Changes in cerebral oxygenation based on intraoperative ventilation strategy
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Background
- A key component of intraoperative care is the assurance of adequate cardiac output and oxygen delivery to the tissues.
- Tissue oxygenation can be monitored using cerebral oximetry (rSO2) with near infrared spectroscopy (NIRS).
- Monitoring and maintaining rSO2 may improve perioperative neurological outcomes.
- Changes in rSO2 have been shown to precede those in pulse oximetry, providing an earlier detection of deterioration.
- Various factors including changes in ventilation may affect rSO2 and therefore, understanding this effect may help guide clinical care.
- The current study evaluates changes in rSO2 during intraoperative changes in mechanical ventilation.

Methods
- Following IRB approval, patients undergoing a surgical procedure requiring placement of an arterial cannula were enrolled.
- Tissue and cerebral oxygenation were monitored using NIRS.
- Prior to anesthetic induction, the NIRS monitor was placed on the forehead and over the deltoid to obtain baseline values.
- NIRS measures were repeated each minute over a 5-minute period during general anesthesia at four phases of ventilation:
  1. normocarbia and low FiO2
  2. hypocarbia and low FiO2
  3. hypocarbia and high FiO2
  4. normocarbia and high FiO2.
- Normocarbia was defined as ETCO2 = 35-40 mmHg (correlated with an arterial sample), hypocarbia as ETCO2 = 25-30 mmHg, low FiO2 = 30%, and high FiO2 = 60%.
- Average values of NIRS measurements obtained during each phase were used to compare sequential phases using paired t-tests.

Table 1: Changes in NIRS values during the changes in ventilation and oxygenation strategies

<table>
<thead>
<tr>
<th>Phase</th>
<th>Mean ± SD</th>
<th>Change from previous time point</th>
<th>Mean ± SD</th>
<th>95% CI</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>Cerebral</td>
<td>80 ± 9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tissue</td>
<td>87 ± 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase 1</td>
<td>Cerebral</td>
<td>82 ± 8</td>
<td>2 ± 7</td>
<td>(-1, 5)</td>
<td>0.284</td>
</tr>
<tr>
<td></td>
<td>Tissue</td>
<td>87 ± 8</td>
<td>-1 ± 7</td>
<td>(-4, 2)</td>
<td>0.472</td>
</tr>
<tr>
<td>Phase 2</td>
<td>Cerebral</td>
<td>79 ± 8</td>
<td>-3 ± 4</td>
<td>(-5, -2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Tissue</td>
<td>88 ± 8</td>
<td>2 ± 2</td>
<td>(1, 3)</td>
<td>0.002</td>
</tr>
<tr>
<td>Phase 3</td>
<td>Cerebral</td>
<td>80 ± 9</td>
<td>1 ± 3</td>
<td>(0, 2)</td>
<td>0.029</td>
</tr>
<tr>
<td></td>
<td>Tissue</td>
<td>88 ± 7</td>
<td>0 ± 2</td>
<td>(-1, 1)</td>
<td>0.923</td>
</tr>
<tr>
<td>Phase 4</td>
<td>Cerebral</td>
<td>82 ± 8</td>
<td>2 ± 3</td>
<td>(1, 4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Tissue</td>
<td>87 ± 8</td>
<td>-2 ± 3</td>
<td>(-3, 0)</td>
<td>0.017</td>
</tr>
</tbody>
</table>

* P-value comparing sequential time points by paired t-test

Results
- The study cohort included 25 adolescents (11 males and 14 females, age 15 ± 3 years, and weight 52 ± 14 kg).
- Baseline cerebral and tissue oxygenation were 80 ± 9 and 87 ± 5, respectively.
- During phase 1, cerebral rSO2 was 82 ± 8 and decreased to 79 ± 8 in phase 2 (hypocarbia and low FiO2).
- Cerebral oxygenation recovered during phase 3 (80 ± 9) and phase 4 (82 ± 8).
- Each sequential change (e.g., phase 1 to phase 2) in cerebral oxygenation was statistically significant (p<0.05).
- Tissue oxygenation remained at 87-88 throughout the 4 study phases.
- During the study sequences, heart rate, blood pressure, and the depth of anesthesia remained unchanged.

Conclusion
- Cerebral oxygenation declined during GA with the transition from normocarbia to hypocarbic conditions, although this decrease was likely of no clinical significance given the high starting rSO2 value.
- The decrease was easily reversed by the administration of supplemental oxygen (60% versus 30%).
- Changes in tissue oxygenation over this time were very minor, with mean rSO2 varying by ≤1 between study phases.
- These data suggest that the decrease in rSO2 that occurs with hyperventilation may be offset by increasing the FiO2.

References