Assessment for Spinal Cord Injury in Neonates Undergoing Surgical Repair of Coarctation of the Aorta

N Deutsch1, P Sinha2, RA Jonas2, G Vezina3, T Chang4, RJ Levy1
1Divisions of Anesthesiology and Pain Medicine, 2Cardiovascular Surgery, 3Radiology, 4Neurology
Children’s National Medical Center, The George Washington University School of Medicine and Health Sciences

Introduction:
Paraplegia is a rare but devastating complication that can result from aortic cross clamping (ACC) during repair of coarctation of the aorta. Infants are at high risk for spinal cord injury due to lack of collateral arteries. However, no active intraoperative spinal cord protective measures are routinely employed. Although prolonged ACC can result in overt spinal cord injury, it is unknown if mild ischemia leads to sub-clinical injury (1, 2). In this work, we aimed to identify evidence of sub-clinical spinal cord injury during surgical repair of coarctation of the aorta.

Methods:
We evaluated 5 neonates with critical coarctation presenting for surgical repair. Neurologic exam (3, 4) and MRI imaging of the brain and spinal cord were performed preoperatively and again 24-48 hours postoperatively. General endotracheal anesthesia was maintained with fentanyl, pancuronium, and oxygen (100%). All patients were allowed to passively cool to 35°C (rectal). Somatosensory evoked potentials (SSEPs) and lumbar spinal tissue oxygenation (TOI) using near-infrared spectroscopy (NIRS) were recorded. ABGs and lactate levels were measured prior to ACC (baseline) and 15 minutes post release of the clamp.

Results:
Mean ACC time was 36 minutes (±27 min). Post ACC lactate levels increased significantly from baseline. SSEP amplitude significantly decreased following ACC, with slow return toward baseline after removal of the clamp. Spinal TOI did not change with ACC, however significantly increased upon reperfusion. Although all preoperative neurologic exams were unremarkable, two patients demonstrated new-onset clonus in both lower extremities postoperatively. Postoperative spinal MRI, however, demonstrated no interval change from baseline study in all patients.

Conclusions:
Taken together, decreased SSEP amplitude during ACC, a rise in post-ACC lactate, along with new onset clonus suggest sub-clinical to mild spinal cord injury following surgical repair of coarctation of the aorta in neonates. Further work will investigate the importance, natural history, and incidence of such injury. In addition, we will explore the need to develop therapeutic interventions targeting intraoperative spinal cord protection in infants undergoing surgical repair of coarctation of the aorta.

Table 1: Neurologic Studies Pre- and Post-Repair

<table>
<thead>
<tr>
<th>PT #</th>
<th>MRI-pre</th>
<th>MRI-post</th>
<th>Neuro exam-pre</th>
<th>Neuro exam-post</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal study spinal cord and brain</td>
<td>unchanged</td>
<td>normal</td>
<td>mild clonus</td>
</tr>
<tr>
<td>2</td>
<td>Normal study spinal cord and brain</td>
<td>unchanged</td>
<td>normal</td>
<td>mild clonus</td>
</tr>
<tr>
<td>3</td>
<td>Normal study spinal cord and brain</td>
<td>unchanged</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>4</td>
<td>Dorsal epidural fluid collection, normal study brain</td>
<td>unchanged</td>
<td>normal</td>
<td>orthostasia</td>
</tr>
<tr>
<td>5</td>
<td>Normal spinal cord, immature appearing brain</td>
<td>unchanged</td>
<td>normal</td>
<td>normal</td>
</tr>
</tbody>
</table>

Fig. 1. Spinal TOI, SSEP Latency, and Amplitude over time
Fig. 2. MRI Spine
Fig. 3. Lowest SSEP Amplitude
Fig. 4. Maximum SSEP Latency
Fig. 5. Spinal TOI
Fig. 6. Lactate

References:
Armit-Tison C. Pediatric Neurol 2002; 27:196-212.