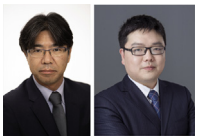

REPLY FROM AUTHORS: RESCUE BIDIRECTIONAL CAVOPULMONARY SHUNT FAILURE

Reply to the Editor:

Patients requiring bidirectional cavopulmonary shunt (BCPS) takedown may represent some of the greatest-risk candidates for stage II palliation for single-ventricle anatomy. The mortality associated with BCPS takedown is as high as 25% to 71.4% in the literature.[1,2] Perseveration of antegrade pulmonary blood flow may help borderline patients to maintain reasonable arterial saturation early after BCPS. The trade-off is that the antegrade blood flow causes some competing flow with the superior vena cava (SVC) flow, thereby potentially causing SVC syndrome. In this issue of the *Journal*, Türköz and Doğan[3] reported an alternative approach of providing additional antegrade pulmonary blood flow by adding an aortopulmonary shunt to the left side of the BCPS without takedown and placing a tight band between the aortopulmonary shunt in 2 patients with BCPS failure. Systemic oxygen saturation was dramatically increased from 50% and 60% to 70% to 75%, whereas only a +1- to 2-mm Hg increase was seen in the BCPS pressure. Another attractive aspect of their approach is they used a 6-0 polypropylene suture, which allows the subsequent transcatheter debanding during follow-up.

The authors should be congratulated for their innovative, life-saving approach to BCPS failure. Indeed, more questions may be raised. Is this approach suitable for all patients with BCPS failure? How can we decide the location of aortopulmonary shunt on the left pulmonary artery? What is clear is that the growth and development of the pulmonary vasculature are abnormal in most patients with a univentricular heart. Decreased pulmonary blood flow may present in fetal life, and an initial systemic-to-pulmonary shunt may cause pulmonary artery distortion as well as a maldistribution of flow to the pulmonary arteries, resulting in a combination of pulmonary hypoplasia some areas and vascular disease in other areas. Also, aortopulmonary collaterals (APC) flow is not insignificant in this population according to our cardiac magnetic resonance measurement.[1] The APC flow maybe maldistributed as well and could cause a significant retrograde flow to the affected lung, competing at SVC flow. Given that the pulmonary artery flow, APC flow, and pulmonary resistance could be substantially different between both lungs, adding an aortopulmonary shunt to the left of the BCPS may further compromise the BCPS physiology if the SVC-supplied right lung is mainly affected by pulmonary vascular abnormalities.

This report adds another option of rescue treatment for BCPS failure, although we would be cautious using this strategy for the aforementioned reasons. The mechanisms associated with BCPS failure are complex, as the abnormalities in the cerebral and pulmonary vascular beds may both contribute to the unsuccessful BCPS physiology. A better understanding of the anatomy and physiology of these 2 vascular beds can facilitate the choice of treatment in this inherently high-risk population.

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The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Shuhua Luo, MD, PhD[a]
Osami Honjo, MD, PhD[b,c]
[a]*Department of Cardiovascular Surgery*
West China Hospital of Sichuan University
Chengdu, China
[b]*Division of Cardiovascular Surgery*
The Labatt Family Heart Centre
The Hospital for Sick Children
[c]*Department of Surgery*
University of Toronto
Toronto, Ontario, Canada

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