Effects of Tracheal Suctioning on Respiratory Resistances in Mechanically Ventilated Patients*

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**Objective:** To evaluate the effects of tracheal suctioning (TS) on respiratory resistances in sedated critical care patients receiving mechanical ventilation (MV).

**Setting:** Surgical ICU of Bichat Hospital, Paris.

**Patients and participants:** Thirteen sedated critical care patients receiving MV for various conditions.

**Measurements and results:** Airway resistances ($R_1$), airway and pulmonary resistances ($R_2$), and intrinsic positive end-expiratory pressure (PEEPint) were measured according to the end-inspiratory and end-expiratory occlusion methods before and after TS. $R_1$ and $R_2$ increased by 49.1% and 46.3%, respectively, 0.5 min after TS ($p<0.01$) but returned to baseline values at 1 min without any change thereafter. PEEPint decreased progressively following TS to reach a significant level ($-13.3\%$) at 10 min ($p<0.05$) and was persistently reduced at 30 min ($p<0.01$). Nine patients received 500 µg of inhaled albuterol before another suctioning procedure. $R_1$ and $R_2$ decreased by 11.5% and 9.9%, respectively, 20 min after inhalation ($p<0.05$), but the $R_1$ and $R_2$ initial increase following TS did not differ between the two suctioning procedures.

**Conclusions:** TS evokes only a transient bronchoconstrictor response, but thereafter, does not reduce respiratory resistances below presuctioning values. However, the decrease of PEEPint following TS suggests an increase of expiratory flow. Effective $\beta_2$-adrenergic receptor blockade fails to suppress the TS-induced bronchoconstrictor response. (CHEST 1998; 113:1335-38)

**Key words:** $\beta_2$-adrenergic agonists; mechanical ventilation; respiratory mechanics; respiratory reflexes; tracheal suctioning

**Abbreviations:** MV = mechanical ventilation; $P_1$ = inflection point; $P_2$ = plateau pressure; PEEP = positive end-expiratory pressure; PEEPint = intrinsic PEEP; PEEPtot = total PEEP; $P_{peak}$ = maximum inspiratory pressure; $R_1$ = airway resistance; $R_2$ = pulmonary resistance; TS = tracheal suctioning; $V_{ins}$ = inflation flow

Mechanical ventilation (MV) impairs airway secretion clearing in critical care patients. Furthermore, cough reflex and mucociliary clearance are depressed by sedation, glottic closure inability, high cuff pressure, or tracheal mucosal damage. Therefore, tracheal suction (TS) is periodically warranted in MV patients to prevent airway obstruction, and to a lesser extent, to decrease the work of breathing resulting from retained secretions.

However, TS can lead to serious and even life-threatening complications. Hypoxemia is the most commonly reported complication, but cardiac arrhythmias and even cardiac arrest have been described. It can equally increase oxygen consumption and could lead to silent tissular ischemia in hemodynamically compromised patients. Tracheal mucosal damage and impaired mucociliary clearance induced by the suction catheter could also enhance tracheal bacterial colonization and then promote ventilator-associated pneumonia.

Although TS is performed routinely, no clinical evidence supports its benefit on respiratory mechanics and especially on the expected decrease of respiratory resistances resulting from secretion removal. Furthermore, tracheal receptor stimulation elicited by this maneuver could increase rather than de-
crease respiratory resistances. The aim of this study was to assess the effects of TS on respiratory resistances in critical care patients receiving MV.

**Materials and Methods**

Thirteen ICU patients requiring MV for various conditions were studied. The investigation was conducted according to the local ethics committee guidelines, and informed consent was obtained from each patient or relative.

All patients were sedated with IV midazolam and fentanyl. They were well adapted to the ventilator and no triggering activity was detected.

MV was performed through a cuffed translaryngeal tube or through a tracheostomy tube. MV was achieved in a volume-controlled mode with a ventilator (Servo 900 C; Siemens-Elema, Solna, Sweden). The latter delivered a constant inflation flow and an accurate tidal volume and allowed end-inspiratory and end-expiratory occlusions. No changes in individual ventilatory settings were made for the purpose of the study. All patients were in the supine position with a 30° head lift inclination.

Tracheal pressures were measured using a 2.7-mm-diameter multiperforated catheter positioned 1 cm above the distal tip of the tracheal tube and connected to a pressure transducer (Valdyne CD 15; Northridge, Calif). Flow and volume values were obtained from the electronic signal of the ventilator. Tracheal pressures and flow were recorded continuously on a polygraph (Gould) at a paper speed of 10 mm/s (Gould ES 1000; Gould Instruments; Balainvilliers, France). Pressure transducer was calibrated with a water column. Flow was calibrated using incremental airflow delivered by an air flowmeter. Calibrations were performed before each patient record.

Respiratory mechanics were studied according to the end-inspiratory occlusion method. After an end-inspiratory occlusion, an abrupt decrease in tracheal pressure from a maximum pressure (Ppeak) to an inflection point (P1) is observed and followed by a more gradual decrease to a plateau pressure (P2). P2 corresponds to the elastic recoil pressure of the respiratory system. The initial pressure drop (Ppeak-P1) reflects the loss of pressure attributable to airway resistance (Rl) during the constant inflation flow (VIns) preceding occlusion, whereas total pressure decrease (Ppeak-P2) represents both airway and pulmonary resistances (Rl). P1 was measured at the inflection point and P2 was measured 3 s after flow interruption. R1 was calculated as (Ppeak-P1) x Vins and R2 as (Ppeak-P2) x Vins. Total positive end-expiratory pressure (PEEPtot) was measured as previously described.13 3 s after an end-expiratory occlusion. Intrinsic PEEP (PEEPint) was calculated as the difference between PEEPltot and extrinsic PEEP and compliance as the ratio of inspired tidal volume to (PEEPtot-P2-PEEPint). All Ppeak, P1, P2, and PEEPltot values were an average of at least two respiratory cycle measurements except for those performed 0.5 and 1 min following the removal of the suction catheter, which were obtained from a single one. Each occluded cycle was separated by at least two normal respiratory cycles except for those 0.5 and 1 min, which were separated by only one normal cycle.

TS was achieved via a 4.7-mm-diameter catheter (Sherwood Medical; Tullamore, Ireland) through a suction catheter adapter (Sontek Medical; Hingham, Md) allowing no flow interruption during the procedure. It took place during usual nursing care of the patient. Patients did not receive any bronchodilator therapy during the last 6 h. The catheter was introduced until a resistance was met. Negative pressure ( 80 cm H2O) was then applied, and the catheter was rotated and gradually removed. Suction was interrupted when the catheter reached the external outlet of the tube.

Airway pressures were measured, and respiratory resistances were calculated before TS and 0.5, 1, 3, 5, 10, 15, and 30 min after the removal of the suction catheter.

In nine patients, the effects of TS were studied twice, 1 day apart, in a randomized crossover fashion. Measurements were performed once before and immediately after TS as previously described. On the other day, measurements were first done before and 20 min following the inhalation of 500 µg of albuterol (five puffs) to assess the effects of albuterol on bronchial tone; then, the patients underwent TS, and measurements were repeated (see above). Albuterol was delivered with a metered-dose inhaler through a spacer device (Aerovent; Trudell Medical; London, Ontario, Canada) connected between the inspiratory line and the Y-piece. This device has been reported to provide a 32% pulmonary deposition in an in vitro model.12

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**Table 1—Patient Characteristics**

<table>
<thead>
<tr>
<th>Patient/Age, yr/sex</th>
<th>Diagnosis</th>
<th>Duration of MV, d</th>
<th>Compliance, mL/cm H2O</th>
<th>PaO2/FIO2, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/60/M</td>
<td>CHF</td>
<td>10</td>
<td>46.0</td>
<td>375</td>
</tr>
<tr>
<td>2/35/M</td>
<td>AP</td>
<td>28</td>
<td>54.8</td>
<td>343</td>
</tr>
<tr>
<td>3/58/M</td>
<td>Trauma</td>
<td>34</td>
<td>80.6</td>
<td>335</td>
</tr>
<tr>
<td>4/81/F</td>
<td>Sepsis</td>
<td>5</td>
<td>43.6</td>
<td>431</td>
</tr>
<tr>
<td>5/50/M</td>
<td>Trauma</td>
<td>3</td>
<td>43.6</td>
<td>199</td>
</tr>
<tr>
<td>6/61/M</td>
<td>Postop</td>
<td>3</td>
<td>62.2</td>
<td>343</td>
</tr>
<tr>
<td>7/58/M</td>
<td>Trauma</td>
<td>44</td>
<td>49.9</td>
<td>308</td>
</tr>
<tr>
<td>8/63/F</td>
<td>NP</td>
<td>5</td>
<td>36.1</td>
<td>390</td>
</tr>
<tr>
<td>9/35/M</td>
<td>AP</td>
<td>13</td>
<td>53.6</td>
<td>247</td>
</tr>
<tr>
<td>10/58/M</td>
<td>NP</td>
<td>13</td>
<td>65.1</td>
<td>200</td>
</tr>
<tr>
<td>11/60/M</td>
<td>CHF</td>
<td>20</td>
<td>49.8</td>
<td>216</td>
</tr>
<tr>
<td>12/75/F</td>
<td>POP</td>
<td>5</td>
<td>25.6</td>
<td>129</td>
</tr>
<tr>
<td>13/54/M</td>
<td>NP</td>
<td>15</td>
<td>75.5</td>
<td>525</td>
</tr>
<tr>
<td>Mean±SD</td>
<td></td>
<td>58±13 (age)</td>
<td>15.2±15.9</td>
<td>52.8±15.2</td>
</tr>
</tbody>
</table>

*CHF= congestive heart failure; AP= acute pancreatitis; NP= nosocomial pneumonia; Trauma= polytraumatism; Postop= postoperative care; POP= postoperative peritonitis; FIO2= fraction of inspired oxygen.
Statistical Analysis

Data are expressed as mean±SD. Comparison of quantitative variables used a Mann-Whitney U test, a paired Wilcoxon t test, or a one-way analysis of variance for repeated measurements followed by post hoc analysis for comparison to baseline values. A p value <0.05 was considered as statistically significant.

RESULTS

Demographic data regarding the patients in the study are summarized in Table 1.

R1 increased from 7.6±5.0 to 10.4±7.0 cm H2O/L s (p<0.01) and R2 from 10.9±5.2 to 15.3±7.1 cm H2O/L s (p<0.01) 0.5 min after tracheal suction (Table 2). R1 and R2 returned to baseline values 1 min following suction without any change thereafter. There was no change in the (R2-R1)/R2 ratio during the 30 min recording, suggesting that the increase in R1 paralleled this in R2.

PEEPint dropped from 3.70±4.10 cm H2O at baseline to 3.35±3.76 cm H2O 10 min following TS (p<0.05). It was persistently reduced at 30 min (p<0.01) (Table 2).

In the subgroup of nine patients who received albuterol, a significant 11.5% decrease of R1 (p<0.05) and 9.9% of R2 (p<0.05) was observed 20 min after inhalation (Table 3). By contrast, there was no statistically significant difference of R1 or R2 percentage increase from presuctioning value (ΔR1 and ΔR2) between the two suctioning procedures (Table 3).

DISCUSSION

In this study, we found that TS evoked a potent bronchoconstrictor response for a short period. Indeed, R1 and R2 increased >45%, 30 s following this procedure but returned to baseline values 1 min later. More surprisingly, respiratory resistances did not decrease below presuctioning values thereafter, as could have been expected, even on the late recordings (Table 2). Dohi and Gold13 reported a similar evolution of respiratory resistances following mechanical stimulation of the trachea with a suction catheter in anesthetized healthy men undergoing surgery. However, their last recording was performed only 5 min after suction, and a delayed effect on respiratory resistances could not be ruled out. Furthermore, these data were obtained from patients ventilated for a few hours, whereas our patients had been ventilated for many days.

We did not observe a decrease of respiratory resistances below presuctioning values after the procedure. This may be related to the fact that the TS catheter usually reaches the carina or the main bronchi and is thus effective only in clearing proximal airways, whereas medium and small bronchi, up to the seventh generation, are the main site of respiratory resistances.14 However, we observed a late decrease of PEEPint (Table 2) reflecting a decreased expiratory airflow obstruction.11 The mechanism involved in this delayed improvement in expiratory flow remains to be determined. The end-inspiratory occlusion method uses inspiratory pressures and could fail to identify expiratory airflow limitation.15 The absence of decrease of respiratory resistances following TS, contrasting with a persistently reduced PEEPint, could result from this technical limitation.

Mechanical tracheal stimulation with a suction catheter evokes a potent bronchoconstrictor response as demonstrated previously in anesthetized cats16 or healthy humans.13 It allows an increase in rigidity that renders the airway less susceptible to

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Table 3—Effects of 500 μg Inhaled Albuterol on R1, R2, ΔR1, and ΔR2

<table>
<thead>
<tr>
<th></th>
<th>Before Albuterol</th>
<th>After Albuterol</th>
</tr>
</thead>
<tbody>
<tr>
<td>R1, cm H2O/Ls</td>
<td>8.0±4.9</td>
<td>6.3±3.5†</td>
</tr>
<tr>
<td>R2, cm H2O/Ls</td>
<td>10.9±5.6</td>
<td>8.9±3.9†</td>
</tr>
<tr>
<td>TS Without Albuterol</td>
<td>55.4±69.9</td>
<td>45.5±62.4</td>
</tr>
<tr>
<td>TS With Albuterol</td>
<td>49.8±43.8</td>
<td>34.4±35.5</td>
</tr>
</tbody>
</table>

*Data are expressed as mean±SD.
†p<0.05 vs baseline.

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Table 2—Changes in R1, R2, and in PEEPint after TS

<table>
<thead>
<tr>
<th>Time, min</th>
<th>0</th>
<th>0.5</th>
<th>1</th>
<th>3</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>R1, cm H2O/Ls</td>
<td>7.6±5.0</td>
<td>10.4±7.0†</td>
<td>7.7±5.1</td>
<td>6.4±3.0</td>
<td>6.3±2.8</td>
<td>6.6±3.8</td>
<td>6.6±3.5</td>
<td>7.2±4.3</td>
</tr>
<tr>
<td>R2, cm H2O/Ls</td>
<td>10.9±5.2</td>
<td>15.3±7.1†</td>
<td>11.6±5.8</td>
<td>9.7±4.2</td>
<td>9.6±3.5</td>
<td>9.9±4.2</td>
<td>9.7±4.2</td>
<td>10.4±5.0</td>
</tr>
<tr>
<td>PEEPint, cm H2O</td>
<td>3.70±4.10</td>
<td>3.39±4.20</td>
<td>3.49±4.15</td>
<td>3.51±3.90</td>
<td>3.44±3.92</td>
<td>3.35±3.76†</td>
<td>3.37±3.70†</td>
<td>3.32±3.60†</td>
</tr>
</tbody>
</table>

*Data are expressed as mean±SD.
†p<0.01 vs baseline.
‡p<0.05.
collapse during expulsive reflexes, a better impaction and absorption capability of the larger airways for foreign particles and aerosols, and a decrease in dead-space ventilation.\textsuperscript{17} Although this bronchoconstrictor response represents an effective respiratory defense mechanism in normal subjects, it could be attractive to block it in mechanically ventilated patients in order to prevent bronchospasm and/or pulmonary barotraumatic lesions related to increased airway pressure. The effective neural pathway is mediated by acetylcholine,\textsuperscript{18} and inhaled anticholinergic agents such as ipratropium bromide effectively attenuate evoked bronchoconstriction.\textsuperscript{19} It can also be blocked by inhaled or IV local anesthetics,\textsuperscript{20,21} although the mechanisms involved are not fully determined. Inhaled $\beta_2$-adrenergic agonists are frequently administered to ICU patients because of their short onset of action and their absence of cardiac or neurologic toxic reactions, two points of prime importance for critically ill patients. In the subgroup of nine patients, albuterol significantly reduced $R_1$ and $R_2$ but failed to attenuate $\Delta R_1$ or $\Delta R_2$, indicating that despite the achievement of an effective blockade of $\beta_2$-receptors, it was not effective in suppressing the TS-induced bronchoconstrictor response (Table 3).

Some patients studied had underlying bronchopulmonary disease. They might have a more potent bronchoconstrictor response to TS than healthy subjects. Regarding the limited number of patients studied, we cannot rule out this hypothesis. Nevertheless, previous data of Dohi and Gold\textsuperscript{13} about the effects of mechanical irritation of the trachea suggest that patients with reactive airway disease do not respond more vigorously to TS than healthy subjects.\textsuperscript{13} Indeed, they demonstrated that the magnitude of the bronchoconstrictor response elicited by tracheal stimulation did not differ between healthy subjects and patients with chronic obstructive bronchopulmonary disease undergoing surgery under general anesthesia.

In summary, TS evokes only a transient bronchoconstrictor response but does not thereafter reduce respiratory resistances below presuctioning values. However, the decrease in PEEPint following TS suggests that expiratory flow has increased. Effective $\beta_2$-adrenergic receptor blockade fails to suppress the TS-induced bronchoconstrictor response.

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REFERENCES