Respiratory effect of the lower rib displacement produced by the diaphragm

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De Troyer A. Respiratory effect of the lower rib displacement produced by the diaphragm. J Appl Physiol 112: 529–534, 2012. First published December 1, 2011; doi:10.1152/japplphysiol.01067.2011.—The diaphragm acting alone causes a cranial displacement of the lower ribs and a caudal displacement of the upper ribs. The respiratory effect of the lower rib displacement, however, is uncertain. In the present study, two sets of experiments were performed in dogs to assess this effect. In the first experiment, the animals were given a muscle relaxant to abolish respiratory muscle activity, and external, cranially oriented forces were applied to the lower rib pairs to simulate the action of the diaphragm on these ribs. The data showed that 1) holding the lower ribs stationary during spontaneous, isolated diaphragm contraction had no effect on the change in lung volume during unimpeded inspiration and no effect on the fall in pleural pressure (Ppl) during occluded breaths; 2) the procedure, however, caused an increase in the caudal displacement of the upper ribs; and 3) pulling the lower rib pairs cranially induced a cranial displacement of the upper ribs and a small fall in Ppl. These observations indicate that the force applied on the lower ribs by the diaphragm during spontaneous contraction, acting through the interdependence of the ribs, is transmitted to the upper ribs and has an inspiratory effect on the lung. However, this effect is very small compared to that of the descent of the dome.

The studies were carried out on 12 adult bred-for-research dogs (21–30 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 position and intubated with auffed endotracheal tube, and a venous cannula was inserted in the forelimb to give maintenance doses of anesthetic (3–5 mg·kg⁻¹·h⁻¹ iv). The rib cage and intercostal muscles were exposed on both sides of the chest from the 1st to 11th.

THE DIAPHRAGM functions essentially like a piston. As its muscle fibers are activated and shorten, the dome of the diaphragm descends, the pleural cavity expands along its craniocaudal axis, and the abdominal viscera are displaced caudally. As a result, pleural pressure (Ppl) falls, abdominal pressure (Pab) rises, and the ventral abdominal wall moves outward. In addition, measurements of thoracoabdominal motion in humans with complete, traumatic transection of the cervical cord (2, 10, 17, 18) and measurements of rib motion after denervation of all the inspiratory intercostal muscles in dogs (3) have shown that isolated contraction of the diaphragm during breathing induces both a caudal and inward displacement of the upper ribs and a cranial and outward displacement of the lower ribs. Conventional wisdom maintains that the displacement of the upper ribs by the diaphragm is the result of the fall in Ppl and that the displacement of the lower ribs is determined by the combination of two mechanisms, namely 1) the direct, cranially oriented force applied by the muscle fibers of the costal portion of the diaphragm on the ribs into which they insert; and 2) the transmission of the rise in Pab through the zone of apposition of the diaphragm to the rib cage (8, 14, 15).

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rib by reflection of the skin and superficial muscle layers, and two hooks were screwed in the right 5th and 10th ribs in the midaxillary line and connected to linear displacement transducers (Schaevitz Engineering, Pennsauken, NJ) to measure the craniocaudal (axial) displacement of the upper and lower ribs, respectively. This technique has been previously described (4). Additional hooks were implanted in the 10th rib pair so that the ribs could be manipulated later. Two experimental protocols were then followed.

**Experiment 1.** Seven animals were prepared first as described in detail in a recent report (3). Thus the abdomen was opened by a 4-cm-long midline incision cranial to the umbilicus, and a balloon-catheter system filled with 1.0 ml of air was placed between the stomach and the liver to measure Pab. After the abdomen was closely sutured, the external intercostal muscles in interspaces 1–8 were bilaterally severed from the chondrocostal junctions to the spine, and the internal intercostal nerves in these interspaces were sectioned at the chondrocostal junctions to denervate the parasternal intercostal muscles. The parasternal intercostal in the second left interspace, however, was left intact, and a pair of silver hook electrodes spaced 3–4 mm apart was implanted in parallel fibers in the muscle area near the sternum to quantify neural inspiratory drive and to provide a time reference for rib displacement and pressure. The electromyographic (EMG) signal thus obtained was processed as previously described (3).

The animal was allowed to recover for 30 min after surgery, after which a differential pressure transducer (Validyne, Northridge, CA) was connected to a side port of the endotracheal tube to measure airway opening pressure (Pao). A heated Fleisch pneumotachograph coupled with a differential pressure transducer was also connected to the endotracheal tube to measure the changes in lung volume, after which baseline measurements of tidal volume, Pao, Pab, rib motion, and parasternal intercostal EMG activity were obtained. The animal maintained spontaneous diaphragmatic breathing throughout. Every 10–15 breaths, however, the hooks implanted in the 10th rib pair were gently grasped at end-expiration and maintained stationary for one breath so as to prevent the ribs from moving cranially and outward. Ten to twelve trials were recorded in each animal.

After completion of these measurements, the endotracheal tube was occluded at regular intervals at end-expiration for a single inspiratory effort. Four to five occluded breaths during which rib 10 was allowed to move freely alternating with four to five occluded breaths during which rib 10 was maintained stationary were recorded in each animal.

**Experiment 2.** Five animals were studied next to evaluate the response of Pao and the upper ribs to the isolated cranial displacement of the lower ribs. Clamps were attached bilaterally to ribs 9 and 10, i.e., to the two rib pairs into which the diaphragm predominantly inserts in the dog (11), and, as in Expt 1, a differential pressure transducer was connected to a side port of the endotracheal tube to measure Pao. The animal was then injected with a neuromuscular blocking agent (4 mg pancuronium iv) and connected to a mechanical ventilator (Harvard Pump, Chicago, IL). After the animal was disconnected from the ventilator, the endotracheal tube was occluded, and the rib clamps on both sides of the chest were grasped and pulled gradually in the cranial and outward direction until rib 10 was displaced ~5 mm cranially. Three trials were performed in each animal. After the procedure was completed, the intercostal muscles in the third interspace were severed over ~2 cm at the chondrocostal junction to induce a unilateral pneumothorax and, thus, to eliminate the potential influence of Ppl on the mechanical coupling between the lower ribs and the upper ribs, and cranial displacement of the rib clamps was repeated in triplicate.

The animals in Expt 1 were maintained at a constant level of anesthesia. They had no pupillary light reflex and made no spontaneous movements other than respiratory movements both during the surgery and during the measurements, including when external forces were applied on rib 10; the corneal reflex, however, was maintained. The animals in Expt 2 were maintained at a deeper level of anesthesia throughout, so that the corneal reflex was suppressed; they also received an additional dose of anesthetic (10 mg/kg) just before the muscle relaxant was given. Rectal temperature was kept constant between 36 and 38°C with infrared lamps. At the end of the experiment, the animals were given an overdose of anesthetic (30–40 mg/kg iv).

**Data analysis.** For each animal of Expt 1, the change in lung volume, the inspiratory axial displacement of rib 10 and rib 5, the change in Pab, and phasic inspiratory EMG activity in the parasternal intercostal muscle of the second left interspace were measured for each tidal breath during which rib 10 was held stationary and for each preceding, unimpeded (control) breath. The values were then averaged over the 10–12 trials. Rib displacements in the cranial direction were given a positive sign, and rib displacements in the caudal direction were given a negative sign. Also, although inspiratory EMG activity was first quantified by measuring the peak height of the integrated EMG signal in arbitrary units, it was expressed as a percentage of the activity recorded during the control breaths.

The peak values of ΔPao, ΔPab, axial displacement of rib 10 and rib 5, and parasternal intercostal EMG activity obtained for each occluded breath with the rib 10 stationary and the values obtained for each occluded breath with the rib unimpeded (control) were also averaged over the four to five trials. In addition, for each occluded breath in each animal, the axial displacement of rib 5 was measured at 2-cmH₂O increments of ΔPao, and rib displacement was plotted against ΔPao. It should be emphasized that all values of rib displacement and ΔPao were measured relative to the onset of the inspiratory burst in the parasternal intercostal. Consequently, the rib displacements and ΔPao’s that were considered in the data analysis resulted exclusively from the contraction of the diaphragm (and, in half of the breaths, the external force applied on the 10th rib pair) and were not corrupted by the relaxation of the abdominal muscles and the internal interosseous intercostals at the end of expiration.

For each animal of Expt 2, the axial position of rib 5 and rib 10 during each cranial pull of the 9th and 10th rib pairs before and after pneumothorax was measured relative to the relaxation position of the rib, and the values of rib 5 position were plotted against the corresponding position of rib 10. The values of ΔPao before pneumothorax were also plotted against the position of rib 10. The values obtained in the different trials were then averaged, the relationships were fitted by quadratic equations, and the positions of rib 5 and ΔPao at fixed rib 10 positions at 1-mm increments were determined from these equations.

Data in both experiments were finally averaged across the animal group, and they are presented as means ± SE. Statistical assessment of the effect of holding rib 10 stationary on tidal volume, ΔPao, ΔPab, axial rib displacement, and parasternal intercostal EMG activity (Expt 1) was made by using paired t-tests. Statistical analysis of the effect of pneumothorax on the displacement of rib 5 during cranial pull of ribs 9 and 10 (Expt 2) was made using ANOVA with repeated measures. The criterion for statistical significance was taken as P < 0.05.

**RESULTS**

Effects of maintaining the lower ribs stationary on tidal volume (Expt 1). Figure 1 shows the records of lung volume, Pab, rib displacement, and parasternal intercostal EMG activity in the second left interspace obtained in a representative animal during two unimpeded breaths performed with the diaphragm alone followed by one breath during which rib 10 on both sides of the chest was held stationary. During the inspiratory phase of unimpeded breathing, rib 10 moved in the cranial direction in all animals, whereas rib 5 consistently moved in the caudal direction. For the seven animals, the displacement of rib 10 and
rib 5 at peak inspiration was +1.26 ± 0.11 and -0.80 ± 0.16 mm, respectively.

When rib 10 was held stationary during inspiration, so that its cranial displacement was reduced to +0.03 ± 0.02 mm (P < 0.001), the inspiratory EMG activity recorded from the second left parasternal intercostal remained unchanged at 100.5 ± 0.5% of the control value (NS), but the inspiratory caudal displacement of rib 5 showed a small but consistent increase (control: −0.80 ± 0.16 mm; stationary: −0.94 ± 0.15 mm; P < 0.01). ΔPab also showed a small increase in every animal (control: +2.04 ± 0.23 cmH2O; stationary: +2.10 ± 0.23 cmH2O; P < 0.05). However, tidal volume was unaltered (control: 225.6 ± 15.5 ml; stationary: 225.3 ± 15.7 ml; NS).

**Effects of maintaining the lower ribs stationary during occluded breaths.** Holding rib 10 stationary during occluded breaths had essentially similar effects, as shown by the records of a representative animal in Fig. 2. Thus, while the cranial displacement of rib 10 observed at the peak of occluded inspiratory efforts in the control condition was abolished (control: +1.44 ± 0.19 mm; stationary: −0.09 ± 0.06 mm; P < 0.001), the inspiratory EMG activity in the second left parasternal intercostal was unchanged at 100.2 ± 1.3% of the control value. However, the caudal displacement of rib 5 was consistently increased (control: −4.12 ± 0.45 mm; stationary: −4.42 ± 0.47 mm; P < 0.001), and ΔPab was increased in six of seven animals (control: +1.85 ± 0.24 cmH2O; stationary: +1.97 ± 0.22 cmH2O; P < 0.05). ΔPao at peak inspiration, however, was unaltered (control: −14.0 ± 1.5 cmH2O; stationary: −13.9 ± 1.4 cmH2O; NS). As is shown in Fig. 3, therefore, the relationship between the displacement of rib 5 and Pao was consistently displaced downward, so that, for any given value of Pao, the rib was in a more caudal position when rib 10 was held stationary than when it was free to move.

**Effects of isolated cranial displacement of the lower ribs (Expt 2).** When the 9th and 10th rib pairs were pulled manually in the cranial direction, rib 5 was displaced cranially and Pao fell in every animal, as shown in Fig. 4A. The relationship between the displacement of rib 5 and that of rib 10 was slightly curvilinear, such that for a given displacement of rib 10, the displacement of rib 5 increased progressively in mag-
nitude as force increased and rib 10 was further away from its relaxation position (Fig. 5). However, the displacement of rib 5 was small compared with that of rib 10, and ΔPao was also small. Thus, when rib 10 was displaced 2 mm for example, rib 5 in the five animals was displaced only 0.29 ± 0.05 mm, and ΔPao was only -0.21 ± 0.05 cmH2O.

When the procedure was repeated after pneumothorax, so that ΔPao remained zero, the cranial displacement of rib 5 induced by any given displacement of rib 10 was substantially greater (P < 0.05) than it was with the rib cage intact (Figs. 4B and 5). For a 2-mm displacement of rib 10 after pneumothorax, rib 5 moved by 0.50 ± 0.09 mm.

**DISCUSSION**

There are two important new observations in this study. The first is that holding the lower ribs stationary and preventing them from moving cranially during spontaneous, isolated contraction of the diaphragm has no measurable effect on Ppl. The second observation is that applying external, cranially oriented forces selectively to the lower ribs so as to simulate the action of the diaphragm on these ribs causes a cranial displacement of the upper ribs and a fall in Ppl. These observations taken together have significant implications for the action of the diaphragm on the lung and the rib cage during breathing. They also have implications to the mechanism of lung expansion during phrenic nerve stimulation.

**Action of the diaphragm on the lung.** Mead and Loring (16), in their theoretical analysis of the volume contribution of the diaphragm during breathing, pointed out that the cranial displacement of the lower ribs caused by the diaphragm during contraction should induce a cranial displacement of the muscle as a whole and that this motion should oppose lung expansion. If this were the case, then it would be expected that holding the lower ribs stationary during spontaneous, isolated diaphragm contraction would cause an increase in the descent of the dome and, with it, an increase in ΔPpl. Although diaphragmatic displacement was not measured in this study, the finding that ΔPab was slightly greater when the lower ribs were held stationary (Expt 1) is consistent with the idea that the procedure was associated with an increase in the descent of the dome. However, although neural inspiratory drive was unchanged, there was no concomitant increase in tidal volume during unimpeded inspiration and no increase in ΔPao (or ΔPpl) during occluded breaths (Figs. 1 and 2). This indicates that the pressure (volume) contribution of the cranial displacement of the lower ribs produced by the diaphragm during isolated contraction is small, insufficient to be separated from the effect of the descent of the dome.
To assess the isolated effect of the lower rib displacement, therefore, the lower rib pairs were displaced manually in the cranial direction while respiratory muscle activity was abolished and the airway was occluded (Expt 2). In agreement with previous studies of the coupling between the ribs and the lung in dogs (5, 9), such displacement produced a fall in Pao together with a cranial displacement of the upper ribs (Figs. 4A and 5). Thus the force applied to the lower ribs, acting through the interdependence of the ribs, was transmitted to the upper ribs and thereby caused a fall in Ppl that outweighed the potential expiratory effect of the cranial displacement of the diaphragm. However, when the lower ribs were pulled the same amount as during isolated diaphragm contraction (i.e., 1.2–1.4 mm), ΔPao was only −0.14 cmH2O. To the extent that ΔPpl during tidal breathing in the dog is ~4 cmH2O, it follows that the ΔPpl caused by the lower rib displacement alone was ~30 times smaller than that produced during unimpeded inspiration. The ΔPao produced by the isolated displacement of the lower ribs was also 100 times smaller than that recorded during occluded breaths (~14.0 cmH2O). On this basis, the observation that holding the lower ribs stationary did not induce any measurable change in tidal volume during unimpeded breathing and in ΔPao during occluded breaths can be understood.

**Action of the diaphragm on the rib cage.** Previous studies in rabbits and dogs have shown that the inward and caudal displacement of the upper ribs during isolated phrenic nerve stimulation is abolished when stimulation is performed in the presence of pneumothorax (1, 6). The conventional view, therefore, maintains that the action of the diaphragm on the upper ribs is exclusively related to the fall in Ppl. Contrary to this view, when the lower ribs were maintained stationary in Expt 1, the caudal displacement of rib 5 during unimpeded inspiration was consistently increased while tidal volume (and presumably ΔPpl) was constant. The caudal displacement of rib 5 associated with any given ΔPao was also slightly greater during occluded breaths in every animal (Fig. 3), thus suggesting that the displacement of the upper ribs was also affected by the displacement of the lower ribs. And indeed, after a pneumothorax was performed in Expt 2 so as to uncouple the rib cage from the lung, rib 5 moved in the cranial direction when the lower rib pairs were pulled cranially (Fig. 5). Pulling the lower rib pairs still produced a cranial displacement of rib 5 when the rib cage was intact, even though the maneuver in this condition also induced a fall in Ppl.

The agreement between the upper rib displacements measured in the two experiments of the study is not only qualitative but also quantitative. When the lower ribs were held stationary in Expt 1, the normal cranial displacement of rib 10 at the peak of occluded breaths was decreased, on average, by 1.53 mm (from +1.44 to −0.09 mm), and although ΔPao was unchanged, the caudal displacement of rib 5 increased by 0.30 mm (from −4.12 to −4.42 mm). The ratio of the difference in rib 5 displacements to the difference in rib 10 displacements, therefore, was 0.20. Similarly, in Expt 2, a 2-mm cranial displacement of rib 10 in the presence of pneumothorax (so that Ppl was also constant) led to a 0.50-mm displacement of rib 5, i.e., the ratio of rib displacements was 0.25. Based on this qualitative and quantitative agreement, one may therefore conclude that contrary to the conventional view, the action of the canine diaphragm on the upper rib cage is determined not only by ΔPpl but also by the displacement of the lower ribs.

**Mechanism(s) of lung expansion during phrenic nerve stimulation.** Stimulation of the phrenic nerves with tetanic (20 Hz), supramaximal stimuli in dogs produces marked shortening of the muscle fibers of the diaphragm and a large decrease in the zone of apposition, so that the lower ribs become exposed to the expiratory effect of Ppl (3, 7). As a result, whereas the ribs move cranially during spontaneous, isolated diaphragm contraction, they move caudally during such stimulation of the phrenic nerves (3, 7). Based on the idea that the changes in length of the diaphragm muscle fibers during isolated contraction are essentially determined by the relative displacement of the central tendon and the muscle insertions into the lower ribs, De Troyer et al. (7) recently measured diaphragm length and displacement during phrenic nerve stimulation, and they concluded that the caudal displacement of the lower ribs in this condition accounts for nearly a quarter of the descent of the dome. The investigators further speculated that this lower rib displacement contributed ~25% of the total ΔPpl developed by the diaphragm.

In view of the current observation that the displacement of the lower ribs during spontaneous, isolated diaphragm contraction has no detectable effect on pleural pressure, it appears that this prediction is incorrect. It is worth pointing out, however, that the magnitude of the lower rib displacement during stimulation of the phrenic nerves is substantially larger than that during spontaneous diaphragm contraction. Specifically, whereas the lower ribs move ~1.3 mm cranially during spontaneous contraction, they move ~10 mm caudally during phrenic nerve stimulation (3, 7). Therefore, the potential impact of the lower rib displacement on the descent of the dome during phrenic nerve stim-
prediction should be ~7-fold greater than that during spontaneous muscle contraction. In addition, and more important, the prediction made in the previous study overlooked the interdependence between the lower ribs and the upper ribs. It is most likely, in fact, that the caudal displacement of the lower ribs during phrenic nerve stimulation added to the effect of ΔPpl and enhanced the caudal displacement of the upper ribs. This additional caudal displacement should, in turn, cause a rise in Ppl and, thus, reduce or abolish the effect of the lower rib displacement on the descent of the dome.

In conclusion, the present study has demonstrated that in the dog, the cranially oriented force applied on the lower ribs by the diaphragm during isolated, spontaneous contraction is transmitted to the upper ribs. Therefore, although the cranial displacement of the lower ribs may also induce a cranial displacement of the muscle, as suggested by Mead and Loring (16), this force has an inspiratory effect on the lung. Because the lower rib displacement produced by the diaphragm is small, however, this inspiratory effect is very small and has no significant impact on lung expansion during breathing.

These observations cannot be extended to humans without considerable caution. However, there is no reason to believe that the interdependence between the lower ribs and the upper ribs would be weaker in humans than in the dog. To the extent that, in contrast to the dog, the costal cartilages of ribs 6–10 in humans connect directly to each other, it is likely that this interdependence is, in fact, stronger in humans. Therefore, it would be reasonable to speculate that the force applied by the diaphragm on the lower ribs in humans is also transmitted in part to the upper ribs. Thus, in agreement with the earlier hypothesis of Goldman and Mead (12), the diaphragm in humans would have an inspiratory action on the entire rib cage, even though its action on the upper rib cage is outweighed by the expiratory effect of Ppl. The coupling between the ribs and the lung in humans is unknown, however, so it is difficult to predict the effect on the lung of the lower rib displacement produced by the diaphragm.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

A.D.T. conception and design of research; A.D.T. performed experiments; A.D.T. analyzed data; A.D.T. interpreted results of experiments; A.D.T. prepared figures; A.D.T. drafted manuscript; A.D.T. edited and revised manuscript; A.D.T. approved final version of manuscript.

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