Stopping a Silent Killer: Aortic Stenosis

Detection and Evaluation

Its symptoms are quiet and progress late in life, but its effects are deadly. Early detection of aortic stenosis is the best chance for survival.

By Annie Dore, MD, FRCPC

Case

M.P., a 76-year-old man with dyslipidemia and diabetes, recently moved to your region. It is his first visit to your office. He is on glucophage and simvastatin. His blood pressure is 130/85 mmHg and on physical examination you notice a 3/6 systolic murmur in the left parasternal area. It is the first time, according to M.P., that a cardiac murmur is heard. The patient is worried. Should you be worried?

M.P. is an active man who reports dyspnea on exertion for at least two years. He has no chest pain, no orthopnea, and no palpitations. He had a single episode of syncope a month ago while he was climbing stairs with his grocery bags. On physical examination, M.P. has a slow rise in his carotid arterial pulse. He has a loud 3/6 systolic ejection murmur, maximal at the second intercostal space, that radiates to the neck and to the left parasternal area. He also has a fourth heart sound.

See case discussion on page 43.

In this article:

1. What are the symptoms of aortic stenosis?
2. What do examinations tell us?
3. When should patients be referred?
Aortic stenosis is the most common and the most fatal isolated valvular heart lesion in the adult. It results either from fusion and calcification of the commissures in young adults with a bicuspid valve or from degenerative calcific changes in older patients. The functional consequence of aortic stenosis is obstruction to the ejection of blood from the left ventricle, leading to an increase in left ventricular (LV) pressure. LV output is maintained by the development of LV hypertrophy, which may sustain a large pressure gradient across the aortic valve for many years without symptoms, development of ventricular dilatation, or reduction in cardiac output. Patients with aortic stenosis are free of symptoms until relatively late in the course of the disease, but when symptoms develop, the average survival is around two to five years. As the population ages and calcific degenerative changes inevitably occur, detection and evaluation of aortic valvular stenosis become important.

An anatomy refresher

The aortic valve is a three-leaflet, semilunar valve that guards the entrance into the aorta. Its normal area is 3 cm² to 4 cm². Aortic stenosis may occur either on a congenital or an acquired basis.

Congenital valvular stenosis is usually due to a bicuspid aortic valve, which is a frequent cardiac malformation that occurs in approximately 1% of the general population. The commissures can be fused at birth and produce obstruction in infancy, but the majority of patients with a bicuspid aortic valve will develop significant hemodynamic valvular lesion in their fourth or fifth decade of life. Acquired aortic valve stenosis is, however, the most frequent cause of aortic stenosis in adults and results from years of mechanical stress with development of fibrosis, calcifications, and degenerative changes. Otto et al. reported, in a large echocardiographic population-based study of 5,621 adults over age 65, that significant aortic stenosis was present in 2% of this population. They also reported that age-related changes, such as thickening and fibrosis (also called aortic sclerosis), were present in 29%, and were believed to represent an earlier disease process.

What are the symptoms?

Most adults with aortic stenosis have a long latent period without symptoms. In general, the valve orifice must be decreased by 60% to 75% before any significant obstruction is produced and symptoms develop. Adults with a
bicuspid aortic valve develop symptoms in their 40s or 50s, whereas adults with degenerative changes usually present symptoms after age 65. Dyspnea, angina, and syncope represent the classic triad of symptoms of aortic stenosis. Patients may have dyspnea on exertion and subsequently at rest because of the high pressure necessary to fill the hypertrophied left ventricle. Angina pectoris occurs in about two-thirds of the patients and half of them have normal coronary arteries. Syncope typically occurs on exertion secondary to the peripheral vasodilation and the inability to increase the cardiac output in the presence of a fixed obstruction. The development of atrial fibrillation (AF) with the loss of the atrial kick is often associated with worsening of symptoms of congestive heart failure (CHF). Prompt cardioversion is indicated. Sudden death may occur and is probably due to ventricular arrhythmia, which is the first symptom of the disease in 3% to 5% of patients. In general, after the onset of symptoms, the three-year mortality is about 40%, the five-year mortality is 50% and the 10-year mortality is 80%.

What to look for during physical examination

The characteristic arterial carotid pulse in patients with severe stenosis is low in amplitude, with a slow rise and a low-rounded delayed peak (pulsus parvus tardus). The jugular venous pressure is within normal limits. The apex impulse is not enlarged or displaced, but may be sustained due to the forceful systolic impulse of the hypertrophied left ventricle against the chest wall. The aortic component of the second heart sound ($A_2$) is decreased or not audible in the majority of patients with severe obstruction. A fourth heart sound can be heard. The murmur is typically loud, mid-systolic, crescendo-decrescendo maximal in the second intercostal space, and radiates to the carotids and to the left sternal border (Table 1). As the stenosis becomes more severe, the peaking of the loudness of the systolic murmur occurs later in systole (Figure 1). An ejection click can be heard in patients with a bicuspid aortic valve.

Several free Web sites, listed in the Practical Point, have excellent audio documents to help with auscultation skills, visit:

- [www.wilkes.med.ucla.edu/intro.html](http://www.wilkes.med.ucla.edu/intro.html)
ments to help clinicians with their auscultation skills.

**What would laboratory examinations show?**

The electrocardiogram shows a sinus rhythm with LV hypertrophy. On the chest roentgenograph, the heart size is normal, but a prominence of the ascending aorta due to post-stenotic dilatation and calcifications of the valve can be seen.

**Transthoracic echocardiography** is the diagnostic method of choice, as it allows a good visualisation of the valve anatomy and an evaluation of the severity of the stenosis. A stenotic aortic valve is thickened and calcified with a restricted opening. Bidimensional echocardiography is helpful to identify the number of aortic cusps and the presence of LV hypertrophy, as well as to evaluate the LV systolic and diastolic dysfunction. The hemodynamic severity of the stenosis is based on Doppler-derived measurements, such as the mean pressure gradient and the aortic valve area (Table 2).

---

**Table 1**

**Grading heart murmurs**

<table>
<thead>
<tr>
<th>Modality</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barely audible intensity; need special effort</td>
<td>1</td>
</tr>
<tr>
<td>Low intensity murmur</td>
<td>2</td>
</tr>
<tr>
<td>Loud murmur</td>
<td>3</td>
</tr>
<tr>
<td>Loud murmur with palpable thrill</td>
<td>4</td>
</tr>
<tr>
<td>Very loud with palpable thrill (stethoscope not completed on chest)</td>
<td>5</td>
</tr>
<tr>
<td>Loud enough to be heard with stethoscope off the chest</td>
<td>6</td>
</tr>
</tbody>
</table>


---

**Table 2**

**Severity of aortic stenosis**

<table>
<thead>
<tr>
<th>Degree of stenosis</th>
<th>Mean valvular gradient (mmHg)</th>
<th>Valve area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trivial</td>
<td>&lt; 10</td>
<td>&lt; 2</td>
</tr>
<tr>
<td>Mild</td>
<td>10-29</td>
<td>1.5-2</td>
</tr>
<tr>
<td>Moderate</td>
<td>30-49</td>
<td>1-1.5</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt; 50</td>
<td>&lt; 1</td>
</tr>
<tr>
<td>Critical</td>
<td>&gt; 50</td>
<td>&lt; 0.7</td>
</tr>
</tbody>
</table>

Exercise testing is not indicated and may even cause ventricular tachycardia or AF in patients with severe aortic stenosis. Currently, cardiac catheterisation is only performed to confirm diagnosis in patients with difficult transthoracic echocardiograms. Coronary angiography is necessary before surgery in most patients over age 35.

When should you refer?

Once the onset of symptoms occurs (heart failure, angina, and syncope) and the severity of the stenosis is confirmed, patients should be referred promptly for surgery. Asymptomatic adults with severe stenosis should also be referred to determine the timing of surgery. Patients with moderate stenosis should be followed annually for detection of clinical manifestations and echocardiographic evaluation. Patients with moderate or severe stenosis should be advised against sports or activities that require extreme exertion. Vasodilators and diuretics should be used with caution because they can decrease the preload, lower cardiac output, and cause orthostatic hypotension. AF should be cardioverted.

In patients with mild stenosis, echocardiographic evaluation should be performed every two to three years because a gradual increase of 4 mmHg to 8 mmHg per year in Doppler-derived gradients and a decrease of 0.1 cm² per year in valve area have been reported with time. Patients with degenerative changes, but no stenosis (aortic sclerosis), which represent a third of the population over 65, should probably have echocardiographic assessment every five years. Endocarditis prophylaxis is indicated in all patients.

Take-home message

• Symptoms of aortic stenosis, the classic triad of dyspnea, angina, and syncope, appear late in its progression, and once detected, average survival time is short.

• For detection of aortic stenosis, both physical exams (using auscultation skills) and laboratory exams (such as transthoracic echocardiography) are needed.

• It is important to refer patients to a specialist when symptoms occur and severity is established.

• Patients with moderate stenosis should be followed annually.

References