Aggressive Coiling of Aortopulmonary Collaterals in Single-Ventricle Patients Is Warranted

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Aortopulmonary collaterals (APCs) are present in up to 80% of single-ventricle patients undergoing pre-Fontan catheterization [5, 7, 13, 14]. It is theorized that hypoxia is a potent stimulus for the development of APCs [17]. No general consensus exists as to their impact on the mid- to long-term course of single-ventricle patients. Bradley et al. [2] argued that their presence in the immediate postoperative period after Fontan repair had no impact on the duration of pleural effusions or resource use with regard to length of mechanical ventilation, intensive care unit (ICU), stay, or hospital stay. Although their findings were counterintuitive, even to the authors, this single study has been used as justification to ignore aortopulmonary collaterals not only at the pre-Fontan catheterization but also in the long term at a number of centers (informal polling and personal communication).

In the centers performing catheter occlusion of APCs, the criteria for occlusion are subjective and qualitative or based on hemodynamic data that have not been validated in terms of defining what significant APC flow is. Some centers rely on an oxygen step-up from the superior vena cava to branch pulmonary arteries to determine which vessels to occlude in which patients. However, it is clear that many patients would be excluded from catheter occlusion using only this parameter given that collateral flow often enters the lungs distal to the site of sampling. Some centers use filling defects from APC competitive flow noted on pulmonary artery (PA) imaging to discern the degree of negative washout from collaterals (Fig. 1). Some try to gauge the pulsatility of the branch PAs to

assess the significance of flow, and some use the PA pressures and end-diastolic pressure measurement of the single ventricle, assuming elevated values are due to volume overloading. Others use the size of APCs, the density of the pulmonary capillary blush (Fig. 2), and opacification of the pulmonary venous return to decide when to occlude. The aforementioned parameters, either singly or in combination, may provide clues as to the volume of collateral flow but can be used only as rough guides.

Bradley et al. [2] addressed this issue by quantitating APC flow as the volume of blood returning from the pulmonary venous vent during Fontan surgery [2]. They conceded, however, that their methods were nonphysiologic because flow was measured during hypothermic cardiopulmonary bypass and certainly did not represent a real-life situation.

Lim et al. [15] quantitated APC flow using thermal indicator dilution methods in a more physiologic state during the pre-Fontan catheterization, but patients with significant semilunar or atrioventricular valve insufficiency invalidated this technique and were excluded from their study, disqualifying an important subset of patients [10]. In addition, these invasive techniques to evaluate APC flow made it difficult to follow cases in a longitudinal manner. Magnetic resonance imaging (MRI) assessment of PA and pulmonary vein flow using phase-contrast velocity mapping is a promising and accurate noninvasive method for calculating APC flow at the pre-Fontan catheterization and beyond, but it is expensive and time consuming and usually requires general anesthesia [15].

Finally, the long-term impact of permanently occluding vessels off the subclavian arteries and thoracic descending aorta is unknown. A syndrome of chest pain, irritability, and low-grade fevers lasting 3 to 5 days is common after occlusion. Given this backdrop, what is an interventionalist

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Fig. 1 Injection in a cavopulmonary shunt. Note that no flow exists in the right upper lobe despite a patent vessel due to competitive flow from aortopulmonary collaterals (APCs)

Fig. 2 Injection in an intercostal artery off the thoracic aorta. The *small arrows* depict the normal intercostal vessel. The *large arrow* depicts the sizeable collateral supplying much aortopulmonary collateral (APC) flow

to do when he or she encounters APCs during the pre-Fontan catheterization?

It is well known that chronic volume loading raises filling pressures, induces adverse remodeling, triggers

apoptosis, and activates neurohormones, all of which conspire to cause progressive ventricular dysfunction. This pathophysiologic cascade is well described in the adult literature, and compelling data suggest that these same mechanisms apply to the single-ventricle patient [3, 6, 9, 12, 16]. Additionally, Ascuitto and Ross-Ascuitto [1] have demonstrated that competitive flow and pressure elevation from APCs can lead to dramatic energy losses, resulting in elevated filling pressures and a decreased cardiac index in a hydrodynamic model, further implicating APCs as pathologic entities.

Confusing matters further, the anatomy of APCs is poorly described in the literature. Consequently, the techniques to occlude these vessels effectively have been suboptimal. In the study of Bradley et al. [2] cited earlier, it is unclear whether APC flow was effectively occluded because the specifics of the occlusion technique were omitted, and their conclusions therefore may have been flawed with regard to the impact of APC flow in the post-Fontan state [2].

It is critically important to understand that arteries arising from the subclavian arteries (including the internal mammary arteries, thyrocervical trunk, lateral thoracic branches, and so forth) and the intercostal arteries normally have extensive anastomotic connections between them (Fig. 3). Occluding only the origins of these vessels, which have APCs arising from them, is inadequate for occluding APC flow because distal feeder vessels will always revascularize them. The entire length of the feeder vessel, when applicable, must be occluded to prevent this phenomenon from occurring (Figs. 4, 5, 6, 7). This task has been simplified by the availability and use of coaxial microcatheters (Tracker-Target Therapeutics, Fremont, CA, USA; Progreat-Terumo Interv. Systems, Somerset, NJ, USA) and platinum microcoils (Micronester, Cook Inc.-Bloomington, IN, USA; Target platinum coils, Target Therapeutics -Fremont, CA, USA).

Invariably, APCs are encountered that connect to a vast network of collaterals but cannot be entered due to their small origins or extremely acute takeoffs. In these situations, the surgeon can deflate the lungs at Fontan surgery and Bovey electrocauterize these collaterals from the chest wall.

Although methods to calculate APC flow need refining so that APCs are addressed appropriately at the pre-Fontan catheterization as well as longitudinally, we believe that the presence of APCs in single-ventricle patients is detrimental to optimal ventricular function. Phase-contrast MRI may prove to be the most reliable noninvasive technique for evaluating APC flow. But until this method becomes more standard, we believe the parameters mentioned earlier in paragraph 2 all have merit and should be considered in the decision to occlude these vessels. There is a

Fig. 3 Anastomotic connections of the thoracic aorta. From the *Atlas of Vascular Anatomy* (1997) by R. Uflacker (Charleston, SC)

Fig. 4 Injection in the left internal mammary artery (LIMA). The *small arrows* depict anastomotic connections to anterior intercostal arteries supplying a large amount of aortopulmonary collateral (APC) flow. Notice the connections along the entire length of LIMA and the dense filling of the left pulmonary vein (*large arrow*)

Fig. 5 Injection in the left internal mammary artery (LIMA) after coil occlusion using Cook Micronester coils. The *arrow* depicts reflux into the lateral thoracic artery also supplying collateral flow

Fig. 6 Injection in the left lateral thoracic artery. The *small arrows* depict anastomotic connections to the anterior intercostal arteries and the left internal mammary artery (LIMA). The *large arrow* depicts prompt filling of the left pulmonary vein

Fig. 7 Injection in the left lateral thoracic artery after coil occlusion with Cook Micronester coils of the left internal mammary artery (LIMA) and the lateral thoracic artery. No residual collateral flow is seen

disturbing trend toward gradual attrition in single-ventricle patients partly due to ventricular dysfunction [8].

In addition to other well-described anatomic determinants for long-term survival, attention to elimination of

APC flow is important and often overlooked [4]. Using the rationale that early Glenn shunting (at about 4 months) is preferred for unloading the single ventricle and preventing deleterious long-term consequences, aggressive coiling of APCs should be viewed in this same light [11]. Despite our opinions, however, multicenter randomized prospective double-blind studies are needed to evaluate the impact of APCs on neurohormonal activation, volume loading, filling pressures, ventricular function, and correlation, with adverse consequences including protein-losing enteropathy, atrial arrhythmias, pleural effusions, ascites, and death.

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