HUME, R. (1958) *Brit. Heart J.* **20,** 15.
HUSOM, O. (1961) *Scand. J. clin. Lab. Invest.* **13,** 619.
JOYNER, C. R., HORWITZ, O., and WILLIAMS, P. G. (1960) *Circulation,* **22,** 901.
KERR, J. W., PIRRIE, R., MCAULEY, I., and BRONTE-STEWART, B. (1964) Personal observations.
KUO, P. T., WHEREAT, A. F., ALTMAN, A. A., and WEST, J. W. (1960) *Circulation Res.* **8,** 1157.
KWAAN, H. C., and MCFADZEAN, A. J. S. (1957) *Nature,* **179,** 260.
LYON, T. P., YANKLEY, A., GOFMAN, J. W., and STRISOWER, B. (1956) *California Med.* **84,** 325.
MCDONALD, L., and EDGILL, M. (1957) *Lancet,* **2,** 457.
MACFARLANE, R. G., TREVAN, J. W., and ATTWOOD, A. M. P. (1941) *J. Physiol.* **99,** 7P.
MALMROS, H. (1950) *Acta med. Scand.* **246,** Suppl. 137.
MASTER, A. M., JAFFE, H. L., and DACK, S. (1936) *Amer. Heart J.* **12,** 549.
MERSKEY, C., and LACKNER, H. (1959) *Postgrad. med. J.* **35,** 203.
MEYER, J. S., and WALTZ, A. C. (1959) *Neurology,* **9,** 728.
MITCHELL, J. R. A., and BRIERS, S. M. (1959) *Lancet,* **2,** 435.
MORRIS, J. N. (1951) *Lancet,* **1,** 69.
MORRISON, L. M. (1960) *J. Amer. med. Ass.* **173,** 884.
MUSTARD, J. F., MURPHY, E. A., ROWSELL, H. C., and DOWNIE, H. G. (1962) *Amer. J. Med.* **33,** 621.
——— ROWSELL, H. C., MURPHY, E. A., and DOWNIE, H. G. (1963) *J. clin. Invest.* **42,** 1783.
O'BRIEN, J. R. (1956) *J. clin. Path.* **9,** 47.
OGSTON, D., and FULLERTON, H. W. (1962) *Brit. med. J.* **2,** 1288.
PIRRIE, R., KERR, J. W., and BRONTE-STEWART, B. (1964) Personal observations.
POOLE, J. C. F. (1958) *Brit. Med. Bull.* **14,** 253.
——— and ROBINSON, D. S. (1956) *Quart. J. exp. Physiol.* **41,** 31.
REGAN, T. J., BINAK, K., GORDON, S., DEFAZIO, V., and HELLEMS, H. K. (1961) *Circulation,* **23,** 55.
SOLOFF, L. A., and WIEDEMAN, M. P. (1963) *Nature,* **199,** 495.
STRØM, A., and JENSEN, R. A. (1951) *Lancet,* **1,** 126.
SWANK, R. L., and CULLEN, C. F. (1953) *Proc. Soc. exp. Biol. Med.* **82,** 381.
——— GLINSMAN, W., and SLOOP, P. (1960) *Surg. Gynec. Obstet.* **110,** 9.
THOMAS, W. A., and HARTROFT, W. S. (1959) *Circulation,* **19,** 65.
——— DAVIES, J. N. P., O'NEAL, R. M., and DIMAKULANGAN, A. A. (1960) *Amer. J. Cardiol.* **5,** 41.
VARTIAINEN, I., and KANERVA, K. (1947) *Ann. Med. intern. Fenn.* **36,** 748.
WILLIAMS, A. V., HIGGINBOTHAM, A. C., and KNISELY, M. H. (1957) *Angiology,* **8,** 29.

ANATOMICAL LOCALIZATION OF VENOUS THROMBOSIS

by

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CLINICAL EXPERIENCE AND post-mortem studies indicate that the majority of venous thrombi originate in the deep veins of the lower limb. The exhaustive studies of the sites of venous thrombosis carried out by Gibbs (1957) and Sevitt (1959) revealed an incidence of 59 per cent and 60 per cent respectively of thrombi in the legs in routine post-mortem examination. In Gibbs' series of 253 cadavers two main sites were found, the deep veins of the lower leg and of the thigh. In the lower leg the intramuscular sinuses of the soleus muscle were most commonly affected. Next in order of frequency came the posterior tibial and peroneal veins; gastrocnemial veins were least often involved. In the thigh, thrombi were often discovered in the superficial femoral vein, much less often in the profunda femoris vein. Frequently both sites were affected though clearly separated.

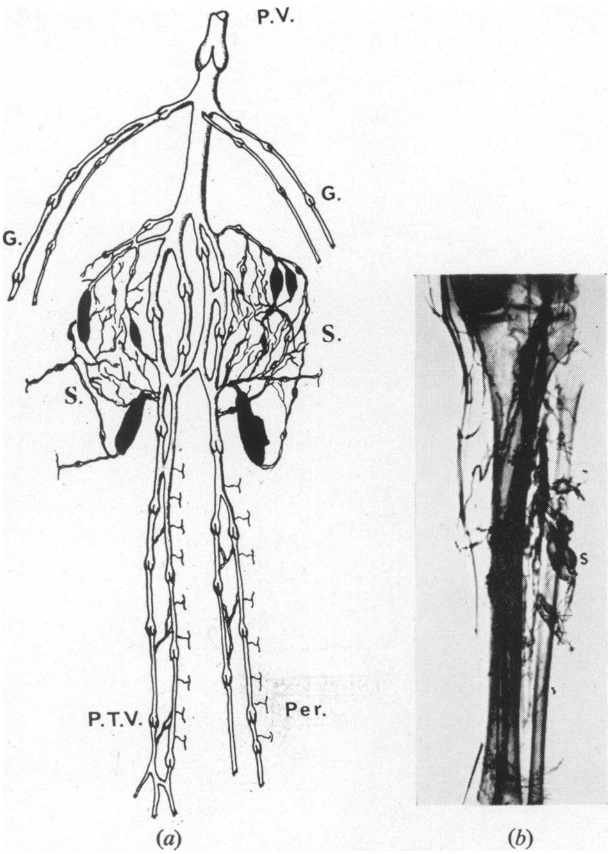


Fig. 1. (*a*) Diagram of anatomy of deep veins. P.V., popliteal; P.T.V., posterior tibial; Per., peroneal; G., gastrocnemial; S., soleal. (*b*) Phlebogram of amputated limb injected with micropaque. Soleal venous sinuses (S).

These observations have prompted us to study the anatomy of the veins of the lower limb, the function of the calf muscles and venous valves and their effects on flow.

Venous anatomy

Examination of veins in their collapsed condition post-mortem cannot fully reveal their state in life. When they are filled with radiopaque media or with resin, from which corrosion casts can be prepared, an idea of their size and spatial anatomy can be appreciated. In this study we have used both amputated limbs and specimens of isolated calf muscles taken from cadavers and have filled the veins either with " micropaque " (barium sulphate suspension) for radiography or Marco resin 26 C, a cold setting

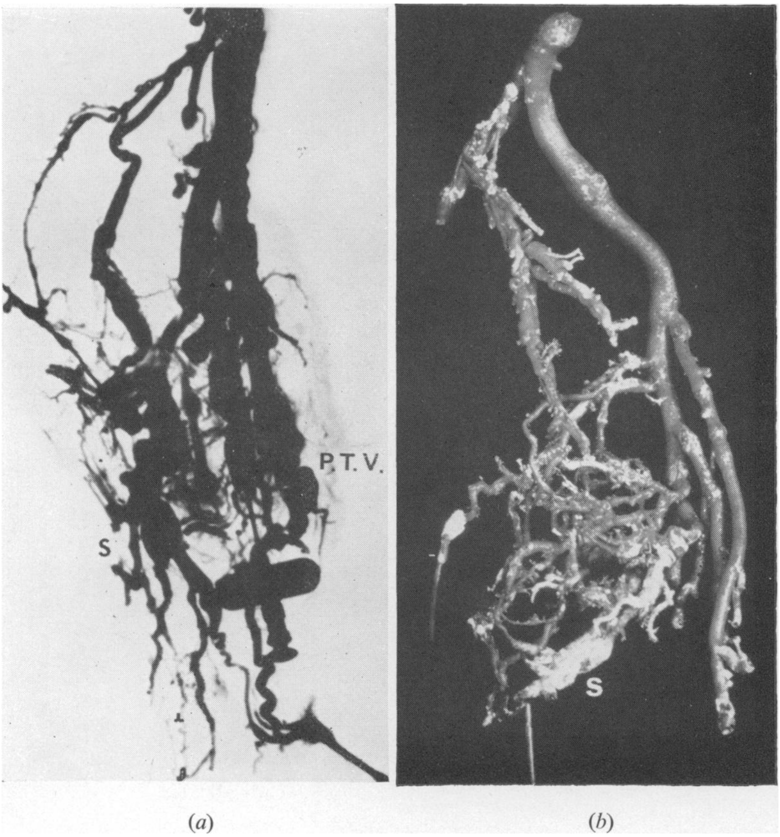


Fig. 2. (*a*) Phlebogram of isolated calf muscles. The size of a soleal sinus (S) is comparable with the size of a posterior tibial vein (P.T.V.). (*b*) Corrosion cast of soleal veins. Relatively thin arborizing veins are seen above a soleal sinus (S) which connects with the posterior tibial veins.

resin prepared and injected by a modification of the technique suggested by Tompsett (1959) (see Appendix, page 224). These methods allow filling of veins under moderate pressure so that they may be fully distended but it is not possible by these means to distend veins beyond the point of valvular competence. Radiographs tend to magnify the size of the veins by a small factor; cold setting resins contract on setting, so that plastic casts are a little smaller than the potential size of the veins.

The anatomy of the deep veins of the lower leg (Fig. 1*a*) was first described in detail by Le Dentu (1867) and more recently by Dodd and Cockett (1956). The venae comitantes of the posterior tibial and peroneal arteries are paired channels closely valved and haphazardly connected by short veins which run both transversely and longitudinally. Into them drain communicating veins that come from superficial vessels and veins

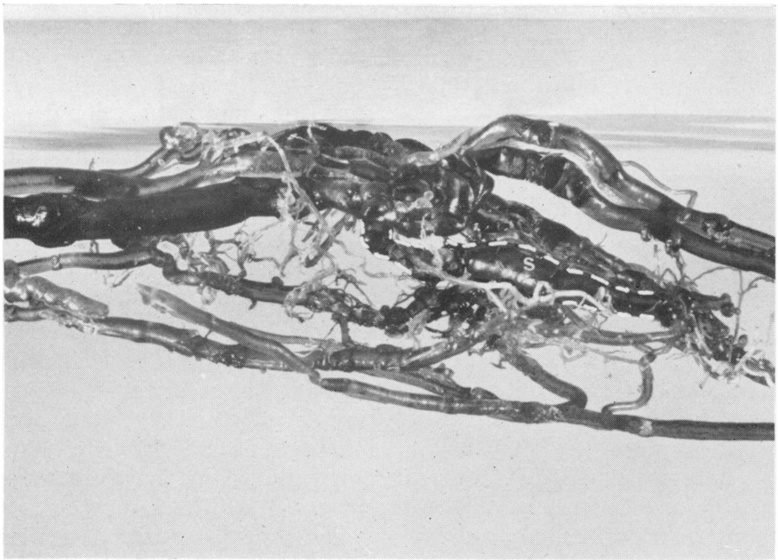


Fig. 3. Corrosion cast of soleal veins. A typical soleal sinus (S) is outlined. It lies dependent in recumbency in the inferior part of the muscle.

from nearby muscles. Proximally they join with the anterior tibial veins to form the popliteal vein.

Each head of gastrocnemius is drained by venae comitantes which join the popliteal vein either separately or as one vein. The gastrocnemius represents a variable proportion of the calf muscle mass; the size of its medial head is also variable. This variability is reflected in the size of the gastrocnemial veins which in some cases are very large, especially those draining the medial head.

The long strap muscles, such as flexors hallucis and digitorum longus, drain into either the peroneal or posterior tibial veins by short thin venous arcades that enter along the course of the veins nearest to them.

The soleus muscle has a distinctive venous anatomy. Dodd and Cockett (1956) have described large venous sinuses in the soleus muscle and their relation to the spread of thrombus from the sinuses to communicating veins near the ankle. Our preparations have demonstrated the size and relations of the veins within the calf muscles.

When amputated limbs are injected with radiopaque media at the ankle via the long saphenous and posterior tibial veins the filling of the soleal veins is usually found incomplete (Fig. 1*b*) because of the very efficient valving of muscular veins which prevents reflux. Soleal veins are connected with communicating veins, and only if the injection medium can be made to flow centripetally via communicating veins can adequate filling be achieved. The most constant results have been achieved after excision of the calf muscles together with the peroneal, posterior tibial and popliteal veins. Injection of the posterior tibial veins and communicating veins demonstrates clearly the size of sinuses in the soleus muscle relative to the size of the posterior tibial, peroneal and popliteal veins (Fig. 2*a*).

Corrosion casts provide permanent and more complete preparations of the soleal venous system. They show that most of the muscle is drained by short thin veins of regular calibre which intercommunicate in an arborizing pattern (Fig. 2*b*). These veins show numerous valves, even in the smallest which may be less than a millimetre in diameter.

Soleal veins form arcades of variable length joining the deep veins above and below at their extremities. Soleal sinuses occur as dilated segments of the arcades, beginning where they arise inferiorly and extending over a variable length of vein; they may measure up to 5 cm. in length and up to 1 cm. in diameter (Fig. 3). Several short stubby veins may join to form one trunk or there may be only one large vessel, which is sausage-shaped, with few or no valves, contorted and sometimes duplicated. These large vessels usually lie in the lower part of the soleus muscle, passing superiorly to enter the posterior tibial or peroneal veins in the upper third of the lower leg. Usually there is one large sinus to each half of soleus and a variable number of the smaller variety. It has been suggested by Gibbs (1957) that the number and size of the sinuses increase with age.

Where both the arterial and the venous systems of the muscle have been injected, the pattern of the arteries adjacent to the venous sinuses has not been found greatly different from the rest of the muscle, except that the sinuses have very large arterial branches closely applied to them, much larger than arteries to the rest of the muscle.

Thrombus in soleal veins

In three of six casts of the soleal venous system thrombus has been found, in one confined to a soleal sinus (Fig. 4*a*) and in two thrombosis had involved both soleal sinuses and the posterior tibial veins (Fig. 4*b*). The plastic casts were prepared by dissolving away all soft tissues with concentrated hydrochloric acid. Where thrombus had been dissolved the cast was stained black; elsewhere thrombus could be seen to be impregnated by the resin and preserved by it. In all three specimens it appeared that valves limited the extent of the thrombus either at a point of junction with major deep veins or within the posterior tibial veins themselves. In one limb soft thrombus had been expressed from the major veins by perfusion with saline leaving more firmly attached and presumably older thrombus in the soleal sinuses. There was evidence in the casts of

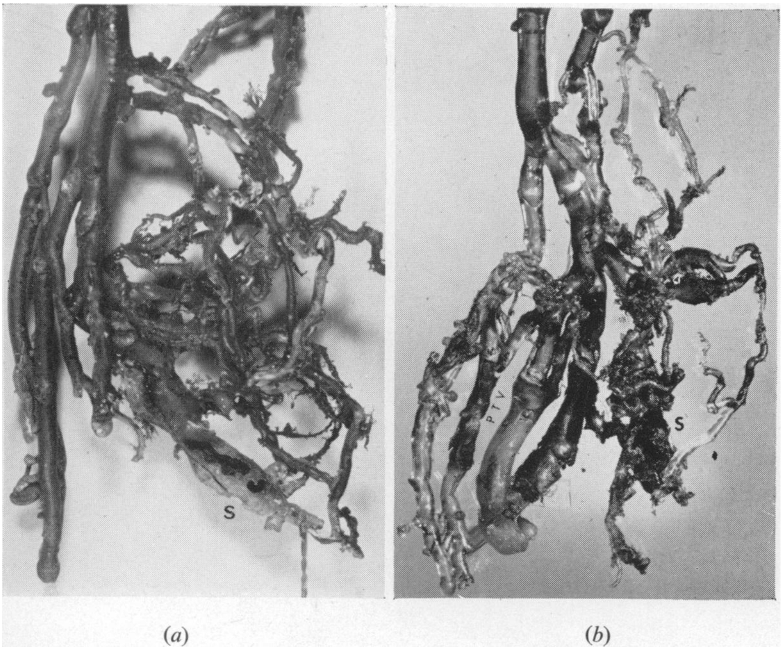


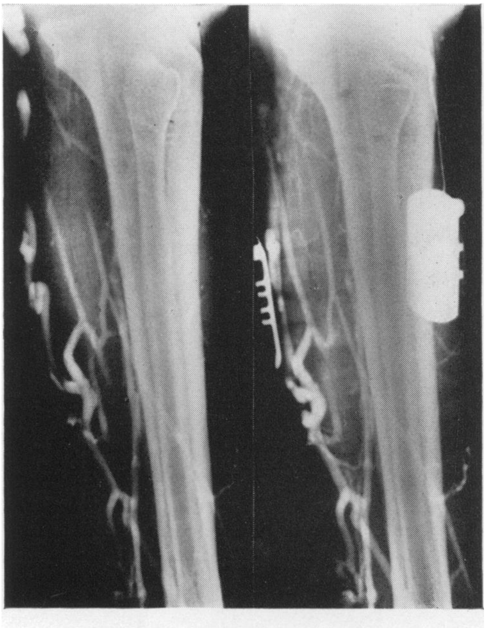
Fig. 4. (*a*) Corrosion cast showing thrombus in a soleal venous sinus (S). Only the sinus is affected. (*b*) Corrosion cast showing thrombus in a soleal sinus (S) and in the posterior tibial vein (P.T.V.) near their junction.

the propagation of thrombus from soleal sinuses into the smaller veins draining into them.

Function of the calf muscle veins

When the calf muscles are excised as a unit with their attached deep veins, the tendo Achillis and an appreciable part of the lower half of the calf muscles can be elevated before any large vein, usually the termination of a soleal sinus, is found entering the posterior tibial or peroneal veins. The soleal sinuses are the largest veins draining the muscles and must be responsible for a large part of the venous flow from them. When the calf muscles contract, their compression of the soleal veins plays an important role in the promotion of venous flow; in the relaxed soleus muscle stagnation of blood must occur because the sinuses are dependent in recumbency. Ludbrook (1964) has produced evidence, plethysmographically, that in change of posture from recumbency to the erect position the volume of blood pooling in the soleus muscles can amount to as much as 300 ml.

It is difficult to prepare casts of soleal veins unless resin is injected centripetally through communicating veins that connect with the calf muscles. It is rare to see muscular veins filled in phlebograms performed in recumbency or the feet-down position. Arnoldi (1961) performed many



(a) (b)

Fig. 5. Arteriograms of a patient with varicose veins. Exposure 18 seconds from beginning of injection. In (a) the muscles are inactive, in (b) rhythmical contraction of the calf muscles has been induced. Only superficial veins are seen.

phlebograms during exercise, but in his illustrations, although the posterior tibial, peroneal and popliteal veins are clearly seen, there is no sign of muscular veins. He commented that soleal veins are rarely seen in phlebograms. McLachlin *et al.* (1960) have shown by serial phlebography and estimation of the clearance of radiopaque medium from the deep veins how stasis occurs in valve pockets or sinuses and how elevation of the lower leg to 15° above the horizontal reduces the clearance time.

We have studied the circulation of the leg by performing arteriography, taking radiographs at six-second intervals. By this means the flow of contrast medium can be followed from arteries to veins. Such arteriophlebograms show that most of the contrast medium flows first into superficial veins (Fig. 5), the deep veins filling much later in the series. No sign of any filling of muscular veins in the calf is seen. The effect of exercise can be simulated by periodic electrical stimulation of the calf muscles, causing them to contract rhythmically. We have used electrocardiograph electrodes applied to the skin over the calf delivering stimuli

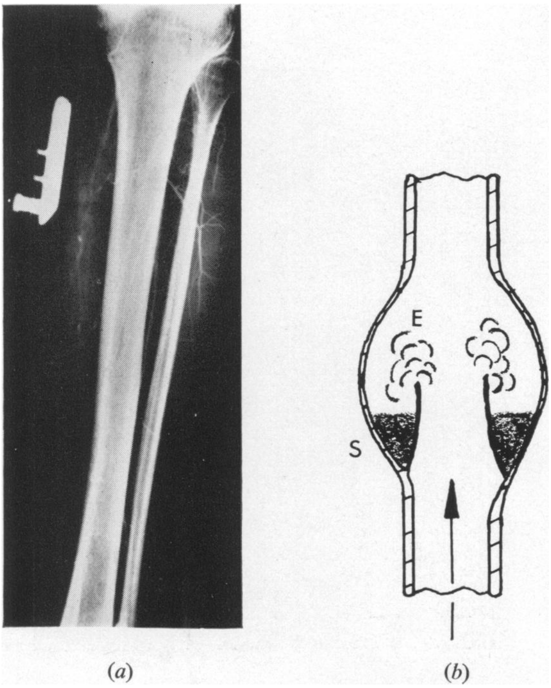


Fig. 6. (*a*) The effect of application of a crêpe bandage. Superficial veins are effectively obliterated. This radiograph taken 18 seconds from the beginning of injection should be compared with Figure 5 (*a*). (*b*) Diagrammatic section of venous valve showing stasis of dye in the valve sinus (S) and fluid eddies at the cusp free borders (E).

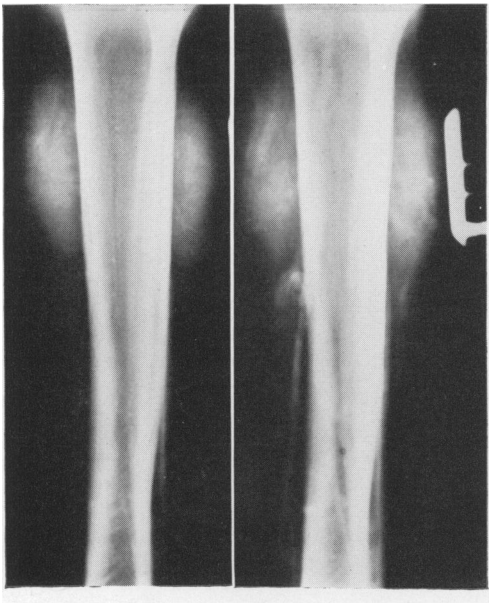
of 30–45 volts intensity for a duration of one millisecond at a frequency of one per second, a method similar to that described by McLachlin (1958).

Electrical stimulation of the calf muscles has little effect on the speed of venous flow unless the superficial veins are compressed by the firm application of a crêpe bandage. This simple manoeuvre produces a dramatic change in the vascular pattern, a complete absence of superficial veins and more rapid filling of the deep veins (Fig. 6*a*). If the calf muscles of the bandaged leg are stimulated electrically then the speed of deep venous filling is increased further and for the first time the veins of the muscles are visualized (Fig. 7).

These radiographic studies confirm that, in recumbency, the veins of the calf muscles contain stagnant blood excluded from the circulation of the limb. Flow through the deep veins can be increased by compression of the superficial veins and by contraction of the calf muscles.

Function of venous valves in relation to thrombosis

Gibbs (1957) and McLachlin (1951) have shown how often thrombi originate in venous valvular sinuses. We have examined a large number of femoral venous valves from cadavers and occasionally found thrombus in a valve sinus making a perfect cast of the sinus and only with difficulty dislodged from it.



(a) (b)

Fig. 7. Radiographs taken 30 seconds from injection; in (*a*) the limb is inactive, in (*b*) the calf muscles are contracting rhythmically. Deep veins and calf muscle veins are seen in the active limb.

Valve behaviour has been investigated in isolated preparations of cadaveric femoral vein. Vein segments are mounted vertically, perfused through the distal end, and valve movement observed from above, through the open proximal end of the vein. The perfusate is a dextran in saline solution having similar flow properties to that of blood. Rhythmical compression of the tubing leading to the vein creates pressure waves which simulate the disturbances caused by the veno-pressor action of calf muscles. This effectively demonstrates the transient valve closure which is thought to occur *in vivo* during exercise.

Venous valves are diaphanous but very strong. Their free borders become wavy and oscillate in a pulsating stream. If Indian ink is injected into the fluid entering the vein it can be seen to pass between the valve cusps, eddying around their free borders (Fig. 6*b*). In the valve sinus stagnation appears to be almost complete, for if Indian ink is injected into it from above, it lies in a pool at the bottom of the sinus with hardly any movement and little diffusion. The deeper the sinus the more extensive is the stagnation. As the deepest sinuses are found in the superficial femoral and common femoral veins it is not surprising that thrombus should form in their valvular sinuses so often.

Discussion

The radiographs of post-mortem calf muscles and corrosion casts of their veins show how the anatomy of the soleal venous sinuses provides favourable conditions for thrombosis. The finding of thrombus in the sinuses clearly demarcated from the remainder of the muscle veins confirms the importance of the anatomical arrangement of the veins. The difficulty of filling muscular veins in post-mortem specimens and in phlebograms suggests that large muscle veins contain, in recumbency and inactivity, stagnant pools of blood of an appreciable volume. The second site of venous stagnation in the lower limb is in the depths of the large valvular sinuses of the femoral vein. It has been shown by Payling Wright *et al.* (1951) that post-operatively there occurs a progressive decrease in the speed of venous flow, reaching its slowest between the 10th and 12th day after operation. This slowing of the circulation must favour the onset of thrombosis. McLachlin (1958) concludes that venous stasis in the lower limb is the prime cause of venous thrombosis.

We have been impressed by the pronounced effect of compression bandaging on the circulation of the lower leg in diverting blood from the superficial to the deep veins. Such bandaging might provide a simple method of prophylaxis against venous thrombosis by hastening deep venous flow. We have found the most striking demonstration of improved venous circulation to be provided by adding muscular contraction to the bandaged limb. It would seem worth while trying the effect of bandaging the lower limb together with regular contraction of the calf muscles as a prophylactic against venous thrombosis.

Summary

The venous circulation of the lower limb has been studied by means of injection of radiopaque media into cadaveric limbs and isolated calf muscles. Corrosion casts of resin have confirmed the distinctive anatomy of the veins of the soleus muscle and, in particular, the soleal venous sinuses in which thrombus is commonly found. Anatomical and radio-

logical evidence of stagnation of venous blood has been found in soleal venous sinuses. A second site of venous stagnation lies in the deep valvular sinuses of the femoral veins. Arteriography has been used to demonstrate the arterial and venous flow in the lower limb. Hastening of venous flow and visualization of muscular veins has been shown best by a combination of compression bandaging and calf muscle activity.

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Appendix

Calf muscles are excised from the limbs of cadavers through a mid-line posterior incision of the skin of the leg from the popliteal fossa to the heel. Skin flaps are dissected down to the plane of the posterior borders of the tibia and fibula. The tendo Achillis is severed from the calcaneum and raised together with the posterior tibial vessels. The calf muscles are dissected free of the tibia and fibula together with the posterior tibial and peroneal vessels.

The muscles are perfused through the popliteal artery with 2 per cent saline and all leaks are sealed off with ligatures. The veins are then perfused via cannulae inserted into the posterior tibial and short saphenous veins. The short saphenous veins are found to communicate with calf muscle veins in the majority of specimens. Any venous leaks are sealed off.

Radiography of the muscular veins can be performed after filling them with micropaque. Corrosion casts have been prepared by injection of the veins with Marco resin 26 C (supplied by Scott Bader and Co.). The constituents used to prepare the resin are Marco resin 26 C 100 g., monomer 30 g., catalyst HCH 6 g., accelerator E 6 g. The mixture is coloured with a blue dye. The plastic is injected through syringes 10 minutes before it is due to gel; enough is injected to fill the veins under moderate tension.

REFERENCES

ARNOLDI, C. C. (1961) *J. cardiov. Surg.* **2,** 116.
DODD, H., and COCKETT, F. B. (1956) *The Pathology and Surgery of the Veins of the Lower Limb.* Edinburgh and London, Livingstone.
GIBBS, N. M. (1957) *Brit. J. Surg.* **45,** 209.
LE DENTU, A. (1867) *Circulation veineuse du Pied et de la Jambe*, Thesis No. 276. Paris.
LUDBROOK, J. (1964) *Amer. Heart. J.* **67,** 493.
MCLACHLIN, A. D., MCLACHLIN, J. A., JORY, T. A., and RAWLING, E. G. (1960) *Ann. Surg.* **152,** 678.
MCLACHLIN, J., and MCLACHLIN, A. D. (1958) *Arch. Surg.* **77,** 568.
———— and MCLACHLIN, J. C. (1951) *Surg. Gynec. Obstet.* **93,** 1.
PAYLING WRIGHT, H. P., OSBORN, S. B., and EDMONDS, D. E. (1951) *Lancet*, **1,** 22.
SEVITT, S. (1959) *Modern Trends in Accident Surgery and Medicine*, Chapter 16. London, Butterworth.
TOMPSETT, D. H. (1959) *Ann. Roy. Coll. Surg. Engl.* **24,** 110.