



The deep venous system and reverse flow flaps

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SUMMARY. The deep venous system of the upper and lower extremities was injected with a lead oxide mixture in 2 fresh human cadavers, dissected, radiographed and the sites of the venous valves located.

These studies confirmed that the *macrovenous* connections between the *venae comitantes* of the distributing arteries were insufficient in number to bypass the venous valves in conventional, distally based reverse flow flaps (e.g. radial, ulnar, peroneal) but revealed an alternative *microvenous* interconnecting pathway which surrounds the artery as the *venae arteriosae*.

This pathway was investigated in a series of distally based reverse flow saphenous flaps in dogs, comparing flaps where the microvenous connections were left intact (non-skeletonised) with those where these vessels were disconnected with the operating microscope (skeletonised). All non-skeletonised flaps survived subtotally or totally whereas total necrosis was observed in 70% of the skeletonised flaps.

Finally a series of haemodynamic studies was performed to test valve competency including extrinsic pressure on the valves. It is concluded that the macrovenous and microvenous pathways, coupled with the variable anatomy of the venous valves, are major factors in determining the survival of reverse flow flaps.

Since Biemer and Stock¹ introduced "the distally based flap" where flow was reversed against the venous valves, the technique has become popular, especially in the reconstruction of defects in the distal limb. The flaps may be raised as an island flap on a stalk of deep vessels or on a skin and subcutaneous pedicle, based on perforators of the deep arteriovenous system. With increasing frequency, new flaps are appearing in the literature which are designed in this way.

By definition, these flaps impose a retrograde venous flow against the anatomical obstruction of the valves. Although they are usually based distally in the body, the terms "distally based" and "reverse flow" are not necessarily synonymous, as there are many situations where venous valves direct blood away from the heart and towards the periphery, for example, the superficial and deep inferior epigastric veins and some of the tributaries of the cutaneous perforators as they converge on a central pedicle.²

The arterial inflow to the flap is easy to comprehend since the arterial framework of the body is a continuous unbroken network of vessels which interconnect by true or choke anastomoses to link with the artery at the base of the flap.³ The precise mechanism of the venous return from the flap however still remains unresolved, although there have been many theories. Obviously, the venous valves must become regurgitant or be bypassed by collaterals since most flaps survive without infarction.

Lin and co-workers⁴ suggested that the venous return "skips" between the *venae comitantes* of the artery, across the interconnecting stepladder of communicating veins, to bypass the valves. These con-

necting veins are very evident in the deep venous system, especially in the limbs, and range from 1 mm up to 3 mm in diameter or more in some cases. We will refer to them as **Macrovenous connections**.

In the same year Timmons⁵ suggested an alternative mechanism and postulated three prerequisites for valve incompetence to allow regurgitant flow.

- (i) There should be venous blood on both sides of the valve
- (ii) The venous pressure proximal (downstream) to the valve should be higher than distal (upstream)
- (iii) The valve should be denervated.

Finally Torii and co-workers⁶ published their work. They postulated that there is reflux of blood between the valve cusps if there is a change in the valve axis, if there is loss of tension in the pedicle of the flap and a high pressure in the proximal pole of the valve.

Although some of the suggestions in each of the three theories are plausible none explains the entire picture as there are certain inconsistencies. Timmons⁷ and Satoh and co-workers⁸ showed that anatomically there were too many valves in the radial pedicle and relatively too few macroconnections between the *venae comitantes* to allow reverse venous flow without encountering the mechanical obstruction of one or more valves, unless the pedicle was short. Torii and co-workers⁹ expanded the studies to encompass other veins in the limbs and obtained comparable results.

A similar criticism can be made about denervation of the valves and the other criteria suggested by Timmons. A free flap or a vein graft containing valves



Fig. 1

Figure 1 Radiographs of the cadaver dissections of the deep venous system of the upper limb (left) and lower limb (right). Lead beads locate the cutaneous perforators. They are labelled: *Upper Limb* (Left): 1. Axillary; 2. Posterior circumflex humeral; 3. Brachial; 4. Profunda brachii; 5. Anastomotic between 2 and 4 veins; 6. Middle collateral; 7. Radial collateral; 8. Superior ulnar collateral; 9. Basilic draining into profunda brachii; 10. Veins accompanying musculocutaneous nerve; 11. Inferior ulnar collateral; 12. Radial recurrent; 13. Anterior ulnar recurrent; 14. Posterior ulnar recurrent; 15. Veins accompanying motor division of radial nerve; 16. Radial; 17. Posterior interosseous; 18. Anterior interosseous; 19. Vein accompanying median nerve; 20. Ulnar; 21. Anastomotic between 17 and 18 perforating interosseous

is denervated when transferred. If placed in the lower limb the flap or graft may be subjected to the other conditions stipulated by Timmons. However, we have never observed varicosities within a free flap and the valves in vein grafts have been shown to remain competent experimentally and in humans.¹⁰⁻¹⁶

Finally it is difficult to accept reflux through the valve as being responsible for flap survival as the pressure needed to produce this is higher than that recorded intraoperatively in a reverse flow flap in man.^{1,6,9}

This paper encompasses three studies which evolved chronologically in an attempt to explore the voids in our knowledge regarding the possible mechanism of the venous flow in reverse flow flaps (RFF). The first is an anatomical appraisal of the location and orientation of the valves in the deep veins of the limbs in a series of fresh human cadaver injection studies. Next is a series of reverse flow flaps in dogs based on the results of these human studies. Finally a number of haemodynamic studies were performed on isolated vein segments.

Human anatomical studies

The investigation was conducted in two fresh subjects. Total body perfusion of the venous system was performed with lead oxide modified from Salmon's original mixture.^{2,17} An upper and a lower limb were disarticulated from each subject. The integument was removed and the cutaneous perforators were identified with lead beads as they emerged from the deep fascia using loupe (2.5 ×) magnification.

The deep neurovascular systems were dissected from each limb. In doing so veins draining muscles were identified with metal staples and black silk ties were used for those veins draining bones or joints. The specimens were radiographed (Fig. 1) and then each vein was dissected under magnification (6 × to 40 ×) to identify the site and orientation of the valves. A tracing was made of each specimen and the veins were colour coded according to the presence (blue) or absence (yellow) of valves (Fig. 2). The study was extremely time consuming and averaged 100 hours per limb. It was completed in 1989.

Results

This study was most rewarding. In addition to confirming published anatomical data of other workers it revealed several new findings. Perhaps the most significant of these was the identification of a rich plexus of

fine veins, free of valves, which surrounded the artery as the vena arteriosa and provided a rich interconnection between the accompanying venae comitantes (Fig. 3). This plexus of tiny veins provided not only a transverse pathway between the venae comitantes, but a longitudinal avenue to bypass valves in the comitant veins. We will refer to them as **Microvenous connections** to distinguish them from their larger counterparts (*vide supra*). They have been mentioned already in our description of the various pathways of the venous drainage of peripheral nerves.¹⁸ This was a constant finding in every situation where an artery was accompanied by venae comitantes, in either the upper or the lower limb, the only difference being the density and disposition of these fine venous radicles.

Their diameters were less than 1 mm and most were between 0.1 and 0.3 mm. Our other findings were as follows:

- 1) Numerous valves were found in the deep venous system of the upper as well as the lower limbs. Except for two unicuspid and one tricuspid they were all of the bicuspid variety. Although more valves were identified in the lower limb the average number of valves per unit length, excluding those identified in collateral channels, was almost identical to that located in the deep venae comitantes of the upper extremity (Table 1). In other words there are more valves in the lower limb because the venous channels are longer.
- 2) A large number of avalvular (oscillating or bidirectional) veins were identified as noted in our previous work^{2,19} which we have once again colour coded yellow (Fig. 2). Typically the macrovenous (as well as the microvenous) connections between venae comitantes were of this avalvular variety. In addition avalvular collateral vein segments were noted in many places which coursed parallel to, and bypassed valved segments in, the main venous channels. Although the avalvular macrovenous connections and collateral vein "detours" reduced the number of valves to be overcome, they did not provide a pathway free of valves to allow in most cases the design of an unobstructed reverse flow flap (Tables 2 and 3).
- 3) In many instances venous valves were noted which directed flow distally in the limb. Typically this occurred at the distal end of an arcade whose "keystone" was a bidirectional or oscillating vein segment or plexus of veins and where the valves in the proximal end of the arch were

membrane; 22. Tributary from anterior carpal (cruciate) network; 23. Posterior terminal division of 18 which pierces interosseous membrane; 24. Anastomosis between 17 and 23 veins; 25. Superficial palmar arch; 26. Deep palmar arch; 27. Anastomosis between radial and cephalic vein in anatomical snuff box; 28. Anastomosis between ulnar and basilic vein at wrist level. *Lower Limb (Right)*: 1. Sciatic nerve; 2. Common femoral vein; 3. Lateral femoral circumflex; 4. Profunda femoris; 5. Superficial femoral; 6. Perforative veins of profunda; 7. Satellite veins of sciatic nerve; 8. Popliteal; 9. Sural entering popliteal; 10. Veins accompanying lateral popliteal (common peroneal) nerve; 11. Nutrient vein of tibia; 12. Tibio-peroneal; 13. Anterior tibial; 14. Peroneal; 15. Posterior tibial; 16. Perforating branch of peroneal; 17. Anterior lateral malleolar; 18. Anastomosis between 16 and 17 veins; 19. Posterior communicating between 14 and 15 veins; 20. Calcaneal branch of peroneal; 21. Dorsalis pedis; 22. Lateral tarsal; 23. Arcuate; 24. Lateral malleolar; 25. Medial plantar; 26. Lateral plantar; 27. Anastomotic between 25 and 26 veins; 28. Anastomosis between dorsalis pedis and long saphenous vein; 29. Stump of first dorsal interosseous; 30. Plantar metatarsal veins.

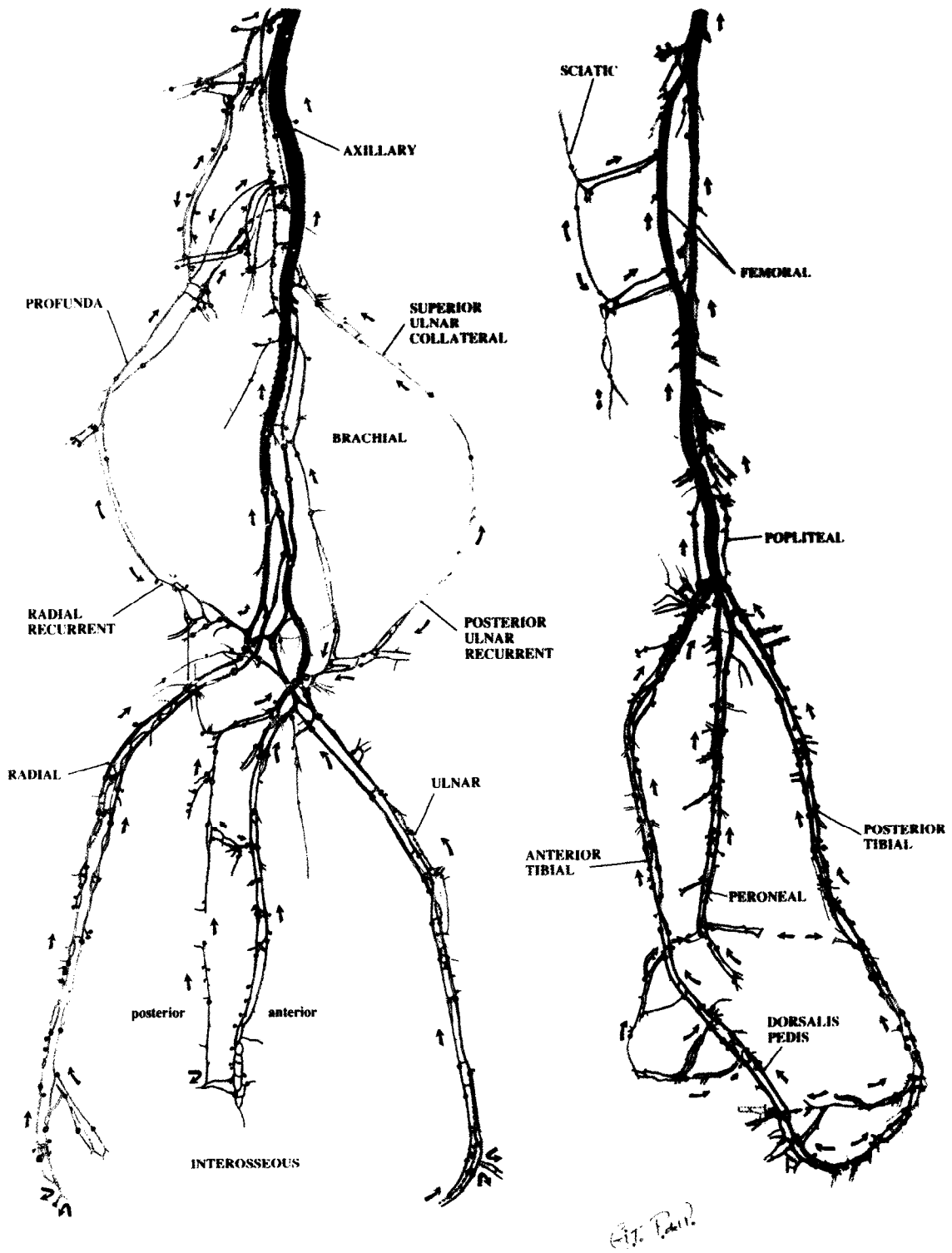


Fig. 2

Figure 2 Diagrams to correspond with Figure 1. Valved veins are coloured blue and avaluular veins yellow. Valve sites (dots and circles) and direction of flow (arrows) are indicated.

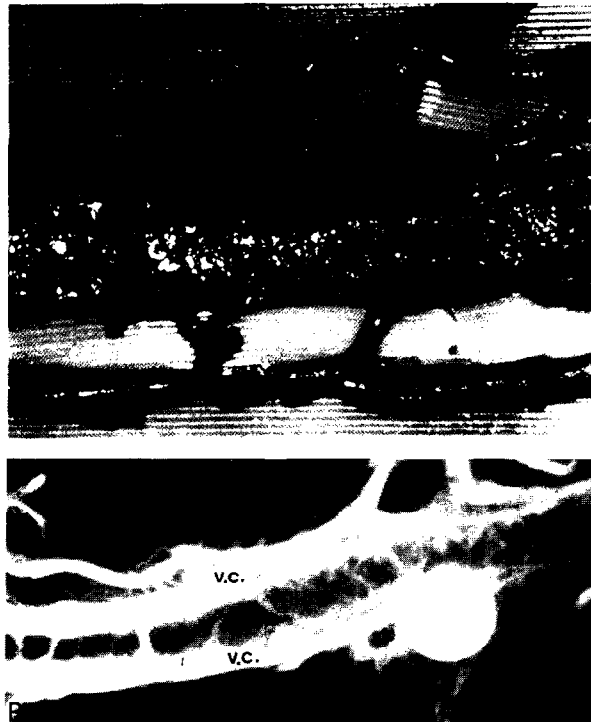


Fig. 3

Figure 3—Human cadaver lead oxide venous injection study (above) and radiograph (below) of the non injected dorsalis pedis artery (a) and its orange stained venae comitantes (v.c.) to demonstrate the macrovenous and microvenous connections. Note the rich periarterial plexus of veins which pass transversely and longitudinally and unite the venae comitantes. The macrovenous connections are partially hidden by the artery in the upper illustration.

Table 1 Valve density and average intervalvular distance in the upper and lower extremities of the two cadavers C1 and C2. All values in a given venous pedicle have been included irrespective of the number of venae comitantes in a given segment. Valves belonging to collateral veins have been excluded.

Pedicle	Length in cm		Number of valves		Inter-valvular distance	
	C1	C2	C1	C2	C1	C2
Anterior tibial	39	37	53	53	0.74	0.70
Posterior tibial	30	29	36	41	0.83	0.72
Peroneal	21	22	19	23	1.10	0.96
Radial	10	20	7	22	1.43	0.91
Ulnar	16	17	19	25	0.84	0.68
Anterior interosseus	13	14	12	13	1.08	1.07
Posterior interosseus	—	16	—	10	—	1.60

oriented in the opposite direction, towards the heart. This was noted, for example, in the arcades formed by the venae comitantes of the superior ulnar collateral and ulnar recurrent arteries, the veins associated with the profunda brachii and radial recurrent arteries or the venous arcades along the sciatic nerve.

Table 2 Maximum unobstructed distance between valves allowing for collateral veins and macroconnections between venae comitantes in cadavers C1 and C2

	Length in cm		Number of valves		Maximal inter-valvular distance		Average
	C1	C2	C1	C2	C1	C2	
Right anterior tibial	39	37	19	19	2.05	1.95	2.08
Left anterior tibial	39	37	18	17	2.17	2.18	
Right posterior tibial	30	29	15	17	2.00	1.71	1.91
Left posterior tibial	30	29	14	16	2.14	1.81	
Right peroneal	21	22	4	9	5.25	2.44	3.08
Left peroneal	21	22	8	11	2.62	2.00	
Right radial	10	20	2	7	5.00	2.85	3.35
Left radial	10	20	3	9	3.33	2.22	
Right ulnar	16	17	9	10	1.77	1.70	1.86
Left ulnar	16	17	7	10	2.28	1.70	

Table 3 Number of valves obstructing reverse flow for different pedicle lengths of commonly used distally based flaps allowing for bypass macroconnections between the venae comitantes. Valves in the radial and ulnar recurrent veins direct flow distally and hence are represented with a negative sign

Pedicle length	Number of obstructing valves to reverse venous flow					
	10 cm		5 cm		2.5 cm	
	C1	C2	C1	C2	C1	C2
Anterior tibial	5	4	3	3	1	1
Posterior tibial	8	5	2	4	1	2
Peroneal	2	5	1	2	0	1
Radial	1	3	0	2	0	1
Ulnar	4	6	1	2	1	1
Posterior interosseus	—	3	—	2	—	1
Radial recurrent	(-1)	0	(-1)	0	(-1)	0
Ulnar recurrent	(-1)	(-2)	(-1)	(-2)	(-1)	(-2)

Canine experiments

Based on the knowledge that in the human the macrovenous connections and collaterals were insufficient to bypass the mechanical obstruction of the valves in most reversed flow flaps, we decided to investigate the part played by the microvenous connections. We chose the dog saphenous arteriovenous system since we were familiar with its anatomy following our vascularised nerve experiments²⁰ as well as the fact that a proximally based island skin flap had been described by Banis and co-workers.²¹ We decided therefore to design a distally based island flap and to compare the survival of skin flaps where the periarterial microvenous connections were left intact (non skeletonised) with those in which this microvenous system was interrupted (skeletonised).

Before this could be done we had to define the anatomy of this new island flap model, to design the experiment and to eliminate spasm as a possible explanation for flap failure. Therefore several initial experiments were conducted.

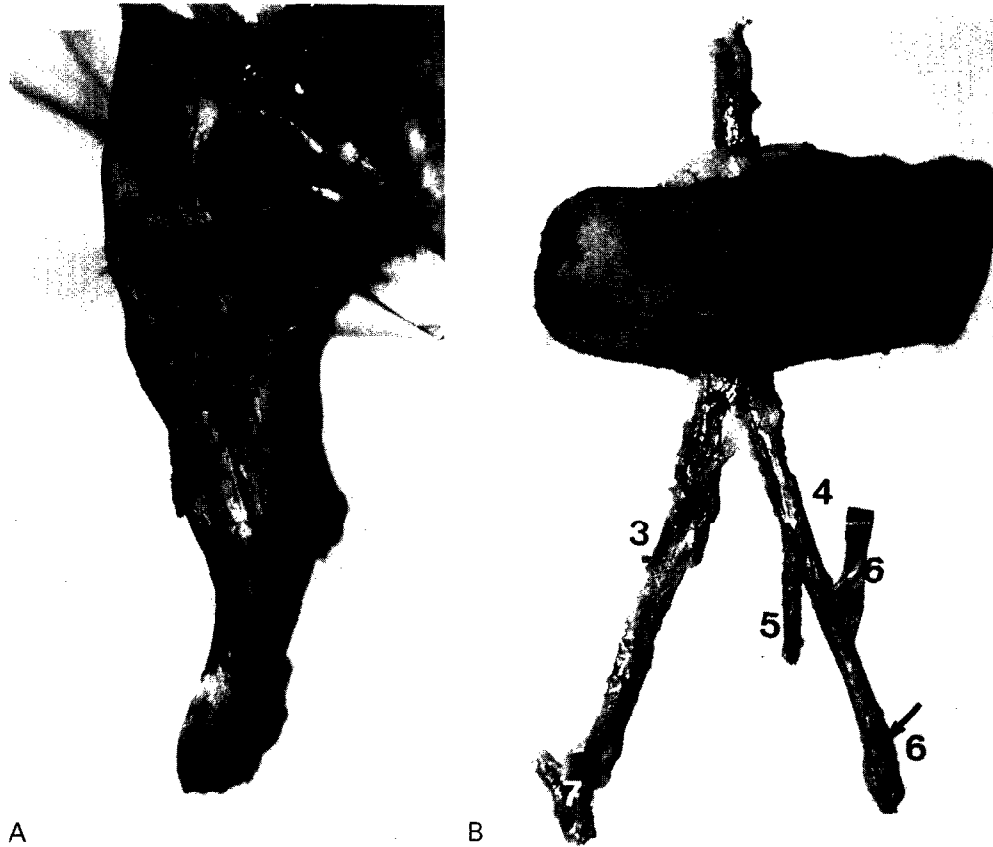


Fig. 4

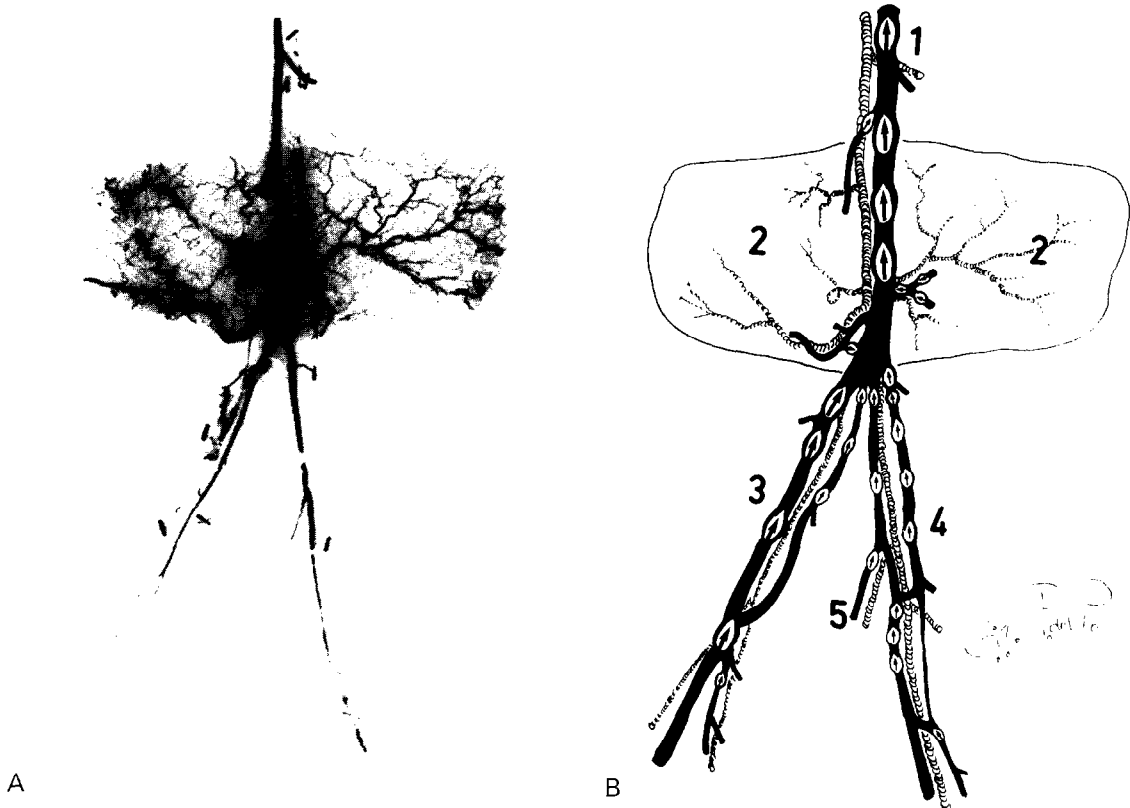


Fig. 5

Figure 4 (Left) Fresh cadaver lead oxide arterial injection and dissection of the dog hind limb showing (1) the saphenous vessels, (2) the saphenous flap, (3) dorsal division of the saphenous system, (4) plantar division of the saphenous system which was used as the pedicle for the reverse flow flap and (5) its peroneal branches. (Right) Island flap removed for radiology and valve mapping. The tibial nerve (6) which courses with the future flap pedicle is evident.

Figure 5 Arteriogram (left) and valve map (right) of the specimen in Figure 4 labelled to match.

Preliminary Studies

First a total body fresh cadaver injection study was performed in the dog in which the arteries were injected with a radioopaque lead oxide mixture²² and a simultaneous injection of the venous system was performed with a radiolucent dye. This allowed us to dissect and locate the valves in the veins of the hind limb of the dog, to remove the proposed flap and to X-ray it in order to locate the site and pattern of supply of the cutaneous arterial perforators.

The saphenous arteriovenous system divides just proximal to the knee into a small dorsal and a large ventral (plantar) system (Fig. 4). The latter was chosen as the pedicle for the distally based skin flap since the venae comitantes were more proportional to their accompanying plantar artery, there were more valves in the system and since the calibre of the dorsal artery was very small and it was feared that it would be of a size insufficient to supply the skin flap (Fig. 5). An unexpected finding was the near absence of macrovenous connections between the venae comitantes and this was verified in all but one of the subsequent *in vivo* experiments. This was fortuitous, as it provided a model where the only collateral circulation which could bypass the obstruction of the venous valves in the pedicle was the periarterial plexus of fine veins.

Next a pilot study was performed in one dog in which bilateral transverse 7.5×5 cm skin flaps were based distally on 9.5 cm pedicles with the microvenous connections divided on one side using the operating microscope. This *in vivo* experiment provided a much clearer picture of the periarterial venous plexus and its communications with the venae comitantes than the canine cadaver study (Fig. 6A).

Both flaps showed venous congestion and necrosed. However on inspection of the pedicles there was an obvious obstruction at a venous valve in the skeletonised pedicle which was not apparent on the other side. In addition the microvenous plexus was clearly evident on the non skeletonised side, suggesting that it was involved in attempting to provide a collateral venous return. We were uncertain whether flap necrosis was related to the siting of the skin flap over sufficient cutaneous perforating veins or whether the pedicle was too long and hence provided an undue overload on the microvenous pathway, especially as there were no macrovenous connections between the venae comitantes. Therefore we decided to increase the dimensions of the skin flap and to shorten the pedicle on each side, a manoeuvre which would favour the possibility of flap survival on the skeletonised side as it would reduce the number of obstructing valves.

Finally, to eliminate trauma or spasm of the pedicle as a possible cause for flap necrosis, we assessed the survival of two skeletonised island flaps where the skin paddle was the same but where the pedicles were designed proximally on the saphenous systems. The pedicles were 7 cm in length and both flaps, whose flow was antegrade, survived totally without evidence of venous congestion.

Definitive Investigation

Ten mongrel dogs were used with weights ranging from 14 to 23 kg. Distally based island flaps were designed bilaterally on the plantar saphenous systems. The skin paddles measured 9×7 cm. The pedicle length was 3.5 cm on the non skeletonised side in every case. In 5 dogs the length of the skeletonised pedicle was the same and in 5 dogs it was shortened by more than 50% to a length of 1.5 cm. One skeletonised flap, which survived totally, was excluded subsequently from the series as dissection revealed the absence of any valves.

Under general anaesthesia the skin flap was elevated and the dorsal division of the saphenous arteriovenous system was ligated. On the skeletonised side the plantar division of the saphenous artery was separated from its venae comitantes, dividing the microvenous connections with the operating microscope (Fig. 6B). A vasodilator (verapamil) was applied to the pedicle and spasm was allowed to decay.

Then the saphenous artery and vein were ligated on each side proximal to the flap. Immediate congestion of all flaps was seen with cyanotic bleeding from the skin margins. In the flap with no valves, which was excluded from the series, there was no evidence of cyanosis. The flaps were sutured in position and post-operative antibiotics (penicillin and benzatylne) and analgesia (buprenorfin) were administered for 3 days. Flaps were inspected regularly and dressings changed daily.

The animals were killed between the 4th and the 15th day, depending on the condition of the wounds. They were subjected to various postmortem studies including antegrade and retrograde phlebography, histological analysis of the pedicles and microdissection of the veins to locate the site of valves.

Results

In each case cyanosis of the flap persisted for 12 to 24 h and either proceeded to necrosis or gradually cleared between 2 and 5 days. The survival rate of the skin flaps is depicted in Figure 7. Total or partial flap survival was noted in every case where the microvenous connections were left intact in the pedicle. When necrosis occurred in these flaps it was confined to one side and was less than 50% of the area.

On the skeletonised side the result was an all or none phenomenon. Three of 10 flaps survived and 2 of these were cases where the pedicle was shortened to 1.5 cm. In the 7 cases of total flap necrosis, the site of obstruction was obvious. The proximal veins in the pedicle were distended with thrombus, the distal vein was collapsed and histology confirmed the obstructive point to be a valve in each case.

Micro dissection of the veins, to locate valve sites either beyond the obstruction or where partial or total flap survival was observed, was abandoned because of tissue friability of the inflamed pedicle. We resorted therefore to phlebography and histology in these cases.

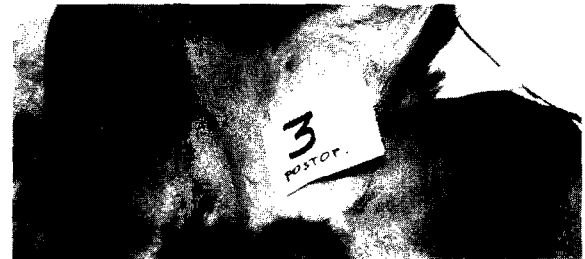


Fig. 7

Figure 6—(Left) *In vivo* study of the saphenous pedicle in the dog showing the periarterial plexus of veins which drain the walls of the artery (a) and interconnect the venae comitantes (v.c.). (Right) Skeletonised saphenous pedicle with forceps indicating obstruction of flow at a valve. **Figure 7** Postoperative course of distally based reverse flow saphenous flaps in 2 of the dogs. In each case the skeletonised flap is on the left of each picture. In the dog on the left there is total necrosis of the skeletonised flap whereas it has survived completely in the other animal. Although the non-skeletonised flap has survived in each case there is some patchy superficial loss in the dog on the right.

Antegrade phlebography, by injecting the lead oxide mixture into a vein on the dorsum of the hind foot, was disappointing. It simply confirmed the site of obstruction in the case of total flap necrosis and showed flow of contrast to the flap when it survived.

Retrograde phlebography however was most rewarding. The stump of the saphenous vein at the proximal margin of the flap was cannulated and injected with the lead oxide mixture. In every case of flap survival venous valve incompetence was noted extending *distal* to the base of the pedicle to reach the foot of the dog. Because of this finding an *in vivo* study was performed before killing the dog. Urografin was injected under fluoroscopic control. It flowed freely to the level of the malleoli, across a short large macro-connection and returned via the saphena parva (sural vein in the human) to the popliteal vein (Fig. 8). It is important to note that the plantar saphenous arterio-venous system courses with the tibial nerve in the dog. Hence valve incompetence occurred over a considerable distance, beyond the field of surgery, where the nerve supply to the veins remained undisturbed.

Haemodynamic studies

Because 3 of the flaps with skeletonised pedicles survived, even though valves were demonstrated intra-operatively and they showed severe postoperative congestion for at least 24 h, we conducted a series of haemodynamic studies which focused on the behaviour of the venous valves. Three different investigations were performed on isolated fresh vein segments. It should be mentioned at this stage that we had measured the venous pressure in the reverse flow saphenous flaps, recorded at the stump of the saphenous vein in both the skeletonised and non skeletonised flaps, which measured 56 cm of water.

Study 1

In 5 experiments a segment of cephalic vein, containing a single valve, was harvested from the forelimb of the dog and submitted to a water column of 275 cm in height. In each case the valve remained competent except for a dribble of no more than a drop every 15 s.

Study 2

In a further 5 experiments multivalvular cephalic vein segments were interposed between 2 columns of water. Proximal (downstream) to the valves the column, which contained methylene blue, was raised to 56 cm to correspond to the venous pressure recorded intra-operatively in the reverse flow flaps. Distally the column of water was raised from 4 to 10 cm to correspond with the physiological venous pressure in the dog.^{23,24} The system was left for a period of 7 h, the time for irreversible venous infarction to take place *in vivo*.^{25,26}

In two cases the system remained competent and the dye did not pass beyond the first valve. In two cases the first valve became incompetent but the remainder remained competent. However in the fifth study the first valve became incompetent after one minute and by 5 min all 3 valves had become regurgitant.

Study 3

A final 5 experiments were conducted in which multivalvular cephalic vein segments were set up as in Study 2. This time extrinsic pressure was applied to the vein just proximal to the first valve in the series (Fig. 9). In 3 of the 5 studies gentle compression of the vein with forceps produced incompetence of the valve and in one study the next valve became incompetent when the manoeuvre was repeated. However the manipulation failed to produce incompetence in the other 2 experiments.

Discussion

One of the basic functions of the venous system is to return blood to the heart while at the same time maintaining a constant pressure in the tissues at the venous end of the capillary bed. Physiologically it must compensate for normal pressure variations which result from factors such as changes in posture, kinking and muscle activity. Anatomically the veins are structured to meet these demands by the presence of valves, a rich interconnecting framework of alternative pathways to bypass potential obstructions and the strategic

Figure 8—Cinefilm of *in vivo* retrograde venogram performed via the proximal stump of the saphenous vein (1) on the seventh postoperative day. The flap overlies the vein between (1) and (2) and its pedicle, highlighted with white arrows, extends to (3). Note that the valves are regurgitant well distal to the field of surgery down to the level of the ankle (black arrows) before the urografin reaches a large connection (4) with the saphena parva (5) which is the sural vein in humans. The urografin then returns via the popliteal (6) and the femoral (7) vein.

Figure 9—Haemodynamic Study 3 in the dog with cannula in proximal end of cephalic vein. (Above) Direction of retrograde flow (white arrow) and site of competent valve (black arrow) are indicated. (Below) Gentle compression renders the valve incompetent and the flow is now arrested at the next valve (smaller arrow). **Figure 10**—Stages of development of the venous valves in a foetus according to Kampmeier and La Fleur Birch (1927) which could explain the variation in contact area of the cusps between valves in the adult. **Figure 11**—Longitudinal section of an obstructing saphenous valve in the dog. Note the diameter of the proximal (D) and distal (d) vein segments, the valve cusps (c), their area of contact (z) and the smooth muscle (r) at the valve base (s). **Figure 12**—Possible explanation of valve behaviour in various situations where pressure is raised in the proximal (downstream) vein segments. The contact area of the cusps is represented as a thick line. (A) Valve remains competent but contact area of the cusps is decreasing as valve expands. Fluid is present in the distal segment. Such a valve

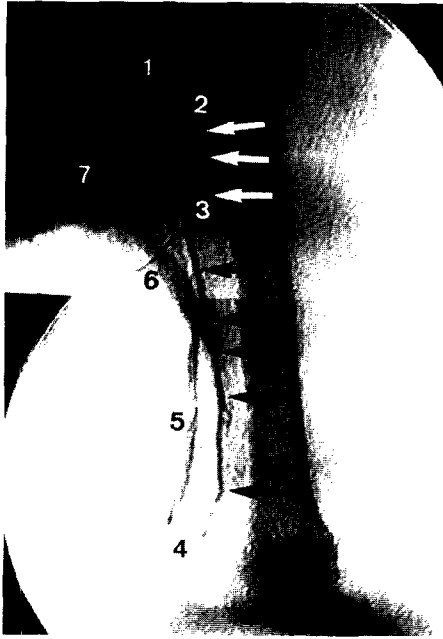


Fig. 8

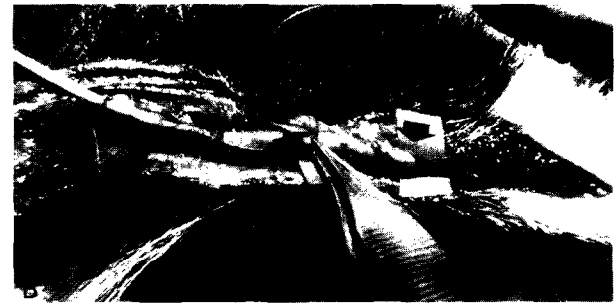


Fig. 9

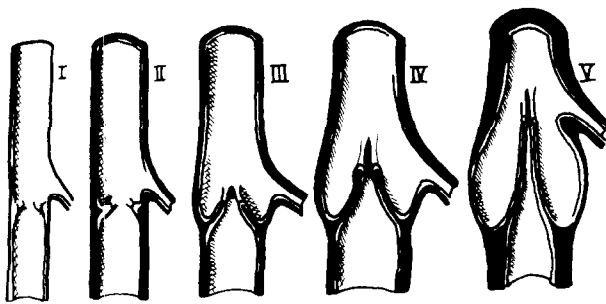


Fig. 10

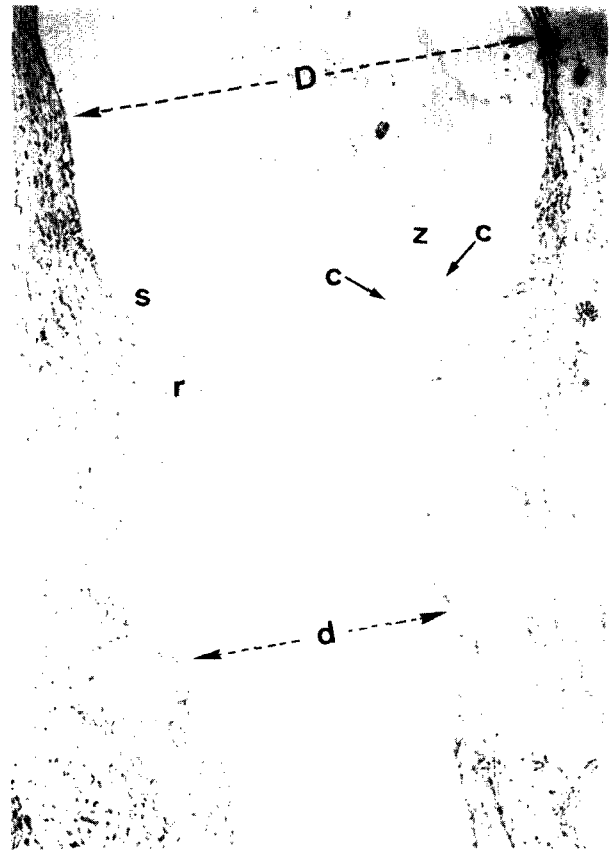


Fig. 11

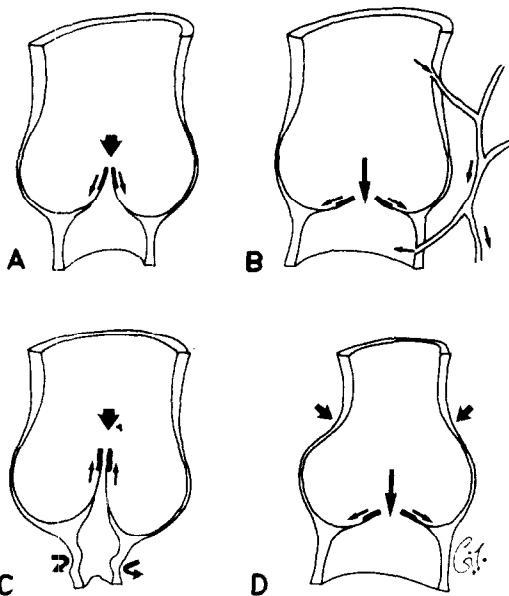


Fig. 12

could become regurgitant in situations where the normal contact area of the cusps is small. (B) Microvenous connections allow blood to bypass valve which becomes regurgitant. (C) Distal (upstream) vein segment is collapsed with no fluid allowing cusps to rotate and resist very high pressure in proximal segment of vein. (D) Distortion of valve due to pinching with forceps or other extrinsic pressure which renders cusps incompetent.

placement of numerous avalvular channels which allow for bidirectional flow. It would seem that these avalvular (oscillating) veins are a clever and sophisticated mechanism which provide for the equilibration of flow and pressure between valved veins which in turn protects the capillary bed from regional fluctuations in pressure.

In our previous anatomical studies of the venous drainage of the integument (skin and subcutaneous tissues), the muscles and the nerves we have noted that the venous network is arranged *within* the tissues as a 3 dimensional system of arcades, usually with avalvular veins situated at their "keystones".^{2,18,19} The study of the deep venous system completes the picture and shows a similar arrangement of the venae comitantes as they course between the tissues. The arcades may be long, for example the loops formed between the anterior tibial, posterior tibial and peroneal systems, or short as constituted by the stepladder of veins which interconnect the venae comitantes of the same system. Although the majority of venous valves direct flow proximally towards the heart there are a significant number of veins, as mentioned already, whose valves direct flow distally. It is for this reason that we prefer the term reverse or retrograde flow for those flaps where the venous return is directed against the obstruction of the valves.

What then is the mechanism and the venous pathway by which island reverse flow flaps, such as the radial, ulna or peroneal, have been shown clinically to survive? Undoubtedly with time the valves become regurgitant. However, as shown in our canine experiment, retrograde flow continues beyond the field of surgery until a major avalvular connection is met with a vein which permits antegrade flow (Fig. 8).

In the dog this continued for a distance of more than twice the length of the dissected pedicle. This would suggest that denervation is not essential for valve incompetence for reasons already stated (*vide supra*). By correlating our results with those of other workers we believe the following factors are involved:

Alternative pathways

Our human cadaver and canine experiments clearly demonstrate avalvular channels at the macroscopic and microscopic level which bypass the valves, either between or along the venae comitantes (Figs 2, 3 and 6).

The large transverse macrovenous connections and longitudinal collaterals provide uninterrupted detours to bypass many of the valves. However, we calculated in the human that they were insufficient to bypass all of the valves until the pedicle length was reduced from 10 cm to 2.5 cm or less (Tables 2 and 3).

On the other hand the microvenous connections do provide an avenue to bypass all of the valves. This is substantiated by our fresh human cadaver studies and the observation that the non skeletonised reverse flow flaps in the dogs survived partially or totally in every case, whereas 70% of the skeletonised flaps showed

total necrosis. However the diameters of these vessels are small and their pathways are plexiform as they surround the artery as the venae arteriosae. This would suggest that a relatively high pressure and a reasonable period of time would be required for the venous return to negotiate this plexus of small veins, for the valves in the venae comitantes to become regurgitant and for the system to equilibrate. This could explain the cyanotic appearance of the non skeletonised flaps, which lasted for 12 to 24 h, especially as macrovenous connections were non existent in the pedicles and the microvenous pathway was the only one available to bypass the valves.

Valve structure

Although the valves in the extremities were nearly all bivalvular this does not imply that their anatomy is identical (Fig. 10). In 1934 Edwards found that the contact area between cusps varied from valve to valve and ranged from 20% to 50% of the vessel diameter. He concluded that distension of a vein necessary to produce valvular incompetence would therefore vary from valve to valve. This could explain why 3 out of 10 of our skeletonised canine flaps survived and why some or all of the valves in 3 of the 5 vein segments became regurgitant in experiment 2 of our haemodynamic studies.

Another observation which may be relevant to valve behaviour is the presence of smooth muscle in the valve cusps (Fig. 11). Smooth muscle normally responds to physical, chemical or nervous stimuli. It is possible that contraction of the smooth muscle may produce valve incompetence and hence further investigation in this area may be warranted.

Intraluminal factors

Our haemodynamic studies would support Timmons' hypothesis that fluid is required on each side of the valve as a prerequisite for reversal of flow. In experiment 1, the vein distal to the valve was collapsed. This could produce increased efficiency of the valve by allowing additional contact of the valve cusps as shown in Figure 12, thus allowing it to withstand a column of water 275 cm in height. This is supported by the fact that 3 of the multivalvular vein segments in experiment 2 became partially or totally incompetent when a fluid column of 4 to 10 cm of water was introduced into the distal end of the vein while at the same time the column of fluid at the proximal end was reduced to 56 cm.

Extraluminal factors

The third experiment in our haemodynamic study suggests that extrinsic pressure on the valve may produce distortion and valve incompetence (Fig. 12). A rise in interstitial fluid pressure:

- (i) would be expected intermittently with muscle activity in the limb and
- (ii) was evident as oedema in the flap and the pedicle when flow was reversed against the venous valves. Whether and how this produces valve incompetence requires further study. However, it could explain why the valves became incompetent in Timmons' experiment, being due to raised interstitial fluid pressure around the vein after injection of local anaesthetic, rather than denervation. This could be resolved by injecting saline around the vein *in vivo* instead of local anaesthetic.

Finally a word of caution should be mentioned with regard to the use of reverse flow free flaps. This has been attempted, but not without problems. Muller and co-workers tried this in 1987²⁷ and the flap became deeply cyanosed requiring an additional antegrade venous anastomosis to salvage the situation. Morrison (personal communication, 1989) encountered the same problem and the flap failed as an additional antegrade anastomosis was not possible. A possible solution to this problem is a staged transfer of the free flap. The strategic use of a delay may allow the valves in the island flap to become regurgitant and the haemodynamics of the system to equilibrate before transfer.

Summary

We feel that the macrovenous and microvenous pathways coupled with the variable anatomy of the venous valves are major factors in determining the survival of reverse flow flaps. However, the effect of extrinsic pressure on the valves and the role of the smooth muscle in the valve cusps warrant further investigation.

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