

Editorial



Beyond the Valve: Lifelong Management of Right Ventricular Outflow Tract Lesion in Adult Congenital Heart Disease

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Adults with congenital heart disease (ACHD) may have native or repaired right ventricular outflow tract (RVOT) issues, such as residual or recurrent RVOT obstruction or pulmonary regurgitation. Isolated or combined RVOT problems cause hemodynamic deterioration, leading to right ventricular dilatation and decreased ventricular function, tricuspid valve regurgitation, arrhythmia, and sudden death.¹⁾ Therefore, intervention in the RVOT may be required, including pulmonary valve repair or replacement, which is necessary for competent valve function along with RVOT procedures. Prosthetic valve replacement is essential if the native pulmonary valve cannot be preserved or valve preservation is not achieved during a previous cardiac surgery. Various prosthetic valves have been introduced for surgical pulmonary valve replacement (SPVR). In particular, bovine and porcine bioprostheses are commonly used in clinical practice because of their easy availability and various valve sizes. These valves are well known for their excellent long-term performance in the aortic valve position. However, there are few long-term outcomes of these valves in the pulmonic position, and there are few head-to-head comparisons of long-term outcomes between bovine and porcine prostheses. Kwak and colleagues²⁾ recently published our experience. A total of 258 SPVR were performed in 248 relatively young patients, with an average of 14.9 years at SPVR. During a median follow-up of 10.5 years, the risk of reoperation or prosthetic valve dysfunction was significantly higher in the bovine bioprosthesis group than that in the porcine bioprosthesis group.

In this journal, Ramchandani and colleagues³⁾ published the results of a propensity score matching analysis on a similar topic. The authors reported their 20-year experience of SPVR in 319 ACHD and comparatively analyzed the outcomes of bovine and porcine bioprostheses used in SPVR. Five years after PVR, the porcine bioprosthesis showed superior reintervention and structural valve deterioration (SVD) compared to the bovine bioprosthesis. However, the differences in age at SPVR, concomitant procedures, and follow-up period between the 2 groups were significant; therefore, propensity matching was performed to overcome these problems. After propensity score matching, the age at SPVR was 29.2 years in the bovine group and 27.8 years in the porcine group. In the matching analysis results, the risk of SVD after 5 years of SPVR was significantly higher in the bovine group, but there was no difference in reintervention. As mentioned by the authors, the same surgical team performed SPVR, but it was performed using different valves at 2 different institutions, and the follow-up period for porcine bioprostheses was relatively limited.

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The reason for the inferiority of bovine bioprostheses in the pulmonic position remains elusive. Recently, Pragt and colleagues⁴⁾ compared the flow and pressure profiles of a pericardial bioprosthesis in aortic and pulmonary artery positions. A pericardial bioprosthesis with excellent long-term performance in the aortic position resulted in decreased leaflet motion and incomplete valve closure in the pulmonic position, suggesting the possibility of leaflet degeneration due to pannus overgrowth into the valve leaflets. The 2 studies mentioned above help choose the better bioprosthesis in adolescent and young adult patients with RVOT dysfunction. Efforts should be made to reduce reintervention-related mortality or morbidity by SPVR using a more durable bioprosthesis in the pulmonic position. However, in retrospective studies, attempts to identify a better bioprosthesis based on SVD and reintervention as the primary endpoint require attention in several aspects. In both studies, SVD was defined as the hemodynamic deterioration of moderate to severe pulmonary regurgitation and/or a peak transprosthetic gradient >50 mmHg as assessed by echocardiography, and reintervention was defined as surgical or catheter replacement of the bioprosthesis. Capodanno et al.⁵⁾ and Dvir et al.⁶⁾ emphasized the importance of standardized definitions for SVD and bioprosthetic valve failure to better assess the long-term durability of bioprostheses. SVD should be considered an acquired intrinsic bioprosthetic abnormality defined as deterioration of the leaflets or supporting structures, leading to degenerative changes of the prosthetic valve materials and eventually associated valve hemodynamic deterioration, manifested as stenosis or regurgitation. For the precise evaluation of SVD, echocardiography, multi-detector computed tomography, and cardiac magnetic resonance imaging (CMRI) are used. Through these imaging modalities, SVD should be evaluated along with morphologic and hemodynamic SVD. In addition, freedom from reintervention does not reflect SVD well, because reintervention of the bioprosthesis can be performed for reasons other than SVD, such as non-SVD, valve thrombosis, and endocarditis. Furthermore, there are cases in which reintervention is not performed since meaningful SVD is not properly detected. A bigger problem is that the standardized definition for SVD and reintervention is mostly studied through the aortic valve; therefore, pulmonic bioprosthesis becomes more complicated. It is believed that the evaluation of bioprosthesis through echocardiography is difficult, and in particular, it is not easy to accurately evaluate the SVD of a bioprosthesis in the pulmonic position.⁷⁾ Caution is required when evaluating stenosis of the pulmonic bioprosthesis because, unlike in the aortic valve position, it is sometimes accompanied by subpulmonary RVOT obstruction or distal pulmonary bifurcation obstruction. In addition, it is not easy to quantitatively and accurately evaluate bioprosthetic pulmonary regurgitation using echocardiography alone; therefore, evaluation is performed using CMRI concurrently. Owing to these limitations, better bioprosthesis selection is possible only when the SVD of the pulmonic bioprosthesis and its reintervention are properly evaluated.

At this point, it is essential to manage RVOT dysfunction for life, especially in young ACHD patients, along with efforts to find a better bioprosthesis.⁸⁾ For pulmonic bioprostheses with severe SVD, surgical replacement and catheter reintervention are being actively carried out. When performing SPVR in ACHD with RVOT dysfunction or reoperation in patients with prosthetic valve failure, valve selection or RVOT reconstruction should be performed after fully considering potential transcatheter PVR (TPVR).¹⁾ The bioprosthesis should be placed at an appropriate tilting angle suited to the RVOT, and severe RVOT aneurysm, residual RVOT hypertrophied muscle, stenosis, or severe dilatation of the branch pulmonary artery should be corrected during SPVR. In addition, RVOT reconstruction should be performed simultaneously so that TPVR-related coronary artery compression does not pose a challenge. The material of the bioprosthesis and the design are essential; therefore, even if a small

bioprosthesis is placed, a TPVR of sufficient size could be possible in the future, and it is necessary to consider whether valve-in-valves and valve-in-valve-in-valves are feasible.⁹⁾¹⁰⁾

In conclusion, a lifelong strategy is mandatory to preserve the right ventricular function substantially through appropriate surgical and catheter reintervention for RVOT problems in relatively young ACHD, along with the effort to find an ideal bioprosthesis in the pulmonic position.

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