Mechanical advantage of sternomastoid and scalene muscles in dogs

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Legrand, Alexandre, Vincent Ninane, and André De Troyer. Mechanical advantage of sternomastoid and scalene muscles in dogs. J. Appl. Physiol. 82(5): 1517–1522, 1997.—Theoretical studies have led to the prediction that the maximal effect of a given respiratory muscle on airway opening pressure (Pao) is the product of muscle mass, the maximal active muscle tension per unit cross-sectional area, and the fractional change in muscle length per unit volume increase of the relaxed chest wall. It has previously been shown that the parasternal intercostals behave in agreement with this prediction (A. De Troyer, A. Legrand, and T. A. Wilson. J. Physiol. (Lond.) 495: 239–246, 1996; A. Legrand, T. A. Wilson, and A. De Troyer, J. Appl. Physiol. 80: 2097–2101, 1996). In the present study, we have tested the prediction further by measuring the response to passive inflation and the pressure-generating ability of the sternomastoid and scalene muscles in eight anesthetized dogs. With 1-liter passive inflation, the sternomastoids and scalenes shortened by 2.03 ± 0.17 and 5.98 ± 0.43%, respectively, of their relaxation length (\(L_{\text{rel}}\)). During maximal stimulation, the two muscles caused similar falls in Pao. However, the sternomastoids had greater mass such that the change in Pao (\(\Delta\text{Pao}\)) per unit muscle mass was \(-0.19 ± 0.02\) cmH\(_2\)O/g for the scalenes and only \(-0.07 ± 0.01\) cmH\(_2\)O/g for the sternomastoids (\(P < 0.001\)). After extension of the neck, there was a reduction in both the muscle shortening during passive inflation and the fall in Pao during stimulation. The \(\Delta\text{Pao}\) per unit muscle mass was thus closely related to the change in length; the slope of the relationship was 3.1. These observations further support the concept that the fractional changes in length of the respiratory muscles during passive inflation can be used to predict their pressure-generating ability.

Mechanics of breathing; respiratory muscles; neck inspiratory muscles

ON THE BASIS of a standard theorem of mechanics, the Maxwell reciprocity theorem, Wilson and De Troyer (18, 19) have postulated that the potential mechanical effect of a given respiratory muscle on the respiratory system (i.e., its respiratory effect) is proportional to the change in length of the muscle during inflation of the relaxed chest wall. Specifically, the change (\(\Delta\)) in airway opening pressure (\(\Delta\text{Pao}\)) produced by a muscle contracting alone against a closed airway would be the product of three factors, namely, the mass (\(m\)) of the muscle, the maximal active muscle tension per unit cross-sectional area (\(s\)), and the fractional change in muscle length per unit volume increase of the relaxed chest wall (\([\Delta L/(L\Delta V_L)]_{\text{rel}}\)). Hence

\[
\Delta\text{Pao} = m\sigma[\Delta L/(L\Delta V_L)]_{\text{rel}} \tag{1}
\]

where \(L\) is muscle length, \(V_L\) is volume of chest wall, and \(\text{rel}\) is relaxation.

To test the validity of this theoretical conclusion, we have initially assessed the respiratory effect of the internal intercostal muscles of the parasternal region of the rib cage (the so-called parasternal intercostals). Indeed, although these muscles are known to be electrically active during inspiration (7, 17) and to play a major role in causing the inspiratory elevation of the ribs (3, 5), the shortening of the parasternal bundles during passive inflation decreases markedly from the sternal to the chondrocostal junctions (8) and decreases also from the third to the second interspace (9). When the parasternal intercostal in a given interspace was maximally activated by electrical stimulation, the medial portion was observed to generate a larger fall in Pao than the lateral portion, in agreement with the prediction (14). The parasternal intercostal in the third interspace also induced a larger fall in airway pressure than the muscles in the more caudal interspaces (9). Furthermore, and perhaps more importantly, the ratio of the fall in airway pressure per unit parasternal muscle mass (\(\Delta\text{Pao}/m\)) to the fractional muscle shortening during passive inflation was invariably \(-3.0\) kg/cm\(^2\), in close agreement with values of maximal active tension measured in vitro (1, 11, 16).

In the present study, we have tested further the validity of Eq. 1 by measuring the response to passive inflation and the respiratory effect of the canine sternomastoid and scalene muscles. These muscles were selected because they are anatomically well individualized and can be activated selectively. In addition, in contrast to the parasternal intercostals, they attach to both the rib cage and the head (sternomastoids) or the spine (scalenes). Therefore, if these muscles also behaved in agreement with Eq. 1, they would strongly strengthen the idea that the fractional changes in length of the different respiratory muscles during passive inflation can be used to predict their respective respiratory effects.

METHODS

The studies were carried out on eight adult mongrel dogs (15–25 kg) anesthetized with pentobarbital sodium (initial dose = 30 mg/kg iv). The animals were placed in the supine posture, intubated with a cuffed endotracheal tube, and connected to a mechanical ventilator (Harvard pump, Chicago, IL). The rib cage and scalene muscles were exposed on both sides of the chest from the first to seventh ribs by deflection of the skin and pectoralis muscles, and the sternomastoids were exposed through a long midline incision of the neck. The animal’s neck and mandible were then secured parallel to the floor, after which we measured the changes in length of the two muscles during passive inflation and their
pressure-generating ability. Only the principal, medial head of the scalenes (pars supracostalis) was evaluated. The level of anesthesia was maintained so that the corneal reflex was abolished throughout the study, and all measurements were made during mechanically induced apneas.

Changes in muscle length. The changes in sternomastoid and scalene muscle length during passive inflation were measured with screws implanted in the bones at the extremities of the muscle fibers, as previously described (8). For the sternomastoid, the screws were inserted into the mastoid process cranially and into the tip of the manubrium sterni caudally, whereas for the scalenes they were inserted into the cervical vertebrae and the fourth and fifth ribs in the anterior axillary line; the long tendinous attachments of the scalene muscle fibers to the seventh and eighth ribs were thus excluded from the measurements. The linear distance between each pair of screws (i.e., the length of each muscle) was then measured with a caliper first at resting end expiration (functional residual capacity [FRC]) and then after a 1-liter passive inflation. Each measurement was made in triplicate.

Pressure-generating ability. When these measurements were completed, two pairs of stainless steel hook electrodes spaced 3–4 mm apart were implanted into the muscles, and their motor nerves were exposed bilaterally. The sternomastoid muscle in the dog is supplied by the cervical accessory nerve, which enters the muscle at its deep aspect about midway between the manubrium and the mastoid process (10), whereas the pars supracostalis of the scalene is innervated by two branches of the second and third internal intercostal nerves (4). The nerves thus exposed were positioned across bipolar stimulating electrodes, and pulses of 0.2-ms duration were delivered at intervals of 1 s. The two electromyographic signals were processed by using 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. For each nerve, stimulus intensity was increased progressively until it was 50% greater than that required to produce a compound muscle action potential of maximal amplitude.

A Validyne differential pressure transducer was subsequently connected to a side port of the endotracheal tube to measure $P_{ao}$. The tube was occluded, and the cervical accessory nerve was stimulated bilaterally at intervals by applying square pulses of 0.2-ms duration and supramaximal voltage at a frequency of 50 impulses/s. The scalene branches of the second and third internal intercostal nerves were then stimulated bilaterally with similar pulses. Each stimulation was performed at least three times. It is important to stress that the animal's head remained firmly tethered throughout these measurements to avoid any confounding influence of neck motion on $P_{ao}$.

In seven animals, the position of the neck was subsequently altered so as to mimic the natural orientation of the sternomastoid and scalene muscles, as can be seen in standing dogs. Thus, with the shoulder girdle still attached to the board, the animal's head was suspended off the edge of the board, and all measurements were repeated. The animal's neck was finally brought back to the initial control position, the costal insertions of the scalene muscles were severed bilaterally, and a last set of tetanic, supramaximal stimulation of the second and third internal intercostal nerves was performed. An overdose of pentobarbital sodium was then given, and the sternomastoid and scalene muscles were harvested bilaterally and weighed.

Data analysis. The length of each muscle in each neck position was averaged over the three measurements that were performed, both at FRC and after passive inflation. The $\Delta P_{ao}$ results measured during stimulation were also averaged over the three measurements performed. To allow comparison between the two muscles in the different animals, $\Delta P_{ao}$ was subsequently divided by muscle mass to yield specific $\Delta P_{ao}$, and the changes in muscle length induced by passive inflation were expressed as percent changes relative to the muscle length at FRC. Because there were only minor and inconsistent differences between the scalene bundles attached to the fourth and fifth ribs, the fractional changes in length of the two bundles were also averaged. Data were finally averaged for the animal group, and they are presented as means ± SE.

For statistical comparisons between the sternomastoid and scalene muscles in the supine position, paired t-tests were done on the mean values thus calculated in each individual animal. Statistical assessment of the effects of neck extension on each muscle was also made by paired t-tests on the individual mean values. The criterion for statistical significance was taken as $P < 0.05$.

RESULTS

Changes in muscle length. Passive inflation in the supine position caused shortening of the sternomastoid and scalene muscles in all animals. However, the fractional shortening of the sternomastoids was invariably smaller than that of the scalenes. For the eight animals, the shortening of the sternomastoids thus averaged 2.03 ± 0.17% of the muscle length at FRC, whereas the shortening of the scalenes amounted to 5.98 ± 0.43% (P < 0.001).

Pressure-generating ability. Stimulating the sternomastoid or the scalene muscles in the supine position resulted in a considerable cranial displacement of the rib cage and induced a fall in $P_{ao}$, as shown in Fig. 1.

![Fig. 1. Changes in airway opening pressure ($P_{ao}$) recorded during bilateral, tetanic stimulation of sternomastoid (top left) and scalene (top right) muscles in a representative animal in supine posture. EMG activity recorded from muscles is also shown.](image_url)
For the animal group, the ΔPao produced by the sternomastoids averaged $-4.96 \pm 0.75 \text{cmH}_2\text{O}$, and that produced by the scalenes was $-3.97 \pm 0.46 \text{cmH}_2\text{O}$. This difference was not statistically significant. However, the mass of the sternomastoids was greater than that of the scalenes ($66.07 \pm 8.64 \text{g}$ vs. $20.76 \pm 2.17 \text{g}$; $P < 0.001$). As a result, specific ΔPao was substantially smaller for the sternomastoids than for the scalenes ($-0.07 \pm 0.01$ vs. $-0.19 \pm 0.02$ cmH$_2$O/g; $P < 0.001$).

Effects of neck extension. Extension of the neck induced an increase in muscle length at end expiration, particularly in sternomastoid length. For the seven animals studied, the sternomastoid lengthened by $12.94 \pm 1.32\%$ of its control length, and the scalene lengthened by $6.59 \pm 0.67\%$ ($P < 0.001$ for both). However, both muscles shortened less during passive inflation with the neck extended than in the control, supine position (Fig. 2). One animal even had mild lengthening of the sternomastoids ($+0.09\%$). As a result, whereas in the control position the sternomastoid shortening in the seven animals averaged $1.96 \pm 0.19\%$, after extension of the neck, it was only $0.48 \pm 0.14\%$ of the new FRC length ($P < 0.01$). Corresponding values for the scalenes were $5.85 \pm 0.48$ and $3.73 \pm 0.46\%$, respectively ($P < 0.01$).

The effects of neck extension on the pressure-generating ability of the muscles are shown in Fig. 3. In all animals, stimulation of the sternomastoids resulted in a smaller fall in Pao than in the control position, so that specific ΔPao decreased from $-0.07 \pm 0.01$ to $-0.02 \pm 0.01 \text{cmH}_2\text{O/g}$ ($P < 0.001$). Six of seven animals also had a smaller fall in Pao during stimulation of the scalenes, such that specific ΔPao decreased from $-0.19 \pm 0.02$ to $-0.13 \pm 0.01 \text{cmH}_2\text{O/g}$ ($P < 0.01$).

When the costal insertions of the scalenes were severed, stimulating the scalene branches of the internal intercostal nerves did not induce any ΔPao. Thus the falls in Pao previously recorded during stimulation of these nerves were not contaminated by the actions of the intercostal muscles and resulted entirely from the contraction of the scalenes.

Relationship between changes in muscle length and ΔPao. The values of specific ΔPao obtained for the sternomastoids and scalenes in both neck positions are plotted against the corresponding fractional changes in muscle length during passive inflation in Fig. 4. Specific ΔPao was closely related to the change in muscle length such that a larger muscle shortening corresponded to a greater fall in Pao per unit muscle mass. The slope of this relationship was 3.1.

**DISCUSSION**

When the sternomastoid or scalene muscles in the dog are activated selectively by electrical stimulation,
the sternum and the ribs are displaced cranially and lung volume increases (7, 15). Thus, although these two muscles are not active during breathing (4), they have clear-cut inspiratory actions. One would anticipate, therefore, that both would shorten during inflation of the relaxed chest wall, and indeed passive inflation caused sternomastoid and scalene muscle shortening in all supine animals. The smaller cranial displacement of the sternum than the ribs during passive inflation (2, 6), combined with the longer length of the sternomastoid at FRC, accounts for the observation that the fractional shortening of this muscle was smaller than that of the scalenes.

The fractional shortening of the sternomastoids in our supine animals, in fact, was three times smaller than that of the scalenes, and the specific ∆Pao measured during maximal contraction was similarly about three times smaller for the sternomastoids than for the scalenes. This similarity was consistent with the theoretical prediction, summarized in Eq. 1, that the potential ∆Pao values produced per unit muscle mass by the different respiratory muscles are proportional to the fractional changes in length of the muscles during passive inflation (19). Previous studies by Farkas and Rochester (12), however, have shown that in supine dogs, the neck muscles operate on the ascending limb of their active length-tension curves. Specifically, when the animal's neck was positioned as it was initially in the present study, the relaxation length of the sternomastoid and scalene muscles at FRC corresponded, respectively, to 85 and 87% of their in vitro optimal force-producing length. However, when the neck was extended, there was considerable lengthening of the sternomastoids and moderate lengthening of the pars supracostalis of the scalenes. Consequently, specific ∆Pao for the sternomastoids in the control, supine position might have been significantly underestimated relative to the scalenes.

In agreement with Farkas and Rochester (12), the sternomastoids lengthened by 13% with neck extension, whereas the scalenes lengthened by only 6%. Thus, with this postural change, both muscles and particularly the sternomastoid moved toward their optimal force-producing length, yet the muscles' pressure-generating abilities were observed to be reduced.

This apparent paradox can be understood if one considers the effect of neck extension on the orientation of the muscle fibers (Fig. 5). The rib cage moves essentially through a rotation of the costovertebral joints, and hence the sternomastoids exert a torque when they contract. This torque (τ) depends on the force (F) generated by the muscles, the distance (r) from the point of rotation of the ribs to the point where the force acts on the rib cage (i.e., the manubrium sterni), and the angle (θ) between r and F, such that

\[ τ = rF \sin θ \]  

(2)

When the animal was supine with the neck in the control position, the long axis of the sternomastoid fibers was almost along the craniocaudal axis of the sternum. Gross measurements indicated that in this position, θ was actually ~80°. On the other hand, when the neck was extended, θ was only ~15°. All other things being equal, this reduction in θ would lead to a 74% reduction in τ. Because the increase in muscle length with neck extension was 13%, the potential increase in F was only 10–15% (12). Thus the magnitude of this increase in intramuscular F could not offset the reduction in θ, resulting in a marked decrease in τ. Extending the neck should affect the scalenes in a similar way, but because the pars supracostalis herein studied inserts into the ribs of the middle and caudal portions of the rib cage, rather than into the tip of the sternum, its orientation should be less affected by neck extension than that of the sternomastoids and so its inspiratory effect should be better preserved.

Extending the neck reduced not only specific ∆Pao but also the shortening of the muscles during passive inflation. More importantly, these two variables were
reduced in proportion. When the neck was extended, specific ΔPao for the scalenes was, on average, 68% of the control, supine value, and the fractional shortening of the muscle was 64%. Similarly, specific ΔPao and the fractional shortening of the sternomastoid with the neck extended represented, respectively, 32 and 24% of the corresponding control value. This finding provided additional support to the prediction (19) that the fractional changes in length of the different respiratory muscles during passive inflation are proportional to their respiratory effects.

Even stronger evidence in support of this prediction was obtained. According to Eq. 1, the coefficient of proportionality (ν) between specific ΔPao and the fractional change in muscle length during passive inflation should be the maximal active muscle tension per unit cross-sectional area. When measured in vitro, maximal active tension in limb and respiratory muscles, including the scalenes and sternomastoids, is known to be a fairly constant value, ranging between 2.2 and 3.5 kg/cm² (1, 11, 12, 16). If the prediction were correct, therefore, the slope of the relationship between the fractional change in muscle length and specific ΔPao should be ~3.0. We have previously demonstrated that, indeed, the ratio between the specific respiratory effects of the canine parasternal intercostals in the different interspaces and their fractional changes in length during passive inflation is ~3.0 (9), and, similarly, the ratio calculated from the present data on the neck inspiratory muscles was 3.1 (Fig. 4). It appears, therefore, that the specific respiratory effects of the different muscles are uniquely related to their fractional changes in length during passive inflation, and hence, that these changes in length reflect well the muscles’ respective mechanical advantages (i.e., ΔPao/mν). As a corollary, if the respiratory effect of a particular muscle cannot be studied directly, it could be calculated from measurements of the muscle’s mass and fractional change in length during passive inflation.

We have previously demonstrated that the distribution of inspiratory electrical activity among the canine parasternal intercostals during breathing is precisely matched with the distribution of inspiratory mechanical advantage (8, 9, 13). Thus, in a given intercostal space, the medial parasternal bundles, which have the greatest inspiratory mechanical advantage, are strongly activated, whereas the lateral bundles, which have no inspiratory mechanical advantage, remain silent (8). Similarly, parasternal activity is greatest and starts first in the third interspace, where the inspiratory mechanical advantage is greatest, and it is smallest and occurs later in the seventh interspace, where the inspiratory mechanical advantage is also smallest (9, 13). Consequently, on the basis of the fact that the sternomastoid and scalene muscles are not used during breathing in the dog (4), we expected that both muscles would have smaller mechanical advantages than the parasternals. This was indeed the case for the sternomastoids, which, in the control, supine position, shortened by only 2% for a 1-liter passive inflation, i.e., less than the parasternal in the seventh interspace (3.7%). On the other hand, the fractional shortening of the scalenes in this position averaged 6%, which is more than the parasternal in the seventh interspace and only slightly less than the parasternal in the fifth interspace (7%).

The reason that dogs use the parasternal intercostals in all interspaces and not the scalenes during breathing is certain, but it must be remembered that the supine position is not physiological in this animal. When the neck was extended to simulate the natural orientation of the neck muscles, the inspiratory mechanical advantage of the sternomastoid became very small (0.5%), and that of the scalene was reduced to 3.7%; that is, it was similar then to the parasternal intercostal of the seventh interspace. Furthermore, it is well established that the topographic distribution of parasternal activity results from a central, rather than reflex, mechanism (13). It is possible that an inspiratory mechanical advantage of 3–4% corresponds to the threshold of inspiratory activity during resting breathing and, if that were the case, the scalenes in the dog would be inactive in their physiological position and would remain inactive in the supine position irrespective of their greater inspiratory mechanical advantage in this posture.

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