CEREBRAL OXIMETRY IN INFANTS WITH HIE

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OBJECTIVES

1. Understand how cerebral oximetry works
2. Understand how cerebral oximetry may guide intervention
3. Learn that cerebral oximetry trends may have prognostic value
WHAT IS CEREBRAL OXIMETRY?

• Cerebral oximetry is a non-invasive tool based on near-infrared spectroscopy (NIRS) that can monitor the regional hemoglobin oxygen saturation (rScO2) of the frontal cortex.

• It provides continuous information about brain oxygenation and it provides a measurement of the brain as a sentinel organ indexing overall organ perfusion and injury.
Cerebral & Somatic rSO$_2$: A Real-time Guide to Perfusion and Interventions

- Left and right peri-renal NIRS
- Left and right cerebral NIRS
HOW DOES IT WORK?

• An emitter sends light of the near-infrared spectrum (wavelength of 700-1100nm) through cerebral tissue in a semi-curved shape to a detector, approximately 2–3 cm in depth.

• Differences in NIR light absorption are detected by the sensor and the ratio between $O_2$Hb and HHb is expressed as the $r$Sc$O_2$ or tissue oxygenation index (TOI), depending on the manufacturer of the NIRS device.
CEREBRAL OXIMETRY VALUES

• Felt to be consistent with a mixed venous measurement.
• Good correlation with jugular venous oxygen saturation.
• \( r\text{SCO}_2 \)/ systemic arterial oxygen saturation = cerebral oxygen utilization
• \( r\text{ScO}_2 \) is between approximately 40 and 56% directly after birth
  • increases up to 78% in the first 2 days after birth
  • stabilizes during 3–6 weeks after birth with values between 55 and 85%
• Trend is more useful than single absolute value
MODIFIERS OF CEREBRAL OXYGENATION

• Ventilation impacts cerebral circulation
  • High mean airway pressures can reduce oxygen saturation

• pCO2: hypercapnia induces cerebral vasodilation and hypocapnia induces vasoconstriction
  • Increased pCO2 increases oxygen saturation and decreases oxygen extraction

• SGA infants: much higher rScO$_2$ in first few postnatal days
  • Likely a function of intra-uterine preservation of brain blood flow
MODIFIERS OF CEREBRAL OXYGENATION

• Hypotension: true hypotension will affect rScO$_2$
  • Consider permissive hypotension unless cerebral saturation affected

• Significant PDA: shunting away from the brain can have a profound effect on rScO2
  • rScO2 rarely used as a marker of PDA significance

• Blood transfusions: anemic infants who undergo transfusion have resultant increase in rScO2

• Dysfunction cerebral autoregulation: RDS, surgery, high concentrations of pressors
  • Impaired autoregulation linked to poor ND outcomes.
CEREBRAL OXIMETRY IN HIE

• Infants with HIE have increased rScO2 and decreased cFOE during days following asphyxia.
  • Cerebral hyperoxygenation likely a result of decreased metabolism leading to low oxygen utilization, impaired cerebral autoregulation despite hyperperfusion after injury.
  • Higher rScO2 have correlated with adverse outcomes at 2 years (both with and without TH)
CAN CEREBRAL OXIMETRY BE OF PROGNOSTIC VALUE?

• Retrospective review, N=38 neonates with HIE with rScO2 data during cooling between 2013-2016 (total N of babies cooled during that time: 62).

• data: continuous vEEG, CO values throughout cooling, post rewarming MRI

• Hypotheses:
  • Persistently abnormal vEEG tracings would correlate with severe injury.
  • Higher rScO2 during cooling will correlate with greater severity of the hypoxic-ischemic injury as seen on brain MRI.
  • Cerebral oximetry values and vEEG results can be combined to construct an injury prediction model.
VEEG PATTERNS

• Continuous:
  normal continuity, or amount of uninterrupted activity, for age with only discontinuous periods during quiet sleep.

• Discontinuous:
  at least one hour of burst activity (with some normal features) separated by low voltage intervals with no discernible activity and is not explained by tracé alternant (quiet sleep)

• Maximal suppression:
  No discernible background activity for at least one hour
  OR seizure activity on a maximally depressed background
EEG Background Pattern

No/Mild Injury

Moderate Injury

Severe Injury

% of patients

Hours of Life

Maximal suppression
Discontinuous voltage
Continuous voltage

*p<0.05
Electrographic Seizure Activity

% of Patients

* p<0.05

Hours of Life

0-12 12-24 24-36 36-48 48-60 60-72 72-96

Severe Injury
Moderate Injury
No/Mild Injury
Cerebral Oxygenation Saturations

Mean Cerebral Saturation

Hours of Life

* p<0.05

Severe Injury
Moderate Injury
No/Mild Injury

* indicates significant difference.
### No SWC

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### Seizures

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RECEIVER OPERATOR CURVES

Black line = model A (cerebral oximetry + seizures + SWC + vEEG background pattern)
Grey line = model B (cerebral oximetry only)
What is a prism, Dad?

A place for light waves that commit minor refractions.