

Right Heart Percutaneous Valve Therapies

An update on percutaneous valve options for treating right ventricular outflow tract dysfunction in patients with congenital heart disease.

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The use of a surgically placed, valved conduit between the subpulmonary ventricle and the pulmonary artery in patients with congenital heart defects was first reported in the mid 1960s.¹ Such conduits made possible the repair of many complex congenital cardiac lesions involving atresia or hypoplasia of the right ventricular outflow tract (RVOT). Patients with multiple types of congenital cardiac lesions benefited from reparative surgery involving RVOT conduits and, as technical advances in surgery occurred, these procedures were performed in younger (and smaller) patients.²⁻¹¹ Although initial results were exciting, it soon became apparent that conduit failure in the form of progressive valve/conduit stenosis from calcification or intimal proliferation, and/or valvular degeneration causing progressive pulmonary regurgitation (PR), was inevitable for all types of RVOT conduits.¹²⁻²¹ In the growing child, somatic outgrowth of the conduit(s) occurs. Irrespective of the cause of conduit failure, the need for multiple surgical procedures for conduit revisions was to become the rule for these patients. Furthermore, some found that second and subsequent conduits had shorter freedom from failure than the originally placed conduits.¹⁵

Transcatheter therapies, such as balloon valvuloplasty/angioplasty and/or bare-metal stenting, have been instituted to provide palliative relief for conduit stenosis in hopes of extending the functional life span of conduits and have been shown to be somewhat beneficial in prolonging interval(s) between conduit replacement(s).²²⁻²⁵ However, especially in the case of conduit stenting, patients are left with significant PR. Multiple long-term follow-up studies have suggested that chronic volume overload of the RV can lead to impaired systolic and diastolic function of the RV, dilation of the

tricuspid valve annulus leading to significant tricuspid regurgitation, impaired exercise capacity, atrial and ventricular arrhythmias, heart failure, and an increased risk of sudden death.²⁵⁻³⁰ Furthermore, restoration of pulmonary valve competence at an appropriate time has resulted in improvement in RV function, exercise tolerance, and incidence of arrhythmias.^{28,30,31} Until recently, there has been no transcatheter or less-invasive treatment for PR and therefore, these patients have either been managed medically or referred back for repeat surgery.

For nearly 20 years, investigators have attempted to come up with a nonsurgical treatment for failed conduits and valves.³²⁻³⁴ Dr. Philipp Bonhoeffer was the first (in 2000) to perform percutaneous replacement of the pulmonary valve in a right ventricular–pulmonary artery (RV-PA) prosthetic conduit of a human with valve dysfunction.³⁵ Since then, rapid advancements in the development of percutaneous catheter-based therapies for cardiac valve repair have occurred.³⁶⁻⁴¹

Currently, in the United States, there are only two transcatheter heart valves that have been used in the pulmonary position for RVOT dysfunction: the Medtronic Melody transcatheter pulmonary valve (Medtronic, Inc., Minneapolis, MN) and the Edwards Sapien transcatheter heart valve (THV) (Edwards Lifesciences, Irvine, CA).⁴²⁻⁴⁶ The basic concept of both devices involves intravascular stent and bioprosthetic valve technologies.

This article provides an update on percutaneous valves used in the treatment of RVOT conduit dysfunction. It also reviews recommended criteria for use, special procedural considerations for transcatheter valve placement in the RVOT position, and recently published outcomes regarding the use of these valves for RVOT dysfunction.

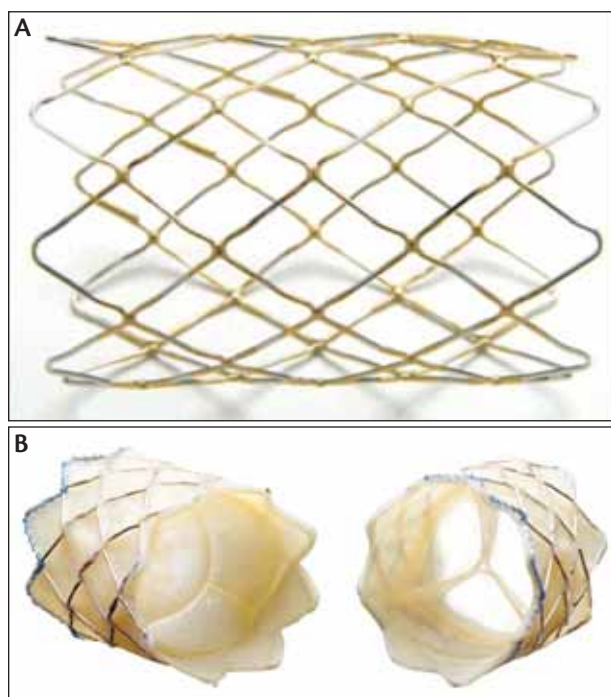


Figure 1. Platinum-iridium stent frame for the Melody valve (A). Melody valve looking from the inflow end on the left (white sutures at inflow) and looking from the outflow end on right (blue sutures at outflow) (B) (Reprinted with permission from Medtronic, Inc).

MEDTRONIC MELODY TRANSCATHETER PULMONARY VALVE

The current Melody valve is a modification of the initial valve used by Dr. Bonhoeffer and consists of a platinum-iridium stent with a valved segment of natural bovine jugular vein.^{34,39} The Melody valve has an initial stent length of 28 mm and an initial diameter of 18 mm (Figure 1A). The venous segment is sutured to the stent around its entire circumference at inflow and outflow and at each stent node using 5-0 polypropylene sutures. The suture is blue at the outflow to signify the outflow end of the valve for appropriate alignment of the valve onto the delivery system and at implant (Figure 1B). The valve can be crimped down to 6 mm for loading and re-expanded up to 22 mm in diameter, depending on which size delivery system is used.

Delivery System

The Melody valve is implanted using the Ensemble catheter delivery system (Medtronic, Inc). The Ensemble system is composed of a balloon-in-balloon deployment catheter, which comes in three different sizes (18, 20, and 22 mm) based on the outer balloon diameter of the balloon-in-balloon catheter onto which the Melody valve is

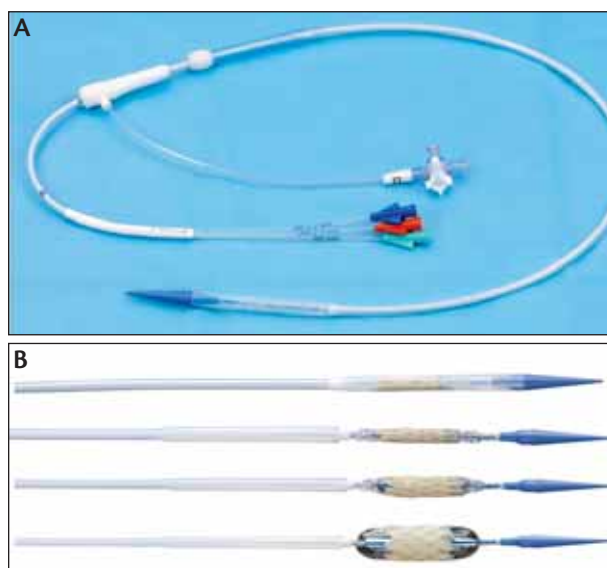


Figure 2. Ensemble transcatheter delivery system (A). Melody valve loaded onto the Ensemble delivery catheter with retractable sheath initially covering the valve for delivery (top) and then retracted for inner balloon inflation followed by outer balloon inflation at deployment. Shown sequentially from top to bottom (B) (Reprinted with permission from Medtronic, Inc).

front-loaded and hand crimped. There is an outer retractable sheath that covers the valve during delivery and is pulled back just before deployment. The sheath has a side arm for flushing or delivering contrast. The entire system has a 22-F crossing profile no matter which outer balloon diameter is used (Figure 2).

Melody Outcomes and Follow-Up Data

Initial outcomes data involving use of the Melody valve in the pulmonary position in human subjects with RVOT dysfunction are from Dr. Bonhoeffer's group in Europe.^{35,36,39,47} In September 2006, the first prospective, nonrandomized, multicenter trial involving the Melody valve was initiated in the United States under an investigational device exemption using a standardized protocol. The objectives of the trial were to determine safety, procedural success, and short-term effectiveness of the Melody valve by assessing 6-month outcomes. Inclusion criteria included patients aged more than 5 years, weight > 30 kg, presence of a surgically placed conduit with an initial implant diameter of > 16 mm, and evidence of conduit dysfunction defined as either: 1) New York Heart Association (NYHA) class II, III, IV with RVOT mean Doppler gradient by echo > 35 mm Hg or moderate-to-severe PR; or 2) NYHA class I with RVOT mean gradient > 40 mm Hg or severe PR with RV dilation and dysfunction (RVFS < 40%) by

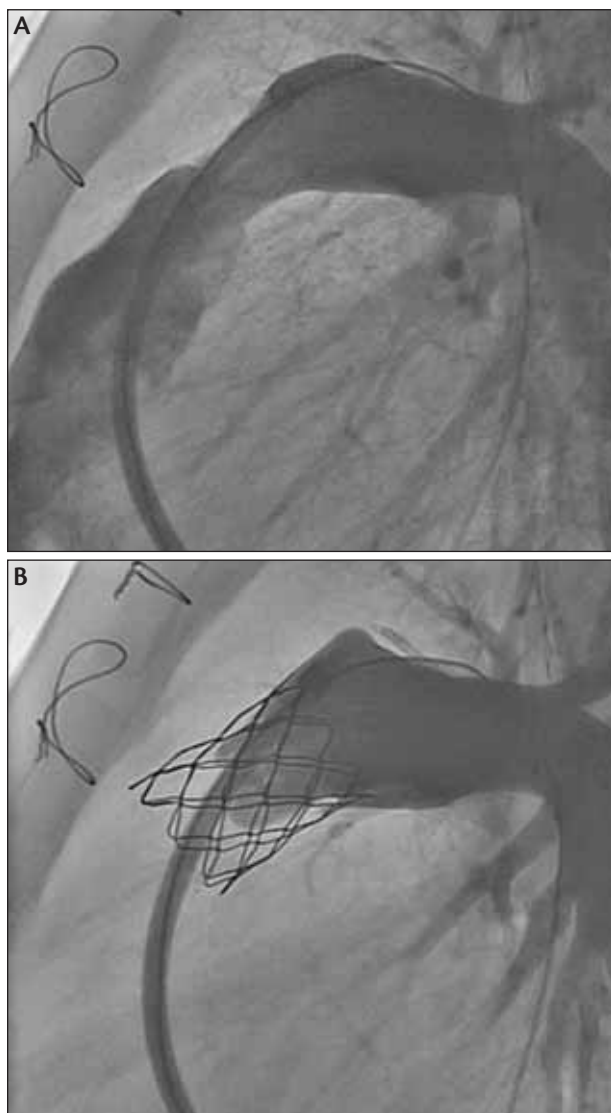


Figure 3. Lateral projection of a PA angiogram demonstrating stenosis and regurgitation of a previously placed RV-PA conduit (A). Follow-up PA angiogram after Melody valve placement demonstrating no PR (B).

echocardiography. The initial short-term results involving the first 34 patients enrolled in the study were reported by Zahn et al.⁴² The short- and medium-term outcomes of 136 patients enrolled through August 2009 were recently published by McElhinney et al.⁴³ In the updated report of these patients by McElhinney et al, the procedural success rate was high at 98%, with only 6% experiencing serious procedural adverse events, which included uncontained conduit rupture in two patients, guidewire-induced pulmonary artery perforation in two patients, and femoral vein thrombosis in one patient. There was one death after implantation that was not device related.

This patient died 20 days after implantation after experiencing complications due to coronary dissection with diagnostic coronary angiography for coronary assessment before valve implantation. Although only 15% of the patients were categorized as NYHA class I before implantation, at every follow-up interval after implantation at least 75% were categorized as NYHA class I, with no patients considered class III or IV. Before implantation, more than 80% of the patients had moderate to severe PR; however, at each follow-up interval out to 2 years, more than 90% of the patients had no/trivial PR and no patients had more than mild PR (Figure 3). Paired preimplantation and 6-month postimplantation cardiac MRI data were consistent with a significant decrease in RV end-diastolic volumes, RV mass, and PR fractions at follow-up. Stent fractures were observed in 26% of the patients, with the majority occurring in patients who did not undergo prestenting of their conduit before valve implantation. Most of these patients had been enrolled early on in the study when concomitant procedures (such as prestenting of the conduit) were not allowed. McElhinney et al did report that 10 of 122 patients (8%) required reintervention during follow-up, with all reinterventions performed due to increased RVOT gradients. All but one of these patients were noted to have stent fractures at the time of reintervention. Initial reintervention was balloon angioplasty in two patients and placement of a second Melody valve in eight patients. Only one patient required surgical conduit replacement at follow-up, and late mortality was zero.

EDWARDS SAPIEN TRANSCATHETER HEART VALVE

The Edwards Sapien (formerly known as the Cribier-Edwards) THV was initially developed as a percutaneous valve option for aortic valve disease and was first successfully placed in a human for calcified aortic stenosis by Cribier.³⁷ In 2005, a previous version of the Sapien valve was used under a compassionate use protocol for percutaneous placement in an obstructed RV-PA homograft of a patient who had previously undergone a Ross procedure.⁴⁴ Most recently, the Edwards Sapien THV was used in the treatment of seven patients with RV-PA conduit failure.⁴⁵ The Sapien valve also combines balloon-expandable stent and bioprosthetic valve technology. The frame is a radiopaque, stainless steel, expandable support structure with an integrated, unidirectional, trileaflet, tissue valve fabricated from three equal sections of bovine pericardium. The valve leaflets are hand-sewn to the stent frame (Figure 4). The valve is available in 23-mm- and 26-mm-diameter sizes, with heights of 14.5 and 16 mm, respectively.⁴⁵

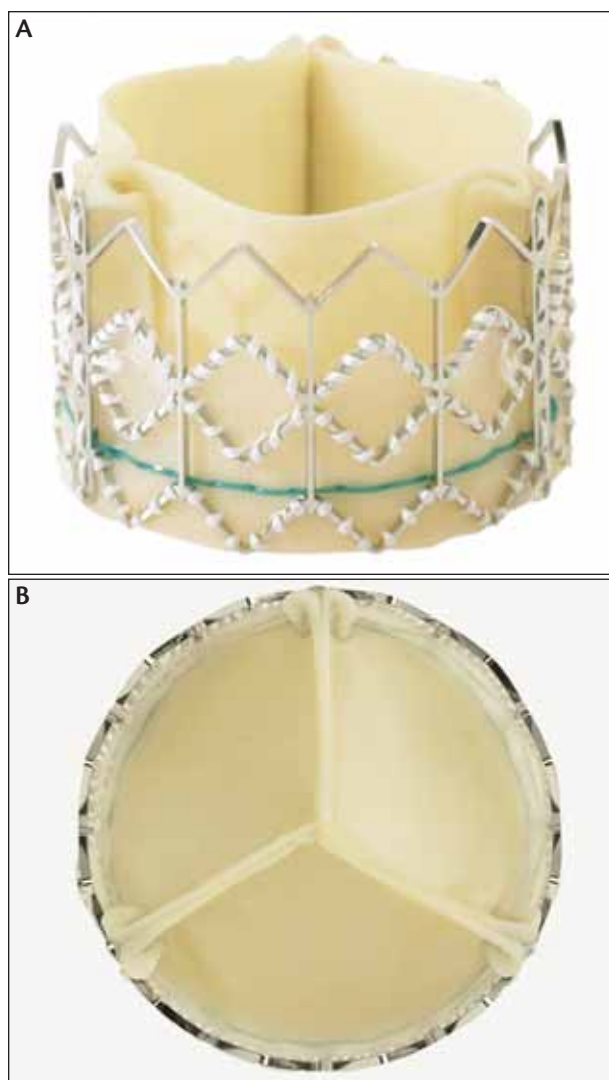


Figure 4. Sapien THV viewed from the side (A). Sapien THV on-end view (B) (Reprinted with permission from Edwards Lifesciences).

Delivery System

The Sapien valve has been delivered via a transfemoral route using the RetroFlex 3 delivery system (Edwards Lifesciences). The RetroFlex 3 system consists of the introducer sheath set, a dilator kit, and the RetroFlex balloon catheters. The delivery system is an articulating “flex” catheter with a handle that provides a rotational grip for articulation of the distal portion of the catheter, a tapered tip at the distal end of the delivery system to facilitate tracking of the Sapien valve into the RVOT, and a balloon for deployment of the valve. This valve requires a specialized crimper that symmetrically reduces the overall diameter of the valve from its expanded size to its collapsed (mounting) size on the delivery balloon catheter.

PROCEDURAL CONSIDERATIONS

Coronary Artery Testing

A few comments should be made regarding the risk of coronary artery compression with stent and/or valve placement in the RVOT position, because this is the most common reason why patients who would otherwise meet criteria for a percutaneous valve do not receive one. As has been discussed in previous studies, it is imperative that assessment for possible coronary compression be performed before placing a stent and/or valve in the RVOT conduit.^{24,42,48,49} In the patient population requiring RVOT conduits, the respective cardiac defects may either have anomalous coronary artery anatomy, such as a single coronary giving rise to significant branches that cross over the front of the heart, and/or the aorta may be in such an anterior or rotated position that even normal origins of the coronaries may still be relatively displaced. Still in other patients, surgery for their congenital cardiac defects may include reimplantation of the coronary arteries, such as in an arterial switch operation or a Ross procedure. The reimplanted coronaries, particularly the left coronary artery, may be at risk of compression during RVOT conduit stenting or valve placement, depending on the postsurgical anatomic relationship between the more anteriorly placed RV-PA conduit and the coronaries. Often, it is not possible to predict the risk of coronary compression with noninvasive testing; therefore, the risk of coronary compression must be evaluated at catheterization before bare-metal stenting and/or valve placement. Initial screening is done by aortic root or selective coronary angiography with a wire or catheter in the RV-PA conduit. If there is any concern of a close anatomic relationship between the conduit and a significant coronary branch, simultaneous inflation of a balloon within the conduit and aortic root and/or selective coronary angiography should be performed (Figure 5). A few important technical considerations include: (1) be sure the appropriate balloon size and inflation pressure is used to best approximate the resultant diameter of the conduit after stent/valve implantation; (2) multiple imaging projections may be necessary to provide the best assessment of the conduit-coronary relationship; (3) remember that selective coronary angiography may mask ostial stenosis or very proximal obstruction during simultaneous conduit balloon dilation; (4) use very dilute contrast to inflate the balloon because a more concentrated mixture may make it difficult to see the course of the coronary arteries behind or underneath the balloon; (5) make sure the widest part of the balloon being used to dilate the conduit is actually positioned over the region or coronary in question; and (6) remember that ringed conduits or bioprosthetic valve rings do not remove the risk of coronary compression.

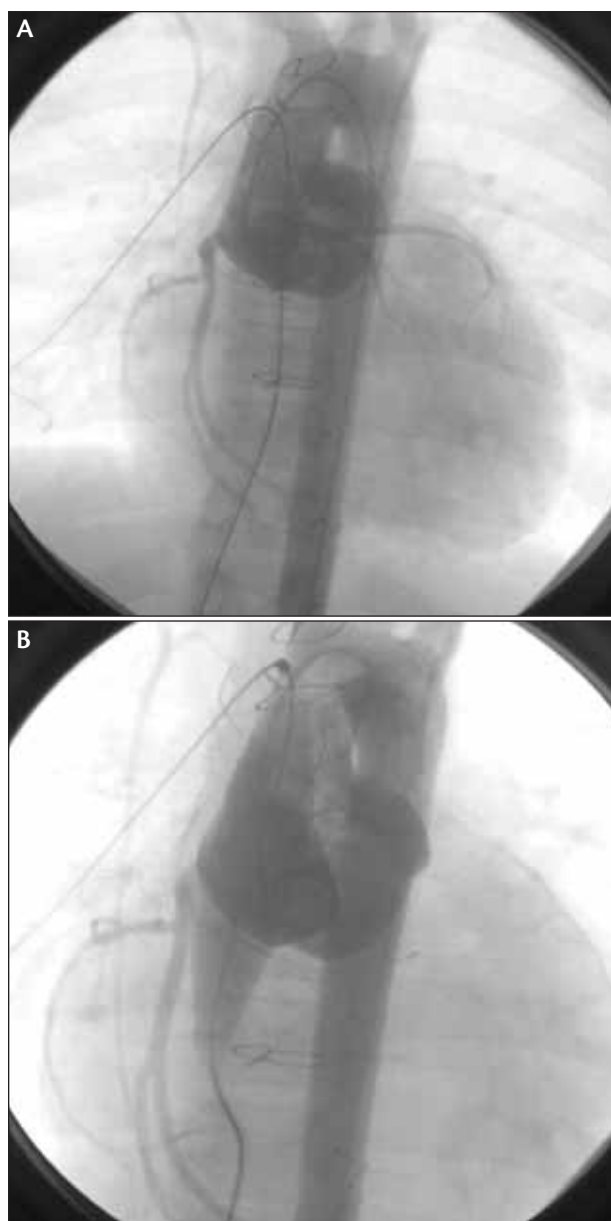


Figure 5. Aortic root angiogram demonstrating filling of the right and left coronary arteries without balloon dilation of the anterior RV-PA conduit (A). Note that the origin of the left coronary artery is fairly rightward (and possibly directly behind the RV-PA conduit). Repeat aortic root angiogram with simultaneous balloon dilation of the conduit demonstrates complete occlusion of the left coronary artery due to compression from the expanded conduit (B).

Conduit Rupture

It is not uncommon for contained conduit tears to occur with balloon dilation for conduit stenosis. Most often these tears are hemodynamically insignificant. In the United States investigational device exemption trial

involving the Melody valve, uncontained conduit ruptures occurred in two patients. In the first instance, the patient was temporarily palliated with a chest tube and then underwent emergent surgical conduit replacement. The second patient was treated under emergent use protocol with a large-diameter, covered stent. Of note, the Melody valve itself functions as a covered stent, with the venous wall segment attached to the stent frame; however, ruptures of the conduit may not be in the same region that optimal valve placement would be and/or the ruptures may be longer than the postimplantation valve length.

Pulmonary Artery Injury

Pulmonary artery injury (perforation) is another serious procedural adverse event noted in the Melody trial. Vessel injury was presumed to have occurred due to wire perforation. Percutaneous valve implants require use of very stiff guidewires and great care should be taken in attaining appropriate and stable wire position before valve deployment to keep the risk of vessel injury to a minimum.

PREPARATION OF CONDUITS FOR VALVE IMPLANT

Despite improved stent technologies over the years, stent fractures are a common complication after implanting intravascular stents into cardiovascular-related positions.^{23,24,50-53} This has been shown to be true for the Melody valve, as well.^{43,47,54,55} The etiology of stent fractures is thought to be secondary to the dynamic implant environment and the intrinsic metal characteristics of the stents. With ongoing implantation experience, the Melody investigators have implemented certain practices to prepare conduits for implantation in the hopes of decreasing the incidence of stent fractures. These include: (1) aggressive predilation of calcified and stenotic conduits using high-pressure balloons and a balloon diameter < 110% of the conduit diameter at implantation; (2) prestenting conduits with bare-metal stent(s) until evidence of recoil of the conduit during balloon deflation is gone; and (3) attempting to implant the valve in a position that is not directly under the sternum, when possible. Preliminary evaluation of the Melody data suggests that when these techniques are implemented, the incidence of stent fractures at follow-up is significantly decreased, with 35% of fractures occurring in conduits not prestented compared to only 7% of fractures occurring in prestented conduits. When valve stent fracture occurs and reintervention is indicated, placement of bare-metal stent(s) within the initially placed valve with subsequent placement of a second valve has been shown to be successful.^{43,47}

DEVICE AVAILABILITY IN THE UNITED STATES

On January 25, 2010, the United States Food and Drug Administration formally approved the Melody valve for placement in dysfunctional RVOT conduits under a Humanitarian Device Exemption, making it the first transcatheter heart valve to receive United States Food and Drug Administration approval. Enrollment into the United States feasibility trial involving use of the Edwards Sapien THV in the pulmonic position was completed in May 2010. Plans for a pivotal study are in progress.

CONCLUSION

Percutaneous valve therapies for RVOT conduit dysfunction have been shown to be feasible, safe, and effective, with high procedural success rates and low adverse event rates. Procedural considerations, such as careful assessment for risk of coronary compression, cautious use of stiff guidewires, and awareness of the potential for serious adverse events such as conduit rupture, as well as knowing how to prevent and manage such events, will lead to safe and successful percutaneous valve implantations. ■

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1. Ross DN, Somerville J. Correction of pulmonary atresia with a homograft aortic valve. *Lancet*. 1966;2:1446-1447.
2. McGoon DC, Rastelli GC, Ongley PA. An operation for the correction of truncus arteriosus. *JAMA*. 1968;205:69-73.
3. Rastelli GC, McGoon DC, Wallace RB. Anatomic correction of transposition of the great

- arteries with ventricular septal defect and subpulmonary stenosis. *J Thorac Cardiovasc Surg*. 1969;58:545-552.
4. Rastelli GC, Wallace RB, Ongley PA. Complete repair of transposition of the great arteries with pulmonary stenosis. A review and report of a case corrected by using a new surgical technique. *Circulation*. 1969;39:83-95.
5. Bowman RO, Hancock WD, Malm JR. A valve containing Dacron prosthesis: its use in restoring pulmonary artery-right ventricular continuity. *Arch Surg*. 1973;107:724-728.
6. Ross DN. Homograft replacement of the aortic valve. *Lancet*. 1962;2:487.
7. Ross DN. Replacement of the aortic and mitral valves with a pulmonary autograft. *Lancet*. 1967;2:956-958.
8. Brown JW, Ruzmetov M, Rodefeld M, et al. Valved bovine jugular vein conduits for right ventricular outflow tract reconstruction in children: an attractive alternative to pulmonary homograft. *Ann Thorac Surg*. 2006;82:909-916.
9. Reddy VM, McElhinney DB, Hanley FL. The Ross procedure in children. *Isr J Med Sci*. 1996;32:888-891.
10. Bove EL, Lupinetti FM, Pridjian AK, et al. Results of a policy of primary repair of truncus arteriosus in the neonate. *J Thorac Cardiovasc Surg*. 1993;105:1057-1065.
11. Perron J, Moran AM, Gauvreau K, et al. Valved homograft conduit repair of the right heart in early infancy. *Ann Thorac Surg*. 1999;68:542-548.
12. Clarke D, Campbell D, Hayward A, Bishop DA. Degeneration of aortic valve allografts in young recipients. *J Thorac Cardiovasc Surg*. 1993;105:934-942.
13. Bando K, Danielson GK, Schaff HV, et al. Outcome of pulmonary and aortic homografts for right ventricular outflow tract reconstruction. *J Thorac Cardiovasc Surg*. 1995;109:509-517.
14. Baskett R, Ross D, Nanton M, Murphy DA. Factors in the early failure of cryopreserved homograft pulmonary valves in children: preserved immunogenicity? *J Thorac Cardiovasc Surg*. 1996;112:1170-1179.
15. Stark J, Bull C, Stajevic M, et al. Fate of subpulmonary homograft conduits: determinants of late homograft failure. *J Thorac Cardiovasc Surg*. 1998;115:506-516.
16. Tweddell JS, Pelech AN, Frommelt PC, et al. Factors affecting longevity of homograft valves used in right ventricular outflow tract reconstruction for congenital heart disease. *Circulation*. 2000;102(19 suppl 3):III130-III135.
17. Forbess JM, Shah AS, St. Louis JD, et al. Cryopreserved homografts in the pulmonary position: determinants of durability. *Ann Thorac Surg*. 2001;71:54-60.
18. Wells WJ, Arroyo H Jr, Bremner RM, et al. Homograft conduit failure in infants is not due to somatic outgrowth. *J Thorac Cardiovasc Surg*. 2002;124:88-96.
19. Baskett RJ, Nanton MA, Warren AE, Ross DB. Human leukocyte antigen-DR and ABO mismatch are associated with accelerated homograft valve failure in children: implications for therapeutic interventions. *J Thorac Cardiovasc Surg*. 2003;126:232-239.
20. Oosterhof T, Meijboom FJ, Blieden HW, et al. Long-term follow-up of homograft function after pulmonary valve replacement in patients with tetralogy of Fallot. *Eur Heart J*. 2006;27:1478-1484.
21. Sierra J, Christenson JT, Lahlaide NH, et al. Right ventricular outflow tract reconstruction: what conduit to use? Homograft or Contegra? *Ann Thorac Surg*. 2007;84:606-611.
22. Sreeram N, Hutter P, Silove E. Sustained high pressure double balloon angioplasty of calcified conduits. *Heart*. 1999;81:162-165.
23. Powell AJ, Lock JE, Keane JF, Perry SB. Prolongation of RV-PA conduit life span by percutaneous stent implantation: intermediate-term results. *Circulation*. 1995;92:3282-3288.
24. Peng LF, McElhinney DB, Nugent AW, et al. Endovascular stenting of obstructed right ventricle-to-pulmonary artery conduits: a 15-year experience. *Circulation*. 2006;113:2598-2605.
25. Aggarwal S, Garekar S, Forbes TJ, Turner DR. Is stent placement effective for palliation of right ventricle to pulmonary artery conduit stenosis? *J Am Coll Cardiol*. 2007;49:480-484.
26. Jones EL, Conti CR, Neill CA, et al. Long-term evaluation of tetralogy patients with pulmonary valvular insufficiency resulting from outflow-patch correction across the pulmonic annulus. *Circulation*. 1973;48:11-18.
27. Bove EL, Byrum CJ, Thomas FD, et al. The influence of pulmonary insufficiency on ventricular function following repair of tetralogy of Fallot: evaluation using radionuclide ventriculography. *J Thorac Cardiovasc Surg*. 1983;85:691-696.
28. Therrien J, Siu SC, McLaughlin PR, et al. Pulmonary valve replacement in adults late after repair of tetralogy of Fallot: are we operating too late? *J Am Coll Cardiol*. 2000;36:1670-1675.
29. Oosterhof T, Meijboom FJ, Vliegen HW, et al. Long-term follow-up of homograft function after pulmonary valve replacement in patients with tetralogy of Fallot. *Eur Heart J*. 2006;27:1478-1484.
30. Therrien J, Provost Y, Merchant N, et al. Optimal timing for pulmonary valve replacement in adults after tetralogy of Fallot repair. *Am J Cardiol*. 2005;95:779-782.

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31. Eyskens B, Reybrouck T, Bogaert J, et al. Homograft insertion for pulmonary regurgitation after repair of tetralogy of Fallot improves cardiorespiratory exercise performance. *Am J Cardiol.* 2000;85:221-225.
32. Andersen HR, Knudsen LL, Hasenkam JM. Transluminal implantation of artificial heart valves. Description of a new expandable aortic valve and initial results with implantation by catheter technique in closed chest pigs. *Eur Heart J.* 1992;13:704-708.
33. Pavcnik D, Wright KC, Wallace S. Development and initial experimental evaluation of a prosthetic aortic valve for transcatheter placement: work in progress. *Radiology.* 1992;183:151-154.
34. Bonhoeffer P, Boudjemline Y, Saliba Z, et al. Transcatheter implantation of a bovine valve in pulmonary position: a lamb study. *Circulation.* 2000;102:813-816.
35. Bonhoeffer P, Boudjemline Y, Saliba Z, et al. Percutaneous replacement of pulmonary valve in a right-ventricle to pulmonary-artery prosthetic conduit with valve dysfunction. *Lancet.* 2000;356:1403-1405.
36. Bonhoeffer P, Boudjemline Y, Qureshi SA, et al. Percutaneous insertion of the pulmonary valve. *J Am Coll Cardiol.* 2002;39:1664-1669.
37. Cribier A, Eltchaninoff H, Bash A, et al. Percutaneous transcatheter implantation of an aortic valve prosthesis for calcific aortic stenosis: first human case description. *Circulation.* 2002;106:3006-3008.
38. Eltchaninoff H, Tron C, Cribier A. Percutaneous implantation of aortic valve prosthesis in patients with calcific aortic stenosis: technical aspects. *J Interv Cardiol.* 2003;16:515-521.
39. Khambadkone S, Coats L, Taylor A, et al. Percutaneous pulmonary valve implantation in humans: results in 59 consecutive patients. *Circulation.* 2005;112:1189-1197.
40. Grube E, Laborde JC, Zickmann B, et al. First report on a human percutaneous transluminal implantation of a self-expanding valve prosthesis for interventional treatment of aortic valve stenosis. *Catheter Cardiovasc Interv.* 2005;66:465-469.
41. Feldman T, Leon MB. Prospects for percutaneous valve therapies. *Circulation.* 2007;116:2866-2877.
42. Zahn EM, Hellenbrand WE, Lock JE, McElhinney DB. Implantation of the Melody transcatheter pulmonary valve in patients with a dysfunctional right ventricular outflow tract conduit: early results from the U.S. clinical trial. *J Am Coll Cardiol.* 2009;54:1722-1729.
43. McElhinney DB, Hellenbrand WE, Zahn EM, et al. Short- and medium-term outcomes after transcatheter pulmonary valve placement in the expanded multicenter US Melody valve trial. *Circulation.* 2010;122:507-516.
44. Garay F, Webb J, Hijazi ZM. Percutaneous replacement of pulmonary valve using the Edwards-Cribier percutaneous heart valve: first report in a human patient. *Catheter Cardiovasc Interv.* 2006;67:659-662.
45. Boone RH, Webb JG, Horlick E, et al. Transcatheter pulmonary valve implantation using the Edwards SAPIEN transcatheter heart valve. *Catheter Cardiovasc Interv.* 2010;75:286-294.
46. Bertels RA, Blom NA, Schaliij MJ. Edwards SAPIEN transcatheter heart valve in native pulmonary valve position. *Heart.* 2010;96:661.
47. Lurz P, Coats L, Khambadkone S, et al. Percutaneous pulmonary valve implantation: impact of evolving technology and learning curve on clinical outcome. *Circulation.* 2008;117:1964-1972.
48. Maheshwari S, Bruckheimer E, Hehgme RA, et al. Single coronary artery complicating stent implantation for homograft stenosis in tetralogy of Fallot. *Cathet Cardiovasc Diagn.* 1997;42:405-407.
49. Perret X, Bonvini RF, Aggoun Y, et al. Aberrant right coronary artery occlusion during the percutaneous pulmonary trunk stenting in a patient with tetralogy of Fallot. *Heart Vessels.* 2008;23:140-143.
50. Breinholt JP, Nugent AW, Law MA, et al. Stent fractures in congenital heart disease. *Catheter Cardiovasc Interv.* 2008;72:977-982.
51. Knirsch W, Haas NA, Lewin MA, Uhlemann F. Longitudinal stent fracture 11 months after implantation in the left pulmonary artery and successful management by a stent-in-stent maneuver. *Catheter Cardiovasc Interv.* 2003;58:116-118.
52. O'Laughlin MP, Slack MC, Giffka RG, et al. Implantation and intermediate-term follow-up of stents in congenital heart disease. *Circulation.* 1993;88:605-614.
53. Law MA, Shamszad P, Nugent AW, et al. Pulmonary artery stents: long-term follow-up. *Catheter Cardiovasc Interv.* 2010;75:757-764.
54. Nordmeyer J, Khambadkone S, Coats L, et al. Risk stratification, systematic classification, and anticipatory management strategies for stent fracture after percutaneous pulmonary valve implantation. *Circulation.* 2007;115:1392-1397.
55. Schievano S, Taylor AM, Capelli C, et al. Patient specific finite element analysis results in more accurate prediction of stent fractures: application to percutaneous pulmonary valve implantation. *J Biomech.* 2010;43:687-693.