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COMPARISON OF FLUID TRANSPORT SYSTEMS IN LYMPHATICS AND VEINS

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ABSTRACT

In the anesthetized sheep, pressure pulses generated in the feet are transmitted downstream in the veins but not in the lymphatics at normal intralymphatic pressure. When the sheep is tilted on a tilt table, gravitational changes occur in venous pressure but not in the pressure in adjacent lymphatics. These results suggest that in limb lymphatics, unlike limb veins, the column of fluid is incomplete. This makes extrinsic pumping less effective for propelling fluid in lymphatics than in veins. At normal intralymphatic pressures, intrinsic pumping seems to be mainly responsible for lymph propulsion. The incompleteness of the fluid column in lymphatics might also protect these vessels against the hydrostatic problems experienced by veins during gravitational stress.

Though veins contain valves, the fluid column along them is usually complete. Accordingly, when a person changes from the recumbent to the upright position, venous pressure in the feet rises by the hydrostatic equivalent of the column of the blood between the heart and the feet (1). This tends to cause venous pooling in the lower extremities and a decrease in venous return to the heart. The problem can be overcome to some extent by the "venous" or "muscle" pump. Compression of the veins by surrounding structures such as contracting skeletal muscles will, in the presence of valves, drive blood back towards the heart. There is little evidence that the lymphatic system is affected by similar gravitational problems, and this may be because the column of fluid in the lymphatic system is not normally continuous. The present experiments were designed to study this problem.

MATERIALS AND METHODS

Experiments were performed on the limbs of sheep anesthetized with pentobarbitone (10-30mg/kg IV) and halothane (1-3% in O_2). Afferent lymphatics were cannulated in the metacarpal or metatarsal regions with PVC tubing (0.28-0.5mm internal diameter) and connected to appropriate transducers to measure lymph outflow pressure and flow (2). Intravenous and intralymphatic pressures were measured in some experiments by connecting intravenous and intralymphatic catheters to pressure transducers. Pressure pulses were applied to the hoof region by a pneumatic cuff inflated rhythmically from an automated pressure source. In some experiments the sheep was strapped by harness to a tilt table to study the effects of change in posture on lymphatic and intravenous pressure.

RESULTS

Transmission of pressure pulses in lymphatics and veins

Fig. 1 shows the effect of intermittent compression of the sheep's foot on lymph flow and outflow pressure from a

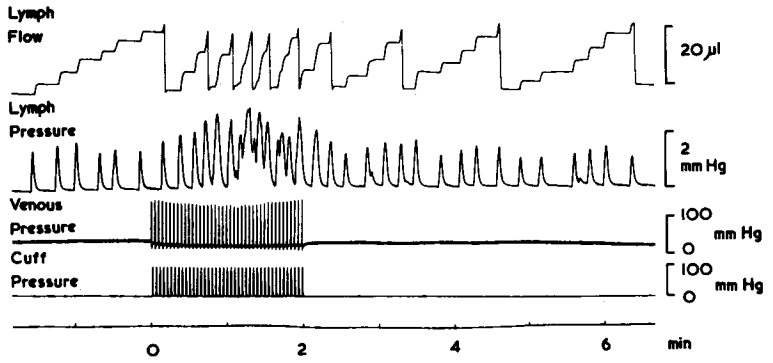

Fig. 1. The effect of intermittent compression of the foot with a pneumatic cuff on lymph flow, lymph outflow pressure, and venous pressure in the metatarsal region.

catheterized lymphatic in the metacarpal region about 10cm downstream and on venous pressure from a catheter in an adjacent vein. At the beginning of the experiment the lymphatic contracted spontaneously, and each contraction extruded a small quantity of lymph which appeared as a step increment in the flow record. During the 2 min period when the foot cuff was inflated intermittently, each foot compression caused a synchronous pressure transient in metacarpal venous pressure but similar transients were not seen in the lymphatic outflow pressure record. The frequency and amplitude of the spontaneous lymphatic contractions increased, and this was associated with a concomitant increase in lymph flow.

The fact that pressure pulses were transmitted downstream in veins but not in the lymphatics would suggest that either the column of fluid in the lymphatics was not continuous or that the walls of the lymphatic were so compliant that the pulses were damped out between the compression and the recording site. The former is the more likely explanation. It seemed possible that the failure of lymphatics to transmit pulsations in this sort of experiment might be due to the fact that the outflow catheter was open to atmosphere. If this allowed the intralymphatic pressure to fall to an abnormally low value, the lymphatic might collapse between contractions and so damp out transmitted pulses by breaking the fluid column. To test this hypothesis we looked at the effect of intermittent foot compression at normal intralymphatic pressure using a fine catheter (24G, 3/4" Abbocath) to catheterize the lymphatic. The results are shown in *Fig. 2*. Before compression, intralymphatic pressure was about 5mmHg and showed the typical irregular pulsations (rate 2-4mm, amplitude 2-10mmHg). Venous pressure was about 20mmHg and fairly steady. Again, foot compression caused the typical synchronous pressure transients in the vein but in the lymphatic, similar transients were not seen. However, the rate and amplitude of the spontaneous lymphatic contractions increased due presumably to the associated increase in lymph flow. The amplitude of the pressure pulses rose to almost 20mmHg. When foot compression stopped, the venous pressure transients ceased immediately but the enhanced spontaneous activity in the lymphatic took several minutes to return to normal. This sort of experiment showed that even at normal intralymphatic pressure values, pressure pulses were not transmitted along lymphatics as they are in veins.

However, since veins and lymphatics have basically similar structures it was felt that there must be some intralymphatic pressure at which the lymphatic system would be sufficiently full of

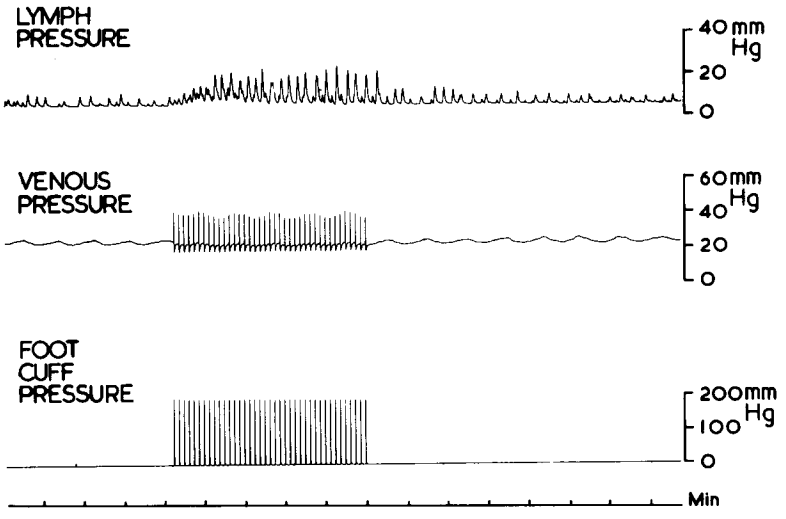

Fig. 2. The effect of intermittent compression of the foot with a pneumatic cuff on intralymphatic and intravenous pressure in adjacent vessels in the metatarsal region.

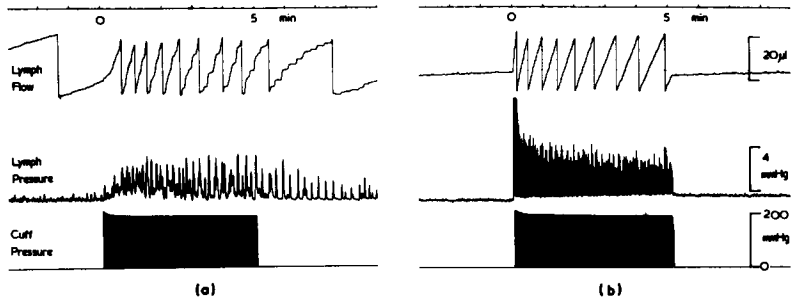

Fig. 3. The effect of increasing lymphatic outflow pressure on the lymph flow and pressure responses to intermittent foot compression with a pneumatic cuff. a) outflow pressure = 0. b) outflow pressure = 80mmHg.

lymph to be able to transmit pulsations. Two sorts of experiment were carried out to test this concept. One method was to obstruct lymph outflow from a cannulated lymphatic by raising the catheter outlet to a level where the lymphatic "failed" and could not expel lymph against the gradient by its own intrinsic contractions. *Fig. 3* shows an experiment of this type. The left hand record (a) shows the effect of intermittent compression when the outflow pressure was zero. The cuff pressure transients did not cause synchronous lymphatic pressure transients but caused an increase in the rate and amplitude of the spontaneous lymphatic pressure pulses associated with the increase in lymph flow. In the right hand record (b), outflow pressure was raised to 80mmHg at which level resting lymph flow was reduced to almost zero. In this case each hoof compression caused a synchronous pressure transient in lymph outflow pressure and resulted in the extrusion of a small quantity of lymph. Lymph flow started abruptly with the first compression and ended abruptly with the last. At these very high intralymphatic pressures where intralymphatic volume was presumably raised, pressure pulses were transmitted readily downstream in the lymphatics. In this situation lymph propulsion was due entirely to external factors, i.e., the hoof com-

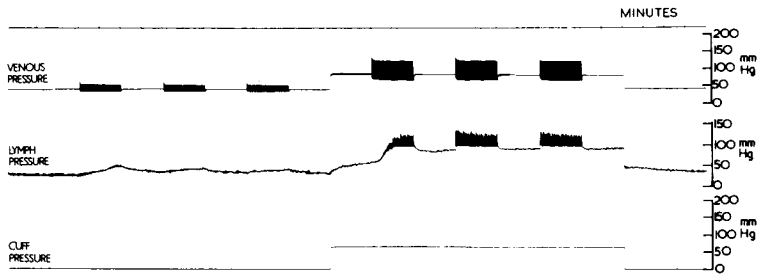

Fig. 4. The effect of inflating a congesting cuff to 70mmHg on the proximal limb on the responses of intralymphatic pressure and intravenous pressure to intermittent foot compression with a pneumatic cuff.

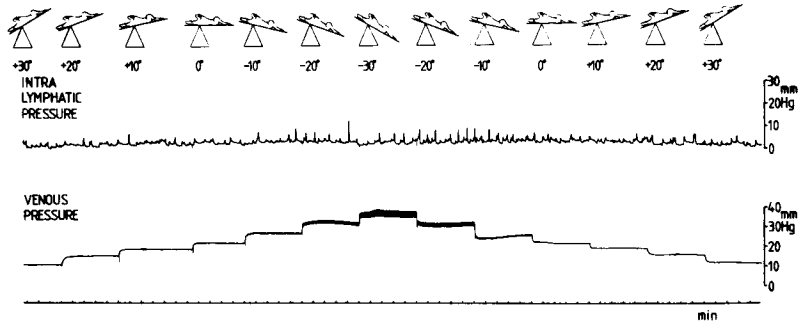

Fig. 5. The effect of tilting the sheep on a tilt table on pressure in an adjacent lymphatic and vein in the metatarsal region.

pression. At these high pressures "extrinsic pumping" was very effective for lymph propulsion.

A second method used to raise lymphatic pressure was to inflate a pneumatic cuff on the proximal part of the limb. *Fig. 4* shows an experiment in which intravenous and intralymphatic pressure was measured in adjacent vessels in the metacarpal region. In the first part of the experiment, three 2-minute periods of intermittent foot compression caused the typical synchronous transients in venous pressure. In the adjacent lymphatic, pressure rose, but similar transients were not seen. When the pneumatic cuff on the proximal part of the limb was inflated to 70mmHg, venous pressure rose rapidly and lymphatic pressure rose more slowly toward that level. During this phase, intermittent foot compression caused pressure transients in both the vein and the lymphatic. However, the lymphatic transients were only seen when intralymphatic pressure had risen to about 100mmHg.

Changes in venous and lymphatic pressure with changes in posture

Fig. 5 shows the result of an experiment with an anesthetized sheep on a tilt table. At the beginning of the experiment the sheep was tilted feet up at an angle of 30° above horizontal. Venous pressure was steady at about 10mmHg and lymphatic pressure about 3mmHg and showed the usual irregular pulses. As the table was tilted in stages to 30° below horizontal, venous pressure rose in step increments. Lymphatic mean pressure did not change much, but there may have been a slight increase in the frequency and amplitude of the pressure pulses. *Fig. 6* shows the result of a similar experiment where the resting level of lymph flow was increased throughout by applying intermittent foot compression.

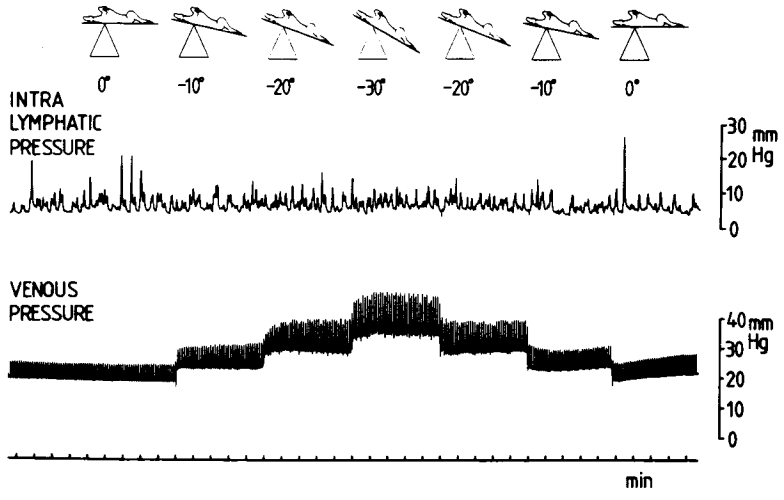

Fig. 6. The effect of tilting the sheep on pressure in an adjacent lymphatic and vein in the metatarsal region when lymph flow from the foot was increased by intermittent foot compression.

At the higher lymph flow rates there was more spontaneous activity in the lymphatic, but again there was no change in the mean intralymphatic pressure with gravitational stress. Intravenous pressure on the other hand increased in steps as the sheep was tilted feet downwards. Each foot compression caused the usual pressure transient in the vein, but this was not seen in the lymph pressure record.

DISCUSSION

Though lymphatics resemble veins in that they are thin-walled valved conduits that return fluid from the periphery, they face different physiological problems. In veins the energy to drive blood comes mainly from the heart. The heart's contractions provide a pressure head in the peripheral veins and venules which is sufficient to overcome venous vascular resistance. This energy may be supplemented from time to time, especially in dependent parts, by the "muscle" or "venous" pump; in the presence of valves, compression of the veins by surrounding structures will drive blood towards the heart.

In lymphatics, the heart does not provide the energy to drive lymph. Lymphatics are blind-ended tubes and have no direct connections with the blood vessels in the tissues. Present evidence suggests that the energy for lymph propulsion comes mainly from the lymphatics themselves. Lymphatic smooth muscle behaves rather like heart muscle in that it beats rhythmically (3) and, with its valves, can propel fluid (4). This behavior allows lymphatics to act as pumps where the output can be varied by altering pump rate, pump stroke volume, or both (2). At high flow rates lymphatics can generate pressures as high as those seen in arteries and such high pressures are needed to overcome the considerable resistance offered by the lymphatic system (5). The pump action can be modulated by local factors, such as filling pressure and temperature to meet the needs of local lymph clearance. It can also be modulated by sympathetic nerve activity (6,7) and circulating hormones (2) to meet the general needs of the body for lymph return. The energy required for lymph transport could also be augmented by a lymphatic analog of the "venous pump", i.e., external rhythmic compression of the lymphatics by external structures. However, there is not much evidence that this phenomenon plays a major part in lymph transport in

the limbs. Olszewski and Engeset found that muscular activity in and massage of the human foot caused increases in local lymph flow (8). However, lymph propulsion did not occur synchronously with the muscle or massage movements. Lymph only flowed when the spontaneous contractions of the lymphatics themselves raised intralymphatic pressure.

The results of the present experiments in sheep feet are in complete agreement with this finding. At normal intralymphatic pressure the lymphatics are insufficiently filled with lymph for external pumping to be a very effective means of lymph propulsion, perhaps because the "pump" is inadequately "primed". In these conditions intrinsic lymphatic pumping would be mainly responsible for lymph propulsion.

However, if intralymphatic pressure is raised to very high levels, as might happen when the intrinsic contractions were not strong enough to expel the lymph they contained, or when the lymphatic was partially obstructed, the "pump" would become sufficiently "primed" for extrinsic pumping to become a very effective means of lymph transport.

The fact that lymph pressure was not much affected by gravity would again support the idea that the column of fluid along the lymphatic is not continuous at normal intralymphatic pressures. This situation should help the lymphatics to clear tissue fluid from dependent limbs since they would not need to work at the high pressure levels seen in the veins in the upright position. However, in lymphatic obstruction it is probable that intralymphatic pressures would rise to the level where the fluid column was continuous so that this advantage would be lost.

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