 7, 8).

Certain features about valves in veins must also be considered, as the whole concept of a pump and the propulsion of venous blood towards the heart depends

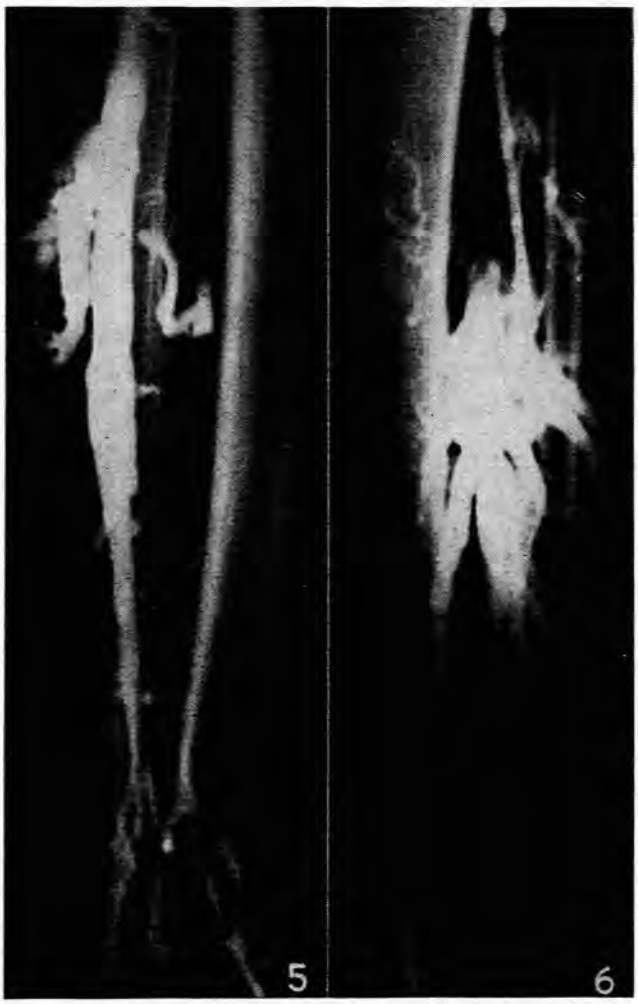

Fig. 5. Dilated deep veins in the dependent position.
Fig. 6. Dilated deep veins with a light tourniquet applied to the thigh.

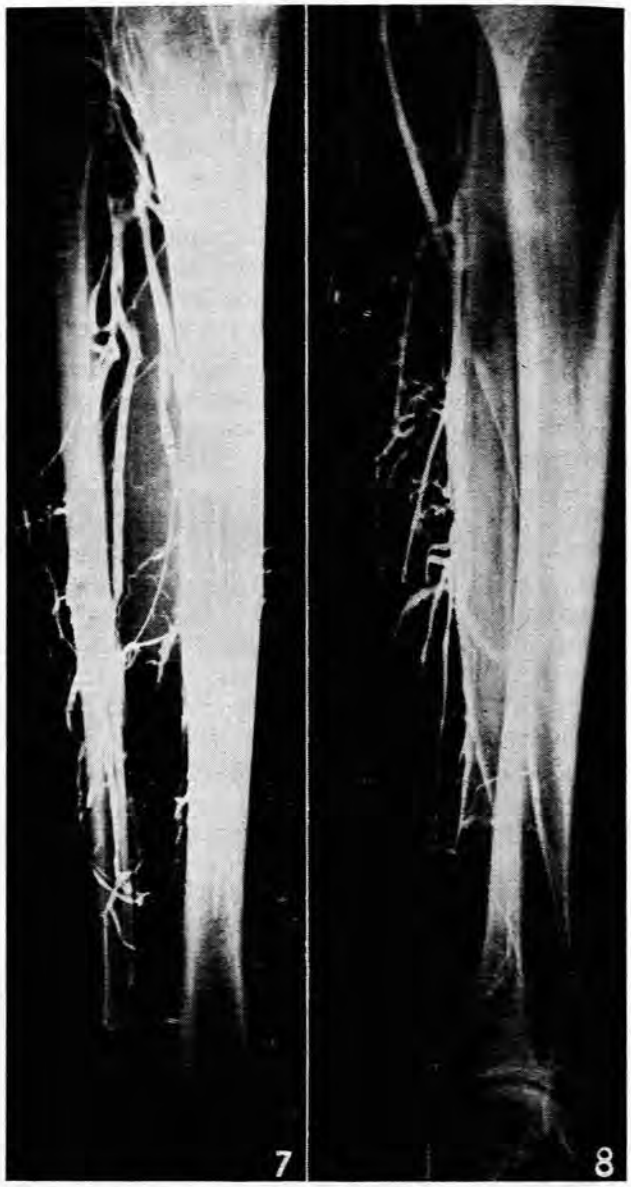

Fig. 7. Collaterals forming after deep-vein thrombosis.
Fig. 8. Numerous collaterals after deep calf thrombosis.

on the patency of valves or on other factors already mentioned.

Jäger's work on valves is basic. He studied the veins on the inner surface of the frog's leg. He is quoted by Franklin [10] as follows: 'When the circulation was rapid, the valve cusps were completely flattened against the vein wall, but with slowing of the blood stream they came out from the wall and eddies were often seen in the concave sinus or at the free border of the cusps. With a still further fall of blood pressure the cusps came out still further into the stream and when the blood flow was reversed, by raising the pressure in the main vein, the cusps met to form a single hemisphere blocking the tributary. On re-establishment of the normal direction of blood flow the two cusps separated'. Franklin [11] states: 'The valve closes intermittently whenever the pressure in the segment of vein proximal to the valve rises above that in the segment of vein distal to it . . . .

'In general the valves prevent back-flow of blood or give irre-

versibility of direction to the blood flow. After a valve closes the blood flowing into the segment of vein distal to it raises the pressure until the normal gradient is re-established and the valve opens . . . .

'To Jäger we are indebted for the first direct observation of the valve play during muscular contraction. When the thigh muscles of a frog were tetanized, there was a temporary closure of the valves of the vv. peronea and suralis, peripheral to the muscles. The closure of the valves next to the muscle was effected by a back-flow of blood in the veins. The more distal ones shut without any observable back-flow, but presumably the backward displacement of the nearest valves raises the pressure in the next distal segment of vein. After a short period of time the valves reopened even though the muscles were still visibly contracting, i.e. the inflow of blood from the capillaries had re-established the venous gradient at a higher level . . . .

'A vein leaving the gastrocnemius was also studied. It had a valve just proximal to its exit from the muscle. The valve remained open during the contraction of the gastrocnemius but shut for a moment as the muscle relaxed, because the muscle vein dilated with the relaxation and tended to refill in part from the proximal extramuscular portion, i.e. the pressure gradient was temporarily reversed. Jäger (1937) considers that the main function of valves in the frog's veins is to protect the capillaries and venules from *sudden* rises of pressure during muscular contraction'. Franklin concludes: 'It will be seen that even slight muscular contractions can temporarily reduce the venus pressure in the veins of the foot. It must be assumed that after the contractions valves are kept closed by hydrostatic pressure until fresh inflows of blood from the periphery raise the venous pressure distal to these valves above that obtaining proximal to them. In such cases therefore the valves will be acting as antigravity mechanisms.'

OTHER SUPPORTING AGENCIES

The role of contraction of the smooth-muscle wall of the veins, particularly the superficial veins, must not be overlooked. This property may explain why congenital deficiency of the valves exists for many years before dilated veins occur. The thickening which is usually present in varicose veins denotes a long period of increased muscular activity capable of maintaining contraction even in the abnormal vein against increased venous pressure. Antigravity failure only occurs on failure of this compensatory mechanism to maintain contraction. The muscular contraction of the veins is also influenced by the higher centres, and by drugs (e.g. nicotine) and hormones (e.g. progesterone), as well as by weather conditions.

The subcutaneous tissues themselves may play a part. It is a clinical impression that fatty tissues provide deficient support, whereas the healthy areolar tissue of young persons supplies much better support. There is a considerable variation in the density and construction of subcutaneous tissue and in the thickness of the skin. These factors also play a part in the general support of the neighbouring veins.

DISCUSSION

The physiological processes surveyed suggest that an investigation should be made of some of the clinical phenomena constantly seen. In further studies it is hoped that a full discussion of these may be possible. However, in the light of the concepts discussed, it is possible to make certain statements and to outline certain syndromes.

In general there is no such thing as stasis in the legs, because in legs suffering from the effects of gravity the blood flow through the leg as a whole is more rapid. Blalock, for example, has shown that the oxygen content of the venous blood is normally increased in the femoral vein in varicosities and leg ulcers. A rise in venous pressure stimulates increased arteriolar pressure to allow forward propulsion. If the compensatory arterial response cannot occur, stasis and cyanosis will result.

Generally speaking there is also no such thing as venous obstruction, because it leads to physiological changes which overcome the obstruction. These physiological changes are dependent on the arteriolar circulation and the arterial system. Hence when there is arterial deficiency or arteriolar spasm, the venous obstruction cannot be met by the development of an effective collateral circulation. Oedema then results.

In cases of early thrombosis, lumbar sympathetic block with paralysis of sympathetic fibres to the leg causes a dramatically rapid response with improvement in the oedema.

Tying of deep veins has become recognized through the work of Bauer [12] (who recommends ligation of the popliteal vein) and Linton [13] (who, depending on the pressure readings, ties the superficial femoral vein and strips the superficial venous tree when it is varicose; he also does a radical below-knee division of perforators). There is every indication that the limb will overcome such an assault on the venous tree for the normal response in every patient without a defective arterial tree permits the development of collaterals.

These surgeons have satisfactory results in a high proportion of patients. However, Oschner and others have demonstrated that tying of deep veins not only has no effect on antigravity failure, but also has no effect on the reductions of venous pressures on exercise. These observations would therefore seem to give no indication for this type of operation, and they support the rejection of what appears to be a safe and helpful procedure in the hands of other surgeons, thus pointing to a paradox in this field at present. Antigravity failure occurs even in normal subjects and it is unlikely that the tying of one vein in a column, when there are many side channels which will immediately fill the already dilated systems, will cure antigravity failure. Moreover ligations of femoral or popliteal veins will not have the effect of creating negative pressure aspiration in the pumping system discussed in this article, and cannot be expected to alter the venous pressures below the knees.

In proved arterial deficiency one can appreciate what happens (1) when the patient is lying in bed with the leg elevated and (2) when he stands. Certainly on standing the increased venous pressure will tend to dilate the capillary bed and will stimulate what arterial circulation there is to compensate for the condition. The concept of Makins, that when an artery is tied the vein should be tied simultaneously in order to keep a viable leg, can also be understood on this basis.

In arterio-venous aneurysm the arterial pulse, in the early stages, is diminished; later, however, an excellent collateral circulation comes into being in which it is perfectly safe to do a complete quadruple ligation and excise the aneurysm. We suggest that in these cases

the arteries fill the veins and increase the venous pressure, and the increased venous pressure (increased by standing) causes an increased stimulus to the arterial tree. In the late stages the excellent collateral circulation is due to this stimulus from the venous side.

This concept also applies to the patients with gravitational leg syndromes and postphlebitics with or without ulcers, who show marked pulsations on oscillometric study.

On occasion a patient with warm toes may present with venous disease, varicosities and even ulcers. Because of the warm toes one is disinclined to consider the case as an arterial problem. Admittedly the arterial pulses may be impalpable in these legs, but this is not unusual in cases with oedema or induration. However, on oscillometric studies in some of these cases, it is apparent that complete blockage at the level of the popliteal artery is present. The author introduces the term 'masked arterial disease' for these cases and stresses that it is only by oscillometric study that the presence of arterial disease is detected. In fact, it is possible to describe some cases as true arterial disease, and others—late phlebitics with popliteal or femoral artery blockage—as masked arterial disease.

In certain late phlebitics with gross venous disease and varicosities, oedema, pigmentation, ulceration and sometimes arterial deficiency, there is marked rubor on standing and pallor on elevation. The rubor comes on as a flush of deep pink congestion on standing and an ulcer may bleed. The author has introduced the term 'flush syndrome' in these cases.[14] The flushing indicates a dilatation of the capillary bed on standing and the pallor on elevation indicates a drainage of the blood along the dilated veins, emptying of the capillaries and even possibly contraction of the now unnecessarily dilated arterioles. In support of this concept is the finding at operation when these thickened tissues are excised. Areas of gross thickening with dense white scar will bleed profusely with bright red blood spurting from numerous points; but with or without pressure within a few seconds the bleeding ceases, leaving a dry field without need of ligatures.

It is assumed that in these cases there has been a failure of protection of the capillaries due to (1) constant back-pressure on the venous side and (2) loss of pumping action with consequent loss of the accessory protective mechanism of the pump in reducing venous pressure. Moreover, chronic infection with persistent hyperaemia adds to the continuous dilatation of the capillaries. These patients usually have pale, bloodless muscles and straight intermuscular channels, without valves plastered against the oedematous fascial interosseous membrane. The flush syndrone in no way indicates arterial deficiency, as is so often premised clinically when there is dependent rubor and elevation pallor. Operations can be performed with safety in these cases, although it is inadvisable to try to save the worst legs, where periostitis and irreparable changes are present. But in less severely affected legs operations can be performed on the foot with excellent healing.

When considering gravitational syndromes one must consider also the problems arising from what may be termed dependent granulation tissue. In the standing position the granulation tissue with its buds of capillaries is subjected even in the normal person to high venous pressures. Any leg ulcer will thus be associated with an increased flow of blood through the granulation tissue and be subject to excessive oedema. It is well known that healing in dependent areas is slow. How much more does this apply when an ulcer is in an area where there are dilated varicose veins and incompetent veins with high venous pressures aggravated by standing. This is the state in the so-called ulcer area of the leg.

It remains to discuss the problems of sympathectomy in 'gravitational' legs. Sympathectomy will undoubtedly increase the circulatory flow and the warmth and colour of the leg in the lying position. This will be particularly effective in the toes and feet. It is doubtful whether the operation has an effect on the ulcer area, where there is already the full stimulus of an increased flow of blood. After sympathectomy the stimulus to increased blood flow is already present on standing and it is possible that it may even aid the development of the so-called flush syndrome. Whether this is good or not, it is highly probable that the natural protection afforded by an arteriolar tree capable of contracting and varying under a differing series of conditions is lost. However, there is no doubt about its value in the patients with persistent oedema and cold, sweaty feet, especially when the symptoms are aggravated by cold weather.

A few points should be made concerning other factors in the pump. One cannot overlook the function of the feet in muscular pumping. Deformities of the feet may be the direct result of congenital deformity; in other cases a congenital deformity is aggravated, e.g., by hallux valgus, hallux rigidus, clawed toes, callosities, or fungus infection in the feet. It is one of the mainstays in treatment that if the patient is able to walk long distances and keep the feet strong he will have little trouble with his legs. It is patent that patients with deformed and painful feet are not good walkers.

The author has now completed a series of 120 instances of what he has termed strip grafting, in chronic indurated leg ulcers. This operation, commenced in 1945, is designed to excise (1) irreparable tissues in the area of the isolated cuff, and (2) dilated communicating veins passing through into this area on the medial aspect of the leg. Strip grafting combines the so-called Linton procedure, which is a subfascial operation, and lengthening of the tendo Achillis, and provides a skin graft on muscle. This thickens to provide support against all tendencies of antigravity failure to form new 'blow-outs'. By attention to the principles of prevention of dependent granulation tissue, most patients have been given a new lease of life. The operation recently described by Cockett,[15] in which perforators were excised in the medial part of the lower leg (he reported 2 cases) has been part of the procedure described by the author[14] since the earliest cases in the present series. It is possible now, with the long follow-up and the large series, to know what the results over the years really are. In a later article it will be

shown that the results are really worth while and are based upon physiological premises.

SUMMARY

1. The early history of varicosities and venous ligations is reviewed briefly.

2. The normal peripheral mechanism of propulsion of blood from the arterial to the venous side is assessed critically, with attention to the compensatory mechanism resulting from a rise in venous pressure.

3. The role of the arterio-venous shunts and of the capillary and the venous subpapillary plexuses in venous return is considered.

4. The hypothesis is put forward of a muscular pump in the calf which aids the efficient return of blood from the periphery of the leg by a mechanism of systole and diastole present in walking.

5. Significant differences in the anatomy and the physiology of venous return in the medial and lateral sides of the leg are reported.

6. The skin and the fascia on the lower medial side of the leg are described as an isolated cuff, prone to complications arising from their anatomical features.

7. The clinical implications of these observations are considered.

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