Role of rib cage elastance in the coupling between the abdominal muscles and the lung

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Submitted 12 January 2004; accepted in final form 12 March 2004

Cappello, Matteo, and André De Troyer. Role of rib cage elastance in the coupling between the abdominal muscles and the lung. J Appl Physiol 97: 85–90, 2004. First published March 19, 2004; 10.1152/japplphysiol.00032.2004.—The abdominal muscles expand the rib cage when they contract alone. This expansion opposes the deflation of the lung and may be viewed as pressure dissipation. The hypothesis was raised, therefore, that alterations in rib cage elastance should affect the lung deflating action of these muscles. To test this hypothesis and evaluate the quantitative importance of this effect, we measured the changes in airway opening pressure (Pao), abdominal pressure (Pab), and rib cage transverse diameter during isolated stimulation of the transversus abdominis muscle in anesthetized dogs, first with the rib cage intact and then after rib cage elastance was increased by 77% (P < 0.001). The observed increase in Pao was still unchanged, the change in Pao was increased by 77% (P < 0.001). In a second experiment, the transversus abdominis was stimulated before and after rib cage elastance was reduced by removing the bony ribs 3–8. Although the change in Pab after removal of the ribs was unchanged, the change in Pao was increased by 62% (P < 0.001). These observations, supported by a model analysis, indicate that rib cage elastance is a major determinant of the mechanical coupling between the abdominal muscles and the lung. In fact, in the dog, the effects of rib cage elastance and Pab on the lung-deflating action of the abdominal muscles are of the same order of magnitude.

response to a given abdominal muscle stimulation is further enhanced.

However, even though the rectus abdominis in the dog pulls the sternum and the ribs in the caudal direction when it contracts alone (11), the abdominal muscles expand the rib cage through the rise in pleural pressure and Pab (6). Such an expansion opposes the deflation of the lung and, hence, may be viewed as a dissipation of pressure. As a corollary, one would expect that, for a given rise in Pab and a given diaphragmatic elastance, an increase in rib cage elastance leading to a reduction in rib cage expansion would induce a larger deflation of the lung. Conversely, with a decrease in rib cage elastance, it would be expected that a given abdominal muscle contraction would result in a smaller deflation of the lung.

The present studies were thus designed to test the hypothesis that the magnitude of the lung deflating action of the abdominal muscles is also determined by rib cage elastance. In a first series of experiments, the pressure-generating ability of the transversus abdominis, the predominant respiratory muscle of the abdominal wall both in quadrupeds (9, 12, 15, 16) and in humans (1, 8), was measured in a group of anesthetized dogs, first before and then after rib cage elastance was increased by 77% (P < 0.001). The observed increase in Pao was still unchanged, the change in Pao was increased by 62% (P < 0.001). These observations, supported by a model analysis, indicate that rib cage elastance is a major determinant of the mechanical coupling between the abdominal muscles and the lung. In a second series of experiments, therefore, the pressure-generating ability of the transversus abdominis was also evaluated before and after rib cage elastance was reduced by the removal of several bony ribs.

METHODS

The experiments were carried out on 14 adult mongrel dogs (13–25 kg body weight) 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. Animals were placed in the supine posture, intubated with a cuffed endotracheal tube, and connected to a mechanical ventilator (Harvard pump, Chicago, IL). A venous cannula was inserted in the forelimb to give maintenance doses of anesthetic (3–5 mg·kg⁻¹·h⁻¹), and a catheter was inserted into the left femoral artery to monitor blood pressure and heart rate. In each animal, the level of anesthesia was regulated to abolish the corneal reflex throughout the surgery and the measurements. The rib cage and intercostal muscles were then exposed bilaterally from the 1st to the 10th rib by deflection of the skin and underlying muscle.

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layers, and a pair of linearized magnetometers (Norman H. Peterson, Boston, MA) was attached to the external intercostal muscles in the 4th and 5th interspaces on the right and left sides to measure the changes in rib cage transverse diameter (17). A differential pressure transducer was also connected to a side port of the endotracheal tube to measure Pao, and a balloon-catheter system filled with 1.0 ml of air was positioned in the stomach to measure Pab.

The skin of the abdominal wall was subsequently incised on the right and left anterior axillary line from the rib cage margin to the iliac crest. The obliquus externus and obliquus internus muscles were severed to expose the transversus abdominis, and a pair of stimulating electrodes was inserted 3–4 cm apart in parallel fibers on either side of the abdomen; these electrodes were silver hooks insulated with polyethylene tubing except for the last 0.8 cm. The lower limbs were then firmly tethered to avoid any subsequent motion of the pubis and iliac crests, and after a recovery period of 15 min, the animal was made apneic by mechanical hyperventilation. Measurements of Pao and rib cage transverse diameter were first obtained during passive inflation of the respiratory system. In each animal, five levels of inflation (100, 200, 300, 400, and 500 ml) were applied in triplicate, after which square pulses of 0.2-ms duration and 50-Hz frequency were delivered at intervals to the right and left transversus abdominis while the endotracheal tube was occluded at resting end expiration. The stimulus intensity was set by increasing the voltage from 5 to 30 V by 5-V increments. At least three trials were obtained at each voltage in every animal; after completion of the measurements, two experimental protocols were followed.

Protocol 1. In seven animals, the rib cage was stiffened by using a procedure that was recently described (7). Thus multiple clamps were attached to adjacent bony ribs on both sides of the chest, as shown in Fig. 1A. Also, two inverted V-shaped metallic bars were secured to the sternum and two pairs of ribs in the midaxillary line to prevent the sternum from moving relative to the ribs; one bar was positioned at the level of the second or third rib pair, and the other was positioned at the level of the sixth or seventh rib pair. In addition, the rib clamps were tethered, through metallic threads, to a rigid frame placed on both sides of the animal. It should be stressed that the rib clamps, the metallic bars, and the metallic threads were locked while the animal was apneic at resting end expiration; when stiffened, therefore, the relaxed rib cage had the same configuration as in the control condition. The measurements were then repeated, first during passive lung inflation from 100 to 500 ml and then during isolated, tetanic stimulation of the transversus abdominis. As in the control condition, three trials were performed at each stimulus intensity.

Protocol 2. In the other seven animals, rib cage elastance was decreased by removing the bony ribs 3–8 on both sides of the chest. The procedure used to remove the bony ribs has been previously described in detail (3). Briefly, the periosteum of each rib was incised from the costochondral junction ventrally to the rib angle dorsally, and it was then carefully slit and peeled on both the external and the internal aspect of the rib with a curved, chisel-edged instrument. The bony ribs thus exposed were subsequently sectioned at their dorsal and ventral ends, such that a large fraction of the right and left lateral walls of the rib cage was made of bands of periosteum connected by intact intercostal muscles (Fig. 1B). A second set of measurements was obtained after a 15-min recovery period.

Data analysis. For each animal, the changes in Pao, Pab, and rib cage diameter recorded at each stimulus intensity in each condition were averaged over the three trials. The increases in Pao and rib cage diameter recorded during passive inflation were also averaged over the three trials, and the values of rib cage elastance were calculated by linear regression techniques. Data were then averaged for the animal group, and they are presented as means ± SE. Statistical comparisons between the changes obtained during stimulation in the different conditions were made by ANOVA with repeated measures, and multiple comparison testing of the mean values was performed, when appropriate, using Tukey’s honest tests. Statistical comparisons between the values of rib cage elastance in the different conditions were made by paired t-tests. The criterion for statistical analysis was taken as P < 0.05.

RESULTS

Effects of rib cage stiffening. Clamping the ribs and the sternum did not induce any change in the rib cage transverse diameter at functional residual capacity (before, 112.3 ± 3.6 mm; after, 113.2 ± 3.7 mm; P = not significant). As anticipated, however, the relationship between Pao and rib cage diameter during passive inflation was markedly altered such that a given rise in Pao was associated with a smaller increase in diameter (Fig. 2). As a result, whereas rib cage elastance in the control condition averaged 2.57 ± 0.39 cmH2O/mm, with the ribs and the sternum clamped it amounted to 19.54 ± 3.75 cmH2O/mm (P < 0.005).

A representative example of the traces obtained during bilateral stimulation of the transversus abdominis before and after clamping the ribs and sternum is shown in Fig. 3, A and B, and the changes in Pao, Pab, and rib cage transverse diameter recorded at all stimulus intensities in the seven animals are summarized in Fig. 4. Stimulating the transversus abdominis with the endotracheal tube occluded induced a rise in Pao and Pab and caused an increase in rib cage diameter in both conditions. All these changes increased progressively as the stimulus intensity increased (P < 0.001). However, as was the case during passive inflation, the increase in rib cage diameter during stimulation was consistently
smaller after clamping the ribs and sternum than in the control condition \((P < 0.001)\). Furthermore, the rise in \(P_a\) was similar to that measured during control, but the rise in \(P_o\) was much greater at all stimulus intensities \((P < 0.01)\). At the highest stimulus intensity, the rise in \(P_a\) for the seven animals was thus increased from \(3.30 \pm 0.52\) to \(5.85 \pm 1.16\) cmH\(2\)O \((P < 0.001)\).

**Effects of rib removal.** Removing the bony ribs induced disappearance of the normal outward curvature of the lateral walls of the rib cage, as described in our previous investigation \((3)\). After removal of the ribs, therefore, the rib cage transverse diameter at functional residual capacity was reduced from \(100.1 \pm 3.2\) to \(78.4 \pm 2.6\) mm \((P < 0.001)\). Also, the relationship between \(P_a\) and rib cage diameter during passive inflation was altered such that a given rise in \(P_a\) was associated with a greater increase in diameter (Fig. 5). For the seven animals, rib cage elastance was reduced from \(2.71 \pm 0.69\) to \(0.62 \pm 0.07\) cmH\(2\)O/mm \((P < 0.02)\), and this reduction was associated with marked alterations in the response to transversus stimulation, as shown in Fig. 3, \(C\) and \(D\), and Fig. 6. Thus, at all stimulus intensities, the rise in \(P_a\) after rib removal was essentially similar to that in the control condition, but the increase in rib cage diameter was greater \((P < 0.001)\), and the rise in \(P_o\) was lower \((P = 0.002)\). At the highest stimulus intensity, the rise in \(P_a\) for the seven animals averaged \(3.41 \pm 0.53\) cmH\(2\)O in the control condition, whereas after rib removal it was only \(1.29 \pm 0.25\) cmH\(2\)O \((P < 0.001)\).

**DISCUSSION**

In agreement with our hypothesis, increasing rib cage elastance by clamping the ribs and the sternum induced an increase in the rise in \(P_a\) generated by the transversus abdominis (Fig.

**Fig. 2.** Relationship between the rise in airway opening pressure (\(P_a\)) and the increase in RC transverse diameter during passive inflation (100–500 ml) before (●) and after (○) the ribs and sternum were locked. Values are means ± SE recorded in 7 animals.

**Fig. 3.** Representative example of the changes in \(P_a\), abdominal pressure (\(P_b\)), and RC transverse diameter recorded during bilateral stimulation of the transversus abdominis with the RC intact \((A)\) and after the ribs and sternum were locked \((B)\). Example of the changes in \(P_a\), \(P_b\), and RC diameter recorded during stimulation with the RC intact \((C)\) and after removal of the bony ribs 3–8 on the both sides of the chest \((D)\). The stimulus intensity in both examples was 30 V.
4). The increase in the change in Pao (ΔPao) was substantial, amounting to ~75% of the value recorded with the rib cage intact, yet the rise in Pab produced by the muscle was the same. With the ribs and sternum clamped, therefore, the pressure difference across the diaphragm was smaller than with the rib cage intact, and, hence, the cranial displacement of the diaphragm was also smaller. This should have resulted in a decrease, rather than an increase, in ΔPao. Conversely, when rib cage elastance was decreased by removal of the bony ribs, the rise in Pao produced by the transversus abdominis was reduced by 65% relative to the control condition, although the rise in Pab was unchanged (Fig. 6). Thus the pressure difference across the diaphragm and, with it, the cranial displacement of the diaphragm was greater, and this should have led to an increase, rather than a decrease, in ΔPao. The alterations in ΔPao observed in the two experiments of this study must, therefore, be exclusively attributed to the changes in rib cage elastance. In view of the magnitude of the alterations in ΔPao and the concomitant changes in diaphragmatic displacement, the conclusion can be drawn that rib cage elastance is, in fact, a major determinant of the mechanical coupling between the abdominal muscles and the lung.

The quantitative importance of the role played by rib cage elastance in determining this mechanical coupling was further evaluated by developing a two-compartment model of the chest wall, as shown in Fig. 7. The rib cage compartment and the diaphragm in this model are represented by the pistons and springs shown at the top and bottom of the cylinder, respectively, the lung is represented by the spring between the two
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in Pab (ΔPab) during transversus stimulation in our control animals was 0.35, which would correspond to a $K_R / K_{Di}$ of 0.55. In addition, the measurements obtained during passive inflation have shown that the elastance of the rib cage was increased by a factor of seven after the ribs and sternum were clamped (Fig. 2) and that it was reduced by a factor of four after removal of the ribs (Fig. 5). If $K_R$ in Eq. 6 is multiplied by 7, the relationship in Fig. 8 predicts that $Pao/Pab$ would be increased to ~0.80. On the other hand, if $K_R$ in Eq. 6 is replaced by $K_R/4$, $Pao/Pab$ would be reduced to ~0.12. Thus, according to this model analysis, a comparable ΔPab would be associated with a twofold increase in ΔPao when rib cage elastance is increased by clamping the ribs and sternum and would elicit a 66% decrease in ΔPao when rib cage elastance is decreased by removing the ribs. Although the increase in ΔPao observed after clamping the ribs and the sternum was only 75% of the control value, these results agree well with the experimental observations, and, hence, they further strengthen our conclusion that rib cage elastance is a major determinant of the lung-deflating action of the abdominal muscles.

Because $Pab$ is conventionally considered the main determinant of this action, we also compared the quantitative effect of rib cage elastance with that of $Pab$. The two experiments reported in this study indicate that, with the rib cage intact, a 50% reduction in ΔPab induces a 43% decrease in ΔPao; or, alternatively, a twofold increase in ΔPab causes a 76% increase in ΔPao. The results of the present study, combined with the relationship shown in Fig. 8, indicate that, with a 50% reduction in rib cage elastance, the decrease in ΔPao would amount to 37%. Also, with a twofold increase in rib cage elastance, the increase in ΔPao would be 51%. Thus, in the dog, the effect of rib cage elastance on the expiratory action of the abdominal muscles is slightly smaller than but of the same order of magnitude as the effect of $Pab$.

The relative importance of rib cage elastance and $Pab$ in determining the lung-deflating action of the abdominal muscles in humans is uncertain. On the basis of the present findings, however, one would predict that the ability of these muscles to increase pleural pressure during coughing would be greater than normal when the rib cage is made abnormally stiff by

\[ Pao = K_R V_R + K_L V_L \]  
\[ Pao = K_{Di} V_{Di} + K_L V_L + Pab \]

and

\[ Pab = K_A V_A + P_a \]

The volume displacement of the lung is the sum of the rib cage and diaphragm volume displacements, so that

\[ V_L = V_R + V_{Di} \]

and substituting for $V_R$ from Eq. 1 and for $V_{Di}$ from Eq. 2 yields the following relation

\[ V_L = \frac{Pao (K_R + K_{Di}) - K_R Pab - K_L V_L (K_R + K_{Di})}{K_{Di} (K_R + K_{Di})} \]

When the abdominal muscles contract alone with the endotracheal tube occluded ($V_L = 0$), Eq. 5 thus yields

\[ Pao = \frac{[K_R/(K_R + K_{Di})]}{Pab} \]

In other words, the ratio between the rise in $Pao$ and the rise in $Pab$ would exclusively depend on the relative values of $K_R$ and $K_{Di}$.

The relationship thus computed between $Pao/Pab$ and $K_R/K_{Di}$ is shown in Fig. 8. The ratio between Δ$Pao$ and the change...
ankylosing spondylitis (13, 14, 21). The increased rib cage elas-tance should also enhance the pressure-generating ability of the abdominal muscles in patients with severe kyphoscoliosis (2, 14), although the distortion of the diaphragm in such patients might be associated with an increase in passive tension in a number of diaphragmatic muscle fibers and might, therefore, impede the transmission of Pab to the pleural cavity. Furthermore, one would also predict that open-window thoracostomy would lead to opposite alterations in patients with chronic empyema. Indeed, this procedure involves resecting two to five adjacent ribs and the intercostal soft tissues so as to expose widely the empyema cavity, aspirate the pus and necrotic debris, and then fill the cavity with gauze and antibiotic solution (4, 5, 20, 23). In the last 4 yr, four patients with postpneumonectomy empyema have been treated by extensive open-window thoracostomy (3–4 ribs) at our institution. The procedure was successful in all patients; there was no recurrence of empyema and a marked improvement in the general condition, yet three patients complained of ineffective cough. Although no pressure measurement was performed, this is consistent with the idea that the ability of the abdominal muscles to increase pleural pressure was indeed diminished.

GRANTS

This study was supported by a grant (3.4521.01) from the Fonds National de la Recherche Scientifique, Belgium.

REFERENCES