Transcatheter aortic valve implantation (TAVI) is becoming a reality in the management of patients with severe aortic stenosis and high or prohibitive risk for standard surgical management. Current understanding of the potential adverse events associated with this procedure is limited. Risks associated with TAVI differ from those related to surgical valve replacement and include vascular injury, stroke, cardiac injury such as heart block, coronary obstruction, and cardiac perforation; paravalvular leak; and valve misplacement. The clinical experience of multiple centers with different valve implantation systems and techniques was reviewed. Awareness of how complications occur might help in their avoidance, recognition, and management. Ultimately, improved understanding of the potential complications associated with TAVI might help improve outcomes and allow wider application of this therapy.

Transcatheter aortic valve implantation (TAVI) is increasingly recognized as a viable therapeutic option for patients with severe, symptomatic aortic stenosis and high risk for conventional surgery. Registries from multiple centers have shown TAVI can be accomplished in selected high-risk patients with outcomes that compare favorably with the outcome of standard valve replacement as predicted by validated operative risk assessment tools (1–6).

Despite being less invasive than open-chest aortic valve replacement, TAVI remains associated with the potential for serious complications. We review the potential complications of TAVI and discuss their prevention, diagnosis, and management.

**Overview of procedure.** Two TAVI systems have seen wide clinical application: the balloon-expandable Edwards valve (Edwards Lifesciences, Irvine, California), and the self-expandable CoreValve ReValving system (CoreValve, Irvine, California). Both systems have been extensively described elsewhere (6–8). Retrograde transarterial or antegrade transapical approaches are currently used to access the aortic valve. Balloon aortic valvuloplasty is performed before valve insertion to facilitate passage of the prosthesis through the stenotic native valve. With the balloon-expandable valve, ventricular burst pacing is used to decrease transvalvular flow and avoid expulsion of the system toward the aorta upon deployment.

**Access and Delivery**

**Arterial injury.** The relatively large diameter of the delivery catheter has been a major limitation of transarterial TAVI. Early systems used 22- to 25-F sheaths (outer diameter 9 to 10 mm), and in the absence of adequate screening the inci-
idence of arterial dissection and perforation was relatively high. Newer low-profile systems (e.g., CoreValve and Edwards NovaFlex) are compatible with smaller 18-F sheaths (outer diameter approximately 7 mm). It is reasonable to assume that the risk of vascular complications is reduced with the use of these lower-profile delivery systems. With technological advances, delivery catheter and sheath size will likely decrease further, which should be associated with further reductions in the risk of vascular injury and less stringent criteria for a transarterial approach.

Angiography and multislice computed tomography are the main imaging modalities used to assess the presence and severity of ilio-femoral disease and determine the feasibility of an arterial approach. Minimal lumen diameter as well as the amount and distribution of atheroma, tortuosity, and calcification will determine the risk for vascular injury related to sheath insertion. Ideally the minimal lumen diameter should exceed the diameter of the delivery system. However, in the absence of extensive calcification, bulky atheroma, or severe tortuosity, short segments of relatively compliant artery 1 to 2 mm smaller in diameter than the intended sheath can often be safely cannulated.

Dissection or perforation of the ilio-femoral arteries might occur in the presence of excessively traumatic sheath insertion (Fig. 1A). Dissection of the ascending or descending aorta can similarly occur due to catheter trauma (Fig. 2) or as an unpredictable complication of aortic valvuloplasty (1). Nonocclusive retrograde arterial dissection will commonly heal once antegrade flow is restored; therefore limited dissections are often best managed conservatively. More extensive arterial dissection can be managed with endovascular stenting although on occasion surgical repair might be necessary.

Vascular perforation leading to retroperitoneal hemorrhage is a more dramatic potential complication of TAVI. Successful management requires a high level of suspicion, should sudden, unexplained hypotension appear. When the large arterial sheath is occlusive, perforation might become evident only after sheath removal. Volume expansion and angiographic assessment should be performed without delay. Immediate reinsertion of the occlusive sheath over a guidewire or placement of a highly compliant occlusion balloon proximal to the area of suspected perforation typically provides rapid and relatively reliable control of bleeding (9), allowing time for definitive management (Figs. 1B and 1C). Covered stents or percutaneous endografts might be adequate, although surgical repair might be necessary. After uncomplicated vascular closure, ilio-femoral angiography performed from the contralateral femoral access site allows rapid identification and, if necessary, management of vascular complications.

An unusual presentation peculiar to a large, occlusive femoral sheath is a tendency for the sheath and endothelium to adhere. Sheath withdrawal is met with resistance and possible complete arterial avulsion and sudden hemorrhage (Fig. 3). The risk of ilio-femoral adherence and

**Figure 1. Vascular Injury**

(A) Dissection of the right iliac artery. (B) Occlusion balloon (Occlusion Catheter, Boston Scientific, Natick, Massachusetts). (C) Occlusion balloon (Coda Occlusion Balloon Catheter, Cook Medical, Inc., Bloomington, Indiana) inflated in the left iliac artery.
avulsion can be minimized with smaller sheaths, periodic sheath rotation, and early sheath removal. When this phenomenon is suspected, pre-emptive placement of an occlusion balloon and preparation for possible surgical repair is prudent.

For patients with unsuitable femoral access, alternatives include the apical, subclavian (10), open iliac or ascending aorta (11) approaches, or reconstruction of ilio-femoral axis with stents or grafts. Although a large experience exists with the apical procedure, experience with the other approaches is limited.

**Apical access issues.** Direct access to the left ventricle (LV) is typically obtained through an intercostal mini-thoracotomy. The risk for lung injury, pneumothorax, or pleural bleeding seems low. Perhaps the most common concern related to the mini-thoracotomy is chest wall discomfort and associated potential for respiratory compromise and prolonged ventilation (12).

Access to the LV cavity is obtained by needle puncture lateral to the apex. The ventriculotomy is dilated to accommodate a large sheath and on completion of the procedure is repaired with pre-inserted pledgeted sutures. Short bursts of rapid ventricular pacing to decrease LV systolic pressure can be helpful during repair. Post-procedural low-grade bleeding from the access site might result in cardiac tamponade and require further repair, whereas management of large tears might require institution of cardiopulmonary support. In rare cases, pseudoaneurysm formation at the site of ventricular repair has been observed weeks to months after TAVI (Fig. 4). Although pseudoaneurysms might be initially asymptomatic, they are typically progressive and might require intervention.

**Stroke.** The most frequent etiology of procedural stroke is likely to be atheroembolism from the ascending aorta or the aortic arch. Other potential causes include calcific embolism from the aortic valve, thromboembolism from catheters, air embolism from LV cannulation, prolonged hypotension, and dissection of arch vessels (13). Balloon dilation of the native valve is typically performed before valve implantation to ease insertion of the crimped prosthesis. Repeated or overly aggressive valvuloplasty might be associated with an increased risk of embolization of calcific material from the aortic valve (14) and should be avoided.

The incidence of stroke varies in the published reports as the consequence of the learning curve, the evolution in technique, and equipment but also the completeness of neurologic assessment. With current devices and experience, stroke rate ranges from 0% to 10% (2,3,5,15,16). Some authors have suggested that stroke risk might be lower with transapical access due to less manipulation in the aortic arch (1,2), but this has not been a universal finding (5,15). In the future, increased procedural experience, less traumatic valve delivery systems, screening for
thick aortic atheroma, and possibly embolic protection devices currently under development might lower the risk of stroke. Procedural anticoagulation to reach a target activated clotting time over 250 s is generally suggested.

The longer-term thromboembolic risk associated with transcatheter valves is currently unknown. Empiric dual oral antiplatelet therapy is generally recommended for 3 to 6 months, followed by long-term daily low-dose aspirin.

**Positioning and Deployment**

**Improper positioning.** An ideal transcatheter aortic prosthesis would restrain the native leaflets and relieve stenosis without unnecessary contact with the surrounding structures. A valve extending excessively into the ventricle or the aorta might be associated with adverse events such as mitral insufficiency, arrhythmias or aortic injury.

Prosthesis embolization immediately after deployment is generally the result of a gross error in positioning or ejection of the device by an effective ventricular contraction during deployment (Fig. 5). Embolization to the aorta is well-tolerated so long as coaxial wire position is maintained, preventing the valve from flipping over to obstruct antegrade flow. Typically the valve can be snared or repositioned with a partially inflated valvuloplasty balloon into a stable position in the aorta. A TAVI reattempt is often successful, although an alternative approach might be advisable when the reason for initial failure cannot be addressed (17). Embolization to the LV is far less likely, but in such cases surgical removal might be the only option (18). The ability to recapture and

**Figure 4. Apical Pseudoaneurysm**

(A) Pseudoaneurysm arising from the left ventricular apex apparent several weeks after a transapical procedure. The **black arrow** indicates the valved stent. (B) Pseudoaneurysm formation after a local wound infection. Ao = aorta; LV = left ventricle; PA = pseudoaneurysm.

**Figure 5. Embolization**

(A) The embolized balloon-expandable valve orientation is maintained by the wire position. (B) The prosthesis is secured in the aorta with no detectable gradient across it.
reposition a valve after deployment would clearly be advantageous, and such prostheses might become available in the upcoming years (19).

**Coronary obstruction.** Coronary obstruction might occur if an obstructive portion of the valve frame (Fig. 6A) or the sealing cuff is placed directly over a coronary ostium; however, this is exceedingly rare. The presence of open cells over a coronary ostium is well-tolerated, but although percutaneous coronary interventions have been performed after valve implantation (20), it is likely that frame struts will prevent or complicate selective coronary cannulation. Of more concern is the possibility of displacing an unusually bulky, calcified native leaflet over a coronary ostium (Fig. 7)(7). Although this might be fatal, some cases have been successfully managed by immediate percutaneous angioplasty or bypass surgery (2). The risk of coronary occlusion is low but difficult to assess and most likely depends on the bulkiness of the native leaflets, height of the coronary ostia, and dimensions of the sinus of Valsalva. Echocardiography, aortog-

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**Figure 6. Coronary Obstruction and Frame Deformation**

(A) Normal flow in the left coronary artery despite the presence of a stent strut at the left main coronary ostium. (B) Oval shape of the transcatheter valve, possibly the result of chest compressions received during a transient hypotension episode.

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**Figure 7. Left Main Obstruction**

(A) Left main coronary artery occlusion resulting from a bulky leaflet displaced over the ostium. (B) Successful percutaneous intervention restored left coronary flow. (C) In a second patient, calcifications from the native aortic leaflet and left main (arrows) are approximated after valve implantation. (D) At autopsy, the leaflet (not the stent itself) seemed to obstruct the ostium.
raphy, and multislice computed tomography have been used to assess these relationships (21). On occasion aortography during aortic valvuloplasty might help clarify potential concerns. At this time no definite criteria exist to exclude patients on the basis of the risk for coronary obstruction, but some have suggested that the coronary ostia should be minimally located 14 mm away from the leaflets insertion.

**Mitral valve injury.** Mitral valve injury was first reported in the setting of the transvenous, transseptal approach to the aortic valve (22). With this approach the delivery system crosses the mitral valve with the potential for temporary mitral incompetence (23) or abrasion and laceration of the anterior mitral leaflet (24). Although this approach is no longer used, mitral injury might still occur. With the antegrade apical approach, a wire can be passed underneath a mitral chordae. Advancing a large catheter over this wire might result in temporary distortion of the mitral valve apparatus or avulsion of a mitral chordae, leading to acute mitral regurgitation. Resistance to catheter advancement through the ventricle or transient mitral regurgitation assessed by transesophageal echocardiography should alert the operator to this possibility.

Rewiring or use of a balloon flotation catheter might be considered to avoid subchordal passage.

The ventricular end of a transcatheter prosthesis can be expected to contact the anterior mitral curtain. A prosthesis extending too far into the LV might interfere with movement of the mitral leaflet and cause acute mitral regurgitation. Surgical removal of such prosthesis might be necessary, although this seems exceedingly rare. The long-term effects of lesser degrees of prosthesis-anterior mitral leaflet contact are unknown, but isolated instances of late mitral valve injury have been documented (25) (Fig. 8).

**Paravalvular regurgitation.** Minor paravalvular regurgitation is ubiquitous with current transcatheter valves, but the incidence of moderate or severe paravalvular leaks was greatly reduced by the routine insertion of prostheses larger than the measured annulus diameter. Initial reports suggested significant paravalvular leak in many patients after implantation of the first-generation balloon-expandable TAVI (22,23), but more recent publications report infrequent cases of moderate or severe paravalvular regurgitation with both systems (3–5).

Mild and even moderate degrees of paravalvular regurgitation seem well-tolerated, and clinically significant

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**Figure 8. Delayed Mitral Valve Injury**

(A) The stent (double arrow) is in contact with the anterior mitral leaflet. Prosthetic valve endocarditis 1 year after implantation associated with perforation of the mitral leaflet at the point of contact (single arrow). (B) Ensuing severe mitral regurgitation. (C) In a second patient prolapse of the anterior mitral leaflet secondary to chordal rupture created (D) severe mitral regurgitation several months after the procedure. Ao = aorta; LA = left atrium; LVOT = left ventricular outflow tract.
hemolysis has not been observed to date (26). However, moderately severe or severe paravalvular, albeit infrequent, is likely to be hemodynamically significant. The initial sign is typically an unexpectedly low aortic diastolic pressure. Rising ventricular filling pressure might lead to myocardial ischemia, ventricular dysfunction, and ultimately shock. The diagnosis is confirmed with aortography or, more reliably, echocardiography. Most likely causes are incorrect positioning, undersizing, or underexpansion. Balloon re-expansion might be helpful in cases of incomplete expansion, whereas a second overlapping prosthesis might be the most effective solution for leaks caused by malposition (Fig. 9). Exclusion of patients with an annulus larger than the largest available prosthesis is prudent to avoid significant paravalvular regurgitation. TAVI in stenotic, congenitally bicuspid valves is possible, but experience is limited.

**Annular and root rupture.** Rupture of the aortic annulus is a rare complication of aortic balloon valvuloplasty (27). Similarly, rare cases of annular or root rupture with subsequent hemodynamic collapse occurred with TAVI. Excessive balloon dilation, aggressive valve oversizing, and extensive annular calcification might increase the likelihood of this uncommon complication. When the annulus and/or subannular tissues are markedly calcified or when the root is unusually small, it seems prudent to be less aggressive with balloon dilation and valve oversizing. Unexplained hypotension after balloon dilation or valve expansion should prompt echocardiographic or angiographic assessment of the LV outflow tract and aortic root. A tear created at the level of the valve inflow can result in either ventricular septal defect (Fig. 10) or LV to left atrial shunt (Fig. 11), whereas aortic root rupture will likely cause massive bleeding and tamponade. Ventricular or root rupture in the setting of TAVI carries a very poor prognosis and is likely to require emergent cardiopulmonary bypass and open surgical repair.

**Cardiac perforation.** Unexplained hypotension should prompt the consideration for cardiac tamponade. With a retrograde approach, wire or catheter-induced LV perforation can cause tamponade. Use of a stiff wire with an appropriately shaped curve and a standard J-curve at the tip is likely the best method to avoid perforation of the LV. Right heart perforation by the transvenous pacemaker is also possible. The reported incidence of tamponade after TAVI varies from 0% to 7% (3–5,16). Typically, pericardiocentesis is adequate; however, thoracotomy might be required.

**Heart block.** Atrioventricular block is a known complication of surgical aortic valve replacement with reported incidence up to 8.2% (28). Not surprisingly, block can also occur with TAVI, presumably as a consequence of the pressure applied on the conducting tissues located

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**Figure 9. Paravalvular Regurgitation**

(A) Self-expanding valve implanted too low, resulting in severe paravalvular regurgitation. (B) A second prosthesis was implanted in the correct position (arrows indicate the distal edge of both prostheses). (C) Mild residual paravalvular leak.

**Figure 10. VSD**

Small tear in the intra-ventricular membranous septum with left to right shunting. LV = left ventricle; RV = right ventricle; VSD = ventricular septal defect.
subendocardially in the LV outflow tract and interventricular septum. In initial reports of TAVI-induced heart block with the 2 currently used systems, permanent pacemaker implantation rate was 7% (29) and 18% (30), respectively. Early experience suggests that prostheses extending farther in the ventricle are associated with a higher incidence of conduction abnormalities, most likely new-onset left bundle branch block (30). Potential risk factors also include aggressive oversizing and the presence of pre-existing infranodal conduction anomalies such as right bundle branch block or second-degree heart block.

Heart block typically manifests immediately after valvuloplasty or valve implantation. Consequently, placement of a temporary pacemaker is desirable during the procedure. In rare cases heart block has appeared days after the procedure; post-procedural monitoring for 48 h has been suggested (30,31).

Other Complications

Arrhythmia. Atrial fibrillation or ventricular ectopy might be precipitated by cardiac manipulation and is often poorly tolerated in the setting of aortic stenosis. Repositioning the ventricular wire is often all that is necessary in cases of frequent ventricular ectopy. Sustained ventricular arrhythmias might occur spontaneously or as a consequence of rapid pacing but generally are responsive to prompt defibrillation; preparatory placement of defibrillator pads is advisable. Timely management of tachyarrhythmias is important to help prevent adverse hemodynamic consequences.

Cardiogenic shock. Patients with severe aortic stenosis often have little myocardial reserve, particularly in the presence of LV dysfunction, hypertrophy, or coronary artery disease. Tachycardia of any cause, including burst pacing, should be minimized, and hypotension should be avoided. Whatever the cause, hypotension or tachycardia might initiate a downward spiral of ischemia and myocardial dysfunction, leading to shock. Vasopressor agents (phenoxyphrine or norepinephrine) to maintain adequate perfusion pressure are often helpful (32), whereas agents with a more pronounced chronotropic or inotropic effect should be avoided when possible. Rarely, temporary femoral cardiopulmonary support might be required, although most often relief of aortic stenosis is associated with prompt improvement of the LV function and improvement of hemodynamic status can be expected. Should chest compressions be required, post-resuscitation evaluation
of the stent position and expansion (Fig. 6B) might be considered.

Acute renal failure. Aortic stenosis is often associated with renal dysfunction due to renal pathology, medications, and low cardiac output. Angiographic contrast injection, hypotension, and atheroembolism might contribute to further reduction of the glomerular filtration rate. However, improved renal perfusion after relief of aortic stenosis has a salutary effect on renal function, and although severe renal dysfunction and dialysis requirement might occur, improvement in renal function is most common.

Structural valve failure. Acute valve failure has been documented very rarely. Potential causes include manufacturing defects, leaflet damage during crimping or implantation and inadequate closing pressure due to abnormal flow characteristics. If structural valve failure is suspected to be the cause for significant valvular regurgitation, implantation of a second valve within the failed valve has been shown to be a successful strategy (1).

Conclusions
Symptomatic aortic stenosis is associated with a dismal prognosis. Any intervention designed to relieve aortic stenosis carries both the potential for benefit and risk. Improved understanding of these potential risks will likely improve the safety and widen the potential application of transcatheter aortic valve replacement.

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REFERENCES


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