Mechanics of the canine diaphragm in pleural effusion

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Submitted 6 April 2012; accepted in final form 5 July 2012

De Troyer A, Leduc D, Cappello M, Gevenois PA. Mechanics of the canine diaphragm in pleural effusion. J Appl Physiol 113: 785–790, 2012. First published July 12, 2012; doi:10.1152/japplphysiol.00446.2012.—Pleural effusion is a complicating feature of many diseases of the lung and pleura, and its effects on the mechanics of the diaphragm have not been assessed. In the present study, radiopaque markers were attached along muscle bundles in the midcostal region of the diaphragm in anesthetized dogs, and the three-dimensional location of the markers during relaxation before and after the stepwise introduction of liquid into the left or right pleural space and during phrenic nerve stimulation in the same conditions was determined using computed tomography. From these data, accurate measurements of diaphragm muscle length and displacement were obtained, and the changes in pleural and abdominal pressure were analyzed as functions of these parameters. The effect of liquid instillation on the axial position of rib 5 was also measured. The data showed that 1) liquid leaked through the dorsal mediastinal sheet behind the pericardium so that effusion was bilateral; 2) effusion caused a caudal displacement of the relaxed diaphragm; 3) this displacement was, compared with passive lung inflation, much larger than the cranial displacement of the ribs; and 4) the capacity of the diaphragm to generate pressure, in particular pleural pressure, decreased markedly as effusion increased, and this decrease was well explained by the decrease in active muscle length. It is concluded that pleural effusion has a major adverse effect on the pressure-generating capacity of the diaphragm and that this is the result of the action of hydrostatic forces on the muscle.

PLEURAL EFFUSION IS A COMMON complicating feature of many diseases of the lung and pleura, and its occurrence is frequently associated with dyspnea. In an attempt to identify the mechanism(s) that underlie this symptom, pulmonary function studies have been performed in patients before and shortly after the removal of pleural liquid, and these studies have shown that the increase in lung volume after thoracocentesis is substantially smaller than the volume of liquid withdrawn (3, 9, 15). Brown et al. (3), for example, reported in nine patients with large pleural effusions that the withdrawal of a mean liquid volume of 1.11 liters was associated with a mean increase in functional residual capacity (FRC) of only 0.32 liter. Similarly, in nine patients studied by Estenne et al. (9), FRC increased 0.46 liter following the removal of 1.82 liters of liquid. This difference in volume implies that pleural effusion causes not only a partial deflation of the lung but also an expansion of the chest wall.

The effects of pleural effusion on the volume and shape of the lung and chest wall were examined by Dechman et al. (8). With the use of computed tomography (CT), these investigators confirmed that lung volume decreased, and chest-wall volume increased after infusion of 60 ml/kg saline into the pleural space of supine dogs. In agreement with the observations in patients, the decrease in lung volume accounted for only one-third of the total volume of liquid infused. In addition, the lung-volume loss occurred mostly in the caudal region of the lungs, whereas the increase in chest-wall volume appeared to be uniform. However, Dechman et al. (8) noted that the estimates of the combined changes in lung- and chest-wall volume were smaller than the volume of liquid instilled, and they pointed out that the CT images in their study essentially covered the rib-cage compartment of the chest wall. They suspected, therefore, that effusion caused both an expansion of the rib cage and a caudal displacement of the diaphragm. The effect of effusion on the diaphragm, however, was not investigated.

The objective of the present study was to assess the impact of pleural effusion on the mechanics of the diaphragm. Thus progressively increasing pleural effusion was induced in anesthetized dogs, and the following hypotheses were tested: 1) effusion causes expansion of the chest wall with both an expansion of the rib cage and a descent of the diaphragm; 2) the increase in pleural pressure (Ppl) in this setting is likely nonuniform so that the displacements of the rib cage and diaphragm might be different from those anticipated on the basis of their relative compliances; and 3) because the passive diaphragm descends with effusion, its muscle fibers are shorter, and their capacity to develop pressure during contraction is impaired.

METHODS

The experiments were carried out on adult bred-for-research dogs (20–32 kg) anesthetized with pentobarbital sodium (initial dose, 30 mg/kg iv), as approved by the Animal Ethics and Welfare Committee of the Brussels School of Medicine. The animals were placed in the supine posture, intubated with a cuffed endotracheal tube, and connected to a mechanical ventilator (Harvard Apparatus, Holliston, MA). A venous cannula was inserted in the forelimb to give maintenance doses of anesthetic, after which, the C5 and C6 phrenic nerve roots were isolated bilaterally in the neck. Two experimental protocols were then followed.

Experiment 1. Five animals were studied first to assess the effect of pleural effusion on the rib cage and the pressure-generating ability of the diaphragm. In each animal, the rib cage and intercostal muscles were exposed on both sides of the chest from the first to seventh rib by reflection of the skin and superficial muscle layers, and a pleural cannula, 3 mm outer diameter (Lepine Society, Lyon, France), was introduced into the pleural space through the third right (three animals) or left (two animals) intercostal space near the costochondral junction. The cannula was held in place with a purse-string suture and connected to a catheter and a three-way stopcock so that a graded pleural effusion could be induced later. A hook was also screwed in the right fifth rib in the midaxillary line and connected to a linear displacement transducer (Lucas Schaevitz Engineering, Pennsauken, NJ) to measure the cranio-caudal (axial) displacement of the rib, as described previously (4). In each animal, the abdomen was then...
opened by a 4-cm-long midline incision cranial to the umbilicus, and a balloon-catheter system filled with 1.0 ml air was placed between the stomach and the liver to measure abdominal pressure (Pab). After the abdomen was closely sutured in two layers, the C5 and C6 phrenic nerve roots were laid over insulated stainless-steel stimulating electrodes, and a differential pressure transducer (Validyne Engineering, Northridge, CA) was connected to a side port of the endotracheal tube to measure airway-opening pressure (Pao).

The animal was allowed to recover for 20 min after instrumentation, after which, it was made apneic by mechanical hyperventilation. After it was disconnected from the ventilator, the respiratory system was passively inflated to a transrespiratory pressure of \(\sim 20\) cm H\(_2\)O, the endotracheal tube was occluded, and the system was allowed to deflate in small-volume steps to FRC. The endotracheal tube was occluded at each step so that the relationship between axial rib position and Pao during relaxation could be established. The maneuver was performed in duplicate, after which, the animal was reconnected to the ventilator. After the ventilator was stopped, the endotracheal tube was occluded at FRC, and the changes in Pao and Pab (\(\Delta\) Pao and \(\Delta\) Pab, respectively) were measured while square pulses of 0.1-ms duration and supramaximal voltage were applied at a frequency of 20 impulses/s to the left and right phrenic nerves. Two to three trials were performed. After the animal was reconnected to the ventilator, a volume of 10 ml isoosmotic liquid (Dianeal glucose 1.36%; Baxter, Deerfield, IL)/kg body wt was introduced into the pleural space. The axial position of the rib was monitored closely during the instillation, and after a 10- to 15-min recovery period, stimulation of the phrenic nerves was repeated. Liquid volume in the pleural space was subsequently increased by increments of 10 ml/kg body wt up to a total of 40 ml/kg, and data were recorded after each increment. As during control, all stimulations were performed while the animal was apneic, and the endotracheal tube was occluded at resting end-expiration.

At postmortem examination, pleural effusion in each animal was bilateral and appeared to be symmetrical, as reported previously (8). Experiment 2. Six animals were studied next to evaluate the effect of pleural effusion on the displacement and length of the diaphragm during relaxation and during phrenic nerve stimulation. In each animal, the abdomen was opened by a midline incision from the xiphisternum to the umbilicus, and rows of five polyethylene spheres were stitched superficially to a muscle bundle in the midcostal region of both the left and the right hemidiaphragm using the same method that has been described previously in detail (6, 13). A balloon-catheter system was also placed between the liver and the stomach to measure Pab, and a pleural cannula was introduced into the pleural space through the third intercostal space on the right (three animals) or the left (three animals) side of the chest, as described in experiment 1.

After the abdomen was closely sutured and the pleural cannula was firmly attached to the intercostal muscles, the animal was transferred to a V-shaped board and placed in a four-channel multidetector CT scanner (Somatom Volume Zoom 4; Siemens Healthcare, Forchheim, Germany), and the C5 and C6 phrenic nerve roots were laid over insulated stainless-steel stimulating electrodes. The animal was made apneic by mechanical hyperventilation, and a first helical data acquisition was obtained at that time, and the \(\Delta\) Pao and \(\Delta\) Pab were recorded. A gradually increasing pleural effusion was subsequently induced using four instillations of 10 ml isoosmotic liquid/kg body wt, and a new set of CT data acquisitions was obtained after each instillation, first during relaxation and then during phrenic nerve stimulation. As in experiment 1, all measurements were performed while the animal was apneic.

The animals in both experiments were maintained at a constant, rather deep level of anesthesia throughout the study with supplemental doses of pentobarbital sodium (2–3 mg·kg\(^{-1}\)·h\(^{-1}\)). Thus at no time in the experiment did they have a corneal reflex or movements of the fore- or hindlimbs. Rectal temperature was maintained constant between 36°C and 38°C with infrared lamps. At the conclusion of the experiment, the animal was given an overdose of anesthetic (30–40 mg/kg iv).

Data analysis. For each animal of experiment 1, the values of axial position of rib 5 at end-expiration in the presence of different volumes of pleural effusion were measured relative to the relaxed FRC position in the control condition. The values of axial position of the rib obtained for the different volume steps during passive deflation were also measured relative to the relaxed FRC position, and these values were plotted against the corresponding values of Pao. The relationship between axial rib position and Pao was then fitted by a quadratic regression equation, and the rib positions at fixed Pao values at 4 cm H\(_2\)O increments were determined from the equation by interpolation. The values of rib position during pleural effusion and during passive deflation were finally averaged across the animal group, and they are presented as means \(\pm\) SE.

For each volume of effusion in each animal of experiment 1, the \(\Delta\) Pao and \(\Delta\) Pab values recorded during phrenic nerve stimulation were averaged over the three trials. The \(\Delta\) Pao and \(\Delta\) Pab values recorded during stimulation in each animal of experiment 2 were also measured relative to the relaxed FRC position, and these values were averaged across the two animal groups, are also presented as means \(\pm\) SE.

Analysis of the CT data (experiment 2) was made as described previously (6, 13). For each lung volume during passive inflation and for each volume of pleural effusion during relaxation and during phrenic nerve stimulation in each animal, 1.25-mm-thick transverse CT sections were reconstructed at 1.0-mm intervals by using 360° linear interpolation and a standard algorithm (AB 40f; Siemens Healthcare). Sagittal and coronal images were also reconstructed, and these multiplanar reconstructions were used in a workstation (Leonardo; Siemens Healthcare) to define the three-dimensional coordinates of each diaphragm marker and to measure the length of each hemidiaphragm. Particular attention was given to the markers situated near the central tendon. By convention, the coordinates of the markers along the craniocaudal axis were expressed in millimeters relative to their relaxed FRC position in the control condition; a caudal displacement of the markers relative to that position was given a negative sign. The length of the diaphragm muscle fibers was also expressed in millimeters. To allow comparison among the different animals, however, muscle lengths during relaxation and during phrenic nerve stimulation in the different conditions were then expressed as percentages of muscle length during relaxation at FRC in the control condition (\(L_{\text{FRC}}\)).

One of the most conspicuous features of the CT images after the induction of pleural effusion was the presence of liquid in both the right and the left pleural space in five of six animals, in agreement with the observations of Dechman et al. (8) and the postmortem observations made in experiment 1. In addition, although the amount of liquid on the side of instillation was usually larger than that on the opposite side when effusion was 10 ml/kg, it appeared symmetrical on the two sides when the volume of effusion increased. Only one animal did not show any liquid on the opposite side until effusion was 30 ml/kg; the amount of liquid on this side also remained smaller than that on the side of instillation. As a result, for the animal group, there were no consistent differences in the axial displacement of the
markers near the central tendon or in muscle length between the hemidiaphragm on the side of instillation (ipsilateral) and the hemidiaphragm on the other side (controlateral). For each volume of effusion, therefore, the values for the two hemidiaphragms were averaged for each individual animal. These values were then averaged across the animal group, and they are also presented as means ± SE.

Statistical analysis. Statistical assessments of the effects of increasing pleural effusion on ∆Pao, ∆Pab, rib position, diaphragm marker position, and diaphragm muscle length were made by ANOVA with repeated measures, and multiple comparison testing of the mean values was performed, when appropriate, using Tukey honestly significant difference tests. The criterion for statistical significance was taken as P < 0.05.

Additional observation. Four animals were finally studied to identify the route by which the liquid introduced on one side of the chest moved to the pleural cavity on the other side. These animals had been previously involved in another protocol, and by the time of this study, they were in the supine posture, deeply anesthetized, and connected to a mechanical ventilator. A pleural cannula was inserted in the third left intercostal space, and the pleural cavity on the right side was widely exposed by resecting the bony ribs 4–8 and the intercostal soft tissues. The right lung was also carefully lifted upward toward the costal cartilages so as to have better exposure of the dorsal mediastinum. Two, 10-ml/kg instillations of liquid were then performed. The liquid was identical to that used in experiments 1 and 2 but was made more apparent by the addition of minute amounts of methylene blue.

RESULTS

Pressure. The values of ∆Pao and ∆Pab obtained during phrenic nerve stimulation in the presence of increasing pleural effusion are shown for the 11 animals in Fig. 1. With an effusion of 10 ml/kg, ∆Pao decreased (P < 0.001) to 85.5 ± 1.4% of the value measured in the control condition, and this trend continued as the volume of effusion increased. As a result, when effusion was set at 40 ml/kg, ∆Pao was only 40.8 ± 2.2% of the control value (P < 0.001). ∆Pab also decreased progressively and continuously (P < 0.001) with increasing effusion.

Position of the rib at end-expiration (experiment 1). The changes in axial position of rib 5 during increasing pleural effusion are shown for the five animals studied in Fig. 2A. As effusion increased, the rib at resting end-expiration moved gradually in the cranial direction (P < 0.001) relative to its control position. The rib also moved in the cranial direction during passive lung inflation in the control condition, as shown in Fig. 2B.

Position of the dome of the diaphragm at end-expiration (experiment 2). The changes in end-expiratory axial position of the diaphragm markers situated near the central tendon during increasing pleural effusion are shown for the six animals in Fig. 3A. As the volume of effusion increased, the central tendon moved progressively and continuously in the caudal direction (P < 0.001).

The displacement of these markers during passive inflation in the control condition was analyzed using the same method as that described for the position of the rib (see above). Thus in each animal, the values of axial position of the markers obtained at the different lung volumes were plotted against the corresponding values of Pao, the relationship between these values was fitted by a quadratic regression equation, and the marker positions at fixed Pao values at 4.0-cm H2O increments were determined from this equation by interpolation. The relationship obtained for the six animals is shown in Fig. 3B; in each animal in the control condition, the central tendon moved caudally as lung volume was increased passively above FRC.

Fig. 1. Effects of graded pleural effusion on the changes in airway opening pressure (ΔPao) and abdominal pressure (ΔPab) during bilateral stimulation of the C5 and C6 phrenic nerve roots. Mean ± SE values obtained from 11 animals. ΔPao and ΔPab decreased gradually and continuously as the volume of effusion increased.

Fig. 2. A: effect of graded pleural effusion on the axial position of rib 5 during relaxation. B: relationship between the axial position of the rib and transrespiratory pressure during passive inflation in the control condition. Mean ± SE values obtained from 5 animals. Positive values in both panels denote rib displacement in the cranial direction relative to the position at functional residual capacity (FRC) in the control condition.

J Appl Physiol • doi:10.1152/japplphysiol.00446.2012 • www.jappl.org
Combining the data shown in Fig. 2 and those shown in Fig. 3, one could plot the values of axial displacement of rib 5 obtained at end-expiration during increasing effusion against the values of axial displacement of the diaphragm markers near the central tendon, and the plot thus obtained could be compared with that during passive inflation. The two plots are shown in Fig. 4. Note that these plots are analogous to the conventional Konno-Mead diagram (11), in which the rib-cage displacements during breathing are displayed against the displacements of the ventral abdominal wall. The relationship during pleural effusion clearly lay to the right of that during passive inflation, so that for any given rib displacement, the diaphragm displacement was approximately twofold of that observed during passive inflation.

Diaphragm muscle length. The values of diaphragm muscle length during relaxation in the presence of increasing pleural effusion and during phrenic nerve stimulation in the same conditions are shown in Fig. 5. Muscle length during relaxation decreased progressively ($P < 0.001$) to $84.7 \pm 1.6\%$ of $L_{FRC}$ as the volume of effusion increased to 40 ml/kg. Muscle length during stimulation, although much shorter than during relaxation in all conditions, also decreased gradually ($P < 0.001$) as effusion increased. Therefore, whereas active muscle length in the control condition was $61.0 \pm 2.2\%$ of $L_{FRC}$, with 40 ml/kg effusion, it was only $54.0 \pm 1.8\%$ of $L_{FRC}$.

Passage of liquid through the mediastinum. When instillation in the left pleural space began, liquid immediately appeared in the dorsal aspect of the left pleural cavity behind the pericardium. As the instillation progressed to 10 ml/kg, the height of the pool of liquid in the left pleural cavity increased, and the mediastinal sheet behind the pericardium gradually bulged to the right, leading to liquid exsudation into the right pleural cavity. As instillation progressed further from 10 to 20 ml/kg, the passage of liquid through the dorsal-caudal mediastinal sheet became prominent, and liquid accumulated in the caudal portion of the right pleural cavity as well.

DISCUSSION

The effects of pleural effusion on the diaphragm have not been assessed, and the present study reports two important
observations. The first is that in supine dogs, the expansion of the relaxed chest wall produced by an acute pleural effusion involves a prominent descent and shortening of the diaphragm. The second observation is that effusion markedly impairs the capacity of the muscle to generate pressure, in particular, Ppl. In the following sections, we will first analyze the pattern of chest-wall expansion during graded effusion. We will then show that the decrease in the pressure-generating capacity of the diaphragm is well accounted for by the changes in active diaphragm length, and finally, we will discuss the limitations and pathophysiologic implications of the study.

Effects of pleural effusion on the relaxed chest wall. Agostoni and Mead (1), in an earlier theoretical analysis, considered that air introduced into the pleural space during pneumothorax is essentially a space-occupying lesion. They inferred, therefore, that pneumothorax should produce partial deflation of the lung and expansion of the chest wall in accordance with the static pressure-volume relationships of the structures. As a corollary, pneumothorax would displace the rib cage outward and the diaphragm caudally so that the chest wall would expand along its relaxation characteristics.

In agreement with this analysis, Dechman et al. (8) showed with the use of CT that the introduction of liquid into the pleural space in supine dogs leads to a rather uniform expansion of the rib cage. In addition, noting that the estimates of the combined changes in lung and rib-cage volume were smaller than the volume of liquid instilled, these investigators suspected that pleural effusion also caused a caudal displacement of the diaphragm. The present findings confirmed this suspicion, and indeed, as effusion progressed, our animals showed both a progressive cranial displacement of the ribs (Fig. 2A) and a progressive caudal displacement of the diaphragm (Fig. 3A). However, the causal displacement of the diaphragm after effusion was, relative to passive lung inflation, twice as large as the cranial rib displacement so that the chest wall departed to the right of its relaxation characteristics (Fig. 4). This result is fully consistent with our hypothesis that with a pleural effusion, unlike a pneumothorax, the increase in Ppl is not uniform. Apparently, in the dog, the pressure rise over the base of the lung (i.e., in the vicinity of the diaphragm) is larger than that over the apex of the lung.

The visual inspection of the pleural cavity during the liquid instillation further supports this idea. As would be expected in dogs in the supine posture, liquid accumulated first in the dorsal aspect of the ipsilateral pleural cavity. However, although the instillation was made in a rostral intercostal space, this accumulation took place in the caudal one-half of the cavity, which in supine dogs, is dependent relative to the cranial one-half. As a result, the mediastinal membrane behind the pericardium bulged toward the opposite side and eventually leaked so that liquid appeared in the contralateral pleural cavity. On the other hand, the membrane in the cranial one-half of the pleural cavity did not bulge, and no leakage was observed in this region as the volume of liquid was increased up to 20 ml/kg.

Effects of pleural effusion on the active diaphragm. As the muscle fibers of the diaphragm are activated and shorten, the dome descends, Ppl decreases, and Pab increases, and at equilibrium, the pressure generated by the diaphragm [transdiaphragmatic pressure (Pdi)] balances the load imposed on the muscle by Ppl and Pab (6, 7, 16). As discussed previously, the relaxed diaphragm was displaced caudally in the presence of effusion. If, during subsequent phrenic nerve stimulation, the muscle were to reach the same axial position as it does in the control condition, the volume swept by the muscle would therefore be smaller, and the changes in Ppl would be smaller as well. Consequently, the load imposed on the muscle would be smaller, and thus the muscle would continue to shorten until a new equilibrium position is reached (8, 16). In agreement with this analysis, diaphragm muscle length during phrenic nerve stimulation decreased progressively as effusion increased (Fig. 5); when effusion was set at 40 ml/kg, the diaphragm shortened to 54.0% of LFRM, whereas in the control condition, it only shortened to 61% of LFRM.

This difference in active muscle length is not large. However, it is well established that the relationship between the pressure developed by the diaphragm and muscle length is very steep when muscle length decreases to ~60% of LFRM and below (6, 7, 10); this phenomenon is likely the result of the decrease in the curvature of the muscle that occurs at such short muscle lengths (2, 7). In our previous study of the effect of lung inflation on diaphragm function in dogs, for example (6), we found that when the phrenic nerves were maximally stimulated at FRC in animals with the airway occluded, the diaphragm shortened to ~60% of LFRM, and ∆Pao (or ∆Ppl) was ~35 cm H2O. However, when the same stimulation was delivered at total lung capacity (TLC), muscle length was ~51% of LFRM, but ∆Pao was only ~3.0 cm H2O. Thus as lung volume increased from FRC to TLC, and active diaphragm length decreased from 60% to 51% of LFRM, the ∆Ppl generated by the muscle decreased by a factor of ~12. The current finding that in the presence of 40 ml/kg pleural effusion, the ∆Ppl generated by the diaphragm decreased 60% relative to the control value is therefore consistent with the measured decrease in active muscle length.

The role played by diaphragm muscle length in determining the pressure developed by the muscle in the presence of pleural effusion is more quantitatively examined in Fig. 6. To compute

![Fig. 6. Relationship between transdiaphragmatic pressure (Pdi) and diaphragm muscle length during phrenic nerve stimulation in the presence of gradually increasing pleural effusion (closed circles); the numbers next to the circles refer to the volume of effusion expressed as ml/kg body wt. Mean ± SE values obtained from 6 animals; SE values for Pdi are not shown for the sake of clarity. The open circles are the mean ± SE values previously obtained from 6 animals during phrenic nerve stimulation at 5 different lung volumes between FRC and total lung capacity (TLC) (6). A single relationship (dashed line) fits both the data for pleural effusion and those for passive lung inflation.](http://jap.physiology.org/doi/10.1152/japplphysiol.00446.2012)
the values of Pdi during pleural effusion, it was assumed that the relationship between Pdi and diaphragm length at relaxed end-expiration was the same as the passive Pdi-length relationship found in previous studies (6) and thus that Pdi at end-expiration progressively decreased from 7.0 cm H2O in the control condition to 2.5 cm H2O with an effusion of 40 ml/kg. These estimated values of passive Pdi were then added to the Δ Pao and Δ Pab measured during phrenic nerve stimulation, and the values thus obtained from the six animals of the study were used to plot the active Pdi-length relationship (Fig. 6). The results of this analysis clearly show that the relationship between Pdi and diaphragm length for increasing pleural effusion is the same as that during progressive lung inflation. In other words, the prominent adverse effect of effusion on the action of the diaphragm is well explained by the impact of effusion on muscle length.

Limitations and pathophysiologic implications. Liquid instillations in our animals were made in an attempt to simulate pleural effusion as observed in clinical practice. The volume of liquid was increased up to 40 ml/kg, which is equivalent to an effusion of 3.0 liters in humans with 75 kg body wt. Such an effusion is not uncommon in thoracic medicine. Also, because pleural effusion in humans with lung or pleural disease is usually unilateral, liquid instillations were made on one side of the sternum. Liquid, however, leaked through the dorsal-caudal mediastinal membrane so that effusion was bilateral and nearly symmetric. The present findings, therefore, cannot be extended to humans without caution.

Regardless of this limitation, the current findings have two important pathophysiologic implications. First, it is well established that passive lung inflation in the dog affects nearly equally the pressure-generating capacity of the diaphragm and that of the inspiratory intercostal muscles, in particular, the parasternal intercostals (14). By demonstrating that in pleural effusion, the descent of the diaphragm is proportionately greater than the cranial displacement of the upper ribs, the current findings therefore imply that the decrease in the pressure-generating capacity of the diaphragm in this setting is greater than that of the inspiratory intercostals in the rostral interspaces, i.e., in the intercostal muscle areas known to have the greatest inspiratory drive (5). If it is assumed that the distribution of neural drive between the diaphragm and the inspiratory intercostals remains unaltered, then it would be expected that the contribution of the inspiratory intercostals to the pressure (or volume) changes during breathing would gradually increase with increasing effusion and that the contribution of the diaphragm would progressively decrease.

The second implication is that the impact of effusion on the inspiratory muscle pump should be affected by body position. Because of the effect of gravity on the abdominal contents, a change from the supine to the upright posture causes an outward displacement of the ventral wall of the abdomen and with it, a caudal displacement and shortening of the diaphragm. If the effect of pleural effusion on diaphragm length in the upright posture were the same as in the supine posture, it would therefore be expected that in the presence of a given effusion, the pressure-generating capacity of the muscle in the upright posture would be smaller than in the supine posture. In addition, the pleural cavity, both in the dog and in humans, has a larger surface area along its dorsal wall than along its caudal wall. In pleural effusion, therefore, the height of the column of pleural liquid should increase with a change from the supine to the upright posture. As a result, the hydrostatic forces applied on the diaphragm should be greater in this posture, and the decrease in the pressure-generating capacity of the muscle should also be greater. On the other hand, hydrostatic forces on the upper ribs should be less in the upright posture, and indeed, Krell and Rodarte (12) did not detect any change in the orientation of the ribs during graded pleural effusion in head-up dogs. It would be expected, therefore, that in the presence of a given pleural effusion, the pressure-generating capacity of the inspiratory intercostals in the upright posture would be essentially preserved and thus that the relative pressure (volume) contribution of these muscles during breathing in this posture would be even greater than that in the supine posture.

ACKNOWLEDGMENTS

The authors are grateful to T. A. Wilson (University of Minnesota) for helpful discussions.

GRANTS

Financial support for this study was provided by the Brussels School of Medicine.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS


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