Cardiac Critical Care After Transcatheter Aortic Valve Replacement

Matthew I. Tomey, MD*, Umesh K. Gidwani, MD*,
Samin K. Sharma, MD

KEYWORDS

• Transcatheter aortic valve replacement • TAVR • Critical care • Complications

KEY POINTS

- Transcatheter aortic valve replacement (TAVR) is a new approach to management of severe aortic stenosis, first described in 2002, that is now available for clinical use in the United States.
- Vulnerability is a defining feature of the initial cohort of patients eligible for TAVR, whose advanced age and comorbidities confer high or extreme surgical risk.
- Patients undergoing TAVR experience acute hemodynamic changes and risk several complications, including hypotension, vascular injury, anemia, stroke, new-onset atrial fibrillation, conduction disturbances and acute kidney injury.
- Critical care after TAVR centers on anticipation, prevention and management of complications. We present our approach to this challenge and identify avenues for future research.

Transcatheter aortic valve replacement (TAVR) has been described as "game changing," providing a novel, less invasive therapeutic option for patients with severe aortic stenosis (AS) whose age and comorbidities make the operative risk of surgical aortic valve replacement (SAVR) high or extreme. Based on the results of the pivotal Placement of Aortic Transcatheter Valves (PARTNER) trial, US Food and Drug Administration (FDA) approval, announcement of coverage by the Centers for Medicare and Medicaid Services in 2012, and after 10 years' experience in Europe, the volume of TAVR procedures in the United States is poised to increase substantially.

Exuberance for adoption of TAVR is balanced by recognition of multisystem complications and a persistently high rate of mortality (24.3% at 1 year and 33.9% at 2 years in PARTNER) only partly attributable to cardiovascular causes.[1] Although this risk reflects, in part, the advanced age and comorbidity that define TAVR candidates, it also highlights the role of care after TAVR, beginning in the intensive care unit (ICU), and our challenge to improve it.

A hybrid of interventional cardiology and cardiac surgery, TAVR demands the collaboration of a heart team, marrying skills from both disciplines, and incorporating multiple additional specialties, including anesthesiology, vascular surgery, and cardiac imaging. This synthesis extends to the ICU, where optimal care after TAVR requires integration of knowledge and experience previously housed separately in cardiothoracic, surgical, medical, and cardiac ICUs. In this respect, care after TAVR epitomizes a paradigm shift in cardiovascular critical care, emphasizing cross-training of

Disclosures: None.
The Zena and Michael A. Wiener Cardiovascular Institute, The Icahn School of Medicine at Mount Sinai, One Gustave L. Levy Place, New York, NY 10029, USA
* Corresponding author. The Zena and Michael A. Wiener Cardiovascular Institute, The Icahn School of Medicine at Mount Sinai, One Gustave L. Levy Place, Box 1030, New York, NY 10029.
E-mail addresses: matthew.tomey@mountsinai.org; umesh.gidwani@mountsinai.org

Cardiol Clin 31 (2013) 607–618
http://dx.doi.org/10.1016/j.ccl.2013.07.006
0733-8651/13/$ – see front matter

physicians and nurses and the management of cardiac and surgical problems in the context of complex medical illness.

The objective of critical care after TAVR is to guide patients, often elderly and ill, through a period of acute hemodynamic changes and potential for multisystem complications to maximize chances for survival and functional recovery. In this review, our approach to this challenge is presented, acknowledging limitations to evidence where they exist to prompt future research.

PREPARATION: UNDERSTANDING THE PATIENTS AND THE PROCEDURE

Patients

Patients undergoing TAVR are characterized by advanced age and prevalent comorbidities, including coronary and peripheral artery disease, lung disease, pulmonary hypertension, and cerebrovascular disease. The PARTNER trial, which provides the basis for the initial rollout of TAVR offers a useful preview of the comorbidities and risk profiles of high-risk and extreme-risk patients presenting for TAVR (**Table 1**).[2,3] Many patients assigned to cohort B were believed to be at extreme risk due to comorbidities not captured by standard risk prediction tools, such as extensive aortic calcification (15.1%), chest wall abnormalities (13.1%), oxygen-dependent respiratory insufficiency (23.5%), or frailty (23.1%).

Table 1
Age, comorbidities and risk profile in the PARTNER trial

	Cohort A (High Risk)	Cohort B (Extreme Risk)
Estimated 30-d mortality risk after SAVR (%)	15	50
Age (y)	83.6 ± 6.8	83.1 ± 8.6
Society of Thoracic Surgeons score	11.2 ± 5.8	11.8 ± 3.3
Logistic euroSCORE	26.4 ± 17.2	29.3 ± 16.5
Coronary artery disease (%)	74.9	67.6
Peripheral artery disease (%)	43.0	30.3
Cerebrovascular disease (%)	29.3	27.4
Chronic obstructive pulmonary disease (%)	43.4	41.3
Pulmonary hypertension (%)	42.4	42.4
New York Heart Association (NYHA) class III/IV symptoms (%)	94.3	92.2

At present, the indication for TAVR is severe valvular AS, which is consequently a defining feature of the initial cohort of patients undergoing TAVR in the United States. Fixed obstruction to left ventricular outflow at the level of the aortic valve impedes forward flow and subjects the left ventricle to chronic pressure overload and variable degrees of hypertrophy, diastolic, and ultimately systolic dysfunction. Although classically characterized by a high transvalvular pressure gradient (mean, >40 mm Hg), severe AS can present with a low gradient in the presence or absence of left ventricular systolic dysfunction. Augmented myocardial oxygen demand increases susceptibility to ischemia and angina. Reduction in cardiac output, highly sensitive to changes in left ventricular preload, can lead to organ hypoperfusion, and symptoms of lightheadedness and syncope. Increased left ventricular end diastolic pressure leads to pulmonary vascular congestion and dyspnea.

As technical comfort with TAVR increases, both clinical trials and off-label indication creep may shift use to comparatively younger, healthier patients or different valvular lesions,[4] altering the makeup of patients presenting to the ICU. The Society of Thoracic Surgeons/American College of Cardiology Transcatheter Valve Therapy Registry, akin to counterparts in Europe,[5] will provide insight into real-world patient selection, device application, and outcomes.

Procedure

Intensivists should become familiar with the TAVR technique, for which a thorough description, exceeding the scope of this review, has been published.[6] Briefly, TAVR entails transcatheter delivery and implantation of a prosthetic valve within the stenotic native valve.

Prosthesis delivery may be antegrade (via the left ventricle) when vascular access is obtained via the left ventricular apex or the systemic venous system with transseptal puncture, or retrograde when vascular access is obtained via arterial puncture. Potential arterial access sites include the femoral, iliac, axillary, and subclavian arteries, as well as the aorta. Access selection depends on arterial calcification and caliber, as informed by preprocedure imaging.

The first generation of valves includes the Edwards SAPIEN Transcatheter Heart Valve (Edwards Lifesciences, Inc, Irvine, CA), consisting of bovine

pericardial tissue with a balloon-expandable stainless steel frame and a polyethylene terephthalate skirt; the related Edwards SAPIEN XT valve, using bovine pericardium and a balloon-expandable cobalt chromium frame; and the Medtronic CoreValve (MCV, Medtronic, Minneapolis, MN), consisting of porcine pericardial tissue with a self-expanding nitinol frame (**Fig. 1**).[7] Several newer valve designs are under investigation to address limitations of first-generation models, in particular, paravalvular aortic regurgitation.

Patients are typically brought to the operating suite in an elective manner, with some having undergone preceding balloon aortic valvuloplasty (BAV) as a temporizing measure before return for TAVR.[8] Preprocedure evaluation includes a battery of testing, including laboratory studies, electrocardiography, chest radiography, echocardiography, coronary angiography, and computed tomography or magnetic resonance angiography.

At a given hospital, TAVR may occur in a catheterization laboratory, surgical operating room, or hybrid of the two, provided capacity to emergently convert to cardiopulmonary bypass is available. The TAVR team involves a team of specialists, including a structural interventionalist and assistants, cardiac surgeon, vascular surgeon, anesthesiologist, cardiac imager, nurses, and technicians.

The procedure is performed under general endotracheal anesthesia. Support lines and tubes typically include a pulmonary arterial catheter, transvenous pacemaker, arterial line, and urinary catheter, along with peripheral venous access. Arterial access may be completely percutaneous or may require cut-down. BAV may be performed before valve delivery. Rapid ventricular pacing is typically performed at the time of valve expansion to minimize cardiac motion and optimize placement. Echocardiography is used during and after TAVR to assess valve positioning and function.

Hemodynamic Changes

Successful replacement of the aortic valve, whether by TAVR or SAVR, yields an acute increase in aortic valve effective orifice area and acute decrease in the mean and peak transaortic pressure gradient. This causes relief of afterload related to left ventricular outflow obstruction but also broader reduction in global left ventricular hemodynamic load, as reflected in significant reduction in valvuloarterial impedance and end-systolic meridional wall stress.[9] The resultant reduction in myocardial oxygen consumption, combined with increased myocardial blood flow, as a result of reduced coronary microvascular compression and increased diastolic perfusion time, contribute to improved myocardial energetics.[10]

Acutely, this hemodynamic change is usually well tolerated, but can be destabilizing. Left ventricular hypertrophy and diastolic dysfunction resulting from compensation for chronic pressure overload can predispose to development of a dynamic intraventricular pressure gradient when valvular obstruction is abruptly relieved, akin to hypertrophic obstructive cardiomyopathy. Patients with small left ventricular end diastolic diameter, high ejection fraction, high ratio of interventricular

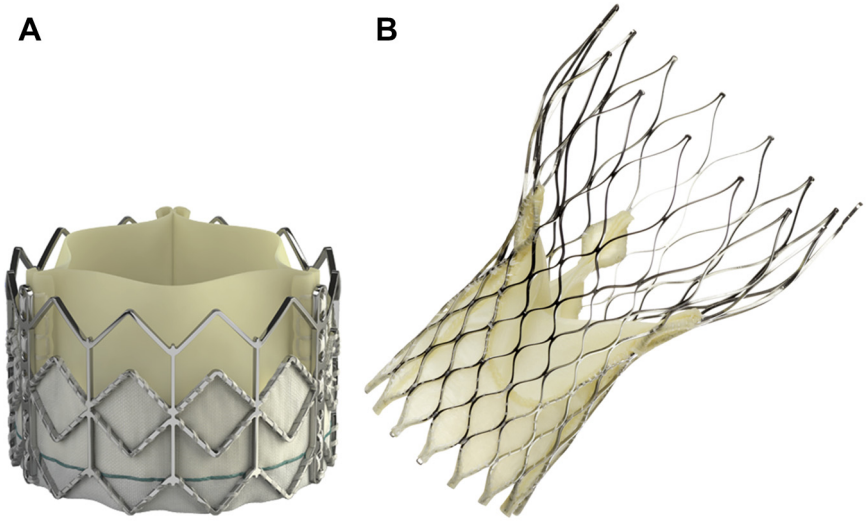

Fig. 1. Transcatheter valves currently in use in the United States. (*A*) Edwards SAPIEN valve (FDA approved). (*B*) Medtronic CoreValve (in phase 3 clinical trials). ([*A*] *Courtesy of* Edwards Lifesciences LLC, Irvine, CA; with permission. [*B*] *Courtesy of* Medtronic, Minneapolis, MN; with permission.)

septum to posterior wall thickness, high valve gradients, and small left ventricular mass are at particular risk.[11] Dubbed "suicide left ventricle,"[12] this phenomenon can lead to hypotension and shock. Endogenous catecholamines and exogenous β-adrenergic agonists exacerbate hypotension by increasing contractility and intraventricular gradients. With volume expansion and temporary use of pure vasopressors, such as phenylephrine or vasopressin, the condition typically resolves over several days.

POSTPROCEDURE COMMUNICATION

Close communication is required between operating and critical care teams (both physicians and nurses) to ensure safe transition of care. Essential elements of handover are listed in **Box 1**. Checklists may be useful to ensure completeness of communication. Whenever possible, ICU staff should meet patients before TAVR to ascertain baseline health status, including neurologic function, and goals of care.

GLOBAL APPROACH TO CRITICAL CARE AFTER TAVR

The role of the intensivist after TAVR is to guide patients through a gauntlet of acute hemodynamic changes and potential complications to optimize the chances for survival and functional recovery. The 3 core elements of post-TAVR critical care are communication, rapid identification and triage of complications, and orderly progression through the ICU.

We advocate a fast-track approach to post-TAVR critical care, paralleling current practice after cardiac surgery (**Fig. 2**). Barring complications, extubation should occur within the first 12 hours after arrival in the ICU, and support lines and tubes should be removed within the first 24 hours. Formal echocardiographic and neurologic assessments are performed 12–24 hours postprocedure. On the second day, diet is advanced and patients are mobilized out of bed and encouraged to ambulate. By the third day, the patient is ready for discharge. To facilitate this, discharge planning should begin by the second ICU day.

Box 1
Key elements of post-TAVR handover

- Preprocedure comorbidities
  - Prior stroke or neurologic deficits
  - Chronic lung disease
  - Coronary artery disease
  - Left ventricular dysfunction
  - Chronic kidney disease
- Periprocedural events
  - Hypotension
  - Arrhythmia and conduction disturbance
  - Fluid management
  - Bleeding and transfusion
  - Vascular access and complications
  - Sedation and paralytics
  - Difficulties with intubation and mechanical ventilation
  - Indwelling lines and tubes
  - Technical outcome, including paravalvular regurgitation and requirement for postdilatation

MANAGING THE VENTILATOR

We advocate a strategy of early extubation, drawing from trials of fast-track care after cardiac surgery. In a Cochrane systematic review of 25 randomized clinical trials comparing fast-track with conventional care, involving 4118 patients undergoing coronary artery bypass graft surgery (CABG), aortic or mitral valve replacement, early extubation was associated with no significant difference in risks of mortality, myocardial infarction, reintubation or major sepsis, but significant reduction in time to extubation (3.0–10.5 hours vs 3.4–35.1 hours) and ICU length of stay.[13] Additional benefits of early extubation include improved patient comfort, reduced endotracheal tube and ventilator complications (such as vocal fold injury, ciliary dysfunction, cough impairment, and ventilator-associated pneumonia), and reduced costs.[14]

Whether fast-track data from the cardiac surgery population can be extrapolated to patients undergoing TAVR is uncertain, as a fast-track strategy in TAVR has not yet been subjected to a randomized trial. TAVR is less invasive than SAVR, minimizing postoperative pain and tissue injury, and avoiding cardiopulmonary bypass and ventricular dysfunction. However, unlike the low-risk to moderate-risk patients in fast-track trials, patients undergoing TAVR are, by definition, high risk, with a high prevalence of coronary artery disease and chronic lung disease. Deleterious effects of withdrawal of mechanical ventilatory support, including increases in the work of breathing and myocardial oxygen consumption,[15] may be magnified in patients undergoing TAVR. Although we believe early extubation is uniformly desirable, timing of extubation in each case must be

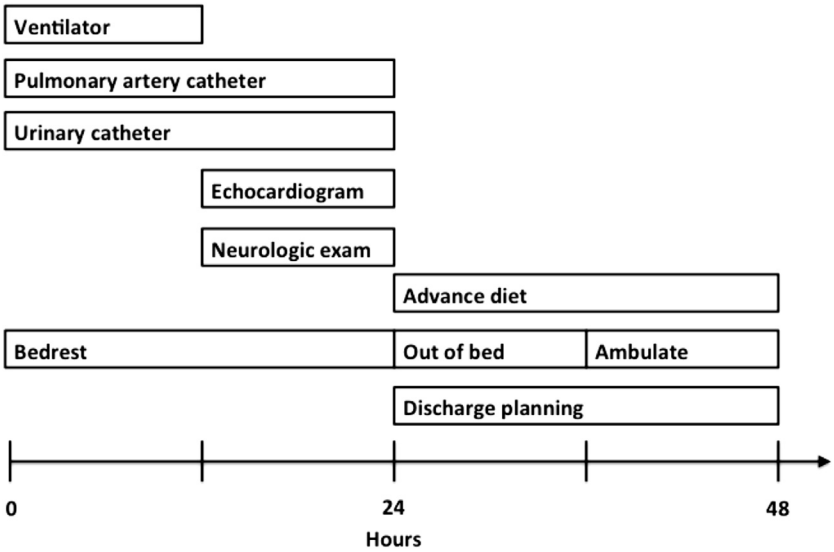

Fig. 2. Post-TAVR care pathway.

individualized. Nurses are integral to this assessment, and protocols for nurse-led early extubation, developed for CABG,[16] may be adaptable to the TAVR setting.

OPTIMIZING NUTRITION

Malnutrition is common among the elderly, and increases in prevalence with increasing frailty and physical dependence.[17] Among adults undergoing cardiac surgery, including aortic valve surgery, surrogate markers for malnutrition, including a low serum albumin level (<2.5 g/dL) and low body mass index (BMI <20 kg/m^2 or <24 kg/m^2), predict postoperative mortality.[18,19] The advanced age, severe AS, multiple comorbidities, and frequent frailty of patients undergoing TAVR suggest that in this highly selected cohort, malnutrition is common. Frailty status, of which malnutrition (measured as serum albumin level) is 1 of 4 components, predicts 1-year mortality after TAVR.[20] It is plausible that malnutrition adversely affects both functional and myocardial recovery after correction of AS.

In the acute care setting, current emphasis is on restarting enteral nutrition as soon as possible after extubation, with a diet tailored to the individual patient. The optimal caloric provision post-TAVR is unknown. Pending further data, a diet containing 120% to 130% of basal energy expenditure (estimated for gender, height, weight, and age), consistent with current practice for patients with congestive heart failure,[21] is reasonable.

Directions for further research include evaluation of the relationship between preprocedure nutritional status (beyond serum albumin and BMI) and outcomes; description of resting energy expenditure after TAVR using the metabolic cart; and trials of nutritional interventions to improve recovery and functional outcomes after TAVR.

ANTICIPATING, PREVENTING, AND MANAGING COMMON COMPLICATIONS

Hypotension

Hypotension is common after TAVR and mandates rapid evaluation and triage to guide management. A high-quality postprocedure handover prepares the intensivist for differential diagnosis of subsequent hypotension by calling attention to potential mechanisms, such as difficult vascular access (a source of bleeding) or significant paravalvular regurgitation requiring postdilatation (a source of aortic injury). When confronted with acute hypotension in the ICU, our approach, illustrated in **Fig. 3**, begins with a focused physical examination, with particular attention to the vascular examination. If the pulmonary arterial catheter has not yet been removed, its data can be useful to corroborate the findings of the physical examination. Complete blood count and arterial blood gas are essential to identify acute anemia and acidosis. Electrocardiography is used to identify new arrhythmia or signs of myocardial infarction. Echocardiography is useful to reassess valvular and biventricular function, as well as to exclude mechanical complications of TAVR, including tamponade, aortic injury, and ventricular septal defect formation.

As an initial management strategy, fluid resuscitation is appropriate for most cases of acute

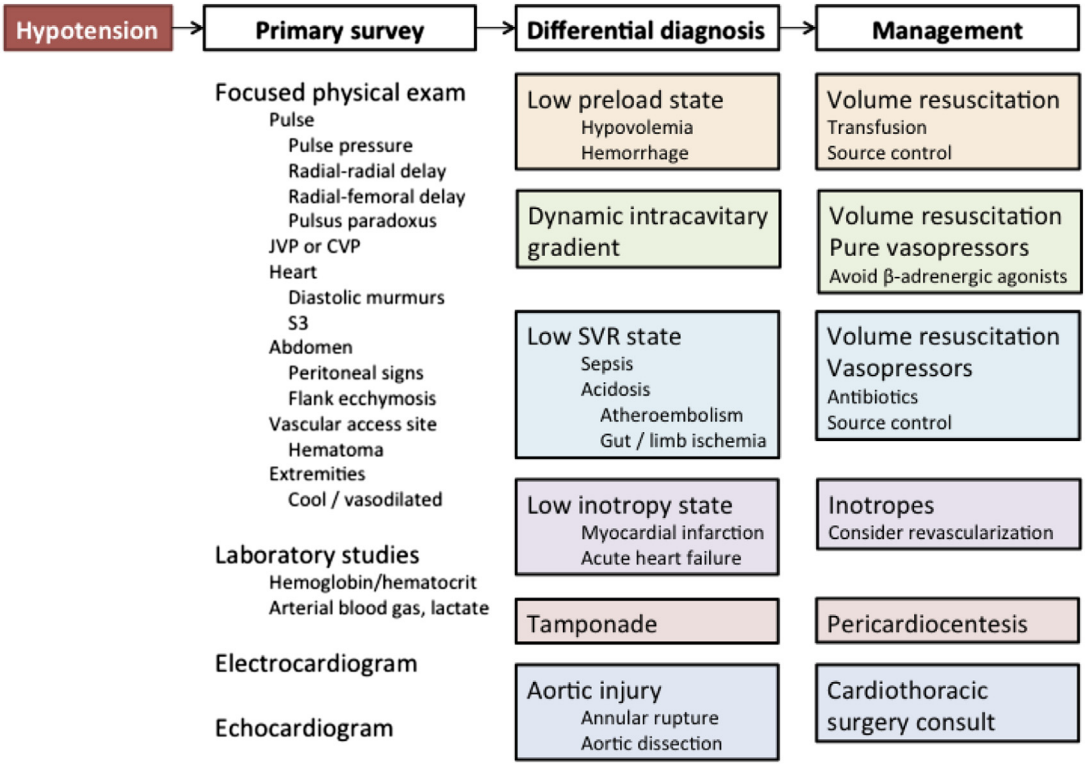

Fig. 3. Approach to hypotension after TAVR.

hypotension after TAVR, coupled with directed management for the source of hypotension (eg, hemostasis for bleeding, revascularization for limb ischemia, antibiotics for infection, pericardiocentesis for tamponade). For patients in whom incipient cardiogenic shock is suspected, as suggested by jugular venous distension, a third heart sound (S3), and cool extremities, invasive hemodynamic monitoring with the pulmonary arterial catheter may be helpful to guide volume management.

Acute Anemia and Vascular Complications

Acute anemia after TAVR represents bleeding until proved otherwise. Bleeding is common; in a series of 943 consecutive patients undergoing TAVR between 2005 and 2011, 20.9% experienced major bleeding and 13.9% had life-threatening bleeding according to Valve Academic Research Consortium (VARC) definitions,[22] of which 23.2% were associated with vascular complications.[23] Furthermore, bleeding is important; requirement for blood transfusion was associated with increased risk of acute kidney injury, major stroke, and mortality at 30 days and 1 year.

We begin our approach to acute anemia with meticulous examination of the vascular access site (**Fig. 4**). Any hematoma should be measured and marked to facilitate future comparison. If hemostasis cannot be achieved with manual compression, vascular surgical consultation is required. If hematoma is stable or absent, attention should turn to occult sources of bleeding, including the abdomen and retroperitoneum. In this scenario, we advocate early use of abdominal computed tomography, ideally without use of intravenous contrast given proximity to TAVR. Retroperitoneal hemorrhage requires emergent vascular surgery and/or interventional radiology consultation.

With first-generation devices and transfemoral access, major vascular complications have been reported in 5% to 23% of cases.[24] Female gender and small arterial caliber, as demonstrated by preprocedure imaging, confer increased risk. Classification of vascular complications after TAVR is outlined in **Table 2**.

In the absence of vascular complications, blood loss may also occur via the gastrointestinal tract and stool should be examined for frank and occult blood. The combination of acquired deficiency of von Willebrand factor and submucosal angiodysplasias in severe AS, known as Heyde syndrome,[25] may render patients particularly susceptible to gastrointestinal hemorrhage. Rapid improvement in hemostatic abnormalities has been observed in patients after both SAVR and

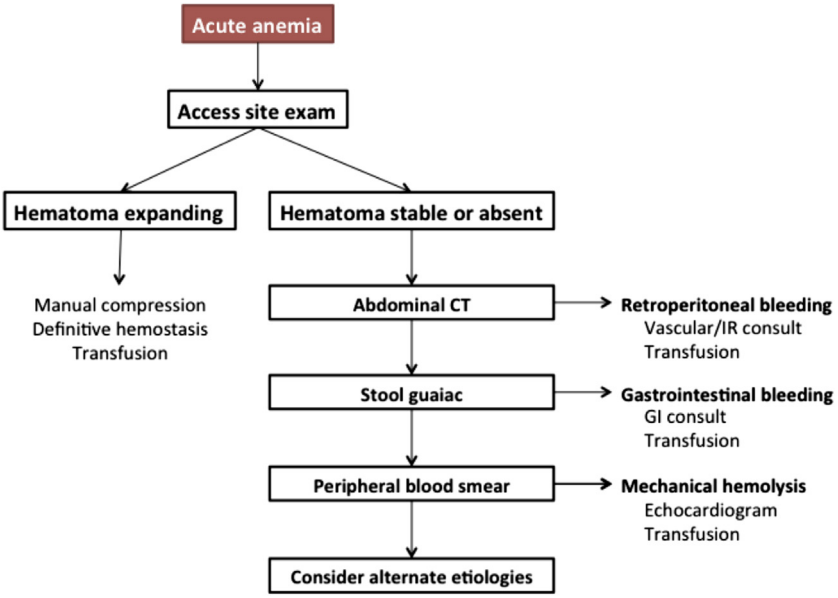

Fig. 4. Approach to acute anemia after TAVR.

BAV,[26,27] and a recent retrospective study suggests that over the long term, TAVR may reduce the risk of recurrent gastrointestinal bleeding.[28] Further research is needed to characterize hemostatic parameters, including von Willebrand factor multimers, after TAVR.

In the absence of evidence of bleeding, alternate causes of anemia should be considered, beginning with examination of the peripheral blood smear. Causes of particular interest for patients undergoing TAVR include disorders of red cell production, including nutritional deficiencies, erythropoietin deficiency (in the setting of comorbid chronic kidney disease), and primary bone marrow disease including myelodysplastic syndromes, as well as disorders of red cell destruction, including mechanical hemolysis (with either native valvular stenosis or prosthetic paravalvular regurgitation). Baseline anemia is common, present in more than 50% of patients before TAVR.

Optimal targets for red blood cell transfusion after TAVR have not been defined. In a recent randomized comparison of liberal versus restrictive transfusion strategies (with hemoglobin cutoffs of 10 g/dL and 8 g/dL, respectively) in elderly patients at high cardiovascular risk undergoing orthopedic surgery, there was no difference in the

Table 2
Vascular complications after TAVR

	Major	Minor
Aortic	Any thoracic aortic dissection	—
Access site or access-related vascular injury[a]	Death	—
	Transfusion >4 units	Transfusion ≥2 but <4 units
	Need for unplanned percutaneous or surgical intervention	Need for compression or thrombin injection therapy
	Irreversible end-organ damage	—
Embolic (noncerebral)	Requiring surgery	Treated by embolectomy/ thrombectomy
	Amputation	—
	Irreversible end-organ damage	—
Other	Left ventricular perforation	—

[a] Examples of vascular injury: dissection, stenosis, perforation, rupture, arteriovenous fistula, pseudoaneurysm, hematoma (including retroperitoneal hemorrhage), irreversible nerve injury, compartment syndrome, failure of percutaneous access site closure device.

primary outcome of death or inability to walk across a room without human assistance at 60-day follow-up.[29] Whether these data can be extrapolated to the TAVR population is uncertain, noting the high prevalence of comorbid lung disease and pulmonary hypertension. Pending randomized study in the TAVR setting, we favor a restrictive approach, targeting a hemoglobin level of 8 g/dL.

Fever and Leukocytosis

Fever and leukocytosis are common after TAVR, but infection is documented in only a few cases. In a single-center experience involving 270 patients (61.6% ES, 38.5% MCV), fever (defined as temperature >37.5°C) was observed in 102 patients (37.8%), of whom only 28 (27.4%) had infections documented, including respiratory (50.5%), urinary (25.0%), access site (14.0%), bacterial endocarditis (3.5%), and bacteremia without overt focus (11.0%).[30]

This infective or inflammatory response is clinically important, with approximately one-third of mortality in the period between 48 hours and 30 days post-TAVR attributed to infection or sepsis in observational series.[31] Why it occurs and how to prevent it are uncertain, and the subject of controversy.[32] Further research is needed to clarify optimal management, including the role of empirical antibiotics. Our approach to evaluation is outlined in **Box 2**.

Box 2
Approach to new-onset fever after TAVR

History, with focus on new localizing symptoms
- Cough
- Dysuria
- Diarrhea
- Abdominal pain
- Access site pain

Physical examination, with particular attention to
- Indwelling support lines and tubes (remove or exchange)
- Access site erythema, warmth, induration, or tenderness
- Abnormalities of pulmonary percussion and auscultation
- New murmurs of valvular regurgitation
- Abdominal or suprapubic tenderness
- Signs of atheroembolism

Laboratory investigation, with a preliminary survey including
- Blood cultures
- Urinalysis and culture
- Chest radiograph

Stroke and New-Onset Atrial Fibrillation

Stroke, as defined by VARC,[22] occurs in approximately 9% of patients after TAVR, of which 5% are major, 2% are minor, and 3% are transient ischemic attacks.[33] Subclinical cerebral infarction is far more common, evident in most patients on post-TAVR magnetic resonance imaging.[34] In PARTNER, stroke was more common with TAVR than with SAVR.[3]

Intraprocedurally, based on study with transcranial Doppler ultrasonography during transapical TAVR, most strokes occur during BAV, catheter manipulation across the stenotic valve, and valve implantation.[35] This finding is consistent with a hypothesis that most strokes result from embolization of atherosclerotic debris from the aorta and aortic valve.[36] Thrombosis and thromboembolism at the time of valve manipulation may also contribute, noting recent data identifying tissue factor,[37] fibrin, and in vivo fibrin clot formation[38] in stenotic aortic valves. Strategies under investigation to reduce intraprocedural stroke risk include different modalities of anticoagulation, minimization of catheter exchanges and valve manipulation (such as by foregoing preparatory BAV before prosthesis implantation),[39] and use of embolic protection devices.[40]

The observation that symptom onset is often delayed more than 24 hours after TAVR, rather than immediately, has heightened interest in the mechanisms and modifiable risk factors for postprocedure stroke.[33] Two potential mechanisms include late embolization of valvular or prosthetic debris and thromboembolism related to valvular injury or atrial fibrillation. Current practice favors an antithrombotic strategy of aspirin (indefinitely) plus clopidogrel (to be continued for 1–6 months), but this approach is incompletely tested, and the primacy of antiplatelet versus anticoagulant therapy is subject to controversy.[41]

Choice of antithrombotic therapy is complicated by atrial fibrillation, which is prevalent at baseline and often new onset in the post-TAVR setting. In a series of 138 patients with no known previous atrial fibrillation, new-onset atrial fibrillation (defined as any episode lasting >30 seconds on electrocardiographic monitoring) was observed in 32% of cases.[42] Predictors included left atrial enlargement and transapical access. The mean

CHADS2 score was 3. As is the case outside the TAVR setting, new-onset atrial fibrillation was an important predictor of stroke and systemic embolism (13.6% vs 3.2% at 1 month, $P = .047$ after adjustment), particularly among those not treated immediately with anticoagulant therapy (40% vs 2.9%, $P = .008$). Although these data would seem to favor timely initiation of anticoagulation for new-onset atrial fibrillation, the addition of anticoagulation to dual antiplatelet therapy (so-called triple therapy) is perilous, associated according to recent data with increased risk of bleeding in patients requiring anticoagulation after coronary stenting,[43] and increased risk of death, stroke, embolism, or major bleeding after TAVR in patients in a German registry.[44]

Pending additional trial data, we currently favor the following strategy for antithrombotic therapy after TAVR. For patients with no indication for oral anticoagulation, we prescribe aspirin 81 mg daily indefinitely and clopidogrel 75 mg daily for 1 month, to be continued for up to 6 months in the absence of major bleeding. For patients with baseline or new-onset atrial fibrillation and no indication for dual antiplatelet therapy, we prescribe aspirin 81 mg daily indefinitely and warfarin, titrated to achieve an international normalized ratio of 2.0 to 3.0. For patients with indications for both dual antiplatelet therapy (eg, recent intracoronary stent placement) and anticoagulation, we prescribe clopidogrel 75 mg daily and warfarin. Additional data are needed to help understand the comparative safety and efficacy of dual antiplatelet therapy versus aspirin monotherapy; antiplatelet therapy versus anticoagulant therapy; novel oral anticoagulants; novel antiplatelets; and left atrial appendage exclusion devices in patients after TAVR.

Conduction Disturbances

Expansion of the new valve in the left ventricular outflow tract, adjacent to the left bundle branch, confers a high risk of left bundle branch block (48% and 21% at 1 month) and permanent pacemaker requirement (18% and 4% at 1 month) with MCV and ES, respectively.[45,46] New conduction defects may not be immediately evident, but may emerge in the initial 24 to 48 hours after TAVR. For patients with risk factors for high-grade atrioventricular block, such as MCV implantation or preexisting right bundle branch block, continuous electrocardiographic monitoring is advised for 72 hours after TAVR,[7] and preemptive permanent pacemaker placement may be considered before TAVR. For patients with new conduction defects immediately after TAVR, the appropriate duration of observation with a temporary venous pacemaker is uncertain before implantation of a permanent device. Better prediction tools are needed to discriminate patients who will recover native conduction. Next-generation models with decreased protrusion into the left ventricular outflow tract are designed to reduce the risk of new conduction defects.

Acute Kidney Injury

Acute kidney injury (AKI) is observed in 7% to 12% of patients after TAVR, with reported risk factors including transfusion, hypertension, chronic lung disease, chronic kidney disease, and higher euroSCORE (European System for Cardiac Operative Risk Evaluation).[7] Key potential mechanisms, which may overlap, include prerenal azotemia and ischemic acute tubular necrosis, as may be seen secondary to intraprocedural hypotension or post-TAVR hypovolemia; atheroembolism; contrast-induced nephropathy; allergic interstitial nephritis; and urinary obstruction. When AKI occurs after TAVR, it is associated with increased length of stay, cost, and mortality.[47,48] Although this risk of adverse events likely reflects, in part, covariance of kidney disease with other risk factors for poor outcomes, including microvascular disease, malnutrition, and frailty, AKI can contribute directly to mortality, accounting for 12.5% of non–cardiac-related deaths after TAVR.[49] The cardiovascular intensivist plays a key role in the prevention, identification, and management of AKI after TAVR.

An optimal strategy for prevention of AKI has not been defined specifically for TAVR, but drawing on experience from percutaneous coronary intervention, we favor a strategy of careful periprocedural hydration, minimization of intravenous contrast, and consideration of *N*-acetylcysteine administration for patients deemed to be at high risk, in particular those with chronic kidney disease.[50]

Surveillance in the ICU setting is facilitated by continuous urine output measurement for the first 24 hours by urinary catheter and daily measurement of blood urea nitrogen, serum creatinine, and electrolytes. The VARC provides a 3-stage classification system for reporting of AKI after TAVR in clinical trials, based on a modification of the RIFLE (Risk, Injury, Failure, Loss, End-stage) classification system.[22] We advocate aggressive investigation for modifiable sources of AKI when serum creatinine increases by more than 50% or 0.3 g/dL greater than baseline or when urine output decreases to less than 0.5 mL/kg/h for 6 hours or more. Basic evaluation should include

exclusion of urinary obstruction (via urinary catheter or renal ultrasonography), urine analysis and examination of the urine sediment, and identification and correction of states contributing to renal hypoperfusion and ischemia. Offending nephrotoxins should be withheld. Care is supportive. When AKI leads to severe acidemia or hyperkalemia, hypervolemia, or uremia refractory to medical therapy, suitable vascular access may be required to initiate temporary dialysis.

SUMMARY

TAVR offers a pathway to functional recovery for patients with symptomatic severe AS whose age and comorbidities put safe SAVR out of reach. This pathway begins in the cardiovascular ICU. Next-generation devices, interventional techniques, and pharmacologic strategies under investigation offer promise to improve outcomes and reduce complications after TAVR, including stroke, bleeding, and vascular injury. Additional key advances will come in the arena of post-TAVR critical care. As a model of acute care for patients with a primary cardiac problem and a background of advanced age and multiple comorbidities, post-TAVR critical care will further provide a training ground for the future of cardiovascular intensive care.

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