Impact of acute ascites on the action of the canine abdominal muscles

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Leduc D, De Troyer A. Impact of acute ascites on the action of the canine abdominal muscles. J Appl Physiol 104: 1568–1573, 2008.—Although ascites causes abdominal expansion, its effects on abdominal muscle function are uncertain. In the present study, progressively increasing ascites was induced in supine anesthetized dogs, and the changes in abdominal (ΔPab) and airway opening (ΔPao) pressure obtained during stimulation of the internal oblique and transversus abdominis muscles were measured; the changes in internal oblique muscle length were also measured. As ascites increased from 0 to 100 ml/kg body wt, Pab and muscle length during relaxation increased. ΔPab also showed a threefold increase (P < 0.001). However, ΔPao decreased (P < 0.001). When ascites further increased to 200 ml/kg, resting muscle length continued to increase and muscle shortening during stimulation became very small so that active muscle length was 155% of the resting muscle length in the control condition. Concomitantly, ΔPab returned to the control value, and ΔPao continued to decrease. Similar results were obtained with the animals in the head-up posture, although the decrease in ΔPao appeared only when ascites was greater than 125 ml/kg. It is concluded that 1) ascites adversely affects the inspiratory action of the abdominal muscles on the lung; 2) this effect results primarily from the increase in diaphragm elastance; and 3) when ascites is severe, the abdomen cross-sectional area is also increased and the abdominal muscles are excessively lengthened so that their active pressure-generating ability itself is reduced.

Methods

The experiments were carried out on 14 adult cross-breed dogs (22–32 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 supine on a tilt table and intubated with auffed endotracheal tube, and a venous cannula was inserted in the forelimb to give maintenance doses of anesthetic. The abdomen was then opened by a midline incision from the xiphisternum to the umbilicus, and a balloon-catheter system filled with 1.0 ml of air was placed between the liver and the stomach to measure abdominal pressure (Pab) in the vicinity of the diaphragm. A catheter was also inserted through the rectus abdominis, 2–3 cm cranial to the umbilicus, such that liquid could easily be introduced into the abdominal cavity later. After the abdomen was sutured in two layers, the skin of the abdominal wall was incised on the right and the left anterior axillary line from the rib cage margin to the iliac crest. The external oblique to the inspiratory muscles, in particular the parasternal intercostals (13), the impact of ascites on the other respiratory muscles has not been studied. One of the most prominent features of this condition, however, is a distension of the ventrolateral wall of the abdomen. At end expiration, therefore, the muscles in the wall should be lengthened. In addition, because ascites lengthens the diaphragm, it also induces passive diaphragmatic tension (11–13). Consequently, it would be expected that the load imposed on the abdominal muscles during contraction would also increase and, hence, that the amount of muscle shortening would decrease. On this basis, one would therefore predict that in the presence of ascites, the contracting abdominal muscles would be longer and that their capacity to raise abdominal pressure would be greater. At the same time, however, the greater passive tension in the diaphragm should impede the pressure transmission from the abdominal to the intrathoracic cavity, and this might compensate, at least in part, for the greater force-generating ability of the abdominal muscles.

The objective of the present study was specifically to assess the effect of ascites on abdominal muscle function. Thus progressively increasing ascites was induced in dogs, and the changes in abdominal and airway opening pressure were examined during isolated stimulation of the internal oblique and transversus abdominis muscles, the predominant respiratory muscles of the abdominal wall in quadrupeds (7, 10, 14) and in humans (1, 6). The results indicated that moderate ascites improves the pressure-generating ability of the muscles but reduces their lung-deflating action, in particular in the supine posture. With severe ascites, however, the pressure-generating ability of the muscles was also impaired.
muscle on either side was reflected in part to expose the internal oblique, and a pair of stimulating electrodes was inserted 3–4 cm apart in parallel fibers through both the internal oblique and the underlying transversus abdominis. These electrodes were silver hooks insulated with polyethylene tubing except for the last 0.8 cm; after they were inserted, therefore, their tip was brought back to the surface and anchored. In addition, in each animal, two 3-0 black suture threads were stitched 20–30 mm apart to the superficial muscle fibers of the right internal oblique, 3–4 cm lateral to the umbilicus; these threads were oriented perpendicular to the long axis of the muscle fibers and were used to assess the changes in muscle length (see below). The lower limbs were finally tethered to avoid any subsequent motion of the pubis and iliac crests, and a differential pressure transducer (Validyne, Northridge, CA) was connected to a sideport of the endotracheal tube to measure the changes in airway opening pressure (ΔPao).

Protocol. The animals were allowed to recover for 15 min after instrumentation, after which they were connected to a mechanical ventilator (Harvard pump, Chicago, IL) and made apneic by hyperventilation. The ventilator was stopped, so that the respiratory system was allowed to relax to equilibrium, and the linear distance between the two suture threads inserted in the internal oblique [i.e., the length of the muscle bundle at functional residual capacity (L_FRC)] was measured with calipers in duplicate. The animals were then reconnected to the ventilator and made apneic, the endotracheal tube was occluded at resting end expiration, and square pulses of 0.2-ms duration and 20-Hz frequency were applied to the left and right abdominal muscles. The stimulus intensity was initially set at 20 V, and it was subsequently increased by increasing the voltage by 10-V increments to achieve a strong, symmetric muscle contraction associated with a significant rise in Pab without any visible motion of the pelvis; the intensity that was eventually selected for the study corresponded to a voltage of 60–80 V. Two trials of stimulation were then obtained, after which seven animals were tilted to the 45° head-up posture; in these animals, measurements of muscle length during relaxation and of the pressure changes during stimulation were repeated after each infusion. Muscle length was also measured during the stimulations in the presence of 175 and 200 ml/kg.

The animals were maintained at a constant, rather deep level of anesthesia throughout the surgery and the measurements. They had no corneal reflex and no movement of the fore- or hindlimbs. Rectal temperature was maintained constant between 36 and 38°C with infrared lamps. At the end of the experiment, the animal was given an overdose of anesthetic (30–40 mg/kg iv).

Data analysis. For each volume of ascites in each animal, the changes in Pab and Pao recorded during abdominal contraction were averaged over the two trials. The values of Pab during relaxation were also measured, and they were expressed relative to the value before ascites (control) in either the supine (7 animals) or the head-up (7 animals) posture.

For each volume of ascites, internal oblique muscle length during relaxation was also averaged over the two measurements and expressed in millimeters. To allow comparison between the different animals, however, muscle lengths were then expressed as percentages of muscle length during control in the supine posture (L_FRC). Muscle length during contraction at 175 and 200 ml/kg ascites was analyzed similarly.

Data were finally averaged across the animal group, and they are presented as means ± SE. Statistical assessments of the effects of increasing ascites on pressure and muscle length were made by ANOVA with repeated measures, and multiple comparison testing of the mean values was performed, when appropriate, using Student-Newman-Keuls tests. The criterion for statistical significance was taken as P < 0.05.

RESULTS

Relaxation in the supine posture. The effects of increasing ascites on Pab and the internal oblique muscle length during relaxation in the supine posture are shown for the seven animals in Fig. 1. With increasing ascites, the resting Pab increased gradually and the muscle lengthened progressively (P < 0.001 for both). As shown in Fig. 2, the relationship between Pab and muscle length was curvilinear, such that for
a given increase in pressure, the increase in muscle length was smaller as Pab was greater. When ascites amounted to 200 ml/kg, therefore, resting Pab was 38.4/110.0 cmH2O and muscle length was 160.8/16.2% Lfrc.

Pressure-generating ability in the supine posture. The values of Pab and Pao obtained during abdominal muscle contraction in the supine posture are shown in Fig. 3. Pab first increased progressively and markedly with increasing ascites. Thus, whereas ΔPab in the control condition was 12.2 ± 0.6 cmH2O, with 100 ml/kg ascites it was 41.8 ± 4.4 cmH2O (P < 0.001). As ascites increased further, however, ΔPab decreased rapidly, such that at 200 ml/kg, it was returned to the control value. In contrast, ΔPao decreased gradually and continuously in every animal (P < 0.001), even in the presence of small volumes of ascites.

Stimulation in the control condition and in the presence of moderate volumes of ascites induced substantial muscle shortening, although these length changes could not be quantified with precision. However, when stimulation was performed in the presence of large volumes of ascites, it hardly caused any change in muscle length. Specifically, with an ascites of 175 ml/kg, muscle shortening was only 6.6 ± 4.5% Lfrc. Active muscle length in this condition, therefore, was still 147.8 ± 5.6% Lfrc. Similarly, during stimulation at 200 ml/kg, the muscle shortened by 6.1 ± 4.9% Lfrc, so that its active length was 154.7 ± 6.4%.

Head-up posture. The effects of ascites in the 45° head-up posture were, by and large, similar to those observed in the supine posture. Thus resting Pab and abdominal muscle length during relaxation increased progressively as the volume of ascites increased (Fig. 1), and the relationship between the two variables was also curvilinear (Fig. 2). The Pab-length relaxation curve for the head-up posture was, in fact, similar in shape to the curve for the supine posture. Also, as shown in Fig. 4, ΔPab during stimulation in the head-up posture increased progressively as ascites increased to 100 ml/kg and then decreased rapidly as ascites increased further. Muscle length during stimulation at 200 ml/kg also remained virtually unchanged relative to its value during relaxation (151.5 ± 3.6% vs. 156.8 ± 5.1% Lfrc).

However, whereas ΔPao in the supine posture decreased continuously with increasing ascites, in the head-up posture,
\( \Delta P_{\text{ao}} \) remained similar to the control value until ascites was 125 ml/kg (Fig. 4). \( \Delta P_{\text{ao}} \) in this posture decreased only when ascites increased further from 125 to 200 ml/kg.

**DISCUSSION**

The present observations confirmed that increasing ascites induces, through the increase in Pab, a progressive lengthening of the relaxed abdominal muscles (Fig. 1). The curvilinear shape of the Pab-length relaxation curve, combined with the fact that the shape of the curves for the supine and the head-up posture was identical (Fig. 2), is evidence that this change in muscle length is exclusively determined by the muscle passive length-tension characteristics. In addition, the present observations also showed that ascites causes a reduction in the amount of abdominal muscle shortening during contraction; when ascites was set at 200 ml/kg, muscle contraction was, in fact, nearly isometric. This reduction in muscle shortening added to the increase in resting muscle length such that the muscles during contraction were longer, and indeed, in agreement with our hypothesis, our animals when supine had a threefold increase in \( \text{Pab} \) as ascites increased to 100 ml/kg (Fig. 3).

This increase in the active pressure-generating capacity of the abdominal muscles, however, did not translate into a greater lung-deflating action. With the animals in the supine posture, \( \Delta P_{\text{ao}} \) in fact continuously decreased as ascites increased, thus indicating that the increase in active pressure-generating capacity of the muscles was more than offset by the increase in passive diaphragmatic tension. Similarly, when the animals were in the head-up posture, \( \Delta P_{\text{ab}} \) increased markedly as ascites increased to 100 ml/kg, but \( \Delta P_{\text{ao}} \) remained unchanged (Fig. 4). In this posture, however, the diaphragm at resting end expiration is shorter and develops lower passive tension than in the supine posture (15). Apparently, therefore, diaphragm elastance in the head-up posture was sufficiently increased with moderate ascites so as to prevent the larger \( \Delta P_{\text{ab}} \) from being transmitted to the pleural cavity.

A more quantitative evaluation of the alteration in diaphragm elastance in the presence of ascites and of the role of this alteration in determining the change in the coupling between the abdominal muscles and the lung can be provided by using the two-compartment model of the chest wall recently developed by Cappello and De Troyer (5). According to this model, \( \Delta P_{\text{ao}} \) during isolated contraction of the abdominal muscles would be related to \( \Delta P_{\text{ab}} \) and to the elastance of the rib cage (\( K_R \)) and the diaphragm (\( K_{\text{di}} \)), such that

\[
\Delta P_{\text{ao}} = [K_R(\Delta P_{\text{ab}} + K_{\text{di}})] \cdot \Delta P_{\text{ab}}
\]

In other words, the ratio between \( \Delta P_{\text{ao}} \) and \( \Delta P_{\text{ab}} \) would exclusively depend on the relative values of \( K_R \) and \( K_{\text{di}} \).

The relationship thus computed between \( \Delta P_{\text{ao}}/\Delta P_{\text{ab}} \) and \( K_{\text{di}}/K_R \) for the different volumes of ascites up to 100 ml/kg is shown in Fig. 5. When our animals were in the supine posture, the ratio \( \Delta P_{\text{ao}}/\Delta P_{\text{ab}} \) during abdominal muscle stimulation in the control condition was 0.34, which corresponds to a \( K_{\text{di}}/K_R \) of 1.94; these values are very close to those recorded in our previous study (5). On the other hand, as ascites increased to 100 ml/kg, \( \Delta P_{\text{ao}}/\Delta P_{\text{ab}} \) was gradually reduced to 0.05, thus suggesting that \( K_{\text{di}}/K_R \) was increased to 20.0. Similarly, when the animals were in the head-up posture, \( \Delta P_{\text{ao}}/\Delta P_{\text{ab}} \) decreased from 0.82 during control to 0.41 with 100 ml/kg ascites, and the corresponding values for \( K_{\text{di}}/K_R \) would be 0.22 and 1.49, respectively. To the extent that moderate ascites does not affect rib cage elastance (in another study, we measured in 4 animals the relationship between the axial motion of the ribs and transthoracic pressure during passive inflation in the presence of increasing amounts of ascites, and we found that the slope of the relationship remained unaltered), one may therefore draw the conclusion that both in the supine and the head-up posture, the procedure increases diaphragm elastance by a factor of 7 to 10.

Whereas \( \Delta P_{\text{ab}} \) increased in the presence of moderate volumes of ascites, with larger volumes, it did rapidly and markedly decrease. As a result, with the animals in the supine posture, \( \Delta P_{\text{ab}} \) during stimulation with 200 ml/kg ascites was similar to the value in the control condition (Fig. 3). A similar decrease in \( \Delta P_{\text{ab}} \) was also found when the animals were head-up (Fig. 4), and this decrease added to the increase in diaphragm elastance to accentuate the detrimental effect of ascites on the lung-deflating action of the abdominal muscles.

This prominent decrease in \( \Delta P_{\text{ab}} \) in the presence of severe ascites was not the result of a deterioration of the muscles in the course of the experiment. Indeed, after the standard protocol was completed, in three supine animals, liquid was removed from the abdominal cavity, such that the total volume was brought back first to 150 ml/kg, then to 100 ml/kg, and abdominal muscle stimulation was repeated at each volume. The values obtained for \( \Delta P_{\text{ab}} \) at both volumes were much greater than those obtained at 200 ml/kg and close to the values obtained during the initial stimulations, thus indicating that the preparation was stable throughout. On the other hand, severe ascites led to a considerable increase in length of the passive internal oblique muscle. Specifically, with the animals in both the supine and the head-up posture, muscle length in the presence of 200 ml/kg ascites was \( \sim 160\% L_{dRC} \) (Fig. 1B). Also, when the muscles were stimulated in this condition, diaphragm elastance...
was so high that the muscles shortened only a little and remained at \(-155\% \text{L}_{FRC}\). If, as is the case for the diaphragm (8, 16), the relaxed internal oblique and transversus abdominis muscles in supine dogs were at the in vitro optimum force-producing length (\(L_o\)), such results would therefore imply that severe ascites causes these muscles to operate well beyond \(L_o\), i.e., at lengths where their active force-generating capacity is dramatically reduced (3, 9).

Although the operating length of the canine internal oblique and transversus abdominis has not been assessed, two lines of evidence support the idea that with severe ascites, these muscles do indeed operate on the descending limb, rather than the ascending limb, of their active length-tension curve. First, Farkas and Rochester (9) have previously shown that in supine dogs, the resting FRC length of the external oblique muscle of the abdomen corresponds to 0.83 \(L_o\). If it is assumed that the \(L_{FRC}\) of the internal oblique and transversus abdominis in our animals was also at 0.83 \(L_o\), then the length changes observed during ascites would indicate that at 200 ml/kg, muscle length during relaxation was (0.83 \times 1.60) or 1.33 \(L_o\), and that muscle length during stimulation was (0.83 \times 1.55) or 1.29 \(L_o\). The second evidence is provided by the finding that the largest value for \(\Delta P_{ab}\) in our supine or head-up animals was obtained when ascites was 100 ml/kg (Figs. 3 and 4). This finding suggests that \(L_o\), for the internal oblique and transversus abdominis taken together corresponds to the active muscle length at this volume of ascites. Also, as is shown in Fig. 1, muscle length during relaxation in this condition was 136\% \(L_{FRC}\), and in view of the amount of muscle shortening measured during stimulation at 200 ml/kg, a reasonable estimate for muscle shortening at 100 ml/kg would be 15–20\% \(L_{FRC}\). Consequently, in agreement with the observation by Farkas and Rochester (9), \(L_o\) for the muscles would be 116–120\% \(L_{FRC}\). To the extent that with 200 ml/kg ascites, active muscle length was increased to 156\% \(L_{FRC}\), this length would correspond to at least (156/120) or 1.30 \(L_o\).

Sharp et al. (17) postulated in earlier studies that the decrease in the pressure-generating ability of the diaphragm observed in morbidly obese subjects assuming the supine posture was the result of the excessive lengthening of the muscle. This mechanism, however, was not readily demonstrated. The present study would provide the first direct evidence that overstretching may actually alter respiratory muscle function in vivo.

We do not mean to imply, however, that overstretching entirely accounts for the decrease in \(\Delta P_{ab}\) in severe ascites. In fact, increasing ascites also causes a progressive increase in the cross-sectional area of the abdomen. Since the two muscles investigated in this study, in particular the transversus abdominis, run primarily circumferentially around the abdominal cavity, such an increase implies that the muscles undergo not only an increase in length but also an increase in the radius of curvature. Consequently, the \(\Delta P_{ab}\) associated with a given muscle tension should decrease (Laplace’s law). Therefore, it would be reasonable to speculate that the increase in abdomen cross-sectional area in our animals both reduced the increase in \(\Delta P_{ab}\) observed in the presence of moderate ascites and enhanced the decrease in \(\Delta P_{ab}\) in severe ascites. It is worth pointing out, however, that the concomitant decrease in the radius of abdominal curvature in the sagittal plane might compensate, in part, for the increase in radius of curvature in the transverse plane.

As with all animal models of disease, there is a limitation with the present study in that ascites was produced over a few hours. In contrast, in patients with liver or peritoneal diseases, ascites develops slowly over several weeks or months, and studies on limb muscles in mice and cats have shown that chronic muscle lengthening elicits an addition of sarcomeres in series along the muscle fibers (19, 20). The result of this remodeling is that the length of individual sarcomeres is virtually restored to its initial value. Therefore, the possibility exists that, in patients with severe ascites, the increase in passive diaphragmatic tension and, with it, the increase in diaphragm elastance is partly offset by an addition of sarcomeres. Addition of sarcomeres might also occur in the abdominal muscles, such that the force-generating capacity of the muscles would be better maintained. Nonetheless, by demonstrating that in the dog, severe ascites markedly reduces the capacity of the abdominal muscles to raise intrathoracic pressure, the present study implies that cough may be less effective in patients with ascites than in healthy individuals and may, therefore, lead to retention of airway secretions. This mechanism might account, at least in part, for the frequent occurrence of lung atelectasis and bronchopneumonia in such patients (21).

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