The Role of Cerebral and Splanchnic Oxygen Saturation in Predicting NEC in Children with HLHS

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I have no disclosures
Overview

• NEC literature review in congenital heart surgery

• The concept of using Cerebral Oximetry to detect NEC

• Study Design

• Results

• Conclusions
Necrotizing Enterocolitis

• Tissue in small or large intestine is injured
  • Intestine becomes inflamed
  • Bacteria and waste pass through intestines into abdominal cavity or bloodstream

• Patients at increased risk:
  • Prematurity 0.3-0.6% (McElhinney et al., 2000; Palmer et al., 1989; Stoll et al., 1980)
  • FT infants with congenital heart defects 3.3-6.8% (Leung et al., 1988; McElhinney et al., 2000)
  • Hypoxemia and hypotension
  • IUGR
Necrotizing enterocolitis in neonates with congenital heart disease

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• NEC and CHD significant causes of morbidity and mortality

• Circulatory disturbances (LVOTO, Single V physiology) along with stressors (surgery and CPB), elevations of proinflammatory cytokines all play a role in pathogenesis of NEC

• Cyanotic (71%) vs Acyanotic (39%)
HLHS and NEC

- HLHS higher incidence of NEC due to alterations in systemic blood flow: 7.6% vs. 2.1% in other CHD (McElhinney et al., 2000).

- Superior mesenteric artery (SMA) perfusion impaired in neonates with HLHS (Cheung et al., 2003)

- The underlying condition predisposing term infants to the development of NEC may be altered mesenteric blood flow rather than gut immaturity (Gellen et al., 2003)
Splanchnic Near-Infrared Spectroscopy and Risk of Necrotizing Enterocolitis After Neonatal Heart Surgery

Aaron G. DeWitt · John R. Charpie · Janet E. Donohue · Sunkyung Yu · Gabe E. Owens

- Prospective, n=64, neonates, single and biventricular repair
- 0% developed NEC in biventricular repair group
- 32% developed NEC in single ventricular repair group
- Single V had significantly lower splanchnic SrO2 before and during feeds
- Compared with suspected NEC, proven NEC had lower average splanchnic SrO2
- Splanchnic NIRS may help predict NEC
Fore-Sight ® Absolute Tissue Oximetry

- Non-invasive, continuous monitoring of regional oximetry
- Detects rSO₂ utilizing 5 wavelengths for accuracy
- Utilized to assess splanchnic ischemia (Matcher SJ et al., 1994)
Initial palliative repair for HLHS

• Low cardiac output state may lead to:
  • Global impact that may lead to rise in lactate and BNP:
    • Could be monitored by the rise in lactate and B-type natriuretic peptide (BNP) levels in blood.
  • Regional impact by decreased mesenteric blood flow and ultimately NEC:
    • Could be monitored by near infrared spectroscopy.
• Rise in lactate and BNP levels in blood could be an early indication of low cardiac output state, which may be associated with NEC

• Monitoring changes in the ratio between Cerebral Regional oxygen saturation to Splanchnic Regional Oxygen Saturation (CSOR) could be a valuable tool to predict these changes
Objective

• Our primary objective in this study was to evaluate the validity of changes in regional saturation (cerebral and splanchnic) as a predictor of changes in the tissue perfusion, as evident by changes in the levels of lactate and BNP.

• Our secondary objective was to determine if these correlations can ultimately predict GI complications.
Methods

• IRB approval and waiver of consent were obtained

• Patients with HLHS that were scheduled for Hybrid Stage I procedure or Balloon Atrial Septostomy were included.

• Regional oximeters were applied as follow:
  • Cerebral saturation (right forehead)
  • Abdominal regional saturation (epigastric quadrant)
Methods

Data collected included:

1. All Cerebral saturation data that was stored on the device for each patients. (Data were collected while patients remained in the CTICU)
2. Arterial blood gas results
3. BNP and Lactate levels that were drawn as standard of care
4. Clinical evidence of feeding intolerance or other clinical signs of GI complications
## Results

<table>
<thead>
<tr>
<th></th>
<th>Cardiac lesion</th>
<th>Associated anomaly</th>
<th>Feed and NEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient A</td>
<td>HLHS (MA/AA)</td>
<td>None</td>
<td>Tolerated feeds</td>
</tr>
<tr>
<td>Patient D</td>
<td>HLHS (MS/AA)</td>
<td>Atrial flutter</td>
<td>Tolerated feeds</td>
</tr>
<tr>
<td>Patient M</td>
<td>HLHS (MA/AA)</td>
<td>None</td>
<td>Tolerated feeds</td>
</tr>
<tr>
<td>Patient S</td>
<td>HLHS (MS/AA)</td>
<td>None</td>
<td>Tolerated feeds. Developed bloody stool at home and NPO for 5 days, but no NEC</td>
</tr>
<tr>
<td>Patient C</td>
<td>HLHS (MA/AA)</td>
<td>Agenesis of corpus callosum, Jacobsen syndrome, single kidney</td>
<td>Tolerated feeds</td>
</tr>
</tbody>
</table>
BNP vs. Cerebral saturation in patient D

(Mean difference: for BNP 2480 vs BNP 753 = -1.8, p value <0.0001)
BNP vs. Splanchnic saturation in patient D

(Mean difference for BNP 2480 vs BNP 753 = -0.77, P < 0.0001)
BNP vs. CSOR in patient D

Mean difference: CSOR for BNP 2480 vs CSOR for 753 = -0.02, p < 0.0001
### Results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Mean Cerebral Saturation at baseline BNP</th>
<th>Mean Cerebral Saturation at highest BNP</th>
<th>Changes in mean Cerebral Saturation</th>
<th>Mean Splanchnic Saturation at baseline BNP</th>
<th>Mean Splanchnic Saturation at highest BNP</th>
<th>Changes in mean Splanchnic Saturation</th>
<th>Mean CSOR at baseline BNP</th>
<th>Mean CSOR at highest BNP</th>
<th>Changes in mean CSOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient A</td>
<td>53%</td>
<td>61%</td>
<td>+ 8%</td>
<td>79%</td>
<td>82%</td>
<td>+ 3%</td>
<td>68%</td>
<td>74%</td>
<td>+ 6%</td>
</tr>
<tr>
<td>Patient D</td>
<td>60.1%</td>
<td>59.8%</td>
<td>-0.3%</td>
<td>66.4%</td>
<td>65.6%</td>
<td>- 0.8%</td>
<td>93%</td>
<td>91%</td>
<td>- 2%</td>
</tr>
<tr>
<td>Patient M</td>
<td>54%</td>
<td>58.8%</td>
<td>+ 4.8%</td>
<td>70%</td>
<td>73%</td>
<td>+ 3%</td>
<td>77%</td>
<td>81%</td>
<td>+ 4%</td>
</tr>
<tr>
<td>Patient S</td>
<td>59.8%</td>
<td>58.7%</td>
<td>- 0.9%</td>
<td>80%</td>
<td>75%</td>
<td>- 5%</td>
<td>74%</td>
<td>78%</td>
<td>+ 4%</td>
</tr>
<tr>
<td>Patient C</td>
<td>64%</td>
<td>66.3%</td>
<td>+ 2.3%</td>
<td>69%</td>
<td>67%</td>
<td>- 2%</td>
<td>91%</td>
<td>93%</td>
<td>+ 2%</td>
</tr>
</tbody>
</table>
Conclusion

• This pilot study demonstrates lack of correlation between changes in Cerebral saturation, splanchnic saturation and CSOR with changes in lactates or BNP.

• There is a higher correlation between the rise in BNP and lactate with CSOR. Four of our patients had a positive elevation of CSOR with the rise of BNP and lactate.

• It is no surprise that each comparison between plots was statistically significant since the sample size for each plot was large (7000+)
Conclusion

• While each comparison between plots showed statistical significance, the directions were not the same, hence we have concluded there were no correlations.

• This lack of correlation could be due to the fact that these changes represent global decrease in the cardiac output rather than actual decrease in the mesenteric blood flow, since no patient in our study developed NEC.
Limitations

- Sample size:
  - Original data were collected for 13 patients.
  - Technical issues lead to loss of data of 8 patients.
- We are currently recruiting more patients in the study.
Thank you

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Kristin Kirchner, RD
Yongjie Miao

AmSECT