stented and had an intact interventricular septum. In contrast, our patient did not have a PDA and the small muscular VSD was restrictive.

It is possible that the patient could have undergone a primary ASO with a similar outcome. But the improvements in LV parameters afforded a certain degree of safety that we believe helped the outcome. The intraoperative difficulties in removing the stent were substantial and perhaps might decrease with more experience. The appropriate size of the stent remains unclear. A larger VSD could perhaps be more helpful. Creating or increasing the VSD size by using cutting balloons and stent has been reported for different clinical applications. The Boston group⁶ reported in detail stenting of the systemic ventricular outflow tract and creation or enlargement of a VSD using stents as potential therapeutic options in rare patients with congenital heart disease who develop significant ventricular hypertension due to outflow obstruction. We are not aware of any previous report of use of this strategy to retrain the LV in d-TGA.

CONCLUSIONS

A regressed LV may be prepared for ASO by stenting a muscular VSD in carefully selected patients. To the best of our knowledge, this approach has not been reported earlier.

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EDITORIAL COMMENTARY

A new approach to retrain the deconditioned left ventricle in transposition of the great arteries

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Delayed presentation of transposition of the great arteries can be a challenging issue when the patient is seen late with a deconditioned left ventricle. These patients require a fair bit of judgment regarding how best to proceed, and every patient needs to be treated as an individual case. The authors Talwar and colleagues¹ from New Delhi present a novel solution that uses stenting of a restrictive ventricular septal defect (VSD) to retrain the left ventricle before proceeding with an arterial switch procedure.

Typical solutions for these patients include banding of the pulmonary artery for “retraining” of the left ventricle and planning for temporary mechanical support after an arterial

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Central Message

A new method is presented for retraining the deconditioned left ventricle in transposition of the great arteries by stenting the VSD.

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switch procedure. Pulmonary artery banding is not a benign procedure, and mechanical support after surgery can be unpredictable, with the potential for significant bleeding issues as a result of anticoagulation. In this particular case, the presence of a restrictive muscular VSD provided the opportunity to stent the VSD open to allow preparation of the left ventricle. Stenting the VSD obviated the need for initial palliative surgery and allowed easy monitoring of the progress of the left ventricle. One caveat with this technique is that not all VSDs are amenable to stenting, because the risk of inducing heart block is high in the more commonly seen perimembranous type of VSD. In this reported case, the VSD was muscular, however, which made the risk of heart block low.

The use of interventional cardiology procedures for initial palliation is a relatively new and promising alternative. Some groups have explored stenting of the right

ventricular outflow tract in patients with symptomatic tetralogy of Fallot.² The ductus arteriosus has also been stented in lieu of a surgically placed systemic-to-pulmonary arterial shunt for temporary palliation of ductus-dependent lesions.³ The ultimate benefit of this pathway remains to be fully elucidated, but the incorporation of these new techniques is an outstanding way to expand and improve the care of these challenging patients.

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Tailored therapy for aggressive dilatation of systemic veins and arteries may result in improved long-term Fontan circulation

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High central venous pressure (CVP) is a key pathophysiologic factor underlying increased morbidity and mortality in patients undergoing the Fontan procedure. Therefore, maintaining the lowest possible CVP is a primary therapeutic goal for improving patient outcome after this procedure. Currently, reduction of pulmonary resistance is used as a means to achieve this goal. However, even patients who have low pulmonary resistance can have CVP that is much higher (generally, >10 mm Hg) than levels observed in healthy 2-ventricle circulation. We hypothesized that therapy tailored to increase systemic venous capacitance (venous pool) using venodilators would yield a Fontan

Modulation of venous capacitance yielded Fontan circulation with minimal central venous pressure.

Central Message

Venous capacitance can be a viable and novel therapeutic target to yield Fontan circulation with minimal CVP.

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circulation with a lower CVP than is generally accepted for this procedure, but that is close to healthy CVP values.