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Commentary: Innominate turndown—a better insurance for the Fontan circulation?

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Protein-losing enteropathy and plastic bronchitis are significant complications of Fontan circulation. Although protein-losing enteropathy or plastic bronchitis develops in up to 5% of Fontan patients, these complications can lead to circulatory failure or death in almost a one-third of the patients with these complications.¹

In their article in this issue of *JTCVS Techniques*, Hraska and coauthors² not only demonstrate the lymphatic pathophysiology behind these severe complications, but also present the techniques and outcomes of a surgical remedy in the form of innominate vein turndown procedure. Out of 14 patients who underwent innominate vein turndown procedure presented in this series, 2 underwent the procedure concomitantly with Fontan completion and 12 did so after the development of Fontan failure. After a follow-up of 3 to 36 months, there were 3 deaths in the series, all in patients with end-stage Fontan failure. Almost one-third of the survivors developed either narrowing at the innominate vein atrial anastomosis or thrombosis and required percutaneous reintervention on the pathway. In addition to innominate vein turndown and direct anastomosis, the authors also describe the use of a ring-supported expanded polytetrafluoroethylene tube as an interposition graft to allow a tension-free innominate vein-to-atrial connection using the “dunked” technique, which may reduce the anastomotic reintervention rate and even allow performance of the procedure without cardiopulmonary bypass. Additional

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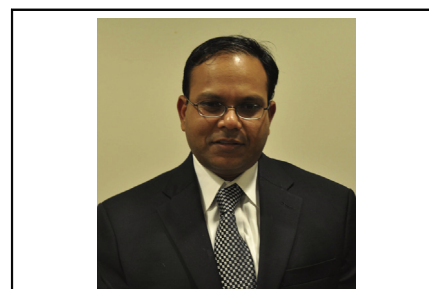
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CENTRAL MESSAGE

Innominate vein turndown may offer the benefits of fenestration with the added advantage of thoracic duct decompression.

banding of the jugular vein may be necessary postoperatively to avoid/correct clinical hypoxemia by reducing the amount of left-to-right shunting.

Despite the short-term advantages of fenestrating a Fontan,³ its long-term benefits have been debatable.⁴ As we look for additional palliative methods to ensure a well-functioning Fontan free of complications in the long term, the theoretical premise of the innominate vein turndown is promising. Like a fenestration, it can provide the right-to-left pop-off shunt for the Fontan circuit, but unlike a fenestration alone, it can also achieve a physiologically normal thoracic duct pressure. The performance of innominate vein turndown in a small subset of patients in this series shifts the indications of this novel technique from the salvage/therapeutic realm to prophylactic applications, where in theory it could replace fenestration of the Fontan.

Given the establishment of lymphatic hypertension after stage II palliation with superior cavopulmonary connection even before the Fontan,⁵ would it be far-fetched to consider this procedure at stage II, or as an added interim step before Fontan, to preserve the lymphatic architecture? Or would this compromise the adequacy of pulmonary blood flow?

Given the propensity of the venous system to form collaterals and thromboses, the long-term efficacy of innominate vein turndown could deteriorate over time. Hepatic and splanchnic venous congestion leading to increased lymphatic production and Fontan failure is not primarily addressed by the innominate vein turndown. Would lymphatic decompression alone reverse/prevent the sequelae of hepatic and splanchnic venous congestion? A balance of these factors will eventually determine the

role, timing, and efficacy of innominate vein turn-down as insurance for a longer-term healthy Fontan circulation.

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