Hypercarbia vs Hypoxia to Increase PVR in Single Ventricle Patients

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Preoperative Single Ventricle Physiology

• The distribution of blood flow to the systemic and pulmonary circulations is in parallel rather than series.
• Blood flow depends primarily on the relative resistances of the respective vascular beds.
• Entire cardiac output is managed by the single ventricle and must somehow divide itself between the systemic and pulmonary circuits.
• As pulmonary vascular resistance decreases after birth, many of these children will suffer from overcirculation into the pulmonary circuit.
  – In patients with ductal-dependent systemic circulation, such as hypoplastic left heart syndrome, this can result in a paucity of systemic circulation with concomitant acidosis.
Fig. 1. A: physiology of univentricular patient as pulmonary vascular resistance falls and systemic vascular resistance rises after birth

Management Goals

• The goal of preoperative circulatory management in patients with single ventricle hearts is balancing the blood flow to lungs (Qp) and body (Qs)

• Keeping pulmonary-to-systemic blood flow ratio (Qp/Qs) <1 is the goal in these patients

• Qp/Qs ratio can be manipulated by modifying pulmonary vascular resistance
  – An increase in pulmonary vascular resistance can be obtained by decreasing the concentration of inspired oxygen by adding supplemental nitrogen (hypoxia) via nasal cannula or adding supplemental inspired CO2 to the ventilator circuit (hypercarbia)
• Compares the impact of hypoxia (17% FiO2) vs hypercarbia (2.7% FiCO2) on oxygen delivery under conditions of fixed minute ventilation

• 10 anesthetized, paralyzed pts preop with HLHS

• Prospective, randomized, crossover trial

• Treated in random order (10 mins) then recovery period in RA (15-20 mins)

• Arterial (SaO2), SVC SvO2, Cerebral ScO2 (NIRS)

(Circulation. 2001;104[suppl I]:I-159-I-164.)
NIRS – Theory of Operation

- Near-infrared light sensor applied to area of interest
- Near-infrared light photons are injected into the tissue
- Allows for direct measurement of oxygen saturation in blood in capillary bed below sensor
- Subtraction of shallow signal from deep yields average oxygenation (rSO2) of tissue 2-3 cm deep
- Values reflect surplus of oxygen remaining after the tissues have taken what they require

The mean photon path in tissue is a "banana" shape.
NIRS Concept

- Non pulse-oximetry
- Reflects balance between O2 delivery and O2 consumption
- Correlates best with venous blood saturation
- As oxygen delivery to a region ↓ or consumption ↑, rSO2 declines from baseline
Cerebral Regional Monitoring

- Gives indication of cerebral blood flow/perfusion
- Prevention neuronal injury enhanced by ability to assess cerebral perfusion continuously
- Critically ill children are at risk for neuronal injury due to their disease process, complications or treatments
- Often difficult to assess neurologic function as indicator of adequate cerebral perfusion due to sedation
NIRS Values

- **Cerebral** (rSO2C) normal – acyanotic or cyanotic
  - 30% less than arterial sat
- Abnormal & associated with ischemic brain injury
  - A change of 20% from baseline
  - A rSO2 of less than 45%

- **Renal/Somatic** (rSO2S) normal – acyanotic or cyanotic
  - 10-15% less than arterial sat
- Abnormal & associated with ischemic tissue injury
  - A change of 20% from baseline
  - A rSO2 <60% indicates a significant change in tissue perfusion
TABLE 2. Hemodynamic Data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Hypoxia (Baseline)</th>
<th>Hypoxia (Hypoxia)</th>
<th>P</th>
<th>Hypercarbia (Baseline)</th>
<th>Hypercarbia (Hypercarbia)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen saturation, %</td>
<td>93.2±1.5</td>
<td>89.8±2.8</td>
<td>0.004*</td>
<td>92±1.3</td>
<td>90.2±1.8</td>
<td>0.04</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>160±6</td>
<td>153±4</td>
<td>0.30</td>
<td>150±5</td>
<td>149±4</td>
<td>0.56</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>61.7±3.6</td>
<td>61.2±3.4</td>
<td>0.79</td>
<td>60.6±2.6</td>
<td>68.5±4.6</td>
<td>0.02</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>35±2.1</td>
<td>33.7±1.9</td>
<td>0.57*</td>
<td>35±1.7</td>
<td>40±2.5</td>
<td>0.002</td>
</tr>
</tbody>
</table>

BP indicates blood pressure. Values are mean±SEM. Oxygen saturation is transcutaneous measurement. Difference between condition and baseline was determined by paired t test.

*Data not normally distributed, with P value determined by Wilcoxon signed rank test.

Hemodynamic Data

Table 2 shows the transcutaneous oxygen saturation, heart rate, and systolic and diastolic arterial blood pressures. There was a significant decrease in transcutaneous oxygen saturation for both hypoxia and hypercarbia compared with baseline. There was a significant increase in both systolic and diastolic blood pressure with hypercarbia but not with hypoxia. There was no significant change in heart rate with either hypoxia or hypercarbia.
TABLE 3. Arterial Blood Gas Data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>Hypoxia</th>
<th>P</th>
<th>Baseline</th>
<th>Hypercarbia</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.44±0.02</td>
<td>7.46±0.02</td>
<td>0.012</td>
<td>7.43±0.02</td>
<td>7.33±0.02</td>
<td>0.002*</td>
</tr>
<tr>
<td>Po₂, mm Hg</td>
<td>50.3±1.9</td>
<td>42.2±1.8</td>
<td>&lt;0.0001</td>
<td>49.6±1.6</td>
<td>50.8±1.9</td>
<td>0.64</td>
</tr>
<tr>
<td>PCO₂, mm Hg</td>
<td>37.5±1.8</td>
<td>35.7±2.0</td>
<td>0.055</td>
<td>39.4±1.6</td>
<td>53.7±1.6</td>
<td>0.002*</td>
</tr>
</tbody>
</table>

Values are mean±SEM. Difference between condition and baseline was determined by paired t test. *Data not normally distributed, with P value determined by Wilcoxon signed rank test.

Arterial Blood Gas Data
Table 3 shows the arterial pH, oxygen tension (Po₂), and carbon dioxide tension (PCO₂) for hypoxia and hypercarbia compared with baseline. Hypoxia resulted in a significant decrease in Po₂, a small but significant increase in pH, and an insignificant decrease in PCO₂. Hypercarbia did not affect Po₂ but significantly decreased pH and increased PCO₂.
TABLE 4. Co-Oximetry Data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Hypoxia</th>
<th></th>
<th>Hypercarbia</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Hypoxia</td>
<td>P</td>
<td>Baseline</td>
<td>Hypercarbia</td>
</tr>
<tr>
<td>Sao₂, %</td>
<td>90.7±1.9</td>
<td>85.6±2.1</td>
<td>0.001</td>
<td>90.0±1.2</td>
<td>87.4±1.7</td>
</tr>
<tr>
<td>Svo₂, %</td>
<td>62.4±3.3</td>
<td>56.7±3.8</td>
<td>0.009</td>
<td>61.5±3.5</td>
<td>67.5±3.6</td>
</tr>
<tr>
<td>AVO₂ difference, %</td>
<td>28.3±3.2</td>
<td>28.8±3.8</td>
<td>0.77</td>
<td>28.5±3.6</td>
<td>19.9±3.8</td>
</tr>
</tbody>
</table>

Values are mean±SEM. P values were determined by paired t test.

Hypoxia significantly decreased both Sao₂ (−5.2±1.1%, P=0.0014) and Svo₂ (−5.6±1.7%, P=0.009), but AVO₂ difference (0.44±1.4, P=0.76) and Sco₂ (−0.4±1.5%, P=0.8) remained unchanged. Hypercarbia decreased arterial saturation Sao₂ (−2.6±0.6%, P=0.002) but increased both Sco₂ (9.6±1.8%, P=0.0001) and Svo₂ (6±2.2%, P=0.022) and narrowed the AVO₂ difference (−8.5±2.3%, P=0.005).
Figure 1. Absolute difference between condition and baseline (mean±SEM) for arterial co-oximetry (SaO₂), superior vena caval co-oximetry (SvO₂), arteriovenous saturation difference (AVO₂ difference), and cerebral oxygen saturation (ScO₂). P values were determined by paired t test comparing condition and baseline.
Figure 2. Difference in Qp:Qs between condition and baseline (mean±SEM). Qp:Qs was calculated as \((\text{Sa}_0 - \text{Sv}_0) / (\text{Spvo}_2 - \text{Sa}_0)\), where \(\text{Sa}_0\) and \(\text{Sv}_0\) were directly measured, and \(\text{Spvo}_2\) was assumed as 99% at baseline, 98.2% for hypoxia, and 98.5% for hypercarbia. \(P\) values represent difference between condition and baseline as determined by paired t test.
Figure 3. Difference in oxygen delivery between condition and baseline (mean±SEM). Oxygen delivery was calculated as $\text{SaO}_2 \div (\text{SaO}_2 - \text{SvO}_2)$, where $\text{SaO}_2$ and $\text{SvO}_2$ were directly measured. $P$ values were determined by paired $t$ test comparing condition and baseline.
Take Home Points

- Under hypercarbia, BP sig increased with no sig change in HR
- Using SvO2 as a marker, hypercarbia sig increased DO2 and hypoxia sig decreased DO2
- AVO2 diff unchanged with hypoxia and decreased with hypercarbia (ie increased O2 delivery compared to O2 consumption ratio)
- Hypoxia had no affect on ScO2, hypercarbia sig increased ScO2
- Hypoxia and hypercarbia both lowered Qp:Qs, but only hypercarbia increased CO
- Limitation is inability to measure “true” SvO2 in HLHS, and thus unable to differentiate cerebral vs systemic O2 delivery

Conclusions—In preoperative infants with HLHS, under conditions of anesthesia and paralysis, although Qp:Qs falls in both conditions, oxygen delivery is unchanged during hypoxia and increased during hypercarbia. These data cannot differentiate cerebral from systemic oxygen delivery. (Circulation. 2001;104[suppl I]:I-159-I-164.)