Near-Infrared Spectroscopy (NIRS): Principles and Clinical Applications

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What is Near Infrared Spectroscopy (NIRS)?

- NIRS can be used as a non-invasive monitoring technique for cerebral and somatic oxygenation and hemodynamics.
- Data is acquired from vascular beds (cerebral, renal, and splanchnic) with varied flows and extraction ratios.
- While pulse oximetry provides a measure of arterial oxygen saturation reflecting oxygen supply to the tissues, NIRS-measured regional oximetry measures the balance between local oxygen delivery and consumption beneath the sensor.
- It provides a non-invasive measure of end-organ oxygenation and perfusion.
How a NIRS sensor works

Placement of NIRS sensor on Forehead. The two black circles are the light source and detector.

Light passes from light source through the scalp, skull, and brain tissue then to the detector.

Cerebral saturation (rSO2) reflects a ratio of arterial to venous blood of 25%:75%
Regional saturation reflects oxygen balance

- rSO2 increases with more oxygen delivery or less demand while rSO2 decreases when delivery falls or rise in demand

- Oxygen delivery is influenced by:
  - Hemoglobin concentration
  - Hemoglobin saturation
  - Cardiac output (HR, preload, contractility and afterload)

↑ Oxygen demand
  - fever, shivering, cold stress, infection, seizures, pain

↓ Oxygen demand
  - hypothermia, sedation/paralysis, decreased extraction
Target rScO2 ranges for newborns

Dix et al., Pediatr Res (2014);
Alderliesten et al., Pediatr Res 2015
What can you do if the rScO2 is abnormal?

If cerebral saturation is too low:

• Hypocarbia (decrease ventilation)
• Hypotension (treat with fluid or inotropes)
• Anemia (give packed red blood cell transfusion)
• Low arterial saturation (increase FiO2)

If cerebral saturation is too high:

• Supranormal arterial saturation (wean FiO2)
• Hypercarbia (increase ventilation)
Who may benefit from NIRS monitoring

- Preterm infants < 29 weeks gestation
- Infants with suspected hemodynamically significant PDA
- Hypoxic ischemic encephalopathy
- Grade III/IV intraventricular hemorrhage
- Complex Congenital heart disease
- Congenital diaphragmatic hernia
- Critically ill infants with hemodynamic instability (pre-ECMO or ECMO)
Hypocarbia during mechanical ventilation

26 4/7 weeks gestation, 925 g, chorioamnionitis, day 1 of life

van Bel F, Brain monitoring conference (2015)
HFOV can affect cerebral saturation

26 week gestation, 780 grams, on HFOV for RDS
ScO2 increases and FTOE decreases following transfusion in preterm infants.

There is a poor correlation of pre-transfusion hematocrit with rScO2. This suggests that hematocrit alone is a poor predictor of cerebral saturation.

rScO2, perfusion and symptoms of anemia improve in infants with rScO2 <55% but not in infants with rScO2 ≥55%

rScO2 or FTOE may be better indicators of need for transfusion

Sood B et al., J Near InfraRed Spectrosc (2014)
Hypotension in preterm infants

Objective: To compare neurodevelopmental (ND) outcome, mean arterial BP, and rScO2 between neonates treated for low mean arterial pressure (MAP) and controls

Results: Infants treated for low MAP spent more time with MAP < gestational age than controls (9 versus 0%, p<0.001) but there were no differences in ND outcome or rScO2

rSO2 <50% for >10% time was associated with lower ND outcome

Conclusion: This suggests that rScO2 is a surrogate marker for cerebral blood flow and could be used in hypotension treatment protocols.

Alderliesten T et al., J Pediatr (2014)
NIRS and the PDA

A hemodynamically significant PDA (hsDPA) is associated with increased pulmonary blood flow and decreased systemic blood flow due to ductal steal.

This is associated with lower blood pressure as well as decreased brain and other organ perfusion.

NIRS has been used to study cerebral and somatic effects of PDA as well as response to medical and surgical treatment.
**rScO2 and hemodynamically significant PDA**

<table>
<thead>
<tr>
<th>Variable</th>
<th>hsPDA (n= 20)</th>
<th>Controls (n=20)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean blood pressure (mm Hg)</td>
<td>33</td>
<td>38</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Mean rScO2 (%)</td>
<td>62± 9</td>
<td>72± 10</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>FTOE</td>
<td>0.34± 0.1</td>
<td>0.25± 0.1</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

**Conclusions:** In infants with PDA, mean blood pressure and cerebral saturation were lower and FTOE higher compared with control infants without a PDA.

Renal saturation and hemodynamically significant PDA

<table>
<thead>
<tr>
<th>Variable</th>
<th>hsPDA (n=21)</th>
<th>No PDA (n=14)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal saturation (%)</td>
<td>61± 3</td>
<td>70± 3</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Low renal saturation is associated with hs PDA.

Renal saturation <66% was associated with hsPDA:
Sensitivity of 81%
Specificity of 77%
AUC = 0.786, p<0.0001.
Is the PDA significant in this infant?
What about the PDA in this infant?