Miles to go before we sleep

Susan R. Hupp, MD, MSCR,^a and R. D. B. Jaquiss, MD^b

From the ^aSection of Critical Care Medicine, Department of Pediatrics, and ^bDepartment of Cardiovascular and Thoracic Surgery, UT Southwestern Medical Center and Children's Medical Center, Dallas, Tex.

Disclosures: Authors have nothing to disclose with regard to commercial support.

Received for publication Aug 27, 2017; accepted for publication Aug 30, 2017; available ahead of print Sept 30, 2017.

Address for reprints: R. D. B. Jaquiss, MD, 1935 Medical District Dr, Dallas, TX 75235 (E-mail: robert.jaquiss@utsouthwestern.edu).

J Thorac Cardiovasc Surg 2018;155:301-2

0022-5223/\$36.00

Copyright © 2017 by The American Association for Thoracic Surgery

<http://dx.doi.org/10.1016/j.jtcvs.2017.08.101>

With survival after surgery for complex congenital cardiac malformations a matter of near routine, the opportunity and imperative to broaden our focus to detection and prevention of associated neurologic impairment has emerged.¹ With improved clinical instruments to quantify neurodevelopmental status,²⁻⁴ focus has turned to the quantitative description of associated neuroanatomic pathology. Peyvandi and colleagues⁵ have demonstrated that neonates with hypoplastic left heart syndrome (HLHS) and transposition of the great arteries (TGA) have different but substantial rates of preoperative brain injury and postoperative new brain injury, and different rates of early postoperative brain growth.

Some findings in the study are not surprising: As might be expected with abnormal fetal circulation, patients with HLHS and TGA have delayed but similar brain development at preoperative assessment.⁶ Another previously described⁷ (but no less alarming) finding was the marked rate of new brain injury (30% in TGA and 56% in HLHS). A novel finding was the difference in rates of postoperative brain growth, which was significantly faster in the TGA patients, a difference not accounted for by the differential rates of brain injury between groups. In fact, on a positive note for developing brains, despite volume loss from injury, an injured brain continues to develop at a rate similar to brains without injury.

How are we to use the new and provocative information presented in the study? At a minimum, skeptics should be moved to contemplate adding magnetic resonance imaging to routine assessment of neonates before and after surgery. Further, our community must view the prevalence of new injury as a call to action: If present strategies result in neurologic injury in 30% to 50% of our patients, there is room for improvement. A major limitation of the study is the inability to precisely time the new brain injury, hampered in part by lack of information regarding the conduct of the operations, but also by the necessary 2- to 3-week delay before follow-up imaging. It is tempting, but likely wrong, to assign complete causality to the perfusion technique used. It is perhaps useful to contemplate the differential rates

Susan R. Hupp, MD, MSCR

Central Message

Magnetic resonance imaging should be considered for routine assessment of neonates before and after surgery. Focus should be paid to advancing care strategies with the goal of improving outcomes beyond survival.

See Article page 291.

of injury while remembering that after surgery 1 group remains vulnerable to paradoxical embolism (the HLHS group), whereas the other is essentially protected. This might lead a cynic to suggest the period of risk that is most different is the phase that follows the operating room.

On the other hand, fundamental differences between groups must be acknowledged. Whereas repair for TGA results in nearly normal cardiovascular physiology, stage I palliation for HLHS leaves patients cyanotic and at risk for low cardiac output and cardiac arrest (indeed more common in the HLHS cohort in the present study). The difference in postoperative brain growth in these 2 populations should not be surprising. Ultimately, although Peyvandi and colleagues⁵ may provoke more questions than they answer (a common result of good studies), they remind us that whereas we have achieved great progress in ensuring survival of our patients, there remains much to do in protecting them and their brains.

References

1. Marino BS, Lipkin PH, Newburger JW, Peacock G, Gerdes M, Gaynor JW, et al. Neurodevelopmental outcomes in children with congenital heart disease: evaluation and management: a scientific statement from the American Heart Association. *Circulation*. 2012;126:1143-72.
2. Wechsler D. *Wechsler Preschool and Primary Scale of Intelligence-3rd Edition (WPPSI-III)*. San Antonio, TX: Pearson Clinical; 2002.
3. Beery KE, Buktenica NA. *Beery-Buktenica Developmental Test of Visual-Motor Integration, 6th Edition, the (BEERYTM VMI)*. San Antonio, TX: Pearson Clinical; 2010.
4. Achenbach TM, Rescorla LA. *ASEBA Child Behavior Checklist for Ages 1.5-5 (CBCL/1.5-5) and ASEBA Caregiver-Teacher Report Form for Ages 1.5-5 (C-TRF)*. Burlington, VT: Psychological Assessment Resources; 2001.

5. Peyvandi P, Kim H, Lau J, Barkovich AJ, Campbell A, Miller S, et al. The association between cardiac physiology, acquired brain injury and postnatal brain growth in critical congenital heart disease. *J Thorac Cardiovasc Surg.* 2018;155:291-300.e3.
6. Limperopoulos C, Tworetzky W, McElhinney DB, Newburger JW, Brown DW, Robertson RLJR, et al. Brain volume and metabolism in fetuses with congenital heart disease; evaluation with quantitative magnetic resonance imaging and spectroscopy. *Circulation.* 2010;121:26-33.
7. McQuillen PS, Barkovich AJ, Hamrick SEG, Perez M, Ward P, Glidden DV, et al. Temporal and anatomic risk profile of brain injury with neonatal repair of congenital heart defects. *Stroke.* 2007;38(2 Suppl):736-41.