The arterial switch procedure (ASO) has been adopted as the technique of choice for repair of transposition of the great arteries. Survival after neonatal repair exceeds 95%, with early mortality most often related to difficulties involving coronary transfer. As a result of many improvements in care, survival into adulthood now exceeds 90%, yet a number of residual lesions—neoaortic root dilation, neoaortic regurgitation, neopulmonary stenosis, and coronary artery disease—can persist. Of these, supravalvular neopulmonary stenosis is the most commonly reported complication, ranging from 2% to 40%, and it is the number one reason for reintervention. It has been well documented that a proportion of adults exhibit diminished exercise capacity, which has been linked to right-sided lesions. The article by Baggen and colleagues in this issue of the Journal gives us insight into the hemodynamic basis underlying these physiologic limitations: flow restriction at the main pulmonary artery.

The etiology of right ventricular outflow tract obstruction after an ASO procedure can be multifactorial, with contributors including the smaller native aorta often seen in cases with associated aortic arch obstruction, unusual spatial relationships of the aorta and pulmonary arteries or inadequate mobilization of the branch pulmonary arteries leading to distortion after the LeCompte maneuver, the technique of coronary artery harvesting and reconstruction of the neopulmonary sinuses, and postoperative cicatrix formation. In the past, perhaps we have been too cavalier in accepting mild to moderate right ventricular outflow tract gradients after the ASO procedure, leaning on the knowledge that the neonatal right ventricle is well prepared to handle modest degrees of afterload. Evidence is mounting, however, that this may be harmful with time. Absent any overt narrowing, increased peak flow velocities have been identified in the pulmonary trunk after the ASO, probably secondary to surgically induced loss of distensibility, resulting in delayed pulmonary flow propagation, right ventricular hypertrophy, and abnormalities in right ventricular relaxation despite preservation of systolic right ventricular function. Although they were not nearly as abnormal as in patients after atrial switch procedures, ventricular myocardial deformation (right ventricular strain parameters) were reduced in patients after ASO relative to control subjects. In addition, patients with pressure-loaded right ventricles displayed up-regulation of numerous microRNAs, which correlated negatively with systemic ventricular myocardial acceleration.

It is interesting that Baggen and colleagues have specifically identified main pulmonary artery narrowing as the primary culprit. Reduced cross-sectional main pulmonary arterial area was correlated with right ventricular stroke volume and systolic pressure at rest and likely limited effective increases in these parameters during exercise. They dutifully sought to eliminate the potentially deleterious effects of unilateral or bilateral branch pulmonary artery stenosis by evaluating the association between percentage peak oxygen uptake and the combined left and right pulmonary arterial areas, as well as the smallest pulmonary arterial branch area; however, they once again found that the main pulmonary arterial area remained the strongest independent predictor of reduced exercise capacity. Notably, main pulmonary arterial area was not associated with exercise capacity in patients younger than 18 years. This may represent an era effect. Close scrutiny of changes in the group’s technique with time reveals that the older patients underwent many more pulmonary arterial bandings before repair and also had the neopulmonary sinuses repaired with separate glutaraldehyde-treated pericardial patches. In contrast, the more current cohort of patients routinely had “pantaloon type” neopulmonary reconstructions with untreated pericardium which afforded them larger main pulmonary arterial areas.
If we now accept perforce that right ventricular outflow tract obstruction after the arterial switch procedure may have significant negative consequences, we must strive to relieve any residual obstruction as completely as possible. In light of the compelling data provided by Baggen and colleagues, more aggressive primary main pulmonary artery augmentation may be necessary in cases exhibiting any significant potential for obstruction. For those patients already “postswitch,” diligent follow-up is required. Ad interim, we can take some solace in the knowledge that reoperation and patch augmentation of the main pulmonary artery has been shown to have good intermediate results.

References