Thoracic Epidural Anesthesia Increases Diaphragmatic Shortening after Thoracotomy in the Awake Lamb

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Background: Prolonged inhibition of diaphragmatic function occurs after thoracic and upper abdominal surgery. It was hypothesized that thoracic epidural anesthesia on the day after a thoracotomy could block inhibitory neural pathways and increase the shortening of costal and crural diaphragmatic segments.

Methods: Pairs of sonomicrometer crystals were implanted into the costal and crural regions of the diaphragm through a right lateral thoracotomy in 14 30-kg, 4–5-month-old lambs. One day after surgery, a thoracic epidural catheter was placed at the T8–T9 level. Regional diaphragmatic shortening normalized to end-expiratory length (ΔL_{TOT}), was measured by sonomicrometry in these awake lambs. Changes in gastric (ΔP_{ga}), esophageal (ΔP_{es}), and transdiaphragmatic (ΔP_{di}) pressures were measured with transnasal balloon catheters. End-tidal carbon dioxide (PETCO_{2}), costal and crural electromyogram (EMG), and tidal volume (VT) were measured. Inductance plethysmography was used in four lambs to assess relative contributions of the rib cage and abdomen to VT. Control values were obtained during quiet breathing and while re-breathing at up to 10% PETCO_{2}. To block thoracic dermatomes, 1% or 2% lidocaine was injected through the epidural catheter. Measurements were repeated after each lidocaine injection.

Results: There was no change of resting length with 1% lidocaine; costal resting length increased by 22% with 2% lidocaine. After 2% lidocaine, costal EMG increased from control both during quiet breathing (8.7 ± 0.7 to 18.1 ± 1, S ± SEM%) and at PETCO_{2} 10% (22.1 ± 2 to 33.7 ± 3%). VT during quiet breathing was unchanged after 1% lidocaine but increased from 235 ± 16 to 283 ± 28 ml after 2% lidocaine. At 10% PETCO_{2}, ΔP_{di} was unchanged after 1% lidocaine and decreased from 36.5 ± 4.3 to 26.3 ± 4.9 cmH_{2}O after 2% lidocaine. Regional ΔE_{TOT} was unchanged with both 1% and 2% lidocaine at rest and during carbon dioxide re-breathing. Plethysmography in three lambs showed a reduction in rib cage contribution to tidal volume with 2% lidocaine during quiet breathing.

Conclusions: Improved postoperative tidal volume and diaphragmatic shortening after thoracic epidural blockade may be due to changes of chest wall conformation and resting length and a shift of the workload of breathing from the rib cage to the diaphragm caused by intercostal muscle paralysis. (Key words: Anesthetic techniques; epidural. Diaphragm: inhibition. Lung: postoperative respiratory function. Measurement techniques: sonomicrometry. Surgery: thoracic.)

INHIBITION of diaphragmatic function occurs after thoracic and upper abdominal surgery in both humans and experimental animals and is believed to be one of the major factors contributing to respiratory complications after a thoracotomy. The postoperative reduction of diaphragmatic contraction is both profound and long lasting. The precise mechanism of diaphragmatic dysfunction is unknown, but it is hypothesized to arise from stimulation of afferent nerves in the chest and abdominal walls, the viscera, and/or the diaphragm, producing inhibition of phrenic nerve motor drive. An inspiratory inhibitory reflex originating in the intercostal muscle spindles has been described in human neonates. The postoperative contractile function of the diaphragm is not impaired, since a normal shortening fraction can be elicited by electrical stimulation of the phrenic nerve. Pain does not appear to be the major mediator of this inhibitory phenomenon, as several studies have shown minimal or no alteration in the various indirect indices of diaphragmatic function despite profound analgesia produced by either parenteral or epidural opioids. Other investigations have demonstrated some improvement of indirect measures of diaphragmatic function after clinical postoperative intercostal blockade or thoracic...
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epidural anesthesia,12 suggesting that reduced diaphragmatic inhibition might result from the neural blockade of inhibitory afferent pathways that ascend in the spinal cord.

We undertook this study to examine the hypothesis that post-thoracotomy diaphragmatic dysfunction is due to inhibitory afferent pathways arising from the chest wall and to learn whether segmental epidural anesthesia could block afferent pathways, resulting in increased diaphragmatic shortening. Therefore, in awake lambs, we used implanted sonomicrometers to directly measure the shortening fraction and resting length of the costal and crural regions of the diaphragm, electromyograms (EMGs) to assess diaphragmatic activation, balloon-tipped catheters to measure transdiaphragmatic pressure, and a pneumotachograph to measure tidal volume and inspiratory and expiratory times. Inductance plethysmography (Respirtrace, Ambulatory Monitoring, Ardsley, NY) was employed in four lambs to assess rib cage and abdominal dimensions during quiet breathing before and after epidural blockade.

Methods

Implantation

This study was approved by the Subcommittee on Animal Studies of the Massachusetts General Hospital. Our monitored lamb model has been described in detail in previous reports.5,6,13 A rigid lateral thoracotomy was performed in 14 4–5-month-old lambs at the 9th intercostal space during general endotracheal anesthesia with 2–2.5% halothane. The animals weighed 30 ± 1 (X ± SEM) kg. Pairs of sonomicrometer crystals (Dimension 3, Hacienda Heights, CA), mounted on stalks of PE 190 tubing and affixed to 15-mm triangles of Dacron with RTV silicone rubber were implanted between muscle fibers on a relatively planar surface of the costal and crural diaphragm regions. Placement of the crystals in the region of the diaphragm over the liver provided increased stability and improved signals from these devices. The crystal faces, 15–20 mm apart, were oriented perpendicular to the muscle fibers, and proper alignment was confirmed by visualizing the sonomicrometer signal on an oscilloscope (Tektronix, Beaverton, OR) before closure of the chest. Corresponding EMG leads made of Teflon-coated multi-stranded stainless steel wire (Medwire, Mt. Vernon, NY) were implanted adjacent to the sonomicrometers. All leads were tunneled subcutaneously and brought out through separate skin incisions. Before closure of the chest, the diaphragm was electrically stimulated with a supramaximal tetanic stimulus at 100 Hz (Microstim, NeuroTechnology, Houston, TX) to assess the maximal attainable shortening fraction. The chest was evacuated with a 24-Fr thoracostomy tube at the time of closure, and a chest radiograph documented the absence of any residual pneumothorax. A tracheostomy was performed, intercostal nerve blocks with 0.5% bupivacaine were administered, and the animals were allowed to recover overnight. The lambs received daily intramuscular antibiotics (Combiotic, Pfizer, New York, NY) after surgery. All implantations were performed by the same thoracic surgeon (J.C.W.) using the same techniques.

Experimental Protocol

Lambs were studied 12–24 h after surgery. They were not fed for 24 h before the study, to avoid gastric distension, but were allowed unlimited access to water. All studies were performed with the animals standing, awake, and unsedated.

Epidural Catheter Placement. Auffed 7-mm tracheostomy tube was inserted and inflated. A brief (less than 30 min) anesthetic with 1–2% halothane was administered to facilitate placement of a 20-G thoracic epidural catheter. The catheter (Portex, Keene, NH) was placed at the T9–T10 level through a 17-G Weiss needle using a modified paramedian approach with loss of resistance to saline and threaded 2–3 cm cephalad. Position was confirmed with a lateral radiograph after an epidural injection of iopamidol nonionic contrast media (Isovue 360, Squibb, New Brunswick, NJ). In the animals that received a 1% lidocaine epidural blockade, the contrast injection was mixed with an equal volume of 2% lidocaine (producing a 1% lidocaine/half-strength iopamidol solution) and administered after the control studies. One hour of awake recovery breathing air was allowed before the beginning of the study period.

Measurements. After topical nasal anesthesia with 4% lidocaine jelly, two balloon-tipped catheters (#85842, National Catheter, Mallinckrodt, Argyle, NY) were inserted through the nares. One was placed in the stomach and inflated with 3 ml of air; the other was placed in the lower third of the esophagus and inflated with 1 ml of air. These were connected to differential pressure transducers (Validyne MP45) to measure gastric (ΔPga) and esophageal (ΔPeo) pressure changes. Esophageal balloon position was confirmed

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by recording large negative pressure waves with transient tracheal occlusion and by noting transmitted cardiac pressure artifacts.

Tracheostomy flow was measured with a Fleisch pneumotachograph (model 3700, Hans Rudolph, Kansas City, MO) calibrated for volume with a 1-l giant syringe. This signal was integrated to obtain tidal volume (VT). Expired gas was continuously sampled at the tracheostomy, and end-tidal carbon dioxide (PETCO2) was measured with an infrared analyzer (Beckman LB-2, Sensormedics, Anaheim, CA).

Before each study, this was calibrated with air and 5% CO2.

The EMG signals were amplified (Grass, Quincy, MA), band-pass filtered (30 Hz to 2 kHz), and processed through a Paynter filter using a 0.1-s period. The raw EMG signal was integrated and recorded.

Dynamic measurements of the distance between each crystal pair were obtained with a sonomicrometer (model 120, Triton Technology, San Diego, CA). Each piezoelectric crystal pair consists of a transmitter and receiver; when electrically stimulated, the transmitter emits ultrasonic waves that induce a voltage in the receiver. A quartz clock in the sonomicrometer measures the transit time of sound waves between the crystals, and, because the speed of sound in muscle is a known constant, the distance between crystals can be determined accurately.

Control Studies. Measurements were obtained while lambs breathed air for several minutes until a steady state was reached. Following this period of quiet breathing, a 5-l anesthesia reservoir bag containing 5% CO2 in 95% O2 was attached to the tracheostomy, and the animal rebreathed this gas mixture until the PETCO2 reached 10% to stimulate central respiratory drive. Two control studies were performed in each animal, and a 20-min recovery period was allowed between studies.

Epidural Blockade. After the control studies, eight lambs received an epidural blockade with 1% lidocaine. Equal parts of iopamidol and 2% lidocaine were mixed, yielding a half-strength iopamidol in 1% lidocaine solution, and 1.5 ml was injected through the epidural catheter. A lateral radiograph was taken to confirm catheter placement in the epidural space. The distribution of blockade was assessed by loss of the patulysma twitch response (a visible twitch of platsysma muscle underlying the skin in response to a light touch) and by lack of withdrawal to cutaneous electrical stimulation at 100 Hz. Quiet breathing and carbon dioxide-stimulated rebreathing studies were obtained as described for the control studies.

In four lambs, the 1% lidocaine blockade was allowed to dissipate, a second block was established with 1.5 ml 2% lidocaine, and the protocol was repeated. Three hours were allowed to elapse after the 1% lidocaine study to allow any residual blockade to dissipate. In the six subsequent lambs, only 2% lidocaine was injected after the control studies. These animals had epidurograms performed with iopamidol alone after the placement of the epidural catheter. Thus, four lambs received 1% lidocaine, four received both 1% and 2% lidocaine, and six received only 2% lidocaine.

In four animals that received a 2% lidocaine blockade, inductance plethysmography (Respirtrack) was used to quantify the relative contributions of the rib cage and abdomen to ventilation during resting breathing. The plethysmograph was calibrated for volume using the method of Abraham et al.14

Data Collection and Analysis. Signals were recorded on a Hewlett Packard 7758A strip chart recorder (Palo Alto, CA) and digitized using CODAS, an IBM compatible data acquisition and analysis system (Dataq, Akron, OH). Pressures were measured according to the conventions we described previously.6 ΔPgas and ΔPes were measured as the difference between the trough of the tracing at end expiration and its crest at peak inspiration. The pneumotachograph trace provided the timing points for these measurements. Expiratory ΔPgas was measured as the difference between peak and trough measurements during exhalation, seen as the second inflection point on the Pgas tracing. ΔPes was calculated as the difference between ΔPgas and ΔPes. Data either were measured directly from the strip charts and entered into a computer (320 Microstation, Northgate Computer, Plymouth, MN) or were measured using CODAS, when available. Five sequential breaths were measured for each lamb at resting breathing and from PETCO2 7% to 10% in 0.5% increments. All values are expressed as mean ± SEM. Each animal served as its own control, and paired t tests assessed significance at the P < 0.05 level.

Results

Two of the 14 lambs (2% lidocaine only group) were excluded from the analysis because of an abnormal postoperative respiratory pattern and carbon dioxide retention at rest (PETCO2 6.8% and 6.4%) before epidural

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blockade. The other 12 lambs all exhibited inhibition of diaphragmatic segmental shortening after the thoracicotomy similar to the level of postoperative inhibition measured in our previous reports. Crural sonomicrometers did not function in one lamb, and EMG electrodes malfunctioned in two lambs. Thus, the number of animals for which data were analyzed includes seven lambs in each group for data involving EMG, seven lambs for data involving crural shortening, and eight lambs for data involving costal shortening. Costal shortening fractions of 8.7 ± 0.7% at rest and 22.1 ± 2% at 10% \text{F}e\text{CO}_2 were measured before epidural blockade (fig. 1). Direct tetanic stimulation of the diaphragm during general anesthesia at the time of implantation elicited regional shortening fractions of 50 ± 5% (n = 14).

An epidural injection of 1.5 ml lidocaine in iopamidol mixture (1% lidocaine final concentration) produced an epidural block approximately spanning the T4 to L1 dermatomes. Changes in segmental shortening fraction, ΔPdi, and ΔEdi during epidural blockade are shown in figures 1 and 2. After 1% lidocaine blockade, the fractional shortening of the costal segment during quiet breathing increased by 10% from control, from 8.7 ± 0.7% to 10.7 ± 1%, but did not reach statistical significance (P = 0.08; fig. 1 and table 1). At 10% \text{F}e\text{CO}_2, the shortening fraction was unchanged from control.

Following 2% lidocaine blockade, the costal shortening fraction significantly increased both during quiet breathing and during 10% \text{CO}_2 rebreathing (fig. 1), while ΔPdi decreased and ΔEdi was unchanged from control (fig. 2). Crural shortening increased during quiet breathing after 2% lidocaine, and although there was a tendency toward an increase at 10% \text{F}e\text{CO}_2, the large variability precluded statistical significance (fig. 1). Crural ΔEdi remained unchanged after epidural blockade. Abdominal muscle relaxation was produced, as evidenced by the marked decrease of expiratory ΔPhib during \text{CO}_2 rebreathing (table 1).

Table 1 displays the values of the respiratory variables. There were no changes of tidal volume (\text{V}_T), minute ventilation (\text{V}_E) or respiratory rate following 1% epidural blockade, but after 2% lidocaine blockade, tidal volume during quiet breathing increased to 283 ± 28 ml from 235 ± 16 ml before blockade (P < 0.05), while \text{V}_T/T_i (the mean inspiratory flow rate) was unchanged. The costal velocity of shortening (%LFR/s) increased after 2% lidocaine blockade both during quiet breathing and at 10% \text{F}e\text{CO}_2 but was unchanged during 1% lidocaine blockade.

Inductance plethysmograph (Respirtrac) measurements could not be calibrated in one lamb. In the remaining three lambs after 2% lidocaine blockade, the rib cage's contribution to ventilation decreased, and the abdominal contribution increased from control levels during quiet breathing (table 2). Rib cage volume (volumetric equivalents) decreased after epidural blockade with 2% lidocaine, and functional residual capacity, calculated from the rib cage and abdominal

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motion coefficients, decreased in two lambs and increased in one (table 2).

Discussion

In awake lambs 24 h after a thoracotomy, thoracic epidural administration of 2% lidocaine increases diaphragmatic shortening and increases tidal volume during quiet breathing. Concomitant with these improvements, the costal diaphragmatic segmental resting length increased by nearly 25% (table 1); the rib cage contribution to ventilation, as measured by inductance plethysmography, decreased from 50% to 10% (table 2); and $\Delta P_{di}$ decreased despite the increased costal segment shortening fraction. These major respiratory muscle alterations after thoracic epidural anesthesia using 2% lidocaine were not anticipated and demonstrate that the mechanism widely believed to be the cause of improved postoperative diaphragmatic shortening after epidural anesthesia, the blockade of afferent inhibitory neural pathways, may be overly simplistic in the sheep after a thoracotomy. The relationship between these changes and the interactions between the diaphragm and rib cage muscles under the different conditions of control state and after epidural blockade, however, may help to explain our results.

Investigators of diaphragmatic dysfunction after both abdominal and thoracic surgery have suggested that respiratory function is impaired by inhibition of phrenic nerve motor drive. Neither intravenous, intrathecal nor epidural administration of opioid analgesia to patients after surgery reduces phrenic inhibition. Mankikian and his colleagues, using indirect measurements of diaphragmatic function in humans after a cholecystectomy, suggested that diaphragmatic function may improve following an epidural blockade. In their investigation, thoracic epidural blockade with 0.5% bupivacaine increased $\Delta P_{pm}/\Delta P_{es}$ (a ratio of pressure measurements reflecting the diaphragmatic contribution to ventilation) and augmented diaphragmatic motion as inferred from linear differential transducers placed on the rib cage and the abdomen. Their data indicated that neural blockade might block the transmission of the afferent impulses decreasing phrenic nerve drive. The limitations of non-invasive methodologies to accurately describe diaphragmatic function did not allow these investigators to directly measure human diaphragmatic contraction, and a prior study from our laboratory casts some doubt on the use of cavitary pressures to reflect ovine diaphragmatic shortening.

Epidural injection of local anesthetics may cause considerable muscular weakness, resulting in difficulty distinguishing the effects of motor blockade from blockade of inhibitory afferent pathways. We believe the increased diaphragmatic shortening we measured in lambs after epidural anesthesia with 2% lidocaine (fig. 1) primarily may be due to a shift of the workload of breathing from the chest wall (following intercostal
motor blockade) to the diaphragm. This was manifested by an increase in costal resting length, a decrease in expiratory $\Delta P_{gas}$, a decrease in rib cage tidal expansion, and a decrease in rib cage size (in volumetric equivalents; tables 1 and 2). The shift in workload may explain why the increase of diaphragmatic segmental shortening measured after 1% blockade, where minimal impairment of thoracic and abdominal motor function probably occurred, was less than after 2% lidocaine.

To further test this hypothesis, we studied three lambs using inductance plethysmography during the control state and after epidural blockade with 2% lidocaine. We found that, while the rib cage contribution to tidal ventilation and expiratory $\Delta P_{gas}$ was markedly diminished after 2% lidocaine (table 2), the tidal volume increased, and respiratory rate and minute ventilation increased.

### Table 1. Respiratory Variables during Quiet Breathing and Carbon Dioxide Rebreathing at Control and after Thoracic Epidural Blockade with 1% and 2% Lidocaine

<table>
<thead>
<tr>
<th>Variable</th>
<th>Quiet Breathing</th>
<th>10% End-tidal Carbon Dioxide</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>1% Lidocaine</td>
</tr>
<tr>
<td>Costal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$L_{pneu}$ (mm)</td>
<td>23.0 ± 1.1</td>
<td>23.6 ± 1.3</td>
</tr>
<tr>
<td>% $L_{pneu}$ (%)</td>
<td>8.7 ± 7</td>
<td>10.7 ± 1</td>
</tr>
<tr>
<td>$\Delta E_{p}$ (units)</td>
<td>19.7 ± 3.1</td>
<td>24.4 ± 6.2</td>
</tr>
<tr>
<td>$\Delta l/\Delta E_{p}$</td>
<td>0.98 ± 0.1</td>
<td>1.0 ± 0.07</td>
</tr>
<tr>
<td>% $L_{pneu}$/s</td>
<td>0.09 ± 0.01</td>
<td>0.11 ± 0.01</td>
</tr>
<tr>
<td>Crural</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$L_{pneu}$ (mm)</td>
<td>19.8 ± 0.5</td>
<td>19.4 ± 0.4</td>
</tr>
<tr>
<td>% $L_{pneu}$ (%)</td>
<td>4.9 ± 0.5</td>
<td>5.0 ± 0.6</td>
</tr>
<tr>
<td>$\Delta E_{p}$ (units)</td>
<td>24.2 ± 2.9</td>
<td>27.6 ± 3.2</td>
</tr>
<tr>
<td>$\Delta l/\Delta E_{p}$</td>
<td>0.04 ± 0.01</td>
<td>0.04 ± 0.01</td>
</tr>
<tr>
<td>$\Delta P_{es}$ (cmH$_2$O)</td>
<td>6.6 ± 0.5</td>
<td>6.8 ± 0.5</td>
</tr>
<tr>
<td>$\Delta P_{es}$ (cmH$_2$O)</td>
<td>5.0 ± 0.3</td>
<td>5.46 ± 0.4</td>
</tr>
<tr>
<td>$\Delta P_{gas}$ (cmH$_2$O)</td>
<td>1.4 ± 0.2</td>
<td>1.0 ± 0.1</td>
</tr>
<tr>
<td>Expiratory $P_{gas}$ (cmH$_2$O)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>FeTco2</td>
<td>5.08 ± 0.11</td>
<td>5.21 ± 0.08</td>
</tr>
<tr>
<td>RR (breaths/min)</td>
<td>21.2 ± 1.2</td>
<td>21.4 ± 1.2</td>
</tr>
<tr>
<td>$V_T$ (ml)</td>
<td>235 ± 16</td>
<td>230 ± 16</td>
</tr>
<tr>
<td>$V_T/T_i$ (ml/s)</td>
<td>271 ± 24</td>
<td>241 ± 18</td>
</tr>
</tbody>
</table>

- $L_{pneu}$ = segment length at end-expiration; % $L_{pneu}$ = segmental shortening fraction normalized for end-expiratory length; $\Delta E_{p}$ (values in arbitrary units normalized to 100) = value at 10% end-tidal CO$_2$ fraction (FeCO$_2$) during control; $\Delta l/\Delta E_{p}$ = change in segment length per unit electromyographic activity; % $L_{pneu}$/s = velocity of shortening; $\Delta P_{es}$ = change in transdiaphragmatic pressure; $\Delta P_{gas}$ = change in esophageal pressure; $\Delta P_{gas}$ = change in gastric pressure; $V_T$ = tidal volume; $V_T/T_i$ = mean inspiratory flow rate.

- * Value differs from control, $P < 0.05$.
- † Value differs from control, $P < 0.01$.

### Table 2. Respiratrace Data During Quiet Breathing in Three Lambs

<table>
<thead>
<tr>
<th>Animal</th>
<th>Rib Cage</th>
<th>Abdomen</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control (% $V_T$)</td>
<td>2% Lidocaine (% $V_T$)</td>
</tr>
<tr>
<td>1</td>
<td>58.9</td>
<td>9.3</td>
</tr>
<tr>
<td>2</td>
<td>62.3</td>
<td>15.9</td>
</tr>
<tr>
<td>3</td>
<td>46.4</td>
<td>14.4</td>
</tr>
</tbody>
</table>

- $V_T$ = tidal volume; $\Delta V$ = calculated change in compartmental volume (volumetric equivalents); $\Delta FRC$ = change in functional residual capacity, calculated from the rib cage and abdominal motions and $V_T$ during resting breathing at control and after 2% lidocaine epidural blockade.

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were unchanged (table 1). These respiratory parameters, therefore, were maintained or increased by increasing the diaphragm's contribution to ventilation.

It is possible that the differences between our ovine and human epidural study results also depend in part on a shift (or lack of shift) of respiratory workload. In our human investigation, no significant rib cage function changes were measured by the inductance plethysmograph. Since no shift in workload from the rib cage to the diaphragm occurred in our patients, no increase of diaphragmatic shortening was measured. This suggests that the dominant physiologic mechanism governing diaphragmatic shortening after epidural anesthesia may be the degree of transfer of workload from the rib cage to the diaphragm.

One of the changes measured in this investigation was the increase of costal segment resting length after epidural blockade (table 1). Torres et al., using the same animal model, found similar changes in magnitude of the costal segment resting length measured after implantation surgery compared to its length measured after recovery. One day after implantation, Torres et al. found the resting length of the costal segment to be 83% of the length of that segment measured during recovery at both 7 and 28 days after surgery. In our lambs, the resting length before epidural blockade was 81.5% of that measured after blockade. We speculate that the epidural block may have returned the costal resting length to that of the preoperative state; that is, the “normal” resting length was reduced after surgery, and the changes in chest wall and abdominal conformation caused by the epidural blockade (an average decrease in rib cage volume of 131 ± 45 ml) restored the diaphragm to its “normal” resting length. If this longer resting length is at a more efficient point on the muscle’s length-tension curve, the contractile efficiency of the diaphragm may be increased by this change, but this is uncertain because the optimal resting length cannot be determined from our data.

If neural inhibition of phrenic motor activation was partially or completely reversed by epidural blockade, one would expect the amount of phrenic neural activation, estimated by ΔEad, to increase. ΔEad did not change in either this study or our human investigation, suggesting that inhibition was not appreciably altered by epidural blockade. However, the increased resting length of the costal segment in this lamb investigation altered the diaphragm’s position on its length-tension curve and presumably changed both the maximal contractile force and the level of neural activation if the tidal volume were to be maintained. An increase in diaphragmatic segmental resting length after epidural blockade would have been reported previously, and thus it is not known what changes in ΔPdi would occur under similar conditions in the uninhibited diaphragm. Nevertheless, this increase in the costal diaphragm’s resting length makes the comparison of ΔPdi and ΔEdi at differing segment lengths potentially misleading. Since the diaphragm, like any muscle, has an optimum resting length at which maximal contractile force is greatest, function at various points on the length-tension curve will produce differing amounts of shortening and ΔPdi for any given amount of neural input, assuming a constant load. The increased costal segment resting length after 2% lidocaine appears to be associated with a greater shortening fraction in response to the same degree of neural activation (estimated by ΔEad). Thus, the contractile efficiency of the diaphragm (tired shortening fraction per unit EMG activation) appears to increase; that is, the amount of shortening increased with a constant neural input, and the resulting tidal volume increased. Furthermore, while shortening increased, the amount of force that was generated (ΔPdi) decreased. Because negative intrathoracic pressure (ΔPad) is generated by both rib cage and diaphragmatic contraction and because the costal diaphragm and intercostal muscles behave as if arranged mechanically in series, a decrease in rib cage contraction would be likely to lead to increased diaphragmatic contraction. Thus, motor blockade of the rib cage muscles may have decreased the force placed on the diaphragm by the rib cage, thereby reducing one limitation to diaphragmatic excursion. This reduced diaphragmatic afterload induced by epidural motor blockade of the rib cage also may have led to an increased velocity of shortening that further reduced diaphragmatic force generation.

Other changes induced by thoracic epidural blockade may have unmasked the effect of the diaphragm upon the rib cage, that is, to expand the rib cage through the action of the diaphragm both at the zone of apposition and at its insertion to the rib cage. Our inductance plethysmograph data (table 2) suggest that the diaphragm’s direct action on the rib cage, in the absence of intercostal and accessory muscle activity, is to lift and expand the lower rib cage. After thoracic epidural blockade, the diaphragm produced far less rib cage expansion (without help from the rib cage or accessory muscles). The action of the accessory and possibly the upper parasternal muscles on the rib cage,
however, most likely was unchanged, and the relative contributions of the diaphragm and these remaining inspiratory muscles on rib cage expansion cannot be quantified from these data.

Epidural motor blockade, by relaxing the abdominal muscles during inspiration, could increase abdominal compartment compliance and thereby increase the shortening fraction of the costal diaphragm segment. This would occur if there were significant tonic inspiratory abdominal muscle activity before blockade. Recruitment of expiratory abdominal muscles (expiratory ΔP_gas) during carbon dioxide rebreathing was nearly abolished by 2% lidocaine in our study, suggesting that tonic inspiratory activity in the abdominal muscles should have been abolished, too. The work of Mankikian et al., which demonstrated increased abdominal compliance in patients after epidural blockade with 0.5% bupivacaine, suggests that decreased abdominal muscle tone might have occurred in our lambs after epidural blockade with 2% lidocaine. However, if a release of tonic abdominal muscle activity by thoracic epidural anesthesia were important in our study, there should have been a decrease of resting segmental length rather than the increase we observed (table 1), due to less restriction of diaphragmatic descent at end expiration. The net effect of the loss of expiratory ΔP_gas would be to decrease shortening after epidural blockade rather than to increase it as we measured, since the level of expiratory ΔP_gas measured during control conditions would be expected to provide an assistance of at least 50% to tidal diaphragmatic shortening (fig. 1).

Before epidural blockade, when there was normal abdominal muscle activity during carbon dioxide rebreathing, abdominal muscle recruitment was released at the end of expiration, resulting in a fall of intraabdominal pressure. This effectively increased abdominal compliance by reducing the difference between P_gas at end expiration and P_gas at maximal inspiration, making ΔP_gas independent of the volume change. Because, after thoracic epidural anesthesia, the costal diaphragmatic segment was lengthened both during quiet breathing and at 10% Fe(floor) and the abdominal muscles were derecruited at 10% Fe(floor), changes of rib cage conformation appeared to be of greater importance than changes of abdominal muscle activity in influencing the mechanics of breathing in the awake standing lamb.

Epidural blockade, by reducing resting sympathetic vascular tone, may have produced a change in thoracic blood volume. In part, this also could account for some of the changes that we measured; but because these changes were not seen after 1% lidocaine also, it appears unlikely that it occurred. We did not monitor the ovine blood pressure for hemodynamic changes that may have occurred in response to thoracic epidural blockade.

Species differences may produce significant influences upon the outcome of this investigation and need to be borne in mind when attempting to extrapolate our ovine results to patients. The lamb's thoracic cage forms an ellipse in the anteroposterior axis, in contrast with human chest wall conformation, which is a transverse ellipse. Also, the abdomen of the sheep is highly distensible, and its four-legged standing posture differs from that of the human. Our lambs were free of pulmonary disease and did not have lung tissue resected, which was not the case for our patients studied after a thoracotomy. In addition, our lambs, like young children, may have a more compliant and easily distorted chest wall than the adult, although such distortion is unlikely to be as extensive as in the case of human infants because of the age and relative maturity of our lambs. Despite these differences, both our ovine study and our clinical study demonstrate that epidural anesthesia produces an increased tidal volume during quiet breathing but did not increase regional ΔP_ao.

The results of our investigation suggest that the effects of epidural anesthesia on diaphragmatic function after thoracic surgery are complex. The increased tidal volume and segmental diaphragmatic shortening measured in this model suggest that epidural anesthesia can produce beneficial effects on respiratory function under certain conditions after thoracotomy. Although decreased phrenic nerve inhibition may result from epidural anesthesia, other dramatic changes of respiratory mechanics, including an increased diaphragmatic segmental resting length and decreased intercostal motor function, appear to play a more important role in this animal model. The lack of major effect of epidural blockade on diaphragmatic inhibition in this study suggests that the major afferent pathways for the putative inhibitory impulses may reside in the vagus or phrenic nerve, which could not be reached by our thoracic blockade. Although the increase in shortening and decrease in rib cage function measured in our lamb model was not identical to that seen in our clinical investigation, both studies suggest that changes in respiratory mechanics may have a greater effect than the reduction of neural inhibition on the pulmonary function changes seen after thoracic epidural anes-
theses. In the future, invasive monitoring in an animal model such as we have described may further decipher the complex neurophysiology of post-thoracotomy phrenic inhibition.

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References