Effects of body position and lung volume on in situ operating length of canine diaphragm

SUSAN S. MARGULIES, GASPAR A. FARKAS, AND JOSEPH R. RODARTE
Mayo Foundation, Rochester, Minnesota 55905

MARGULIES, SUSAN S., GASPAR A. FARKAS, AND JOSEPH R. RODARTE. Effects of body position and lung volume on in situ operating length of canine diaphragm. J. Appl. Physiol. 69(5): 1702-1708, 1990.—The performance of the diaphragm is influenced by its in situ length relative to its optimal force-generating length ($L_o$). Lead markers were sutured to the abdominal surface of the diaphragm along bundles of the left ventral, middle, and dorsal regions of the costal diaphragm and the left crural diaphragm of six beagle dogs. After 2-3 wk postoperative recovery, the dogs were anesthetized, paralyzed, and scanned prone and supine in the Dynamic Spatial Reconstructor (DSR) at a total lung capacity (TLC), functional residual capacity (FRC), and residual volume (PV). The location of each marker was digitized from the reconstructed DSR images, and in situ lengths were determined. After an overdose of anesthetic had been administered to the dogs, each marked diaphragm bundle was removed, mounted in a 37°C in vitro chamber, and adjusted to $L_o$ (maximum tetanic force). The operating length of the diaphragm, or in situ length expressed as percent $L_o$, varied from region to region at lung volumes studied; variability was least at RV and increased with increasing lung volume. At FRC, all regions of the diaphragm were shorter in the prone posture compared with the supine, but there was no clear gravity-dependent vertical gradient of in situ length in either posture. Because in vitro length-tension characteristics were similar for all diaphragm regions, regional in vivo length differences indicate that the diaphragm’s potential to generate maximal force is nonuniform.

SKELETAL MUSCLES generate maximal force at one length, known as the optimal length ($L_o$). The force-length relationship for muscle dictates that active muscle force generated during isometric tetanic stimulation decreases at lengths above or below $L_o$. The force-generating potential of the in situ diaphragm may be assessed by relating diaphragm length to $L_o$ for a variety of physiological conditions.

Most investigators have confined their studies of diaphragm length changes to one region in the costal and/or crural diaphragm (1-4, 6, 9-11). However, Sprung et al. (12, 13) found variations in diaphragm length and shortening within the costal region, as well as between the costal and crural diaphragm segments.

In this communication, we relate optimal force-generating length $L_o$ to in situ length of three regions in the costal diaphragm and one region in the crural diaphragm. We determine the regional force-generating properties of the canine diaphragm and how they are affected by variations in body posture and lung volume. In addition, we relate the unstressed excised length ($L_{exc}$) to $L_o$ for the different diaphragmatic regions to investigate the passive tension present in the diaphragm.

METHODS

Six bred-for-research beagle dogs (8-11.5 kg) were anesthetized with pentobarbital sodium (30 mg/kg iv). A midline laparotomy was performed. Lead markers (1 mm diam) with a small hole drilled through the center were sutured to the abdominal surface of the left hemidiaphragm. Marker pairs were placed along muscle bundles ~1 cm apart in the ventral (CoV), middle (CoM), and dorsal (CoD) regions of the costal diaphragm, and in the crural (Cr) diaphragm. Where possible, three or more markers were placed along a muscle bundle from origin to insertion to yield information from more than one marker pair for that region.

After a 2- to 3-wk postoperative recovery period, the dogs were anesthetized with pentobarbital sodium (30 mg/kg) and intubated with an endotracheal tube. The depth of anesthesia was considered sufficient when the pupillary reflexes were abolished and the dog was able to breathe unassisted by a ventilator. The dogs were positioned prone in the Dynamic Spatial Reconstructor (DSR), a unique fast volumetric computed tomographic-imaging device, and supported in a sling with forelimbs bent slightly under the chest in a neutral position. Between DSR scans they were maintained on a ventilator (Harvard pump) at 15-20 breaths/min with a 200-ml tidal volume. Airway pressure (Pao) was monitored at the oral end of the endotracheal tube with a Validyne DP9 pressure gauge. Inspiratory capacity (IC) was determined by inflating the lungs from functional residual capacity (FRC) to total lung capacity (TLC, defined as Pao = 30 cmH2O) with a calibrated supersyringe connected to the endotracheal tube.

The lungs were inflated with the supersyringe to TLC, the endotracheal tube was clamped closed, and the thorax was scanned for 4 s. The clamp was then released, the lungs were allowed to deflate passively to FRC (Pao = 0 cmH2O), and the thorax was scanned again. Finally, the lungs were deflated to residual volume (RV, when Pao was approximately -30 cmH2O), the endotracheal tube was clamped, and the thorax was scanned for the third time. The dogs were then paralyzed with a short-acting agent (20 mg succinylcholine), and the scan sequence was repeated twice. The dog was rotated to the supine...
posture, the forelimbs were again placed in a slightly bent neutral position, and the dog was allowed to recover from the paralysis. The scan sequence was repeated once in the supine posture, then the dog was paralyzed (3 mg pancuronium), and finally the scan sequence was repeated two more times.

At the completion of the DSR study, each dog was administered an overdose of pentobarbital sodium, the abdomen was opened, and the diaphragm was removed entirely. The excised diaphragm was quickly placed in cooled oxygenated Krebs solution (4°C) containing (in mM) 137 NaCl, 4 KCl, 1 MgCl2, 1 KH2PO4, 12 NaHCO3, 2 CaCl2, and 6.5 glucose. The four diaphragm segments containing the rows of markers were removed from the intact diaphragm and inspected to ensure that the lead markers were correctly oriented along the long axis of the fibers. On this basis, the CoM row from one dog and the CrO row from another dog were eliminated from the study. Individual strips were immersed in a film of oxygenated Krebs solution at room temperature, and the intermarker distance between each pair of adjacent markers was measured in triplicate with a micrometer. After \( L_\infty \) had thus been determined, each bundle was returned to the cooled oxygenated Krebs solution.

To determine each segment's \( L_\infty \), the bundles were placed in an in vitro muscle bath that was filled with Krebs solution, maintained at 37°C, and perfused with 95% O2-5% CO2. After \( L_\infty \) had thus been determined, each bundle was returned to the cooled oxygenated Krebs solution.

The bundle was removed from the apparatus and blotted dry, the markers were removed, and the bundle was weighed on an analytic balance. Muscle cross-sectional area was estimated by dividing the muscle mass by its length and density (1.056 g/cm³). Average cross-sectional area at \( L_\infty \) was \( 0.12 \pm 0.03 \) cm². Muscle tension was expressed as force per unit cross-sectional area in newtons per centimeters squared.

The elapsed time between the death of the dog and in vitro testing of the last diaphragm bundle was ~2 h. Anoxia reduces the maximum tension generated by a muscle in vitro, and we were concerned that the delay between death and isometric testing might also affect \( L_\infty \). In a separate study, four mongrel dogs (15–25 kg) were anesthetized and maintained on a ventilator while a 5-mm-wide strip extending from origin to insertion was excised from the CoM region. Two 1-mm lead markers were sutured to the diaphragm along a muscle bundle ~1 cm apart. \( L_\infty \) was measured as described earlier, and the bundle was mounted in the in vitro chamber maintained at 37°C. The initial \( L_\infty \) was measured at maximum tension using the same technique described earlier, and the initial maximum tension \( P_0 \) was recorded. The muscle bundle was removed and placed in hypoxic Krebs solution at room temperature. A second bundle was removed from the contralateral hemidiaphragm, and the process was repeated. After ~1 h in the hypoxic Krebs solution, the bundles were remounted in the in vitro chamber and equilibrated, and \( L_\infty \) and maximum tension were reevaluated.

### Data analysis

The DSR scans were reconstructed into a 128 × 128 × 101 array of volume elements or voxels. Each voxel is a cube, 1.4 mm on a side, that represents the X-ray density information for a specific location in the image. The right white lead markers were easily recognizable on the diaphragm surface in every reconstructed image. By use of customized digitizing program, the markers were located and coordinates of the markers were determined (±1 voxel), and the in vivo intermarker distances between adjacent markers were calculated. The volume sequence was performed twice for each body position during paralysis, and the intermarker distances for each marker pair from the two scans were averaged for each lung volume step.

The three micrometer measurements of each in vitro marker pair were averaged. The in vivo and in vitro intermarker lengths were measured on separate occasions.

The in vitro intermarker length at \( L_\infty \) of each marker pair was compared with the in vivo paralyzed and anesthetized length of the same marker pair determined from the DSR scan data. Intermarker length at TLC, FRC, and RV in the supine and prone dog was normalized by the corresponding intermarker distance at \( L_\infty \). If more than one marker pair was studied in a row in a dog, \( L/ L_\infty \) for the marker pairs were averaged. Sprung et al. (12) reported variability in shortening along a row of markers, but it was not systematic. Then the ratios were averaged across dogs in each of the four regions studied. Values are reported as means ± SD. Paired t tests were used to compare the diaphragm lengths in the prone and supine postures, with \( P < 0.05 \) significant. Because some regions were eliminated from the study, it was impractical to use analysis of variance to investigate regional variations in \( L_\infty \) or \( L_\infty \). Instead, multiple paired t tests were performed, with \( P < 0.01 \) significant.

### Results

There were no significant differences between diaphragm lengths with and without paralysis, so we report only the data for the paralyzed condition. Close agreement between our findings in the anesthetized dog and after paralysis indicates that there is little tonic muscle activity in the anesthetized diaphragm during passive lung inflation.

Means ± SD across the dogs for the ratio between diaphragm lengths with and without paralysis, so we report only the data for the paralyzed condition. Close agreement between our findings in the anesthetized dog and after paralysis indicates that there is little tonic muscle activity in the anesthetized diaphragm during passive lung inflation.

### Table 1. Diaphragm length expressed as percentage of \( L_\infty \)

<table>
<thead>
<tr>
<th>Diaphragmatic Region</th>
<th>TLC</th>
<th>FRC</th>
<th>RV</th>
<th>TLC</th>
<th>FRC</th>
<th>RV</th>
</tr>
</thead>
<tbody>
<tr>
<td>CoV</td>
<td>78±13†</td>
<td>83±15†</td>
<td>96±12</td>
<td>78±13†</td>
<td>84±11†</td>
<td>93±8</td>
</tr>
<tr>
<td>CoM</td>
<td>74±10†</td>
<td>88±10*</td>
<td>104±8</td>
<td>84±6†</td>
<td>100±7†</td>
<td>104±5</td>
</tr>
<tr>
<td>CoD</td>
<td>58±8†</td>
<td>86±10†</td>
<td>104±12</td>
<td>87±10†</td>
<td>90±11†</td>
<td>97±16</td>
</tr>
<tr>
<td>Cr</td>
<td>69±7†</td>
<td>78±10†</td>
<td>102±12</td>
<td>99±5†</td>
<td>91±9†</td>
<td>99±11</td>
</tr>
</tbody>
</table>

Values are means ± SD for 6 dogs expressed as percentage of optimal length (\( L_\infty \)). TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume. CoV, costal ventral; CoM, costal middle; CoD, costal dorsal; Cr, crural. * \( P < 0.05 \) prone vs. supine; † \( P < 0.05 \) vs. \( L_\infty \).
The comparison between in vivo length at FRC (L_{FRC}) and in vitro L_o of the four diaphragmatic regions for individual dogs is presented in Fig. 1 (n = 6 in CoV, n = 5 in all other regions). For the costal diaphragm, supine L_{FRC} averaged 84 ± 11, 100 ± 7, and 90 ± 11% L_o for the CoV, CoM, and CoD regions, respectively, and 91 ± 9% L_o for the Cr. At supine FRC, only the CoV row was operating at a length significantly shorter (P ≤ 0.05) than L_o.

In the prone position compared with the supine, L_{FRC} was shorter in all rows, but the difference reached statistical significance only for the CoM and Cr regions. For the costal diaphragm, prone L_{FRC} averaged 83 ± 15, 88 ± 10, and 86 ± 10% L_o for CoV, CoM, and CoD regions, respectively, and 78 ± 10% L_o for Cr. In the prone posture the CoV, CoD, and Cr regions were significantly shorter (P ≤ 0.05) at FRC than L_o.

Figure 2, top, shows the effects of lung volume on the in vivo operating length expressed as a percentage of L_o in each of the diaphragmatic regions studied. The diaphragm consistently shortens between RV and TLC, ranging from 18–46% L_o in the prone position to 15–30% L_o in the supine. The CoV row appears unique in that it shortens very little in both positions between FRC and TLC; all regions shorten from RV to TLC. Diaphragm length shows regional variability at each lung volume in the upper panels, but the rank order changes with lung volume and posture, and there is no obvious gravity-dependent gradient in diaphragm length. Figure 2, bottom, presents the length of the diaphragm normalized to the FRC length for that posture.

The means ± SD across the dogs for L/L_o were calculated for each of the four regions at TLC, FRC, and RV, and the data are shown in Table 2. At FRC and RV in both postures, all regions are longer than L_o; at TLC only the CoM region in the supine dog differs significantly from L_o.

The means ± SD across dogs of L_o/L_o in the CoV, CoM, CoD, and Cr diaphragm were 68 ± 4, 67 ± 2, 68 ± 5, and 68 ± 5%, respectively. Figure 3 displays this nearly constant relationship between the in vitro measurements of L_o and L_o. The mean and SD of L_o/L_o across the four regions was 68 ± 4%.

In the first in vitro study, peak active muscle tension averaged 15.5 ± 4.8 N/cm² (1.58 ± 0.49 kg/cm²). The second study compared L_o of a freshly excised muscle bundle with that of the same bundle after a period of anoxia. L_o was not significantly different from the origin.
Tables

**TABLE 3. Studies comparing L\textsubscript{FRC} in supine posture with optimal length**

<table>
<thead>
<tr>
<th>Study</th>
<th>CoM Region</th>
<th>Crural Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present study</td>
<td>106±3 (CoM)</td>
<td>91±4</td>
</tr>
<tr>
<td>Farkas and Rochester (2)</td>
<td>95±4</td>
<td>84±8</td>
</tr>
<tr>
<td>Farkas and Rochester (3)</td>
<td>97±2</td>
<td>89±3</td>
</tr>
<tr>
<td>Kim et al. (6)</td>
<td>94±3</td>
<td></td>
</tr>
<tr>
<td>Road et al. (11)</td>
<td>105</td>
<td>92</td>
</tr>
</tbody>
</table>

Values are means ± SE expressed as %L\textsubscript{FRC}, L\textsubscript{optimal}, length at FRC, CoM, costal middle.

Conal L\textsubscript{o} (102 ± 5\%L\textsubscript{o}), even though P, decreased 60 ± 5\% after the anoxia from its initial value of 27.1 ± 3.2 N/cm\textsuperscript{2} (2.76 ± 0.33 kg/cm\textsuperscript{2}) to 16.3 ± 2.3 N/cm\textsuperscript{2} (1.66 ± 0.23 kg/cm\textsuperscript{2}). Individual L\textsubscript{ex} and L\textsubscript{o} data for the fresh bundles are shown in Fig. 3 and identified as study 2.

**DISCUSSION**

Our study compares in vitro optimal length of the diaphragm to the in vivo passive muscle length in the intact dog and has several advantages over other studies reported in the literature. First, the lead markers were implanted several weeks before the study date so that the data could be obtained from intact dogs and the results would not be affected by laparotomy or acute minor surgery to the diaphragm. Second, several regions of the diaphragm were studied simultaneously to focus on any in vivo or in vitro regional variations in muscle bundle length and force-generating capacity. Third, passive diaphragm length was measured at lung volumes studied ranging from RV to TLC, in both supine and prone body postures. The following discussion compares these results with those of other studies in the literature. Tables 3 and 4 present pertinent measurements from this study and others to facilitate cross-comparisons. For the purpose of this discussion, we define L\textsubscript{o} as the length at which the diaphragm generates maximum active force.

**Table 4. Studies comparing L\textsubscript{TLC} and L\textsubscript{RV} with L\textsubscript{FRC} in supine posture**

<table>
<thead>
<tr>
<th>L\textsubscript{TLC}/L\textsubscript{FRC}</th>
<th>L\textsubscript{RV}/L\textsubscript{FRC}</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Costal</td>
</tr>
<tr>
<td>Present study</td>
<td>84 (CoM)</td>
</tr>
<tr>
<td>Farkas and Rochester (2)</td>
<td>63</td>
</tr>
<tr>
<td>Newman et al. (9)</td>
<td>54-85</td>
</tr>
<tr>
<td>Decramer et al. (1)</td>
<td>67</td>
</tr>
<tr>
<td>Hubmayr et al. (5)</td>
<td>78</td>
</tr>
<tr>
<td>Sprung et al. (13)</td>
<td>82</td>
</tr>
</tbody>
</table>

Values are means expressed as %L\textsubscript{FRC}, L\textsubscript{TLC}, length at total lung capacity; L\textsubscript{RV}, length at residual volume.

**Diaphragm length at FRC.** The force-generating capacity of the diaphragm at FRC is given by the ratio L\textsubscript{FRC}/L\textsubscript{o} (Table 1 and Fig. 1). In the prone dog, all regions except CoM were significantly shorter (P ≤ 0.05) than L\textsubscript{o}. However, in the supine dog only the CoV region was significantly shorter than L\textsubscript{o}. We noted no gravity-dependent regional variation in L\textsubscript{FRC}/L\textsubscript{o} in either posture. In the prone posture there were no significant regional differences in the force-generating capacity of the diaphragm. In the supine posture only the CoV-CoM comparison reached significance. Table 3 presents our findings in the supine posture with L\textsubscript{FRC}/L\textsubscript{o} ratios reported by other investigators. To our knowledge, no studies have compared L\textsubscript{o} and in vivo diaphragm lengths in prone animals.

Using sonomicrometers, Farkas and Rochester (2, 3) found that L\textsubscript{FRC}/L\textsubscript{o} was larger in CoM than in Cr and that only Cr was significantly shorter than L\textsubscript{o} (2). Similarly, Kim et al. (6) found that the CoM was operating at ~94\% of its maximum force-generating length at FRC. Road et al. (11) did not measure L\textsubscript{o} directly but estimated it to be the in situ length at which maximum transdiaphragmatic pressure was generated in response to phrenic stimulation. They found L\textsubscript{FRC}/L\textsubscript{o} to be larger in CoM than in Cr. Two of the previous studies (6, 11) used indirect methods to measure L\textsubscript{o} in dogs with open abdomens, and all previous studies (2, 3, 6, 11) were acute in nature. The present study confirms the previous findings in a chronic preparation. To summarize, in the supine dog the costal region at FRC operates closer to its optimum force-generating capacity than the Cr.

**Changes in diaphragm length with lung volume.** The length of a region of the diaphragm in vivo compared with its L\textsubscript{o} can be considered an indicator of the force-generating potential of that region. Muscle generates maximum force at L\textsubscript{o}, and force production becomes increasingly attenuated as length increases or decreases from L\textsubscript{o}. Table 1 and Fig. 2, top, show that as lung volume decreases, the in vivo length in every region of the diaphragm increases from a length at TLC that is significantly below L\textsubscript{o} to a length at RV that is not significantly different from L\textsubscript{o}. As described earlier for FRC, we compared the four regions at TLC and at RV in multiple paired t tests with a level of significance equal to $P \leq 0.01$. At TLC, only the CoM-CoD regional length differences reached significance in the prone dog, whereas none reached significance in the supine dog. At RV, there was no significant regional variation in diaphragm length.
in the prone dog, and only the CoV-CoM regional differences reached significance in the supine dog.

Farkas and Rochester (2) reported the ratio between in vivo length and $L_o$ in supine dogs at RV, FRC, and TLC ($P_{ao} = 30\, \text{cmH}_2\text{O}$) as 102, 95, and 60%, respectively, in the costal diaphragm (Cos) and 88, 84, and 66% in the Cr. Our findings in the supine dog indicate the length ratios average 104, 100, and 84%, respectively, in the CoM and 99, 91, and 69% in the Cr. The two studies show comparable results, with the exception of TLC in the Cos and RV in the Cr. We have no explanation for the differences in these findings.

Most investigators choose to normalize the length of the diaphragm to its $L_{FRC}$ in that posture. Figure 2, bottom, displays our data in this format, and Table 4 presents our data for the CoM and Cr regions along with the findings of other studies of the supine dog. When the results of Farkas and Rochester (2) are normalized to $L_{FRC}$, our results are in agreement except at TLC in the Cos region. In an acute sonomicrometer crystal study, Newman and colleagues (9) inflated the lungs of supine dogs to TLC (transpulmonary pressure = 25 cmH$_2$O) and found considerable variability across dogs in costal and crural diaphragm length. Our results fall within the upper portion of their data range, perhaps because of differences in our definition of TLC. In another acute sonomicrometer crystal study, Decramer et al. (1) measured Cos and Cr diaphragm length during a passive inflation from RV to TLC and found that both regions shortened as lung volume increased. Their results are comparable to ours at RV, but they found that the costal diaphragm was considerably shorter at TLC, more in agreement with Farkas and Rochester (2) than the present study. Hubmayr et al. (5) sutured lead markers along muscle bundles in four regions of the diaphragm (CoV, CoM, CoD, and Cr) in an acute study of supine dogs, inflated the dogs' lungs from FRC to TLC ($P_{ao} = 30\, \text{cmH}_2\text{O}$), and found that whereas all regions shortened as lung volume increased, the CoV region reached its minimum length at a volume below TLC and the other three regions shortened in a linear manner with lung volume. Our results are similar to those of Sprung et al. (13), who found $L_{FRC}$ was 118-133% $L_o$ in Cos and 135% in the Cr of the prone dog and 121-137% $L_o$ in the Cr of the supine dog. Road and colleagues (11) also found that $L_{FRC}$ was greater than 100% $L_o$. They reported $L_{FRC}/L_o$ in the supine dog only; 126% $L_o$ in the CoM and 115% in the Cr.

Is there passive tension in the diaphragm even at TLC? Sprung et al. (13) report that in both prone and supine dogs, length $L_o$ at TLC was not significantly different from $L_o$ in any diaphragmatic region. In the present study the only region that was significantly different from its $L_o$ was the CoM region in the spine position. Although the CoM and Cr regions were significantly longer in the supine posture at TLC, because the relationship between passive muscle tension and length is nonlinear, the tension at TLC is quite small in magnitude and similar in both body positions. We therefore conclude that whereas the diaphragm has passive tension at FRC, at TLC the diaphragm is stress free.

Changes in diaphragm length at FRC with posture. All regions are longer at FRC in the supine posture than in the prone posture, but only the changes in the CoM and Cr regions reach statistical significance (Table 1, Fig. 1). A decrease in lung volume in the supine posture compared with the prone position would be associated with consistently longer diaphragm lengths in the supine posture. We did not attempt to keep lung volume constant with body rotation in our study. However, Lai et al. (7) and Sprung et al. (13) found no difference in lung volumes between the prone and supine postures at either TLC or FRC in anesthetized dogs. Differences in diaphragm length due to postural changes could be attributed to a redistribution of volume between the chest wall and abdomen. Margulies and Rodarte (8) reported that when dogs are rotated from the prone to the supine posture while supported in a sling, the ventral portion of the diaphragm displaces caudad and dorsal and the dorsal regions move cephalad. These movements are consistent with larger increases in muscle length in the dorsal region compared with the ventral regions of the diaphragm (Table 1).

Other investigators have compared $L_{FRC}$ in the prone
and supine body positions \( (L_{\text{prone}}/L_{\text{sup}}) \). In the present study, \( L_{\text{prone}}/L_{\text{sup}} \) was 88% in the CoM and 86% in the Cr, and both ratios were significantly less than 1.0. Newman and colleagues (10) also document significantly shorter lengths in both regions in the prone posture, with the Cr region shortening more than the Cos region \( (L_{\text{prone}}/L_{\text{sup}} \text{ at end expiration} = 92 \text{ and } 89\% \text{ in the Cos and Cr regions, respectively}) \). Sprung et al. (13) found that of the four diaphragmatic regions, only the shortening in the CoM and Cr regions reached significance \( (L_{\text{prone}}/L_{\text{sup}} \text{ equal to 86 and 93\% in the CoM and Cr regions, respectively}) \).

Implications for spontaneous breathing in awake dogs.

All measurements presented in this report are for the passive diaphragm, but we would like to speculate on the implications of these results for diaphragm shortening during spontaneous breathing. Muscle shortening depends on, among other factors, the force-per-unit area generating capacity of a muscle, which is determined by the initial muscle length \( (L/L_0) \).

Before extrapolating our findings in regional diaphragmatic force-generating capacity to muscle shortening in awake dogs breathing spontaneously, however, we must first address two issues: the effect of anesthesia and paralysis on muscle length and the relationship between muscle length and active muscle shortening. In this study, the in vivo length measurements were determined in dogs anesthetized with pentobarbital sodium, with and without paralysis. The addition of paralysis did not alter the diaphragm lengths significantly from those during anesthesia alone. In addition, our results in paralyzed dogs are similar to those of Sprung et al. (13) in anesthetized dogs. We conclude that paralysis has little effect on diaphragm length. In contrast, it is unclear whether muscle length in the awake dog changes with the induction of anesthesia.

Fitting et al. (4) evaluated diaphragm length in awake dogs and after the induction of pentobarbital anesthesia. They found that whereas the Cr length increased 7–8% with anesthesia, the Cos length remained unchanged. It was concluded that the lengthening was due to a loss of diaphragmatic tone after anesthesia and that it was more pronounced in the Cr than in the Cos segment. On the other hand, Lai et al. (7) found no significant alterations in TLC or FRC during the induction of anesthesia. No measure of diaphragm length was attempted, but it should be noted that the relationship between lung volume and diaphragm length may change in awake animals when other muscles are acting in the respiratory system.

Shortening is not necessarily proportional to the diaphragm's contribution to ventilation. Hubmayr et al. (5) showed that muscle shortening and diaphragm geometry together affect the ability of the diaphragm to produce a change in transdiaphragmatic pressure. In addition, the tension, or force per unit length, in the muscle will influence the shape of the diaphragm and transdiaphragmatic pressure. Tension is proportional to diaphragm thickness and the force per unit area the muscle generates. Because there is no information in the literature at this time about the regional variation in diaphragm thickness, we focus on muscle shortening, with the knowledge that it is not the only determinant of diaphragmatic contribution to ventilation.

Our results at FRC indicate that the force-generating capacity of the diaphragm is nonuniform, although regional differences rarely reach significance. In the supine dog the CoV region would shorten least because it is the only region not at its \( L_0 \), and in the prone dog the CoV, CoD, and Cr would shorten less than the CoM region for the same reason.

Neural activation of the muscle and the afterload of the muscle also influence muscle shortening. Regions receiving more neural stimulation will contract more than those regions activated to a lesser degree. Decreased afterload would also increase muscle shortening. Despite the complex interplay among the factors affecting shortening, Sprung et al. (12, 13) found no systematic regional variation in fractional shortening in prone and supine spontaneously breathing anesthetized dogs. Because regional variations in neural activation probably do exist during spontaneous breathing, it is difficult to extend our results for the passive diaphragm to the contracting diaphragm in the awake dog.

Conclusions. The ratio \( L/L_0 \), an indicator of the diaphragm's potential to generate maximal force, is not uniform over the diaphragm, and therefore all regions of the diaphragm are not at the same force-generating length. Regional variability in length is least at RV, when all regions are approximately at \( L_0 \), and increases with lung volume. In addition, there is no clear gravity-dependent vertical gradient in diaphragm length. The regional differences in operating length did not reach statistical significance, perhaps indicating that the diaphragm operates as a unit at the lung volumes studied, with no single region at a more advantageous length than the others.

As body position is changed from supine to prone at TLC or FRC, the diaphragm shortens in all regions but reaches statistical significance in only the CoM and Cr regions. There is no posture-dependent shortening at RV.

The relationship between \( L_0 \) and \( L_{\text{prone}} \) is independent of region, and therefore \( L_0 \) can be estimated from \( L_{\text{prone}} \).