Effect of Anesthesia on Canine Diaphragm Length

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General anesthesia has been shown to induce a cephalad shift of the end-expiratory position of the diaphragm in recumbent human subjects. The authors used the technique of sonomicrometry in chronically instrumented dogs to measure the length changes occurring in the costal and crural diaphragm during anesthesia. Seven dogs were studied in lateral decubitus; first awake, and then during pentobarbital anesthesia. The end-expiratory length (LFRG) of the crural segment increased gradually and reached a plateau after 30 min of anesthesia. Costal LFRG did not change. The results were similar when the hemidiaphragm under study was placed in a gravity-dependent or in a non-dependent position. In the awake state, variable levels of post-inspiratory or tonic diaphragmatic EMG activity were observed, which disappeared during anesthesia. The authors conclude that anesthesia induces a 7–8% increase in end-expiratory length of the crural, but not of the costal, diaphragm. This selective adjustment is not due to a pressure gradient effect, but is compatible with a loss of tone in the crural diaphragm. (Key words: Lung; diaphragm length; FRC. Measurement techniques: EMG; sonomicrometry. Respiratory muscle tone.)

A REDUCTION IN functional residual capacity (FRC) has been repeatedly observed in humans during general anesthesia.1-9 Although the exact mechanism underlying this change in lung volume is still unknown, it has been proposed that the initial effect of anesthesia is to decrease the outward recoil forces of the chest wall.5 Such an alteration of the pressure-volume characteristics of the chest wall could be explained by a loss of muscle tone, as suggested by the fact that FRC does not decrease further when paralysis is added to anesthesia.2,5 In fact, both a reduction in internal rib cage dimensions5 and a cephalad shift of the diaphragm5,6 have been observed with the induction of anesthesia.

Using fluoroscopy, Froese and Bryan demonstrated that general anesthesia induces a cephalad shift of the end-expiratory position of the diaphragm.6 This displacement was predominant in the dependent parts of the muscle. However, this technique provides only a lateral view of the diaphragm silhouette and does not quantitate the change in muscle length. Furthermore, it does not dissociate the contribution of the two segments of the diaphragm, costal and crural, which are distinct muscles and may, therefore, react differently to anesthesia.7

The length of respiratory muscles can be measured accurately with the method of sonomicrometry.8 Using this technique in chronically instrumented dogs, we measured the length of costal and crural diaphragm in the awake state and during anesthesia.

Methods

MODEL

Seven mongrel dogs weighing 20–30 kg were instrumented during halothane anesthesia. Through a midline laparotomy, two pairs of piezoelectric crystals were implanted between the muscle fibers of the left hemidiaphragm, one pair in the costal segment and one pair in the crural. Each pair was implanted along the same fiber bundle, 15–25 mm apart. Fine wire electromyogram (EMG) electrodes were implanted in adjacent areas of each segment. A 5 cm long latex balloon attached to a polyethylene catheter was placed in the mid-abdomen, close to the anterior wall. The abdomen was closed and the wires and catheter were externalized through a subcutaneous tunnel. During the same procedure, a chronic tracheostomy was performed. The dogs were then allowed to recover from the anesthesia and the operation. After an initial period of rapid shallow breathing and diaphragmatic inhibition, the contractility of the diaphragm increased progressively and reached a plateau at the 10th postoperative day. The recovery course of diaphragmatic function after implantation and the validation of the model have been described previously.9 The present study was performed, on the average, on the 12th postoperative day.

TECHNIQUES

The piezoelectric crystals were connected to a sonomicrometer (Triton Technology, San Diego, CA) via fine isolated wires. The sonomicrometer measures accurately and continuously the distance between the crystals of each pair on the basis of the transit time of ultrasonic waves propagating from one crystal to the other. The application of sonomicrometry to the measurement of diaphragm length has been described previously.8

The EMG was recorded and amplified (TECA TE4, White Plains, NY), band-pass filtered between 100 and
600 Hz, rectified and integrated by an RC circuit with a 100 ms time constant. Inspiratory flow was measured with a pneumotachograph (Fleisch® no. 3) connected to the endotracheal cannula. Tidal volume was measured as the integration of flow over time (Respiratory Integrator®, Hewlett-Packard, Waltham, MA). During the measurements, the abdominal balloon was filled with 1.0 ml of air. All measurements were recorded on an eight-channel paper recorder. In some dogs, EMG and flow signals were recorded on a tape recorder.

**_protocol**

Awake recordings were performed with the dogs in the right lateral decubitus (RLD) position. Before starting the recording, time was allowed for each dog to achieve a breathing pattern which was regular and similar to that recorded on the previous days. After the awake recording had been completed, the dogs were anesthetized with an intravenous injection of pentobarbital sodium (20 mg · kg⁻¹). Care was taken to keep the anesthetized dogs in exactly the same position as they had been in when awake. The measurements were recorded continuously during the first 30 min of anesthesia.

In three dogs, the recordings in the awake state were made first with the dogs in the left lateral decubitus (LLD) position, and then in the RLD position. Anesthesia was then induced in the same RLD position. After 30 min of anesthesia, the measurements were made with the dogs again in the LLD position, within 5 min after the position change.

In three dogs, the anesthesia protocol was performed twice. On the first occasion, the dogs were studied in the RLD position, first awake and then anesthetized. On the second occasion, they were studied in the LLD position, first awake and then anesthetized.

The crystals being implanted in the left hemidiaphragm, the non-dependent diaphragm was studied in the RLD position, and the dependent diaphragm in the LLD position (fig. 1).

**Analysis of Results**

The distance between crystals at end expiration was termed LFRC. This intertransducer distance reflects the change in length of the whole fiber, since the magnitude of contraction has been shown to be uniform along a fiber. In each position, the value of LFRC was normalized using the awake state as 100%. Measurements were made on 20 breaths in the awake state and on 10 breaths every 5 min during anesthesia. Reported values are means ± SD. In the RLD position (n = 7), the difference in LFRC of each segment of the diaphragm between the awake state and after 30 min of anesthesia was analyzed with the Wilcoxon Signed-Rank test for two groups arranged as paired observations.

**Results**

**RLD Position (n = 7)**

Breathing frequency was 15.0 ± 3.1 breaths per min in the awake state, and 10.1 ± 3.9 breaths per min after 30 min of anesthesia. Tidal volume was 0.255 ± 0.060 l in the awake state, and 0.316 ± 0.105 l after 30 min of anesthesia.

Costal diaphragm LFRC did not change significantly with anesthesia, increasing by 0.8 ± 1.8% after 30 min (P > 0.1). Crural diaphragm LFRC increased gradually during anesthesia, approaching a plateau after 30 min (fig. 2). At this time, crural LFRC had increased by 7.6 ± 3.4%, which represents a significant change from the awake state (P < 0.01).

**Effect of Position on Diaphragmatic Fiber Length**

In the same anesthesia (n = 3). In the awake state with dogs in the LLD position, costal LFRC was 3.7 ± 1.0% longer and crural LFRC 1.7 ± 0.3% longer than in the RLD position.

With the awake LFRC normalized as 100% in each position, comparisons were made between the LFRC in the awake state and after 30 min of anesthesia. Costal LFRC decreased by 0.5 ± 0.9% in RLD, and by 2.1 ± 1.6% in LLD. Crural LFRC increased by 6.8 ± 5.8% in RLD, and by 6.7 ± 4.3% in LLD (fig. 3). Thus, crural LFRC increased and costal LFRC did not in the two positions.
In separate anesthetics (n = 3). Comparisons were made between the LFRG in the awake state and during anesthesia, with the dogs in both LLD and RLD positions. After 30 min, costal LFRG had decreased by 0.3 ± 0.9% in RLD, and by 0.6 ± 1.2% in LLD. Crural LFRG increased gradually in both positions to reach a plateau after 30 min. At that time, the increase was 6.8 ± 3.8% in RLD, and 9.9 ± 7.7% in LLD (fig. 4). Thus again, crural LFRG increased and costal LFRG did not in the two positions.

Electromyography

The EMG could be measured in the costal and crural segments in three dogs, but only in one of the segments in four dogs. Therefore, a quantitative comparison of the costal and crural EMG patterns was not undertaken. In the awake state, it was found in every animal that the electrical activity of the diaphragm persisted during part of expiration, as measured from flow. As can be seen in figure 5, the EMG activity increased during inspiration and then decreased very gradually over most of the expiratory time. In some breaths, this post-inspiratory activity persisted even until the onset of the next inspiration. In contrast, in every animal, the EMG activity ceased abruptly at the end of inspiration during anesthesia. This change usually occurred within the first breath following induction of anesthesia.

Abdominal Pressure

Abdominal pressure could be measured successfully in four dogs. From the awake state to anesthesia in the same position, the end-expiratory abdominal pressure decreased minimally in two dogs and did not change in the other two dogs. The mean change was -0.3 ± 0.4 cm H₂O.

Discussion

A cephalad displacement of the end-expiratory position of the diaphragm has been previously documented during anesthesia in human subjects, both by fluoroscopy and by computerized tomography. The present study provides, for the first time, a direct measurement of the length changes occurring in the diaphragm with the induction of anesthesia.

The results show a clear difference between the two segments of the diaphragm during anesthesia, the crural segment increasing its end-expiratory length up to +7.6% after 30 min, whereas the costal segment length did not change. To interpret our data, we must take into account the observation of Froese and Bryan that the cephalad shift of the diaphragm occurring with anesthesia or with paralysis predominates in the dependent parts of the muscle. The authors explained this effect by the difference in gradients of hydrostatic pressure existing across the diaphragm in recumbent positions. In the awake state, pressure increases by 0.2 cm H₂O per cm of height on the thoracic side, and by 1.0 cm H₂O per cm of height on the abdominal side. Therefore, in horizontal positions, higher transdiaphragmatic pressures (Pdi) must exist.

![Diaphragm resting length (LFRG)](image)

**FIG. 2.** End-expiratory length (LFRG) of costal and crural diaphragm in the awake state and during anesthesia. Right lateral decubitus position. Values are means ± SE. The change in LFRG between the awake state and after 30 min of anesthesia is significant in the crural segment (P < 0.01), and not significant in the costal segment.

![Diaphragm resting length (LFRG)](image)

**FIG. 3.** End-expiratory length (LFRG) of costal and crural diaphragm in the awake state and after 30 min of anesthesia. Comparison between the nondependent side (right lateral decubitus) and the dependent side (left lateral decubitus) during the same anesthesia. Values are means ± SE.
across the dependent parts than across the non-dependent parts of the diaphragm. This vertical gradient of Pdi could explain that the cephalad displacement predominates in the dependent parts when all activity is suppressed in the diaphragm. However, it is unknown if the gradient of pleural pressure remains unchanged or increases during anesthesia.

In our study, the dogs were lying in the RLD position, i.e., in a situation where the left costal segment was in the least dependent position and the crural segment in a more dependent position (fig. 1, left schema). Therefore, the possibility existed that the selective lengthening of the crural segment observed during anesthesia was due to a vertical pressure gradient across the diaphragm. To distinguish between a postural effect and a segmental effect, we studied three dogs in both lateral positions. In the LLD position, the left costal segment is in the most dependent position and the crural is in a relatively less dependent position (fig. 1, right schema). If the adjustments in the segmental end-expiratory lengths were due to a pressure gradient effect, the pattern should be reversed in LLD, i.e., costal LFRC should increase more than crural LFRC during anesthesia. This was not the case; the crural segment always lengthening, and the costal not lengthening, regardless of the position. This result was found whether the two positions were studied during the same anesthesia or each position was studied during a separate anesthesia. We conclude, therefore, that the difference in length change between costal and crural diaphragm during anesthesia is not due to a postural effect, but to distinct segmental properties. These results do not contradict with those of Froese and Bryan. Indeed, fluoroscopy revealed a predominant shift of the diaphragm silhouette in the dependent areas, but does not allow interpretation of the contribution of each segment to this movement.

The cephalad shift of the diaphragm occurring with anesthesia has been attributed to a loss of tone in this muscle. The presence of tone in the diaphragm is debated because it is impossible to distinguish it with certainty from a noisy signal. Muller et al. addressed this problem by studying step changes in baseline EMG activity between the awake state and REM sleep or anesthesia. They observed consistently more baseline activity in the awake state than in the other two conditions, which suggested the presence of tone rather than noise. Our findings are similar to those of Muller et al., in that we observed post-inspiratory activity and, sometimes, tonic activity in the awake state, which disappeared during anesthesia. Moreover, our EMG signals were recorded via implanted, rather than surface, electrodes, and were thereby free of interference from other muscles. If the selective lengthening of the crural segment was due to this mechanism, the loss of tone should be more important in this segment than in the costal segment. The small number of complete EMG recordings did not allow us to quantitate differences between the two segments. However, some reasons support the hypothesis that tonic activity may be more important in the crural than in the costal segment. When muscle spindles have been described in the diaphragm, they were found in the crural segment, either exclusively or predominantly. The spindles play an important role in regulating tonic activity by their facilitating effect on alpha-motoneurons, and their activity varies in proportion to the level of cortical activity.

It can be argued that our results might be due to the light level of anesthesia, rather than to anesthesia per se. A phasic expiratory activation of abdominal muscles has been documented in a majority of subjects during light anesthesia, decreasing with deepening anesthesia. Therefore, it is theoretically possible that the increase in end-expiratory diaphragm length might be due to an increase in abdominal pressure secondary to abdominal muscle contraction, and not to a loss of tone in the diaphragm itself. However, this mechanism was ruled out in humans by Froese and Bryan, since they measured a similar cephalad displacement of the diaphragm during anesthesia and during muscle paralysis. In the absence of EMG recording of abdominal muscles, we cannot comment on their activation, but we think, nevertheless, that this mechanism did not account for the length changes that we observed. Firstly, there is no apparent reason for an abdominal muscle contraction to affect the length of only the crural part of the diaphragm, especially since the
costal part represents about five-sixths of the diaphragm area. Secondly, according to the in vivo passive pressure-length characteristics of the diaphragm measured by sonomicrometry, the baseline abdominal pressure should increase by approximately 7 cm H2O to produce the crural lengthening that we observed. On the contrary, the baseline abdominal pressure fell slightly or did not change in the four dogs in which it could be measured.

It should be mentioned that, in contrast to humans, dogs do not manifest, as a rule, a fall in FRC with anesthesia. During halothane anesthesia, FRC has been shown to decrease in supine, but not in prone, dogs. During thiopental anesthesia, FRC did not change significantly in dogs in prone, supine, and lateral positions. FRC was not measured in this study, but we attempted to indirectly evaluate the volume change induced by the diaphragmatic lengthening. Taking the actual length of each segment and the area of the diaphragm dome measured in dogs of similar weight, assuming a piston-like displacement of the diaphragm and considering the relative areas of each segment, and finally assuming an FRC of 1 l, we calculated that the length changes that we measured would produce a 5–6% fall in FRC. The interspecies difference in FRC decrement during anesthesia, which is still not explained, raises the possibility that the increase in diaphragm LfRC may be greater in humans than in dogs. However, the decrease in FRC observed in humans is not only due to diaphragmatic displacement, but also to changes in rib cage dimensions and intrathoracic fluid volume.

Finally, the time course of crural lengthening is noteworthy. Most of the length change occurred within the first minutes of anesthesia, but an additional elongation appeared slowly over the next 20 min. In man, FRC decreases within minutes after induction of anesthesia, and remains unaltered over the next half-hour. It is unknown
if the slow elongation that we measured in dogs occurs in humans. If it does, the lack of further change in FRC could be due to the small size of this additional lengthening, or to compensatory volume changes from other thoracic structures.

In summary, we demonstrated that, in dogs, anesthesia induces a 7–8% increase in end-expiratory length of the crural diaphragm with no change in the costal diaphragm. This difference seems to be due to segmental properties and not to a pressure gradient effect. In partial data, we observed prolonged post-inspiratory EMG activity and, in some cases, tonic activity of the diaphragm in the awake state, which disappeared during anesthesia. It remains to be established if the selective lengthening of the crural diaphragm is secondary to a preferential loss of tone in this segment.

References