A 92-Year-Old Woman With Severe Aortic Stenosis Scheduled for Open Reduction and Internal Fixation of Fractured Hip: Lessons From and Impact of the TAVR Experience

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Stem Case and Key Questions Content
Part 1a Preoperative Clinic
You are asked to consult on a 92-year-old female for treatment of a hip fracture who has severe aortic stenosis. The orthopedic surgeon states that the patient has been “cleared by cardiology” and wants to do the case as soon as possible.

You obtain the following additional history after reviewing the chart and interviewing the patient and her family. She is 5 feet tall, weighs 50 kg, and is mildly demented. Past medical history includes long-standing hypertension, one episode of gastrointestinal bleeding, a “mild stroke,” and a cholecystectomy.

The laparoscopic cholecystectomy one year prior was complicated by congestive heart failure requiring a one-week stay in the intensive care unit. A transthoracic echocardiogram done one year prior, at the time of the cholecystectomy, revealed an aortic valve area of 0.6 cm², peak aortic valve area velocity of 3.2 m/sec, peak gradient of 40 mm Hg, moderate mitral regurgitation, and pulmonary artery pressure of 50 mm.

Key Question 1:
Why do you think the patient went into congestive heart failure perioperatively at the time of her cholecystectomy one year ago when she had a relatively low gradient and low aortic valve area peak velocity?

After that hospitalization a year ago, the family (the husband is a retired cardiologist), acting for the patient, refused surgical aortic valve replacement, balloon valvuloplasty (BAV) or transcatheter aortic valve replacement (TAVR) because of the risk of these procedures.
Current Physical Examination:
She is awake and alert but is complaining of moderately severe leg pain. She is oriented to place and person but not to time.

Her current vital signs include heart rate of 90 beats per minute, irregularly irregular heart rhythm, blood pressure of 100/45 mm Hg, respiratory rate of 26 breaths per minute, temperature of 37.4° Celsius, and SaO2 of 95% on 3 liters oxygen.

A 4/6 systolic murmur is heard over the left and right subclavian arteries going into the carotids, and a 2/6 diastolic murmur is present in the same distribution. A 2/6 holosystolic murmur is heard at the apex. You hear rales at the bases on auscultation of the lungs.

Current Laboratory:
Hemoglobin and hematocrit are 10 and 30, respectively. Her glomerular filtration rate is 40% of predicted with a serum creatinine of 2 mg/dl. B-type natriuretic peptide is 400 iU. Electrocardiogram shows atrial fibrillation with a ventricular response of 90 beats per minute. Chest X-ray is read as “signs suggestive of congestive heart failure.”

Key Question 2:
Her cardiologist husband is worried about delaying the case. What are the risks of waiting? How long can you safely delay the case?

Her cardiologist for this admission did not, in fact, “clear” the patient but wrote, “Patient at very high risk for general anesthetic given severe aortic stenosis. Would consider BAV or TAVR prior to orthopedic surgery if patient cannot have spinal.”

Key Question 3:
Is the cardiologist correct that doing a spinal would reduce the risk?

Key Question 4:
Should she have a TAVR? Should she have a BAV?

After a family conference (to which you are invited to discuss the risks of anesthesia), the patient and family agree to proceed with whatever it takes to get the surgery done because of the pain she is suffering. They would like to know how you assess her risk.

Key Question 5:
To assess her current risk, what additional information do you want? Is a repeat transthoracic echocardiogram/transesophageal echocardiogram needed? Why or why not? Does she need a dobutamine stress echocardiogram? Why or why not?
The cardiologist orders a repeat transthoracic echocardiogram and dobutamine stress echocardiogram in order to more accurately assess the current risk of proceeding with any surgery. A repeat transthoracic echocardiogram and stress dobutamine echocardiogram reveal the following: with dobutamine, ejection fraction increases to 45% from 30%, peak velocity across the aortic valve is 5.2 m/sec, and the aortic valve area remains 0.5 cm².

Key Question 6: What is the significance of the new findings? How do the new results influence your decision to recommend preoperative BAV or TAVR?

The cardiologist performs a BAV. The aortic valve area increases from 0.5 cm² to 0.9 cm² and the mean gradient decreases from 70 mm Hg to 35 mm Hg.

Key question 7: What results do you expect from BAV? How long after the BAV should you wait before proceeding to the operating room for the hip surgery?

The orthopedic procedure planned is a total hip replacement because the hip fracture is not amenable to a simple open reduction internal fixation.

Key question 8: What monitoring would you use and why? Does the choice of anesthetic (regional versus general) dictate your monitoring?

After induction of general endotracheal anesthesia, the blood pressure drops from 130/55 mm Hg to 70/30 mm Hg, and the patient’s heart rate goes from 90 beats per minute (atrial fibrillation) to 130 beats per minute. The central venous pressure, which was 12 mm Hg prior to induction, rises to 22 mm Hg.

Key question 9: What might account for the change in the hemodynamics? How could the change have been prevented?

Additional norepinephrine bolus and drip restores the hemodynamics and the case continues uneventfully. The orthopedic surgeon is surprised that you have requested a bed in the intensive care unit.

Key question 10: What problems do you anticipate that need a bed in the intensive care unit?
The colleague who suggested a continuous spinal for the case stops by and asks if you are going to do a single-shot intrathecal morphine injection to help reduce the chance of delirium postoperatively.

Key question 11:
What are the advantages and disadvantages of a single-shot morphine injection in this patient?

Model Discussion Content
AORTIC STENOSIS DIAGNOSIS AND NEW CLASSIFICATION:
Aortic stenosis is the most frequent type of valvular heart disease in North America, most often presenting as calcific in adults of advanced age (incidence of 2-7% in the population age >65 years). The second most frequent cause is congenital. Elderly patients adapt to the slowly progressive cardiac disability associated with aortic stenosis, therefore careful questioning by clinicians to elicit symptoms is required. Typical symptoms at presentation include shortness of breath on exertion, angina, dizziness and syncope.

Echocardiography, the method of choice for evaluating aortic stenosis, permits Doppler quantification of the valve area, elucidates the exact anatomy, defines the degree of accompanying left ventricular hypertrophy, and determines left ventricular function. Additionally, echocardiography permits evaluation of any accompanying aortic pathology or other valvular disease. However, as with all Doppler and echocardiographic measurements, caveats apply to valve area measurements. Specifically, the examination is operator-dependent and, most importantly, flow-dependent (thus, in actuality, left ventricular load- and function-dependent). The methods used to quantitate aortic valve area in the catheterization and echocardiography laboratories are in Appendix 1. Importantly, beyond measuring aortic valve area, clinical evaluation of patients with aortic stenosis should take into account functional status, blood pressure control, volume status, etc. when considering a specific valve area obtained at a single time point. The 2014 guidelines outline a useful division of patients by stages and quantitation of the disease, including the “low-flow/low-gradient” patient (see Table 1).

ROLE OF DOBUTAMINE STRESS TESTING:
Our patient probably fits into the D2 category of low-flow/low-gradient hemodynamics (LFLG) with reduced ejection. Why perform a dobutamine stress test on a patient like ours? Dobutamine stress testing has been used to further classify patients with aortic stenosis who present with LFLG. Recall that the traditional diagnostic criteria for severe aortic stenosis is aortic valve area <1.0 cm², mean transvalvular gradient of at least 40 mm Hg and an aortic valve velocity of 4 m/sec. LFLG aortic stenosis is defined as aortic valve area <1 cm², transvalvular gradient <40 mm Hg and aortic valve velocity <4 m/sec. Importantly, this group is being increasingly recognized and better understood. Although LFLG aortic stenosis patients do indeed have severe aortic stenosis, the flow across the valve is reduced, either because of a small left ventricle (<35 mm/m²) or because of left ventricular...
dysfunction. This flow reduction, in turn, reduces the mean pressure gradient and velocity below the normal diagnostic criteria for LFLG aortic stenosis.

Dobutamine stress testing also can be used to identify another important group of patients with a condition labeled “pseudo-aortic stenosis.” These patients meet traditional criteria but, when challenged with dobutamine, the calculated valve area increases above 1.1 cm², expanding with the augmented flow from the dobutamine. These patients actually have moderate aortic stenosis with reduced flow -- again, either from left ventricular dysfunction or reduced left ventricular size. Postoperative congestive heart failure was comprehensively addressed in a review by Watson and Fleischer in 2008. Common causes of congestive heart failure in this type of patient with severe aortic stenosis who is undergoing a cholecystectomy include: 1) vasodilatory challenge of sepsis with ensuing tachycardia, 2) volume overload, 3) atrial fibrillation, 4) myocardial infarction/ischemia, and 5) pain-induced tachycardia. Of interest is the association of postoperative delirium and exacerbation of congestive heart failure in patients with severe aortic stenosis.

OPTIMAL TIMING OF HIP FRACTURE REPAIR:
Optimal timing of hip fracture repair has been extensively reviewed. In this elderly population, the risks of delay (i.e. urinary tract infection, atelectasis/pneumonia, deep vein thrombosis/pulmonary embolism, and decubitus ulcers) must be balanced against the time for medical evaluation and treatment of comorbidities. Recent literature suggests a delay of 48 hours is not associated with any increase in mortality or morbidity. A retrospective study by Orosz et al. of aggressively timed surgical intervention for hip fractures within 24 hours of admission evaluated functionality, pain and length of stay and found that all of these markers improved with early intervention. However, this study was small and did not adjust for patient comorbidities. In our patient, early intervention is appealing because any delay, with the consequent continued narcotic use, may exacerbate her preexisting dementia. However, in addition to her aortic stenosis, she has evidence of congestive heart failure, new atrial fibrillation and elevated serum creatinine, all of which should be addressed prior to surgery.

Collard et al. and Fuzier et al. both reported successful use of continuous spinal for hip fracture anesthesia in patients with severe aortic stenosis. In an anesthesia-centered review, Istanphalous challenged the dogma of “no spinal for aortic stenosis patients” by pointing out that there are no prospective or randomized trials to evaluate the role of neuraxial blockade for noncardiac surgery patients with aortic stenosis and further noting that regional anesthesia has the benefits of earlier return of bowel function, decreased rate of deep venous thrombosis and decreased blood loss. Eaton used intrathecal sufentanil to good effect in three patients with aortic stenosis having shock wave lithotripsy. Murphy et al. showed that a dose of 0.1 mg of intrathecal morphine sulfate provided the best balance of pain relief and side effects in patients (without aortic stenosis) having hip arthroplasty. These side effects include troublesome nausea, pruritus, urinary retention, respiratory depression and relative hypotension. No patients were excluded because of comorbidities. Sparse
literature exists for using intrathecal morphine in patients like ours with her specific challenging comorbidities. The duration of intrathecal morphine and the potential side effects (including hypotension from vasodilation) last as long as 24 hours. Given the risk of ischemia and worsening perfusion from the hypotension, she may not be a good candidate for a 0.1-mg dose of intrathecal morphine. However, if she has significant hypertension and pain postoperatively (and a continuous spinal technique had been used for the case), a much smaller dose of intrathecal morphine or other shorter-acting narcotic under very close monitoring might be reasonable.

However, any technique in aortic stenosis patients, regional or general, must recognize the grave jeopardy of severe ischemia from low systemic vascular resistance. Collard and Fuzier both reported using intrathecal anesthesia and invasive monitoring successfully in this patient population. The role of intrathecal narcotics in this group merits further discussion.

BAV and TAVR for AS and NCS:
As early as 1989, the Mayo Clinic reported a successful series of patients with severe aortic stenosis who had balloon valvuloplasty (BAV) prior to noncardiac surgery. Considered “too sick” for (or refusing) aortic valve replacement, the 15 patients had immediate preoperative increase in valve area from 0.49 cm$^2$ to 0.85 cm$^2$ and a reduction in the gradient from 58 mm Hg to 32 mm Hg. All the patients subsequently tolerated their noncardiac procedure well. However, the 2014 guidelines for valvular heart disease catalogue the relatively high incidence of complications (10-20% risk of aortic regurgitation, stroke or myocardial infarction). Also, the failure of the BAV to increase valve area above 1 cm$^2$, and subsequent restenosis within 6-12 months, makes BAV a poor long-term solution. BAV is assuming a larger role in scenarios like ours.

Transcatheter aortic valve replacement (TAVR) may play an increasing role in the management of this patient population depending on institutional experience. Patients are now stratified according to the feasibility of iliac access for the CoreValve device (Medtronic Inc., Minneapolis, MN), size of the aortic annulus, and the available commercial approval versus investigative protocols (some of which require two cardiac surgeons to assess the patient). Mobilizing the TAVR team to analyze a patient like ours can be a time-consuming and daunting task. The most common complications of TAVR include aortic regurgitation, iliofemoral injury secondary to the large access catheters required, stroke (embolization from balloon inflations of the calcified valve), aortic rupture, coronary occlusion, valve embolization, and left ventricular rupture. (See www.newheartvalve.com for a complete description of the TAVR procedure, exclusions, complications, etc.; see Appendix 2 for a discussion of what we have learned from our first 400 cases.)

Prior to surgery, an updated quantification of the patient’s current aortic valve area is needed since aortic stenosis progresses over time (average rates of progression are 0.1 cm$^2$/year reduction in aortic valve area, 4-7 mm Hg increase in mean arterial pressure and 0.3 m/sec/year increase in aortic jet velocity). Additional important information for our patient includes functional class, the nature of her
stroke and residual deficit, any evidence of congestive heart failure (i.e. B-type natriuretic peptide level), extent of coronary disease (present in 50% of aortic stenosis patients over age 75), and renal and hepatic function.

This patient’s risk is high according to the revised criteria -- which list six risk factors: serum creatinine >2 mg/dl, ischemic heart disease, congestive heart failure, cerebral vascular disease, insulin-dependent diabetes mellitus, and high-risk surgery -- especially if her new atrial fibrillation is ischemic in origin. Additionally, her pulmonary hypertension places her at increased risk (although pulmonary hypertension is not included in the revised risk criteria, ample literature documents pulmonary hypertension as a significant independent risk factor). A reassessment of the degree of pulmonary hypertension by transthoracic echocardiography is desirable, as the pulmonary artery pressure could be driven higher by fluid overload, hypercarbia, hypoxemia, and/or worsening mitral regurgitation, all of which can easily occur perioperatively.

Older anesthesia studies indicated a markedly elevated risk of complications in patients with aortic stenosis subjected to noncardiac surgery. Those studies do not reflect the current use of arterial lines, echocardiography, or what our specialty has learned from the TAVR experience. Two studies from the modern era indicate that the perioperative risk for patients with aortic stenosis facing noncardiac surgery has declined. A tertiary care hospital series of nonemergency noncardiac surgery in patients with moderate or severe aortic stenosis quantitated the increased risk relative to controls as: 1) 30-day mortality of 2.1% versus 1%, 2) postoperative myocardial infarction 3% versus 1.1%, and 3) primary outcomes (composite of mortality and perioperative myocardial infarction) for severe aortic stenosis of 5.7% versus 2.7%. In 2014 Tashiro et al. published the results of their study of more than 250 patients with severe aortic stenosis who underwent emergency and nonemergency noncardiac surgery at the Mayo Clinic. The authors concluded: 1) the 30-day mortality was not different between the control group and the aortic stenosis group (5.9% vs. 3.19%), 2) the 1-year mortality was, in fact, higher in the aortic stenosis group, 3) the incidence of medical adverse events was higher in the aortic stenosis group (18.8% vs. 10.5% - mainly heart failure), and 4) emergency surgery, atrial fibrillation, and serum creatinine >2 mg/dl were predictors of perioperative mortality, with emergency surgery being the strongest predictor.

The TAVR experience has helped us manage patients with critical aortic stenosis undergoing noncardiac surgery. Preload augmentation prior to any manipulation is key. Afterload and contractility must be scrupulously protected, especially when any vasodilator challenge is involved. Heart rate is maintained in a narrow range of not too fast or too slow.

Lability on induction that occurred in this patient might have been avoided by applying some of the lessons from our TAVR patients. These interventions include augmenting and measuring preload, maintaining critical afterload/systemic vascular resistance, and augmenting left ventricular function with epinephrine or norepinephrine by carefully titrated bolus.
POSTOPERATIVE CARE:
The immediate postoperative volume shifts, changes in vascular resistance from rewarming, pain management, and continued blood loss provide dynamic challenges in the open reduction internal fixation population, who are uniquely intolerant of vasodilation or arrhythmias. They demand intense vigilance to prevent a downward spiral of hypotension, i.e. the consequent ischemia and then further ischemia-induced hypotension. Thus, intensive care unit (ICU) surveillance postoperatively is reasonable. Exactly how long the patient should remain in the ICU is problematic. Congestive failure may develop when the patient’s heart faces the challenge of rehabilitation with the consequent increase in myocardial oxygen consumption. As indicated by Tahsiro and colleagues, the major risk is postoperative congestive heart failure. Heart rate control and avoidance of atrial fibrillation also is critical.

SUMMARY AND CONCLUSIONS:
The anesthetic considerations for patients with severe aortic stenosis facing noncardiac surgery represent a dynamic challenge. The risks have clearly decreased as our understanding of the pathology and manipulation of the physiology have improved. As transcutaneous aortic valve replacement technology and balloon valvuloplasty evolve, the approach to these patients may change. The exploding aging population, with its attendant uptick in aortic stenosis diagnoses, should provide ample opportunities to apply what we learn in the cath lab to the operating room.
Appendix 1

Catheterization laboratory assessment of aortic valve gradient. The peak-to-peak gradient is always less than the instantaneous gradient (i.e. the echo-derived gradient). (Image used with permission from www.e-echocardiography.com and JLS Interactive LLC.)

Echocardiography-derived aortic valve area is shown below. The panel on the left shows that we assume the stroke volume across the left ventricular outflow track is equal to the stroke volume across the aortic valve. Based on that assumption, we can use the continuity equation (right panel) to solve for aortic valve area. (Images used with permission from www.e-echocardiography.com and JLS Interactive LLC.)

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SV_{LVOT} = SV_{AV}
\]

\[
SV_{LVOT} = \frac{Area_{LVOT} \times VTI_{LVOT}}{VTI_{AV}}
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SV_{AV} = AVA \times VTI_{AV}
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Area_{LVOT} \times VTI_{LVOT} = AVA \times VTI_{AV}
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\[
AVA = \frac{Area_{LVOT} \times VTI_{LVOT}}{VTI_{AV}}
\]
Table 1. Stages of Valvular Aortic Stenosis

<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
<th>Valve Anatomy</th>
<th>Valve Hemodynamics</th>
<th>Hemodynamic Consequences</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At risk of AS</td>
<td>• Bicuspid aortic valve (or other congenital valve anomaly)</td>
<td>• Aortic $V_{max} &lt; 2 \text{ m/s}$</td>
<td>• None</td>
<td>• None</td>
</tr>
<tr>
<td>B</td>
<td>Progressive AS</td>
<td>• Mild-to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or • Rheumatic valve changes with commissural fusion</td>
<td>• Mild AS: aortic $V_{max}$ 2.0–2.9 m/s or mean $\Delta P &lt; 20 \text{ mm Hg}$ • Moderate AS: aortic $V_{max}$ 3.0–3.9 m/s or mean $\Delta P 20–39 \text{ mm Hg}$</td>
<td>• Early LV diastolic dysfunction may be present • Normal LV EF</td>
<td>• None</td>
</tr>
<tr>
<td>C</td>
<td>Asymptomatic severe AS</td>
<td>• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>• Aortic $V_{max}$ ≥ 4 m/s or mean $\Delta P ≥ 40 \text{ mm Hg}$ • AVA typically ≤ 1.0 cm$^2$ or (AVA) ≤ 0.6 cm$^2$/m$^2$ • Very severe AS is an aortic $V_{max}$ ≥ 5 m/s or mean $\Delta P ≥ 60 \text{ mm Hg}$</td>
<td>• LV diastolic dysfunction • Mild LV hypertrophy • Normal LV EF</td>
<td>• None: Exercise testing is reasonable to confirm symptom status</td>
</tr>
<tr>
<td>C1</td>
<td>Asymptomatic severe AS</td>
<td>• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>• Aortic $V_{max}$ ≥ 4 m/s or mean $\Delta P ≥ 40 \text{ mm Hg}$ • AVA typically ≤ 1.0 cm$^2$ or (AVA) ≤ 0.6 cm$^2$/m$^2$</td>
<td>• LVEF &lt;50%</td>
<td>• None</td>
</tr>
<tr>
<td>C2</td>
<td>Asymptomatic severe AS with LV dysfunction</td>
<td>• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>• Aortic $V_{max}$ ≥ 4 m/s or mean $\Delta P ≥ 40 \text{ mm Hg}$ • AVA typically ≤ 1.0 cm$^2$ or (AVA) ≤ 0.6 cm$^2$/m$^2$</td>
<td>• LVEF &lt;50%</td>
<td>• None</td>
</tr>
<tr>
<td>D</td>
<td>Symptomatic severe AS</td>
<td>• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>• Aortic $V_{max}$ ≥ 4 m/s or mean $\Delta P ≥ 40 \text{ mm Hg}$ • AVA typically ≤ 1.0 cm$^2$ or (AVA) ≤ 0.6 cm$^2$/m$^2$ but may be larger with mixed AS/AR</td>
<td>• LV diastolic dysfunction • LV hypertrophy • Pulmonary hypertension may be present</td>
<td>• Exertional dysnea or decreased exercise tolerance • Exertional angina • Exertional syncope or presyncope</td>
</tr>
<tr>
<td>D1</td>
<td>Symptomatic severe high-gradient AS</td>
<td>• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening</td>
<td>• Aortic $V_{max}$ ≥ 4 m/s or mean $\Delta P ≥ 40 \text{ mm Hg}$ • AVA typically ≤ 1.0 cm$^2$ or (AVA) ≤ 0.6 cm$^2$/m$^2$ but may be larger with mixed AS/AR</td>
<td>• LV diastolic dysfunction • LV hypertrophy • Pulmonary hypertension may be present</td>
<td>• Exertional dysnea or decreased exercise tolerance • Exertional angina • Exertional syncope or presyncope</td>
</tr>
<tr>
<td>D2</td>
<td>Symptomatic severe low-flow/low-gradient AS with reduced LV EF</td>
<td>• Severe leaflet calcification with severely reduced leaflet motion</td>
<td>• AAVA ≤ 1.0 cm$^2$ with resting aortic $V_{max}$ ≤ 4 m/s or mean $\Delta P &lt; 40 \text{ mm Hg}$ • Dobutamine stress echocardiography shows AAVA ≤ 1.0 cm$^2$ with $V_{max}$ 24 m/s at any flow rate</td>
<td>• LV diastolic dysfunction • LV hypertrophy • LV EF &lt;50%</td>
<td>• HF • Angina • Syncope or presyncope</td>
</tr>
<tr>
<td>D3</td>
<td>Symptomatic severe low-gradient AS with normal LV EF or paradoxical low-flow severe AS</td>
<td>• Severe leaflet calcification with severely reduced leaflet motion</td>
<td>• AAVA ≤ 1.0 cm$^2$ with aortic $V_{max}$ &lt; 4 m/s or mean $\Delta P &lt; 40 \text{ mm Hg}$ • Indexed AAVA ≤ 0.6 cm$^2$/m$^2$ and • Stroke volume index &lt;35 mL/m$^2$ • Measured when patient is normotensive (systolic BP &lt;140 mm Hg)</td>
<td>• Increased LV relative wall thickness • Small LV chamber with low stroke volume • Restrictive diastolic filling • LV EF ≥50%</td>
<td>• HF • Angina • Syncope or presyncope</td>
</tr>
</tbody>
</table>

AR, aortic regurgitation; AS, aortic stenosis; AAVA, aortic valve area; AAVA, aortic valve area indexed to body surface area; BP, blood pressure; HF, heart failure; LV, left ventricular; LV EF, left ventricular ejection fraction; $\Delta P$, pressure gradient; $V_{max}$, maximum aortic velocity. (Reprinted from Nishimura R et al. 2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary. J Am Coll Cardiol 2014;63:2438-88, with permission from Elsevier.)
Appendix 2: Anesthetic Considerations for Percutaneous Transcatheter Aortic Valve Replacement (TAVR)

One of the most dynamic aspects of cardiology/cardiac surgery practice, TAVR offers unique challenges for the anesthesiologist. The three approaches to TAVR are iliofemoral, direct aortic (upper sternotomy), and transapical. All attempt to avoid a full sternotomy and are usually done in a catheterization or so-called “hybrid” laboratory. In each approach, a wire is placed across the aortic valve and a large introducer railroaded over the wire. With echocardiographic and fluoroscopic guidance, the new valve is then positioned in the old calcified one and ballooned into place. TAVR is generally performed on patients who were (once upon a time) considered “too sick” for general anesthesia.

The commonest issues and other lessons derived from our institution’s first 400 TAVR cases include:

1. Prewarming and prehydration are desirable. Cath labs are ice cold, and keeping patients warm is a low priority for everyone except the anesthesiologist. Underbody warming blankets and prewarming the patient may help some, but this is still a significant challenge.

2. Monitoring, arterial and venous access decisions will vary greatly depending on the learning curve of the operators, their experience with the device being employed, and the individual patient’s comorbidities (e.g., active congestive heart failure, peak pulmonary hypertension >60 mm Hg, or concurrent other vascular heart disease). We have learned that a transthoracic echocardiogram immediately preinduction can help with decision-making.

3. Patients require a transvenous pacemaker for two reasons: 1) because of significant incidence of complete heart block, and 2) to use rapid pacing to produce ideal hypotension (mean 50–60 mm Hg) for deploying the balloon and/or the valve. The pacemaker MUST work. We have learned to check its function frequently and to be aware of any migration by fluoroscopy as the patient and C arm are manipulated. The pacemaker function is checked on arrival in the ICU to make sure its position was not changed in transit. “Late” complete heart block up to 3 days after the procedure is not uncommon. Patients who have a permanent pacemaker are the obvious exception.

4. Aortic stenosis patients coming for TAVR now come in a very wide spectrum from easy to very difficult. They may be relatively healthy — the patient with good right and left ventricular function, no other significant comorbidities, and who has a permanent pacemaker and good transthoracic echocardiographic windows and who can be done as a monitored anesthetic represents one end of the spectrum, whereas the patient added on as an urgency on the weekend after hours who is in biventricular failure, already getting supplemental oxygen, has significant mitral/tricuspid disease and significant pulmonary hypertension, and who is getting a new aortic valve via a transapical approach as “last resort” represents the other end. This latter patient will require the entire armamentarium of vasoactive support, nitric oxide or epoprostenol sodium, and postoperative ventilator support.

5. These cases frequently require the removal of arterial and venous access lines, with a 20- to 30-minute “holding” period for safe removal after reversal of heparin. During that time period, the anesthesiologist obviously remains in attendance, planning the “landing.” In a high-volume environment (like ours), the cardiologist/cardiac surgeon are ready for the next case and looking for another anesthesiologist and cath lab crew. Thus, additional anesthesia personnel are airlifted away from the operating room cases.

6. Prior to balloon dilation or valve deployment, the operative team may ask for blood pressure to be reduced (mean of 50–60 mm Hg) through rapid pacing. We have learned that vasoactive support just prior to pacing is necessary/desirable to avoid a postpacing crash. Minidoses of epinephrine (10–20 mcg), phenylephrine (100–200 mcg), and norepinephrine (0.1–0.2 mcg) to get the blood pressure to 110–120 systolic are desirable prior to rapid pacing and may be required post-pacing.

7. Typically, the hypertrophied left ventricle, when relieved of its constricting burden by the new aortic valve, will produce impressive hypertension. Nitride, nitroglycerin, fenoldopam, or nicardipine is required until the patient is warmed and volume resuscitated, just as in any open aortic valve replacement. For the patient having the more painful direct or transapical approach, additional narcotic and/or dexmedetomidine is useful.
References
27. Fleisher LA; American College of Cardiology/American Heart Association: Cardiac risk stratification for noncardiac surgery: update from the American College of Cardiology/American Heart