THE EFFECT OF BLOOD VESSEL PULSATIONS ON LYMPH PRESSURE IN LARGE LYMPHATICS

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Rhythmic alterations in lymph pressure at the same rate as the heart beat have long been believed on circumstantial grounds to be an important factor in the propagation of lymph. It was not until 1939, however, that the presence of such lymphatic pulsations was demonstrated by Cressman and Blalock (1), who suggested that the impulses were transmitted from contiguous arteries. By measuring lymph output from local areas to which extrinsic pulses could be imparted or withdrawn at will, Parsons and McMaster (2, 3) have shown that lymph formation and drainage are remarkably facilitated in regions subjected to transmitted arterial pulsations.

Because it had never been proved that rhythmic intralymphatic pressure changes are actually due to transmitted arterial pulsations, the present study of the origin and fate of intralymphatic pulsations was undertaken.

METHODS

Thirty-five large mongrel dogs were used, and technically satisfactory conditions were realized in 20 experiments. The dogs were given condensed milk and 30 minutes later were anesthetized with 30 mgm. per Kg. of nembutal given intravenously. In most of the experiments the chest was opened and a positive pressure respirator used thereafter. Umbilical tapes for controlled occlusions were placed about the aorta, the two vessels arising from the aortic arch, and the left innominate vein through a thoracotomy in the left 3rd interspace. The abdominal aorta was isolated through a separate incision. Arterial, venous and lymphatic vessels were cannulated with polyethylene catheters or blunt 20 gauge steel needles filled with heparin. Inductance type transducers were used to measure arterial and venous pressures; lymphatic pressures were detected with a capacitance manometer. All pressures were recorded simultaneously on a multichannel direct writing oscillograph. The frequency response of the measuring and recording systems employed was in excess of 50 cycles per second when tested by recording pressure within a balloon during explosion.

Two sites were chosen for study: the lower portion of the thoracic duct, which was exposed at the level of the 11th and 12th thoracic vertebrae through a thoracotomy in the 11th left interspace, and the terminal portion of the thoracic duct, which was exposed through a left supraclavicular incision. In each location pressures were obtained either by cannulating a tributary so that the main lymphatic channel was unobstructed, or by directly cannulating the thoracic duct in which case the central lymph flow was blocked. In this paper the former method is referred to as *side pressure measurement*, and the latter as *end pressure measurement*.

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In order to measure end pressures the lymphatic channel was mobilized over a distance of 8 to 10 mm. and lifted carefully so that the recording system and lymph vessel would be completely free of contact with any other structure, insuring that any pressure alterations recorded were discretely intralymphatic. Side pressures were obtained by placing the polyethylene tube or blunt steel needle through a tributary until its tip reached the main lymphatic channel. In this situation all other vessels in the immediate vicinity which might impart movement artefact to the recording system were retracted or removed.

RESULTS

Recording from the thoracic duct just above the diaphragm. Mean side pressures ranged from 35 to 55 mm. water. After several minutes of obstruction to flow,

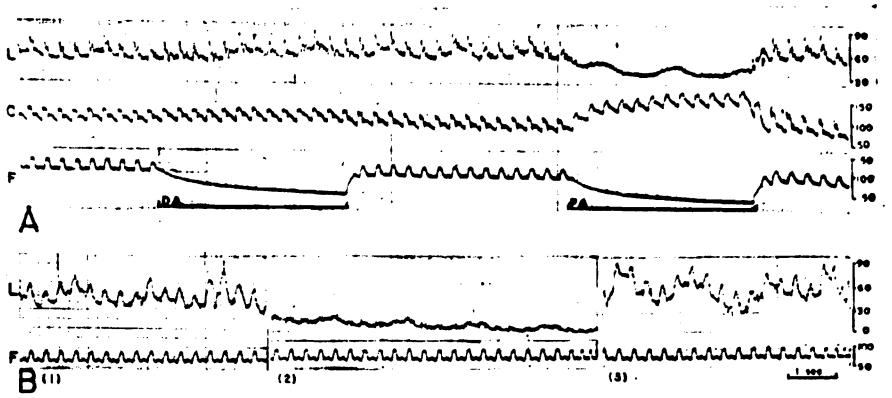

FIG. 1A. Side pressure recording from thoracic duct at the level of T_{12} showing lymphatic pulsations (L) and how these were unaffected by aortic occlusion below the renals (DA) and eliminated by occlusion at the first intercostal level (PA). Concurrent right common carotid (C) and left femoral (F) arterial tracings are shown.

B. Similar record (1) in which a ligature tightened in position to prevent lymph from reaching the tip of the recording catheter obliterated both pressure and pulsations (2), with restoration of the original pattern after ligature was loosened (3). Lymph pressure calibrations are in mm. saline, and arterial pressures in mm. mercury.

end pressures were as high as 350 mm. water, but by intermittent release of the lymph between recordings the pressures were generally maintained at less than 100 mm. water. There were only two types of demonstrable variations from the mean level. The first type consisted of respiratory alterations (Figs. 1–3), which could be kept at a minimum by maintaining the positive pressure respirator at a low exchange. The second type of pressure variation consisted of rapid lymphatic pulsations in phase with the heart beat, which were essentially the same irrespective of whether side or end pressure furnished the background. The pulse pressure of these rapid lymphatic cycles was surprisingly great, usually being 20 to 30 mm. water and amounting to a significant fraction of the peak lymph pressure. Thus typical pressures in millimeters of water could be tabulated as 70 systolic and 40 diastolic, or 55 systolic and 35 diastolic.

The pattern of the lymphatic pulse tracing was variable from animal to animal and at different times in the same animal, sometimes being very sharp with complex form (Fig. 4A), and at other times being a simple wave (Fig. 1B). In all instances, however, the chief (always the first) component had a fixed time relationship to the arterial pulse (Fig. 4A), starting after the carotid and before the femoral pulsations in the position one would predict for adjacent aortic pulsations.

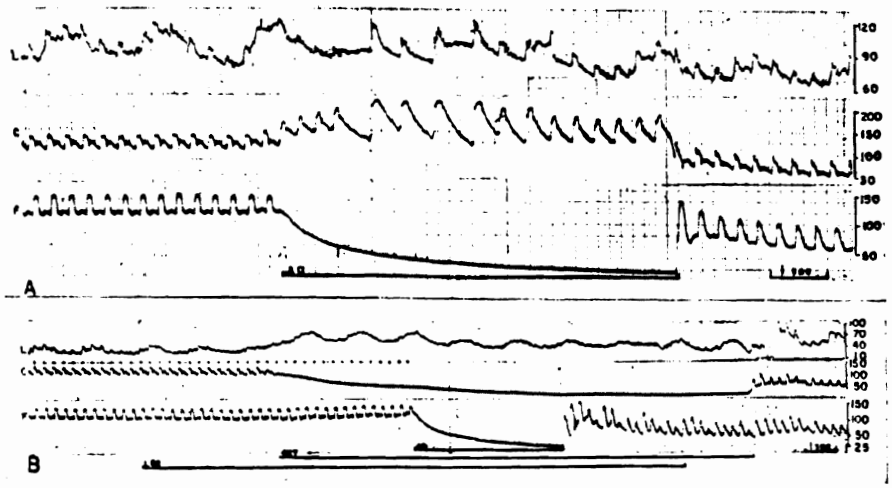

FIG. 2. Side pressure recordings showing effect upon pulsations in the cervical thoracic duct (L) of: A. Occlusion of the aorta at the first intercostal (AO), causing a carotid hypertension with concurrent augmentation of the lymphatic pulsations. B. Serial occlusion and release of the left subclavian (LSC) and brachiocephalic (BCT) arteries, and the thoracic aorta at the first intercostal (AO).

Right common carotid (C) and left femoral (F) arterial pressures are included. The vertical indicator dashes beneath the lymph recording in B show the time of onset of femoral pulses. Lymph and venous pressure are in mm. saline, and arterial pressures are in mm. mercury.

Occlusion of the aorta distal to the site of the lymphatic catheter had little or no effect on the lymphatic pulsations (Fig. 1A), whether the occlusion was at the level of the superior mesenteric artery, renal arteries, or at the bifurcation. Similarly, occlusion of the ascending lymphatic channels at any point 2 or 3 cm. or more below the level of the cannulation did not appreciably alter the pulsations although the mean lymphatic pressure generally fell.

On the other hand, occlusion of the aorta proximal to the lymphatic cannula always completely obliterated the intrinsic lymphatic pulsations (Fig. 1A), usually with an attendant fall in mean lymphatic pressure. Pulsations promptly reappeared upon release of the aorta.

That the pulsations recorded were representative of pressure events in the

lymph rather than of rhythmic catheter movement or other artefact, was evident from several considerations. With either end or side pressure measurement, when the tip of the lymph filled catheter was trapped by a ligature *in situ* so lymph could not reach it (Fig. 1B), the pressure fell and pulsations ceased, to return again when intralymphatic contents were brought back into

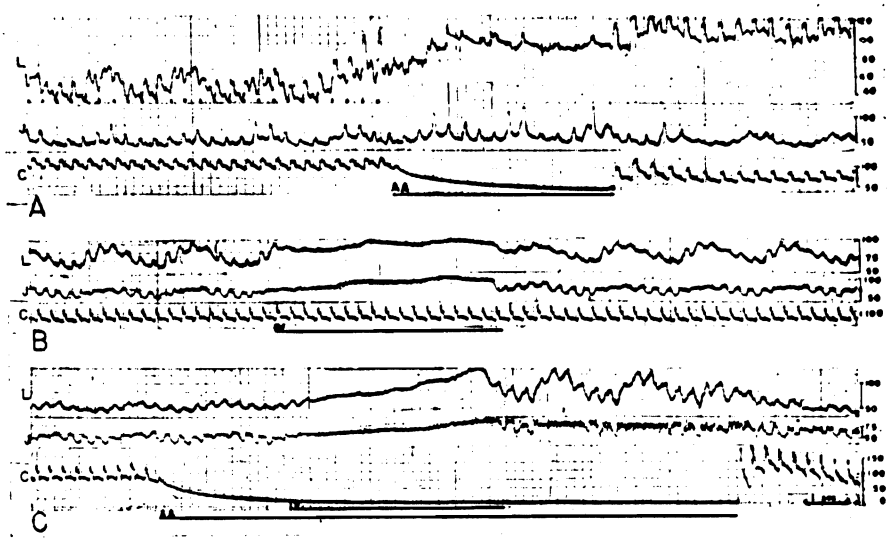

FIG. 3. Side recordings from the cervical thoracic duct (L) showing in each case a transmitted venous contribution to the lymphatic pulsations. Concurrent pressures were taken from the internal jugular vein (J) and left common carotid artery (C). Lymph and venous pressure calibrations are in mm. saline and arterial pressures in mm. mercury.

A. Effect of ascending aortic occlusion (AA), showing elimination of the arterial component of the lymph pulsations. Vertical indicator dashes in lymph channel mark onset of left common carotid pulses.

B. Different animal showing disappearance of pulse and elevation of mean intralymphatic pressure upon occlusion of innominate vein (IV).

C. Immediately after record shown in B the thoracic duct was ligated at its entry into the left subclavian vein. Pulsations persisted, were not affected by ascending aortic occlusion (AA), but were abolished by innominate vein occlusion (IV).

contact with the recording system. Furthermore, it was demonstrated grossly by several techniques that the contents of lymphatic vessels pulsated with considerable sharpness and vigor. When a catheter was placed in the thoracic duct, the lymph could clearly be seen to pulsate as it rose in the tube; or if a small opening was made in the vessel wall, the escaping lymph was seen to issue in a pulsatile manner. Moreover, if either a small air bubble or a tiny amount of ink was injected into the duct, it slowly passed rostrally, moving about with each aortic pulsation, pausing transiently at the valves, and finally passing out of sight into the neck.

Recording from the cervical thoracic duct. Mean side pressures ranged from 25 to 100 mm. water, and mean end pressures were maintained at approximately 100 mm. water by intermittent lymph letting to relieve the obstruction. Intralymphatic pulsations, similar to those described above, were evident. The lymph pulsated either in synchrony with adjacent arteries (Fig. 2) or in

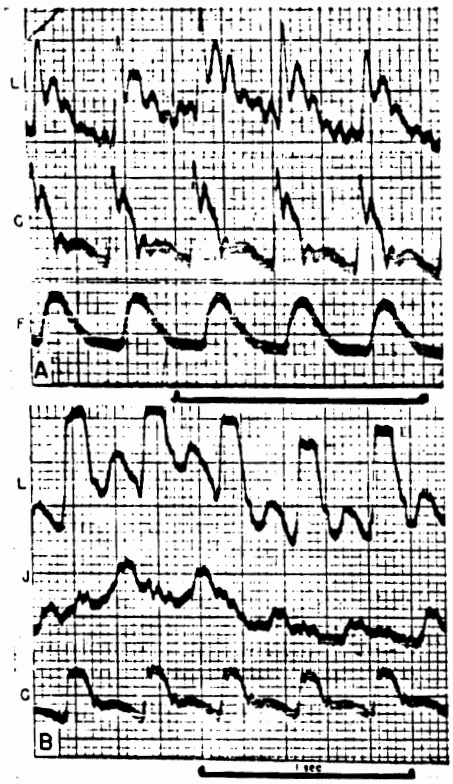

FIG. 4. Lymphatic pulsations in magnified detail showing time relationship to pulsations of adjacent vessels.

A. Side pressure recording from thoracic duct at the T-12 level. The record is an enlarged segment of that illustrated in Fig. 1A, and has the same legend.

B. Similar enlarged segment of record obtained from the cervical thoracic duct. The record is a magnified portion of that shown in Fig. 3A, and has the same legend.

phase with the pulsations of the neck veins (Fig. 3B, C). Often pressure changes were coordinated with both arterial and venous pulses, resulting in an irregular double lymphatic pulse for every cardiac cycle (Figs. 3A, 4B).

The transmitted arterial component of the lymphatic pressure waves was synchronous with the carotid (Figs. 2, 3A, 4B) or subclavian pulse. Accordingly, if the blood or pulse pressure in these vessels was altered, dramatic changes occurred in the lymphatic pulsations. When the third portion of the

aortic arch was occluded (Fig. 2A) carotid hypertension and widening of the arterial pulse pressure occurred immediately, accompanied by augmentation of the lymphatic pulsations. Conversely, if the arteries of the head and neck were serially blocked a decrement or a fall-out of lymph pulsations was demonstrable. Such an instance is shown (Fig. 2B) in which left subclavian occlusion caused a reduction in size of lymph pulsations, followed by virtual disappearance of pulsations when the brachiocephalic artery was occluded. Minute pulsations which remained could then be abolished by occlusion of the third portion of the aortic arch. More simply, occlusion of the ascending aorta also eliminated the transmitted arterial element of the lymphatic pulse, leaving, in those instances where it had been present before (Fig. 3A), a lymphatic pressure wave related only to venous pulsations.

Alterations of lymphatic pressure that occurred in phase with the jugular or subclavian venous pulsations could be recorded with either end or side pressure methods, suggesting at the outset that they were transmitted by contiguity rather than by retrograde flow from the subclavian vein into the thoracic duct. This component of the lymphatic pulse was not significantly affected by occlusion of the ascending aorta (Fig. 3C) but was eliminated by occlusion of the innominate vein (Fig. 3 B, C) or the superior vena cava. Ligation of the thoracic duct at its entry into the subclavian vein, which broke intraluminal contact between the two systems, failed to eliminate this type of lymphatic pulse (Fig. 3 B, C)—emphasizing that any venous component of the lymphatic pulses is chiefly activated by adjacent pulsating veins rather than by venous reflux into the duct.

DISCUSSION

With recording techniques and by direct observation, it is evident that movements from the great arterial trunks are imparted to the larger collecting lymphatics. These transmitted pressure changes apparently keep the lymph in continual pulsatile motion which, because of the high degree of valve competence (1), is of necessity oriented toward the venous drainage sites. In the neck, where the thoracic duct is in close relation to large pulsating veins, venous pulsations are similarly transmitted to the lymph, a process which is probably peculiar to a few anatomic areas such as the subclavian triangle and of limited functional significance.

The type of experimentation performed on the lower thoracic duct yielded valuable information on the fate of the transmitted intralymphatic pressure alterations. Occlusion of the aorta just proximal to the level of pressure measurement routinely obliterated the lymphatic pulses at distal levels, indicating that retrograde lymphatic conduction of these transmitted effects does not occur. Similarly, blockage of ascending lymph channels or elimination of

distal aortic pulsations did not appreciably alter the lymphatic pulsations, suggesting that rostral propagation of the lymph pressure changes is likewise for very short distances.

The information presented is directly relevant only to the effect of the great arteries on the larger lymphatic channels. The possible extension of such a concept to the distal vascular and lymphatic beds has been stressed in the skilful experimentations of Parsons and McMasters (2, 3), although at present one can only speculate upon the role of this mechanism in peripheral lymph clearance.

SUMMARY

A study was made in dogs of rhythmic intralymphatic pressure changes in the supradiaphragmatic and cervical thoracic duct. In both areas lymphatic pulses were found to be transmitted from contiguous large arteries, the lymphatic pulse pressure representing a significant fraction of the peak lymphatic pressure. In the neck venous pulsations were also shown to be transmitted to the lymph in a similar manner. These pulsations were shown to be propagated for only a few millimeters. By "tagging" the lymph it could be demonstrated that the pulsations were associated with its proximal flow. The possible functional significance of such a mechanism in general lymph clearance is reemphasized.

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