

Effect of body position on regional diaphragm function in dogs

JURAJ SPRUNG, CLAUDE DESCHAMPS, SUSAN S. MARGULIES, ROLF D. HUBMAYR, AND JOE R. RODARTE (With the Technical Assistance of Bruce J. Walters)
Departments of Medicine and Physiology, Baylor College of Medicine and The Methodist Hospital, Houston, Texas 77030; and Thoracic Disease Research Unit, Mayo Foundation, Rochester, Minnesota 55905

SPRUNG, JURAJ, CLAUDE DESCHAMPS, SUSAN S. MARGULIES, ROLF D. HUBMAYR, AND JOE R. RODARTE. *Effect of body position on regional diaphragm function in dogs.* *J. Appl. Physiol.* 69(6): 2296–2302, 1990.—The in situ lengths of muscle bundles of the crural and three regions of the costal diaphragm between origin and insertion were determined with a video roentgenographic technique in dogs. At total lung capacity (TLC) in both the prone and supine positions, the length of the diaphragm is not significantly different from the unstressed excised length, suggesting that the diaphragm is not under tension at TLC and that there is a hydrostatic gradient of pleural pressure on the diaphragmatic surface. Except for the ventral region of the costal diaphragm, which does not change length at lung volumes >70% TLC, all other regions are stretched during passive deflations from TLC. Therefore below TLC the diaphragm is under passive tension and supports a transdiaphragmatic pressure (Pdi). The length of the diaphragm relative to its unstressed length is not uniform at functional residual capacity (FRC) and does not follow a strict vertical gradient that reverses when the animal is changed from the supine to the prone position. By inference, the length of muscle bundles is determined by factors other than the vertical gradient of Pdi. During mechanical ventilation, regional shortening is identical to the passive deflation length-volume relationship near FRC. Prone and supine FRC is the same, but the diaphragm is slightly shorter in the prone position. In both positions, during spontaneous ventilation there are no consistent differences in regional fractional shortening, despite regional differences in initial length relative to unstressed length.

respiratory muscles; thoracoabdominal mechanics; mechanical ventilation

THE DIAPHRAGM is the major muscle of inspiration. In vivo its function is usually assessed from measurements of transdiaphragmatic pressure (Pdi) or thoracoabdominal volume changes. These measurements are used to estimate tension, contraction velocity, and muscle fiber length, which are variables characterizing muscle function in vitro. We have recently described a video roentgenographic technique for measuring muscle length and shortening of the crural and multiple regions of the costal diaphragm in vivo (13). In this publication, we have examined regional diaphragmatic shortening in supine anesthetized dogs during spontaneous and mechanical ventilation and found no consistent difference in diaphragm shortening between regions at different vertical heights. This may seem surprising according to the reasoning of Froese and Bryan (5), which assumes that

there is a vertical gradient in Pdi that should expose muscle bundles to a nonuniform axial stress. Accordingly, dependent regions of the diaphragm would be expected to be longer relative to this unstressed length and shorten more than nondependent regions because of the length-tension relationship of skeletal muscles.

This conventional model of regional diaphragm mechanics assumes uniform neuromuscular activation, uniform tissue material properties, and a simple diaphragm shape with a single radius of curvature. To gain further insights into the determinants of regional diaphragm function, we measured the unstressed lengths of excised muscle bundles in vitro and compared these with the in situ lengths of regions at lung volumes between total lung capacity (TLC) and residual volume (RV) in the prone and supine positions. Regional shortening during spontaneous breathing and mechanical ventilation was then related to the passive in situ length-volume relationships.

METHODS

Five bred-for-research beagle dogs weighing between 11 and 13 kg were surgically prepared via a midline laparotomy. Two-millimeter silicon-coated lead spheres with a central hole drilled through them were stitched to the peritoneal surface and superficial muscle fibers of the left hemidiaphragm. Rows of markers were placed along the course of a muscle bundle from the insertion of the diaphragm on the lateral rib cage to its origin on the central tendon. The markers were spaced at 1- to 2-cm intervals so that the chord length between them would closely approximate the arc length along the diaphragm. Four regions on the ventral, middle, and dorsal portions of the costal diaphragm and on the crural diaphragm were evaluated (Fig. 1). The animals were allowed to recover for at least 10 days before study. Thickening of the parietal peritoneum that alters the passive mechanical properties of the region was not present in any of these dogs (13).

The animals were anesthetized with pentobarbital sodium (30 mg/kg iv), intubated with a cuffed endotracheal tube, and placed in the position of interest in a radiolucent volume displacement body plethysmograph. Functional residual capacity (FRC) was measured according to Boyle's law technique. Flow was measured at the endotracheal tube opening with a Fleisch pneumotachograph. Flow and lung volume signals were recorded on a

strip chart recorder and simultaneously encoded on video tape along with the fluoroscopic images. The marker tracking technique as applied to the diaphragm has been previously described (13). Briefly, orthogonal biplane fluoroscopic images of the animal's lower thorax and abdomen are combined into a single video image that is recorded on a stop-action videotape (Ampex VPR2) along with simultaneously encoded analog signals. After the experiment, frames of interest were identified by decoding the analog information, and an operator-interactive computer program was used to identify the projection images of a given marker in each view. The location of each marker was then determined from the known geometry of the video roentgenographic system. From these data, the distances between markers of a row are computed.

The animals were initially placed either prone or supine in a fabric sling in the body plethysmograph. The forelimbs were extended along the head, and, in the supine position, the hindlimbs were gently extended. In the prone position, the hindlimbs were tucked under the animal so that the position was very similar to an awake animal lying prone. The abdomen was not supported and did not contact the sling. The rib cage was supported partially by the sling and partially by the forelimbs. A calibrated super syringe was used to determine the inflation volume that increased airway pressure to 30 cmH₂O. The lung volume at airway pressure of 30 cmH₂O is defined as TLC. This convention is used in our laboratory because it produces the same TLC prone and supine, which approximates the volume of excised lungs at 20 cmH₂O transpulmonary pressure, even if the measured esophageal pressure differs between the body positions (see Ref. 8 and Y. L. Lai, R. E. Hyatt, and J. R. Rodarte, unpublished data).

After several inflations to TLC, the animals were allowed to resume spontaneous breathing, and the airway was occluded at end expiration to determine FRC by Boyle's law. This procedure was repeated at least five

times and the results were averaged. The animals' lungs were inflated twice to TLC to ensure a constant volume history, and then data were recorded after a third inflation during stepwise deflation in 25% inspiratory capacity (IC) decrements to FRC. This maneuver was repeated five times and the data were averaged.

The animals' lungs were then inflated twice with TLC and allowed to resume breathing. Once a stable breathing pattern was obtained, usually within 3-5 breaths, 15 spontaneous breaths were recorded on videotape for subsequent analysis. The animals' lungs were then again inflated twice to TLC, and mechanical ventilation was begun with a Harvard mechanical ventilator at the same tidal volume and a slightly higher frequency than the immediately preceding spontaneous breaths. After a few breaths the frequency was reduced to match the spontaneous pattern, and 15 breaths were recorded. During this time there was no evidence of spontaneous respiratory efforts, as judged from the airway or esophageal pressure recordings or by inspection of the fluoroscopic images of the thorax. After these initial procedures, the animals were placed in the other recumbent position and the study was repeated. Supplemental anesthetic was periodically administered intravenously to maintain the animal anesthetized and breathing with a regular pattern.

At the end of the procedure, the animals were given a lethal overdose of barbiturate. The abdomen and chest were opened widely and the diaphragm was excised intact as quickly as possible. The excised diaphragms were laid flat on towels moistened with saline. Strips of muscle (1.5-2 in.) containing the rows of markers were excised from the diaphragm and incubated in 4°C Krebs solution containing (in mM) 137 NaCl, 4 KCl, 1 MgCl₂, 1 KH₂PO₄, 12 NaHCO₃, 2 CaCl₂, and 6.5 glucose, equilibrated with 95% O₂-5% CO₂. At the end of the incubation, the strips were gently removed and placed on moist towels, and the cumulative intermarker distances were measured with a ruler.

Data were analyzed by repeated-measures analysis of variance and by paired and unpaired *t* tests. Although regional differences within individual dogs were reproducible and highly statistically significant, we report only differences across dogs. Data from three of the five dogs during ventilation in the supine position were included in a previous communication (13).

RESULTS

Effects of lung volume on diaphragm length. FRC, the volume at end expiration during spontaneous ventilation, was not different between the two body positions [532 ± 38 (SE) ml supine, 536 ± 91 ml prone]. FRC was 41 ± 3 and 40 ± 4% of TLC in the supine and prone positions, respectively. Mean regional diaphragm lengths during deflation from TLC to RV in the supine position are shown in Fig. 2. At TLC, all regions of the diaphragm are ~15-20% shorter than at FRC. Near FRC, the costal ventral region shortens significantly more than the rest of the diaphragm, but its length remains constant above 70% TLC. Regional diaphragm length-lung volume relationships below FRC are similar to those immediately above FRC.

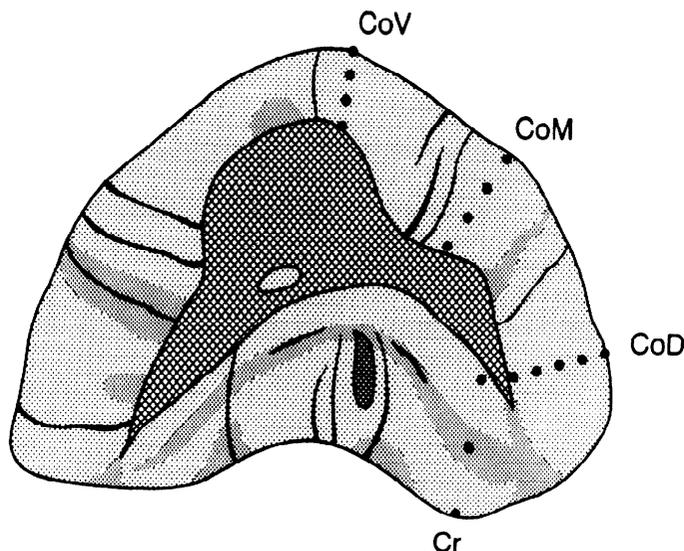


FIG. 1. Schematic view of the abdominal surface of canine diaphragm showing location of metallic markers along muscle bundles of costal and crural regions. CoV, CoM, and CoD, ventral, medial, and dorsal costal diaphragm; Cr, crural diaphragm.

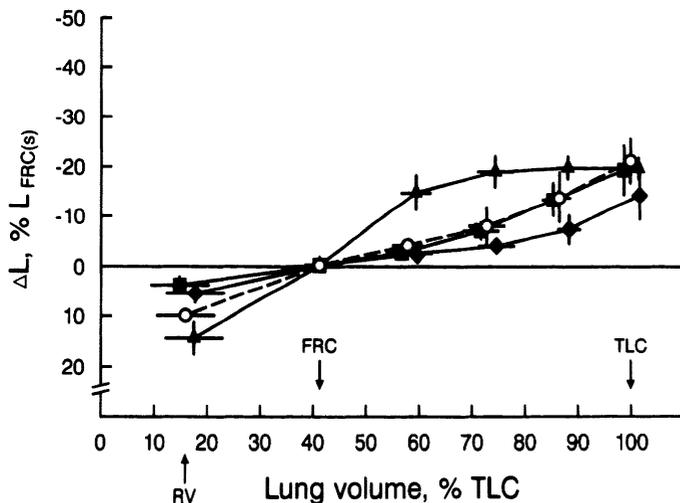


FIG. 2. Regional diaphragm muscle lengths as a function of lung volume during passive deflation in supine dogs. Changes of length (ΔL) are expressed as percentage of length at supine FRC ($\%L_{FRC}$). \circ , Crural region; \blacktriangle , costal ventral; \blacksquare , costal medial; and \blacklozenge , costal dorsal. Error bars, SD.

Diaphragm length-lung volume relationships in the prone position are shown in Fig. 3. In Fig. 3A, the regional lengths are expressed as a percentage of those at prone FRC. In contrast to the supine position, all regions shorten similarly at volumes near FRC. The dorsal region shortens most during an inflation to TLC. Figure 3B shows the diaphragm length-lung volume relationship in the prone position, with length expressed relative to the length at supine FRC. At FRC, the crural and middle regions are significantly shorter in the prone than in the supine position. At TLC, the middle and dorsal regions are significantly shorter in the prone position than in the supine position.

Figure 4 shows the lengths of various regions at the TLC expressed as a fraction of the excised length. None of the lengths are significantly different from the excised length, but, as noted above, the middle and dorsal regions are shorter in the prone than in the supine position.

Diaphragm lengths expressed as fractions of the excised lengths in the prone and supine positions are shown in Fig. 5. In both positions, at FRC the costal diaphragm is 20–40% longer than in its unstressed state *in vitro*. In the supine position, there is an approximate vertical gradient in length at FRC; the ventral costal region is shortest, the crural is longest, and the middle and dorsal are intermediate. In the prone position, the order between ventral costal, dorsal costal, and crural lengths is the same as in the supine posture, even though the Pdi gradient is reversed. The only difference is that the middle costal, rather than the dorsal, region is shortest. Lengths at RV are remarkably similar in the two positions, with the crural being stretched more than the costal diaphragm.

Effects of mode of ventilation on diaphragm shortening. Changes in regional shortening with body position and mode of ventilation are shown in Table 1. In both postures, regional diaphragm lengths at FRC were the same during spontaneous and mechanical ventilation. Tidal volumes are also similar (supine: 210 ± 38 ml spontaneous ventilation, 210 ± 31 ml mechanical ventilation;

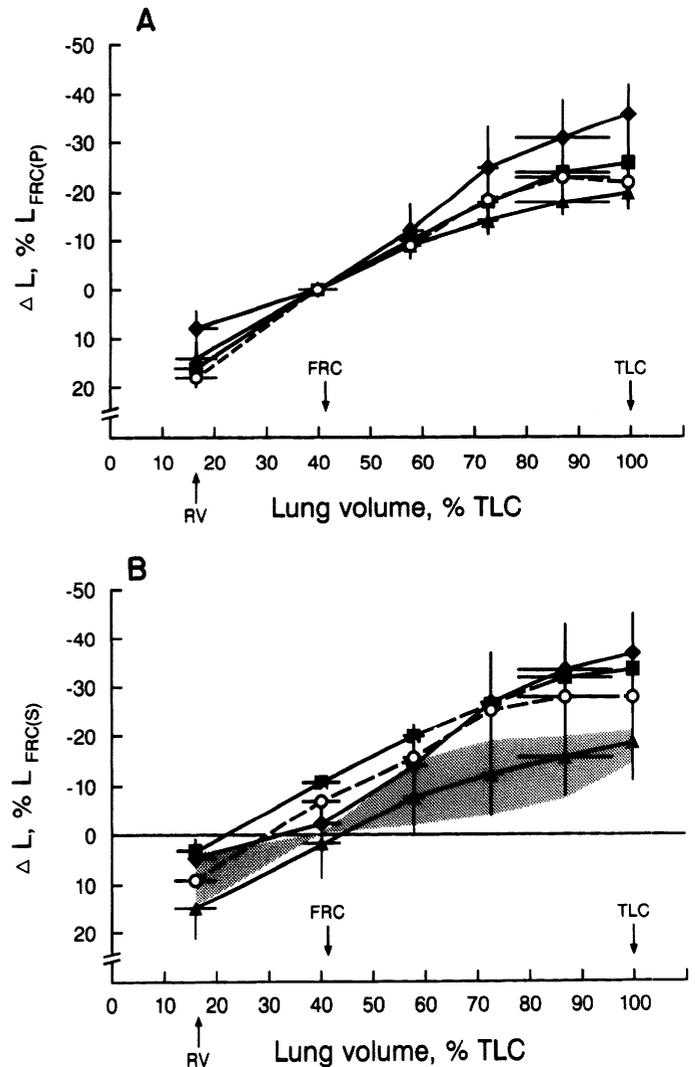


FIG. 3. Regional diaphragm muscle lengths as a function of lung volume during passive deflation in prone dogs. Symbols as in Fig. 2. A: length changes (ΔL) expressed as percentage of length at prone FRC ($\%L_{FRC}$). B: lengths expressed as percentage of length at supine FRC. Shaded area, range of data from Fig. 2.

prone: 230 ± 49 ml spontaneous ventilation, 222 ± 45 ml mechanical ventilation).

Because these tidal volumes are very similar to 25% IC, we compared regional shortening during mechanical ventilation and between FRC + 25% IC and FRC by paired *t* test; there was no significant difference. We also computed fractional length change per liter. Although there was a trend for the costal diaphragm to change length more during the passive deflation maneuver, the difference was not statistically significant (data not shown).

Mean diaphragm shortening was greater in the prone than in the supine position, but this difference did not achieve statistical significance by repeated-measures analysis of variance ($P = 0.078$). During mechanical ventilation, the crural and middle regions shortened more in the prone posture ($P < 0.05$ and $P < 0.01$, respectively). In both positions, the diaphragm shortens less during mechanical ventilation than during spontaneous ventilation ($P = 0.012$). In contrast to the other regions of the diaphragm, the decrease in costal ventral

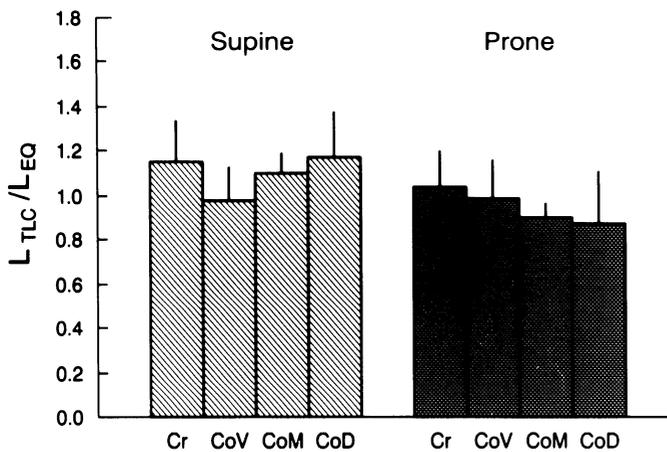


FIG. 4. Ratio of in vivo muscle lengths at passive TLC (L_{TLC}) to unstressed lengths after excision (L_{EO}). Abbreviations as in Fig. 1. Error bars, SD.

shortening during mechanical ventilation in the supine position was small and inconsistent.

Regional differences in diaphragm shortening during ventilation. During mechanical ventilation in the supine position, the ventral region of the costal diaphragm shortened most ($P = 0.001$). With this exception, there were no other significant differences of shortening between regions in either body position during either mode of ventilation.

DISCUSSION

Resolution of the method. The video roentgenographic technique utilized in this study has been previously described in detail (13). The standard deviations of intermarker distances at end inspiration and at FRC approximate 0.05 cm. The cord distance between markers placed at 1-cm intervals along the course of a muscle bundle closely approximates the arc length. The error introduced by a straight-line measurement depends on the ratio of intermarker distance to the local radius of curvature. If the ratio of intermarker distance to the radius of curvature is 0.25, the error from using the cord rather than the arc length is 0.23%. If this ratio is 0.50, the error is 1%. The error does not exceed 5% until the intermarker distance is greater than the radius of curvature. Therefore cord length is an accurate estimate of the arc length. Summing the intermarker distances over the entire length of the muscle bundle from the central tendon to the rib cage provides a more reproducible estimate of regional muscle length and shortening than a single two-point measurement (13).

Effects of lung volume on diaphragm length. For dogs in the supine posture, we observed a 20% shortening of the relaxed diaphragm between FRC and TLC, which is at the low end of the range of previous measurements made with sonomicrometry (10). In the absence of muscle activity, there is no mechanism by which the diaphragm could shorten to less than its unstressed length. However, the diaphragm could buckle, and if the marker technique did not detect it, an erroneous shortening would be computed. In the supine dog, the ventral costal diaphragm shortens more than the other regions, up to 50%

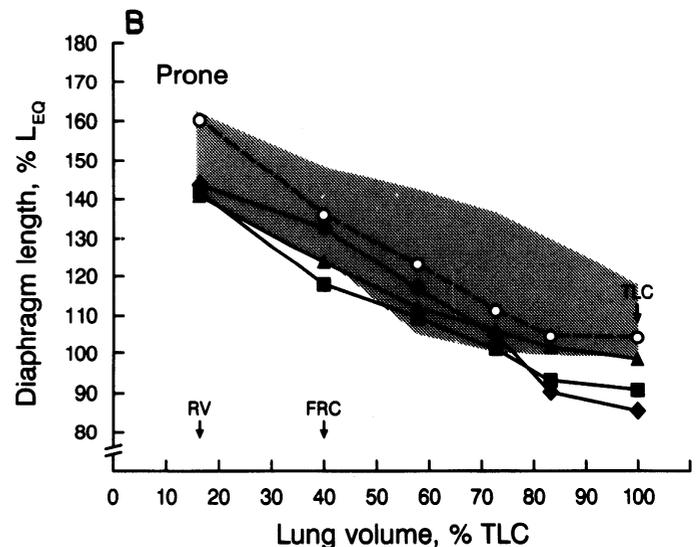
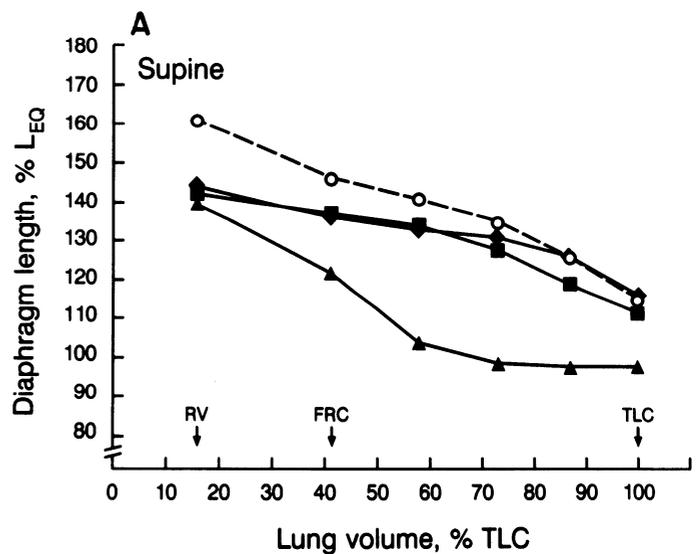


FIG. 5. Regional diaphragm muscle lengths as a function of lung volume during passive deflation. Symbols as in Fig. 2. Length is expressed as percentage of excised unstressed length ($\%L_{EO}$). Shaded area, range of data from A.

IC; thereafter its length remains constant. The muscle bundles in the ventral region are almost parallel to the midsagittal plane. The projection of markers from the ventral row midsagittal plane of a representative dog is shown in Fig. 6 during deflation from TLC to FRC. Above 50% IC, the diaphragm has a double curvature produced by the ventral edge of the cardiac lobe pushing caudally beyond the insertion of the diaphragm on the rib cage. Despite this buckling, the length of the muscle fiber above 50% IC was constant. We were unable to demonstrate buckling of other regions at TLC and thus believe the length measurements at TLC are not an artifact.

The law of Laplace indicates that the pressure difference across a membrane is proportional to the ratio of tension and radius of curvature. At the point where the diaphragm changes from concave to convex toward the abdomen, the radius changes sign and passes through zero. At that point, this relationship can only be satisfied if tension in that direction along the muscle bundle is

TABLE 1. Regional diaphragm shortening during two modes of ventilation and in two body positions

Dog No.	Spontaneous Ventilation				Mechanical Ventilation			
	Cr	CoV	CoM	CoD	Cr	CoV	CoM	CoD
<i>Supine</i>								
T979	12.2	18.0	15.6	9.1	7.2	12.1	4.6	2.9
T980	14.2	16.2	17.2	15.1	3.0	8.9	3.9	3.4
T982	7.6	10.2	8.7	4.4	3.5	10.0	3.1	1.1
T983	7.2	14.7	6.5	3.5	3.6	10.4	1.3	0.8
T984	10.2	10.7	11.5	13.4	3.9	10.9	1.6	2.7
Mean ± SD	10.3±2.9	14.0±3.4	11.9±2.0	9.1±5.2	4.2±1.7	10.1±1.6	2.9±1.4	2.2±1.2
<i>Prone</i>								
T979	16.6	24.1	18.6	14.7	8.8	6.3	1.01	8.5
T980	22.6	19.5	20.0	26.2	13.4	12.7	10.5	19.9
T982	7.4	14.6	12.3	7.0	7.3	7.9	8.7	4.7
T983	15.0	14.7	10.1	9.2	5.3	3.1	3.7	-0.8
T984	12.3	10.1	8.7	9.3	11.1	7.1	4.2	4.5
Mean ± SD	14.9±5.6	16.7±5.5	13.9±5.1	13.3±7.8	9.2±3.2	7.4±3.5	7.4±3.3	7.4±7.8

Cr, crural diaphragm; CoV, CoM, and CoD, ventral, medial, and distal costal diaphragm.

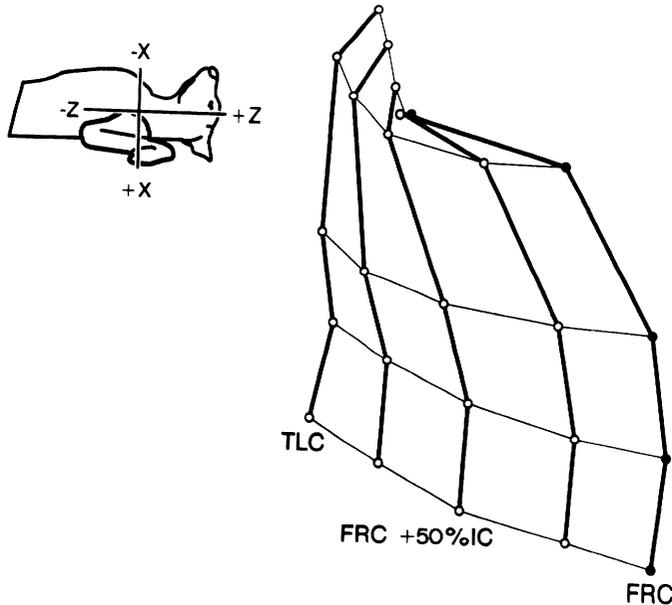


FIG. 6. Shape of ventral costal diaphragm during passive inflation from FRC to TLC in a supine dog. Circles, projection on midsagittal (X, Z) plane of 4 metal markers on ventral costal diaphragm. Markers on *top* are on origin of diaphragm on rib cage near sternum, and markers on *bottom* are at junction of muscle and central tendon. Closed circles, positions at FRC; thick solid lines, contour of diaphragm; thin lines, displacement of markers with inflation. Note double curvature of surface at FRC + 50% IC and higher.

also zero. The absence of a pressure difference across the diaphragm at TLC implies that the vertical gradients in pleural and abdominal pressure are equal. Because of the fluidlike nature of the abdominal contents, both gradients should be hydrostatic, i.e., 1 cmH₂O/cm vertical height. In a previous study, we have shown that the lung is uniformly expanded at TLC (1). The lung is quite stiff at TLC, such that regional inhomogeneities in pleural surface pressure would not cause a detectable deformation of the lung parenchyma. This observation is therefore not inconsistent with a hydrostatic gradient in pleural pressure at TLC.

None of the regional diaphragm lengths at TLC in

vivo are significantly different from the excised lengths in vitro. The length of the ventral costal diaphragm at FRC in the supine dog agrees closely with the data of Road et al. (12). However, in the present study the excised length for the crural diaphragm is considerably shorter. We considered the possibility that the freshly excised diaphragm might have some "tone" because of ischemia and stimulation from cutting the nerves. We hoped incubation in cold oxygenated Krebs solution would block any neural discharge and prevent hypoxia of central fibers. Nevertheless, we cannot exclude the possibility there was tone in our excised diaphragms or that the cold solution induced shortening of the diaphragms. Therefore the excised lengths are a minimum estimate of the passive unstressed length. The finding that the diaphragm reached the same length at TLC in vivo provides evidence that the diaphragm is not under tension at TLC. Below 50% IC for the ventral costal region in the supine dog and 85% IC for all other regions, the diaphragm is stretched, indicating that it must be under some passive tension.

Many studies have shown that the optimal length (L_o) of the excised diaphragm is very near the length at which passive tension develops (3, 4, 7, 9). However, we do not believe that L_o of the diaphragm in vivo is our unstressed length, which occurs near TLC. In the studies on excised rodent and canine diaphragm strips cited above, the pleural and peritoneal membranes had been removed. It is likely that these tissue membranes represent elastic elements in parallel with the muscle fibers and that they can support passive tension at muscle lengths significantly below L_o . We have no data on the length-tension relationships in the excised diaphragms. In in vitro studies, muscle strips are "cycled" several times to "condition" them before L_o is determined, and the length at which passive tension occurs may be quite different from the unstressed length in this study. Previous estimates of the resting length of the costal diaphragm at FRC in the supine posture range between 95 and 105% of L_o (4, 12), whereas the crural diaphragm tended to be shorter (84% of L_o). Our results (Fig. 5) indicate that the crural

diaphragm stretches ~10% between FRC and RV. We consider it unlikely that the crural diaphragm is shorter than L_0 at RV. However, because we have no data on L_0 in this study, we cannot comment on this apparent inconsistency. Although the evidence is not conclusive, the data in the literature suggest that, in the supine anesthetized dog at FRC, the diaphragm is shorter than L_0 .

De Troyer et al. (2) have shown that expiration is not passive in supine anesthetized dog. Dogs expire below the relaxation volume primarily by activation of the triangularis sterni. Previously, we have reported that the end-expiratory lung volume is lower during spontaneous breathing than during mechanical ventilation in prone and supine anesthetized dogs, consistent with expiratory activity of the triangularis sterni in both postures (6). Yet we found no difference in the length of the diaphragm at end expiration between spontaneous and mechanical ventilation in this study. It is possible that the triangularis sterni acts predominantly on the upper rib cage and has little effect on the abdomen. Our results appear inconsistent with the study of Warner and associates (14), who found large differences in diaphragm position at end expiration between mechanical and spontaneous ventilation. However, muscle lengths were not measured directly in this computerized tomography study.

It seems counterintuitive that the diaphragm would be operating at less than optimal length during spontaneous breathing, but the supine position is not physiological for the dog. However, Newman et al. (11) reported that, at FRC in the prone posture, both costal and crural diaphragm are even shorter. We also found small but significant decreases in the length of the middle costal and crural diaphragm in the prone position (Fig. 3B). In animals that are supported in a sling in the prone position, part of the weight is borne by the rib cage. This should also be true in the standing position, because the forelimbs are coupled to the thorax by muscular attachments to the rib cage. If these forces tend to compress the rib cage and the effect of gravity had an inspiratory effect on the abdomen, there could be shortening of the diaphragm with no change in FRC. This is consistent with the findings of our study. FRC does not change with the induction of anesthesia in prone animals (8), suggesting that diaphragm length may be similar in awake animals. The diaphragm may operate below L_0 in dogs at rest if the muscle is designed to function most efficiently during exercise and if the dog, like humans, reduces end-expiratory volume markedly during exercise.

At FRC, a vertical gradient of Pdi resulting from the difference between the vertical gradients of pleural and abdominal pressures would result in a gradient of passive tension, unless there are regional differences in diaphragm curvature. A vertical gradient in tension would produce a gradient of muscle fiber lengths, unless it were counterbalanced by differences in the passive elastic properties and/or thickness of regions. Although the diaphragm could have uniform muscle stress because of differences in thickness and natural curvature in one horizontal position, it is difficult to imagine a structure that would produce uniform muscle tension both prone and supine. Given nonuniform muscle lengths relative to

the unstressed length in the prone position, one would expect it to be oriented along a hydrostatic gradient and to reverse in the supine position. The results shown in Fig. 5, A and B, in which regional lengths at FRC are expressed as a percentage of their unstressed lengths in vitro, are not consistent with this hypothesis. For example, in the prone posture (Fig. 5B), the crural and dorsal costal diaphragm are stretched most and the middle and ventral regions least. This is the opposite of what would be predicted from a dorsal-to-ventral gradient in Pdi. If the difference in length between the middle and dorsal diaphragm in the prone position was explained by the middle region of the costal diaphragm being stiffer, and thus stretched less by the greater local Pdi, a large difference between middle and dorsal costal behavior would be expected in the supine position. This, however, is not the case (Fig. 5A); therefore the lack of expected gradients in regional lengths cannot be explained entirely by differences of regional diaphragm compliance. We conclude that the absence of an orderly gradient in diaphragm muscle length consistent with the gradient in Pdi must be explained by differences of radii of curvature or by stresses aligned normal to the muscle bundles.

Regional tidal shortening. During mechanical ventilation, the diaphragm length-volume relationship is the same as during stepwise static deflations from TLC, indicating that dynamic factors did not contribute to the behavior of the relaxed diaphragm. With the exception of the most ventral portion of the diaphragm, there is no consistent pattern of regional shortening in either body posture or mode of ventilation. This would be expected if tension in the diaphragm were uniform, all regions were at the same length relative to their optimal length, and there were no consistent topographical patterns of neural activation during quiet breathing. The vertical gradient of Pdi does not preclude the possibility that muscle tension is uniform. A dorsal ventral gradient of membrane thickness could produce equal muscle fiber tension in the face of a gradient of transmural pressure in one position but would exaggerate a gradient in tension when the animal was rotated from prone to supine. Our data indicate that various regions of the diaphragm are not uniformly stretched relative to the excised length in either body position, although the stretch does not directly follow the vertical height. It is not clear what proprioceptive mechanisms would allow neural activation to be modulated to achieve a uniform contraction. A better understanding of the mechanics of the diaphragm and the relationship between muscle tension and Pdi will be required to suggest a mechanical explanation for the absence of vertical gradients of fractional shortening during spontaneous breathing.

Summary. During passive inflation, the ventral costal diaphragm is at its unstressed length from 70 to 100% TLC. Above 70% TLC, the ventral costal diaphragm has a double curvature consistent with its being under no passive tension. The other regions of the diaphragm shorten monotonically to TLC. However, the diaphragm length at TLC is not significantly different from the unstressed length in the excised state. These results imply a hydrostatic gradient of pleural pressure at TLC. At FRC, the diaphragm muscle fibers are not uniformly

stretched, although the relative stretch does not follow a vertical gradient. Despite differences in initial length, there are no consistent differences in fractional shortening during spontaneous ventilation. During mechanical ventilation, the diaphragm shortens less, and there are, again, no consistent regional differences except for the ventral costal diaphragm, which shortens more in the supine animal. These data suggest a complicated relationship between regional muscle tension in the diaphragm and local Pdi and point out a need for more detailed information of anatomy and geometry.

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Address for reprint requests: J. R. Rodarte, Pulmonary Section, 6550 Fannin, Suite 1225, Houston, TX 77030.

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