Rostrocaudal gradient of mechanical advantage in the parasternal intercostal muscles of the dog

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1. Previous theoretical studies have led to the predictions that, in the dog, the parasternal intercostal muscles in the rostral interspaces shorten more during passive inflation than those in the caudal interspaces and have, therefore, a greater inspiratory mechanical advantage. The present studies were undertaken to test these predictions.

2. The effects of passive inflation on the length of the parasternal intercostals in interspaces 1 to 7 were evaluated with markers implanted in the costal cartilages. Although the muscles in all interspaces shortened with passive inflation, the fractional shortening increased from the first to the second and third interspaces and then decreased continuously to the seventh interspace.

3. To understand this peculiar distribution, a geometric model of the parasternal area was then developed and a relation was obtained between muscle shortening and the angles that describe the orientation of the muscle and costal cartilage relative to the sternum. Measurement of these angles indicated that the rostrocaudal gradient of parasternal shortening resulted from the different orientations of the costal cartilages and their different rotations during passive inflation.

4. The changes in airway pressure generated by the parasternal intercostals in interspaces 3, 5 and 7 were finally measured during selective, maximal stimulation. The fall in pressure was invariably greatest during contraction of the third interspace and smallest during contraction of the seventh.

5. These observations indicate that, in the dog, the rostrocaudal gradient in rib rotation induces a rostrocaudal gradient of mechanical advantage in the parasternal intercostals, which has its climax in the second and third interspaces. These observations also support the concept that the respiratory effect of a given respiratory muscle can be computed from its behaviour during passive inflation.

Although the actions of most respiratory muscles on the chest wall have been qualitatively described, the question of how much lung expansion (or deflation) each muscle can produce has not been answered. This is a difficult question because a number of muscles cannot be maximally activated in isolation, but recent theoretical studies by Wilson & De Troyer (1992, 1993) have proposed an indirect approach. Thus, using a standard theorem of mechanics, the Maxwell reciprocity theorem, these investigators have postulated that the potential change in airway pressure (ΔP_{ao}) produced by a muscle contracting alone against a closed airway is related to the mass (m) of the muscle, the maximal active muscle tension per unit cross-sectional area (σ), and the fractional change in muscle length per unit volume increase of the relaxed chest wall $[\Delta L/(L\Delta V_L)]_{Rel}$, such that:

$$\Delta P_{ao} = m\sigma[\Delta L/(L\Delta V_L)]_{Rel}. \quad (1)$$

Based on this conclusion, Wilson & De Troyer (1993) computed the fractional changes in length of the canine intercostal muscles during passive inflation. These computations suggested that, compared with the other intercostal muscles, the internal intercostals of the parasternal area (the so-called parasternal intercostals) have a large inspiratory mechanical advantage; that is, they cause a large fall in P_{ao} per unit muscle mass and unit active stress ($\Delta P_{ao}/m\sigma$). Indeed, since the electromyographic

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studies performed by Taylor (1960) in normal humans, these muscles are known to be electrically active during inspiration (De Troyer & Kelly, 1982; Whitelaw & Feroah, 1989; De Troyer & Farkas, 1994) and to play an important role in causing the inspiratory elevation of the ribs (De Troyer, Farkas & Ninane, 1988; Di Marco, Romaniuk & Supinski, 1990; De Troyer, 1991; De Troyer & Farkas, 1993, 1994). These computations, however, also led to two predictions concerning the mechanics of these muscles. First, they predicted that the mechanical advantage of the parasternal intercostal in a given interspace may not be uniform. Second, they predicted that the inspiratory mechanical advantage of the parasternal intercostal in the third interspace is larger than in the sixth, thus suggesting that the advantage of these muscles is distributed along a rostrocaudal gradient.

We have recently confirmed, in agreement with the first prediction, that the inspiratory mechanical advantage of the parasternal intercostal in a given interspace decreases markedly from the sternum toward the chondrocostal junction (De Troyer & Legrand, 1995; Legrand, Wilson & De Troyer, 1996). In the present studies, we have tested the second prediction. We have thus measured, in a group of anaesthetized, paralysed dogs, the changes in length of the parasternal intercostals in the different interspaces during passive chest wall inflation. Although the muscle in the third interspace shortened more than in the sixth interspace, the rostrocaudal gradient of muscle shortening was not monotonic. To understand this result, we have subsequently developed a geometric model of the parasternal area and have obtained a relation between muscle shortening and the angles describing the orientation of the muscle and costal cartilage. We have then measured these angles to identify the factors that are responsible for the observed gradient of muscle shortening. Finally, we have tested the validity of eqn (1) directly by evaluating the inspiratory effect of the parasternals in the different interspaces.

METHODS

Experiment 1

We first studied eight adult mongrel dogs (14–22 kg body weight) to assess the passive changes in length of the parasternal intercostals in all interspaces from the first to the seventh. The apparatus and experimental protocol were similar to those described in the previous study (De Troyer & Legrand, 1995), but the measurements in this study were limited to the sternal bundles, which have a greater mechanical advantage and play a more important role in the act of breathing (De Troyer & Legrand, 1995; Legrand *et al.* 1996). The animals were deeply anaesthetized with pentobarbitone sodium (initial dose, 30 mg kg^{-1} I.V.), placed in the supine posture and intubated with a cuffed endotracheal tube, after which the parasternal region of the ribcage was exposed on the right side of the chest from the first to the ninth interspace. The insertions of the sternal bundles in the different interspaces were carefully defined, and pairs of small screws were inserted into the costal cartilages at the points of insertion of the bundles thus selected. The animals were then paralysed with an intravenous injection of 2 mg pancuronium and ventilated mechanically. The animals studied in Experiments 1 and 2 received an additional dose of pentobarbitone sodium (6–8 mg kg^{-1} I.V.) every 30 min throughout the surgery and the measurements. This anaesthetic regime produces deep anaesthesia in dogs, and the pupils remained constricted and unresponsive to light throughout.

The ventilation was stopped and the chest wall was allowed to relax to equilibrium, and the linear distance between the screws of each pair (i.e. the length of each muscle bundle at functional residual capacity (FRC)) was measured with callipers. The tracheal tube was then connected to a calibrated super-syringe, lung volume was increased by 1 l, and the measurements were repeated. All measurements were made in triplicate. In each individual animal, the measurements obtained in a given interspace were averaged, and the changes in muscle length caused by passive inflation were expressed as percentage changes relative to the muscle length at FRC (L_{FRC}). The animals were then given an overdose (50 mg kg^{-1}) of anaesthetic.

Data were finally averaged for the animal group, and they are presented as means ± S.E.M. Comparisons between the different interspaces were made by analysis of variance (ANOVA) with repeated measures, and multiple comparison testing of the mean values was performed using Student–Newman–Keuls tests. The criterion for statistical significance was taken as $P < 0.05$.

Model

Figure 1 shows a simple model of the geometry of the parasternal region. Muscle length L is related to the distance d along the cartilage between the sternum and the muscle attachment, the distance s along the sternum between the attachments of the muscle and cartilage, and the angle α between the sternum and the costal cartilage by the law of cosines:

$$L^2 = d^2 + s^2 + 2ds\cos\alpha. \quad (2)$$

The values d and s are constant with changes in lung volume. Therefore, if the values of L and α before and after a 1 l passive inflation are denoted by subscripts 1 and 2, respectively,

$$L_2^2 - L_1^2 = 2ds(\cos\alpha_2 - \cos\alpha_1), \quad (3)$$

and the fractional change in muscle length, $\Delta L/L_1 = L_2/L_1 - 1$, is given by the following equation:

$$\Delta L/L_1 = [1 + (2ds/L_1^2)(\cos\alpha_2 - \cos\alpha_1)]^{1/2} - 1. \quad (4)$$

If the angle between the sternum and the muscle fibres is denoted β, simple trigonometric relations yield the results $d/L_1 = \sin\beta/\sin\alpha_1$ and $s/L_1 = \sin(\alpha_1 - \beta)/\sin\alpha_1$. With these substitutions, eqn (4) becomes:

$$\Delta L/L_1 = \{1 + [2\sin\beta\sin(\alpha_1 - \beta)/\sin^2\alpha_1](\cos\alpha_2 - \cos\alpha_1)\}^{1/2} - 1. \quad (5)$$

(This analysis of the geometry of the parasternal region is similar to that given in our previous communication (De Troyer & Legrand, 1995). However, in the previous communication, approximations were made that are valid for small changes in α. Here both the initial angle between the costal cartilage and the sternum α_1 and the final angle α_2 are shown explicitely, and eqn (5) is valid for any initial and final angles. If $\Delta\alpha = \alpha_2 - \alpha_1$ is small, eqn (5) reduces to the equation given in the earlier paper.)

Experiment 2

Six adult mongrel dogs (15–25 kg body weight) were subsequently studied to identify the factors responsible for the observed rostrocaudal gradient of muscle shortening. As in Expt 1 the animals were anaesthetized, placed in the supine posture and

intubated with a cuffed endotracheal tube, and the ribcage and intercostal muscles in the parasternal area were exposed from the first to the ninth rib, after which the animals were paralysed. The lower edge of a protractor was then aligned with the sternum, and the acute angles between the sternum and the direction of the fibres of the sternal parasternal bundles (β) in interspaces 1, 3, 5 and 7 were measured at FRC. The acute angles between the sternum and the costal cartilages of ribs 2, 4, 6 and 8 at FRC (angle α_1) were measured as well. Lung volume was then increased by 1 l, and the angles between the sternum and the costal cartilages (angle α_2) were measured again. All these measurements were also obtained in triplicate, and their statistical analysis was made with the technique used in Expt 1.

Experiment 3

Finally, six dogs (16–25 kg body weight) were studied to assess the pressure-generating ability of the parasternal intercostals in different interspaces. The animals were prepared as in Expt 1 (except that no muscle relaxant was used), after which the internal intercostal nerves in interspaces 3, 5 and 7 were exposed on both sides of the sternum. Nerve exposure in each interspace was made 1–2 cm lateral to the chondrocostal junction by using the procedure described in our previous communication (De Troyer & Legrand, 1995). A pair of stainless-steel hook electrodes spaced 3–4 mm apart was then implanted in the sternal bundles of each parasternal to record compound muscle action potentials and determine the voltage for supramaximal nerve stimulation.

After completion of this procedure, a Validyne differential pressure transducer was connected to a side-port of the endotracheal tube to measure airway pressure. Each nerve was then sectioned ~2 cm dorsal to the site of stimulation, the animal was made apnoeic by mechanical hyperventilation, and the distal end of the nerve was stimulated bilaterally by applying square pulses of 0·2 ms duration and supramaximal voltage at a frequency of 50 impulses s^{-1}. In so doing, we could ensure simultaneous, maximal contraction of all parasternal muscle fibres in each interspace studied while avoiding antidromic stimulation of the internal interosseous intercostal muscle in the same interspace. Sectioning the nerves also avoided stimulation of the spindle afferent fibres, which are known to have extrasegmental projections (Eccles, Sears & Shealy, 1962) and could have produced contraction of intercostal muscles in adjacent interspaces. The nerve in each interspace was stimulated at least three times, each stimulation being performed while the endotracheal tube was occluded.

The exposed internal intercostal nerves were then covered with tissue and warm mineral oil, and the muscle fibres of the sternal half of the parasternals being studied were sectioned along their caudal insertions; the distal part of the internal intercostal nerves, including the branches providing the motor supply to the triangularis sterni, was thus left intact. Bilateral, supramaximal stimulation of the nerves in the third, fifth and seventh interspaces was then repeated. As in the control condition, each nerve was stimulated three times, and the difference between the fall in airway pressure obtained during stimulation of the intact parasternal and that measured after section of the sternal half was considered to be due to the action of the sternal half. When these measurements were completed, the sectioned sternal parts of the parasternal intercostals were excised, their mass was measured, and the change in airway pressure (ΔP_{ao}) generated by each interspace was divided by muscle mass to yield specific ΔP_{ao}.

Comparison between the ΔP_{ao} and specific ΔP_{ao} produced by the different interspaces studied was also made by analysis of variance (ANOVA) with repeated measures coupled with Student–Newman–Keuls tests.

RESULTS

Changes in muscle length (Expt 1)

The effects of passive inflation on the length of the sternal parasternal bundles are shown in Fig. 2 for the eight animals studied. With passive inflation, the muscles shortened in all interspaces. However, the different interspaces behaved differently in that the fractional shortening increased from the first to the second interspace in all animals and then decreased continuously and progressively from the third to the seventh interspace. For the animal group, the shortening of the muscle in the third interspace (mean ± S.E.M.) was 10·0 ± 0·6% of the muscle length at FRC, whereas it was only 5·2 ± 0·5% in the first interspace ($P < 0·001$), 7·2 ± 0·4% in the fifth interspace ($P < 0·001$) and 3·7 ± 0·3% in the seventh interspace ($P < 0·001$).

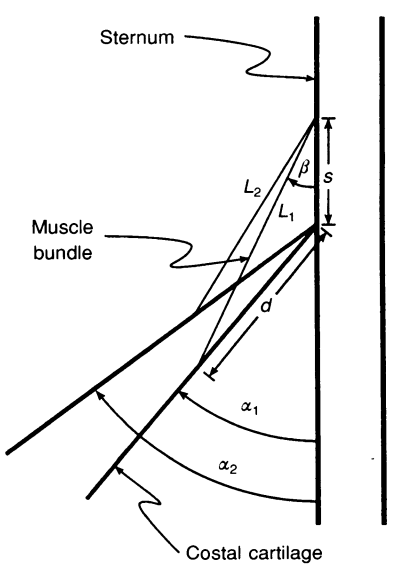

Figure 1. Model of the geometry of the parasternal region of the ribcage

Muscle length is denoted L, and the angles between the sternum and the costal cartilage and between the sternum and the muscle fibres are denoted α and β, respectively. Subscripts 1 and 2 denote length and angles before and after, respectively, a 1 l passive inflation. The distance along the sternum between the attachments of the muscle fibres and cartilage, denoted s, and the distance along the cartilage between the sternum and the muscle attachment, denoted d, remain constant during cartilage rotation.

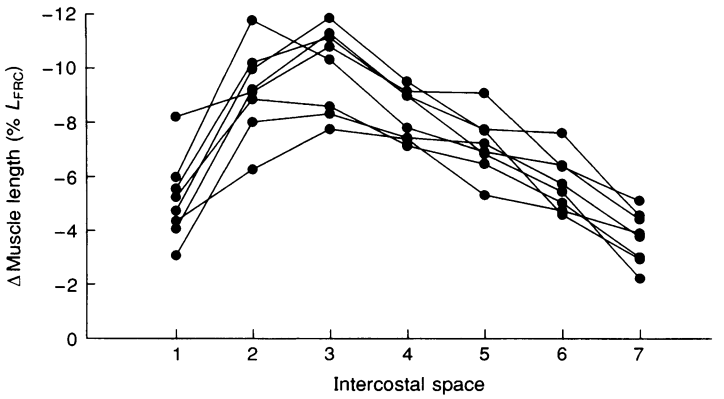

Figure 2. Changes in length of the parasternal intercostals in the different interspaces during passive inflation

Individual data obtained from eight animals. The changes in length produced by passive inflation (1 l) are expressed as percentage changes relative to the muscle length at FRC (L_{FRC}); negative changes in length represent muscle shortening.

Fibre and cartilage orientation (Expt 2)

Figure 3 shows the angles between the sternum and the parasternal fibres (β) in interspaces 1, 3, 5 and 7 and the angles between the sternum and the corresponding costal cartilages at FRC (α_1). Although the acute angle between the sternum and the muscle fibres in the seventh interspace was smaller than in the first and third interspaces ($P < 0{\cdot}05$), the differences were small, amounting to only 2·5 deg. In contrast, the angle between the sternum and the costal cartilage at FRC (α_1) decreased continuously and markedly from the first interspace caudally ($P < 0{\cdot}001$). Whereas this angle in the six animals studied averaged 51·0 ± 1·4 deg in the first interspace, it was only 31·5 ± 1·3 deg in the seventh interspace ($P < 0{\cdot}001$).

The angles between the sternum and the costal cartilages of ribs 2, 4, 6 and 8 after passive inflation (α_2) are also shown in Fig. 3. With inflation, the cartilage of rib 4 rotated by 23·7 ± 0·9 deg, whereas the cartilage of rib 2 rotated by only 11·8 ± 1·9 deg ($P < 0{\cdot}001$) and the cartilage of rib 8 rotated by only 11·2 ± 0·9 deg ($P < 0{\cdot}001$). Consequently, whereas at FRC the angle between the sternum and the costal cartilage was greatest in the first interspace, after passive inflation it was greatest in the third interspace ($P < 0{\cdot}001$).

Computed changes in muscle length

If we substitute into eqn (5) the values of β, α_1 and α_2 shown in Fig. 3, we can compute the changes in length of the parasternal intercostals in interspaces 1, 3, 5 and 7 and we can therefore test the validity of our model of the parasternal area (see Methods). The values thus computed are compared with the measured values in Fig. 4. The computed muscle shortening was greatest in the third interspace and smallest in the seventh interspace, in agreement with our measurements. Moreover, the computed values for the first, third and fifth interspaces agreed closely with the measured values, the differences ranging from 0 to only 11%.

The difference was larger for the seventh interspace, such that the computed value was 44% lower than the measured value. This difference is probably accounted for by the shape of the costal cartilage of rib 8. That is, we measured the orientation of the costal cartilages right at the chondrosternal junction, thus implying that the cartilages between the sternum and the points of insertion of the sternal parasternal bundles were straight, whereas in fact, the cartilage of rib 8 is curved with a rostral concavity. The 'effective' angles α_1 and α_2 for the seventh interspace were therefore slightly greater than those reported in Fig. 3, so

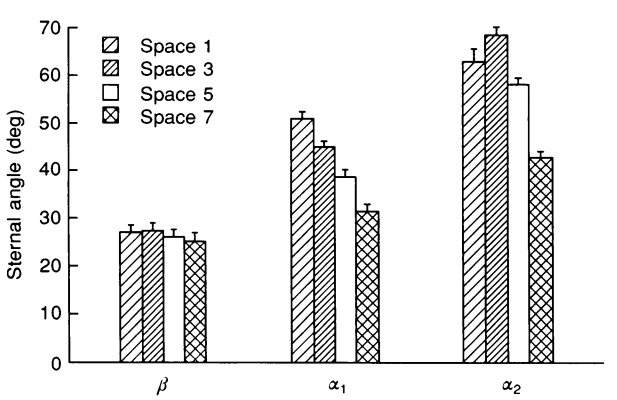

Figure 3. Orientations of the parasternal intercostals and costal cartilages

The values shown are the acute angles between the sternum and the sternal parasternal fibres (β), the acute angles between the sternum and the costal cartilages at FRC (α_1), and the acute angles between the sternum and the cartilages after a 1 l passive inflation (α_2) in interspaces 1, 3, 5 and 7. Data are means ± S.E.M. obtained from six animals.

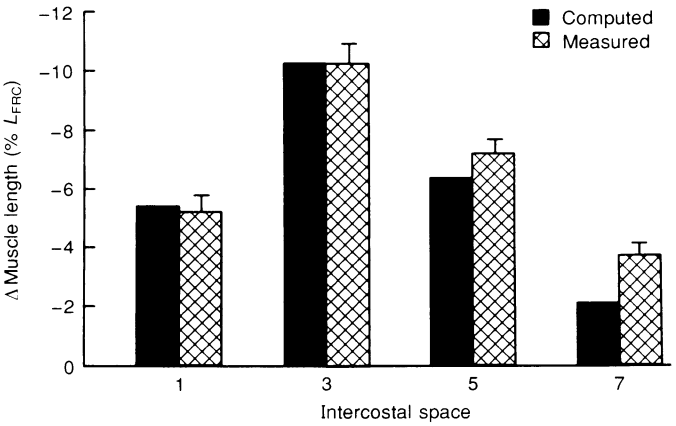

Figure 4. Validation of the geometric model of the parasternal area

The values shown are the measured fractional changes in length of the parasternal intercostals (mean ± S.E.M. of the data shown in Fig. 2) and those computed from the model shown in Fig. 1. Same conventions as in Fig. 2.

that the computed shortening of the corresponding parasternal muscle was underestimated.

Inspiratory effect (Expt 3)

Figure 5*A* shows the changes in airway pressure produced by the sternal portion of the parasternals in interspaces 3, 5 and 7 in the six animals studied. When stimulated, the muscles in all interspaces caused a fall in airway pressure. In all animals, however, the fall in pressure obtained during stimulation of the third interspace was larger than that obtained during stimulation of the fifth ($P < 0{\cdot}01$), and the latter, in turn, was larger than the fall in pressure produced by the seventh interspace ($P < 0{\cdot}001$). This pattern persisted after the changes in pressure were corrected for muscle mass (Fig. 5*B*); specific ΔP_{ao} for the fifth and seventh interspaces represented, respectively, 68 and 25% of the specific ΔP_{ao} for the third interspace ($P < 0{\cdot}005$ for each).

DISCUSSION

The main findings of the present studies agree, by and large, with our theoretical predictions (Wilson & De Troyer, 1992, 1993) and can be summarized as follows. (1) The length response of the canine parasternal intercostal muscles to passive inflation varies along the rostrocaudal axis of the ribcage. However, this response is not distributed along a simple monotonic gradient and has its climax in the second and third interspaces. (2) As shown by the close agreement between the measured and the computed values of length changes (Fig. 4), this peculiar distribution results from a combination of two factors, namely the different orientations of the costal cartilages at FRC and their different rotations during ribcage expansion. (3) The topographic distribution of parasternal shortening during inflation reflects well the distribution of inspiratory effect.

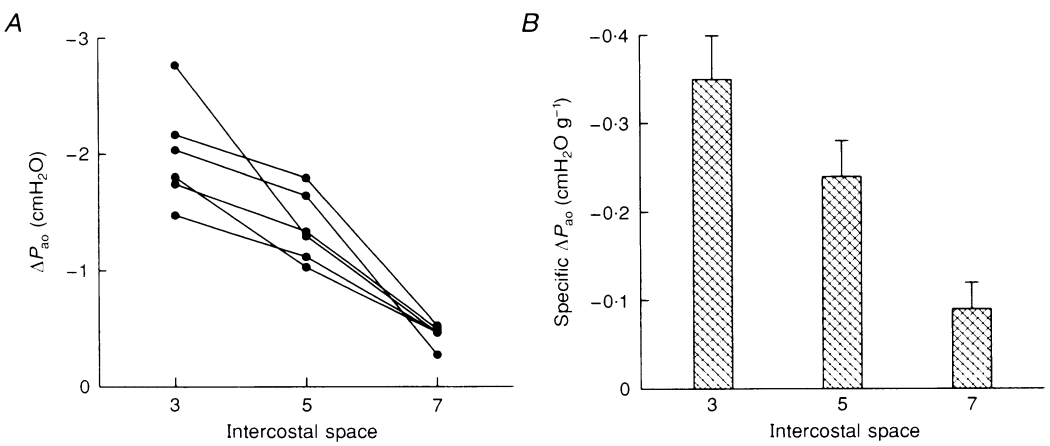

Figure 5. Inspiratory effect of the parasternal intercostals in interspaces 3, 5 and 7

A shows the individual changes in airway pressure (P_{ao}) measured during bilateral, isolated stimulation of the parasternal intercostals in six animals. *B* shows the mean ± S.E.M. changes in P_{ao} corrected for muscle mass (specific ΔP_{ao}).

Gradient of shortening

Although a number of investigators have measured by sonomicrometry the respiratory changes in length of the canine parasternal intercostals (Decramer & De Troyer, 1984; van Lunteren & Cherniack, 1986; De Troyer *et al.* 1988; Di Marco *et al.* 1990; De Troyer & Farkas, 1993), only one study has evaluated the changes in muscle length in the second to seventh interspaces during inflation of the relaxed chest wall (Decramer & De Troyer, 1984). No systematic difference between the degree of shortening of the muscles in the upper *vs.* the lower interspaces was found, in contrast to the current study. Measurements obtained by sonomicrometry, however, are very sensitive to technical factors, such as the orientation of the piezoelectric crystals relative to the muscle fibres and the stability of the crystals during muscle shortening. More importantly, in that study, the location of the muscle bundles along the costal cartilages was not defined systematically. We have recently shown, however, that the shortening of the parasternal bundles in any given interspace decreases markedly from the sternum to the chondrocostal junction (De Troyer & Legrand, 1995). By implanting screws in the costal cartilages along the axis of the parasternal bundles situated in the immediate vicinity of the sternum, we could overcome these problems and thus obtain a precise evaluation of the rostrocaudal distribution of the passive changes in length of the parasternal intercostal muscles.

Mechanism of the gradient

Margulies, Rodarte & Hoffman (1989) have previously studied the configuration of ribs 3–8 at FRC and total lung capacity (TLC) in four beagle dogs with the dynamic spatial reconstructor (DSR). By fitting a plane to each of these ribs, they found that the bucket-handle angle of the ribs at FRC, expressed relative to the transverse plane of the body, increased progressively from rib 3 to rib 8, thus indicating that the ribs in the dog are slanted more caudally as one moves from the apex of the ribcage toward the base. Consistent with this observation, we found that the acute angle α_1 between the sternum and the costal cartilage decreased gradually from rib 3 to rib 8. Margulies *et al.* (1989) also reported that the change in bucket-handle angle between FRC and TLC decreased progressively from rib 3 to rib 8. Although these investigators did not measure the displacements of ribs 1 and 2, this gradient of rib rotation is also consistent with the gradient of cartilage rotation measured in the present studies.

The rotation of the costal cartilages measured in this study was larger than the bucket-handle component of rib rotation reported by Margulies *et al.* (1989). For example, they reported a bucket-handle rotation of 14·5 deg in rib 4, whereas we observed that the costal cartilage of rib 4 rotated by 23·7 deg; corresponding values for rib 8 are 4·5 and 11·2 deg, respectively. Some of this difference may relate to the fact that the animals in the two studies were of different breeds and had different sizes. However, it is likely that the pump-handle component of rib rotation also contributes to cartilage rotation. Margulies *et al.* (1989) reported that the pump-handle component of rotation amounted to ~6 deg. The distance from the spine to the sternum is 10–14 cm. Therefore, if the bony rib extended to the sternum, the cranial displacement of the ventral tip of the ribs would be >1 cm. On the other hand, we have previously observed that the cranial displacement of the sternum during a 1 l passive inflation is only ~0·4 cm (Wilson & De Troyer, 1992). The difference between the cranial displacement of the rib and the cranial displacement of the sternum must be accomodated by a rotation of the costal cartilage. This rotation, with a magnitude of several degrees, would add to the rotation due to the bucket-handle component of rib rotation.

The question then arises as to why ribs 3 and 4 show a larger bucket-handle rotation with passive inflation than ribs 2 and 8. The smaller rotation of rib 8 could be related, at least in part, to passive tension in the diaphragm. Since the studies of Agostoni & Rahn (1960), it has been recognized that the diaphragm at FRC is passively stretched in the supine posture because of the action of gravity on the abdominal contents (Sprung, Deschamps, Margulies, Hubmayr & Rodarte, 1990). This passive diaphragmatic tension is likely to lift the lower ribs and rotate them outward in much the same way that an active diaphragmatic contraction does (De Troyer, Sampson, Sigrist & Macklem, 1982; Loring & Mead, 1982). Therefore, a positive pressure applied at the airway opening in the supine posture would have two opposing effects on the lower ribs: a direct effect, which would rotate these ribs cranially and expand the lower ribcage, and an indirect effect, which would reduce the cranial rotation of the lower ribs via the caudal displacement of the diaphragmatic dome and the decrease in passive tension in the diaphragm. In addition, these ribs are also connected to the muscles of the abdominal wall. These muscles are stretched and develop passive tension with passive inflation (Leevers & Road, 1989), which may also oppose the cranial displacement of the lower ribs.

Since the diaphragm and abdominal muscles have no insertions into the rostral ribs, these mechanisms cannot account for the smaller rotation of ribs 1 and 2. However, the cartilaginous attachments between the bony ribs and the sternum are known to be shorter and more restrictive for the rostral than the caudal ribs, and previous studies in dogs (De Troyer & Decramer, 1985) and in humans (De Troyer, Estenne & Vincken, 1986) have shown that the respiratory displacements of the rostral ribs are more tightly coupled to the sternum than those of the caudal ribs. One might expect, therefore, that the rotations of ribs 1 and 2 and of the costal cartilages of these ribs during passive inflation would be smaller than those of the more caudal ribs. There might also be differences in the costovertebral articulations that contribute to the lower rotational compliance of these ribs, but we do not know of any data that would support this conjecture.

Gradient of inspiratory effect

Based on the Maxwell reciprocity theorem, we have previously speculated that the potential effect of a given respiratory muscle on the respiratory system is proportional to the fractional change in muscle length per unit volume increase of the relaxed chest wall (Wilson & De Troyer, 1992, 1993). If this conclusion were correct, the observed shortening of the parasternal intercostal muscles during passive inflation would therefore indicate that, for a given tension and a given mass, the sternal portion of the parasternal intercostal in the third interspace would produce a larger fall in airway pressure or a larger increase in lung volume than the sternal portion of the parasternal intercostals in the first, fifth and seventh interspaces.

We have not been able to test this prediction completely because in the dog, the internal intercostal nerve in the first interspace is very thin and can be exposed only over a short distance. We did not feel confident, therefore, that we could evaluate the pressure-generating ability of this particular muscle. When we maximally stimulated the parasternals in interspaces 3, 5 and 7, however, specific ΔP_{ao} was consistently greater for the third interspace than for the fifth (Fig. 5). The latter was also greater than specific ΔP_{ao} for the seventh interspace, in agreement with the prediction. As shown in Fig. 6, specific ΔP_{ao} for these muscles was, in fact, proportional to the fractional changes in muscle length during passive inflation. In addition, according to eqn (1), the slope (σ) of the relationship between the fractional change in muscle length during passive inflation and specific ΔP_{ao} should be the maximal active muscle tension per unit cross-sectional area. Previous *in vitro* measurements of maximal active tension in limb and respiratory muscles, including the parasternal intercostals, have yielded values ranging between 2·2 and 3·5 kg cm^{-2} (Close, 1972; Farkas, Decramer, Rochester & De Troyer, 1985; Farkas, 1991; Tao & Farkas, 1992). Although such measurements are made during isometric contractions, whereas our assessment of ΔP_{ao} involved substantial muscle shortening, a line with a slope of 3·0 fits the current data remarkably well (Fig. 6).

Implications

These findings have two important implications. First, we have recently demonstrated that, in the dog, the medial portion of the parasternal intercostal muscles has a greater inspiratory effect than the lateral portion, and we have shown that this gradient is well reflected by the gradient of muscle shortening during passive inflation (Legrand *et al.* 1996). By demonstrating that the gradient of parasternal shortening also mirrors the gradient of inspiratory effect along the rostrocaudal axis of the ribcage, the present studies thus provide additional support to the idea (Wilson & De Troyer, 1992) that the mechanical advantage of a given respiratory muscle can be assessed from its fractional change in length during passive inflation. If the mass of the muscle is also measured, then its potential effect on the respiratory system could be calculated. This important finding should be particularly useful in humans, in whom the respiratory effects of the muscles cannot be studied directly.

In addition, the observation that the mechanical advantage of the parasternal intercostal muscles is largely determined by the rotational compliance of the ribs may also be

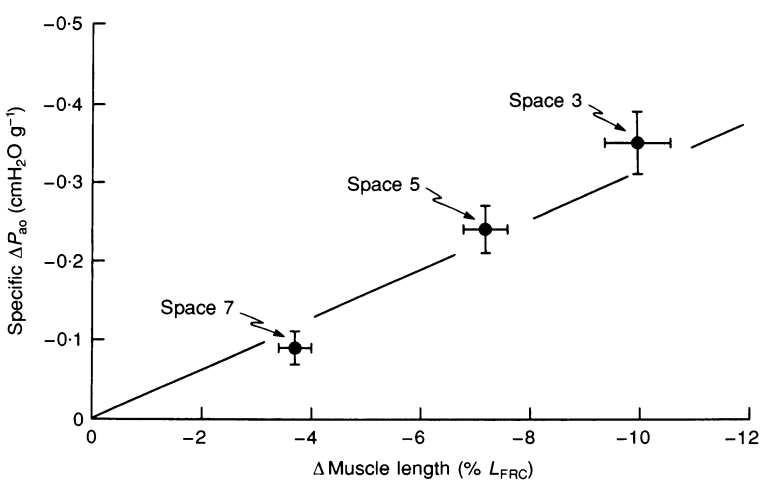

Figure 6. Relationship between the fractional changes in muscle length and the changes in airway pressure per unit muscle mass (specific ΔP_{ao})

The fractional changes in muscle length were obtained during passive inflation in eight animals (mean ± S.E.M. of the data shown in Fig. 2) and they are expressed as percentage changes relative to the muscle length at end-expiration (L_{FRC}). The values of specific ΔP_{ao} were obtained during tetanic stimulation of the internal intercostal nerves in six animals (mean ± S.E.M.; see Fig. 5) and they are expressed as cmH_2O (g muscle mass)$^{-1}$. The line shows the theoretical relationship between muscle length and specific ΔP_{ao}, based on the idea that the maximal active muscle tension per unit cross-sectional area is 3·0 kg cm^{-2} (Farkas, 1991).

pertinent to the behaviour of the external and internal interosseous intercostals. Thus, the cartilage that forms the caudal boundary of the third interspace rotates more than the cartilage of the first interspace during inflation of the relaxed chest wall. This implies that the distance between ribs 2 and 4 decreases as lung volume increases. Compression of these interspaces would induce shortening of intercostal muscles, regardless of their orientation. Both the external and internal interosseous intercostals in the rostral interspaces might therefore shorten during lung inflation and have an inspiratory mechanical advantage. Conversely, the magnitude of cartilage rotation decreased from the third to the seventh interspace. Therefore, both layers of interosseous intercostal muscles in the caudal interspaces may lengthen during inflation of the relaxed chest wall and have expiratory mechanical advantages. The topographic distribution of the mechanical advantage of these two sets of intercostal muscles will be examined in detail in future studies.

AGOSTONI, E. & RAHN, H. (1960). Abdominal and thoracic pressures at different lung volumes. *Journal of Applied Physiology* **15**, 1087–1092.

CLOSE, R. I. (1972). Dynamic properties of mammalian skeletal muscles. *Physiological Reviews* **52**, 129–197.

DECRAMER, M. & DE TROYER, A. (1984). Respiratory changes in parasternal intercostal length. *Journal of Applied Physiology* **57**, 1254–1260.

DE TROYER, A. (1991). Inspiratory elevation of the ribs in the dog: primary role of the parasternals. *Journal of Applied Physiology* **70**, 1447–1455.

DE TROYER, A. & DECRAMER, M. (1985). Mechanical coupling between the ribs and sternum in the dog. *Respiration Physiology* **59**, 27–34.

DE TROYER, A., ESTENNE, M. & VINCKEN, W. (1986). Rib cage motion and muscle use in high tetraplegics. *American Review of Respiratory Disease* **133**, 1115–1119.

DE TROYER, A. & FARKAS, G. A. (1993). Mechanics of the parasternal intercostals in prone dogs: statics and dynamics. *Journal of Applied Physiology* **74**, 2757–2762.

DE TROYER, A. & FARKAS, G. A. (1994). Contribution of the rib cage inspiratory muscles to breathing in baboons. *Respiration Physiology* **97**, 135–146.

DE TROYER, A., FARKAS G. A. & NINANE, V. (1988). Mechanics of the parasternal intercostals during occluded breaths in dogs. *Journal of Applied Physiology* **64**, 1546–1553.

DE TROYER, A. & KELLY, S. (1982). Chest wall mechanics in dogs with acute diaphragm paralysis. *Journal of Applied Physiology* **53**, 373–379.

DE TROYER, A. & LEGRAND, A. (1995). Inhomogeneous activation of the parasternal intercostals during breathing. *Journal of Applied Physiology* **79**, 55–62.

DE TROYER, A., SAMPSON, M., SIGRIST, S. & MACKLEM, P. T. (1982). Actions of costal and crural parts of the diaphragm on the rib cage in dog. *Journal of Applied Physiology* **53**, 30–39.

DI MARCO, A. F., ROMANIUK, J. R. & SUPINSKI, G. S. (1990). Parasternal and external intercostal muscle shortening during eupneic breathing. *Journal of Applied Physiology* **69**, 2222–2226.

ECCLES, R. M., SEARS, T. A. & SHEALY, C. N. (1962). Intracellular recording from respiratory motoneurones of the thoracic spinal cord of the cat. *Nature* **193**, 844–846.

FARKAS, G. A. (1991). Mechanical properties of respiratory muscles in primates. *Respiration Physiology* **86**, 41–50.

FARKAS, G. A., DECRAMER, M., ROCHESTER, D. F. & DE TROYER, A. (1985). Contractile properties of intercostal muscles and their functional significance. *Journal of Applied Physiology* **59**, 528–535.

LEEVERS, A. M. & ROAD, J. D. (1989). Mechanical response to hyperinflation of the two abdominal muscle layers. *Journal of Applied Physiology* **66**, 2189–2195.

LEGRAND, A., WILSON, T. A. & DE TROYER, A. (1996). Mediolateral gradient of mechanical advantage in the canine parasternal intercostals. *Journal of Applied Physiology* (in the Press).

LORING, S. H. & MEAD, J. (1982). Action of the diaphragm on the rib cage inferred from a force-balance analysis. *Journal of Applied Physiology* **53**, 756–760.

MARGULIES, S. S., RODARTE, J. R. & HOFFMAN, E. A. (1989). Geometry and kinematics of dog ribs. *Journal of Applied Physiology* **67**, 707–712.

SPRUNG, J., DESCHAMPS, C., MARGULIES, S. S., HUBMAYR, R. D. & RODARTE, J. R. (1990). Effect of body position on regional diaphragm function in dogs. *Journal of Applied Physiology* **69**, 2296–2302.

TAO, H. Y. & FARKAS, G. A. (1992). Predictability of ventilatory muscle optimal length based on excised dimensions. *Journal of Applied Physiology* **72**, 2024–2028.

TAYLOR, A. E. (1960). The contribution of the intercostal muscles to the effort of respiration in man. *Journal of Physiology* **151**, 390–402.

VAN LUNTEREN, E. & CHERNIACK, N. S. (1986). Electrical and mechanical activity of respiratory muscles during hypercapnia. *Journal of Applied Physiology* **61**, 719–727.

WHITELAW, W. A. & FEROAH, T. (1989). Patterns of intercostal muscle activity in humans. *Journal of Applied Physiology* **67**, 2087–2094.

WILSON, T. A. & DE TROYER, A. (1992). Effect of respiratory muscle tension on lung volume. *Journal of Applied Physiology* **73**, 2283–2288.

WILSON, T. A. & DE TROYER, A. (1993). Respiratory effect of the intercostal muscles in the dog. *Journal of Applied Physiology* **75**, 2636–2645.

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