nutrient delivery to the rapidly developing neonatal brain earlier, by preponed surgery in d-TGA, might be an effective strategy to reduce brain injury. Considering the findings of Peyvandi and colleagues, patients with d-TGA and low TBV could especially benefit from this strategy. Further studies are needed to investigate the origin and predictors of preoperative brain injuries and strategies to prevent them.

These findings add an important piece to the puzzle of myriad risk factors for neurodevelopmental delays in children with CHD. As a result, TBV analysis in prenatal brain MRI can be used for a fetus with d-TGA as a predictor for increased risk of postnatal brain injury, and accordingly, adapted perinatal management in this high-risk group can be planned in advance. We congratulate Peyvandi and colleagues for their excellent work.

References
of intense clinical and research interest. Initially, it was believed that our surgeries, by the exposure of the developing brain to cardiopulmonary bypass and its associated systemic inflammatory response, were responsible for the observed white matter injury (WMI) and delayed motor skills commonly reported. Over the past 2 decades with increased utilization of prenatal and preoperative brain imaging, there is now growing evidence that brain abnormalities are actually present before the surgical repair. It is becoming increasingly understood that impaired brain maturation influences more late neurologic outcomes than punctual injuries inflicted by surgery.

Peyvandi and colleagues test the hypothesis that delayed brain development in fetuses with d-transposition of the great arteries (d-TGA) or hypoplastic left heart syndrome (HLHS) increases their postnatal susceptibility to acquired WMI. A total of 63 patients were prospectively enrolled across 3 study sites. Fetal and preoperative brain magnetic resonance imaging scans have been done to measure total brain volume (TBV) and assess the presence of WMI. In patients with d-TGA, an association with lower TBV and moderate-severe postnatal WMI has been demonstrated. In patients with HLHS, no association was noted between TBV and postnatal WMI.

This study reinforces once again our knowledge that fetuses with congenital heart disease have impaired brain maturation, which now seems the primary anatomical anomaly leading to the observed impairments. For the first time, this study seems to bring an argument to the suggestion that newborn infants with impaired brain maturation might be more susceptible than others to compromised hemodynamic status, in particular hypoxia, because newborn infants with d-TGA seem to be more affected than those with HLHS. The findings of this study present only 1 piece of a complex puzzle that warrants further investigation. It also demonstrates the multifactorial etiology of brain anomalies in patients with critical congenital heart defects.

Our minds can now drift to a distant future when in utero intervention targeted at improving cerebral circulation and oxygenation will take place. In a less distant future, we may conceive that newborn infants identified to have worse brain maturation would be directed toward earlier intervention to provide them as early as possible with the best possible brain perfusion and oxygenation. This goes along with the recently published study showing a progressive decrease in cerebral oxygenation during the time between birth and heart surgery. We can now conceive the day when the decision on time of delivery and neonatal procedures will be made by heart teams, including obstetricians and neurologists, in discussions taking place during fetal life.

References