

The second case remained stable with patent aortopulmonary shunt and BCPS. Both patients still remain stable with a systemic saturation of 75% to 87%.

By adding an aortopulmonary shunt to the left of the BCPS without takedown and placing a tight band between the aortopulmonary shunt and the BCPS, systemic oxygen saturation raised over 75% while only a +1- to 2-mm Hg increase in the Glenn pressure was seen in our cases. For attaching each tip of band, we used 6-0 polypropylene suture, which is thin enough to allow and facilitate transcatheter dilation of banding segment. Thus, it enabled transcatheter debanding during follow-up (Figure 1, B).

This approach has been used for unilateral pulmonary arterial obstruction/hypoplasia to increase the pulmonary blood flow. Casella and colleagues⁴ referred this approach as the “Super Glenn” approach. In conclusion, this technique can be a life-saving strategy in early deep hypoxemia and BCPS failure without BCPS takedown.

Rıza Türköz, MD
Abdullah Doğan, MD

Department of Cardiovascular Surgery
Acibadem Bakirkoy Hospital
İstanbul, Turkey

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**REPLY: ADDITIONAL
“CUSTOMIZED”
AORTOPULMONARY
SHUNT MAY INCREASE
“VIS A TERGO” TO
RESCUE A FAILING
BIDIRECTIONAL
CAVOPULMONARY
SHUNT**



Reply to the Editor:

I discussed in my first Commentary that the necessity of an unrestricted passive systemic blood flow to the pulmonary artery is of paramount importance for the optimal function of a Fontan circulation.^{1,2} As additional information, Türköz and

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Dogan³ report on 2 cases in which an additional aorto-to-left pulmonary artery shunt with a banding performed between the Glenn anastomosis and the shunt to pulmonary artery anastomosis helped to manage a situation of a failing bidirectional cavopulmonary shunt. This shunt, with a minimal pulsatility, helped to increase the “vis a tergo” (a power from behind) that I had postulated as important feature for the success of a Glenn shunt. In fact, the solution proposed by the colleagues from Turkey was successful and contributed most probably to reduce the perioperative risks that may have been associated with a take-down. Both patients survived, had an improved oxygen saturation, and central venous pressure decreased slightly within hours.

I believe there are important aspects that may be discussed in relation to this additional report: the size of the shunt (3 instead of 4 mm?) and the optimal technique to regulate the flow (and therefore the pressure gradient) across the shunt.

Another important point would be to discuss the optimal time point to disconnect the main pulmonary artery from the pulmonary bifurcation during the index procedure. One advantage of preserving the continuity would be that the shunt could be anastomosed to the main pulmonary trunk (perhaps easier in some instances because of the size) and the banding could be placed proximal to the bifurcation. This would eliminate the risk of additional intervention on the left pulmonary artery, even though this was successfully performed in the described case.

This may appear to be in contradiction to what I wrote in my first Commentary, eg, that one of the most important strategies to increase superior vena cava blood flow is probably the elimination of antegrade pulmonary blood flow that competes with superior vena cava flow. With the interposition of a tight banding, this contradiction may become relative only.

Thierry Carrel, MD

From the Department of Cardiovascular Surgery,
University Hospital and University of Bern, Bern,
Switzerland.

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REPLY: SALVAGING A PATIENT WITH A FAILING BIDIRECTIONAL CAVOPULMONARY SHUNT REQUIRES CAREFUL ANALYSIS



AND TIMELY CORRECTIVE ACTION

Reply to the Editor:

The bidirectional cavopulmonary shunt (BCPS) is the cornerstone of the staged palliation of a functional single ventricle. When a BCPS “fails,” the causation may be anatomic, physiologic, or a combination of both. Anatomic abnormalities that are associated with hypoxemia following a properly constructed BCPS could be related to stenosis, distortion, or hypoplasia of one or both pulmonary arteries. Physiologic causes of BCPS failure include increased pulmonary vascular resistance (which can be due to reversible or permanent pulmonary pathology), pulmonary venous obstruction, atrioventricular valve regurgitation, and ventricular dysfunction.

The selection of patients for BCPS palliation is being continually refined in the modern era. Luo and colleagues¹ reported a retrospective analysis of a group of patients in which BCPS failure was predicted by low preoperative superior vena cava (SVC) blood flow detected by cardiac magnetic resonance imaging (CMRI). In their model, low preoperative CMRI-derived SVC blood flow is probably a surrogate for ventricular dysfunction, systemic atrioventricular valve regurgitation, or maldistribution of systemic blood flow.

Turkoz and Dogan² successfully salvaged 2 patients suffering from hypoxemia immediately following a BCPS procedure. In both cases, a systemic to pulmonary artery shunt was added to a BCPS in the setting of documented acute elevation of the pulmonary artery pressure. To prevent SVC syndrome, the authors banded the proximal shunted pulmonary artery. The technique used by Turkoz and Dogan can salvage a difficult situation, thereby creating reasonable palliation at the cost of proximal pulmonary artery stenosis. We do not know the long-term outcomes for their patients. Ideally, anatomic abnormalities are corrected, not created, at the time of the BCPS connection.

Luo and colleagues’ report demonstrates that sophisticated preoperative analyses might not always predict ideal

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outcomes. The 65 consecutive patients they selected were deemed to have appropriate anatomy and physiology for a BCPS. Yet, 7 patients had BCPS takedown, with 5 late deaths. In addition, 4 other patients died with an intact BCPS. Eleven BCPS failures in 65 patients who had been carefully selected for BCPS indicates that the process of preoperative analysis requires additional refinement. In contradistinction to the report of Turkoz and Dogan, none of Luo and colleagues’ patients were immediate/intraoperative failures. Whether any of these patients were candidates for the addition of accessory pulmonary blood flow (APBF) is unclear from their report. Adding APBF can salvage a failing BCPS, but it is unknown whether the addition of APBF makes the patient a better long-term candidate for the Fontan–Kreutzer circulation.^{3,4} Some patients may do well for a prolonged period after palliation with a combination of BCPS and APBF. When the cause of acute hypoxemia following BCPS is due to reversible pulmonary pathophysiology, adding APBF may not only be life-saving, but also relatively harmless. Long-term follow-up will ultimately determine whether adding APBF to a failing BCPS has a negative impact on the early, mid-term, or late Fontan–Kreutzer outcome. In the meantime, further analyses are needed to determine how low SVC blood flow at CMRI reveals a propensity for BCPS failure.⁵

John J. Lamberti, MD
Department of Cardiothoracic Surgery
Stanford University
Palo Alto, Calif

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