Role of pleural pressure in the coupling between the intercostal muscles and the ribs

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Submitted 12 December 2006; accepted in final form 16 February 2007


The inspiratory intercostal muscles elevate the ribs and thereby elicit a fall in pleural pressure (ΔPpl) when they contract. In the present study, we initially tested the hypothesis that this ΔPpl does, in turn, oppose the rib elevation. The cranial rib displacement (Xr) produced by selective activation of the parasternal intercostal muscle in the fourth interspace was measured in dogs, first with the rib cage intact and then after ΔPpl was eliminated by bilateral pneumothorax. For a given parasternal contraction, Xr was greater after pneumothorax; the increase in Xr per unit decrease in ΔPpl was 0.98 ± 0.11 mm/cmH2O. Because this relation was similar to that obtained during isolated diaphragmatic contraction, we subsequently tested the hypothesis that the increase in Xr observed during breathing after diaphragmatic paralysis was, in part, the result of the decrease in ΔPpl, and the contribution of the difference in ΔPpl to the difference in Xr was determined by using the relation between Xr and ΔPpl during passive inflation. With diaphragmatic paralysis, Xr during inspiration increased approximately threefold, and 47 ± 8% of this increase was accounted for by the decrease in ΔPpl. These observations indicate that 1) ΔPpl is a primary determinant of rib motion during intercostal muscle contraction and 2) the decrease in ΔPpl and the increase in intercostal muscle activity contribute equally to the increase in inspiratory cranial displacement of the ribs after diaphragm paralysis.

respiratory muscles; diaphragm

IT IS NOW well established, both in humans and in animals, that the intercartilaginous portion of the internal intercostals (the so-called parasternal intercostals) and the external intercostals in the dorsal portion of the rostral interspaces contract in concert with the diaphragm during the inspiratory phase of the breathing cycle (see Ref. 9 for review). These two sets of intercostal muscles, as their primary action, elevate the ribs and expand the rib cage; in so doing, they cause a fall in pleural pressure (ΔPpl) and an expansion of the lung. Moreover, by causing a ΔPpl, these muscles also prevent the diaphragmatic muscle fibers from shortening excessively. As a result, the diaphragm develops greater pressure during simultaneous diaphragm-intercostal contraction than it does during isolated contraction (6, 16).

Whereas the inspiratory intercostal muscles enhance the force developed by the diaphragm, the diaphragm has no synergistic or antagonistic effect on the force developed by the parasternal intercostals (10). Thus the insertions of these muscles on the sternum, the costal cartilages, and the ribs impose significant constraint on their length; thus, although isolated contraction of the diaphragm causes lengthening of the parasternal intercostals (4), these length changes are small and leave the force-generating ability of the muscles essentially unchanged. In a recent study, however, we (10) found that the rib elevation produced by contraction of the parasternal intercostal in a single interspace is smaller when this contraction is superimposed on diaphragmatic contraction than when the same parasternal contraction occurs with the diaphragm relaxed. Concomitantly, the ΔPpl produced by the parasternal intercostal was larger in the first instance (i.e., when diaphragmatic elastance was greater) than in the second. No attempt was made in the study to identify the mechanism of the reduction in rib elevation during combined parasternal intercostal-diaphragm contraction, but the rationale was offered that the ΔPpl generated by the inspiratory intercostals, although being the result of the cranial rib displacement, would act as a load on the ribs, tending to pull them inward and caudally. If so, the rib elevation associated with a given parasternal force would be reduced as ΔPpl is increased by a concomitant contraction of the diaphragm (10).

The present study was initially undertaken to test the hypothesis that the ΔPpl generated by the inspiratory intercostals does effectively oppose the rib elevation. The rib displacement produced by selective activation of the parasternal intercostal was measured, first with the rib cage intact and then after ΔPpl was eliminated by a bilateral pneumothorax. The cranial rib displacement was greater after pneumothorax than before, and the difference in rib displacement was similar to the difference, for a comparable difference in ΔPpl, during isolated diaphragmatic contraction. In a second set of experiments, therefore, we also tested the related hypothesis that the marked increase in inspiratory rib elevation observed after diaphragmatic paralysis (2, 5, 7, 14) is not exclusively the result of the increase in inspiratory intercostal activity as conventionally thought but is, in part, related to the decrease in ΔPpl induced by paralysis.

METHODS

The studies were carried out on 12 adult cross-breed dogs (18–25 kg body wt); studies were approved by the Animal Ethics and Welfare Committee of the Brussels School of Medicine. The animals were anesthetized with pentobarbital sodium (initial dose of 30 mg/kg iv), placed in the supine posture, and intubated with a cuffed endotracheal tube. A venous cannula was inserted in the forelimb to give maintenance doses of anesthetic (3–5 mg·kg−1·h−1 iv), and a venous Cannula was inserted in the form to give maintenance doses of anesthetic

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first to the ninth rib by reflection of the skin and the superficial muscle layers. Two experimental protocols were then followed.

**Experiment 1.** Five animals were studied first to define the role played by Ppl in determining the coupling between the inspiratory intercostals and the ribs and to compare quantitatively this role with the effect of Ppl on rib motion during isolated diaphragmatic contraction. A pair of stimulating electrodes was thus implanted in the parasternal intercostal muscle in the fourth interspace on either side of the sternum. These electrodes were silver hooks insulated with polyethylene tubing except for their terminal 8 mm, and their implantation was made superficially along muscle bundles situated in the vicinity of the sternum, i.e., in the area of the parasternal intercostal with the greatest inspiratory mechanical advantage (11). In addition, the internal intercostal nerve in the fourth interspace was bilaterally sectioned at the chondrocostal junction such that stimulation of the parasternal intercostal would not elicit any antidromic stimulation of the internal interosseous intercostal muscle in the same segment and/or stimulation, through spindle afferents, of intercostal muscles in contiguous segments (17, 19). It is worth pointing out that great care was taken not to damage intercostal vessels during nerve section; blood supply to the parasternal intercostal, therefore, was kept intact throughout the experiment.

After the internal intercostal nerve was sectioned, the C5 phrenic nerve roots were isolated bilaterally in the neck and laid over two pairs of insulated stainless steel stimulating electrodes, and a differential pressure transducer (Validyne, Northridge, CA) was connected to a side port of the endotracheal tube to measure airway opening pressure (Pao). A hook was also screwed into the fifth rib in the midaxillary line and connected to a linear displacement transducer (Schaevitz Engineering, Pennsauken, NJ) to measure the cranio-caudal (axial) rib displacement (Xr), as previously described (7).

The animal was allowed to recover for 15–20 min after instrumentation, after which it was made apnoeic by mechanical hyperventilation. Square pulses of 0.2-ms duration and 20-Hz frequency were then delivered at intervals to the parasternal intercostal muscle. The stimulus intensity was increased by increasing the voltage of stimulation. At least four voltages between 6 and 15 V were used in each animal, and, for each voltage, two trials of stimulation were performed while the endotracheal tube was occluded at functional residual capacity. When this procedure was completed, square pulses of 0.1-ms duration and supramaximal voltage were applied at a frequency of 20 impulses/s to the left and right C5 phrenic nerve roots. This stimulation was also performed after the animal was hyperventilated to apnoea and the endotracheal tube was occluded. Isolated stimulation of the phrenic nerves, however, causes a marked caudal displacement of the upper ribs (3, 10), and it is well established that the rib cage in the dog becomes less compliant when it contracts below its resting, end-expiratory volume (8, 15). Consequently, during phrenic nerve stimulation at functional residual capacity, a given force applied on the ribs, such as a fall in Ppl, would produce a smaller rib displacement.

To overcome the influence of this confounding factor, stimulation of the C5 phrenic nerve roots in our animals was therefore performed after passive inflation of the respiratory system. The level of inflation was adjusted between 500 and 900 ml so that, in each animal, the ribs during stimulation were maintained above their resting, end-expiratory position. Three trials of phrenic nerve stimulation were performed.

After completion of these measurements, the animal was reconnected to the ventilator and the intercostal muscles in the second interspace were severed over ~2 cm at the chondrocostal junction to induce a bilateral pneumothorax. Stimulation of the parasternal intercostal muscle and the C5 phrenic nerve roots was then repeated. Phrenic nerve stimulation after pneumothorax, however, was made without any prior inflation of the respiratory system because it no longer produced a caudal displacement of the ribs (see RESULTS).

**Experiment 2.** Seven animals were studied next to evaluate the role of Ppl in causing the increase in inspiratory rib elevation after diaphragm paralysis. The vagi in each animal were isolated in the neck, infiltrated with 2% lidocaine (lidocaine), and sectioned. Also, the C5, C6, and C7 phrenic nerve roots were bilaterally isolated, and loose ligatures were placed around them so that they could be easily sectioned later.

All measurements were made while the animals were spontaneously breathing. The axial displacement of the fifth rib was measured as described for experiment 1, and the changes in Ppl were measured with a balloon-catheter system placed in the midesophagus; the balloon was filled with 0.5 ml of air, and its position was adjusted by use of the occlusion technique (1). In addition, the changes in lung volume were measured with a heated Fleisch pneumotachograph coupled with a differential pressure transducer, and the EMGs of the parasternal and external intercostal muscles were recorded with pairs of silver hook electrodes spaced 3–4 mm apart. Each electrode pair was placed in parallel fibers and inserted in the muscle area receiving the greatest neural inspiratory drive. Implantation of the parasternal intercostal electrodes, therefore, was made in the third interspace in the muscle bundles situated near the sternum (11, 22, 24), and implantation of the external intercostal electrodes was made in the dorsal portion of the second interspace, immediately ventral to the rib angle (18, 20, 23). The two EMG signals were processed with amplifiers (model 830/1; CWE, Ardmore, PA), band-pass filtered below 100 and above 2,000 Hz, and rectified before their passage through leaky integrators with a time constant of 0.2 s.

After a 30-min recovery period, baseline measurements of tidal volume, Ppl, rib motion, and intercostal EMG activity during resting breathing were obtained. Three runs of 15 breaths were recorded over a 30-min period, after which the phrenic nerve roots were infiltrated with lidocaine and sectioned. Three runs of resting breathing were also obtained in this condition. After these measurements were completed, the animal was given supplemental anesthesia (5–8 mg/kg) and atracurium besylate (0.5 mg/kg iv), and the animal was connected to the ventilator to assess the relationship between axial rib motion and Ppl during passive inflation.

The animals in experiment 1 were maintained at a constant, rather deep level of anesthesia throughout. They had no corneal reflex and no limb movement, including during phrenic nerve stimulation. In contrast, to avoid obscuring the response to diaphragm paralysis, we regulated the anesthesia in the animals of experiment 2 to keep the corneal reflex present throughout the measurements during spontaneous breathing. Rectal temperature in these animals was also maintained constant between 36 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 changes in Pao (∆Pao) and Xr induced by each parasternal intercostal contraction (i.e., each stimulation intensity) before and after pneumothorax were averaged over the two trials. The difference between Xr after vs. before pneumothorax (∆Xr) was then divided by the ∆Pao measured before pneumothorax, and the values of ∆Xr/∆Pao thus obtained for the different stimulation intensities were averaged to quantify the rib displacement corresponding to a ∆Ppl of 1 cmH2O during parasternal contraction. The ∆Pao and Xr induced by phrenic nerve stimulation were analyzed similarly.

The evaluation of the role played by Ppl in causing the increase in inspiratory rib elevation after diaphragm paralysis (experiment 2) was made in two stages. First, for each individual animal, tidal volume, ∆Ppl, Xr, and phasic inspiratory EMG activity in the parasternal and external intercostals before and after phrenic nerve section were averaged over 10 consecutive breaths from each run. Inspiratory EMG activity in each muscle was first quantified by measuring the peak height of the integrated EMG signal in arbitrary units, and it was then expressed as a percentage of the activity recorded before phrenic section (control). The parasternal intercostal EMG signal was also used as a marker of inspiration and, therefore, as a time reference for Xr; consequently, the Xr values that were considered in the study’s
calculated results exclusively from the contraction of inspiratory muscles and were not corrupted by the relaxation of the expiratory muscles, in particular the triangularis sterni, at the end of expiration (12). Second, the slope of the relationship between Xr and ΔPpl during passive inflation was measured, and this value was multiplied by the difference between ΔPpl measured during breathing after vs. before phrenic section to yield the increase in Xr attributable to the loss in ΔPpl after phrenic section. The value thus calculated was compared with the increase in Xr observed after phrenic section.

The data were finally averaged over the animal group, and they are presented as means ± SE. Comparisons between the values of Xr obtained during parasternal intercostal contraction after vs. before pneumothorax (experiment 1) and between the values of ΔXr/ΔPao derived from parasternal contraction vs. phrenic nerve stimulation were made by paired t-tests. Statistical assessments of the effects of phrenic nerve section on tidal volume, ΔPpl, Xr, and intercostal EMG activity during spontaneous breathing (experiment 2) were made similarly. The criterion for statistical significance was taken as P < 0.05.

RESULTS

Experiment 1. The records of ΔPao and Xr obtained in a representative animal during contraction of the fourth parasternal intercostal muscle with the rib cage intact and after pneumothorax are shown in Fig. 1A, and the values of ΔPao and Xr obtained for all stimulation intensities in the same animal are shown in Fig. 1, B and C, respectively. Stimulating the parasternal intercostal with the rib cage intact produced a cranial displacement of the fifth rib and a fall in Pao, and these effects increased in magnitude as the stimulation intensity was increased. A similar pattern of rib displacement was observed after the pneumothorax was performed and the fall in Pao was abolished. For any given stimulation, however, Xr was larger. When the data obtained for all stimulation intensities were averaged, Xr in this particular animal therefore increased from 3.50 to 9.67 mm. Phrenic nerve section, however, caused an abolition of the inspiratory cranial displacement of the fifth rib and a fall in Pao, and these effects increased in magnitude as the stimulation intensity was increased. A similar pattern of rib displacement was observed after the pneumothorax was performed and the fall in Pao was abolished. For any given stimulation, however, Xr was larger.

The records of ΔPao and Xr obtained in the same animal during isolated stimulation of the C5 phrenic nerve roots with the rib cage intact and after pneumothorax are shown in Fig. 2. During stimulation with the rib cage intact, Pao fell by 8.0 cmH2O and the rib moved 6.56 mm caudally. During stimulation after pneumothorax, however, no ΔPao occurred and the rib remained stationary. As a result, the value obtained for ΔXr/ΔPao during phrenic nerve stimulation in this animal was 0.85 mm/cmH2O.

Every animal showed similar responses to pneumothorax. Xr during parasternal contraction thus increased from 3.13 ± 0.07 mm/cmH2O and the rib moved 6.56 mm caudally. During stimulation after pneumothorax, however, no ΔPao occurred and the rib remained stationary. As a result, the value obtained for ΔXr/ΔPao during phrenic nerve stimulation in this animal was 0.85 mm/cmH2O.

Experiment 2. The effects of sectioning the phrenic nerve roots on tidal volume, ΔPpl, rib motion, and intercostal EMG activity during resting breathing are illustrated by the records of a representative animal in Fig. 3. With phrenic nerve section, the peak height of the integrated parasternal and external intercostal EMG signals in this animal increased, respectively, to 135 and 387% of the control value, and the inspiratory cranial displacement of the fifth rib increased from 3.50 to 9.67 mm. Phrenic nerve section, however, also caused
a decrease in tidal volume from 456 to 281 ml and a decrease in ΔPpl from 5.79 to 3.60 cmH2O. The decrease in ΔPpl thus amounted to 2.19 cmH2O. Because Xr/ΔPpl during passive inflation in this animal was 1.01 mm/cmH2O, the increase in inspiratory cranial rib displacement attributable to the loss in ΔPpl after phrenic section was, therefore, 2.19 × 1.01 mm, i.e., 2.19 mm, which corresponded to 35% of the observed increase in rib displacement (6.17 mm).

The results of the analysis for the individual animals are shown in Table 1. Parasternal and external intercostal EMG activities after phrenic section were, respectively, 144 ± 12 (P < 0.01) and 429 ± 95% (P < 0.02) of control, and the increase in cranial rib displacement amounted to 6.72 ± 0.30 mm (before: 2.87 ± 0.39 mm; after: 9.60 ± 0.47 mm; P < 0.001). The concomitant decrease in ΔPpl was 2.78 ± 0.49 cmH2O (before: 6.28 ± 0.55 cmH2O; after: 3.49 ± 0.09 cmH2O; P < 0.001), and Xr/ΔPpl during passive inflation was 1.12 ± 0.10 mm/cmH2O. Therefore, the increase in inspiratory cranial rib displacement corresponding to the loss in ΔPpl was 3.09 ± 0.61 mm and represented, on average, 47 ± 8% of the observed increase in rib displacement. It must be pointed out, however, that this computed fraction was variable among the different animals, ranging from 14% (dog 3) to 79% (dog 5). As shown in Fig. 4, this fraction showed a negative linear relationship with the amplitude of the increase in parasternal intercostal activity (r = 0.811; P < 0.05). This fraction also tended to decrease as the increase in external intercostal activity was greater, but the relationship did not reach the level of statistical significance (r = 0.670).

**DISCUSSION**

One of the main results of this study is the observation that the cranial displacement of the ribs produced by isolated contraction of the parasternal intercostals was larger when the contraction occurred after a pneumothorax was performed and ΔPpl was eliminated than when the same contraction took place with the rib cage intact (Fig. 1). The results also showed that this increase in rib displacement was similar to the difference, for a given difference in ΔPpl, during isolated stimulation of the phrenic nerves. This similarity is important because previous studies in dogs and rabbits by D’Angelo and Sant’Ambrogio (3) have shown that isolated phrenic nerve contraction produces a similar increase in rib displacement. Since the cranial displacement of the ribs was greater after phrenic paralysis, it appears that the increase in rib displacement is due to the decrease in ΔPpl rather than to any direct effect of phrenic paralysis on the intercostal muscles. The results also showed that the computed fraction was variable among the different animals, ranging from 14% (dog 3) to 79% (dog 5). As shown in Fig. 4, this fraction showed a negative linear relationship with the amplitude of the increase in parasternal intercostal activity (r = 0.811; P < 0.05). This fraction also tended to decrease as the increase in external intercostal activity was greater, but the relationship did not reach the level of statistical significance (r = 0.670).

**Table 1. Computed contribution of pleural pressure to the increase in inspiratory Xr after diaphragm paralysis**

<table>
<thead>
<tr>
<th>Dog</th>
<th>Increase in Xr After Paralysis, mm</th>
<th>Decrease in Ppl After Paralysis, cmH2O</th>
<th>Xr/ΔPpl During Passive Inflation, mm/cmH2O</th>
<th>Increase in Xr Due to ΔPpl, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6.86</td>
<td>4.61</td>
<td>0.90</td>
<td>60</td>
</tr>
<tr>
<td>2</td>
<td>5.42</td>
<td>2.43</td>
<td>1.20</td>
<td>54</td>
</tr>
<tr>
<td>3</td>
<td>6.62</td>
<td>0.99</td>
<td>0.93</td>
<td>14</td>
</tr>
<tr>
<td>4</td>
<td>6.81</td>
<td>3.99</td>
<td>0.96</td>
<td>56</td>
</tr>
<tr>
<td>5</td>
<td>7.27</td>
<td>3.47</td>
<td>1.65</td>
<td>79</td>
</tr>
<tr>
<td>6</td>
<td>6.17</td>
<td>2.19</td>
<td>1.01</td>
<td>35</td>
</tr>
<tr>
<td>7</td>
<td>7.92</td>
<td>1.86</td>
<td>1.18</td>
<td>28</td>
</tr>
<tr>
<td>Mean</td>
<td>6.72</td>
<td>2.78</td>
<td>1.12</td>
<td>47</td>
</tr>
<tr>
<td>SE</td>
<td>0.30</td>
<td>0.49</td>
<td>0.10</td>
<td>8</td>
</tr>
</tbody>
</table>

Xr, cranial rib displacement; Ppl, pleural pressure; Δ, change.
stimulation causes a decrease in the cross-sectional area of the rostral portion of the rib cage; when the stimulation was repeated after pneumothorax, however, the cross-sectional area of the rostral rib cage remained unchanged. Similarly, every animal in the present study showed a large caudal displacement of rib 5 when the C5 phrenic nerve roots were stimulated with the rib cage intact and no rib displacement when the nerves were stimulated after pneumothorax (Fig. 2), thus confirming that the action of the diaphragm on the rostral portion of the rib cage is exclusively related to ΔPpl. The finding that the Xr per unit ΔPpl during parasternal contraction was the same as that observed during phrenic nerve stimulation is, therefore, unequivocal evidence that the increase in rib displacement after pneumothorax was related to the suppression of ΔPpl per se, rather than any alteration in regional rib cage elastance or parasternal muscle length that the induction of pneumothorax might have introduced.

On the basis of these results, the conclusion can therefore be drawn that the net cranial displacement of the ribs produced by contraction of the inspiratory intercostals is determined by the balance between two forces acting in opposite directions. Thus the muscles exert a direct force on the ribs, which displaces them cranially and outward. However, because this displacement induces a ΔPpl, a second force operates, which is oriented inward and, therefore, opposes the cranial and outward rib displacement.

This balance of forces can be analyzed in a more formal way by using a simple, two-compartment model of the chest wall; this model was recently developed to evaluate the interaction between the diaphragm and intercostal muscles on the lung (6) and is shown in Fig. 5. The rib cage and abdominal compartments in the model are represented by the pistons, springs, and muscles shown at the top and the bottom of the cylinder, respectively, and the lung is represented by the spring between the two pistons. If the volume displacements of the rib cage and abdominal compartment are denoted VR and VA, the elastances of the compartments are denoted KR and KA, the volume displacement and the elastance of the lung spring are denoted VL and KL, and the effective pressures (i.e., an index of the forces) developed by the inspiratory intercostals and the diaphragm are denoted PR and PA, then the equations of static equilibrium of the two pistons are

\[
P_{\text{ao}} + P_R = K_R V_R + K_L V_L
\]  
and

\[
P_{\text{ao}} + P_A = K_A V_A + K_L V_L
\]

The volume displacement of the lung is the sum of the compartmental volume displacements, so that

\[
V_L = V_R + V_A
\]

and substituting for VR and VA from Eqs. 1 and 2 yields the following relation:

\[
V_L = \left[ P_{\text{ao}}(K_R + K_A) + P_A K_R + P_R K_A - K_L V_L(K_R + K_A) \right]/K_R K_A
\]

When the endotracheal tube is occluded, as was the case in our animals, Vl = 0 and Pao = Ppl. Therefore, Eqs. 1 and 4 yield, respectively,

\[
V_R = (P_R + P_{\text{pl}})/K_R
\]

and

\[
P_{\text{pl}} = -(P_A K_R + P_R K_A)/(K_R + K_A)
\]

From Eq. 5, it can be seen that VR (i.e., the volume equivalent of the cranial rib displacement) is directly related to the sum of the force generated by the intercostal muscles and pleural pressure and inversely related to the elastance of the rib cage. Thus, during isolated contraction of the inspiratory intercostals (P_A = 0) in the presence of pneumothorax (P_pl = 0), VR = P_R/K_R. If the rib cage is intact, however, the same PR is effective in causing a fall in Ppl, so that VR is smaller. In addition, VR is further decreased if 1) the rib cage is stiffer, leading to an increase in KR, or 2) the same Prc takes place with a simultaneous contraction of the diaphragm, such that Pab is no longer zero and Ppl is more negative (Eq. 6). This model analysis, combined with the results of experiment 1, thus confirms our previous speculation (10) that the decrease in Xr observed during combined parasternal intercostal-diaphragm contraction is partly due to the increase in ΔPpl.

Another implication of this force-balance analysis is that an isolated reduction in ΔPpl during breathing should lead to an increase in Xr. To test this prediction, we sectioned the phrenic nerve roots so as to induce a complete paralysis of the diaphragm; indeed, in agreement with previous studies (2, 5, 7, 14), phrenic nerve section resulted in a significant reduction in ΔPpl associated with a large increase in the inspiratory cranial displacement of the ribs. Phrenic nerve section, however, also elicited a substantial increase in the inspiratory activity recorded from the parasternal and external intercostals, thus implying that there was a concomitant increase in the force applied by these muscles on the ribs. To overcome this confounding factor and quantify the contribution of the loss in ΔPpl to the increase in rib displacement after phrenic section, we thus multiplied, for each individual animal, the pressure

![Fig. 5. Diagram of the chest wall. The rib cage and abdominal compartments are represented by the 2 pistons at the top and the bottom of the cylinder; see text for further explanation. VR, volume displacement of the rib cage; VA, volume displacement of the abdomen; KR, elastance of the rib cage; KL, elastance of the lung; KA, elastance of the abdomen.](Image)
loss by the ratio of $X_r$ over $\Delta P_{pl}$ obtained during passive inflation. Whereas it is usually considered that the increase in rib displacement after diaphragmatic paralysis is primarily related to the increase in intercostal activity, the results of these calculations indicated that, on average, nearly half of the increase in rib displacement is, in fact, the result of the pressure loss.

Although this conclusion was obtained by use of a rather indirect method, its validity is supported by two lines of evidence. First, it would be expected that the loss in $\Delta P_{pl}$ after diaphragmatic paralysis would make a larger contribution to the increase in rib displacement as the increase in intercostal activity is smaller. That is, if diaphragmatic paralysis did not elicit any compensatory increase in intercostal activity, the loss in $\Delta P_{pl}$ would contribute the entire increase in rib displacement; conversely, if the increase in intercostal activity after paralysis was large enough to keep $\Delta P_{pl}$ constant, the contribution of $\Delta P_{pl}$ to the increase in rib displacement would be zero. As shown in Fig. 4, our animals did show an inverse relationship between the magnitude of the increase in parasternal intercostal activity and the contribution of $\Delta P_{pl}$ to the increase in rib displacement.

The second line of evidence is provided by calculating, for each animal in the two conditions of the experiment, the $X_r$ that would be theoretically produced by the intercostal muscles if there were no concomitant $\Delta P_{pl}$. On the basis of the force-balance previously developed (see above), one can compute this displacement by adding the observed (net) rib displacement to the product of the observed $\Delta P_{pl}$ and the value of $X_r/\Delta P_{pl}$ measured during passive inflation. For the seven animals, the theoretical rib displacement thus computed after diaphragmatic paralysis represented, on average, 143 ± 13% of that computed for the control condition. Thus, although these computed values were based exclusively on mechanical measurements, the result agrees well with the magnitude of parasternal intercostal EMG activity after paralysis, and it confirms that, after phrenic nerve section in our animals, the force exerted by the inspiratory intercostals on the ribs increased by ~40%. The threefold increase in the $X_r$ must, therefore, involve another mechanism, i.e., the loss in $\Delta P_{pl}$.

Alterations in $\Delta P_{pl}$ could be involved in determining the pattern of rib cage motion in conditions other than diaphragmatic paralysis. For example, in a recent study of the effects of ascites on inspiratory muscle function (21), it was found that severe ascites in dogs, acting through a marked increase in abdominal elastance, impairs the lung-expanding action of the diaphragm and elicits a compensatory increase in parasternal intercostal activity. The inspiratory $X_r$ was also increased, and conventional wisdom would maintain that this increased rib displacement is the result of two mechanisms working in combination, namely, the increased inspiratory intercostal activity and the greater rib cage-expanding action of the diaphragm. Indeed, inasmuch as abdominal elastance in this condition is markedly increased, one would expect that the insertional and appositional forces of the diaphragm would be increased, such that a given activation of the muscle would lead to a greater cranial displacement of the lower ribs (13, 25). A thorough examination of the effects of ascites, however, indicates that severe ascites induced both an increase in parasternal activity and a large reduction in tidal volume (21). It would be reasonable to speculate, therefore, that the increase in rib displacement in ascites is also related, in part, to the mechanism described in the present study. Although the role of this factor cannot be quantified at this stage, this example highlights the fact that the present findings may provide not only a better understanding of the mechanical interaction between the diaphragm and the intercostal muscles but also new insights into the mechanisms of chest wall displacement in disease.

GRANTS

This study was supported in part by the Fonds National de la Recherche Scientifique (Belgium; Grant 3-4509-04).

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