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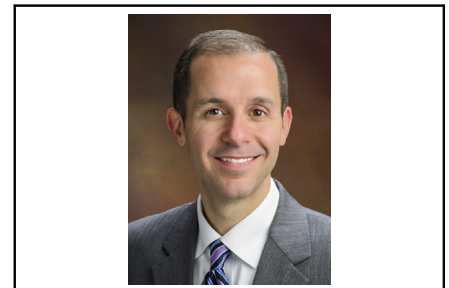


Commentary: Truncus among us

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The first complete repair of truncus arteriosus (TA) was performed in 1962 by Sloan, but not published until 1974 by Behrendt and colleagues¹ as part of a larger cohort of patients. Ebert and colleagues^{2,3} published a series of complete repair of TA in both 1976 and 1984 on infants aged 6 months or less and reported an operative mortality of 11% to 20%. Because the patients were a few months of age or older, not surprisingly, the most common cause of mortality was pulmonary hypertension. Because of weight loss and congestive heart failure in addition to pulmonary hypertension, Bove and colleagues⁴ advocated and reported success repairing TA in the neonate in 1989.

Naimo and colleagues⁵ present a 40-year perspective on TA repair in Australia at multiple centers. There are interesting findings that contradict conventional wisdom about preoperative TA physiology and timing of operation among other things. The authors report a neonatal



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CENTRAL MESSAGE

TA has been repaired successfully in the neonate for 30 years. Advances in imaging, operative techniques, and perioperative care have contributed to improving morbidity and mortality.

mortality of 23.7% compared with 7.4% in older patients. More specifically, they state that neonates in week 1 of life (because of concomitant anomalies like truncal insufficiency, arch obstruction, and coronary anomalies) and week 4 of life (because of congestive heart failure/pulmonary over circulation) had the worst neonatal outcomes. Only 53% of those undergoing TA repair in the most recent era (1999-2018) had the operation during the neonatal period. Their mortality and timing of operation are contrasted with the North American approach to TA. Today, it is standard of care to repair TA in the first week of life. The Society of Thoracic Surgeons Congenital Heart Surgery Database reports a mortality for TA of 9.5% (interquartile range, 0.0%-15.4%).⁶

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Many institutions have mortality less than 5%.⁷ It seems that mortality from pulmonary hypertension or congestive heart failure could be avoided by offering operation to all patients with TA in the first week of life. This would eliminate the less complex patients at risk for pulmonary hypertension while waiting for an operation during week 4 of life or later. Coronary anomalies can make any repair, including TA, more complex. However, better pre-operative coronary imaging (including magnetic resonance imaging and computed tomography), lessons learned about coronary arteries crossing the right ventricular outflow tract, and having a high index of suspicion for intramural coronary arteries with TA have lessened the impact of coronary anomalies as a risk factor. This is partially evidenced by the coronary artery anomaly manuscripts the authors cite from 20 to 30 years ago, save one of their own, which includes many of the cohort reported herein.

The 40-year report is impressive and commendable. However, I would argue for a slightly different approach: offering repair to all TA in the first week of life (including those with arch anomalies and truncal regurgitation),

proper imaging of the coronary arteries if concerned, and not intervening on moderate truncal regurgitation or less. This usually avoids the development of pulmonary hypertension and congestive heart failure without adversely affecting risk.

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