Commentary: Window of brain protection after congenital heart surgery—Is it still open?

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Central Message

Impaired cerebral autoregulation and low cerebral oxygenation do not account for white matter injury after neonatal heart surgery.

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Near-infrared spectroscopy has been adopted by many congenital heart programs during the last 2 decades. This modality allows one to monitor regional oxygenation at the bedside. The frequent clinical observation of low regional cerebral oxygenation (rSO₂) in the postoperative period leaves the bedside clinician to ponder about the long-term implication of this finding. Patients with congenital heart disease, particularly those with single-ventricle physiology, will often have rSO₂ of less than 50%. The consequences of this finding on a child's developmental trajectory remain poorly characterized. In their article in this issue of the *Journal*, Claessens and colleagues¹ attempt to pair the rSO₂ data with simultaneous blood pressure values to provide insights not only into regional cerebral perfusion but also into the potential role of cerebral autoregulation. The investigators seek to determine whether indices of cerebral oxygenation and autoregulation might be related to the development of white matter injury, a potential marker of subsequent neurodevelopmental impairment.

The study demonstrated, as other investigators before them have as well, that cerebral autoregulation is impaired in many children after congenital heart surgery. The study failed, however, to show a relationship between either rSO₂ or autoregulation with the development of white matter injury, as measured by magnetic resonance imaging. The lack of association between these indices and white matter injury raises as many questions as it answers. It is possible that the relatively heterogeneous population in this study may have made it more difficult to discern associations. At least one previous study has suggested that prolonged low rSO₂ (<45% for >180 minutes) is associated with the development of new white matter injury on magnetic resonance imaging scanning.² Given such conflicting data, one cannot say definitively that low rSO₂ or impaired cerebral autoregulation do not affect brain injury. It is also important to realize that the true significance of white matter injury after

congenital heart surgery is not well established. White matter injury described in the congenital heart surgery population is often relatively mild compared with that seen in the preterm population and has been shown to resolve with time. The relationship of white matter injury to subsequent developmental test scores has been variable, with a number of investigators failing to show a strong association. It is possible that impaired rSO₂ or impaired autoregulation may indeed affect developmental outcomes without demonstrating a clear association with the white matter injury seen in the postoperative period. Importantly, one team of investigators found that cerebral tissue oxygenation index, which can be considered a surrogate for cerebral autoregulation, has been shown to correlate with developmental test scores in the congenital heart disease population.³ Accordingly, Claessens and colleagues¹ would do well to follow this cohort through time and consider formal developmental testing.

For the near term, it is unclear how much focus clinicians should place on cerebral rSO₂ in terms of perioperative brain protection. The work by Claessens and colleagues¹ adds to the growing but important literature that provides insights into the early operative and postoperative hemodynamics of the process of brain injury and subsequent developmental outcome.

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