Small Vein and Artery Pressures in Normal and Edematous Extremities of Dogs Under Local and General Anesthesia

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The pressures within small veins are important because the capillary pressure in open beds cannot be lower than those pressures. Therefore small vein pressures represent minimal values for venous capillary pressures. Furthermore there need be no fixed correlation between small vein pressures and those in the larger, more central veins. Therefore if small vein pressures are regulated, as they may well be, in part by the state of constriction or dilation of the venous bed it seems possible that knowledge about such pressures may shed light upon the mechanism of edema production and tissue water storage in general.

This paper will report measurements in 55 normal and operated dogs of pressures in small and large veins and arteries under several experimental conditions. An attempt has been made to determine the normal values for pressures in arteries and veins less than 0.5 mm diameter in tissues of the foot, and their relation to large artery and vein pressures. Changes in such values under several circumstances will also be reported.

These results and others were reported previously in abstract form (1).

METHODS

Subcutaneous small vein pressure was measured under general anesthesia by the following methods. Mongrel dogs were anesthetized with sodium pentobarbital 33 mg/kg, and placed on their sides. A dorsal foot vein about 1 mm in diameter was selected and dissected free. The proximal end was ligated and a glass capillary tube or a stiff plastic catheter with an outside diameter of 0.2-0.5 mm was inserted in a peripheral direction. The catheter was about 7 cm long and was attached to a 25-cm length of flexible translucent polyethylene tubing (outside diameter 1.0 mm). The polyethylene tubing was attached to a standard 0-6 psi resistance wire pressure transducer (Statham strain gauge) through two three-way stopcocks. To the side arm of the stopcock nearest the catheter was attached a Murphy drip and flask which supplied a continuous flow of heparinized saline except while pressures were being recorded. The side arm of the second stopcock was utilized to expose the pressure transducer to atmospheric pressure at the level of the vein with the foot placed at the height of the dog's heart.

By careful manipulation, the catheter was advanced past valves and bifurcations in a peripheral direction as far as it would go and then withdrawn slightly to a position from which blood could be aspirated. A convenient method for withdrawing blood was by stripping the rubber tubing from the Murphy drip just at the three-way stopcock until blood appeared in the catheter. In this position, the tip of the catheter lay in or near the web between the toes. As determined by dissection, the size of the vessels catheterized by the above methods ranged from 0.2-0.5 mm in diameter at autopsy. With the preparation described, it was possible to record pressure for several hours from the same small vein.

Small ventral foot arteries were catheterized by the same method. The less tortuous course of the small arteries and the absence of valves obviated some of the practical difficulties encountered in small vein catheterization. It was, however, necessary to infuse the saline through the catheter under 100 mg Hg pressure and to heparinize the animal to permit prolonged usefulness of the preparation. The tip of the catheter, in its final position, lay within the substance of the foot pad. The pressure was estimated with a second strain gauge.

Large vein (cephalic or saphenous) pressure was simultaneously measured through a 20-gauge needle inserted into the vessel by venipuncture through the intact skin. Utilizing an indwelling cardiac catheter, femoral artery pressure was also recorded.

Small and large vein pressures were also simultaneously measured under local procaine anesthesia in dogs trained to lie on their sides. The small vein catheter was inserted as described above except that only small stiff plastic catheters were employed. Large vein pressure was measured in the cephalic vein of the same leg by inserting a plastic catheter through a size 21 thin-walled needle placed in the vein by venipuncture through the intact skin. The needle was withdrawn...
leaving the catheter in the vein. Both catheters were sutured to the skin to prevent displacement by movement of the extremity. Catheter patency was maintained during exercise by filling the catheters with heparin solution and tightly occluding their external ends.

RESULTS

Figure 1 presents a typical small vein pressure tracing from a dog under general anesthesia. Several points are to be noted. If the preparations were permitted to remain undisturbed, the mean pressure in small veins in Nembutal-anesthetized animals did not change significantly over long periods of time. However, small cyclic changes in pressure, synchronous with the cardiac and respiratory cycles, were observed in most instances. It is likely that the cardiac and respiratory waves were mainly transmitted from the arterial side since occlusion of the main artery supplying the leg regularly caused these waves to disappear whereas venous occlusion with a tourniquet did not affect them. Further, the contour of the respiratory waves was strikingly similar to and closely followed in time the respiratory waves recorded in large systemic arteries. That transmission of the cardiac and respiratory waves through the tissues was not an important factor was demonstrated by experiments in which open-tipped needles, open-tipped small vein catheters and blunt needles with their tips covered with rubber diaphragms were embedded in the tissues. When connected to the same manometer system as employed in measuring small vein pressure, no regular wave pattern was demonstrated. Figure 1 also demonstrates that venous constriction proximal to the small vein catheter caused a sudden elevation of small vein pressure, a useful test of catheter patency. Small vein pressure was observed to rise as high as 104 mm Hg simply by constricting the leg sufficiently to occlude the venous return. Occlusion of the main artery caused an immediate striking fall in small vein pressure with very little fall in large vein pressure. The pressure promptly returned to its control value following release of either venous or arterial constriction.

Figure 2 presents the frequency distribution of 36 small vein pressures from 33 normal anesthetized dogs. The pressures most frequently observed were in the range 6–10 mm Hg. The median pressure range was, however, 11–15 mm Hg. It will be noted that pressures above 20 mm Hg were occasionally recorded in small veins.

Figure 3 presents the results of simultaneous measurement of small and large (saphenous or cephalic) vein pressures under both local and general anesthesia. Under general anesthesia small vein pressure ranged from 8–28 mm Hg with a mean of 14.6 mm Hg (median 13.0 mm Hg). The large vein pressure from the same leg averaged 4.2 mm Hg (median 4.0 mm Hg). A positive gradient from small vein to large vein was always demonstrable.
However, since there was considerable intra- and inter-individual variation in small vein pressure whereas large vein pressure was relatively constant, the pressure gradient therefore varied with the small vein pressure. Although vessels of approximately the same size were catheterized in each instance, the pressure gradient between the large and small veins in normal dogs under Nembutal ranged from 3–26 mm Hg with a mean of 10.2 mm Hg (median 9.0 mm Hg).

In 19 animals, the femoral artery pressure was recorded simultaneously with small vein pressure. Ten of the animals had mean femoral artery pressures above 110 mm Hg and nine had pressures below that value. Though the former group was found to have a mean small vein pressure of 16 ± 8 mm Hg as compared to 11 ± 6 mm Hg for the latter group, this difference was not found to be significant when subjected to Fisher's $t$ test for comparison of two means, $t = 1.52$, $P = 0.1$–0.2.

Small artery pressure was measured in the pad of the fore foot of 16 dogs under general anesthesia. The mean pressure was 65 ± 25 mm Hg. This is to be compared to a simultaneously measured value of 123 ± 21 mm Hg in the brachial artery of the same extremity. The pressure gradient from large to small artery ranged from 25–85 mm Hg with a mean value of 58 mm Hg.

The contour of the pressure tracings recorded from dogs under local anesthesia was different from those found in animals under general anesthesia in several notable respects (fig. 4). Variations in pressure synchronous with respiration were not apparent. However, in contrast to the stable mean pressure found in anesthetized dogs, most records without general anesthesia exhibited spontaneous fluctuations in pressure of small magnitude (2–6 mm Hg) and short period (5–30 seconds). Further, in two animals, slow, large spontaneous fluctuations in pressure (25 mm Hg

![Fig. 3. Relation between small and large vein pressures recorded simultaneously from the same extremity of dogs under local and general anesthesia.](http://ajplegacy.physiology.org/)

![Fig. 4. Typical subcutaneous small and large vein (cephalic) pressure tracings simultaneously recorded under local anesthesia from fore leg of a normal dog. Note variations in small vein pressure unaccompanied by changes of equal magnitude in the large vein.](http://ajplegacy.physiology.org/)
Small Vein Pressure Unanesthetized Clog

FIG. 5. Large spontaneous fluctuation in subcutaneous small vein pressure observed in a normal dog under local anesthesia. Note relatively stable pressure at about 14 mm Hg for over 4 min. followed by a gradual spontaneous elevation to a maximum of 37 mm Hg at 8 min. Pressure gradually returned to the control value in the following 2 min. Small vein pressure variations were unaccompanied by significant large vein pressure changes.

4.2 mm Hg in the latter. Accordingly, the mean pressure gradient from small vein to large vein was higher by 4 mm Hg in the animals under Nembutal anesthesia.

Table 1 presents the small and large vein pressures in the fore and hind legs of four dogs with hind leg edema occurring subsequent to the establishment of chronic iliac and femoral arteriovenous fistulae. It will be noted that the small vein pressure in the edematous limbs averaged 33 mm Hg as compared to 11 mm Hg in the nonedematous limbs. The former value is well above the expected colloid osmotic pressure of dog plasma (2, 3). Of particular interest was the elevated pressure gradient from small vein to large vein in the edematous limb as compared with the nonedematous leg in three of the four animals studied. Thus, the elevation of small vein pressure cannot be completely accounted for by the degree of large vein pressure elevation. Figure 6 is a pressure record from one of the animals.

The effect of exercise upon fore leg small and large vein pressure was tested in four normal trained dogs. After recording control pressures at rest, the catheters were disconnected from the strain gauges, filled with heparin, tightly stoppered and taped to the animal's leg without disturbing their positions in the veins. The animal was then made to run for 150 yards at approximately 5 miles/hr. and the pressures again measured. The results of the experiments are tabulated in table 2. It will be noted that the pressures increased both in the small and large veins but to a greater extent in the former than the latter. The result was an elevation in small-to-large vein pressure gradient in each case.

DISCUSSION

The pressures in the smaller veins as measured in this study are of interest because they show values which are 'spontaneously' variable and are quantitatively more different from those in the usually studied peripheral veins than might have been predicted. It is not suggested that the small vein pressures measured need be close to capillary pressures, but they cannot be higher than those pressures on the average. This must be true because velocities are higher in veins than in capillaries. In many instances the catheter employed occluded the vein it was in, and the pressure measured was then a collateral vessel pressure (4). The data presented shows that about 8%, on the average, of the pressure-drop around the entire circulatory system occurs between the veins of 0.2-0.5 mm bore and the veins of 5-10 mm bore.

**Table 1. Small and Large Vein Pressure as Related to Edema in Anesthetized Dogs**

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Fore Foot</th>
<th>Hind Foot</th>
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<tbody>
<tr>
<td></td>
<td>Small vein</td>
<td>Large vein</td>
</tr>
<tr>
<td>1</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>2</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>15</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>Average</td>
<td>11</td>
<td>6</td>
</tr>
</tbody>
</table>

* Edema due to chronic iliac and femoral arteriovenous fistulae.
† Edema graded 0–4 where 0 indicates no edema and 4 maximal edema.

*The animals were generously supplied by Dr. C. W. Lillehei and Dr. Hammerstrom of the Department of Surgery.
diameter. The fraction can vary in unanesthetized dogs from 4–25%. Thus the bore of these small veins may play a significant role in determining the pooling and the flow of the blood. Also, by influencing the capillary pressure the small vein pressure will have a controlling effect upon water and salt distribution between the blood and the tissue spaces.

The occurrence of spontaneous cyclic changes in small vein pressure, without concomitant fluctuations in large vein pressure strongly suggests that the small vein system is subject to nervous or humoral control. Positive proof for this suggestion is lacking, however, because small artery pressures and flow rates have not been made under local anesthesia. General anesthesia with Nembutal abolished the cyclic changes in small vein pressure. Therefore the question of mechanism is still open.

The existence of spontaneous fluctuations in small vein pressure provides unequivocal evidence, however, that the pressures in capillary beds are not static over time. This observation and the fact that when measurements are made at two sites in the same animal the values are not identical, leads one to suggest that the opening and closing of vascular beds which is evident upon microscopic examination of capillaries and venules exerts its effect upon pressures. Thus, in addition to a pressure gradient along the length of a capillary, there appears also to be a temporal fluctuation which allows a whole length of capillary to have at one time a pressure well above the colloid osmotic pressure, and sometime later a pressure much lower. So the extrusion of tissue space fluid may alternate with its reabsorption in a temporal as well as, or instead of, a spatial sequence.

The direct measurements of small vein pressures in the normal and pathologic extremities of dogs in which edema had been induced by chronic A-V anastomoses confirm the dependence of edema of this type upon hydrostatic factors. In view of the fact that many clinical investigators have been prone to ascribe the edema of heart failure to a primary action of the kidney in retaining water and salt without reference to blood composition it seems important to stress the fact that in the present situation the excess hydrostatic pressure over normal colloid osmotic pressure levels allows no justification for postulating another mechanism for edema production. It may be noted that the same is true in a variety of situations in which pulmonary edema occurs (2, 5–10),

![Figure 6](http://ajplegacy.physiology.org/)

### Table 2. Effect of Moderate Exercise Upon Fore Leg Small and Large Vein Pressure in Four Normal Dogs*

<table>
<thead>
<tr>
<th>Before Exercise</th>
<th>Immediately After Exercise</th>
<th>5 Min. After Exercise</th>
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<tbody>
<tr>
<td></td>
<td>Small vein pressure</td>
<td>Large vein pressure</td>
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<tr>
<td></td>
<td>Small vein pressure</td>
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<td></td>
<td>Small vein pressure</td>
<td>Large vein pressure</td>
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<tr>
<td>14.0</td>
<td>6.0</td>
<td>8.0</td>
</tr>
<tr>
<td>8.0</td>
<td>6.0</td>
<td>2.0</td>
</tr>
<tr>
<td>10.0</td>
<td>6.0</td>
<td>4.0</td>
</tr>
<tr>
<td>11.0</td>
<td>6.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Average</td>
<td>10.8</td>
<td>6.0</td>
</tr>
</tbody>
</table>

*Values in mm Hg.
and also in the case of peripheral edema following pulmonic stenosis (11) in dogs.

Special attention is called to the observations in relation to muscular exercise. The factual observations are clear-cut and certain deductions appear to be justified, although a complete analysis of the mechanism of the changes must await further information. The rise in small vein pressure to values equal to the normal blood colloid osmotic pressure has obvious implications. There can be filtration of fluid into subcutaneous tissues by this mechanism in exercise. Furthermore it is obvious that the pressure rise in the large veins in exercise is much smaller than the small vein rise. Quantitatively this difference is measured by the change in the 'gradient' from rest to exercise. Reliance upon large vein pressure changes to indicate what is going on at the periphery is obviously improper.

SUMMARY AND CONCLUSIONS

Methods are described for measurement of pressures in arteries and veins of inside bore 0.2–0.5 mm in the dog's foot.

Observations have been made upon such 'small artery' and 'small vein' pressures under various circumstances, in relation to large artery and large vein pressures in the same extremities. In normal waking dogs the small subcutaneous vein integrated mean pressure in the foot ranges from 8–25 mm Hg with 13.1 mm Hg as the mean value. The pressure mean is not greatly different under Nembutal anesthesia. The large vein pressures average higher without general anesthesia; therefore the pressure gradient from small to large vein is greater under Nembutal. A significant correlation was not demonstrated between either small and large vein pressures or between small vein and systemic arterial pressures in dogs under general anesthesia.

Under local anesthesia, large fluctuations occurred in small vein pressure, not associated with significant changes in large vein pressure. The findings suggest that the small vein system may be subject to nervous and humoral control and further, that the pressures in capillary beds are not static over time.

The mean value for small foot pad artery pressures was 65 mm Hg as compared to 123 mm Hg in the large arteries.

Exercise was followed by a large elevation in small vein pressure and a small rise in large vein pressure.

In dogs with large arteriovenous anastomoses involving the hind legs, the local small vein to large vein pressure gradient was elevated in three of four animals. Edema in such preparations was seen only in cases where small vein pressures approached the value of the plasma colloid osmotic pressure.

REFERENCES