Do fluctuations of arterial partial pressure of CO₂ impact on venous-arterial CO₂ gradient?

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Abstract

Venous-arterial difference in CO$_2$ tension ($\Delta$CO$_2$) is described as a useful tool to evaluate the adequacy of tissue perfusion in shock. We hypothesized that arterial partial pressure of CO$_2$ variations could impact on this gap for a same patient. We included 10 patients admitted to the intensive care unit after an elective cardiac surgery. Tidal volume was 8 ml/kg throughout the experiment. The respiratory rate (RR) was set at 10, 13 or 16 breaths/min. We reported a significant increase in $\Delta$CO$_2$ between RR 10 and RR 16; (4.2±1.8 and 7.6±1.7, respectively).

We underline the effect of moderate hyperventilation on CO$_2$ gap.
Findings

The mixed venous-arterial difference in CO$_2$ tension ($\Delta$CO$_2$) has been proposed as an index of the adequacy of tissue perfusion in septic shock. Indeed, $\Delta$CO$_2$ increases with low cardiac output or unadapted microcirculatory perfusion (1, 2). However, because CO$_2$ by itself can influence vascular tone (3), we hypothesized that, for a same patient, arterial partial pressure of CO$_2$ (PaCO$_2$) variations can modify $\Delta$CO$_2$ values.

Methods: After approval by the local ethics committee, 10 patients (66±11 years, SAPS II =35.3±6), admitted to the intensive care unit after an elective cardiac surgery, were included. They were all monitored with a Swan-Ganz catheter. Tidal volume was 8 ml/kg throughout the experiment. The respiratory rate (RR) was set at 10, 13 or 16 breaths/min defining three times points. The RR adjustment order was randomized for each patient. After 30 minutes of stabilization at a given ventilatory condition, arterial and venous blood gases as well as cardiac index, and mean arterial pressure were measured. Venous samples were withdrawn from the central venous catheter. The three series of measurements for one patient were performed within 2 hours. A $\Delta$CO$_2$ $\leq$ 6 mmHg was considered as normal (1). Results are presented as means ± SD. Data were analyzed by repeated measures ANOVA and Scheffé’s post-hoc test or Chi$^2$ test and Bonferroni correction when suitable. A p value<0.05 was considered significant.

Results: PaCO$_2$ varied consistently with the changes in RR, and we reported a significant increase in $\Delta$CO$_2$ between RR 10 and RR 16; this was associated with a significant decrease in the percentage of patients with a normal $\Delta$CO$_2$ value (table1). Interestingly, ScvO$_2$ was also found significantly decreased when RR increased.
Discussion: In ventilated hemodynamically stable postoperative patients, we showed that PaCO$_2$ variations might modify ΔCO$_2$. Similarly, in healthy volunteers, hyperventilation is associated with an increase of the difference between arterial and venous peripheral CO$_2$ (4). A possible explanation is that hypocapnia induces microvascular constriction thus increasing stagnation flow and thus increases the gap. This hypothesis could be an explanation of the increment of gut mucosal-arterial PCO$_2$ gradient observed with acute moderate hypocapnia (5). In this situation the decreased in ScvO$_2$ could be interpreted as an increase of tissular oxygen extraction induced by a low O2 delivery with vasoconstriction (3).

Conclusion: Although CO$_2$ gap is a valuable index to evaluate perfusion in shock state, one must be warned of the effect of moderate hyperventilation on this gradient. The direct effect of CO$_2$ on microcirculation needs to be confirmed by further experiments.
List of abbreviations

ΔCO₂: Venous-arterial difference in CO₂ tension
RR: respiratory rate
PaCO₂: arterial partial pressure of CO2
ScvO₂: central venous saturation
Competing interests

The authors declare that they have no competing interest


Table 1 Blood gas values and hemodynamic data at the different respiratory rates.

<table>
<thead>
<tr>
<th></th>
<th>Respiratory rate 10</th>
<th>Respiratory rate 13</th>
<th>Respiratory rate 16</th>
</tr>
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<tbody>
<tr>
<td>PaCO2 (mmHg)</td>
<td>45.5±9.9</td>
<td>39.7±7.9 §</td>
<td>35.9±7.9 †‡</td>
</tr>
<tr>
<td>∆CO₂ (mmHg)</td>
<td>4.2±1.8</td>
<td>6.6±2.8</td>
<td>7.6±1.7 †</td>
</tr>
<tr>
<td>pH</td>
<td>7.29±0.06</td>
<td>7.32±0.06 §</td>
<td>7.35±0.07 †‡</td>
</tr>
<tr>
<td>Bicarbonate (mmol/L)</td>
<td>21.2±2.5</td>
<td>20.7±2.5</td>
<td>20±2.5</td>
</tr>
<tr>
<td>∆CO₂ ≤6mmHg n (%)</td>
<td>10 (100)</td>
<td>4 (40) §</td>
<td>2 (20) †</td>
</tr>
<tr>
<td>ScvO₂ (%)</td>
<td>77.9±4.1</td>
<td>74.7±7.4</td>
<td>72.6±7.1†</td>
</tr>
<tr>
<td>Cardiac index (L/m²)</td>
<td>2.37±0.5</td>
<td>2.36±0.6</td>
<td>2.36±0.6</td>
</tr>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>71.7±13.3</td>
<td>68±14.5</td>
<td>71.4±13.2</td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>36.9±0.9</td>
<td>36.9±0.9</td>
<td>36.8±0.9</td>
</tr>
</tbody>
</table>

§: p< 0.05 (RR 10 vs RR 13), †: p< 0.05 (RR 10 vs RR 16), ‡: p< 0.05 (RR 13 vs RR 16).

ScvO₂ : central venous oxygen saturation