Doing the right thing at the right time: is there more to pulmonary valve replacement than meets the eye?

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This editorial refers to 'Quantitative assessment of homograft function 1 year after insertion into the pulmonary position: impact of in situ homograft geometry on valve competence†', by J. Nordmeyer et al., on page 2147

Prior to Lillehei’s first intracardiac repair, a child born with tetralogy of Fallot had a 50% chance of being alive at the age of 2 years.1 In the modern era, >90% of patients reach the age of 40.2 This dramatic increase in survival is primarily a reflection of the improvements in surgical technique and peri-operative care at the time of primary repair. Indeed, the overall late mortality and morbidity have not changed substantially during the last decades.2 Consequently there are a growing number of teenage and adult survivors after right ventricular outflow tract (RVOT) surgery, many of whom are destined to become symptomatic as a result of decades of free pulmonary incompetence. Previously thought to be the innocent bystander of successful relief of RVOT obstruction at the time of repair, pulmonary incompetence is now known to be the primary determinant of exercise intolerance, right ventricular dilation and failure, and ventricular arrhythmia and sudden death in these patients3. As a result, surgical pulmonary valve replacement late after repair of tetralogy of Fallot has gone from being relatively unusual to one of the most common operations performed in teenagers and adults with congenital heart disease.

Since Ross and Somerville reported the first aortic homograft conduit insertion for reconstruction of the right ventricular outflow tract in 1966,4 a variety of surgical alternatives have been reported. Largely driven by lack of availability of the homograft, the early alternatives included the now abandoned custom-made fascia lata valves, stented porcine valves, and bovine xenografts in pericardial tubes. In the 1980s, porcine-valved Dacron conduits (Hancock™, Medtronic) as well as bovine jugular venous xenografts (Contegra™, Medtronic) were developed,6 although the most commonly used conduits in the UK and North America remain glutaraldehyde fixed aortic or pulmonary homografts.6 Most recently, percutaneous pulmonary valve replacement using valved stents has become an alternative to surgery in selected cases, primarily those who have already undergone valve or conduit surgery.7,8 While, on average, performing much better than when placed as part of the primary operation,9 between 10 and 25% of RVOT valves/conduits inserted during re-operations will require further re-operation within 5 years.10

The need for reoperation is, at best, a clumsy index of success or failure of such procedures however. Nordmeyer et al.11 refine our understanding of this important topic by using magnetic resonance imaging (MRI) to assess the performance of homografts that were placed in a previously operated RVOT (Figure 1). They found that significant pulmonary incompetence (defined in their study as >20% of forward stroke volume) was present early after conduit insertion in 17% of their patients. While almost certainly an important observation, we would add a word of caution when defining pulmonary regurgitation as a percentage or fraction of total forward flow into the pulmonary artery. Two patients may have similar regurgitant fractions, but strikingly discrepant regurgitant volumes. Consequently, the burden (preload) on the right ventricle and therefore the prognosis of these two patients may be quite different. As an illustration of this point, we recently showed that regurgitant volume (measured in absolute terms) was a better predictor of right ventricular volumes than regurgitant fraction.12 Furthermore, the regurgitant fraction appears of limited value when assessing the timing of pulmonary valve replacement, leading the Leiden group to suggest a composite parameter of ‘corrected RV ejection fraction’ that incorporates stroke, end-diastolic and pulmonary regurgitant volumes, respectively.13

That having been said, Nordmeyer and colleagues are to be commended for their detailed analysis of how RVOT geometry and surgical technique may determine valve function early after insertion. Perhaps the most important message from their study...
is that a procedure that results in kinking of the conduit promotes ‘neo-pulmonary’ insufficiency, even in the absence of significant outflow tract obstruction (Figure 1A). They also found a relationship between the degree of incompetence and the discrepancy between the direct measurement of the conduit size ex vivo and its size (generally larger) after implantation, measured by MRI. The authors speculate that the cause of this discrepancy was the stretching of ‘too small’ a homograft for the size of the RVOT. This is supported by their observation that RVOT size prior to valve implantation was correlated with the degree of post-operative incompetence. No matter what the mechanism, it is clear that pre-operative geometry, and the ability to tailor the surgical procedure to it, may significantly affect the quality of subsequent valve function.

Most patients with tetralogy of Fallot will require several RVOT procedures during the course of their lives. As a result, their post-operative anatomy becomes their pre-operative anatomy. Thus, congenital heart surgeons and paediatric cardiologists must look not only to conduit longevity as a measure of success, but also to subsequent valve function and maintenance of optimal geometry, with an eye on subsequent procedures, either in the operating room or in the catheter laboratory. Currently, the surgeon is limited by the availability of homo- or xenografts which fit, approximately, a patient’s RVOT and pulmonary artery anatomy. While the prosthesis can be tailored to fit by trimming or by extending, this is clearly an imperfect art. Tissue engineering of heart valves holds promise in that it may become feasible to grow outflow tracts that match the recipient’s anatomy, by using a mesh that is based on previously obtained three-dimensional MR data,14 but this is both speculative and a post hoc solution. Rather, the current data might further support the shift towards earlier surgery in this group of patients. Although this shift has largely been in response to the rather unpredictable nature of right ventricular reverse remodeling when dilatation is severe, it may also be that the subsequent longevity of valve implants will be enhanced if surgery is performed prior to gross enlargement of the RVOT itself. While we struggle to define thresholds of right ventricular size or performance that predict a good long-term outcome for the ventricle, it may also be that independent thresholds need to be defined for RVOT geometry, which in turn might depend on the type of valve implant used, in order to optimize the performance of the implanted valve. Nordmeyer and colleagues should be applauded for providing the most detailed quantitative description of the reconstructed RVOT reported to date. Whether the lessons from this study will lead to altered surgical approaches remains to be seen, but they have raised the bar for the physiological assessment and reporting of operative outcomes for surgical procedures on the RVOT, which will serve our patients well.

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References

Figure 1 Right ventricular outflow tract anatomy in a 5-year-old with severe pulmonary insufficiency after implantation of a bovine jugular venous conduit for pulmonary atresia with ventricular septal defect. The white blood magnetic resonance image (A) shows a widely patent and kinked conduit. The volume-rendered reconstruction from a contrast-enhanced magnetic resonance angiogram (B) depicts the calibre irregularity of the conduit and diffuse hypoplasia of the right pulmonary artery. The pulmonary regurgitant flow volume was 2.73 L/min/m², corresponding to 45% of forward flow.


Perventricular implantation of a right ventricular-to-pulmonary artery ‘conduit’

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A 2.2 kg newborn who underwent a cardiac catheterization at 2 days of life showed pulmonary atresia, ventricular septal defect, and confluent pulmonary arteries of 2.5 mm in diameter and a Nakata of 50 mm/m². Pulmonary arteries were supplied from a major aorto pulmonary collateral from the left subclavian artery (Panel A). Oxygen saturation was around 60%. Surgical options (implantation of a modified Blalock–Taussig shunt or of a right ventricular outflow tract conduit) were considered too risky. A hybrid approach was undertaken under general anaesthesia and oro-tracheal intubation. Patient underwent midline sternotomy. Under direct vision a modified Blalock–Taussig shunt or of a right ventricular outflow tract conduit were considered too risky. A hybrid approach was undertaken under general anaesthesia and oro-tracheal intubation. Patient underwent midline sternotomy. Under direct vision a modified Blalock–Taussig shunt or of a right ventricular outflow tract conduit was undertaken under general anaesthesia and oro-tracheal intubation. Patient underwent midline sternotomy. Under direct vision a modified Blalock–Taussig shunt or of a right ventricular outflow tract conduit was undertaken under general anaesthesia and oro-tracheal intubation. Patient underwent midline sternotomy. Under direct vision a modified Blalock–Taussig shunt or of a right ventricular outflow tract reconstruction (19 mm). Oxygen saturation increased from 60% at the beginning of the procedure to 90% at the end. Total procedural time was 60 min. The post-procedural course was uneventful. At a 3-month follow-up, the stent is patent (Panel H), patient’s weight is 5 kg, and oxygen saturation is 85%.