

The role of lymphatics in removing pleural liquid in discrete hydrothorax

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The role of lymphatics in removing pleural liquid in discrete hydrothorax. T. Nakamura, Y. Tanaka, T. Fukabori, Y. Iwasaki, M. Nakagawa, S. Kira.

ABSTRACT: The thoracic duct and right lymphatic duct of the dogs in the experimental group were ligated at the neck. Saline labelled with Evans blue was injected ($10 \text{ ml}\cdot\text{kg}^{-1}$) into the pleural cavity. The colloid osmotic pressure of the saline was adjusted to be exactly equal to that of normal pleural liquid. The change in the volume of the liquid and the decrease of the marker were studied for 3 h. The dynamics of the pleural liquid were investigated. In the control group, the volume of the pleural liquid decreased at the rate of $1.31 \text{ ml}\cdot\text{h}^{-1}\cdot\text{kg}^{-1}$ body weight and 87% of it left *via* the lymphatics. With the ligation of the bilateral lymphatic ducts, the lymphatic removal rate decreased to about one tenth. This result shows that the hydrothorax leaves the pleural cavity mainly *via* the lymphatics. *Eur Respir J.*, 1988, 1, 826-831.

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Pleural liquid is thought to be absorbed into the visceral capillaries [1]. However, we have suggested that it decreases mainly *via* the lymphatics [2, 3]. Some other recent studies have also shown the lymphatics to be an important factor affecting the dynamics of pleural liquid [4-7]. However, the pathophysiology of the dynamics of pleural liquid when increased in volume is largely unknown.

The purpose of this study is to confirm the role of the lymphatics in removing pleural liquid when its volume is increased *i.e.* with a discrete hydrothorax. The thoracic duct and right lymphatic duct of the dogs in the experimental group were ligated at the neck. Saline labelled with Evans blue (T-1824) was injected into the pleural cavity. The dynamics of the pleural liquid were then investigated.

Materials and methods

Experiments were performed with 14 dogs (weight range 7.5-12.0 kg; mean 10.3 kg) in the supine posture, anaesthetized with pentobarbital ($30 \text{ mg}\cdot\text{kg}^{-1}$ body weight) under controlled breathing ($10 \text{ ml}\cdot\text{kg}^{-1}$, 15-min^{-1} intermittent positive pressure ventilation).

In the control group (six dogs), a catheter was inserted into the left pleural cavity at the 6th intercostal space. The air which entered the pleural cavity during this manoeuvre was sucked out. Ten $\text{ml}\cdot\text{kg}^{-1}$ body weight of 0.9% saline containing $1 \text{ g}\cdot\text{dl}^{-1}$ of protein and Evans blue (T-1824) was injected *via* the catheter and observed for 3 h.

According to MISEROCCHI and AGOSTONI [8], the normal pleural liquid in dogs has a mean protein concentration of $1.06 \text{ g}\cdot\text{dl}^{-1}$. The protein concentration in the injected saline was adjusted to $1.0 \text{ g}\cdot\text{dl}^{-1}$ by adding a small amount of the dog's own plasma. We used Evans blue at a concentration of $2 \text{ mg}\cdot\text{dl}^{-1}$, which combines with the plasma protein in the saline. Three ml of pleural liquid were sampled at 30-min intervals and the concentration of Evans blue was measured by spectrophotometry with absorption at 620 nm [9].

In the group with lymphatic duct ligation (LDL) (six dogs), the thoracic duct and right lymphatic duct were ligated at the neck as described by LEEDS *et al.* [10]. A longitudinal incision was made on the right side of the neck through the pectoralis superficialis muscle. The junction of the right axillary vein with the external jugular vein was exposed. The right lymphatic duct emerges from the mediastinum and empties into the ampulla. The ampulla also receives the right cervical lymphatic duct and connects with the jugular-axillary vein angle. A ligature was passed around the ampulla and tied. Another ligature was passed around the right lymphatic duct, at the point where it empties into the ampulla and tied.

A longitudinal incision was then made on the left side of the neck. The thoracic duct was exposed by blunt dissection of the area lateral to the oesophagus in the lower neck. The thoracic duct was ligated at the jugular-axillary vein angle. Soon afterwards, $10 \text{ ml}\cdot\text{kg}^{-1}$ body weight of saline was injected as in the control group.

In all of the dogs, the chest was opened 3 h after the injection of saline. The tip of a vinyl tube (o.d 4 mm)

was placed in the posterior part of the costodiaphragmatic sinus and the residual liquid was aspirated into a syringe. The volume of the liquid collected was measured. The change in volume of the pleural liquid during the experiment was calculated with correction for the amount of pleural liquid withdrawn as samples.

Based on the rate of change in volume of pleural liquid and on the rate of change in the Evans blue concentration in the pleural liquid, the rate of lymphatic removal of the pleural liquid was calculated by the method of STEWART [11]. Under all conditions, the amount of protein was far greater than required for combination with all of the Evans blue [12]. Hence Evans blue was not absorbed into the pleural capillary circulation but could only be removed *via* the lymphatics. The amount of dye (Q) removed *via* the lymphatics was calculated as:

$$Q = V_0 C_0 - V_t C_t - S$$

where V_0 is the volume of the injected saline; V_t is the volume of the residual liquid in the pleural cavity at the end of the experiment; C_0 is the initial concentration of Evans blue in the pleural liquid; C_t is the concentration of Evans blue at the end of the experiment; and S is the amount of Evans blue withdrawn in samples.

The rate of lymphatic removal (L) is:

$$L = \frac{Q}{C_m} \times \frac{1}{t}$$

where C_m is the mean concentration of Evans blue in the pleural liquid; and t is the observation period in hours.

To confirm blockage of the lymphatic flow from the pleural cavity, four preliminary experiments were performed. In two dogs, 10 ml·kg⁻¹ body weight of homologous plasma which contained 50 mg of Evans blue was injected into the left pleural cavity after ligation of the right lymphatic duct and the thoracic duct. Small amounts of blood were sampled from the femoral artery at intervals for 4 h. The concentration of Evans blue in the serum was measured. Two other dogs were subjected to the same protocol but with intact lymphatics.

To investigate whether or not the surgery may have had an effect on the lymphatic flow, a sham operation was performed on two more dogs. The right lymphatic duct and thoracic duct were exposed at the neck but no ligations were made. The amount of the instillate was as in the control group.

Data were analysed using Student's unpaired t-test.

Results

Figure 1 shows the results of four preliminary experiments. In the dogs without any ligation of lymphatic ducts, the Evans blue concentration in the serum rose rapidly and increased to about 0.4 mg·dl⁻¹ 4 h after the injection (open circles). In the two experiments with ligation of the lymphatic ducts, the Evans blue concentration was almost zero in the serum even at 4 h after injection (closed circles). Thus ligation of the lymphatic

ducts had almost completely blocked lymphatic flow from the pleural cavity to the blood vessels.

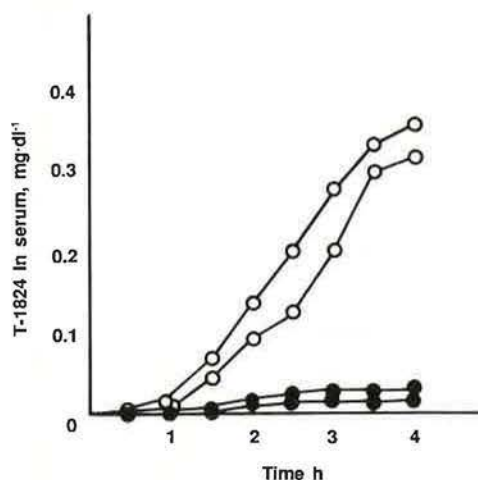


Fig. 1. - The concentration of Evans blue (T-1824) in the serum in four preliminary experiments, expressed as mg·dl⁻¹ and plotted vs time. Open circles refer to the dogs without ligation of lymphatic ducts, closed circles to the dogs with ligation of right lymphatic duct and thoracic duct.

The injected volume of saline and residual volume in each dog is shown in table 1.

Figure 2 shows the rate of decrease in volume of the pleural liquid (ΔV) in the two groups. In the control

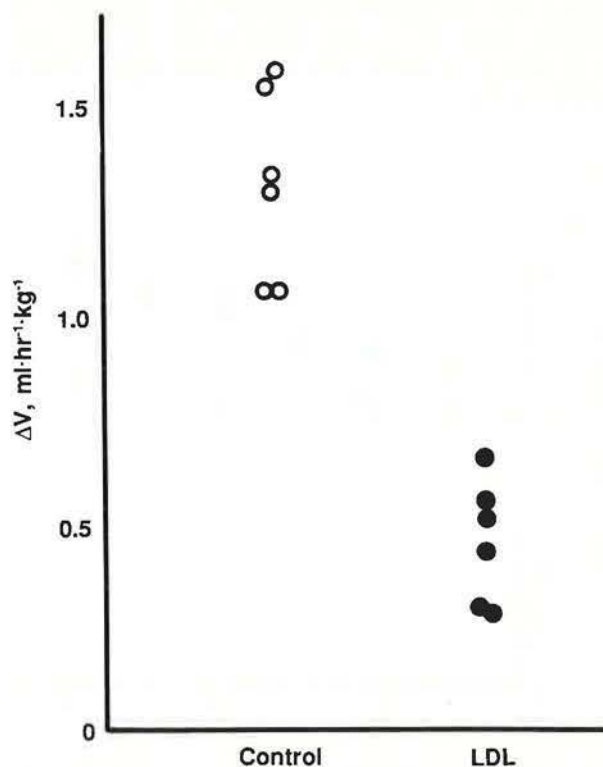


Fig. 2. - The rate of decrease in volume of the pleural liquid (ΔV) of the two groups. Open circles refer to the control group and closed circles to the group with ligation of the lymphatic ducts (LDL).

Table 1. - The change in pleural liquid volume in each dog

Control	BW	Volume of injected saline	Volume of residual liquid	Volume of sampled liquid	Decrease in volume of pleural liquid
	kg	ml	ml	ml	ml
1	8.5	85	27	18	40
2	9.0	90	37	18	35
3	10.5	105	54	18	33
4	11.0	110	48	18	44
5	12.0	120	64	18	38
6	10.0	100	35	18	47
LDL					
1	7.5	75	42	18	15
2	8.5	85	53	18	14
3	11.5	115	79	18	18
4	11.0	110	77	18	15
5	12.0	120	92	18	10
6	11.5	115	88	18	9

BW: body weight; LDL: group with ligation of lymphatic ducts

group the volume of the pleural liquid decreased at the rate of 1.31 ± 0.086 (SE) $\text{ml} \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$ body weight. In the LDL group, the rate of decrease fell significantly to 0.45 ± 0.057 $\text{ml} \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$ ($p < 0.001$).

Figure 3 shows the concentration of Evans blue in the pleural liquid over time. In the control group, the Evans blue concentration increased very gradually and reached about 107% of its initial value 3 h after injection. In the LDL group, it increased more rapidly to about 120% in 3 h.

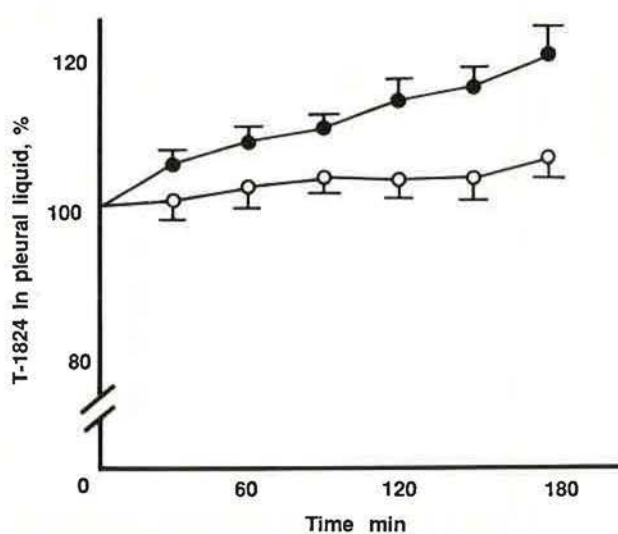


Fig. 3. - The concentration of Evans blue (T-1824) in the pleural liquid, expressed as percentage of initial value and plotted vs. time. Bars indicate mean \pm SEM, open circles refer to the control group and closed circles to the group with ligation of the lymphatic ducts.

Figure 4 shows the rate of lymphatic removal in both groups. In the control group 1.14 ± 0.12 $\text{ml} \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$ of the pleural liquid was removed *via* the lymphatics. In the LDL group, this rate fell significantly to 0.132 ± 0.059 $\text{ml} \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$ ($p < 0.001$).

The rates of removal of pleural liquid in the two dogs with the sham operation were 1.30 and 1.46 $\text{ml} \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$ and the lymphatic removal rates were 1.25 and 1.35 $\text{ml} \cdot \text{h}^{-1} \cdot \text{kg}^{-1}$. They were not significantly different from those of the control group.

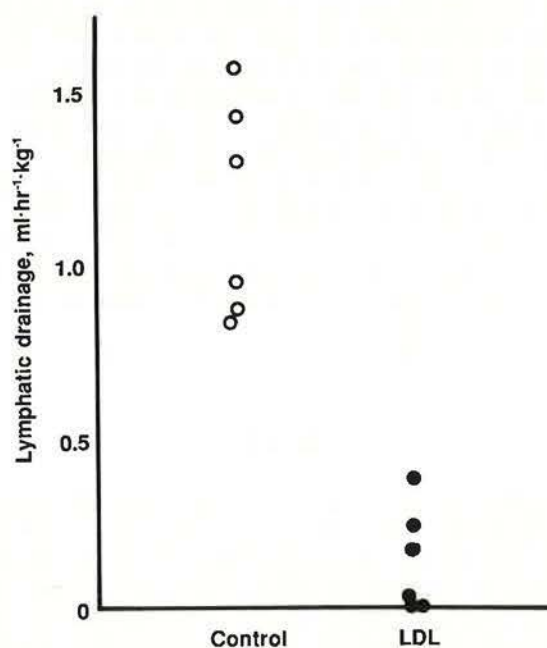


Fig. 4. - The rate of lymphatic drainage of the pleural liquid in the two groups. Symbols as in figure 2.

Discussion

COURTICE and SIMMONDS mentioned the significance of lymphatic drainage of saline injected into the pleural cavity. They confirmed that protein injected into the pleural cavity [13] or peritoneal cavity [14], was almost completely absorbed *via* the lymphatics. On the other hand, the route by which water leaves the pleural cavity has not been definitely determined. Since the visceral pleura is supplied mainly by the pulmonary circulation, the net pressure is absorptive at the visceral capillaries. Pleural liquid is thought to be absorbed into the visceral capillaries. The overall net pressure driving pleural liquid out of the cavity through the visceral pleura into the capillaries is about 4 cmH₂O in the normal supine dog [15]. According to KINASEWITZ and FISHMAN [16], the filtration coefficient of the visceral pleura of the dog is 1.1×10^{-6} ml·s⁻¹·cmH₂O⁻¹·cm⁻². MISEROCCHI and AGOSTONI [8] showed that the surface area of the visceral pleura on both sides in a dog of 10 kg is 979 cm². Based on these data, the rate of absorption of the pleural liquid into the visceral capillaries is about 1.6 ml·h⁻¹·kg⁻¹. This rate is considered to be balanced by the production rate at the parietal capillaries [17].

However, recent studies suggest that the lymphatics may play an important role in absorbing pleural liquid in extremely slight hydrothorax [4–7]. To elucidate to what extent the lymphatics can drain the pleural liquid when its volume is increased, we injected a large volume of saline into the pleural cavity in the present study. Four preliminary experiments showed that the Evans blue injected into the pleural cavity reached blood vessels *via* the lymphatics in unligated dogs. This result agrees with that of BURGEM and STEWART [18]. The two dogs with lymphatic duct ligation in preliminary experiments showed that the bilateral ligation of lymphatic ducts in the neck almost completely blocked this pathway.

In a previous paper [2], we suggested that pleural liquid leaves the pleural cavity mainly *via* the lymphatics despite the rapid exchange of pleural liquid with pleural capillary blood. Therefore, we predicted that ligation of the right lymphatic duct and thoracic duct would inhibit the removal of the injected liquid almost completely.

Out of keeping with our prediction, in the LDL group the rate of removal of the pleural liquid decreased to 34% of the control group rate. One would suspect that in the normal dog 34% of the pleural liquid was absorbed directly into the pleural capillary, and not *via* the lymphatics.

However, the calculated removal rate of the lymphatics shows that 87% of the total volume removed was absorbed *via* the lymphatics in the control group and the bilateral ligation of the lymphatic ducts decreased the lymphatic removal rate to about one tenth of that in control dogs. The calculation of the lymphatic flow by the method of STEWART is based on the assumption that the injected Evans blue leaves the pleural cavity solely *via* the lymphatics and in the concentration present in the pleural liquid [11, 18]. COURTICE and co-workers showed that Evans blue injected into the pleural cavity [13] or peritoneal cavity [14] of rabbits or

rats failed to appear in the bloodstream after the thoracic and right lymphatic ducts had been ligated or cannulated. The preliminary experiments in the present study also showed that almost all of the Evans blue was removed *via* the lymphatics.

Since particulate matter and large particles leave the pleural cavity *via* the lymphatics [19, 20], it is not considered possible that the pleural liquid is subjected to any ultrafiltration at the entrance of the lymphatic vessels. Some investigators have shown that the initial lymphatics concentrate the lymph and that their protein concentration is greater than that in the surrounding tissue fluid [21, 22]. However, CASLEY-SMITH [23] confirmed that the lymph is rediluted in collecting vessels and that the protein concentration becomes approximately equal to that in the tissue fluid; moreover Casley-Smith showed that there are open junctions at the entrance of the initial lymphatics.

These theoretical considerations seem to support the assumption upon which the calculation of the lymphatic flow is based. Therefore, in the control group, the volume of the injected saline seems to have decreased mainly *via* the lymphatics. The rate of decrease in volume of the pleural liquid in the control group was 1.31 ml·h⁻¹·kg⁻¹. However, this value is far in excess of the normal lung lymphatic flow rate from the pleural cavity. MISEROCCHI and co-workers [4] found that the injected saline or plasma was absorbed from the normal pleural cavity at the rate of 0.22 ml·h⁻¹·kg⁻¹. This experiment was performed with the injection of a small volume (2 ml) into a rabbit's pleural cavity. WIENER-KRONISH *et al.* [24] found that plasma instilled into a sheep's pleural cavity decreased at a rate of 0.22 ml·h⁻¹·kg⁻¹. MISEROCCHI and NEGRINI [7] showed that the liquid pressure of the pleural liquid is a factor affecting the rate of lymphatic drainage, *i.e.* the higher the liquid pressure the greater the rate of lymphatic drainage.

In this study, we injected a large volume of saline into the pleural cavity as in our previous study when the liquid pressure at the middle lung height (7.5 cm) was -7.3 cmH₂O [2]. When this value and the data of AGOSTONI and D'ANGELO [25], corrected for the size of the dogs, are taken into account, we could assume that the liquid pressure for the lowermost part of the pleural cavity is about zero in the present study by correcting the difference in the height from the hilum with a gradient of 1 cmH₂O·cm⁻¹. MISEROCCHI and NEGRINI [7] showed the lymphatic drainage to be 0.8–1.6 ml·h⁻¹·kg⁻¹ at this value of liquid pressure. This rate agrees with the present result.

We confirmed that the greater the injected volume the greater the rate of decrease in volume [2]. With a 10 ml·kg⁻¹ injection the volume decreased at the rate of 1.2–1.7 ml·h⁻¹·kg⁻¹. STEWART and BURGEM [26] showed that lymphatic flow from the pleural cavity of the normal dog was increased to about 2.0 ml·h⁻¹·kg⁻¹ by hyperventilation. In the present study 10 ml·kg⁻¹ of saline was injected during positive pressure ventilation, thus, any pumping action by respiratory movement could have been increased.

The difference between the rate of decrease in volume

of the pleural liquid (ΔV) and lymphatic drainage is the rate of the absorption of the pleural liquid into the pleural capillary [11]. In the LDL group this difference was greater than in the control group by $0.15 \text{ ml}\cdot\text{h}^{-1}\cdot\text{kg}^{-1}$, i.e. $0.32\text{--}0.17 \text{ ml}\cdot\text{h}^{-1}\cdot\text{kg}^{-1}$ ($p < 0.1$).

In considering the absorption rate of the pleural liquid into the pleural vessels, one has to analyse the hydrostatic and colloid osmotic pressures of the pleural liquid. According to the change in concentration of Evans blue in the pleural liquid shown in figure 3, the mean protein concentration at the half-time point is $1.04 \text{ g}\cdot\text{dl}^{-1}$ in the control group and $1.10 \text{ g}\cdot\text{dl}^{-1}$ in the LDL group. The colloid osmotic pressure of the pleural liquid estimated by the formula of LANDIS and PAPPENHEIMER [27] was $0.22 \text{ cmH}_2\text{O}$ greater in the LDL group than in the control group.

MISEROCCHI and AGOSTONI [8] showed that the surface area of the left visceral pleura in a dog of 10 kg is 430 cm^2 . Based on these data and the filtration coefficient of the visceral pleura of the dog mentioned above, to increase the absorption rate into the pleural vessels by $0.15 \text{ ml}\cdot\text{h}^{-1}\cdot\text{kg}^{-1}$ the liquid pressure of the pleural liquid must increase (decrease in negativity) by about $1.1 \text{ cmH}_2\text{O}$. If the volume of the pleural liquid decreased with time linearly as suggested by STEWART [11], the mean volume of the pleural liquid (at the half-time point) in the LDL group was greater by $1.3 \text{ ml}\cdot\text{kg}^{-1}$ than the control group due to the difference in ΔV between the two groups. When the data of AGOSTONI and D'ANGELO [25] are taken into consideration, the difference in the mean volume is sufficient to make the change of liquid pressure about $1.2 \text{ cmH}_2\text{O}$. Therefore, a higher liquid pressure in the LDL group might have increased the rate of absorption of the pleural liquid into the visceral capillaries despite the higher colloid osmotic pressure of the pleural liquid.

The results of the experiments on the two sham operated dogs show that the effect of surgery on the lymphatic flow can be excluded.

The present study shows that the main pathway of the drainage of injected saline is the lymphatics in the presence of a discrete hydrothorax

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RÉSUMÉ: L'on a ligaturé au niveau de cou le canal thoracique et la veine lymphatique droite de chiens. L'on a injecté dans la

cavité pleurale 10 ml·kg⁻¹ de solution saline isotonique marquée au bleu Evans. La pression osmotique colloïdale de la solution saline a été ajustée à une valeur égale à celle du liquide pleural normal. L'on a étudié pendant 3 h. les modifications du volume liquidien et la diminution des marqueurs. L'on a investigué la dynamique du liquide pleural. Dans le groupe contrôle, le volume du liquide pleural a diminué à raison de 1.31 ml·h⁻¹·kg⁻¹ de poids corporel et pour 87% par les voies lymphatiques. Après ligature des conduits lymphatiques des deux côtés, le taux de résorption lymphatique est réduit à environ 1/10. Ce résultat montre que l'hydrothorax quitte la cavité pleurale principalement par les lymphatiques.