Effect of carbon dioxide levels post extensive neonatal resuscitation on cerebral and myocardial hemodynamics and oxygenation

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Background

- In the post-resuscitation phase following asphyxia, fluctuations in carbon dioxide could influence cerebral and myocardial perfusion and oxygenation.
- In infants ≥36 weeks GA (n=204) undergoing whole body hypothermia for HIE, cumulative exposure to hypocarbia was associated with increased death/disability at 18-22 months of age.

Objectives

To study the effect of fluctuations of arterial carbon dioxide (PaCO2) level following extensive resuscitation of bradycardic term lambs on hemodynamics and oxygenation based on the following definitions:

- **Hypocapnia** < 35 mmHg
- **Normocapnia** 35 – 50 mmHg
- **Hypercapnia** > 50 mmHg

Methods

- Umbilical cord occluded till heart rate <60 bpm
- Resuscitation with ventilation/chest compressions with 100% O2
- Epinephrine according to Neonatal Resuscitation Program (NRP)
- Return of Spontaneous Circulation (ROSC) = HR>100 bpm & DBP>20mmHg
- O2 titrated according to NRP targets

Results

Carotid blood flow had significant correlation with arterial carbon dioxide

* R=0.64

\[ y = 0.34x + 7.7403 \]

*p <0.001 by correlation

 Coronary blood flow had significant correlation with arterial carbon dioxide

\[ R=0.59 \]

*p <0.001 by correlation

O2 delivery decreased to brain and heart with hypocapnia

Carotid blood flow lowest with hypocapnia

Coronary blood flow lowest with hypocapnia

\[ * p <0.001 by ANOVA \]

Results continued

O2 delivery to the brain

\[ * p <0.0001 \]

O2 delivery to the heart

\[ * p <0.0001 \]

\[ \text{(Kruskal-Wallis)} \]

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Hypocapnia (PaCO2 &lt;35 mmHg)</th>
<th>Normocapnia (PaCO2 35 – 50 mmHg)</th>
<th>Hypercapnia (PaCO2 &gt;50 mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen (%)</td>
<td>30±14</td>
<td>29±9</td>
<td>32±11</td>
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<tr>
<td>Diastolic BP (mmHg)</td>
<td>35±10</td>
<td>37±9</td>
<td>35±9</td>
</tr>
<tr>
<td>Mean BP (mmHg)</td>
<td>46±10</td>
<td>42±11</td>
<td>48±12</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>163±25</td>
<td>172±25</td>
<td>176±20</td>
</tr>
<tr>
<td>Pulmonary Blood Flow (ml/kg/min)</td>
<td>64.5 (IQR:33.6, 68.0)</td>
<td>56.4 (IQR:34.6, 75.8)</td>
<td>51.2 (IQR 40.2, 85.8)</td>
</tr>
<tr>
<td>Heart Blood Flow (ml/kg/min)</td>
<td>7.26±1.2</td>
<td>7.15±1.0</td>
<td>7.13±1.2</td>
</tr>
<tr>
<td>PaO2 (mmHg)</td>
<td>67±27.6</td>
<td>56±27.6</td>
<td>47±27.5</td>
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<tr>
<td>Base deficit (mmol)</td>
<td>-13±3*</td>
<td>-14±4</td>
<td>-7±6</td>
</tr>
</tbody>
</table>

*p<0.05 Data presented as mean ± SD or median & IQR. *A negative DBF value signifies hypokalia in patients who required inotropic support.

References:

- Pappas A et al, Hypocarbia and adverse outcome in neonatal hypoxic-ischemic encephalopathy.
- Wyatt JS et al. Response of cerebral blood volume to changes in arterial carbon dioxide tension in preterm and term infants

Conclusion

- Fluctuations in CO2 led to cerebral and myocardial perfusion changes in the immediate post resuscitation phase.
- Hypocapnia in the immediate post-resuscitation phase led to worsening of hypoxia and decreased cerebral and myocardial oxygen delivery to brain and heart.

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