Unsuspected Hemodynamic Alterations during Endotracheal Suctioning*


Endotracheal suctioning of intubated patients is associated with hemodynamic complications including arterial hypoxemia, cardiac arrhythmias, hypotension and even death. Prior investigations of this subject focus primarily on arterial hypoxemia. Our observations of ETS revealed significant falls in the mixed SvO₂ and we postulated that alterations in VO₂ or CO must be occurring. This study was then designed to determine the alterations in CO, VO₂, SaO₂ and the resulting effect on SvO₂ during ETS. Ten critically ill intubated patients with pulmonary artery catheters in place, were studied during routine ETS by the nursing staff. We found that ETS produced a significant decrease in SvO₂ which was predominantly due to an increased VO₂ accompanied by an inadequate rise or even fall in CO. Alterations in SaO₂ appeared to be modest and were insensitive indicators of alterations in SvO₂. (Chest 1989; 95:162-65)

ETS = endotracheal suctioning

Endotracheal intubation results in impaired cough and retention of respiratory tract secretions. Intubated patients require ETS in order to enhance clearance of secretions and prevent atelectasis. However, ETS is associated with a number of complications including direct damage to the bronchial epithelium from the suctioning catheter, and as well as hemodynamic complications such as arterial hypoxemia, cardiac arrhythmias, hypotension and even sudden death. Previous investigations of complications associated with ETS have focused on arterial hypoxemia as the primary cause of the morbidity and mortality related to ETS, and methods to minimize ETS-induced hypoxemia have been suggested.

During routine care of critically ill patients receiving mechanical ventilation we noted large decreases in mixed SvO₂ occurring during ETS (Fig 1). Decreases in SvO₂ exceeded changes in SaO₂ and suggested to us that changes in CO or VO₂ must also be occurring. Declines in CO or increases in VO₂ are frequently difficult to recognize and can occur without change in arterial O₂ levels, a frequently monitored parameter during ETS. We hypothesized that unrecognized changes in CO and VO₂ could be responsible for some of the life-threatening complications associated with ETS. This study was designed to determine the contributions of alterations in CO, VO₂ and SaO₂ in the decline of SvO₂ during ETS.

Materials and Methods

Patient Population

Our study group consisted of ten acutely ill medical patients (six males) requiring endotracheal intubation and positive pressure ventilation. Seven subjects had hypoxemic respiratory failure and three, hypercapnic respiratory failure. All patients had a severity of illness requiring pulmonary artery catheter placement during the course of their care. At the time of study, patients were hemodynamically stable and did not experience falls in blood pressure with ETS.

Methods

ETS was performed by the nursing staff when clinically indicated as part of routine clinical care. The endotracheal tube was disconnected from the ventilator, the patients' lungs were inflated twice with a 1 l. Ambu bag filled with a continuous flow of 100 percent oxygen, and 3 ml of sterile normal saline solution was instilled into the endotracheal tube. Suctioning was performed with a 14 Fr, 22-inch suction catheter inserted into the airway until resistance was met. The catheter was withdrawn and continual suction applied while simultaneously rotating the catheter. Suction from the wall

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Figure 1. A continuous reading of mixed SvO₂ during three episodes of ETS in a patient with hypoxemic respiratory failure. The first arrow indicates onset of suctioning. Note the prominent fall in SvO₂ from 62 to 40 percent. The second arrow indicates the momentary rise in SvO₂ resulting from pre-ETS hyperinflation with 100 percent oxygen. Note the biphasic fall in SvO₂ which follows the two passes of the suction catheter. The smallest divisions on the X axis represent 1 min. It required 5 to 7 min for SvO₂ to return to baseline values following ETS.
Table 1 — Clinical Status of Study Patients: Baseline Values

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, yr</th>
<th>Diagnosis</th>
<th>FrO₂</th>
<th>PEEP (cmH₂O)</th>
<th>P₀₂ (mm Hg)</th>
<th>P₁₉₀₂ (mm Hg)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>67</td>
<td>Ovarian cancer, sepsis, comatose</td>
<td>0.28</td>
<td>5</td>
<td>54</td>
<td>39</td>
<td>7.36</td>
</tr>
<tr>
<td>2</td>
<td>49</td>
<td>Massive hemoptysis</td>
<td>0.50</td>
<td>0</td>
<td>90</td>
<td>40</td>
<td>7.40</td>
</tr>
<tr>
<td>3</td>
<td>70</td>
<td>Anoxic encephalopathy, comatose</td>
<td>0.50</td>
<td>5</td>
<td>94</td>
<td>30</td>
<td>7.52</td>
</tr>
<tr>
<td>4</td>
<td>26</td>
<td>Sepsis, Hodgkins lymphoma</td>
<td>0.95</td>
<td>18</td>
<td>95</td>
<td>40</td>
<td>7.40</td>
</tr>
<tr>
<td>5</td>
<td>36</td>
<td>Vasculitis</td>
<td>0.65</td>
<td>10</td>
<td>70</td>
<td>40</td>
<td>7.40</td>
</tr>
<tr>
<td>6</td>
<td>71</td>
<td>Aspiration</td>
<td>0.60</td>
<td>8</td>
<td>61</td>
<td>32</td>
<td>7.45</td>
</tr>
<tr>
<td>7</td>
<td>68</td>
<td>Pneumonia</td>
<td>0.50</td>
<td>5</td>
<td>63</td>
<td>36</td>
<td>7.51</td>
</tr>
<tr>
<td>8</td>
<td>59</td>
<td>Pulmonary edema</td>
<td>0.35</td>
<td>10</td>
<td>57</td>
<td>33</td>
<td>7.47</td>
</tr>
<tr>
<td>9</td>
<td>81</td>
<td>Cardiomyopathy postresuscitation</td>
<td>0.60</td>
<td>5</td>
<td>68</td>
<td>35</td>
<td>7.36</td>
</tr>
<tr>
<td>10</td>
<td>61</td>
<td>Pneumonia</td>
<td>0.40</td>
<td>5</td>
<td>66</td>
<td>30</td>
<td>7.50</td>
</tr>
</tbody>
</table>

Mean values ± SD 0.55 ± 0.17 7 ± 15 72 ± 15 35.5 ± 4.1 7.44 ± 0.06

outlet provided a maximum negative pressure of 120 mm Hg and a continuous flow of 20 L/min when fully patent. This procedure was performed twice within approximately 5 min.

Continuous monitoring during the study period included pulse rate, SaO₂ by pulse oximetry (Nellcor N-100 finger oximeter) and mixed SvO₂ (OPTICATH, Oximetrix Corp., Mountain View, CA). Thermodilution CO was measured (mean value of three determinations) prior to ETS and following ETS when SvO₂ was near its nadir. Oxygen consumption was calculated using the Fick equation:

\[ \text{VO}_2 = \text{CO} \times (A-V) \text{O}_2 \text{ content} \]

(ml/min) = (ml/min) × (ml/100 ml)

The arterial and venous O₂ content were calculated using the following equation:

\[ \text{O}_2 \text{ content} = \text{Hgb} \times 1.34 \times \% \text{ saturation} \]

Hgb ml/dl = (gm/dl) × (ml O₂/gm) × (%) (ml)

Because dissolved O₂ content is small, we did not use it in the calculations.

Statistical Analysis

Student's t-test (two-tailed) was used to compare resuscutioning values with values obtained following ETS. A p value less than 0.05 was considered significant.

RESULTS

Table 1 lists the clinical status of the study patients. The ages ranged from 26 to 81 years, with a mean age of 54 years. Causes of respiratory failure included COPD, massive hemoptysis, pulmonary edema, sepsis and aspiration. Two patients were comatose at the time of the study. The FrO₂ ranged from 0.28 to 0.95 and the level of PEEP was from 0 to 18 cmH₂O with a mean of 7 cmH₂O.

Table 2 shows the hemodynamic data before and after ETS. The mean SaO₂ decreased from 96 ± 2 percent to 92 ± 7 percent, but this was not statistically significant. The SvO₂ declined in nine of ten patients during ETS (mean values: 68 ± 3 percent to 54 ± 6 percent [p<0.05]). The change in CO with ETS varied widely with the mean CO rising slightly but insignificantly. The V₀₂ increased in all patients during ETS (281 ± 143 ml/min to 357 ± 165 ml/min [p<0.05]).

Figure 2 displays the poor correlation between the changes in SaO₂ and the changes in SvO₂.

Figure 3 displays changes in V₀₂ and CO associated with ETS. There is good correlation between these values. Of note are the two patients with the smallest increase in V₀₂ but significant drops in CO. These two patients were comatose at the time of the study.

Table 3 further examines the hemodynamic status of these two comatose patients during ETS. Note that the large decrease in SvO₂ is associated with little or no change in SaO₂, and no significant rise in V₀₂. Therefore, all of the decrease in SvO₂ occurred because of decline in CO. The etiology of the fall in CO in these patients is unclear. Because they were comatose, neither responded with cough or agitation to ETS and both had increases in heart rate during ETS as did all study patients.

DISCUSSION

In this study we found large decreases in SvO₂...
frequently accompanying ETS, and increased O₂ consumption was primarily responsible. Changes in SaO₂ were small with ETS and did not correlate with changes in SvO₂ (Fig 2). During the ETS procedure, we attempted to minimize falls in arterial oxygen levels by preoxygenation with FIO₂ = 1.0 and short duration of suctioning as recommended by others.10,16,17 Indeed, we were successful using these techniques because SaO₂ declined modestly from 96 to 92 percent for our patients. Despite the success in limiting the falls in SaO₂ we did not prevent large decreases in SvO₂. Clinicians may be misled with a false sense of security if only arterial oxygen levels are monitored during ETS. Profound tissue hypoxemia may occur despite adequate SaO₂, particularly in the agitated and combative patients who have the greatest increase in O₂ demands.

The mean increase in VO₂ during ETS was 27 percent, with a range of 7 to 70 percent compared with baseline values. The most marked increases in VO₂ were associated with strenuous muscular activity and coughing induced by ETS. These responses are not altogether preventable but realizing the potentially large decrease in SvO₂ accompanying ETS, efforts should be made to minimize the rise in VO₂ during ETS. Nurse-patient interaction to reduce anxiety, sedation, shorter suctioning periods and close obser-

![Figure 3. Change in CO vs change in VO₂. The two points in the lower left corner represent the two comatose patients (r = 0.87 and p = 0.001).](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21587/)

Table 3—Data for the Two Comatose Patients

<table>
<thead>
<tr>
<th></th>
<th>Patient 1</th>
<th>Patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before ETS</td>
<td>After ETS</td>
</tr>
<tr>
<td>SaO₂ (%)</td>
<td>95</td>
<td>90</td>
</tr>
<tr>
<td>SvO₂ (%)</td>
<td>68</td>
<td>48</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>3.0</td>
<td>2.1</td>
</tr>
<tr>
<td>VO₂ (mL/min)</td>
<td>120</td>
<td>127</td>
</tr>
</tbody>
</table>

viation of the patients’ responses to ETS may be valuable in blunting the rise in VO₂.

We noted a strong correlation between rise in CO and increased VO₂ during ETS (Fig 3). Patients demonstrating vigorous cough, agitation or resistance to ETS had the largest increase in VO₂ and CO. Despite the increase in O₂ delivery to tissues with ETS, the rise in VO₂ outstripped the O₂ supply, resulting in substantial falls in SvO₂. The inadequate rise in CO accompanying the increased VO₂ resulting from ETS could have untoward effects in critically ill patients. In a patient with preexisting coronary artery disease or critical aortic stenosis this could result in myocardial infarction, hypotension or sudden death, complications known to be associated with ETS.7,9

The two comatose patients in our study are of interest because they had the smallest rise in VO₂ accompanying ETS and the greatest decrease in CO. These patients may help in understanding the mechanisms of alteration in CO during ETS. A possible mechanism for a decrease in CO is vagally mediated bradycardia;4 however, neither of our patients developed bradycardia, and in fact both experienced tachycardia during ETS.19,20 Because they were comatose, neither coughed or became agitated. A remaining possibility is a decrease in intrathoracic pressure resulting from suctioning intrathoracic gas during ETS, causing intrathoracic venous pooling and a resultant decrease in left ventricular preload. We have no measurements of intrathoracic pressure during ETS in our patients but others have calculated that substantial declines in intrathoracic pressure do occur with ETS.21 The actual drop in pressure depends on suction pressure, catheter length and diameter, as well as endotracheal tube length and diameter.

While the two comatose patients had a decreased CO during ETS, seven of eight conscious patients had increases in CO. The exact mechanism is unclear; however, we believe that increased intrathoracic pressure from vigorous coughing may offset a pressure drop from suctioning. A vigorous cough combined with anxiety and agitation (providing a rise in catecholamines) could result in a rise in CO.

Closed suctioning systems suggested previously to diminish the arterial desaturation from ETS could be deleterious, especially in the less responsive patient. In a closed system, atmospheric gas is prevented from entering the endotracheal tube during ETS.10,11,14,18 This may potentiate the decrease in intrathoracic pressure and thereby possibly depress CO. While this deserves consideration, it will require further study for verification.

Our study advances understanding of the physiologic and hemodynamic consequences of ETS in critically ill patients. Attempts to minimize these hemodynamic changes may be beneficial to the critically ill

Hemodynamic Alterations during Endotracheal Suctioning (Walsh et al)
patient. Prevention of the hemodynamic complications associated with ETS includes consideration of the following: (1) attempting to decrease patient anxiety and fear of ETS by thorough explanation of the procedure; (2) consideration for judicious use of sedation when undue agitation persists during ETS; (3) analysis of the "cost/benefit" of ETS, i.e., does the return of secretions balance the hemodynamic stress; and (4) close observation of SaO₂ and SvO₂ during ETS when possible.

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