Asymptomatic Aortic Stenosis in the Elderly
A Clinical Review

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This article is based on a conference that took place at the Medicine Grand Rounds at Beth Israel Deaconess Medical Center, Boston, Massachusetts, on March 12, 2009.

Case Presentation

Dr Libman

Dr T is an 85-year-old physician with a history of hypertension and aortic stenosis who was in his usual state of health until 2 months ago, when he noticed mild shortness of breath while walking up an incline. Symptoms did not progress and were inconsistent. He was able to walk on level surfaces and pursue his normal daily activities without difficulty. He denied chest pain, orthopnea, paroxysmal nocturnal dyspnea, peripheral edema, lightheadedness, or syncope. He has no history of coronary artery disease. His past medical history is noteworthy for recurrent skin cancers, restless leg syndrome, chronic constipation, and hyperlipidemia.

His current medications include 50 mg of hydrochlorothiazide, 40 mg of lisinopril, 0.25 mg of pramipexole, and 20 mg of simvastatin daily and 50 mg of metoprolol tartrate twice daily.

Dr T is married and lives with his wife. He has 2 grown children. He is a semiretired allergist-immunologist. He has 1 to 2 glasses of wine per week. He has no history of cigarette smoking.

On examination, his blood pressure was 124/78 mm Hg with a regular pulse of 64/min. Jugular venous pressure was 6 cm; carotid arteries were palpable with slightly delayed upstroke and no bruits. His heart beat at a regular rhythm with normal S1, fixed split S2, and no S3 or S4. There was a grade 3/4 systolic ejection murmur, which was loudest at his right upper sternal border radiating toward the carotids. The point of maximal impulse was not displaced.
A transthoracic echocardiograph, performed 2 months prior, showed mild concentric left ventricular (LV) hypertrophy with normal cavity size and normal regional-global systolic function (ejection fraction, >60%; normal ejection fraction, 55%-70%). Peak gradient across the aortic valve was 97 mm Hg (normal peak gradient, <15 mm Hg), and a calculated aortic valve area of 0.7 cm² (normal aortic valve area, >3.0 cm²). There was no aortic regurgitation. There was mild mitral regurgitation. Seven months before, his peak gradient was 67 mm Hg, demonstrating that the severity of aortic stenosis had increased (Figure 1).

A stress nuclear study a month prior was stopped after 6.75 minutes of a modified Bruce protocol due to fatigue and an asymptomatic reduction in blood pressure from 148/84 mm Hg to 122/68 mm Hg. Electrocardiograph showed 2 to 2.5 mm of upsloping-horizontal ST segment depression inferolaterally, which became downsloping in recovery. The nuclear study was interpreted as "probably normal myocardial perfusion."

Dr T was diagnosed with severe aortic stenosis. In light of his ability to function well and in the absence of rest symptoms or reduction in LV function, it was decided to monitor him clinically for the present time.

**Dr T: His View**

I was diagnosed with a heart murmur, which was compatible with aortic stenosis about 8 or 10 years ago. I was asymptomatic at that time. The first symptom I had was this past summer, when I was climbing a steep hill to my summer house. I noticed that I was getting extraordinarily short of breath. I continue, however, to go to the gym at least 3 times a week and work out for 20 to 30 minutes on elliptical machines without symptoms. I talked to my primary care physician who suggested that we get another echocardiograph, which showed severe aortic stenosis.

I was obviously concerned. I live a very active lifestyle for someone who is 85. I see patients on a part time basis, go to school, and live actively. I am concerned that at some point, I will become symptomatic, and then I will have to get a valve replacement. I know that my doctors have suggested watchful waiting, but I am wondering given my age and generally good health whether I should proceed with surgery now.

**Epidemiology and Etiology of Aortic Stenosis**

**Dr Manning** Dr T is a relatively active 85-year-old gentleman who shares a valvular pathology with many older adults. He is fortunate to have lived to an era in which noninvasive imaging technology is readily available both to diagnose and to monitor this common disorder. In addition, highly successful treatment options are widely available. Given the increase in longevity afforded by other advances in health care, degenerative calcified aortic stenosis has now become the most common form of valvular heart disease in the Western World with a prevalence of 3% of those 75 years or older, and representing a major health care burden that is projected to increase with an aging population.1 There is ongoing basic science and clinical research to further our understanding of its pathogenesis and management.2

Although we often think of aortic stenosis as fixed obstructive aortic valve disease, a similar presentation, albeit in a younger population, may occur with supravalvular and subvalvular membranes. These “nonvalvular but fixed” obstructive conditions may present in a similar manner and are readily distinguished by transthoracic echocardiography.

Aortic stenosis is characterized by a progressive narrowing of the aortic valve that leads to a pressure overload state on the LV. Val-
vular aortic stenosis has several etiologies that typically present in different age groups, including unicuspid, bicuspid, and trileaflet aortic stenosis (Figure 2). Rheumatic aortic stenosis leads to leaflet thickening and fusion with a progressively narrow, “oval” residual orifice. Patients with degenerative calcific aortic stenosis are typically older (>70 years), and the residual orifice retains a Y or branch opening configuration. The pathophysiology of degenerative calcific aortic stenosis is not a passive process reflecting the inevitable consequence of aging but is the result of a biologically active process with characteristic features of an osteoblast phenotype. Risk factors for disease progression mirror those of coronary artery disease. Of those risk factors, Dr T has advanced age, hypertension, and hyperlipidemia. Other risk factors include diabetes, smoking, and renal dysfunction. An inflammatory basis for aortic stenosis is supported by studies demonstrating increasing 18-fluorodeoxyglucose levels, a positron emission tomographic ligand marker of macrophage activity, with increasing valve stenosis severity.5

Symptoms and Signs

An increase in the aortic valve gradient results in pressure overload and concentric LV wall thickening or hypertrophy with preservation of LV cavity size and ejection fraction—as was seen in Dr T’s transthoracic echocardiograph. Despite a preserved ejection fraction, subclinical abnormalities of systolic function may be present and identified by assessment of LV strain. The hypertrophy and associated fibrosis likely contributes to LV diastolic dysfunction and increased filling pressure. In a subset of patients with severe aortic stenosis, LV hypertrophy is absent.9

The 3 cardinal symptoms of aortic stenosis indicating a need for mechanical intervention are angina, exertional syncope or presyncope, and heart failure (eg, exertional cardiac dyspnea, orthopnea, paroxysmal nocturnal dyspnea, pedal edema). Heart failure is the result of LV hypertrophy with fibrosis6 and diastolic dysfunction, with increased filling pressure, often despite preserved LV ejection fraction; exertional syncope or presyncope is the result of peripheral vasodilation with an inability of the heart to increase stroke volume; and angina is the result of increased myocardial muscle and increased myocardial demand with limited coronary blood supply without or with obstructive coronary artery disease. Exertional angina may be present in up to 50% of patients with aortic stenosis despite the absence of obstructive coronary artery disease.10

Physical examination findings include a delayed carotid upstroke (pulsus parvus et tardus or slowly rising carotid upstroke; a finding that is often absent in older persons) and a sustained point of maximal impulse due to LV hypertrophy. Auscultation demonstrates a normal or accentuated S1, followed by a harsh, late-peaking systolic murmur (an example of the sound is available at http://www.dundee.ac.uk/medther/Cardiology/as.htm) that is best heard in the right or left upper sternal border and is transmitted to the right clavicular area and carotids (compared with the murmur of mitral regurgitation that is best heard at the left lower sternal border, peaks earlier, and radiates to the axilla and back). As the severity of aortic stenosis progresses, the murmur peaks later in systole or may even decrease in intensity as stroke volume declines. The aortic component of S2 is soft or absent in severe aortic stenosis. Although I have not had the opportunity to examine Dr T, I suspect the aortic valve closure component of S2 was minimal or absent.

Criteria for Clinically Significant Aortic Stenosis

The normal aortic valve has 3 thin, highly mobile leaflets that provide a valve opening of 3 to 4 cm² and minimal gradient (<10 mm Hg). Progression of aortic stenosis in the early stages is very slow,
with estimates of lumen loss of 0.10 cm² per year¹¹ and increase in peak gradient of 10 mm Hg per year,¹² but there is great variation among individual patients. There is considerable valve area reserve. Symptoms of aortic stenosis do not develop until there has been 60% to 75% loss of valve area to less than 1.0 cm². As a result of this large reserve and very slow progression, many patients remain asymptomatic for a decade or more, though a murmur is usually heard when the valve area is 1.5 to 2 cm², a level considered to be mild aortic stenosis. As expected, normalization for body size may remain asymptomatic for a decade or more, though a murmur is usually heard when the valve area is 1.5 to 2 cm², a level considered to be mild aortic stenosis. As expected, normalization for body size may be more meaningful, especially for larger or smaller individuals—with severe aortic stenosis defined as a valve area of less than 1.0 cm² or a body surface area normalized valve area of less than 0.6 cm²/m². Dr T has a valve area of 0.7 cm² (0.35 cm²/m²) placing him in the range of severe aortic stenosis.

### Table 1. Clinical Data From Serial Echocardiographic Studies Performed on Dr T

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Peak velocity, m/s</td>
<td>3.4</td>
<td>3.3</td>
<td>4.1</td>
<td>4.9</td>
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<tr>
<td>Gradient, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Peak</td>
<td>46</td>
<td>44</td>
<td>67</td>
<td>97</td>
</tr>
<tr>
<td>Mean</td>
<td>26</td>
<td></td>
<td>43</td>
<td>68</td>
</tr>
<tr>
<td>Valve area, cm²</td>
<td></td>
<td></td>
<td>1.0</td>
<td>0.9</td>
</tr>
</tbody>
</table>

### Measurement of Aortic Stenosis

Patients with a suspicion of aortic stenosis or with heart failure and exertional syncope will benefit from a resting transthoracic echocardiography, which readily allows for assessment of LV wall thickness (for hypertrophy), cavity size, and regional-global systolic function. In addition, aortic valve morphology (number of leaflets, leaflet thickening, leaflet mobility) can be assessed. Finally, Doppler echocardiography allows for an accurate noninvasive assessment of aortic valve peak and mean gradients, estimation of aortic valve area, and assessment of aortic regurgitation and mitral regurgitation.

Using continuous wave Doppler, the peak and mean gradients across the stenotic aortic valve can be measured. The transthoracic echocardiography peak aortic valve gradient is slightly higher than the gradient reported at cardiac catheterization as the transthoracic echocardiography Doppler gradient measures the maximal instantaneous peak gradient between the aorta and the LV, while invasive measurements report the difference between the peak LV pressure and the peak aortic pressure (Figure 1). The mean gradients derived by both methods are similar.

If Doppler echocardiography is unable to assess the valve, other options include planimetry using moderately invasive 2- or 3-dimensional transesophageal echocardiography, computed tomography,¹³ and cardiovascular magnetic resonance.¹⁴ All of these methods offer good correlation with transthoracic echocardiography and cardiac catheterization but with additional cost, invasiveness, or radiation exposure. They are infrequently needed.

### Medical Management of Significant Aortic Stenosis

Dr T has been monitored for nearly a decade since his initial diagnosis with minimal change in the peak aortic valve gradient from 2003 to 2005 but has experienced marked increase between 2005 and 2008 and again between 2008 and 2009 (Table 1). Progression of aortic stenosis is highly variable, but usually is very slow,¹¹,¹² and there is tremendous reserve so that symptoms may not develop for many decades or until 75% of the valve area has been lost. Although overall progression is approximately 0.10 cm² per year, data suggest that aortic valve calcification is among the strongest predictors of rapid progression.¹²

Medical therapy to prevent progression or complications of aortic stenosis is limited. Retrospective data initially suggested a benefit of statin therapy,¹⁵ in slowing progression, but subsequent prospective studies have demonstrated no benefit for aortic stenosis disease progression (velocity gradient or progression to aortic valve replacement).¹⁶,¹⁷ Prospective studies of statins did demonstrate a decrease in ischemic cardiovascular events,¹⁸ and there is frequent coexistent coronary artery disease. Due to this benefit, I prescribe statin therapy for my patients with aortic stenosis, as was done for Dr T.

Patients with all types of prosthetic valves should receive antibiotic prophylaxis,¹⁸ but current American Heart Association and American College of Cardiology/AHA/ACC prophylaxis recommendations no longer include antibiotic prophylaxis for native valve aortic stenosis (Box 1).¹⁹,²⁰ I advise patients with severe aortic stenosis to stay well hydrated and to avoid hot tubs or baths, steam rooms, and saunas so as to avoid hypotension that may result from peripheral vasodilation with impaired stroke volume augmentation.

For asymptomatic patients with preserved LV ejection fraction, follow-up transthoracic echocardiograms to monitor the gradient and LV systolic function is recommended every 3 to 5 years for mild aortic stenosis, every 1 to 2 years for moderate aortic stenosis, every 6 to 12 months for asymptomatic severe aortic stenosis.¹⁹,²⁰ Transthoracic echocardiography is performed to monitor both the severity of aortic stenosis and associated aortic regurgitation as well as LV ejection fraction. Dr T has had several transthoracic echocardiographies (Table 1) over the years, demonstrating relatively stable peak gradients until last year; with subsequent rapid peak gradient progression from 67 to 97 mm Hg. All of his transthoracic echocardiographies have demonstrated a preserved LV ejection fraction.

### Surgical Treatment of Significant Aortic Stenosis

Class I indications for aortic valve surgery as recommended by both the ACC/AHA and the European Society of Cardiology and European Association for Cardio-Thoracic Surgery (ESC/EACTS) guidelines²⁰,²¹ include symptomatic (heart failure, exertional presyncope or syncope, or exertional angina) patients with severe aortic stenosis (aortic valve area, <1.0 cm² or <0.6 cm²/m²) and asymp-
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Box 1. Recommendations for Patients With Valve Replacements

**Medical therapy**
- Statins
- Endocarditis prophylaxis is not recommended

**Follow-up**
- Asymptomatic patients with preserved LV ejection fraction
- Mild aortic stenosis transthoracic echocardiograph every 3 to 5 years
- Moderate aortic stenosis transthoracic echocardiograph every 1 to 2 years
- Severe aortic stenosis transthoracic echocardiograph every 6 months to a year
- Symptomatic patients and asymptomatic patients with depressed LV ejection fraction are referred for coronary angiography and elective surgery

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Box 2. Recommendations for Surgery

**Surgery**
Nonemergency surgery should be performed 4 to 8 weeks after development of symptoms.

Finally, aortic valve surgery is recommended (class IIa) for asymptomatic patients with mild or greater aortic stenosis at the time of coronary artery bypass graft surgery or other heart valve or thoracic aorta surgery (eg, mitral valve surgery).

**Observation**
In the absence of symptoms and with preserved left ventricle (LV) systolic function, observation with deferral of surgery has an excellent prognosis, especially if the peak aortic stenosis gradient is less than 60 mm Hg.40

Asymptomatic patients with severe aortic stenosis but depressed LV ejection fraction are also class I surgical candidates.29,31

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Table 2. Indications for Aortic Valve Replacement Based on Current Recommendations

<table>
<thead>
<tr>
<th>Indication for Aortic Valve Replacement</th>
<th>ACC/AHA Classa</th>
<th>Level of Evidenceb</th>
<th>ESC/EACTS Classa</th>
<th>Level of Evidenceb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic severe aortic stenosis</td>
<td>I</td>
<td>B</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Severe aortic stenosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Undergoing coronary artery bypass graft surgery</td>
<td>I</td>
<td>C</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Undergoing surgery on the aorta or other heart valves</td>
<td>I</td>
<td>C</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>And left ventricular systolic dysfunction &lt;0.50c</td>
<td>I</td>
<td>C</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Moderate aortic stenosis undergoing coronary artery bypass graft surgery, surgery on the aorta, or surgery on the other heart valve</td>
<td>Ila</td>
<td>B</td>
<td>Ila</td>
<td>C</td>
</tr>
<tr>
<td>Asymptomatic aortic stenosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal response to exercise</td>
<td>Iib</td>
<td>C</td>
<td>Ila</td>
<td>C</td>
</tr>
<tr>
<td>High likelihood of rapid progression</td>
<td>Iib</td>
<td>C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extreme severec</td>
<td>Iib</td>
<td>C</td>
<td>Ila</td>
<td>C</td>
</tr>
</tbody>
</table>

Abbreviations: ACC/AHA, American College of Cardiology and American Heart Association; ESC/EACTS, European Society of Cardiology and European Association for Cardio-Thoracic Surgery.

*a* Not due to another cause.

*b* Based on the ACC/AHA20 and ESC/EACTs21 guidelines.

*c* When the aortic valve is 0.6 cm², the mean gradient is higher than 60 mm Hg, and peak velocity is less than 5 m/s indicates that expected operative mortality is less than 1.0%.

*d* Peak velocity is greater than 5.5 m/s or progression is greater than 0.3 m/s per year.

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Asymptomatic patients with depressed ejection fraction (<50%; Box 2). Of the 3 cardinal symptoms, heart failure is the most ominous with survival of less than a year after heart failure symptoms without valve replacement.22 For patients who present with exertional syncope, survival is less than 2 years after development of syncope without valve replacement (Table 2, Figure 3).

Dr T is largely asymptomatic except for a single episode of dyspnea while walking up a steep incline. He reports being able to perform his usual daily activities, including regular use of elliptical machines without symptoms. Some patients will subconsciously restrict their activity to avoid symptoms. Thus, while they have no symptoms, it is because they have curtailed their activities, often ascribing their diminished activity level to “advancing age.” For patients with equivocal symptoms like Dr T, I often perform a carefully monitored exercise stress test. An abnormal response (found in a third of seemingly asymptomatic patients) includes the development of symptoms or a decrease in blood pressure and defines a high-risk group that is likely to need valve replacement within a year.23 Dr T’s stress test showed a drop in blood pressure and ischemic ST-wave depression suggesting that he is at high risk of mortality (Box 3).

Conventional aortic stenosis treatment options include surgical valve replacement with a mechanical or bioprosthetic valve, a cadaver aortic valve, and the Ross procedure (Table 3). A new option for high-risk patients is transcatheter aortic valve implant.26-27 Both mechanical and bioprosthetic aortic valves give a similar hemodynamic profile. All prosthetic valves increase the risk of endocarditis, so antibiotic prophylaxis is recommended. Mechanical aortic valves are more durable but have higher thrombogenicity, requiring lifelong warfarin (typical target international normalized ratio, 2.0-3.0). Oral warfarin alternatives including dabigatran, rivaroxaban, and apixaban have not been fully assessed for mechanical valves, but adverse events have been reported for dabigatran.28 A prospective trial comparing dabigatran with warfarin in patients with prosthetic valves was prematurely stopped due to increased stroke, myocardial infarction, and mechanical valve thrombi in the dabigatran group.29 Bioprosthetic aortic valves do not require warfarin, but they are less durable,30 especially in younger patients and those with renal dysfunction, failing due to fibrosis or calcification with resultant stenosis, or regurgitation, or both. The longevity of bioprosthetic valves is inversely related to the patient’s age, with a longevity of 1 to 2 decades for patients older than 70 years vs 5 to 10 years for patients younger than 50 years. Thus, otherwise healthy patients younger than 60 years or patients who need long-term warfarin for another condition...
(eg, atrial fibrillation) generally receive mechanical valves, while for those older than 70 years old, such as Dr T, bioprosthetic valves are preferred. These bioprostheses will provide a good hemodynamic result with 15- to 20-year longevity.

In-hospital mortality among patients requiring isolated aortic valve surgery is 2% to 5% and increases to 5% to 10% if coronary artery bypass graft surgery is also needed. Excellent results are obtained even among patients like Dr T who are older than 80 years. Risk factors for in-hospital mortality include hypertension, diabetes, depressed LV ejection fraction, emergency surgery, and prior cardiac surgery. From these data, Dr T has a predicted in-hospital mortality of approximately 3% with excellent long-term survival. For patients who survive without morbidity, the long-term prognosis of isolated aortic valve replacement for aortic stenosis is excellent and approximates the normal population. Adverse long-term risk factors include stroke, smoking, diabetes, and renal failure.

Were Dr T to have numerous comorbidities so as to be at very high risk of cardiac surgery, consideration of transcatheter aortic valve implant would be considered. In the United States, the Edwards Sapien and Medtronic CoreValve options have been the most widely studied. Current data are promising in very high-risk patients with regards to short-term morbidity and mortality with benefit at 2 years for mortality (vs medical therapy). Major advantages of the transcatheter aortic valve implant approach are the rapid patient recovery but with an increased risk of vascular complications, heart block, and stroke. Postprocedure aortic regurgitation appears to be a predictor for adverse outcomes. Current, the Edwards Sapien valve is approved by the US Food and Drug Administration for use in patients with severe symptomatic calcified aortic stenosis at high risk of operative morbidity and mortality. Data suggest similar 30-day and 1-year mortality and major morbidity for the Edwards Sapien and Medtronic CoreValve systems.

Because the presence of angina may be related to aortic stenosis or coexistent coronary artery disease and because patients with aortic stenosis are often older with typical risk factors for coronary artery disease, screening coronary angiography is recommended for most adult patients prior to aortic valve surgery. Data suggest a potential role for coronary computed tomography as a screening test to exclude obstructive coronary artery disease in middle-aged patients. If any stenoses are suggested, a coronary angiogram is performed.

Recommendations for Dr T

Dr T is relatively asymptomatic but has severe aortic stenosis and preserved LV ejection fraction. Although he reports regular exercise on an elliptical, I am particularly concerned by the abnormal hypotensive response on stress testing at a low workload, with accompanying ischemic electrocardiographic changes. Based on this, he has a class IIa recommendation for surgical intervention (Table 2). I recommend that he undergo a coronary angiogram to determine the presence of coexistent coronary artery disease (there is no need to cross the aortic valve to confirm the gradient or to perform left ventriculography) followed by elective aortic valve surgery with a bioprosthetic valve (coronary artery bypass may be necessary for underlying coronary artery disease). Medications may be added before surgery to improve the risk profile.

Table 3. Treatment Options for Aortic Stenosis

<table>
<thead>
<tr>
<th>Option</th>
<th>Target Group</th>
<th>Advantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical treatment</td>
<td>All</td>
<td>Decrease in ischemic cardiovascular events</td>
</tr>
<tr>
<td>Statin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgery options</td>
<td></td>
<td></td>
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<tr>
<td>Valve</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cadaver</td>
<td>Younger adults</td>
<td>No need for warfarin; moderate longevity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>can be used for women of child-bearing age</td>
</tr>
<tr>
<td>Biological</td>
<td>&gt;65 y</td>
<td>No need for warfarin; 15-20 year longevity</td>
</tr>
<tr>
<td>Mechanical</td>
<td>&lt;65 y</td>
<td>Needs warfarin</td>
</tr>
<tr>
<td>Ross procedure&lt;sup&gt;a&lt;/sup&gt;</td>
<td>&lt;40 y</td>
<td>No need for warfarin; moderate longevity</td>
</tr>
<tr>
<td>TAVI</td>
<td>&gt;65 y</td>
<td>Shorter surgical recovery</td>
</tr>
</tbody>
</table>

Abbreviation: TAVI, transcatheter aortic valve implant.

<sup>a</sup> Ross procedure is an operation in which the patient’s native pulmonic valve is moved to the aortic position and a cadaver valve is placed in the pulmonic position.

Figure 3. Algorithm for Management of Severe Aortic Stenosis

Adapted from Bonow et al and Vahanian et al. BP indicates blood pressure and LVEF, left ventricular ejection fraction.

Box 3. American Heart Association Recommendations<sup>*</sup>

I. Evidence or general agreement that a given procedure or treatment is useful and effective.
II. Conflicting evidence or a divergence of opinion about the usefulness or efficacy of a procedure or treatment.
   a. Evidence favors an intervention’s usefulness
   b. Evidence regarding an intervention’s utility is less well established
III. Evidence, general agreement, or both that the procedure or treatment is not useful or effective and in some cases may be harmful

<sup>* Based on Gibbons et al. 24</sup>
A new study by Dr. Manning has been published in the Journal of the American Medical Association (JAMA). The study focuses on the management of asymptomatic aortic stenosis in elderly patients. The study was based on a conference held in March 2009 in Massachusetts.

**Follow-up With Dr T**

Based on the recommendations of Dr. Manning, Dr T decided to undergo surgery. In April 2009, 1 month after the conference, he underwent coronary angiography. The aortic valve was not crossed with a catheter. Angiography demonstrated 90% stenoses of the first and second obtuse marginal arteries. Three weeks later, he underwent uncomplicated aortic valve replacement with a bioprosthetic valve and 2-vessel bypass. He was discharged on postoperative day 6 with home visiting nurse and physical therapy supports.

In 2013, Dr T has just turned 90 years-old and is doing quite well. He remains happy with his decision to proceed with surgery. He continues to be very physically active, exercising on the elliptical machine every other day without any shortness of breath or chest pain. He is currently taking 81 mg of aspirin, 2.5 mg of lisinopril, and 20 mg of simvastatin daily and 100 mg of metoprolol succinate twice a day. He is grateful to Dr Manning for helping him make the decision to proceed with surgery.

**Questions and Discussion**

**QUESTION** He has restless leg syndrome that can be caused by iron deficiency. Do you think he is having intravascular red cell lysis from the deformed valve?

**DR MANNING** Although reported, it is not common to have red cell lysis with native valve aortic stenosis. This condition is more common with prosthetic valve dysfunction either due to a stenosis or a perivalvular leak. Aortic stenosis has been associated with both angiodyplasia and clotting abnormalities (impaired platelet function and decreased levels of von Willebrand factors). 3 9

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Clinical Crossroads Clinical Review & Education

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