A Computed Tomographic Scan Assessment of Endotracheal Suctioning-Induced Bronchoconstriction in Ventilated Sheep

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This study was directed at assessing changes in bronchial cross-sectional surface areas (BCSA) and in respiratory resistance induced by endotracheal suctioning in nine anesthetized sheep. Cardiorespiratory parameters (Swan–Ganz catheter), respiratory resistance (inspiratory occlusion technique), BCSA, and lung aeration (computed tomography) were studied at baseline, during endotracheal suctioning, and after 20 consecutive hyperinflations. Measurements performed initially at an inspired oxygen fraction (FIO2) of 0.3 were repeated at an FIO2 of 1.0. At an FIO2 of 0.3, endotracheal suctioning resulted in atelectasis, a reduction in BCSA of 29 ± 23% (mean ± SD), a decrease in arterial oxygen saturation from 95 ± 3% to 87 ± 12% (p = 0.02), an increase in venous admixture from 19 ± 10% to 31 ± 19% (p = 0.006), and an increase in lung tissue resistance (DRn) (p = 0.0003). At an FIO2 of 1.0, despite an extension of atelectasis and an increase in pulmonary shunt from 19 ± 5% to 36 ± 2% (p < 0.0001), arterial O2 desaturation was prevented and BCSA decreased by only 7 ± 32%. A recruitment maneuver after endotracheal suctioning entirely reversed the suctioning-induced increase in DRn and atelectasis. In three lidocaine-pretreated sheep, the endotracheal suctioning-induced reduction of BCSA was entirely prevented. These data suggest that the endotracheal suctioning-induced decrease in BCSA is related to atelectasis and bronchoconstriction. Both effects can be reversed by hyperoxegenation maneuver before suctioning in combination with recruitment maneuver after suctioning.

Endotracheal suctioning in intubated patients is directed at clearing bronchial secretions, which tend to accumulate because tracheal intubation markedly impairs mucociliary transport. The classical procedure for endotracheal suctioning consists of disconnection from mechanical ventilation, followed by insertion of a suction catheter into the trachea for tracheal suctioning under negative pressure. Each step of this maneuver is potentially harmful, and complications reported with it include arterial hypoxemia and atelectasis (1), bronchospasm (2), cardiac arrhythmias (4), intracranial hypertension (5), and sudden death (6). Most previous studies of endotracheal suctioning have focused on the prevention and treatment of suctioning-induced impairment of arterial oxygenation. Some preventive maneuvers for this have been proposed, such as preoxygenation with an inspired oxygen fraction (FIO2) of 1 (7), hyperinflation before or after suctioning (8, 9), use of a special suction adaptor to avoid disconnection of the patient from the ventilator (10, 11), and insufflation of a constant flow of oxygen during endotracheal suctioning (1).

Recently, it has been shown that endotracheal suctioning may induce a transient bronchoconstrictive response in critically ill patients receiving mechanical ventilation (12). Experimental data suggest that the stimulation of airway irritant receptors produces reflex bronchoconstriction (13), and that this can be prevented with local anesthetic agents that act on sodium channels (14, 15). In ventilated animals or patients, changes in bronchomotor tone can be evaluated by means of the end-inflation airway occlusion technique (16, 17). Unfortunately, this technique provides limited information about the partition between bronchial and parenchymal responses, and no data on the distribution of the regional response. Recent developments in high-resolution computed tomography (HRCT) allow detection and measurement of the cross-sectional surface area of bronchi down to 1 mm in diameter. These developments have been used to assess bronchoconstriction in animal models and in asthmatic subjects (18–21) at a regional level, and appear to be complementary to the measurement of airway resistance.

The aims of this study, which combined these two approaches, were: (1) to assess the global and regional effects of endotracheal suctioning on bronchoreactivity and lung aeration; (2) to assess whether the endotracheal suctioning-induced increase in bronchoreactivity could be prevented by hyperoxegenation or inhalation of lidocaine; and (3) to assess whether the atelectasis resulting from endotracheal suctioning could be reversed by a postsuction recruitment maneuver. The sheep was used as an experimental model.

METHODS

Animal Preparation

Nine anesthetized and ventilated sheep (weight: 48 ± 3 kg [mean ± SD]) were studied while in the supine position. Anesthesia was induced and maintained with flunitrazepam (4 mg/h), fentanyl (200 µg/h), and vecuronium (4 mg/h). The sheep were intubated with an endotracheal tube incorporating a one-sided port that ended at the distal tip of the tube (Hi-Lo Jet No. 8; Mallinckrodt Inc., Argyle, NY). The animals were ventilated (César Ventilator; Taema, Antony, France) in a volume-controlled mode with constant inspiratory flow (tidal volume [VT] = 10 ml/kg, respiratory rate = 12 breaths/min, respiratory duty cycle [VT/Ttot] = 33%, and positive end-expiratory pressure [PEEP] = 5 cm H2O). A fiberoptic thermodilution catheter (Explorer SV05/CO [venous oxygen saturation/cardiac output], Computer²™; Baxter SA, Maurepas, France) was positioned in the pulmonary artery after denudation of the jugular vein. An arterial carotid catheter was placed after denudation of the artery.

Measurements

Systemic and pulmonary arterial pressures were monitored with calibrated pressure transducers (PX-1X2; Baxter) positioned at the level of the radiology table on which pressure monitoring was done. The in-
traversed pressures measured in the right atrium and pulmonary artery were corrected according to the anteroposterior distance between these structures and the table. Airway pressure was measured with a pressure transducer (P23id; Gould, Cleveland, OH) connected to the distal port of the endotracheal tube. A No. 2 Fleish pneumotachograph (Fleisch, Lausanne, Switzerland) linked to a differential pressure transducer (Schumlerberg, Velizy, France) was placed at the proximal end of the endotracheal tube to measure flow (V) and V̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̍
PaO₂, mm Hg 127

Figure 1. PaO₂ (bars) and QVA/Qt (closed circles) measured at an FIO₂ of 0.3 (upper panel), and PaO₂ (bars) and Qs/Qt (closed circles) measured at an FIO₂ of 1.0 (lower panel) under control conditions (C), immediately after endotracheal suctioning (ETS), and after the recruitment maneuver (RM). The endotracheal suctioning induced a decrease in PaO₂ associated with an increase in QVA/Qt at an FIO₂ of 0.3 and in Qs/Qt at an FIO₂ of 1.0. These effects were reversed by the recruitment maneuver after suctioning. With two-way ANOVA, endotracheal suctioning was found to induce significant decreases in Va/T and S/T that were no different for an FIO₂ of 0.3 and an FIO₂ of 1.0 (absence of interaction).

and Table 1). The decrease in PaO₂ was accompanied by a significant increase in mean pulmonary artery pressure and in Rpv, and by a significant decrease in SaO₂ and SvO₂. The post-suction recruitment maneuver (P_{max} = 35 ± 4 [mean ± SD] cm H₂O) reversed these effects and induced a significant decrease in PaCO₂. All other values remained unchanged.

Pretreatment with pure oxygen. Endotracheal suctioning induced a significant decrease in PaO₂, associated with a significant increase in Qs/Qt (Figure 1 and Table 2). Arterial desaturation was not observed after endotracheal suctioning. The postsuction recruitment maneuver reversed these deleterious effects.

TABLE 1

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Endotracheal Suctioning</th>
<th>Recruitment Maneuver</th>
<th>p Value (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
<td>131 ± 12</td>
<td>133 ± 12</td>
<td>127 ± 12</td>
<td>n.s.</td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>136 ± 12</td>
<td>136 ± 12</td>
<td>133 ± 12</td>
<td>n.s.</td>
</tr>
<tr>
<td>Ppa, mm Hg</td>
<td>18 ± 3.4</td>
<td>19 ± 3.1</td>
<td>16 ± 2.1</td>
<td>0.0005</td>
</tr>
<tr>
<td>Rpv, dyn · s · cm⁻³</td>
<td>168 ± 50</td>
<td>189 ± 56</td>
<td>150 ± 50</td>
<td>0.003</td>
</tr>
<tr>
<td>CO₂ L · min⁻¹</td>
<td>5.1 ± 1.1</td>
<td>5.1 ± 0.9</td>
<td>4.6 ± 0.6</td>
<td>n.s.</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>127 ± 36</td>
<td>93 ± 39</td>
<td>137 ± 40</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>QVa/Qt, %</td>
<td>95 ± 5</td>
<td>87 ± 12</td>
<td>96 ± 2</td>
<td>0.02</td>
</tr>
<tr>
<td>SaO₂, %</td>
<td>71 ± 4</td>
<td>63 ± 8</td>
<td>69 ± 6</td>
<td>0.01</td>
</tr>
<tr>
<td>SvO₂, %</td>
<td>544 ± 92</td>
<td>502 ± 82</td>
<td>486 ± 84</td>
<td>n.s.</td>
</tr>
<tr>
<td>VO₂, ml · min⁻¹</td>
<td>142 ± 21</td>
<td>150 ± 33</td>
<td>137 ± 21</td>
<td>n.s.</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>43 ± 6</td>
<td>45 ± 9</td>
<td>40 ± 8</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Definition of abbreviations: CO = cardiac output; DO₂ = oxygen delivery; HR = heart rate; MAP = mean arterial pressure; Pao₂ = arterial oxygen tension; Ppa = mean pulmonary artery pressure; Qva/Qt = venous admixture; Rpv = pulmonary vascular resistance; SaO₂ = arterial oxygen saturation; SvO₂ = venous oxygen saturation; VO₂ = oxygen consumption.

FIO₂ = 0.3, PEEP = 5 cm H₂O.

TABLE 2

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Endotracheal Suctioning</th>
<th>Recruitment Maneuver</th>
<th>p Value (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
<td>131 ± 9</td>
<td>131 ± 13</td>
<td>134 ± 22</td>
<td>n.s.</td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>139 ± 16</td>
<td>139 ± 15</td>
<td>133 ± 18</td>
<td>n.s.</td>
</tr>
<tr>
<td>Ppa, mm Hg</td>
<td>17 ± 5.9</td>
<td>19 ± 4.6</td>
<td>18 ± 4.7</td>
<td>n.s.</td>
</tr>
<tr>
<td>Rpv, dyn · s · cm⁻³</td>
<td>116 ± 25</td>
<td>142 ± 55</td>
<td>140 ± 38</td>
<td>n.s.</td>
</tr>
<tr>
<td>CO₂ L · min⁻¹</td>
<td>4.7 ± 1.1</td>
<td>4.8 ± 0.8</td>
<td>4.4 ± 0.7</td>
<td>n.s.</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>508 ± 52</td>
<td>263 ± 89</td>
<td>484 ± 80</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>QVa/Qt, %</td>
<td>19 ± 5.4</td>
<td>36 ± 2.3</td>
<td>20 ± 5.7</td>
<td>0.0002</td>
</tr>
<tr>
<td>SaO₂, %</td>
<td>99 ± 1</td>
<td>97 ± 2</td>
<td>99 ± 1</td>
<td>n.s.</td>
</tr>
<tr>
<td>SvO₂, %</td>
<td>84 ± 6</td>
<td>79 ± 9</td>
<td>81 ± 8</td>
<td>0.01</td>
</tr>
<tr>
<td>DO₂, ml · min⁻¹</td>
<td>577 ± 80</td>
<td>565 ± 104</td>
<td>563 ± 54</td>
<td>n.s.</td>
</tr>
<tr>
<td>VO₂, ml · min⁻¹</td>
<td>127 ± 19</td>
<td>122 ± 19</td>
<td>127 ± 20</td>
<td>n.s.</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>35 ± 5.3</td>
<td>38 ± 4.3</td>
<td>35 ± 4.2</td>
<td>0.0005</td>
</tr>
</tbody>
</table>

Definition of abbreviations: CO = cardiac output; DO₂ = oxygen delivery; HR = heart rate; MAP = mean arterial pressure; Pao₂ = arterial carbon dioxide tension; Ppa = mean pulmonary artery pressure; Rpv = pulmonary vascular resistance; SaO₂ = arterial oxygen saturation; SvO₂ = venous oxygen saturation; Qt/Qt = pulmonary shunt; VO₂ = oxygen consumption.

FIO₂ = 1.0, PEEP = 5 cm H₂O.

Figure 2. Percentage of change in respiratory resistance (ΔRrs) at an FIO₂ of 0.3 (upper panel) and an FIO₂ of 1.0 (lower panel) under control conditions (C), immediately after endotracheal suctioning (ETS), and after the recruitment maneuver following suctioning (RM). At both levels of FIO₂, endotracheal suctioning induced an increase in R_{max,rs} (squares), DR_{rs} (circles), and R_{min,rs} (triangles). Using with two-way ANOVA, endotracheal suctioning was found to induce increases in R_{max,rs}, R_{min,rs}, and DR_{rs} that were no different for an FIO₂ of 0.3 and an FIO₂ of 1.0 (absence of interaction).
TABLE 3
CHANGES IN RESPIRATORY MECHANICS AND SURFACE OF BRONCHIAL CROSS-SECTIONAL AREA AFTER ENDOTRACHEAL SUCTIONING IN SEVEN SHEEP

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Endotracheal Suctioning</th>
<th>Recruitment Maneuver</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$R_{max,rs}$, cm H$_2$O · L$^{-1}$ · s$^{-1}$</td>
<td>5.8 ± 2.1</td>
<td>9.1 ± 3.7</td>
<td>6.2 ± 2.8</td>
<td>0.0003</td>
</tr>
<tr>
<td>$R_{max,rs}$, cm H$_2$O · L$^{-1}$ · s$^{-1}$</td>
<td>2.5 ± 1.2</td>
<td>3.3 ± 2.2</td>
<td>2.9 ± 1.9</td>
<td>0.03</td>
</tr>
<tr>
<td>$DR_{rs}$, cm H$_2$O · L$^{-1}$ · s$^{-1}$</td>
<td>3.3 ± 1.3</td>
<td>5.7 ± 2.6</td>
<td>3.3 ± 1.2</td>
<td>0.0008</td>
</tr>
<tr>
<td>$Crs$, ml cm H$_2$O$^{-1}$</td>
<td>46 ± 12</td>
<td>33 ± 6</td>
<td>48 ± 9</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>$P_{max}$, cm H$_2$O</td>
<td>17 ± 2</td>
<td>23 ± 2</td>
<td>16 ± 0.8</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>$P_{e,rs}$, cm H$_2$O</td>
<td>15 ± 1.8</td>
<td>19 ± 2</td>
<td>14 ± 1</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>BCSA, mm$^2$</td>
<td>17 ± 28</td>
<td>11 ± 20</td>
<td>13 ± 18</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

Definition of abbreviations: BCSA = bronchial cross-sectional area; $Crs$ = respiratory compliance; $DR_{rs}$ = lung tissue resistance; $P_{e,rs}$ = end-inspiratory plateau pressure; $P_{max}$ = maximum inspiratory pressure; $R_{max,rs}$ = total respiratory resistance; $R_{min,rs}$ = bronchial resistance.

FIO$_2$ = 0.3, mean ± SD.

Pretreatment with lidocaine (FIO$_2$-0.3). Aerosolization of lidocaine did not modify the hemodynamic or respiratory responses to endotracheal suctioning.

Effects of Endotracheal Suctioning on Respiratory Mechanics
FIO$_2$-0.3. Endotracheal suctioning induced a significant increase in $DR_{rs}$ (76 ± 45%) and $R_{max,rs}$ (33 ± 12%) and $R_{min,rs}$ (18 ± 40%) (Figure 2, and Table 3). $R_{max,rs}$ returned to its baseline value after the post suction recruitment maneuver. Endotracheal suctioning induced a 27 ± 19% decrease in $Crs$, associated with a significant increase in $P_{max}$ and $P_{e,rs}$, which returned to control values after the post suction recruitment maneuver.

Pretreatment with pure oxygen. Endotracheal suctioning significantly increased $DR_{rs}$ (96 ± 77%) and $R_{max,rs}$ (72 ± 63%) (Figure 2). The increase in $R_{max,rs}$ was not significant. Endotracheal suctioning-induced changes in $R_{max,rs}$ and $DR_{rs}$ were not different from those observed at FIO$_2$ = 0.3.

Pretreatment with lidocaine. Aerosolization of lidocaine prevented the increase in $R_{max,rs}$ and $DR_{rs}$ after endotracheal suctioning (Figure 3).

Effects on Surface BCSA
FIO$_2$-0.3. Sixty-four bronchi from seven different sheep, with surface BCSA ranging from 1 mm$^2$ to 145 mm$^2$, were found suitable for analysis (Figure 4 and Table 3). As shown in Table 3, a reduction of the surface BCSA was observed after endotracheal suctioning. The reduction was partly reversed by the post suction recruitment maneuver. An illustrative example is shown in Figure 4, which also shows that the bronchial response to endotracheal suctioning differed according to the size of the airways.

Pretreatment with pure oxygen. As shown in Figure 5, endotracheal suctioning reduced surface BCSA by only 7 ± 32% at FIO$_2$ = 1, versus a reduction of 38 ± 15% at FIO$_2$ = 0.3 (p < 0.01).

Pre-treatment with lidocaine. Changes in surface BCSA with and without pretreatment with inhaled lidocaine were compared in 25 bronchi from three sheep (Figure 5). The endotracheal suctioning-induced decrease in BCSA was attenuated by lidocaine (p < 0.05).

Correlation between respiratory resistance and surface BCSA. There was a significant inverse correlation between changes in surface BCSA and $DR_{rs}$ (Figure 6). No correlation of surface BCSA was found with changes in $R_{min,rs}$ and $R_{max,rs}$.

Effects of Endotracheal Suctioning on Lung Aeration
FIO$_2$-0.3. Lung aeration could be analyzed in eight sheep. In four of these sheep, endotracheal suctioning induced an increase in areas of nonaerated parenchyma of at least 30% which was entirely reversed by the post suction recruitment maneuver.

Pretreatment with pure oxygen. At FIO$_2$-1.0, lung aeration could be analyzed in four sheep. Endotracheal suctioning induced an increase in areas of nonaerated parenchyma of more than 30% in one of these four sheep and in more than 60% in the other three sheep. In all animals, the extension of nonaerated lung regions was greater at FIO$_2$-1.0 than at FIO$_2$-0.3. An illustrative example is shown in Figure 7. Atelectasis was completely reversed by the post suction recruitment maneuver.

Pretreatment with lidocaine (FIO$_2$-0.3). Pretreatment with lidocaine did not prevent endotracheal suctioning-induced atelectasis.

DISCUSSION
In mechanically ventilated sheep with normal lungs, endotracheal suctioning induced a significant reduction in the surface BCSA of bronchi of more than 1 mm in diameter. It also induced an increase in respiratory resistance, an impairment of arterial oxygenation, and the appearance of atelectatic lung areas. The reduction of surface BCSA was attenuated by hyperoxygenation before suctioning, and was abolished by the use of aerosolized lidocaine before suctioning. Atelectasis was aggravated by hyperoxygenation before suctioning, and could only be reversed by a post suction recruitment maneuver.
Endotracheal Suctioning-Induced Reduction of Bronchial Diameter

Following endotracheal suctioning, a significant reduction in surface BCSA was observed at $F_{O_2}$ = 0.3. Two mechanisms are likely to have contributed to this reduction of surface BCSA: (1) a loss of lung volume related to the loss of aeration resulting from the negative pressure applied during the suction procedure; and (2) reflex bronchoconstriction (24).

A constriction of bronchial muscles can be provoked by the direct stimulation of bronchial stretch receptors caused by moving a suction catheter in the airways (13). Endotracheal suctioning-induced arterial oxygen desaturation may also independently increase bronchomotor tone (25–27). Fisher and colleagues studied the responsiveness of airway smooth muscle to hypoxia by administering a 10% $O_2$ mixture to paralyzed and ventilated cats (25). They observed a 50% increase in $R_{max,rs}$. Two different mechanisms accounted for the hypoxia-induced bronchoconstriction: direct stimulation of bronchopulmonary afferent branches of the vagal nerve (28), and the release of bronchoconstrictor mediators by mast cells (29).

The results of the present study strongly suggest that endotracheal suctioning induces a true bronchoconstriction. First, the endotracheal suctioning-induced decrease in surface BCSA...
and increase in respiratory resistance were totally prevented by the administration of lidocaine before suctioning, possibly because of a blockade of stretch receptors. Second, hyperoxegenation before suctioning suppressed the endotracheal suctioning-induced decrease in surface BCSA despite an extension of atelectatic lung regions. When endotracheal suctioning was performed at FIO2 = 0.3, significant arterial oxygen desaturation was observed, and was accompanied by a significant reduction of surface BCSA. When endotracheal suctioning was performed at FIO2 = 1.0, arterial oxygenation desaturation and a reduction of surface BCSA were prevented. These findings suggest that hyperoxegenation before suctioning reverses the component of bronchoconstriction related to endotracheal suctioning-induced hypoxemia. However, neither lidocaine- nor oxygen-induced blockade of the decrease in surface BCSA prevented an endotracheal suctioning-induced increase in Qs/Qr, suggesting that atelectasis was largely involved in the impairment of arterial oxygenation.

**Endotracheal Suctioning-Induced Atelectasis**

Pulmonary atelectasis following endotracheal suctioning and identified by thoracic CT was reported by Brochard and co-workers in patients with acute lung injury (1). In these patients, endotracheal suctioning was associated with an increase in lung attenuation and a 400-ml loss of lung volume. In the present study, in which sheep with normal lungs were studied, atelectatic lung areas were observed in half of the animals at an FIO2 of 0.3 and in all animals at an FIO2 of 1. As expected, the loss of lung aeration from atelectasis was associated with a decrease in lung compliance and a significant increase in the resistance of lung tissue.

Atelectasis may have been caused by three mechanisms: (1) a loss of lung volume related to the high negative pressure applied during the suctioning procedure; (2) the closure of bronchi resulting from endotracheal suctioning-induced bronchoconstriction, and (3) the alveolar collapse associated with the administration of pure O2. Mitzner and coworkers have clearly shown that lung compliance, TLC, and RV decrease after bronchial constriction, indicating that the conducting airways play an important role in the regulation of lung elasticity (30). The increase in Qs/Qr observed at an FIO2 of 1.0 matched the greater extension of atelectatic lung regions evidenced in all sheep, and was in accordance with the notion that pure oxygen can induce alveolar collapse (31, 32).

** Relationships Between Respiratory Resistance and Surface BCSA**

We combined HRCT scanning and the constant inspiratory flow occlusion method to assess the bronchial effects of endotracheal suctioning. The HRCT method has the advantages of being quantitative, reproducible, and noninvasive, allowing an accurate assessment of the surface BCSA of different-sized airways down to a diameter of 1 mm. In addition, it is the only existing method that can show locoregional differences in airway reactivity (33, 34). Classically, in animals or humans whose lungs are mechanically ventilated, resistance of the respiratory system, including respiratory tubing and conducting airways, is measured by applying an end-inspiratory occlusion after the administration of a constant inspiratory flow (16, 17). In the present study, we preferred to do this by manual clamping rather than automatic interruption from the ventilator in order to eliminate errors in the measurement of respiratory resistance related to excessive closing time and incomplete sealing by the automatic interrupter device (35). In addition, we measured airway pressure at the distal tip of the endotracheal tube, and Rmax,rs therefore reflected only the resistive properties of conducting airways and lung tissue. In the study, we showed that an endotracheal suctioning-induced decrease in surface BCSA was associated with a predominant increase in DRrs. After endotracheal suctioning, some parts of the lungs became atelectatic, and various degrees of bronchoconstriction were observed in the bronchial tree. As a consequence, lung elasticity decreased, regional time constants of the lungs became more heterogeneous, and DRrs increased. Very logically, a good correlation was found between changes in DRrs and changes in surface BCSA.

**Prevention and Treatment of Endotracheal Suctioning-Induced Hypoxemia, Bronchoconstriction and Atelectasis**

Different maneuvers have been proposed to prevent endotracheal suctioning-induced hypoxemia in patients with acute lung injury (ALI). It has been shown that a constant insufflation of 12 L/min of O2 administered throughout the period of endotracheal suctioning prevents arterial O2 desaturation and loss of lung aeration. However, this procedure requires the reintubation of critically ill patients with a special endotracheal tube incorporating multiple side ports. The use of a closed suctioning system has been proposed as an alternative (36). However, this may result in partial obstruction of the endotracheal tube and may cause an increase in respiratory resistance (37).

A recruitment maneuver after suctioning has also been recommended to prevent arterial oxygen desaturation during and after endotracheal suctioning (8, 32). The present study confirms that a recruitment maneuver consisting of 20 consecutive breaths, each of 20 ml/kg volume and applied immediately at the end of endotracheal suctioning, reverses atelectasis and the increase in respiratory resistance. It should be pointed out that this beneficial effect was obtained in sheep with normal lungs, and that it may not be observed in animals or patients with ALI. Because bronchial reactivity is often increased and nonaerated pulmonary parenchyma is already present before endotracheal suctioning, these effects could increase hypoxemia and bronchoconstriction and render the recruitment maneuver less efficient. Additional studies are required to determine the type of postsuctioning recruitment maneuver that
would be the most effective for reversing endotracheal suctioning-induced loss of lung aeration and bronchoconstriction in the presence of ALI.

In summary, we found that in normal sheep, hyperoxegenation before suctioning prevents endotracheal suctioning-induced bronchoconstriction and arterial oxygen desaturation, but not atelectasis or an increase in Qs/Qt. Aerosolized lidocaine prevents the bronchoconstriction related to suctioning-induced reflex bronchial stimulation, but does not prevent atelectasis or impairment of arterial oxygenation. A recruitment maneuver after suctioning appears to be the only method that can reverse bronchoconstriction, the increase in Qs/Qt and atelectasis resulting from endotracheal suctioning.

References