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CARDIOLOGY (WW LAI, SECTION EDITOR)

Transcatheter Pulmonary Valve Replacement: A Current Review

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Abstract Since first reported in 2000, transcatheter (percutaneous) pulmonary valve (TPV) replacement has become an important tool for the management postoperative right ventricular outflow tract (RVOT) dysfunction in patients with congenital heart disease, either as alternative or adjunct to surgery. Implantation of a pulmonary valve for treatment of RVOT obstruction or pulmonary regurgitation without performing open-heart surgery offers obvious appeal, and short-term results from multiple institutions throughout the world support the effectiveness and safety of this therapy. At present, there are two TPV prostheses available in the U.S.: the Medtronic Melody® valve is available commercially, and the Edwards Sapien® valve is available at limited centers as part of an investigational protocol. Although TPV therapy is likely to have a major impact on the management of postoperative RVOT dysfunction in patients with congenital heart disease or a Ross procedure, the technology is young and there is much that remains to be learned.

Keywords Percutaneous valve · Tetralogy of Fallot · Conduit · Melody valve · Bovine jugular vein · Pulmonary stenosis

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Introduction

Clinical Context

Children with congenital cardiovascular anomalies that affect the right ventricular outflow tract (RVOT), including tetralogy of Fallot, truncus arteriosus, and other conotruncal defects, typically undergo surgical repair early in life. As part of this repair, the RVOT is usually reconstructed, either by augmenting the outflow tract with a patch or inserting a prosthetic conduit or valve to connect the RV to the pulmonary arteries (PAs). Patients who undergo a Ross procedure (pulmonary autograft aortic valve replacement) also undergo RVOT reconstruction with a prosthetic conduit or valve. All of the commonly used RVOT conduits and valves are subject to degeneration and dysfunction over time. Depending on the method of RVOT repair, the patient may develop progressively severe pulmonary regurgitation (PR) or RVOT obstruction, which impose RV volume and pressure loads, respectively. Although PR and RVOT obstruction can be tolerated for extended periods, they often lead to functional limitations and are detrimental to the RV.

Until recently, treatment of RVOT dysfunction almost always required open-heart surgery and implantation of a prosthetic pulmonary valve or valved conduit. Because surgery is associated with morbidity and a risk of adverse outcome, and because replacement pulmonary valve prostheses/conduits will inevitably deteriorate, RVOT dysfunction is often tolerated for many years before the patient is referred for pulmonary valve replacement. Treating RVOT dysfunction as soon as it appears in these patients would ultimately result in their undergoing multiple open-heart surgeries over the course of a lifetime. On the other hand, the risk of such delay in treatment is

progressive and cumulative RV loading that may result in myocardial damage and consequent mechanical and/or electrical dysfunction. However, due to a limited understanding of the balance among risks and benefits, and considerable variability among patients, it is difficult to determine the best course at any given point in a specific patient. In this clinical context, a less invasive approach to the treatment of RVOT dysfunction would be of obvious appeal.

Brief History and Regulatory Status of Transcatheter Pulmonary Valve Replacement

Transcatheter pulmonary valve (TPV) replacement for the treatment of postoperative RVOT obstruction and/or PR is a relatively recent but increasingly important treatment option for patients with complex congenital heart disease or other conditions requiring surgical RVOT reconstruction. There are currently two TPV systems in commercial or investigational use: the Melody® TPV (Medtronic Inc., Minneapolis, MN) and the Sapien® transcatheter heart valve (Edwards Lifesciences LLC, Irvine, CA). The Melody® valve was designed specifically for TPV replacement, whereas the Sapien® valve was initially developed for transcatheter aortic valve replacement and subsequently applied to the pulmonary circulation.

TPV replacement was first reported by Bonhoeffer et al. [1] in a lamb model in 2000, and then later that year in a human patient [2], using the first generation of the Melody® TPV. In 2007, a prospective nonrandomized investigational device exemption (IDE) trial began at three centers in the U.S., with the first Melody® valve implant in the U.S. performed in January of that year [3]. After the addition of two more investigative sites and several protocol amendments that increased the total number of implants allowed, trial enrollment was completed and the 150th implant was performed in January 2010.

The same month, the Melody® valve was officially approved in the U.S. under a Humanitarian Device Exemption (HDE; http://www.accessdata.fda.gov/cdrh_docs/pdf8/H080002a.pdf). The first commercially approved Melody® valve implant in the U.S. was performed in February 2010.

In the U.S., the Sapien® valve is approved for transcatheter aortic valve replacement in high- and extreme-risk patients, but is currently available for TPV replacement only under investigational use, as part of an IDE trial that began in 2008. It is not specifically approved for TPV replacement, and there is limited published information about Sapien® valve therapy for RVOT dysfunction [4], [5•, 6]. Thus, this review will focus primarily on literature about the Melody® valve, with a separate section on the Sapien® valve later.

Indications for TPV Replacement

The instructions for use under the HDE approval for the Melody® valve, which closely conform to the entry criteria for the IDE trial, specify that it is indicated for use as an adjunct to surgery in the management of pediatric and adult patients with the following clinical conditions:

- Existence of a full (circumferential) RVOT conduit that was equal to or greater than 16 mm in diameter when originally implanted, and
- Dysfunctional RVOT conduit with a clinical indication for intervention and either:
  - Regurgitation: ≥ moderate regurgitation, or
  - Stenosis: mean RVOT gradient ≥35 mmHg

Although the Melody® valve is specifically approved only under these conditions, it has been used in other forms of anatomically appropriate postoperative RVOT anatomy and in patients who do not meet these criteria. The instructions for use do not specify a maximum RVOT dimension at the time of TPV replacement, but the delivery system for the Melody® valve comes in 18-, 20-, and 22-mm-diameter sizes, with an outer diameter of the valve about 1.4 mm larger than that. Thus, patients with an RVOT that is larger than 22 mm are generally not candidates for TPV replacement using the Melody® valve and standard delivery methods. However, investigators have also reported innovative approaches to using the Melody® valve in patients with RVOT dysfunction and anatomic or hemodynamic circumstances that are not straightforward and/or do not conform to the instructions for use (including larger RVOT anatomy), even to the point of implanting the TPV in other cardiac valves. Such "off-label" applications are discussed in greater detail later in this review.

There is no age or size specification for percutaneous Melody® valve implant in the instructions for use. The IDE set a lower weight limit of 30 kg [3], but the valve has been implanted in much smaller children. In reported series, patient age and weight range greatly, speaking to the flexibility of this technology. The underlying cardiac diagnosis is not particularly important. In larger series, the most common diagnoses are tetralogy of Fallot and aortic valve disease treated with a Ross procedure.

Outcomes of TPV Replacement

Procedural Outcomes

Reports from single and multicenter studies in Europe, Canada, and the U.S. have consistently found that, in

properly selected patients, the Melody® valve can be implanted in the intended location in the RVOT with few serious procedural complications [3, 7–11]. Serious implant-related or other procedural complications are uncommon but have been reported, including rupture of the conduit with bleeding into the chest, malposition or embolism of the valve, jailing and consequent occlusion of a PA branch, compression of a coronary artery located adjacent to the conduit, and wire-related injury to a PA [3, 7–12]. Most of these complications can be avoided with careful technique and attention, following the recommended instructions for use. However, factors that may predispose to conduit rupture have not been defined, and it has not been determined whether there are reliable measures that can prevent or reduce the likelihood of this rare complication.

Approximately 4–5 % of patients undergoing catheterization for attempted TPV replacement will have a coronary artery that is at risk for compression if the conduit is stented [8–12]. This potentially catastrophic complication is sufficiently common and serious [13, 14] that every patient who goes to the catheterization lab to be evaluated for TPV replacement must have the coronary arterial anatomy defined and assessed for potential compression by the conduit if a TPV is implanted. This relationship can be assessed in reversible fashion by performing simultaneous coronary angiography and balloon angioplasty of the conduit. The distance between the conduit and coronary artery alone is not sufficient to predict compression, which is a function both of the anatomic relationship between the conduit and the coronary, and of how the conduit and any adjacent tissue/material are displaced when the conduit is expanded.

Taggart et al. [15] recently reported a patient who underwent TPV implant for conduit stenosis after a Ross procedure, and was observed acutely to have unexplained elevation in PA pressure. Subsequent evaluations demonstrated LV dilation and elevated LV filling pressures, pleural effusions, and symptoms and signs of congestion. Ultimately, they diagnosed a communication between the ascending aorta and PA, and reported that the patient had experienced erosion of the TPV into the aorta [16]. It seems likely that, rather than an erosion that occurred over time, an acute aortopulmonary window was created during the TPV implant procedure, which would explain the acute and ongoing symptoms. Traumatic aortopulmonary window has been reported after RVOT dilation in other postoperative patients who had both PA and aortic transection and reanastomosis [17, 18], as was the case in this patient, who had undergone a Ross procedure. Whether the reported complication was a traumatic fistula or an erosion, this case is a reminder that all potential complications of TPV implant have not been defined, and illustrates one of the reasons that it is important to measure PA hemodynamics after TPV replacement and be aware of the various potential causes.

Pre-stenting the conduit (i.e., placing one or more bare metal stents) before Melody® valve implant became a common practice after it was widely recognized that stent fracture can limit the effectiveness of TPV therapy [10, 11, 19, 20•, 21, 22]. Although stents in the conduit can potentially complicate advancement of the valve through the RVOT, we are not aware of any serious complications specifically related to the practice of pre-stenting.

Hemodynamic and Cardiac Functional Outcomes

Not surprisingly, given the range of physiologic abnormalities among treated patients, from pure RVOT obstruction to pure PR, with mixed obstruction and PR common, the specific functional and physiologic outcomes of TPV replacement have been found to vary.

RVOT Obstruction

The published studies on TPV replacement have clearly and consistently demonstrated that the Melody® valve provides effective acute relief of RVOT obstruction, although in many cases the obstruction is not eliminated completely (Table 1) [8–11, 23]. Residual gradients in the catheterization lab are often in the 10–20 mmHg range, and are sometimes higher, and mean Doppler gradients tend to range from 15–25 mmHg; higher residual gradients are more common in patients who had important conduit stenosis prior to TPV implant than those with primary PR [8, 9]. In patients who do not develop major stent fractures, the degree of RVOT obstruction appears to remain stable for at least 2–3 years after TPV implant, beyond which adequate data are not yet available [8, 20•]. Progressive obstruction without associated stent fracture has been described, the mechanism of which is not clear [10].

A higher post-implant RVOT gradient is one of the factors that has been associated with shorter freedom from RVOT reintervention [8, 20•], and the vast majority of reinterventions after TPV replacement have been for recurrent RVOT obstruction (see below). Data about the impact of pre-stenting on acute relief of RVOT obstruction are inconclusive [20•, 23]. In addition to those studies, Carr et al., in a study of bare metal stenting in small diameter RVOT conduits, showed that stent implantation reduces gradients beyond what is achieved with balloon angioplasty alone, suggesting that pre-stenting may have improve the hemodynamic outcome when compared with TPV implant alone [24]. However, Lurz et al. did not find any incremental reduction in the RVOT gradient when patients who underwent pre-stenting subsequently had a Melody® valve implanted [25]. These findings suggest that efforts to optimize relief of RVOT obstruction before Melody® valve implant (i.e., with pre-dilation and pre-

Table 1 Summary data from published series of TPV replacement using the Melody® valve

Author	No. of patients	Age (years)	Follow-up duration	Peak RVOT gradient (mmHg)		Mild or greater PR by echo		
				Pre implant	Post implant	Pre implant	Post implant	RVOT Reintervention (no. of patients)
Boshoff et al. [43]	23	16.9 ± 9.7	1.2 ± 1.2 years	24.2 ± 9.6	7.5 ± 4.3	23	1	0
Butera et al. [11]	63	24 (11–65)	2.5 years (14)	45 (35–75)	10 (0–30)	42	4	7
Demkow et al. [22]	10	26.8 ± 4.0	6 months	80.6 ± 22.7	38.8 ± 10.4	6	1	0
Eicken et al. [10]	102	21.5 (16.2–30)	352 days (99–390)	37 (29-46)	14 (9–17)	[a]	[a]	10
Gillespie et al. [40]	104	26 (3–63)	12 months (1–46)	38.7 ± 16.3	10.9 ± 3.7	101	0	2
Lurz et al. [8]	155	21.2 (7–71)	28.4 months (0–83.7)	37.2 ± 20	17 ± 10	99	2	45
Martins et al. [21]	7	9-32	7.8 months (2.8–10.1)	65 ± 28	11 ± 4	7	0	0
McElhinney et al. [9]	136	19 (7–53)	6 months (0–30)	35.6 ± 15.8	14.4 ± 5.7	110	9	11
Vezmar et al. [31]	28	14.9 (10.9–19)	27.6 months (0–37)	36 ± 15	12 ± 7	19	0	6

Data presented as mean ± standard deviation, median (minimum − maximum), or frequency. For centers/trials reported in multiple articles, only the most recent or complete report was included in this table

[a] Echocardiographic data not reported

stenting) may be important means of ensuring that TPV therapy yields the most thorough and lasting benefit.

Pulmonary Regurgitation and Ventricular Volumes

Patients who undergo TPV replacement generally have some degree of PR, and most have moderate or severe leakage. TPV replacement eliminates PR in essentially all patients, and significant paravalvular leak/regurgitation is rare. The longest follow-up data from both European and American studies demonstrated sustained pulmonary valve competence in almost all patients for 3 or more years. We are not aware of any reports in which progression of PR has been cited as a cause of TPV failure or reintervention.

The natural corollary of reduced PR is a decrease in RV volume. Multiple studies have reported acute or early reduction of RV end-diastolic volume after TPV implant [3, 7, 9, 10, 22, 26–28]. The magnitude of reduction varies according to the distribution of patient with significant PR and RVOT obstruction, but tends to range from 15 to 25 mL/m^2 on average. Absolute and effective RV stroke volumes also tend to increase, as does LV end-diastolic volume, presumably due to increased forward flow through the pulmonary circulation and possibly altered ventriculo-ventricular interaction [7, 25, 26, 29].

Ventricular Function

Similar to studies of surgical pulmonary valve replacement, TPV-related elimination of PR and reduction in RV volume are not always accompanied by improved RV function, although some patients demonstrate substantial improvement. Data on changes in RV ejection fraction after TPV replacement are mixed, with some studies finding improvement in the acute or short term [22, 26, 27, 30], and others showing no change [7, 9, 10, 31]. Differences according to baseline physiology appear to be important in considering the RV functional response to TPV replacement. For example, in several focused studies, Bonhoeffer's group observed RV ejection fraction to improve in patients with primary RVOT obstruction but not in those with primary PR [29, 30]. Based on limited data, it appears that there is no substantial improvement after any acute or short-term change [27, 28]. In other small studies, investigators also observed improvement in more subtle measures of RV systolic function, including septal and RV free wall strain [30, 32]. There is limited information on RV diastolic function; Romeih et al. [27] found no early change in diastolic functional indices after TPV replacement but did observe improvement in RV filling at 12 months.

One of the important physiologic consequences of RV dilation and dysfunction in some patients is secondary LV dysfunction. Thus, the impact of TPV replacement on LV systolic and diastolic function is also of interest. As with RV function, the effect of TPV replacement on the LV may vary according to baseline physiology. Improved systolic function, as based on ejection fraction and myocardial velocity imaging, has been reported in several small studies [29, 30]. Improvement in some measures of LV diastolic function has also been reported [26, 33]. Regardless of discrete functional improvement, there is convincing

evidence that LV and RV mechanical efficiency improve, with augmentation of absolute and effective stroke volumes after a Melody® valve is implanted [25, 26, 28–31]. Along with these findings, there are isolated reports describing more efficient ventriculo-ventricular interaction, including a decreased RV-LV mechanical delay [33], reduction in the QRS duration in patients with primary PR [34], and decreased reperfusion heterogeneity in patients with both obstructive and regurgitant physiology [34].

Functional Outcomes

Symptomatic status, most often New York Heart Association (NYHA) classification, has been one of the fundamental outcome measures included in series of TPV replacement. Without exception, studies in which change in symptomatic status has been reported have documented a significant improvement in NYHA class among the cohort, which has largely been maintained for the duration of follow-up [7, 9, 11, 22, 28, 35]. Most patients are in NYHA class II or higher before treatment, but a subset are in NYHA class I. In recognition of the fact that symptoms are not necessarily a prerequisite for intervention in patients with RVOT obstruction or PR, the U.S. IDE trial was designed with more stringent hemodynamic inclusion criteria for patients in NYHA class I than those with symptoms [3]. Although symptomatic improvement beyond NYHA classification has not been systematically reported in patients undergoing TPV replacement, it has been our observation that even patients who were in NYHA class I prior to intervention frequently report feeling better and/or demonstrate improved exercise tolerance after intervention.

Exercise cardiopulmonary function is frequently abnormal in patients with complex congenital heart disease, due to a variety of factors, and is often figured into decision-making about intervention in patients with RVOT dysfunction. Peak oxygen consumption and other metabolic parameters have been evaluated in a number of studies of patients undergoing TPV replacement. [28–30, 36]. In general, pre-intervention measures of exercise cardiopulmonary function vary considerably in these studies, and there is usually not a significant change overall in the short-term after TPV replacement. However, because of the variable pre-intervention physiology (i.e., some patients with primary RVOT obstruction, some with primary PR), general assessment of TPV replacement cohorts may not reveal important differences related to pre-intervention physiologic parameters. For example, several studies found that various measures of cardiopulmonary function—including peak oxygen consumption, ventilatory efficiency, and anaerobic oxygen consumption—improve after TPV replacement in patients with primary or significant RVOT obstruction, but not in patients with primary PR [26, 28–30].

Quality of life measures are an important means of assessing the functional impact of therapy in many areas. To our knowledge, no systematic studies of quality of life have been reported among patients undergoing TPV replacement.

TPV Durability and Longevity

In early experience, both in Europe and in the U.S. IDE trial, the most concerning performance issue was fracture of the Melody® valve stent frame [19, 20•]. In some cases, there was fracture of one or more stent struts with no apparent functional compromise, but in others, there was clear loss of stent integrity and recurrent RVOT obstruction related to the stent fracture. It appears that this problem has been reduced by the practice of pre-stenting, namely, placement of one or more non-valved balloon expandable stents at the site of obstruction, then implanting the Melody® valve into the stent-fortified conduit [19, 20•]. Based on recent studies, pre-stenting has become a common element of the Melody® valve implant procedure. Because stent fracture is a time-dependent phenomenon, it will take some time to determine whether pre-stenting alleviates, or simply delays, fracture of the Melody® valve stent.

Functional failure of the Melody® valve has been uncommon, and has occurred almost exclusively as recurrent RVOT obstruction in the context of a fractured stent. Significant PR of the Melody® valve has been rare, and in this respect, the performance of the Melody® valve has probably exceeded expectations. Accordingly, almost all reinterventions on the RVOT after Melody® valve implant have been for obstruction related to stent fracture, although there are several reports of explant for obstruction without stent fracture [10] and TPV-related endocarditis [10, 11, 37]. One of the appealing features of TPV replacement is that, size permitting, failure of a TPV can often be treated by implanting a second valve concentrically within the first, with the potential for successive Melody® valve-in-Melody® valve therapy limited primarily by the size to which the RVOT can be expanded. This approach, which has been the primary mode of re-intervention reported thus far, appears to be effective and does not seem to differ substantially from primary implants in terms of safety and technical outcome [20•, 38]. Relatively few surgical RVOT interventions have been reported after TPV implant, and there are few data specifically about such cases. However, there has not been any suggestion that removal of a conduit that contains a TPV is more complicated than one without a TPV.

Safety

Serious acute complications, as discussed in the section on procedural outcomes, include conduit rupture, coronary

artery compression, malposition of the TPV, jailing of or injury to a branch PA, and other catheterization-related events. The most common problems that have been observed during follow-up after Melody valve implant have been stent fracture and recurrent RVOT obstruction [8, 19, 20•]. Cases of endocarditis, a known complication of prosthetic and bioprosthetic valves, have been reported, but there is not enough information about this outcome yet to determine whether the extent of the risk differs from that after surgical or other transcatheter RVOT valve implants [10, 11, 37]. Otherwise, there have been no common or significant safety issues remote from the implant. Computed tomography pulmonary angiography was performed at baseline and at 6 months in the first cohort of patients implanted as part of the IDE trial, specifically to evaluate for pulmonary thromboembolism, and no evidence of this complication was found [3].

TPV Replacement Using the Sapien® Valve

The balloon-expandable Sapien® transcatheter heart valve was designed for implantation into the aortic position in adults with acquired calcific aortic stenosis. The device is available in diameters of 23, 26, and in some countries 29 mm, which makes it potentially suitable for implantation into larger conduits and valves. In the U.S., it is available at selected centers under the auspices of an ongoing IDE trial, which began in 2008. Reports describing the early experience with the Sapien® valve in the RVOT suggest similar short-term outcomes as with Melody® TPV replacement [4–6]. At this point, stent fracture does not appear to be a problem with the Sapien® valve, but all reported implants have been performed with pre-stenting, so any comparison with the Melody® valve is confounded. More data will be necessary to ascertain particular populations in which there may be relative advantages or disadvantages of either valve.

Expanded Applications and the Future of TPV Replacement

TPV replacement with the Melody® valve has moved beyond the original indications for use into conditions where hemodynamic and anatomic circumstances are not straightforward. For example, implantation into patients with pulmonary hypertension [35, 39•] and off-label use in patients with failed bioprosthetic valves [40•, 41] have been reported, each with efficacy and safety profiles similar to conventional patients. In particular, implantation within bioprosthetic valves represents a logical extension of TPV technology because the Melody® valve is implanted into the orthotopic position within failed surgical hardware, comparable to placement in an RVOT conduit [40•], which usually implies a fairly uniform landing zone for TPV implant.

Unfortunately, failed RVOT conduits or bioprosthetic valves are present in only a small fraction of patients suffering from postoperative RVOT dysfunction, with the majority having had patch enlargement of the RVOT as part of the initial surgical repair. Interventional cardiologists have responded to this unmet clinical challenge by developing novel and sometimes creative approaches to treating RVOT failure using existing TPV technology in a variety of different non-conduit anatomies. Melody® valve implant in native, augmented RVOT has been described by several groups [19, 42–44]. This approach only works if the device can be anchored within a landing zone that is relatively non-distensible or does not distend beyond $\sim$22 mm. Pre-stenting can help create such a landing zone, and is often employed in such patients. Moreover, pre-stenting may be important in the native RVOT, insofar as this can be a very dynamic implant environment, which was identified by Nordmeyer et al. [19] as a risk factor for stent fracture when pre-stenting was not performed. Aside from stent fracture in their series, other groups reporting TPV replacement with a native augmented RVOT have observed stable valve position and acceptable valve function in the short term [42–44]. It is not yet possible to predict definitively whether a patient with a native or augmented RVOT will be appropriate for this procedure based on pre-catheterization imaging.

Although encouraging results have been reported, due to the size limitations of current devices, the approach of pre-stenting with subsequent TPV implantation into the usual pulmonary position in non-conduit anatomies is only feasible when the RVOT is appropriately sized for stable anchoring of the device (generally $\leq$24 mm in diameter). Many patients with isolated chronic PR may not be eligible for this approach because they typically have a larger RVOT. Several treatment strategies, including Melody® valve implantation into the branch PAs [45, 46] and anchoring via a bare metal stent implanted across the main pulmonary into a PA branch (jailing) [47] have been described as potential options for TPV replacement in patients with a dilated RVOT, but to date have not been widely employed. Another approach, reported recently in a small cases series, is to implant or post-dilate the Melody® TPV using a 24 mm balloon, which does not appear to compromise valve function and may effectively expand the pool of eligible patients [48].

Newer, and as of yet experimental technologies, such as the Medtronic Native Outflow Tract device [49•], which will be entering clinical trials within the year, and infundibular reducer devices [50–52], hold promise and likely represent

future treatment alternatives for patients with a large RVOT. However, surgically augmented RVOTs can vary considerably in their geometry and dimensions [53], and undergo complex and sometimes extreme deformation during the cardiac cycle [54], which can present a unique set of challenges when it comes to developing devices that are feasible and effective for a wide population of patients. Early preclinical experience with other TPV devices, some designed for large outflow tracts, has also been described [55–60].

In patients without suitable vascular access or in whom other anatomic factors may complicate the usual percutaneous delivery, a hybrid approach in which the RV is exposed surgically and the delivery system is introduced directly through the RV may be useful, although limited experience has been reported with this technique [61]. This approach has also been reported as a "bailout" procedure after an unsuccessful attempt at percutaneous implant [62].

The Melody® valve has also been employed in positions other than the RVOT, but that experience is outside the scope of this review [39•, 63].

Conclusions

TPV replacement has rapidly become an important addition to the tool set that pediatric and adult congenital cardiologists and surgeons use in caring for patients with complex congenital heart disease that involves the RVOT and for patients who have undergone a Ross procedure for aortic valve disease. However, the technology is still relatively young, and there is much to be learned about the benefits and potential drawbacks of TPV therapy and of specific technologies. Moreover, currently available TPV technology can be applied to only a small segment of the population in which less invasive management of RVOT dysfunction might be beneficial. Except in rare circumstances, with currently approved devices, TPV replacement is not feasible in patients with a large or aneurysmal RVOT after repair of tetralogy of Fallot, which is the largest cohort of patients in whom pulmonary valve implantation is indicated.

TPV replacement promises to revolutionize the management of RVOT disease in patients with various conditions. Exactly how TPV therapy becomes integrated into the lifelong care of these patients, however, remains to be seen. TPV replacement should not be viewed simply as an alterative to surgical pulmonary valve replacement. In some patients, it may serve that role, while in others it may be used as a temporizing therapy to delay surgery. Moving forward, the task will not only be to learn more about the performance of TPV devices and the outcomes of TPV therapy in various clinical circumstances, but about the long-term functional and clinical implications of more and less aggressive approaches to RVOT dysfunction.

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