Interaction between left and right intercostal muscles in airway pressure generation

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Cappello, Matteo, and André De Troyer. Interaction between left and right intercostal muscles in airway pressure generation. J. Appl. Physiol. 88: 817–820, 2000.—The interactions between the different rib cage inspiratory muscles in the generation of pleural pressure remain largely unknown. In the present study, we have assessed in dogs the interactions between the parasternal intercostals and the interosseous intercostals situated on the right and left sides of the sternum. For each set of muscles, the changes in airway opening pressure (ΔPao) obtained during separate right and left activation were added, and the calculated values (predicted ΔPao) were then compared with the measured ΔPao values obtained during symmetric, bilateral activation (measured ΔPao). When the parasternal intercostals in one or two interspaces were activated, the measured ΔPao was nearly equal to the predicted value. The difference, however, was only 10%. When the interosseous intercostals were activated, the measured ΔPao was equal to or nearly equal to the predicted value. These observations strengthen our previous conclusion that the pressure changes produced by the rib cage inspiratory muscles are essentially additive. As a corollary, the rib cage can be considered as a linear elastic structure over a wide range of distortion.

mechanics of breathing; respiratory muscles

A NUMBER OF ELECTROMYOGRAPHIC (EMG) studies in humans and animals have clearly established that expansion of the lung during breathing involves several groups of rib cage muscles, in particular the parasternal and external intercostals and the scalenes (2–4, 9, 13, 14), but the question as to how the changes in pressure produced by these muscles add to each other remains unanswered. Otherwise stated, how does the pressure produced by a particular rib cage muscle during breathing, when the muscle acts in coordination with other muscles, compare with the pressure produced by this muscle during isolated contraction? If the cage behaved as a linear elastic structure, the total effect of different muscle forces applied simultaneously would simply be the sum of the effects of the individual muscle forces. However, a muscle shortens more when activated in isolation than it does during coordinated activation. As a result, the force exerted by this muscle would be smaller. Furthermore, activation of an individual muscle might cause sufficient distortion of the rib cage so as to alter significantly the geometry and/or the compliance of the structure. Therefore, as Di Marco et al. (7) and Loring and Butler (11) have suggested, the total pressure generated during coordinated muscle contraction might be greater than the sum of the pressures resulting from isolated muscle contractions.

Our laboratory (10) has previously assessed in dogs the interactions between the parasternal intercostals in different interspaces, between the interosseous intercostals in different interspaces, and between the parasternal intercostals and the neck muscles. When the parasternal intercostals or the interosseous intercostals in two interspaces were stimulated selectively and simultaneously on both sides of the sternum, the change in airway opening pressure (ΔPao) was, within 10%, equal to the sum of the ΔPao values produced by bilateral stimulation of the muscles in each individual interspace. The ΔPao produced by the simultaneous, bilateral contraction of the parasternal intercostals in one interspace and either the scalenes or the sternomastoids was also found to be nearly equal to the sum of the ΔPao values produced by the two sets of muscles individually. On the basis of these observations, it was, therefore, concluded that the pressures generated by the rib cage inspiratory muscles are essentially additive (10).

In the present studies, in an attempt to evaluate the range of rib cage distortions for which this conclusion remains valid, we have examined the interactions between the parasternal and interosseous intercostals situated on the left and right sides of the sternum. Indeed, although bilateral contraction of these muscles in only one or two interspaces engenders distortion, it produces symmetrical cranial displacement of the ribs and symmetrical expansion of the cage. On the other hand, contraction of these muscles on one side of the sternum causes cranial and outward displacement of the ipsilateral ribs, but the fall in pleural pressure being transmitted through the mediastinum displaces the contralateral ribs caudally and inward. This asymmetry between the left and right sides of the chest may be further aggravated by the torque exerted by the muscles on the sternum. In these conditions, the chest wall might depart from its linear range, such that the principle of pressure superposition would no longer apply.

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MATERIALS AND METHODS

The experiments were performed on 16 adult mongrel dogs (15–34 kg) anesthetized with pentobarbital sodium (initial dose: 30 mg/kg iv). The animals were placed in the supine posture and intubated with a cuffed endotracheal tube, after which the rib cage was exposed on both sides of the chest from the first through the tenth rib by deflection of the skin and underlying muscle layers. The animal was then connected to a mechanical ventilator (Harvard pump, Chicago, IL); the level of anesthesia was maintained so that the corneal reflex was abolished throughout. Two experimental protocols were followed.

Experiment 1. The interaction between the left and right parasternal intercostals was assessed in nine animals. In each animal, the parasternal intercostals in two or three interspaces between the third and the seventh were thus prepared for electrical stimulation on both sides of the sternum, as previously described (5, 6). The ventral portion of the external intercostal muscle was severed in each interspace, and the caudal border of the rostral rib was cleared of peristeum over the 3–4 cm lateral to the costochondral junction. A curved chisel-edged instrument was then passed under the rib to separate the peristeum from the bone, and the peristeum was incised so as to expose the internal intercostal nerve with little or no injury. A pair of stainless steel hook electrodes spaced 3–4 mm apart was then implanted into the corresponding parasternal intercostal muscle to record compound muscle action potentials (CMAPs) and determine the voltage for supramaximal nerve stimulation; the EMG signal thus obtained was amplified (model 830/1, CWE, Ardmore, PA) and band-pass filtered below 5 and above 2,000 Hz. The freed sector of the nerve was then laid over the corresponding segment of the sternum. The technique used to stimulate the muscles was that described by Ninane et al. (12). After rib cage exposure, pairs of copper threads 0.5 mm in diameter were thus inserted bilaterally between the external and internal intercostal muscles in two contiguous interspaces between the second and the seventh. In each interspace, the threads were introduced near the chondrosternal junction, and they were driven dorsally, parallel to each other, along the cranial and caudal boundaries until their tip lay in the vicinity of the rib angle. The ventral end of the threads, was then bent forward and connected to the stimulator, after which the animal was given an intravenous injection of 2 mg pancuronium. In so doing, we could induce clear-cut unilateral or bilateral contraction of the external and internal intercostal muscles in a single interspace or in two adjacent interspaces (10, 12).

With the animal apneic and the endotracheal tube occluded at FRC, square pulses of 1-ms duration and 60 V were delivered at a frequency of 50 impulses/s to the intercostal muscles in one interspace on the left side of the sternum. The muscles in the same interspace on the right side were subsequently stimulated, after which the muscles on both sides were stimulated simultaneously. The procedure was repeated for the adjacent (rostral or caudal) interspace, after which the muscles in the two interspaces were stimulated together, first on the left side, then on the right side, and finally on both sides simultaneously. For the 7 animals, a total of 27 single interspaces and 19 pairs of interspaces were studied.

Data analysis. The \( \Delta P_{\text{a}o} \) values obtained during nerve (experiment 1) or muscle (experiment 2) stimulation in any given condition were averaged over the three stimulations performed in this condition. For each interspace or each pair of interspaces, the \( \Delta P_{\text{a}o} \) obtained during stimulation of one side was then added to the \( \Delta P_{\text{a}o} \) obtained during stimulation of the other side, and the value thus calculated (this value will

\[ \Delta P_{\text{a}o} = P_{\text{a}o_{\text{right}}} - P_{\text{a}o_{\text{left}}} \]

Fig. 1. Changes in airway opening pressure (\( \Delta P_{\text{a}o} \)) during tetanic stimulation of intercostal nerve in third interspace on left side alone (A), on right side alone (B), and on both sides simultaneously (C) in a representative animal. Electromyographic (EMG) activities recorded from parasternal intercostal muscles (Parast) are also shown. Dashed line in C corresponds to \( \Delta P_{\text{a}o} \) that would be obtained during simultaneous contraction of left and right sides if their effects were perfectly additive.
be referred to here as the predicted $\Delta P_{ao}$ was compared with that measured during stimulation of the two sides simultaneously. For each animal, all values of predicted and measured $\Delta P_{ao}$ were finally averaged, and statistical comparison between these average values was made by using $t$-tests for paired observations. The criterion for statistical significance was taken as $P < 0.05$.

**RESULTS**

Interaction between left and right parasternal intercostals. A representative example of the traces obtained during stimulation of the parasternal intercostal in one interspace first on each side of the sternum separately and then on the two sides simultaneously is shown in Fig. 1. When the parasternal intercostal on the left side was stimulated alone (A), the fall in $P_{ao}$ was 2.25 cmH$_2$O. When the muscle on the right side was subsequently stimulated (B), the fall in $P_{ao}$ was 2.04 cmH$_2$O. Therefore, the predicted $\Delta P_{ao}$ for this muscle was 4.29 cmH$_2$O (C). The $\Delta P_{ao}$ measured during combined stimulation of the left and right sides was, in fact, −5.00 cmH$_2$O.

In agreement with previous observations from our laboratory (6, 10), the $\Delta P_{ao}$ values measured during parasternal stimulation decreased from the third interspace caudally in any particular animal. Therefore, depending on the location and the number of interspaces studied, the predicted $\Delta P_{ao}$ ranged from −6.08 to −0.91 cmH$_2$O. However, 24 of the 32 trials produced results similar to those shown in Fig. 1 and yielded a measured $\Delta P_{ao}$ greater than the predicted value, as shown in Fig. 2. Consequently, the predicted $\Delta P_{ao}$ for the nine animals averaged $-2.89 \pm 0.30$ (SE) cmH$_2$O and the measured $\Delta P_{ao}$ amounted to $-3.17 \pm 0.34$ cmH$_2$O ($P < 0.02$).

Interaction between left and right interosseous intercostal muscles. Figure 3 shows the records of the $\Delta P_{ao}$ values produced by external and internal intercostal stimulation in two adjacent interspaces on the left and right sides of the sternum separately and then simultaneously in a representative animal. In agreement with the previous observations of Ninane et al. (12) and Legrand et al. (10), stimulating the left or right interosseous intercostals in one interspace between the second and the sixth resulted in a fall in $P_{ao}$, whereas stimulating the muscles in the seventh interspace caused little or no change in $P_{ao}$. Depending on the interspace(s), the predicted $\Delta P_{ao}$ values ranged, therefore, between −6.12 and 0 cmH$_2$O.

The $\Delta P_{ao}$ values measured in the 46 trials are compared with the predicted values in Fig. 4. The measured $\Delta P_{ao}$ was greater than the predicted value in 11 trials. However, the measured $\Delta P_{ao}$ was similar to the predicted value in 6 trials, and in 29 trials, the measured $\Delta P_{ao}$ was smaller. As a result, the measured $\Delta P_{ao}$ for the animal group ($-2.40 \pm 0.34$ cmH$_2$O) was lower than the predicted value.
not statistically significantly different from the predicted $\Delta P_{ao} (-2.59 \pm 0.34 \text{ cmH}_2\text{O})$.

**DISCUSSION**

If rib cage distortion were a major determinant of the interactions between the inspiratory intercostal muscles, one would expect that the $\Delta P_{ao}$ generated by a bilateral, symmetrical contraction of the parasternal or interosseous intercostals would be greater than the sum of the $\Delta P_{ao}$ values generated by unilateral left and right muscle contraction. One would further expect that the difference would proportionately increase as the pressure drops produced by unilateral contraction are greater and cause more severe distortion.

In agreement with this prediction, stimulating the parasternal intercostals in one or two interspaces on both sides of the sternum commonly yielded a $\Delta P_{ao}$ that was greater than the sum of the individual left and right $\Delta P_{ao}$ values (Figs. 1 and 2). However, the difference between the measured and predicted $\Delta P_{ao}$ values was only 10%, and it did not increase as the predicted values were greater (Fig. 2). The difference between the measured and predicted $\Delta P_{ao}$ values was even smaller in the case of the interosseous intercostals. In fact, when these muscles contracted on the two sides of the sternum simultaneously, the $\Delta P_{ao}$ was nearly equal to the sum of the $\Delta P_{ao}$ values obtained during unilateral contraction (Figs. 3 and 4). Therefore, even though the right and left parasternal intercostals have a small synergistic action on the lung, these results overall amplify the previous conclusion from our laboratory (10) that the pressure changes due to the rib cage inspiratory muscles are essentially additive. As a corollary, the rib cage can be modeled as a linear elastic system over quite a wide range of distortion, and measurements of the pressures produced by rib cage muscles activated individually can be used to estimate the contribution of these muscles to lung expansion during breathing.

The reason that the $\Delta P_{ao}$ obtained during bilateral contraction is slightly greater than the sum of the unilateral values in the case of the parasternal intercostals but not in the case of the interosseous intercostals is uncertain. However, there is a marked difference between the torque exerted by these muscles on the sternum. The fibers of the parasternal intercostal in a given interspace originate from the lateral aspect of the sternum and the caudal aspect of the costal cartilage of the rib above, and, from these origins, they run caudally and laterally to insert into the cranial aspect of the costal cartilage below. Consequently, when these fibers contract on say the right side of the sternum, the axial components of the force vectors induce a cranial displacement of the ribs below and a caudal displacement of the sternum (3), but, in addition, the lateral components of the force vectors operate to displace the rostral portion of the sternum to the right and the caudal portion of the sternum to the left. On the other hand, the technique used in this study to stimulate the interosseous intercostals affected both the external and internal muscle layers. Because these muscle fibers run approximately perpendicular to each other, the lateral components of the force vectors should cancel each other, and hence contraction of the muscles on one side of the sternum should produce little, if any, sternum rotation. The sternum rotation produced by unilateral contraction of the parasternal intercostals may displace some elements of the rib cage (e.g., the costal cartilages) outside their linear range and cause them to exert large elastic forces. Because the effect of these forces on intrathoracic pressure was small (Fig. 2), no attempt was made, however, to confirm or disprove this mechanism.

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