The first transcatheter pulmonary valve (TPV) implantation was reported in 2000. Drawing from that experience, the Melody valve (Medtronic, Minneapolis, MN) was developed for use in patients with obstructed or regurgitant right ventricular outflow tract (RVOT) conduits. The Melody valve received US Food and Drug Administration approval in 2010 after the initial Investigational Device Exemption (IDE, #G050186) trial. The results from that multicenter trial demonstrated substantial improvements in RVOT obstruction, conduit regurgitation, and RV pressure after valve implantation, similar to previously reported cohorts.

In some patients with a surgically implanted RVOT conduit, the conduit passes directly over or in close apposition to a coronary artery (CA). This proximity may place the CA at risk for distortion or compression if the conduit geometry or size is changed by implantation of a stent. Potential CA compression from an RVOT stent, with or without a valve, was first recognized in 2006. At least 4 cases of CA compression after Melody valve implantation have been described in literature, and the authors are aware of ≥3 others that have occurred. In addition, there have been documented cases of CA compression following stent deployment in the main or right pulmonary artery (PA). The intimate relationship between the main PA and the CAs is also highlighted by reports of CA compression by a dilated and hypertensive main PA in the setting of pulmonary hypertension, an aneurysmal postoperative PA, a surgically implanted RVOT conduit that had not been stented, and pulmonary valve replacement with a bioprosthetic valve.

Congenital and postoperative abnormalities of CA anatomy associated with conotruncal defects are well known. Even

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**Background**—The Melody transcatheter pulmonary valve (TPV) was approved for implantation in obstructed right ventricular outflow tract conduits in 2010 after a multicenter trial demonstrating improvements in conduit obstruction, regurgitation, and right ventricular pressure. A recognized risk and contraindication to TPV implantation is the demonstration of coronary artery (CA) compression during balloon angioplasty or stent placement in the overlying conduit. This study is the first to characterize the risk of CA compression in this population.

**Methods and Results**—From 2007 to 2012, 404 patients underwent 407 catheterizations for potential TPV implantation (median age, 18 years) at 4 centers. Three hundred forty-three patients (85%) underwent valve implantation. Twenty-one patients (5%) had evidence of CA compression with simultaneous right ventricular outflow tract angioplasty and CA angiography. Sixty-eight patients (17%) had abnormal CA anatomy. Fifteen of 21 (71%) patients with CA compression had abnormal CA anatomy. Eight patients with tetralogy of Fallot and 7 patients with transposition of the great arteries demonstrated compression. Of the 34 patients with tetralogy of Fallot and abnormal CA, 7 (21%) demonstrated CA compression.

**Conclusions**—CA compression following TPV implantation can be catastrophic. CA compression was observed in 5% of patients during test balloon angioplasty. No patients in this study developed clinically apparent CA compression after TPV implantation. CA compression was significantly associated with the presence of abnormal CA anatomy, especially in patients with tetralogy of Fallot or transposition of the great arteries. Preimplantation coronary angiography with simultaneous test angioplasty is an important step to evaluate for the presence of CA compression during TPV implantation. (Circ Cardiovasc Interv. 2013;6:00-00.)

**Key Words:** catheterization | heart defects, congenital | tetralogy of Fallot | transposition of great vessels | truncus arteriosus
WHAT IS KNOWN

- Transcatheter pulmonary valve replacement with the Melody valve is safe and of probable benefit as an alternative to repeat surgical replacement in patients with dysfunctional right ventricular outflow tract (RVOT) conduits.
- Coronary artery compression during RVOT conduit dilation or stenting is a well-described risk of these procedures.

WHAT THE STUDY ADDS

- This article describes the prevalence of coronary artery abnormalities in patients undergoing evaluation for Melody valve implantation.
- This article is the first to describe the incidence and risk factors for coronary artery compression in patients undergoing evaluation for Melody valve placement.
- This article describes the techniques necessary for safe and accurate preimplantation coronary artery compression testing.

in patients with normal CA anatomy, rotation or anterior displacement of the aorta, or variations in surgical reimplantation of the CAs (eg, with a Ross procedure or arterial switch), may alter the location of the CAs enough to put the patient at risk, depending on the relative position of the RVOT conduit, which can also vary. Characterizing the anatomy of the CAs before stenting by CA angiography, either alone or with simultaneous balloon dilation of the RVOT, is a crucial step when intervening on an RVOT conduit.2,3

In the initial IDE cohort, 6 of 166 (4%) enrolled patients did not undergo Melody valve implantation because of concern for CA compression based on preimplantation testing,1 which was consistent with estimates from other large series.21 The frequency of potential CA compression and specific patient-related or procedural risk factors for CA compression have not been determined for patients who may otherwise be candidates for Melody valve implantation. This study was designed to estimate the CA compression risk among a larger cohort of patients undergoing catheterization for intended Melody TPV replacement, to characterize factors associated with this risk, and to detail the technical aspects of the procedure that may improve the ability to detect CA compression before valve implantation.

Methods

Patients and Study Protocol

This was a retrospective study designed to assess the incidence and risk factors associated with CA compression in patients undergoing catheterization for Melody TPV implantation. Patients who underwent catheterization at 4 of the original IDE trial sites (Boston Children’s Hospital, Miami Children’s Hospital, Seattle Children’s Hospital, and Nationwide Children’s Hospital), both as part of the IDE trial and outside of the trial (ie, under compassionate use, emergency use, or after humanitarian device exemption approval), were included from the first IDE implantation (January 2007) through February 2012. The multicenter US Melody TPV IDE trial enrolled 166 patients across 5 centers with RVOT conduit or bioprosthetic pulmonary valve dysfunction, 150 of whom received a Melody valve. The study protocol, as well as the short- and medium-term results, was published previously.23 Enrollment was completed in January 2010. The Melody TPV was approved by the US Food and Drug Administration that same month, and each of the study sites continued to implant the valve in patients with dysfunctional RVOT conduits and bioprosthetic pulmonary valves.

Patients were included in this study if they underwent catheterization for possible Melody valve implantation. Aside from patients enrolled in the IDE trial, precatheherization evaluation and patient selection were at the discretion of individual implanters.

Catheterization and Valve Implantation

The technique for Melody valve implantation has been previously described.2 Patients who met criteria for TPV implantation underwent catheterization with hemodynamic and angiographic evaluation of the RVOT conduit or prosthesis valve. Predilation of the conduit or valve was prescribed by the IDE protocol, but placement of a bare metal stent before valve implantation (prestenting) was not allowed for the first 35 patients, after which it was permitted but not required. After US Food and Drug Administration approval, these conduit preparation steps were at the discretion of the operator. Aortic root angiography was routinely performed to characterize CA anatomy and roughly define the spatial relationship between the CAs and the RVOT. If there was suspicion for potential CA compression based on the relationship or proximity of the CAs to the RVOT conduit, dynamic CA compression testing was performed by simultaneous balloon angioplasty of the potential implantation site and either selective CA angiography or aortic root angiography, at the discretion of the operator. CA compression testing was not performed in every patient. Patients with evidence of CA compression were excluded from TPV implantation.

Fluoroscopic/Angiographic Assessments

Study investigators from each study center reviewed hemodynamic and angiographic data from the attempted Melody valve implantation for each patient. Each center collected data on segmental cardiac anatomy, CA anatomy, type of conduit or valve, the performance of compression testing with or without the use of selective CA angiography, the size of balloon used during predilation, and the size of the Melody valve delivery system. For each patient, CA anatomy was characterized as normal or abnormal (for the given cardiovascular anomaly) based on angiography, and descriptive characteristics were provided for patients with abnormal CA anatomy. If CA compression occurred, the nature of the compression was described, including the coronary system involved and the distance between the RVOT and the affected vessel.

Data Analysis

All patients who were taken to the catheterization laboratory with the intention of implanting a Melody valve in the RVOT were included in the analysis. The primary outcome was the angiographic presence of CA compression during preimplantation testing or on deployment of an RVOT stent or Melody valve. Descriptive data regarding anatomic diagnosis, previous RVOT interventions, and aberrations in CA anatomy were also collected and analyzed as risk factors for CA compression. Variables analyzed for association with outcomes included age, diagnosis, RVOT conduit type, conduit size, previous stent placement, sizing balloon diameter, and CA anatomy. The presence or absence of CA compression was considered a categorical variable, and between-group differences in this outcome were assessed using Fisher exact test.

The study was approved by the institutional review board at each participating center. All authors had access to the data and approved the article as submitted.
Results

From 2007 to 2012, 404 patients with an RVOT conduit or bioprosthetic pulmonary valve underwent 407 catheterizations at a median age of 18 years (3–73 years) for possible TPV implantation. Three patients underwent a second catheterization for pulmonary valve reimplantation after fracture of the previously placed Melody valve. Only the initial catheterization was included in the analysis. Additionally, 3 patients underwent a hybrid implantation of the Melody valve following the initial catheterization. CA compression testing was performed at the initial catheterization. Table 1 demonstrates the baseline characteristics of the cohort. The most common diagnosis was tetralogy of Fallot (TOF). The most common RV to PA conduit material was homograft. Of the 404 catheterized patients, 343 (85%) underwent Melody valve implantation (Table 2). Of the 61 patients who did not undergo TPV implantation, 28 had unfavorable conduit dimensions (7%, usually too large), and 12 did not meet criteria for implantation based on hemodynamic parameters (3%).

There were 21 cases (4.7%) of CA compression observed on preimplant testing, with the left anterior descending CA affected in 9 patients (43%), the left main CA in 5 (23%), the right CA in 3 (14%), and other branches in 4 (19%) patients. Of these 21 patients, 19 did not undergo TPV implantation. Two patients underwent TPV implantation despite evidence of compression. One patient experienced compression of a prominent conal branch from the right CA with compression testing in the proximal conduit. Repeat testing more distally did not result in CA compression and a TPV was successfully implanted. A second patient with double-outlet right ventricle demonstrated occlusion of a CA arising from the anterior sinus by an LV to PA conduit with extensive collateralization.

Table 1. Baseline Demographics

<table>
<thead>
<tr>
<th>Description</th>
<th>Number ( % )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at catheterization, y</td>
<td>18 (3–73)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>226 (56%)</td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td>130 (32%)</td>
</tr>
<tr>
<td>Pulmonary atresia</td>
<td>84 (21%)</td>
</tr>
<tr>
<td>Atrioventricular canal defect</td>
<td>3 (1%)</td>
</tr>
<tr>
<td>Absent pulmonary valve</td>
<td>9 (2%)</td>
</tr>
<tr>
<td>Transposition of the great arteries</td>
<td>37 (9%)</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>46 (11%)</td>
</tr>
<tr>
<td>Double-outlet right ventricle</td>
<td>22 (6%)</td>
</tr>
<tr>
<td>Aortic valve disease, before Ross procedure</td>
<td>51 (13%)</td>
</tr>
<tr>
<td>Pulmonary atresia with intact ventricular septum</td>
<td>7 (2%)</td>
</tr>
<tr>
<td>Valvar pulmonary stenosis</td>
<td>7 (2%)</td>
</tr>
<tr>
<td>Other</td>
<td>7 (2%)</td>
</tr>
<tr>
<td>Type of conduit</td>
<td></td>
</tr>
<tr>
<td>Homograft</td>
<td>262 (65%)</td>
</tr>
<tr>
<td>Bioprosthetic valve or conduit</td>
<td>93 (23%)</td>
</tr>
<tr>
<td>Native right ventricular outflow tract</td>
<td>28 (7%)</td>
</tr>
<tr>
<td>Synthetic</td>
<td>14 (3%)</td>
</tr>
<tr>
<td>Unknown</td>
<td>7 (2%)</td>
</tr>
<tr>
<td>Conduit diameter at the time of surgical implantation, mm</td>
<td>20 (9–29)</td>
</tr>
</tbody>
</table>

Data are presented as median (min-max) or number (%).

Table 2. Procedural Data

<table>
<thead>
<tr>
<th>Description</th>
<th>Number (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of attempted Melody valve implantations, n</td>
<td>404</td>
</tr>
<tr>
<td>Number of patients implanted, n (%)</td>
<td>343 (85%)</td>
</tr>
<tr>
<td>Indications for not implanting, n (%)</td>
<td></td>
</tr>
<tr>
<td>CA compression</td>
<td>19 (5%)</td>
</tr>
<tr>
<td>Unfavorable conduit dimensions</td>
<td>28 (7%)</td>
</tr>
<tr>
<td>Favorable hemodynamics</td>
<td>12 (3%)</td>
</tr>
<tr>
<td>Other</td>
<td>5 (1%)</td>
</tr>
<tr>
<td>CA compression testing performed</td>
<td>41%</td>
</tr>
<tr>
<td>IDE trial</td>
<td>52%</td>
</tr>
<tr>
<td>After IDE trial</td>
<td>35%</td>
</tr>
<tr>
<td>Selective CA angiography performed</td>
<td>23%</td>
</tr>
<tr>
<td>CA compression documented after stent/valve implantation</td>
<td>0</td>
</tr>
</tbody>
</table>

CA indicates coronary artery; and IDE, Investigational Device Exemption.

from the opposing CA system. A Melody valve was placed in this case.

Initial CA angiography in 3 patients before compression testing demonstrated significant compression of a CA by the conduit, and another patient was found to have proximal right CA occlusion after a Ross procedure (unrelated to the conduit). In 2 of these patients, the partially compressed CA received collateral blood flow. In addition to the patient with double-outlet right ventricle described above, a second patient with transposition of the great arteries (TGA), a ventricular septal defect, and pulmonary stenosis demonstrated proximal right CA compression with collateralization via the left circumflex CA (Figure 1). A third patient with TOF with pulmonary atresia and a left anterior descending (LAD) artery arising anomalously from the right sinus demonstrated compression of the LAD artery by the conduit during initial CA angiography. A fourth patient who had undergone a Ross procedure for aortic valve disease 12 years earlier demonstrated complete atresia of the reimplanted right CA at the ostium. There was no evidence of compression by the conduit and a Melody valve was successfully placed.

Anatomic CA abnormalities were detected angiographically in 68 (17%) patients. Depending on the anatomic diagnosis, 12% to 33% of patients had CA abnormalities (Table 3). In patients with TOF, the largest group in the study, 34 of 226 patients (15%) had documented CA abnormalities. The majority of the CA anomalies in this group consisted of an anomalous LAD CA or left main CA arising from the right anterior sinus of Valsalva and coursing underneath the RVOT conduit (20 of 34 patients).

Eight of the 21 patients (38%) with CA compression had TOF, 7 (33%) had TGA, and 3 (14%) had undergone a Ross procedure (Table 4). Of the 8 patients with TOF and CA compression, 7 had abnormal CA anatomy, including 6 with an anomalous LAD and a single CA arising from the right anterior sinus. Overall, patients with abnormal CA anatomy were at increased risk of CA compression as compared with patients with normal CA anatomy (22% versus 1.8%; P<0.001). Patients with TOF and abnormal CA anatomy were at significantly increased risk of CA compression relative to the rest of the study population (21% versus 4%; P<0.001). Similarly, patients with TGA and abnormal CA anatomy were also at
increased risk (38% versus 4%; \(P<0.001\)). Of the 7 patients with TGA and CA compression, 3 had CA abnormalities. All 3 patients had undergone a Rastelli procedure and all had LAD compression. The first patient had a circumflex from the right sinus and the LAD artery from the left-facing sinus. The second patient had a single coronary origin arising from the anterior-facing sinus. The third patient had the LAD artery arising from the right anterior-facing sinus. Only 1 patient with TGA and CA compression had undergone the arterial switch operation. There was no apparent difference in the frequency of compression between patients who had undergone an arterial switch and those in whom other types of repair had been performed.

Patients with CA compression were more likely to have abnormal CA anatomy when compared with patients without CA compression (71% versus 14%; \(P<0.001\)). In general, the majority of the patients with CA compression had an anomalous LAD artery from the right sinus and experienced compression of the LAD artery with testing. The proportion of patients in the CA compression group with TGA was higher than in the noncompression group (33% versus 8%; \(P<0.001\)). Otherwise, there were no significant diagnostic, demographic, anatomic, or procedural differences between the 2 groups.

Discussion

In this multicenter series of >400 patients undergoing catheterization for intended TPV implantation, ≈5% of patients did not undergo valve placement because of a documented risk of CA compression. This relatively high frequency of a potentially life-threatening risk highlights the importance of evaluating the CAs thoroughly in every prospective TPV replacement procedure. Patients with abnormal CA anatomy are more likely to be excluded because of risk of CA compression. Patients with TOF and an anomalous left CA branch arising from the right CA are at the highest risk of coronary compression, although this anatomic variation is not always associated with CA compression and should not be considered an absolute contraindication to TPV replacement. However, patients with a wide range of diagnoses and CA anatomy are at risk for CA compression on dynamic testing, reinforcing the fact that this is a potential complication that cannot be predicted on the basis of anatomic diagnosis alone. Of particular note, 3 of 52 (6%) patients with normally related great arteries who underwent a Ross procedure with orthotopic conduit placement were excluded from implantation for CA compression risk, a similar frequency to the overall cohort. Two of these patients had the left CA implanted in a position that was described as high and anterior, which potentially predisposed the vessel to compression during dilation of the overlying conduit. In addition to aortic position and CA anatomy, the varying location and course of the RVOT conduit relative to the CAs is also important. For example, compression, distortion, and even occlusion of a CA by the surgically implanted conduit before implantation of a stent or TPV was identified in 3 patients, and others have reported similar findings.15

Technical Considerations

The goal of CA compression testing is to identify patients at risk for compression of a CA coursing beneath an RVOT conduit or bioprosthetic pulmonary valve before placement of a stent or TPV. Despite the recognized risk of CA compression in this patient population, there have been several documented
cases of CA compression after stent or valve implantation, often with catastrophic consequences. This highlights the importance of recognizing the numerous technical considerations that go into proper assessment of CA anatomy in every patient.

Balloon dilation, stenting, or TPV implantation can all potentially change the geometry or size of the RVOT conduit and alter the anatomic relationship of the RVOT with surrounding structures. Standard angiography often does not appropriately simulate the changes in conduit geometry and the effects on the surrounding structures. Thus, it is often necessary to perform simultaneous balloon dilation of the RVOT and CA angiography to characterize the risk of compression accurately. This process can be broken down into several procedural categories: angiography, fluoroscopy, and simulation of valve implantation.

**Coronary Angiography**

After initial hemodynamic assessment, the anatomy of the CAs and their relationship to the RVOT should be fully characterized. Depending on the circumstances, this can be accomplished by aortic root angiography, selective CA angiography, or both. There are advantages and disadvantages to each method.

Aortic root angiography does not require engagement of the CA ostium, which may be challenging when the anatomy is unusual, the aortic root is dilated, or the coronary ostia are small or multiple or have been surgically reimplanted. However, aortography does not always provide adequate opacification of each coronary system and often requires more contrast. This technique also simultaneously opacifies the aortic root, which may obscure the origins of the CAs and make it challenging to delineate compression of a proximal segment. Also, aortography does not allow for a sustained injection of contrast into the coronary circulation during balloon inflation, which may aid in the accurate identification of CA distortion in subtle cases of compression.

Selective CA angiography allows the operator to take repeated angiograms at different projection angles and with different balloon positions in the RVOT, if necessary. It allows for sustained injection into the CA of interest throughout the duration of balloon inflation to ensure that there is no distortion or compression during all possible changes in RVOT geometry. In cases of very proximal CA compression, deep engagement of the CA with the catheter may limit the degree of compression by stenting open the vessel. In these instances, it is helpful to perform the angiogram with the catheter in the sinus just outside of the CA proper. The other principle concern with selective CA angiography is the risk of damage to the CA itself resulting from coronary spasm, dissection, or air embolism. Thus, use of this technique requires operator judgment based on the initial angiographic assessment of the CAs.

**Fluoroscopy**

In addition to appropriate angiographic techniques, the optimization of fluoroscopic angles is crucial to characterize the relationship accurately between the CAs and the balloon. This may be difficult because of the extreme camera angles
necessary to characterize the anatomy or the presence of hardware in the thorax, such as spinal fusion rods.

In general, camera angles orthogonal to the plane of interest are best. This allows optimal characterization of the relationship between the vessel of interest and the RVOT or balloon. Subtotal compression of the CA will occur most frequently in the anteroposterior or superoinferior direction because the CA at risk usually passes beneath the conduit. Camera angles parallel to the long axis of the RVOT (or balloon) are best, often using caudal and left or right anterior oblique angulation of the anterior camera. This view shows where the balloon is closest to the CA and will provide the least distorted view of the space between the CA and the RVOT. A lateral camera orthogonal to the long axis of the RVOT foreshortens the CA system in question (Figures 2 and 3), helps determine where on the conduit the CA runs, and defines where the balloon waist lies relative to the CA. This may help determine whether a more proximal or distal implantation site may potentially be safe. Finally, one should inflate the compression testing balloon with dilute contrast so that the CA can be clearly distinguished through the contrast-filled balloon, and that the balloon has enough contrast to define the balloon outline (Figure 4).

The distance between the CAs and the RVOT based on CA angiography alone does not necessarily reflect the risk of compression (Figure 4). Calcification, scar tissue, and residual conduits or stents may result in an incompressible mass between the existing conduit and the CAs and, thus, cause CA compression even if there is significant distance between the 2 structures.

Figure 2. Seventeen-year-old man with TOF and anomalous left anterior descending (LAD) coronary artery (CA) from the anterior sinus. A, LAD angiogram with right anterior oblique, caudal, and lateral views. There is no vessel compression. B, Compression testing demonstrates compression of the proximal origin of the LAD CA.

Figure 3. Forty-seven-year-old with bioprosthetic aortic valve who underwent Ross procedure. There are normal coronary artery origins based with no baseline compression on initial angiography. A caudal, right anterior oblique projection demonstrates compression of the left anterior descending artery during simultaneous conduit angioplasty and selective coronary angiography.
Simulation of TPV Implantation

Test balloon angioplasty of the RVOT with simultaneous CA angiography seems to be the most accurate way to simulate the changes that will occur with valve implantation. There are several technical considerations that likely improve the accuracy of this process. Balloon size and type should mimic accurately the effects of valve implantation. Once the sizes of the delivery system and valve have been selected, compression testing should use a balloon that is similar to the diameter at which the TPV will be implanted. Conduit rupture with test dilation was not encountered in this study. However, starting with smaller balloon sizes and gradually increasing to the target balloon size may minimize this potential risk. It may not always be practical to use test balloon sizes equal to the delivery system, but it is often possible to determine if the relationship between the conduit and CA is concerning even with smaller balloon sizes. If the relationship is concerning, a conservative approach should be adopted and every effort should be made to perform test inflations with balloon sizes that closely simulate the deployed valve, recognizing the intrinsic limitations to this method.

There are no data that can help determine which type of balloon is preferable for CA compression testing. Low-pressure balloons may not dilate the conduit adequately, whereas high-pressure balloons may alter the geometry of the RVOT in a manner that does not accurately reflect changes after TPV implantation because of their long lengths and tapers. In some cases, there may be displacement of the CA without compression before there is obvious narrowing of the lumen. Thus, angiography should extend throughout inflation.

Limitations

This study has several limitations. The role of precatheterization imaging was not considered or evaluated. Test angioplasty does not perfectly simulate conduit stent or valve implantation and may create greater conduit displacement, especially when elongated angioplasty balloons are used. Thus, certain patients were excluded from TPV implantation who may have been safely implanted. Only a small number of patients underwent repeat CA angiography after valve implantation; so it is difficult to rule out the possibility of subtle distortion or compression of the CAs not identified on the original study. However, none of the patients manifested any clinical evidence of CA compression after implantation. This study primarily examined the risk of compression of larger CA branches. It is unclear whether compression or occlusion of secondary epicardial branches is clinically significant.

Although this study suggests that patients with abnormal CA anatomy, particularly those with TOF and TGA, are at increased risk of CA compression, the study cohort and the number of cases of compression are small. Thus, the study had limited power to discern other potentially important risk factors, including the era of the procedure and the effects of center/operator variability and experience.

Although this study suggests that patients at risk for CA compression can be identified safely before TPV implantation with the approach of dynamic compression testing outlined above, these methods will not be infallible. It is important to recognize that our means of identifying CA compression risk are imperfect, and to proceed with caution if faced with a potentially high-risk situation. Additional research may help identify other methods of predicting and evaluating CA compression in potential TPV recipients.

Conclusions

This study indicates that a significant number (5%) of cases in which Melody valve implantation was considered were aborted because of the presence of CA compression during RVOT test angioplasty. This potential complication was more common in patients with TOF and coronary anomalies and those with TGA. Careful attention to the technical details of test angioplasty successfully avoided this potentially life-threatening complication in this large study cohort.

Disclosures

Drs McElhinney, Cheatham, Zahn, and Jones receive research grant support and serve as consultants and proctors for Medtronic, the manufacturer of the Melody valve. Dr Cheatham also acts as a consultant for NuMed. The other authors have no conflicts to report.

References


Risk of Coronary Artery Compression Among Patients Referred for Transcatheter Pulmonary Valve Implantation: A Multicenter Experience


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Supplemental Material

Video Legend

Video. 30-year-old with TOF and anomalous origin of the LAD coronary artery from the RCA. A caudal, LAO projection demonstrates the long axis of the angioplasty balloon. This defines the superior-inferior relationship of the conduit to the LAD. There is heavy RVOT conduit calcification. Simultaneous selective LAD angiography demonstrates compression of the CA as it courses inferior to the conduit. The lateral projection allows visualization parallel to the long axis of the LAD and helps identify where along the axis of the conduit the compression occurs.