Pulmonary Hypertension in Patients Undergoing Transcatheter Aortic Valve Replacement
Discussion of the Current Evidence

Mario Gössl, MD; Garvan C. Kane, MD, PhD; William Mauermann, MD; David R. Holmes, MD

Pulmonary hypertension (PH) is a significant, often irreversible, risk factor of early and late morbidity and mortality in patients undergoing transcatheter aortic valve replacement (TAVR). The widely used Society of Thoracic Surgeons (STSs) Predicted Risk of Mortality (STS-Predicted Risk of Mortality) risk score does not include PH and the commonly associated right ventricular (RV) dysfunction. Therefore, procedural risk of patients with severe PH undergoing TAVR is probably underestimated and consideration of its risk relies heavily on clinical, procedural, and anecdotal experience. In addition, literature on the management of patients with severe PH undergoing TAVR is scarce.

Using 1 of our recent transapical TAVR patients as an illustrative example we, therefore, sought to discuss recent data on outcomes of patients with severe PH undergoing TAVR, emerging TAVR risk scores that incorporate severe PH and other TAVR-specific risk factors as well as a guideline on how to clinically manage patients with severe PH undergoing TAVR who often present as difficult hemodynamic challenges.

Case Presentation
An 82-year-old (body mass index, 26.8 kg/m²) insulin-dependent diabetic female (151 cm, 62 kg) with symptomatic severe calcific aortic stenosis (echocardiographically derived: mean transvalvular gradient 43 mm Hg, maximal velocity 4.3 m/s and a calculated valve area of 0.5 cm²) and preserved left ventricular (LV) function with an ejection fraction of 60%. She has severe coronary artery disease, is status post coronary artery bypass graft surgery with patent grafts to left anterior descending and ramus intermedius; the circumflex artery is only moderately diseased and the right coronary artery is small, nondominant with mild, diffuse disease (<1.5 mm in size). The right heart hemodynamics are summarized in Figure 1 and Table 1 with mild-to-moderate right heart failure (increased RV end-diastolic pressure) and severe mixed PH with LV hypertrophy and associated LV diastolic dysfunction. The echocardiogram showed furthermore a preserved RV function with mild RV enlargement. We did not assess her pulmonary vascular reactivity during the right heart catheterization. She had no history of pulmonary thromboembolism or pulmonary disease, her preoperative pulmonary function test was within normal limits.

Her most recent laboratory data are summarized in Table 2. Because of her cardiovascular history, comorbidities, and high STS risk score (10.8%), our heart team decided to offer TAVR. Of significant peripheral vascular disease (<6-mm-sized iliac arteries bilaterally), she underwent transapical TAVR with a 23-mm Edwards Sapien valve.

Discussion

Dr Gössl: Patients like this are a clinical dilemma for any heart team, they are older and often have multiple, severe comorbidities, including various degrees and causes of PH. Because of their complex hemodynamics, they require a high level of care and constant vigilance of the heart team.

Dr Holmes: I fully agree. The literature unequivocally demonstrates that patients with PH undergoing cardiac surgery show increased morbidity and mortality,1–3 with a growing body of evidence in TAVR. Right heart failure with subsequent inadequate filling (preload) of the LV needs to be avoided at any cost because it may lead to rapid decline and ultimately death of the patient.6,7

Dr Gössl: Dr Kane, how would you classify this PH patient?

Dr Kane: The 2009 American College of Cardiology Foundation/American Heart Association expert consensus document6 on PH defines PH as a mean pulmonary artery pressure (PAP) ≥25 mm Hg with precapillary PH or pulmonary arterial hypertension (PAH) as a mean PAP of >25 mm Hg at rest in the setting of a normal PAWP of ≤15 mm Hg with a pulmonary vascular resistance of >3 Wood units. PH has been classified by the World PH Symposium in 5 classes.8,9 Our patient shows features of mixed PH with evidence of high LV filling pressures presumably secondary to her long-standing and severe valvular heart disease with high pulmonary vascular resistance. Unless serial

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measurements of right heart hemodynamics in the same individual are available, it is often impossible to establish if a patient with severe aortic stenosis had preexisting PAH and developed the high LV filling pressures later or if long-term exposure of the pulmonary vasculature to high LV filling pressures led to progressive increasing reactive pulmonary arterial resistance secondary to pulmonary vasoconstriction and remodeling.11

Dr Gössl: We did not perform a vasodilator challenge. Would the additional information have helped in the patient management and procedural planning?

Dr Kane: The extent of pulmonary vascular reactivity may have been useful information. The intraprocedural management would not have changed (management strategies are limited) but the TAVR team would have been able to better predict the responsiveness of the patient to and difficulty of the peri-procedural management. In the setting of marked increase in systemic blood pressure and an elevated PAWP (as in this case), I would normally recommend intravenous challenge with nitroprusside to assess responsiveness of the PAWP and PAP. If there was persistent PAP elevation after normalization of PAWP then an additional challenge of inhaled nitric oxide (NO) would be helpful. However, this hemodynamic assessment is more challenging in the setting of fixed aortic valve stenosis and while not prohibitive may come with additive risk.

Dr Gössl: With a plethora of trial and registry data, it would be great to incorporate TAVR-specific risks into risk calculators or to develop TAVR-specific risk calculators.

Dr Holmes: We are in the final stages of developing an STS/American College of Cardiology/National Cardiovascular Data Registry risk calculator for patients with TAVR based on data from the transcatheter valve therapies registry data, which will, among other more TAVR-specific risk factors, include PH as well.

For now, the most current data come from French Aortic National CoreValve and Edwards (FRANCE) 2 and Observational Study of Appropriateness, Efficacy and Effectiveness of AVR-TAVR Procedures for the Treatment of Severe Symptomatic Aortic Stenosis (OBSERVANT), 2 risk calculators which incorporate more recent data and PH. The OBSERVANT risk score is derived from the largest multicenter study of TAVR in Italy (Observational study of Appropriateness, Efficacy and Effectiveness of AVR-TAVR Procedures For the Treatment Of Severe Symptomatic Aortic Stenosis, n=1911; Figure 2).12 The score ranges from 0 to 29, patients with a score >14 showed a clear increase in mortality. The authors point out that <10% of all studied patients had a score >14 and thus further validation in larger, high-risk cohorts, is warranted.

The investigators of the FRANCE 2 registry (n=3831) developed a 21-point predictive score for 30-day or in-hospital mortality including 9 independently predictive factors of early mortality (Figure 3).13

The German aortic valve risk score is specific to aortic stenosis and achieves a c-index of 0.81 but only 573 TAVR cases are included.14

Arnold et al15 have recently reported predictors of poor outcome from the Placement of Aortic Transcatheter Valves (PARTNER) trial. PH was not further specified but the presence of any PH was not associated with poor outcome. The analysis showed that poor functional capacity, lower mean aortic valve gradients, oxygen-dependent lung disease, renal dysfunction, and poor baseline cognitive dysfunction were the important predictors for poor outcome.

Dr Gössl: Using these currently available risk assessment tools, her calculated STS-Predicted Risk of Mortality mortality risk is 10.8%. Using the OBSERVANT and FRANCE 2 risk scores, her 30-day mortality risk is 10% (14 points) and 10% to 11% (4 points), respectively.

Drs Kane and Mauermann, how would you approach this patient in your pre-TAVR planning?

Dr Kane: A hemodynamic improvement in precapillary PH could not be expected from TAVR, however, conceptually one could expect an improvement in postcapillary PA hemodynamics. Experience tells us that these changes, if they do occur, frequently take time and are not immediately apparent in the postoperative period. Although there seem to be some differences between how the right heart is affected by pre capillary versus post capillary PH, overall it is the RV that invariably dictates outcome in patients with PH.

The finding that PH (systolic pulmonary artery pressure [sPAP] > 60 mm Hg, in some studies even >40 mm Hg) is associated with higher mortality after surgical aortic valve replacement and TAVR is unequivocal. Melby et al14 showed that patients with PH undergoing surgical aortic valve replacement had longer hospital stays with prolonged ventilation and 5-year survival was significantly decreased (45% versus 78% in those with normal pulmonary pressures [<35 sPAP]). Similarly, Rodès-Cabau et al15 showed a 2-fold increase in short- and long-term (<1 year) mortality risk in their TAVR population of 339 patients. Luçon et al16 (FRANCE 2 investigators) showed that even patients with moderate or severe PH (sPAP 40 mm Hg per transthoracic echo) had a 28% 1-year all cause mortality compared with 22% in those with normal pulmonary pressures. Sinning et al17 corroborate these findings with a 2-year mortality of 48.4% in patients with sPAP >60 mmHg compared with 13.9% in patients with sPAP <30 mm Hg. In addition, they found that a decrease in sPAP (< 60 mm Hg) after a successful TAVR procedure carries an improved prognosis, suggesting that a predominantly postcapillary PH might be more beneficial and amendable to TAVR therapy. However, as nicely shown by Roselli et al18 in a surgical aortic valve replacement population of >4000 patients, not only was long-term survival (10 years) again twice as bad in patients with severe PH (31% versus 63%) but initial post surgical aortic valve replacement improvement in RV systolic pressure rose to preoperative levels within 3 to 4 years. This suggests that PH secondary to severe aortic stenosis may simply not reversible, a fact that TAVR teams need to take into consideration.4,5

Dr Mauermann: From the anesthesia/critical care point of view, these cases are challenging and the hemodynamics are complex. Both the perioperative but especially postoperative care require utmost attention. Maintaining adequate RV function and, therefore, sufficient LV preload and cardiac output are the main therapeutic goals for patients with severe PH undergoing TAVR. The normal pulmonary vasculature is a highly compliant, high capacity, but low-resistance, low-pressure system19; the RV is thin-walled (<5 mm), highly compliant and afterload-sensitive (Laplace law). An abrupt increase in PAP or resistance leads to pulmonary vascular remodeling.11

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It is also important to note that, in contrast to the LV, coronary perfusion of the RV is bicyclic, occurring in both systole and diastole because of the gradient between mean systemic arterial pressure and right ventricular systolic pressure (Table 3). In PH, when mean PAP and mean systemic arterial pressure values approach each other, especially when combined with RV hypertrophy, the RV is prone to ischemia because perfusion is limited to systolic coronary flow (Figure 4). If the RV is dilated, RV myocardial oxygen consumption is increased. All of this is especially important to consider when rapid ventricular pacing is required for valve deployment. This period of ischemia combined with high myocardial oxygen demand may be significant enough to move the compensated RV into an uncompensated state with resultant heart failure.

**Dr Gössl:** Dr Holmes, would you consider aortic valve balloon valvuloplasty to assess the change in pulmonary pressures?

**Dr Holmes:** In selected cases, this may be an option, but we have to keep in mind that balloon aortic valvuloplasty often produces only modest decreases in mean transvalvular gradients and carries with it, its own procedural and vascular complication risks. Moreover, based on the results by, for instance, Ben-Dor et al., the acute decrease in pulmonary systolic pressure is on average only \( \approx 12 \) mm Hg and may not last longer than 3 months.

**Dr Gössl:** Drs Kane and Mauermann, do you have specific therapeutic goals in mind? What medical/interventional options would you consider?

**Dr Kane and Dr Mauermann:** Because of the low ischemic threshold of the RV, 1 goal is to maintain an adequate RV perfusion pressure.

**Goal No. 1: Keep Mean Systemic Arterial Pressure>Mean Pulmonary Artery Pressure**

To preserve RV coronary perfusion, some authors also suggest to keep the hemoglobin \( >10 \) g/dL and arterial oxygenation \( >90\% \) to maximize oxygen content.
Goal No. 2: Keep Hemoglobin >10 g/dL and Oxygenation >90%

Using right atrial pressure as a guide, it is important to avoid excessive volume loading and RV distension as this may set up a cycle of worsening RV wall stress, ischemia, leftward septal shift and compromise LV filling through ventricular interdependence.

Goal No. 3: Avoid RV Distension

In PH, the noncompliant, dilated RV depends on atrial contractions to maintain RV systolic function. Maintenance of sinus rhythm and atrioventricular synchrony with a heart rate between 60 and 100 bpm is key.

Goal No. 4: Maintain Sinus Rhythm With Heart Rates of 60 to 100 bpm

Ventilation Strategies

In patients with acute respiratory distress syndrome, an RV-protective ventilation is often used and it is conceivable that such strategy would have a beneficial effect in patients with severe PH. The overall goal in both clinical scenarios is to avoid an increase in pulmonary vascular resistance (ie, hypercapnia, acidosis, and hypoxia). If the pulmonary pressures are dynamic (ie, not fixed), relative hyperoxia and hypocarbia (arterial CO$_2$ =32–35 mm Hg) may decrease the PAP and partially unload the RV. In addition, as noted above the RV exposed to PH is preload dependent. Attention must be paid to the effects of positive pressure ventilation on this preload. Bouferrache et al$^{24}$ proposed plateau pressures of <27 cmH$_2$O, a low level positive end-expiratory pressure of <7 cmH$_2$O, avoidance of intrinsic positive end-expiratory pressure, and the control of hypercapnia.

Goal No. 5: RV Protective Ventilation (Plateau Pressure <27 cmH$_2$O, Positive End-Expiratory Pressure <7 cmH$_2$O)

Dr Gössl: What medical options are in your arsenal?

Dr Kane and Dr Mauermann: There is a multitude of PH medications available per os, intravenously or inhaled that have been tested in clinical trials primarily in the outpatient arena. Although these PH-targeted therapies are relatively specific pulmonary artery vasodilators, their long-term benefit is more probably accrued through reverse remodeling of the pulmonary vasculature with secondary unloading of the right heart rather than a vasodilatory action (Table 4). In patients treated with PAH-specific therapies, it is critical to continue these therapies uninterrupted in the perioperative period to avoid the potential for acute pulmonary hypertensive crisis.$^{25}$

Use of PAH-specific therapies in the setting of acute RV failure for subjects with PH has not been well studied. Short acting titratable therapies with least potential for systemic hypotension are preferable. Inhaled NO leads to pulmonary vasodilation, decreased PAP, and pulmonary vascular resistance by increasing intracellular cyclic guanosine monophosphate. Because of its rapid deactivation (binding to both oxyhemoglobin and deoxyhemoglobin), there are no systemic vasodilatory effects. Because of its additive effects on the pulmonary vasculature, it can be nicely combined with milrinone and dobutamine.$^{26,27}$ Abrupt discontinuation of inhaled NO can lead to significant rebound PH,$^{28}$ it should, therefore, be weaned off slowly with close attention to hemodynamics.

For the dilated or dysfunctional RV, a combination of an inotrope (epinephrine) with an inhaled pulmonary vasodilator that has no systemic effects (NO) may be ideal. In PH, we do not have PA antihypertensives where one size fits all. Therapy may need to be tailored for the underlying mechanism of PH.

Table 2. Laboratory Data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>7.4×(10$^3$/μL)</td>
</tr>
<tr>
<td>RBC</td>
<td>12.7×(10$^3$/μL)</td>
</tr>
<tr>
<td>Platelet</td>
<td>275×(10$^3$/μL)</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1.19 mg/dL</td>
</tr>
<tr>
<td>GFR (Cockcroft–Gault)</td>
<td>36 mL/min per 1.7m²</td>
</tr>
<tr>
<td>Potassium/Sodium</td>
<td>141/3.9 mmol/L</td>
</tr>
</tbody>
</table>

GFR indicates glomerular filtration rate; RBC, red blood cell; and WBC, white blood cell.

Figure 2. OBSERVANT risk scoring system for the prediction of 30-day mortality (left) and bedside chart for estimation of 30-day mortality (right). BAV indicates balloon aortic valvuloplasty; CI, confidence interval; GFR, glomerular filtration rate; LVEF, left ventricular ejection fraction; and OBSERVANT, Observational Study of Appropriateness, Efficacy and Effectiveness of AVR-TAVR Procedures for the Treatment of Severe Symptomatic Aortic Stenosis. Reprinted from Capodanno et al$^{10}$ with permission of the publisher. Copyright ©2014, Elsevier Inc.
Certainly precapillary PH, particularly PAH (Group 1 PH) may respond to PA vasodilator therapy, however, in other forms of precapillary PH such as PH related to hypoxia or parenchymal lung disease (group 3 PH, such as chronic obstructive pulmonary disease or pulmonary fibrosis) PA vasodilator therapy may significantly worsen oxygenation through an exacerbation of ventilation–perfusion mismatch. This is important to recognize, as this is a common cause of PH in patients with TAVR. In postcapillary PH (group 2 PH), selective PA vasodilation may actually lead to a rise in left atrial pressure and a tendency to cause pulmonary edema. The prediction of how the individual may respond is often difficult.

**Dr Gössl:** So what about inotropes?

**Dr Kane and Dr Mauermann:** Vasopressor or inotrope support may be needed in the perioperative and postoperative phase of these critically ill patients. Table 4 summarizes generally available vasopressors/inotropes and their theoretical effect on hemodynamics, RV contractility, and both the systemic and pulmonary vasculature. Typically, they are used in combination where the actions of one may be balanced against the actions of the other with a view to maximizing benefit.

**Dr Gössl:** Is there a role for RV support devices?

**Dr Kane:** The results for the Impella RP (Abiomed Inc, Danvers, MA), a new percutaneous ventricular assist device for RV failure (A Prospective, Multicenter Study to Evaluate a New Percutaneous Ventricular Assist Device for Right Ventricular Failure [RECOVER RIGHT] study) are promising, this and other devices, however, will need to be tested in TAVR patients with RV failure in future clinical trials. It is important to note that assist devices tend to work better in the setting of a lower resistance.

**Dr Holmes:** In addition to the above-mentioned ino-pres- sors as well as vasodilator support, diuresis with the goal of unloading the RV is an important component of the intensive care therapy.

**Dr Gössl:** Is it reasonable to recommend monitoring with a Swan Ganz pulmonary catheter?

**Dr Mauermann and Kane:** Swan Ganz monitoring can be helpful, the use of such a catheter, however, is not without risk. Patients with severe PH are at increased risk of pulmonary

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**Table 3. LV and RV Coronary Perfusion**

<table>
<thead>
<tr>
<th></th>
<th>Systolic BP, mm Hg</th>
<th>Diastolic BP, mm Hg</th>
<th>CPP Systole, mm Hg</th>
<th>CPP Diastole, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RV</strong></td>
<td>25</td>
<td>5</td>
<td>95</td>
<td>75</td>
</tr>
<tr>
<td><strong>LV</strong></td>
<td>120</td>
<td>5</td>
<td>0</td>
<td>75</td>
</tr>
<tr>
<td><strong>Ao</strong></td>
<td>120</td>
<td>80</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

Ao indicates aorta; BP, blood pressure; CPP, coronary perfusion pressure; LV, left ventricle; and RV, right ventricle. Reprinted from Hosseinian20 with permission of the publisher. Copyright ©2014, Elsevier Inc.
artery rupture during Swan-Ganz catheterization. If one avoids wedging the catheter, this risk is likely low and probably outweighed by the benefits. However, one must be careful to avoid focusing too much on the PA pressures. RV function is frequently the determinant of outcome rather than PA pressure. Ultimately, the most useful parameters to follow will include systemic blood pressure, oxygenation status, cardiac output, and right atrial pressure. Bedside echocardiographic evaluation of the right heart size and function can also be helpful.

Dr Gössl: Dr Holmes, do you see a potential usefulness of self-expandable, repositionable TAVR valves in cases similar to ours?

Dr Holmes: The future valve designs will continue to become smaller and facilitate easier transfemoral access or transapical approaches using percutaneous techniques and closure devices. In addition, we will see more valves that can be safely and reliably implanted without the need for rapid ventricular pacing. As was seen in the patient here, rapid ventricular pacing may be poorly tolerated particularly if it has to be repeated because of problems with the optimal implantation site.

Dr Gössl: Dr Mauermann, do you see an advantage between general anesthesia versus local analgesia?

Dr Mauermann: The choice of general anesthesia versus monitored anesthesia care with sedation seems to be largely an institutional preference and based on the depth of experience. One benefit of general anesthesia is that it allows for prolonged imaging with transesophageal echocardiography. At least 1 group has described continuous transesophageal echocardiography monitoring while administering noninvasive ventilation by placing the probe through an opening created in a face mask. However, the benefits of this approach are unclear. Although it may seem intuitively beneficial to perform high-risk cases with sedation only, one must be careful to avoid hypercarbia or hypoxia which may exacerbate the PH. In addition, the relative hyperoxia, hypocarbia, and vasodilation from a general anesthetic often times has a salutary effect on the PAPs during the procedure.

Dr Gössl: Do we need to consider futility in patients like the one we present here with multiple comorbidities?

Dr Holmes: A recent review addressing futility in patients with TAVR highlights severe PH as 1 of the high risk indicators among others like high STS scores (>15), severely impaired LV function and frailty. With the recent addition of risk scores that incorporate these TAVR-specific risks, the heart team should have a detailed discussion with patients and their families about risks and benefits of the procedure.

Clinical Outcome

After the start of anesthesia and with inhaled NO at 20 ppm, our patient’s systemic blood pressure was well controlled and her mean PAP 35 mm Hg. Even with our brief test run of the temporary pacemaker with only 100 bpm, our patient showed significant hypotension and had a slow recovery of her blood pressure with norepinephrine. We, therefore, decided to implant the Edwards Sapien valve without previous balloon aortic valvuloplasty. As expected, our patient did not tolerate rapid pacing during valve deployment well.
Table 4. List of Medications and Dosing, Used Perioperative and Postoperative Phase

<table>
<thead>
<tr>
<th>Ino-pressors</th>
<th>Right Ventricular Inotropy</th>
<th>Systemic Vascular Effect</th>
<th>Pulmonary Vascular Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>...</td>
<td>+++</td>
<td>--</td>
</tr>
<tr>
<td>Dobamine (low/intermediate dose)</td>
<td>...</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Dopamine (high dose)</td>
<td>...</td>
<td>++</td>
<td>---</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>...</td>
<td>+</td>
<td>---</td>
</tr>
<tr>
<td>Ino-dilators</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milrinone</td>
<td>IV and inhaled</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Beyond 10 µg no further improvement in PVR</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>...</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Vasopressors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>...</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>...</td>
<td>---</td>
<td>+</td>
</tr>
<tr>
<td>Pulmonary vasodilators</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitric oxide</td>
<td>Rebound PH</td>
<td>...</td>
<td>+++</td>
</tr>
<tr>
<td>Epoprostenol</td>
<td>Rebound PH, inhaled prostacyclin with less bleeding risk</td>
<td>...</td>
<td>+++</td>
</tr>
<tr>
<td>Iloprost</td>
<td>Less rebound PH than Epoprostenol</td>
<td>...</td>
<td>+</td>
</tr>
<tr>
<td>Treprostenil</td>
<td>Less rebound PH than Epoprostenol and Iloprost</td>
<td>...</td>
<td>++</td>
</tr>
<tr>
<td>Sildenafil</td>
<td>IV:PO=1:2</td>
<td>...</td>
<td>+++</td>
</tr>
</tbody>
</table>

IV indicates intravenous; PH, pulmonary hypertension; PO, per os; and PVR, pulmonary vascular resistance.

* denotes vasoconstriction; +, vasodilation.

(due to inhaled NO as well as low dose milrinone and epinephrine support). She went into PEA arrest and required ≈1 minute of chest compressions and high dose ino-pressor support after valve deployment. After TAVR, her mean PAP did not significantly change and she remained on inhaled NO, intermittent intravenous isoproterenol, epinephrine, and milrinone for hemodynamic support, she was diuresed with intravenous Lasix. She spent a total of 3 weeks in our intensive care unit because of the fact that weaning her off ventilation was difficult with fast deterioration of hemodynamics.

Our patient and her family are happy about the clinical result, she is no longer short of breath and, therefore, feels an increased quality of life which was ultimately our main goal. Her 1-month follow-up echocardiogram showed a normal LV systolic function of 65%, normal RV size and function and an estimated systolic PAP of 75 mmHg (unchanged from pre-TAVR exams).

Disclosures
None.

References
Pulmonary Hypertension in TAVR


Key Words: pulmonary hypertension ■ transcatheter aortic valve implantation
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