Phrenic Nerve Stimulation during Halothane Anesthesia

Effects on Atelectasis

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Background: Atelectasis formation during anesthesia may be due to loss of respiratory muscle tone, in particular that of the diaphragm. This was tested by tensing the diaphragm by phrenic nerve stimulation (PNS) and observing the effect on atelectasis.

Methods: Twelve patients (mean age 48 yr) without preexisting lung disease were studied during halothane anesthesia. PNS was executed with an external electrode on the right side of the neck. Chest dimensions and area of atelectasis were studied by computed tomography of the chest.

Results: Right-sided PNS against an occluded airway at functional residual capacity reduced the atelectatic area in the right lung from 5.1 to 3.8 cm². The atelectasis was reduced to 1.1 cm² after application of positive end-expiratory pressure (PEEP) of 10 cmH₂O and large tidal volumes but increased to 2.5 cm² within 1 min after discontinuation of PEEP. Commencement of PNS immediately after PEEP prevented the atelectasis from increasing, the mean area being 0.9 cm². In seven patients, in whom the trachea was intubated with a double-lumen endobronchial catheter the atelectatic area was smaller during PNS with an open airway than during positive pressure inflation of the lung with the same volume as inspired during PNS (3.5 and 5.2 cm², respectively).

Conclusions: The findings indicate that contracting the diaphragm in the anesthetized subject reduces the size of atelectasis. (Key words: Anesthetics, volatile: halothane. Lung: atelectasis, diaphragm. Nerve, phrenic: stimulation.)

DENSITIES in dependent lung regions have been demonstrated in anesthetized children¹ and adults² by means of computed tomography (CT). The densities occur in individuals who are spontaneously breathing as well as in those who are paralyzed and whose lungs are being mechanically ventilated.³ The dense regions are perfused from the pulmonary artery as evidenced by intravascularly injected contrast,⁴ and the magnitude of right-to-left shunt correlates with the size of the dense region.⁵ In animal experiments, similar densities have been shown to be atelectasis with minor edema and little or moderate vascular congestion.⁶ ⁷ We suggest that the densities in anesthetized humans also represent atelectasis, caused by early or immediate loss of supportive forces rather than resorption of trapped gas.⁸ ⁹ Similar, but larger densities can be seen in patients suffering from the adult respiratory distress syndrome and they have been extensively studied byGattinoni and co-workers.⁸

The mechanism behind the densities in the patient with adult respiratory distress syndrome appears to be the increased weight of the edematous lung, compressing dependent lung tissue.⁹ In the anesthetized patient the mechanism is less clear. However, anesthesia is accompanied by a reduction in the functional residual capacity (FRC) during both spontaneous breathing and mechanical ventilation with muscle paralysis.⁸ ¹⁰ A number of studies suggest a cephalad displacement of the end-expiratory position of the diaphragm,¹¹ ¹² although a more variable effect on the diaphragm, even with caudal displacement, has been reported.¹³ In addition, a reduction in the transverse area of the thorax has been shown.¹² ¹⁴ These findings may support a reduction in inspiratory muscle tone during anesthesia,

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which may bear a causal relationship to the development of atelectasis by several mechanisms. Thus, loss of diaphragmatic tone may facilitate the transmission of abdominal pressure to the pleural space, resulting in an increased vertical gradient of pleural pressure and a preponderant increase of pleural pressure in basal regions. Also, a reduction of FRC will decrease the overall transpulmonary pressure, although this effect may be partly counteracted by a direct action of the inhalational anesthetic on the lung tissue. We hypothesize that these mechanisms reduce the transpulmonary pressure in dependent regions to such an extent that the forces acting on the lung no longer maintain its patency but cause compression of dependent lung tissue. To test the hypothesis we have investigated whether an increase in diaphragmatic muscle tone by right-sided phrenic nerve stimulation (PNS) during general anesthesia reduces the size of atelectasis in dependent lung regions. If PNS reduces atelectasis we consider our hypothesis supported. We did not use PNS with the purpose of developing or refining a ventilatory technique.

Materials and Methods

Twelve subjects, three women and nine men, with an age of 48 ± 12 yr (mean ± standard deviation; range 25–70 yr) were studied. They had normal heights (177 ± 7 cm; range 164–189 cm) and weights (77 ± 10 kg; range 63–96 kg) except for one obese woman (174 cm and 96 kg). Three subjects smoked cigarettes (15–20 cigarettes/day), and one smoked a pipe. Forced spirometry findings were essentially normal (vital capacity and forced expired volume in 1 s: 95 ± 15% of predicted, respectively). The surgical diagnosis was inguinal hernia in the men and gall-bladder disease in the women. The patients gave their informed consent to participate in the investigation, which was approved by the Ethics and Radiological Committees of Huddinge University Hospital.

Anesthesia

All patients received premedication (oxycodeone 7.5–15 mg and scopolamine 0.3–0.6 mg, intramuscularly). Atropine 0.5 mg was given intravenously immediately before anesthesia to counteract vagal effects of PNS. While the patients breathed oxygen, anesthesia was induced with intravenous thiopental 200–425 mg. Intravenous fentanyl 0.05–0.3 mg was given to suppress spontaneous ventilation during the measurements.

Muscular relaxation was achieved with succinylcholine 75–100 mg. In five patients the tracheas were intubated with a conventional endotracheal tube, and in the remaining seven a double-lumen left endobronchial tube was inserted. Anesthesia was maintained with 0.5–1.0% inspired halothane in oxygen. The patients' lungs were ventilated mechanically at a rate of 12 breaths/min with a Servo 900B Ventilator equipped with a carbon dioxide analyzer (Siemens Elema, Stockholm, Sweden). Tidal volume (0.45–0.80 l) was adjusted to maintain a moderate hyperventilation with an end-tidal carbon dioxide concentration of 3.5–4.0%, as a further measure to prevent spontaneous breathing efforts during the investigation. One ventilator was used also in the patients with a double-lumen tube, and the ventilation was distributed according to the impedance of each lung. Airway pressures were read on a manometer connected to the airway tube.

Phrenic Nerve Stimulation

The phrenic nerve was stimulated percutaneously by means of a conventional nerve stimulator (Medelec MS91, Medelec, Surrey, England) connected to a bipolar electrode manually positioned on the skin between the two heads of the sternocleidomastoid muscle. The right phrenic nerve was stimulated in all subjects. A voltage of 100–200 V was applied as square-wave pulses of 200-μs duration at a rate of 10 Hz. This stimulation frequency resulted in a semitonic contraction of the diaphragm, which could be observed by a change in the epigastric configuration.

Computed Tomography

The subject lay supine on the CT table (Somatom 2, Siemens) and a frontal scout view covering the chest and upper abdomen was obtained when the patient was anesthetized. Preliminary tests showed that the shape of the lung was affected by PNS, the tensed diaphragm pulling the lung caudally by approximately 2 cm. To make possible comparisons of the same lung tissue during the control situation and during PNS, two CT scans were obtained 2 cm apart with fixed levels relative to the spine. The upper (cranial) scan during the control situation was then compared with the lower (caudal) scan during PNS (fig. 1): To check to what extent the lung tissue had been displaced in a particular subject during PNS, an analysis of the vascular configuration was made in the CT scans obtained during control and PNS, and the displacement was qualitatively quantified as less, equal or more than 2 cm. It should
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Fig. 1. Elongation of the right lung during phrenic nerve stimulation (PNS). Compared to control, there is caudal displacement of basal parts of the lung. The two computed tomography scan levels 0 and 2 cm above the top of the diaphragm during control (scans 0 and 2) were kept constant relative to the spine. Scan 0 during PNS was compared to scan 2 during control. Control computed tomography scans were obtained at FRC (procedures 1 and 2) or after inflation of the right lung (procedure 3).

be emphasized that repeated CT scans would have been required in each subject to account for individual diaphragmatic shifts. However, such a procedure was not ethically acceptable because of the risk of exposure to excessive amounts of radiation. Scan time was 5 s at 125 kV and 115 mA. Slice thickness was 8 mm.

The transaxial intrathoracic area as well as sagittal and transverse diameters of the thorax were measured on the scans.

In order to calculate the atelectatic area a magnified image (2.5 ×) was made from the dorsal portion of the right and the left lung. The dorsal border between the thoracic wall and the dense area could easily be identified on the magnified image. The dense area was then encircled as a region of interest and the atelectatic area was calculated as picture elements (pixels) with attenuation numbers between −100 and +100 Hounsfield units.20

Procedures

The patients arrived at the x-ray department after receiving premedication and were anesthetized while lying on the CT table. Approximately 15–20 min after induction of anesthesia the scout view and the first CT scans were obtained. The effect of succinylcholine had by then dissipated, as confirmed by eliciting diaphragmatic contractions during PNS. The phrenic nerve was always stimulated during 1 min without mechanical ventilation and PNS was not discontinued until after CT scanning. Also during control situations 1 min of apnea was allowed before CT scanning to ensure standardized volumes. Nine patients took part in two of the procedures below, and three patients in one. All measurements were made before surgery. CT scans were made according to the following procedures (fig. 2).

Phrenic Nerve Stimulation with Occluded Airway (Procedure 1). PNS with occluded airway at resting lung volume (FRC) (n = 8; five patients studied with a single-lumen endotracheal tube and three patients with a double-lumen endobronchial tube). CT scans were performed:

1. during control with apnea and open airway, no PNS
2. after 1 min of PNS with occluded airway.

PROCEDURES

Fig. 2. The three experimental procedures. The apneic period including the computed tomography scan (CT) is 1 min, and the ventilation between apneic periods lasts for 5 min. Procedure 1: Comparison of the atelectasis area at functional residual capacity (FRC) with an open airway (a) and at FRC during phrenic nerve stimulation (PNS) with an occluded airway (b). Procedure 2: CT scans obtained at baseline (FRC, open airway) (a); after application of positive end-expiratory pressure (PEEP) of 10 cmH₂O and ten double tidal volumes (b); after release of PEEP (open airway) (c) and after the same volume history as before b and c but with PNS immediately after release of PEEP (a). Procedure 3: CT scan obtained during PNS with open airway, double-lumen tube (a). After discontinuation of PNS the expired volume (∆V) was recorded, and after a period of tidal ventilation the same volume (ΔV) was given to the lung by mechanical inflation (b).

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This protocol enabled an analysis of the ability of PNS to resolve atelectasis at an essentially constant lung volume. A double-lumen tube was used in a limited number (n = 3) of patients to prevent gas shift between the lungs.

**Phrenic Nerve Stimulation after Previous Hyperinflation (Procedure 2).** PNS after previous hyperinflation by means of positive end-expiratory pressure (PEEP) of 10 cmH2O and after ten double tidal volumes to eliminate or reduce as much as possible the atelectasis (n = 6; endotracheal tube, n = 3; endobronchial tube, n = 3). CT scans were performed:

1. during control at FRC before PEEP, no PNS
2. after 5 min of PEEP and double tidal volume, no PNS
3. at FRC after 1 min of apnea and discontinuation of PEEP, no PNS
4. 1 min after a second period of 5 min with PEEP and double tidal volume. A reliable electrode position for PNS was identified just before the end of PEEP. PEEP was then released to allow the lung to reach its normal FRC; the airway tube was occluded; and PNS was immediately undertaken for 1 min.

This protocol assessed the ability of PNS to prevent the reformation of atelectasis. However, it postulates that FRC is reached before any atelectasis is produced, or that such early atelectasis is easier to eliminate by PNS.

**Phrenic Nerve Stimulation with Open Double-lumen Endobronchial Tube (Procedure 3).** PNS with open double-lumen endobronchial tube compared with isovolumic mechanical inflation (n = 7). CT scans were performed:

1. after 1 min of PNS with the endobronchial tube open
2. The volume previously expired from the right lung when releasing PNS was measured by a water-sealed spirometer. After 10 min of mechanical ventilation, the same volume of air was inflated in the right lung, and the lung was kept at that level for 1 min before CT scanning.

This protocol enabled an analysis of the effect of increase in lung volume by PNS compared to inflation. Also, this procedure did not cause any negative alveolar pressure (as does PNS against an occluded airway).

**Statistics**

Data are presented as mean and standard error of the mean. Student's two-tailed, paired t test was used for statistical inference of two groups of data. Analysis of variance with multiple comparisons according to Newman-Keuls was used for more than two groups. P < 0.05 was considered significant.

**Results**

**Diaphragm Shift**

The caudal displacement of the diaphragm caused by PNS was 2 cm in 8 of a total of 21 measurements (6 of 12 patients). Thus, the same lung segment was studied when the upper, cranial scan during control situation was compared with the lower, caudal scan during PNS. In five measurements the caudal displacement of the diaphragm was less than 2 cm, and in the remaining eight measurements the displacement of the diaphragm was greater than 2 cm as inferred from the shape of the vascular tree in the CT scan. No exact calculation of the diaphragm shift could be made.

**Atelectasis Formation**

All patients developed atelectasis in both lungs and at both scan levels within 15 min after induction of anesthesia (right lung atelectatic area: 5.6 ± 1.2 and 5.4 ± 1.1 cm², upper and lower scan level, 2 cm apart, respectively; left lung: 7.2 ± 1.1 and 6.0 ± 0.7 cm²). The atelectasis corresponded to 3.9% the total intrathoracic area and was similar to earlier findings. There were no significant differences in the atelectatic area between the left and the right lungs or between the two CT scan levels.

**Effects of Phrenic Nerve Stimulation**

**Phrenic Nerve Stimulation with Occluded Airway.** During PNS at FRC with an occluded tube, airway pressure was reduced by approximately 30 cmH2O. The atelectasis on the right side was reduced in seven patients and was unchanged in one patient. The mean reduction amounted to 25% (P < 0.05; table 1 and fig. 3). On the left side during PNS there was a mean increase of the atelectatic area in the caudal scan by 26% (P = 0.10). There was no clear difference between the three patients in whom the tracheas was intubated with a double-lumen tube, and the other five patients.

**Phrenic Nerve Stimulation after Previous Hyperinflation.** PEEP of 10 cmH2O combined with ten double tidal volumes reduced the atelectatic area; in one of the patients it was completely eliminated (table 2). CT scans were repeated 1 min after the discontinu-
Table 1. Atelectatic Area after 1 Minute of Apnea at FRC (Control) and after 1 Minute of Right-sided Phrenic Nerve Stimulation with Airway Closed at FRC (PNS)

<table>
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<th>Right Lung (cm²)</th>
<th>Left Lung (cm²)</th>
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<tbody>
<tr>
<td>Control</td>
<td>5.1 ± 1.1</td>
<td>5.7 ± 1.7</td>
</tr>
<tr>
<td>PNS</td>
<td>3.8 ± 0.8*</td>
<td>7.5 ± 2.0</td>
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FRC = functional residual capacity.  
Values are mean ± SEM; n = 8.  
* Significantly different between control and PNS, P < 0.05.

uation of PEEP. Within this time the atelectasis had partly redeveloped. PEEP was again applied for 5 min and PNS instituted on the right side immediately after the release of PEEP and the occlusion of the airway as described above. As repeated inflations with PEEP may have affected the redeveloped atelectasis we measured the effect of PNS as the difference in atelectatic area on the right and left sides after the release of PEEP without and with PNS. The difference after PEEP: 1.1 ± 0.02 and after PEEP + PNS: −0.6 ± 0.6 cm² was significant (P < 0.05) pointing to a protective effect of PNS against atelectasis. In three patients whose tracheas were intubated with a double-lumen tube there was a similar response to PNS. In the left lung, atelectasis redeveloped to the same extent after discontinuation of PEEP whether right-sided PNS was undertaken or not.

Phrenic Nerve Stimulation with Open Double-lumen Endobronchial Tube. In the third set of measurements PNS was undertaken with the airway open, permitting lung volume to increase, at an atmospheric alveolar pressure. The volume expired from the right lung on discontinuation of PNS varied between 0.3 and 0.9 l. There was no overt diaphragmatic fatigue during PNS for 1 min as indicated by the maintained increase in lung volume (no expiration into the spirometer during PNS). The same volume was then given to the right lung resulting in an airway pressure of 1.0–2.5 kPa (7–19 mmHg). The results are shown in table 3 and figure 4. During PNS there was a significantly smaller atelectatic area on the right side than during mechanical inflation (P < 0.01).

Additional Analysis. A separate analysis of atelectasis formation was made in the eight experiments where identical lung segments were obtained during control and PNS. In these cases the atelectasis area on the right lung was 4.59 ± 1.02 cm² during control, and it was significantly reduced to 3.07 ± 0.78 cm² during PNS (P < 0.01), similar to the results in the separate studies above (procedures 1–3).

Lung Shape

The total transverse area of the lung as well as sagittal and transverse diameters are shown in table 4. In all analyses the lower caudal scan during PNS was compared with the upper, cranial scan during control to account for the downward shift of the lung during PNS. There was no significant change in any of these variables by PNS whether PNS was done against an occluded airway (procedures 1 and 2) or with an open airway (procedure 3). It was not possible to measure the area of each lung separately, because of difficulties of delineating the mediastinum. There was no clear difference between patients whose tracheas were intubated with a single- or double-lumen tube.

Discussion

The study has shown that PNS during anesthesia reduces the atelectatic area on the stimulated side. We consider this to be support of our hypothesis that atelectasis during anesthesia is caused by loss of respiratory muscle tone, in particular of the diaphragm, allowing the higher abdominal pressure to be transmitted to the thoracic space. The assumptions on which our hypothesis is based, the mechanisms behind the findings and possible clinical implications are discussed in the following paragraphs.

Assumptions

Two major assumptions have to be considered. First, comparisons between the control situation and PNS should be made at the same lung volume. An increase in lung volume, for example by PEEP, reduces the amount of collapsed lung tissue both during anesthesia and in adult respiratory distress syndrome. We therefore occluded the endotracheal tube at FRC before starting PNS to prevent the entrance of air and to maintain an unaltered overall lung volume. To prevent an increase in unilateral lung volume caused by a volume shift between the lungs, double-lumen intubation was performed in part of the study. The downward shift of the diaphragm during PNS, despite an occluded airway, can be calculated to have caused a right lung volume increase by around 40 ml, by rarefaction of gas (calculated from an airway pressure of −30 cmH₂O and an assumed right lung volume of 1.3 l). Thus, comparisons...
between PNS and the control situation were made at essentially similar lung volumes.

However, this does not exclude a distortion of the thoracic wall and the lung during PNS. The right hemidiaphragm was pulled caudally by approximately 2 cm during PNS. At the same time there were no significant changes in the intrathoracic dimensions at the studied CT scan level. This may suggest an uneven distribution of the pleural pressure from apex to base by PNS, that is, at an isogravitational level. It has also been dem-
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Table 2. Mean Atelectatic Area during Atmospheric End-Expiratory Pressure (Control); at Positive End-expiratory Pressure of 10 cmH₂O (PEEP) and Double-sized Tidal Volumes; 1 Minute after Discontinuation of PEEP (after PEEP); and after 1 Minute of Right-Sided Phrenic Nerve Stimulation with Airway Occluded at FRC 5 Seconds after the Release of PEEP (after PEEP + PNS)

<table>
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<th>Right Lung (cm²)</th>
<th>Left Lung (cm²)</th>
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<tbody>
<tr>
<td>Control</td>
<td>5.8 ± 1.3</td>
<td>3.8 ± 0.7</td>
</tr>
<tr>
<td>PEEP</td>
<td>1.1 ± 0.7*</td>
<td>0.6 ± 0.5*</td>
</tr>
<tr>
<td>After PEEP</td>
<td>2.5 ± 0.5*</td>
<td>1.4 ± 0.4†</td>
</tr>
<tr>
<td>After PEEP + PNS</td>
<td>0.9 ± 0.2*</td>
<td>1.5 ± 0.5†</td>
</tr>
</tbody>
</table>

Values are mean ± SEM; n = 6.
* Significant difference (P < 0.01) versus previous recording.
† Significant difference between right and left lung.

Oxidated in recumbent dogs that PNS with an open airway resulted in more negative pleural pressure changes at the base of the lung than at the apex on the stimulated side.17,18 Similar, interregional differences in alveolar pressure in response to PNS with occluded airways have also been reported.22 Moreover, in a recent study unilateral hyperinflation caused uneven distribution of pleural pressure in anesthetized dogs and baboons.23 The authors attributed this to thoracic midline structures and the lungs themselves resisting displacement and deformation. All these findings indicate that PNS is not a truly isovolumic maneuver at a regional basis, nor should it be expected so, according to our hypothesis.

Second, the calculation of the atelectatic area should be made in the same lung region during control and PNS; i.e., the CT cut must go through the same lung tissue. We positioned two CT scans 2 cm apart, which made it possible to allow for the descent of the lung base during PNS. In several experiments the cross-sectional CT scan was obtained at exactly the same lung level without and during PNS, as judged from the configuration of the vascular tree. In the other experiments the right lung was shifted more or less than 2 cm in caudal direction by PNS so that slightly different parts were compared. A separate analysis of atelectasis in the eight experiments where identical lung levels had been studied during control and PNS, showed the same effect of PNS as in the total material.

Mechanism of Reduced Atelectasis by Phrenic Nerve Stimulation

Agostoni et al.16 have shown that the vertical abdominal pressure gradient is a major contributor to the vertical pleural pressure gradient. In accordance, studies in awake, supine humans have shown a larger vertical gradient of regional lung expansion (lung expansion used as an index of regional pleural pressure) when the diaphragm is relaxed compared to when it is voluntarily tensed.13 The mechanism by which the tensile state of the diaphragm influences the vertical pleural pressure gradient can be explained by looking at two extremes: if the diaphragm were completely rigid it would take up all the pressure from the abdomen and two completely different pressure gradients would exist on either side of the diaphragm. On the other hand, if the diaphragm were completely flaccid the abdominal contents would encroach on the thoracic cavity until the pressure gradient were the same in the two cavities.

A decrease in diaphragmatic tone has been observed during halothane anesthesia and spontaneous breathing,24 and in an experimental study halothane impaired diaphragmatic muscle function.25 In humans halothane reduces FRC3,5 and a cephalad displacement of the diaphragm has been shown,11,12 although different effects on the diaphragm have also been reported.14 These studies indicate that conditions exist for an increased transmission of the abdominal gradient to the thorax during halothane anesthesia. Tensing the diaphragm at isovolumic conditions would then expand dependent regions at the expense of the nondependent regions.

The results of the current study support this contention. Heneghan and Jones26 reached the same conclusion studying gas exchange in rabbits during bilateral PNS compared with PEEP at isovolumic conditions. The alvoclar-arterial oxygen tension difference was significantly lower during PNS, which they attributed to a more uniform lung expansion.

An anesthetic that does not attenuate the respiratory muscle tone, ketamine, has been shown not to produce atelectasis or shunt,27 nor is FRC reduced.28 Gas exchange was also normal with a good ventilation-per-

Table 3. Mean Atelectatic Area after 1 Minute of Right-sided Phrenic Nerve Stimulation with Open Airway (PNS) and 1 Minute after the Inflation of the Right Lung with the Same Volume of Air as Had Been Inhaled with PNS

<table>
<thead>
<tr>
<th></th>
<th>Right Lung (cm²)</th>
<th>Left Lung (cm²)</th>
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<tbody>
<tr>
<td>PNS</td>
<td>3.5 ± 0.9</td>
<td>5.0 ± 1.4</td>
</tr>
<tr>
<td>Inflation</td>
<td>5.2 ± 1.1*</td>
<td>6.0 ± 1.8</td>
</tr>
</tbody>
</table>

Values are mean ± SEM; n = 7.
* Significant difference between PNS and inflation, P < 0.01.

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fusion match, as assessed by multiple inert gas elimination.\textsuperscript{27} However, when muscle paralysis was created during ketamine anesthesia, both atelectasis and shunt were produced. These findings underscore the importance of a maintained diaphragm function for the patency of the lung tissue and a normal gas exchange.

It should also be mentioned that thoracoabdominal strapping, by means of a corset in awake, healthy volunteers, did not produce atelectasis, despite a lung volume decrease equal to or larger than that caused by anesthesia.\textsuperscript{29} Chest strapping has been shown to increase transdiaphragmatic pressure, implying an increased muscle tone of the diaphragm,\textsuperscript{39} which according to the discussion above, may have protected against atelectasis.
Table 4. Lung Dimensions during Right-sided Phrenic Nerve Stimulation (PNS) and Control

<table>
<thead>
<tr>
<th></th>
<th>Transverse Area (cm²)</th>
<th>Sagittal Diameters (cm)</th>
<th>Transverse Diameter (cm)</th>
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<tr>
<td></td>
<td>Right Lung</td>
<td>Left Lung</td>
<td></td>
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<tr>
<td>PNS against an occluded airway (group 1 and 2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single lumen tracheal tube (n = 8)</td>
<td>334 ± 7</td>
<td>17.0 ± 0.4</td>
<td>17.7 ± 0.4</td>
</tr>
<tr>
<td>Control</td>
<td>340 ± 7</td>
<td>17.2 ± 0.3</td>
<td>17.9 ± 0.4</td>
</tr>
<tr>
<td>Double lumen tracheal tube (n = 6)</td>
<td>320 ± 18</td>
<td>16.1 ± 1.1</td>
<td>17.1 ± 0.8</td>
</tr>
<tr>
<td>Control</td>
<td>324 ± 16</td>
<td>16.4 ± 0.9</td>
<td>16.8 ± 0.9</td>
</tr>
<tr>
<td>PNS with open airway (group 3 (n = 7))</td>
<td>345 ± 17</td>
<td>17.0 ± 0.8</td>
<td>17.5 ± 0.7</td>
</tr>
<tr>
<td>Control</td>
<td>333 ± 13</td>
<td>16.7 ± 0.6</td>
<td>16.9 ± 0.7</td>
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</table>

In conclusion, this study has shown that PNS reduces dependent atelectasis during anesthesia at overall iso-volumic conditions. The findings support the hypothesis that mechanical factors underlie the formation of atelectasis. We suggest that these factors are relaxation of the diaphragm, transmission of the abdominal vertical pressure gradient into the thoracic cavity, and an increase in basal, dependent pleural pressure. PNS tenses the diaphragm, thereby reducing such changes.

References

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