Effect of tracheal suction on oxygenation, circulation, and lung mechanics in newborn infants

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SUMMARY  Transcutaneous Po2, heart rate, and aortic blood pressure were measured in 10 mechanically-ventilated newborn infants to assess the degree and course of hypoxaemia, and to monitor the cardiovascular and respiratory changes during tracheal toilet. Five infants weighed <1250 (mean 994) g, and 5 infants weighed >1750 (mean 2216) g. During tracheal suction the TcPo2 fell from 68 ± 27 (x ± SD) to 43 ± 23 mmHg, and the heart rate from 144 ± 8 to 123 ± 25 beats/minute, but the blood pressure increased from 44 ± 24 to 49 ± 24 mmHg. Hypoxaemia (TcPo2 <50 mmHg) occurred in 7 of 8 initially well-oxygenated infants when suctioned. The decrease in TcPo2 was similar for both groups of infants. It was greater in infants with controlled ventilation and an FIO2 \( \geq 0.8 \) than in infants with intermittent mandatory ventilation and an FIO2 \(<0.8\). The TcPo2 fall correlated well with the TcPo2 during the control period but not during the time that the infants were disconnected from the respirator. A critical re-evaluation of routine tracheal toilet is needed.

Newborn infants who are intubated in order to be ventilated by a respirator have to be routinely suctioned to free them from lung secretions and to prevent the endotracheal tube from becoming blocked. For this tracheal toilet, respirators that have a closed air supply system have to be disconnected from the patient. This interruption of oxygen supply and ventilation often leads to transient hypoxaemia, bradycardia, arrhythmias, decreased lung compliance, and negative intrapulmonary pressure. Even death after tracheal suction has been reported. But the extent and course of hypoxaemia have not been studied, and nor have the cardiovascular and respiratory changes that take place during tracheal suction. We therefore investigated the effects of tracheal toilet on transcutaneous Po2 (TcPo2), heart rate, and blood pressure in newborn infants in an intensive care nursery. We paid special attention to the cardiovascular response of each infant to the sudden hypoxia induced by tracheal suction.

Material and methods

We studied 10 newborn infants with respiratory distress taken at random from our intensive care nursery. Each was being ventilated via a tracheal tube with a Dräger Spiromat* or a Bourns LS 104†

*Drägerwerk AG., Lübeck, West Germany.
†Bourns, Inc., life systems division, Riverside, California, USA.
the open endotracheal tube and ventilated by means of an Ambu-bag supplied with oxygen. For suctioning a negative pressure of about 200 cm water and catheters of 1.0-1.5 mm (inside diameter) and 1.5-2.0 mm (outside diameter) were used. Three measurements were obtained for each infant with a mean time of 11-8 hours (range 4 to 36). When the infant was in a clinically stable condition we recorded the \( \text{TcPo}_2 \) and the aortic blood pressure, and then we did the same during the tracheal toilet, and during the 5-minute period after bag ventilation. The \( \text{TcPo}_2 \) was measured at a skin temperature of 44°C with a Dräger-Hellige Oxyapart with the electrode fixed to the right side of the chest. The heart rate was determined either from an electrocardiogram or from blood pressure tracing. The pressures in the aorta and tracheal tube were measured with pressure transducers (Statham Db 23). The tidal volume was either read from the volume-constant respirator or was determined by pneumotachography (using a Fleisch pneumotachograph 00, a differential pressure transducer (Statham P 15), and electronic integrating circuits). A Beckman R 611 polygraph registered all variables simultaneously.

We checked the calibrations and scale linearity before and after each measurement.

The time immediately before the respirator was disconnected, the time at the end of suctioning, and at 1, 2, and 5 minutes after bag ventilation were chosen for the analysis. The time that the respirator was disconnected was chosen as representing the time of suctioning because the precise time could be read from the tracings. Heart rate and blood pressure were calculated from a 10-second record. The paired Student's \( t \) test was used for statistical analysis.

### Results

The average changes of \( \text{TcPo}_2 \), heart rate, and blood pressure in the 10 infants are shown in Table 2. During tracheal suction the \( \text{TcPo}_2 \) and heart rate decreased but the blood pressure increased. Bag ventilation with pure oxygen reversed all these changes. Five minutes after bag ventilation both the \( \text{TcPo}_2 \) and blood pressure were higher than during the control period. Each patient reacted to tracheal toilet in a similar manner in all 3 studies done on him.

### The course of the \( \text{TcPo}_2 \) in each patient is shown in

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**Table 1**  Details of the 10 infants

<table>
<thead>
<tr>
<th>Cases</th>
<th>Weight at study (g)</th>
<th>Gestational age (weeks)</th>
<th>Age at study* (days)</th>
<th>Diagnosis</th>
<th>( \text{F}_{102} ) at study</th>
<th>Type of ventilation</th>
<th>Duration of disconnection* (seconds)</th>
<th>Duration of bag ventilation* (seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>1020</td>
<td>28</td>
<td>2</td>
<td>RDS</td>
<td>1.00</td>
<td>Controlled</td>
<td>22</td>
<td>18</td>
</tr>
<tr>
<td>2</td>
<td>980</td>
<td>27</td>
<td>0-6</td>
<td>Aspiration</td>
<td>0.81</td>
<td>IMV</td>
<td>41</td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>1000</td>
<td>27</td>
<td>0-5</td>
<td>RDS</td>
<td>0.80</td>
<td>Controlled</td>
<td>90</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>1000</td>
<td>30</td>
<td>0-8</td>
<td>Apnoea</td>
<td>0.45</td>
<td>Controlled</td>
<td>60</td>
<td>27</td>
</tr>
<tr>
<td>5</td>
<td>970</td>
<td>28</td>
<td>11</td>
<td>Aspiration R-L shunt</td>
<td>0.76</td>
<td>Controlled</td>
<td>55</td>
<td>55</td>
</tr>
<tr>
<td>Mean±1 SD</td>
<td>994±19</td>
<td>28±1·2</td>
<td>2·8†</td>
<td>RDS</td>
<td>0·76±0·2</td>
<td>Controlled</td>
<td>56·22</td>
<td>35±32</td>
</tr>
<tr>
<td>Group B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>2040</td>
<td>34</td>
<td>5</td>
<td>RDS</td>
<td>0·39</td>
<td>Controlled</td>
<td>57</td>
<td>22</td>
</tr>
<tr>
<td>7</td>
<td>2150</td>
<td>36</td>
<td>2</td>
<td>RDS</td>
<td>0·86</td>
<td>IMV</td>
<td>46</td>
<td>40</td>
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<tr>
<td>8</td>
<td>2700</td>
<td>40</td>
<td>0-9</td>
<td>Aspiration</td>
<td>0.84</td>
<td>Controlled</td>
<td>44</td>
<td>26</td>
</tr>
<tr>
<td>9</td>
<td>1900</td>
<td>33</td>
<td>22</td>
<td>Congenital heart disease</td>
<td>0·65</td>
<td>IMV</td>
<td>35</td>
<td>17</td>
</tr>
<tr>
<td>10</td>
<td>2290</td>
<td>34</td>
<td>3</td>
<td>RDS</td>
<td>0·53</td>
<td>IMV</td>
<td>24</td>
<td>13</td>
</tr>
<tr>
<td>Mean±1 SD</td>
<td>2216±306</td>
<td>35·4±2·9</td>
<td>36†</td>
<td>RDS</td>
<td>0·64±0·24</td>
<td>IMV</td>
<td>41·12</td>
<td>24±11</td>
</tr>
<tr>
<td>Total mean±SD</td>
<td>1605±675</td>
<td>31·7±4·4</td>
<td>1·8†(0-6-22)</td>
<td>RDS</td>
<td>0·70±0·22</td>
<td>IMV</td>
<td>47·5±19·7</td>
<td>28±23</td>
</tr>
</tbody>
</table>

*Mean values of 3 studies in one patient, †median.

RDS = respiratory distress syndrome, \( \text{F}_{102} \) = flow of inspired oxygen, IMV = intermittent mandatory ventilation.

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**Table 2**  Transcutaneous \( \text{Po}_2 \), heart rate, blood pressure, and dynamic respiratory compliance during tracheal toilet (mean±1 SD) in 10 infants

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Suctioning</th>
<th>Bag ventilation</th>
<th>Minutes after bag ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \text{TcPo}_2 ) (mmHg)</td>
<td>68±27</td>
<td>43±23*</td>
<td>56±27</td>
<td>64±34</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>144±8</td>
<td>123±24*</td>
<td>145±16</td>
<td>148±11</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>44±24</td>
<td>49±24*</td>
<td>46±24</td>
<td>46±24</td>
</tr>
<tr>
<td>Compliance (ml/cm water)</td>
<td>0·9±0·5</td>
<td>NA</td>
<td>NA</td>
<td>0·9±0·4</td>
</tr>
</tbody>
</table>

NA = not applicable. *Statistically significant differences (\( P<0·05 \)) compared with control values.

Conversion; traditional to SI units—7·5 mmHg ≈ 1 kPa, 98·07 cmH2O ≈ 1 Pa.
Fig. 1. During the control period 8 infants had a TcPo₂ $\geqslant 50$ mmHg and 2 infants a TcPo₂ $<50$ mmHg. During the suctioning period all 8 initially well-oxygenated infants showed a similar decrease in the TcPo₂ (27.8 ± 8.3 mmHg; mean ± SD) (range 17.0–43.6). Seven of these 8 infants became hypoxaemic (TcPo₂ $<50$ mmHg) at least once during tracheal toilet (3 infants once, 2 infants twice, and 2 infants three times). Thus in 8 infants, initially well oxygenated, hypoxaemia took place 13 times in 24 studies of tracheal toilet. Their TcPo₂ rise after suctioning and bag ventilation varied considerably. Some infants took 1 to 2 minutes after bag ventilation to reach control values. The other 2 infants, who had been hypoxaemic from the beginning, showed little change in TcPo₂ values.

The amount that TcPo₂ decreased was not related to body weight or gestational age and was similar for groups A and B (27.6 ± 22.4 mmHg). The decrease in TcPo₂ was greater in the 6 infants with controlled ventilation than in the 4 infants with intermittent mandatory ventilation (28.2 ± 14.4 mmHg). It was also greater in the 5 infants with F₁O₂ $\geqslant 0.8$ than in the 5 infants with F₁O₂ $<0.8$ (30.1 ± 19.9 mmHg). If the 2 newborn infants with initial hypoxaemia and cardiovascular disease were excluded the results would not be greatly different. The TcPo₂ decrease correlated well with the TcPo₂ levels during the control period ($r = 0.60$, P $<0.01$), but not with the duration of disconnection from the respirator ($r = 0.18$, NS). The TcPo₂ fall was independent of the disconnection time, irrespective of the method of ventilation, weight, gestational age, or F₁O₂ levels in these patients (Fig. 2). A fairly large TcPo₂ drop took

**Fig. 1** TcPo₂ changes in both groups of infants. Each symbol represents a mean value of 3 studies in one infant. TcPo₂ values below dotted line indicate hypoxaemia. The lowest TcPo₂ values are given for the suctioning period. The numbers on left are the amounts by which TcPo₂ decreased (in mmHg), those on right are case numbers.

Conversion: traditional units to SI—7.5 mmHg $\approx$ 1 kPa.

**Fig. 2** The period that infants were disconnected from the respirator and the decrease in TcPo₂ in the two groups of infants. Each symbol represents the result of one study in one patient. Case 10 had a large decrease in TcPo₂ despite the shortest suctioning time. (Heart rate and blood pressure changes are shown in Fig. 3).
place in one infant (Case 10) who was disconnected from the respirator for the shortest time. It is of note that the TcPo2 did not always reach its lowest value (TcPo2 min) during suctioning. The TcPo2 min was recorded 13 times during and 17 times after the suctioning period.

In each patient the heart rate and the blood pressure (Fig. 3) reacted uniformly to tracheal suction. The heart rate fell in 9 and the blood pressure rose in 8 of the 10 infants. Two infants showed a fall in blood pressure each time they were suctioned; one was initially hypoxaemic, the other (Case 10) developed severe bradycardia when suctioned. While the heart rate returned to control values, the blood pressure in 8 infants was still slightly raised 5 minutes after bag ventilation. The aortic pulse pressure remained unchanged during the entire study period in 6 infants in whom we had obtained an undamped blood pressure tracing. In one infant we observed cardiac arrhythmias.

Discussion

It is common for hypoxaemia to occur during tracheal suctioning and the TcPo2 can fall to hypoxic levels. Our study shows that tracheal suctioning considerably disturbs the oxygenation and causes hypoxaemia on 1 occasion out of 2 in nearly all infants even though they may initially have been well oxygenated. The hypoxaemia is not immediately reversed by bag ventilation with oxygen, but can last as long as 2 minutes in some patients (Fig. 1). We believe this procedure may hinder recovery from the initial disease.

As both groups of infants reacted in a similar manner with falls in TcPo2, we disagree that tracheal suction mainly affects small preterm infants. Surprisingly we found no correlation between the duration that they were disconnected from the respirator and the extent of TcPo2 fall. Although the time that it took to suction the infant was not measured, it is reasonable to assume that it was proportional to the period that the infant was disconnected from the respirator. Thus the lack of correlation cannot be ascribed to differences between these two variables. The severity of the disease seems to be the main determinant for the TcPo2 fall, as patients with high FIO2 and controlled ventilation were affected to a greater extent than the patients with intermittent mandatory ventilation and lower FIO2. However infants receiving intermittent mandatory ventilation also had large decreases in TcPo2 leading to hypoxaemia. In an infant on intermittent mandatory ventilation the oxygen in the alveoli decreases as it is absorbed during suctioning, and as such infants breathe room air the alveolar oxygen concentration is reduced even further. As the rate in the fall of alveolar oxygen is greatly influenced by the rate of TcPo2, these infants are probably equally likely to become hypoxaemic.

The cardiovascular changes noted during tracheal suction were typical of a hypoxaemic response, characterised by a falling heart rate and an increasing blood pressure. The heart rate decrease of 21.5 beats/min and the blood pressure increase of 4-4 mmHg are in good agreement with experimental data in fetal lambs. However we cannot exclude the possibility that the baby's activity, in response to this unpleasant respiratory experience, could bring about similar changes.

Even if a baby did not become hypoxaemic (TcPo2 <50 mmHg) a decrease in TcPo2 to levels
between 60 and 50 mmHg would cause the blood to become less saturated with oxygen. At the same time the heart rate would be lower, the pulse pressure the same, and consequently the cardiac output would be decreased. Thus the oxygen availability (cardiac output \( \times 1.34 \) haemoglobin \( \times \) saturation) for the body’s tissues would be decreased with tracheal suction.

Studies in animals, and in man, indicate that the chemoreceptors are activated when the Po₂ falls below 70 or 100 mmHg respectively. Such a Po₂ fall occurred in our patients; it probably increased the activity of the chemoreceptors and subsequently the pressure area in the brain stem and hence augmented sympathetic outflow. The latter causes vasocostriction. We therefore think that tracheal suction not only causes hypoxaemia in some infants and diminishes oxygen transport in others, but that it also triggers a hypoxaemic response in nearly all infants which is not immediately reversed by high oxygen levels. This conclusion is supported by our finding cardiovascular responses characteristic of hypoxaemia on nearly all, and yet hypoxaemia (TcPo₂ <50 mmHg) on only half of the occasions. Furthermore 8 of these 10 infants had higher blood pressures at 5 minutes after bag ventilation, despite high oxygen levels, than during the control period. The hypoxaemic vasoconstriction is known to affect mainly muscle, splanchnic, renal, and pulmonary vessels. Thus tracheal suctioning and its cardiovascular consequences may well account for many problems in the neonate—such as necrotising enterocolitis, patent ductus arteriosus, or intrapulmonary hypoperfusion.

The fact that hypoxaemic cardiovascular changes took place more often than hypoxaemic TcPo₂ levels, might be explained by a dampened transcutaneous Po₂ measurement. The Po₂ probably fell faster and to lower values than suggested by the TcPo₂ measurement. The TcPo₂ often did not reach its minimum until 2 minutes after bag ventilation. Furthermore, the TcPo₂ measurements in steplike changes of inspired oxygen showed a delay compared with direct Po₂ determinations. It can be argued that the TcPo₂ measurements show falsely low Po₂ values. During suctioning, heart rate (generally) and blood pressure (occasionally) fell. Then skin perfusion might have been decreased and less oxygen been diffused through the skin to the electrode than with normal perfusion. PO₂ measurements during tracheal toilet should be done using a sensor with an immediate response.

All the changes brought about by tracheal toilet call for the procedure to be re-evaluated. At first sight, the long time taken to do the suctioning seems at fault. We measured this time accurately. Other authors who reported a suctioning time of 15 seconds may have underestimated the time. But even if the time the infant is disconnected from the oxygen supply is kept to less than 30 seconds, hypoxaemia and circulatory changes will take place in some patients. We suggest reducing the frequency of tracheal toilet to a minimum, limiting the duration of suctioning, and using respirators which allow simultaneous ventilation and suction.

References


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