Core Competency Review: Aortic Stenosis and Noncardiac Surgery

Douglas E. Wright, MD, PhD1,2 and Daniel P. Hunt, MD1,2*

1Department of Medicine, Harvard Medical School, Boston, Massachusetts; 2Inpatient Clinician Educator Service, Massachusetts General Hospital, Boston, Massachusetts.

Aortic stenosis (AS) poses a risk of adverse cardiac events for patients undergoing surgical procedures. Perioperative mortality for patients with severe AS is as high as 14%. This review examines the accuracy of the history and physical examination in detecting AS and, subsequently, in assessing severity. The utility of echocardiography is addressed, and the relevant pathophysiology of AS is summarized. We also summarize what is known about perioperative risk for patients with AS. Journal of Hospital Medicine 2012;7:655–660. © 2012 Society of Hospital Medicine

Aortic stenosis (AS) is a common problem among aging patients, who often require surgical procedures. The medical consultant must determine whether the presence of a systolic murmur suggesting AS needs additional evaluation before the patient proceeds to surgery. This decision requires interpretation of cardiac murmurs, and understanding the natural history, pathophysiology, and risks of AS.

PATHOPHYSIOLOGY

Aortic stenosis is a progressive disease that leads to predictable impairment of cardiac responses to physiologic stresses of surgery. AS typically results from degenerative calcification or from a bicuspid aortic valve, both of which cause progressive constriction of left ventricular outflow. The heart compensates by left ventricular hypertrophy. Systolic ejection of blood across the stenotic valve requires more time than normal, leaving less time for diastolic refilling. Left ventricular hypertrophy creates a less compliant left ventricle that becomes dependent on left atrial contraction for optimal filling. Atrial fibrillation with loss of the atrial “kick” is particularly problematic for patients with AS and left ventricular hypertrophy. Thickened myocardium increases myocardial oxygen consumption and impairs myocardial perfusion. Myocardial oxygen demand in the hypertrophied ventricle results from increased systolic pressure on the ventricle, increased systolic contraction time, and increased muscle mass. Reduced capillary density in hypertrophied muscle, and diminished perfusion pressure because of a reduced aortic-coronary pressure differential, impair myocardial perfusion. Shortened diastole allows less blood flow to the myocardium.

At rest, with a controlled heart rate and sinus rhythm to allow for left atrial contraction to enhance left ventricular filling, patients may tolerate significant AS. However, increased heart rate in response to physiologic stress reduces diastolic filling time, diminishes somewhat tenuous diastolic perfusion, and increases afterload.

Additionally, the left ventricle depends on adequate filling pressures; the hypertrophied ventricle is prone to reduced cardiac output because of reductions of preload caused by hypovolemia or venodilation. Venodilation has been a particular concern with epidural anesthesia, although recent studies suggest that this modality can be used safely. Many anesthetic agents reduce systemic blood pressure and thereby reduce the aortic-coronary perfusion pressure gradient leading to reduced coronary blood flow. For surgical patients with significant AS, anesthetic management requires appropriate intravascular volume to optimize preload, heart rate control to allow adequate left ventricular filling along with time for coronary artery flow, and sufficient systemic blood pressure to maintain coronary artery blood flow.

IDENTIFYING AORTIC STENOSIS IN PREOPERATIVE PATIENTS AND JUDGING ITS SEVERITY

Many older patients are found to have a systolic murmur consistent with AS prior to surgery. The first step in evaluation is a detailed history to determine exercise capacity and to elicit any history of chest pain, heart failure symptoms, or syncope. A key question for the medical consultant is whether or not patients should have further evaluation of the murmur prior to surgery, typically starting with transthoracic echocardiography. Table 1 outlines echocardiographic criteria for grading AS severity. The history and physical exam inform the decision of whether to pursue echocardiography. Although it is not clear from the
literature whether identification of AS by echocardiography improves outcomes (this question is unlikely to be addressed by randomized trials), anesthesiologists generally want to know if significant AS is present, as it impacts intraoperative monitoring and management. So the question then becomes the following: Can clinicians reliably exclude moderate–severe AS based on history and a careful cardiovascular exam?

For ruling in severe AS, effort syncope provides the highest positive predictive value; stenosis was found to be severe in all patients with a history of effort syncope in a sample of 67 patients with AS. The presence of a loud, late-peaking systolic murmur or significant delay and decrease in the carotid upstroke, argue for severe AS. Etchells et al developed a simple decision rule for detecting moderate–severe AS (defined as an aortic valve area of 1.2 cm² or less, or a peak transvalvular gradient of 25 mmHg or more), based on a study of 162 inpatients who were examined by a senior medical resident and a general internist. If no murmur was heard over the right clavicle, AS was rare (1/69 [1.4%]; likelihood ratio (LR) 0.10 [95% confidence interval (CI) 0.02–0.44]). If there was a murmur radiating to the right clavicle with 3 to 4 associated findings (reduced second heart sound, reduced carotid volume, slow carotid upstroke, and murmur loudest in the second right intercostal space), moderate–severe AS was common (6/7 [86%]; LR 40 [95% CI 6.6–239]). Absence of radiation of a systolic murmur to the right carotid artery is a useful finding to exclude AS, with a negative likelihood ratio of 0.05 to 0.10.

Although no single physical exam finding or combination of findings can reliably exclude hemodynamically significant AS when a systolic murmur radiates to the right neck, the combination of an early-peaking, soft (grade 2 or less) systolic murmur, normal timing and upstroke of the carotids, and an audible aortic second sound substantially lessen the likelihood of severe AS. A recent study of 376 inpatients who underwent meticulous cardiac examination by a single investigator (blinded to the diagnosis in >96% of cases), followed by echocardiography, provides additional information about the operating characteristics of physical examination in determining the etiology of systolic murmurs. Murmurs heard diagonally across the chest from the right upper sternal border to the apex (“broad apical-base pattern”) predicted increased aortic velocity that would be consistent with AS. Other findings that increased the likelihood of aortic valve disease included delayed carotid upstroke, absent second heart sound (S2), radiation to the clavicles and neck on both sides, and a humming quality to the murmur. This study concluded that the physical examination is not reliable in determining the severity of AS. While generally true, this study actually reveals that any pattern of murmur radiation other than the broad apical-base pattern excluded severe AS entirely among 221 patients with murmurs, and excluded moderate AS in all but 3 of these patients.

A retrospective study of 3997 hip fracture patients evaluated 908 echocardiograms done to investigate cardiac murmurs detected during preoperative assessment. These echocardiograms detected 272 patients with AS that had not been previously diagnosed. Thirty patients had severe AS. Detection of AS prompted changes in anesthesia management. The authors argued for preoperative echocardiograms for all hip fracture patients in whom a murmur is detected.

In summary, no finding by history can exclude AS. However, if the murmur is not heard across the precordium and does not radiate to the clavicle or right neck, severe AS is very unlikely. For patients in whom the murmur suggests the possibility of severe AS, echocardiography is prudent.

**PROGNOSIS OF ADVANCED AS**

Symptomatic AS portends poor prognosis in the absence of aortic valve replacement. In a cohort of patients with severe AS who refused aortic valve replacement (AVR), patients survived a mean of 45 months after onset of angina, 27 months following onset of syncope, and only 11 months after the beginning of left heart failure. Recent studies further define the natural history of severe asymptomatic AS. A study of 128 consecutive patients with asymptomatic severe AS identified by echocardiography found 93% survival at 1 year, 91% at 2 years, and 87% at 4 years, suggesting a relatively benign prognosis. However, many patients developed symptoms during follow-up and required aortic valve replacement. A larger study of 622 asymptomatic AS patients with aortic-jet velocity greater than 4 m/s found that 82% of patients were free of cardiac symptoms after 1 year, but only 33% were free of cardiac symptoms or intervention at 5 years. Patients with asymptomatic, very severe AS, defined as peak aortic-jet velocity of 5.0 m/s or greater have an even worse prognosis with an event-free survival of 12% at 4 years and only 3% at 6 years.

Although short-term (1 to 5 years) prognosis for severe symptomatic AS is poor, and asymptomatic but severe AS also carries substantial risk, the major issue

---

**TABLE 1. Classification of the Severity of Aortic Stenosis in Adults**

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jet velocity (m/s)</td>
<td>&lt;3.0</td>
<td>3.0–4.0</td>
<td>&gt;4.0</td>
</tr>
<tr>
<td>Mean gradient (mmHg)</td>
<td>&lt;25</td>
<td>25–40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Valve area (cm²)</td>
<td>&gt;1.5</td>
<td>1.0–1.5</td>
<td>&lt;1.0</td>
</tr>
<tr>
<td>Valve area index (cm²/m²)</td>
<td></td>
<td>...</td>
<td>&lt;0.6</td>
</tr>
</tbody>
</table>

Note: Adapted from Bonow et al.
for the medical consultant evaluating patients prior to noncardiac surgery is the very short-term perioperative risk imposed by AS. Put simply, will the patient survive surgery and the postoperative period of rehabilitation?

### NONCARDIAC SURGERY AND AS

The evidence that AS increases risk of cardiac complications and cardiac death for patients undergoing noncardiac surgery is limited to retrospective studies. In the early 1960s, a retrospective study of cardiac risk among 766 patients found 10% mortality among 59 patients with an aortic valve abnormality.\(^{16}\) The 15 patients who underwent either intrathoracic or intra-abdominal procedures did particularly poorly, with a mortality of 20%. As part of a large cohort study used to develop the first widely employed cardiac risk index for noncardiac surgery, Goldman et al found 13% (3/23 patients) cardiac mortality among patients with “important valvular AS.”\(^{17}\) In comparison, cardiac mortality among 978 patients without identified AS was 1.6% (16/978 patients).

More recent studies demonstrate lower perioperative mortality for AS patients. These studies are summarized in Table 2. A retrospective chart audit of all patients with AS who underwent noncardiac surgery, in Hamilton, Ontario, Canada between 1992 and 1994, identified 55 patients with a mean aortic valve area of 0.9 cm\(^2\) and compared outcome to that of 55 randomly selected control patients.\(^{18}\) The investigators defined cardiac complications as onset of congestive heart failure, myocardial infarction within 7 postoperative days, dysrhythmias requiring cardioversion, unplanned or prolonged intensive care unit stay resulting from cardiac complications, and cardiac death. Cardiac complications occurred in 5 (9%) patients with AS and 6 (11%) control patients. There was 1 cardiac death among patients with AS.

A retrospective analysis of 108 patients with AS who underwent noncardiac surgery, at Erasmus Medical Center in The Netherlands between 1991 and 2000, provides insight regarding severity of stenosis and perioperative outcomes.\(^{19}\) Cardiac complications (cardiac death or nonfatal myocardial infarction within 30 days of surgery) occurred in 15/108 (14%) patients with AS, with the majority of these complications being cardiac deaths. A control group of 216 patients suffered a cardiac complication rate of 1.8%. Multivariate adjustment for other risk factors demonstrated an odds ratio of 5.2 (95% CI 1.6–17.0) for cardiovascular complication in patients with AS. Moderate AS was associated with 11% complication rate (10/92 patients), while severe stenosis was associated with 31% cardiac complications (5/16 patients). Table 3 summarizes cardiac risk among the patients in this study using the Revised Cardiac Risk Index.\(^{20}\)

In contrast, the Mayo Clinic experience with severe AS (defined as an aortic valve area index <0.5 cm\(^2\)/m\(^2\) or mean transvalvular gradient >50 mmHg) suggested substantially lower complication rates among patients undergoing noncardiac surgery.\(^{21}\) In this series of 19 patients undergoing a variety of surgical procedures between 1988 and 1992, there were no intraoperative events, but 2 (11%) major postoperative events (1 myocardial infarction and 1 death related to multiorgan failure). The authors concluded that selected patients with severe AS could undergo noncardiac surgery with acceptable risk, and speculated that their experience of better outcomes was due to “more aggressive intraoperative and postoperative monitoring and therapy, specifically prompt recognition and therapy of intraoperative hypotension.”

A large database study identified 5149 patients undergoing noncardiac surgery, between 1996 and 2002, with a coexistent AS based on International Classification of Diseases, Ninth Revision (ICD-9) discharge codes, and compared these patients to 10,284 controls.\(^{22}\) Acute myocardial infarction occurred more frequently among patients with AS (3.9% vs 2.0%, \(P < 0.001\)), but in-hospital mortality was not more frequent (5.4% vs 5.7%). The association of perioperative nonfatal myocardial infarction persisted after adjustment for comorbidities. While the results of this study might be interpreted as showing no increase in perioperative mortality for patients with AS who are undergoing noncardiac surgery, there is no way to determine the severity of AS among study patients and endpoints were not uniformly sought, but rather, obtained by ICD-9 reporting. A recent study of 30 patients with asymptomatic but severe AS, who underwent low- or intermediate-risk noncardiac surgery, found that 30% of patients required intraoperative vasoressor use for hypotension, but there were no deaths, arrhythmias, or heart failure events.\(^{23}\)

Summarizing evidence on noncardiac surgery for patients with AS, symptomatic AS is associated with an increased risk of adverse cardiac events in patients undergoing noncardiac surgery. Severe, asymptomatic AS increases risk of intraoperative hemodynamic instability and adverse perioperative cardiac outcomes, although mortality appears to be less than that associated with symptomatic AS.

### ECHOCARDIOGRAPHY PRIOR TO NONCARDIAC SURGERY

There are no studies showing that preoperative echocardiograms lessen the perioperative risk for patients with AS. However, as noted earlier, physical examination alone is not adequate to determine the valvular abnormality causing a systolic murmur in many patients, nor is the exam accurate in determining severity of AS in many patients. Echocardiography clarifies both of these issues. Preoperative echocardiography should inform the approach to anesthesia and, for elective surgical procedures, should allow more accurate assessment of operative risk. Because aortic
<table>
<thead>
<tr>
<th>Study (Year)</th>
<th>Study Type</th>
<th>No. of Patients</th>
<th>Summary of Patients</th>
<th>Outcomes</th>
<th>Other Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>McBrien et al (2009)</td>
<td>Database study of all patients with hip fracture admitted to a single hospital in Belfast, UK, 2001–2005</td>
<td>272</td>
<td>Hip fracture, mild (AVA 1.5–2.0, peak velocity 1.7–2.9 m/sec); 168 patients; moderate (AVA 1.0–1.4, peak velocity 3.0–4.0); 64 patients; severe (AVA &lt;1.0, peak velocity &gt;4.0); 30 patients. Control group without AS 3481 patients</td>
<td>30-day mortality; mild AS, 3.9%; moderate AS, 6.2%; severe AS, 5.1%. Controls, 7.4%</td>
<td>Invasive blood pressure monitoring used more frequently for patients with AS</td>
</tr>
<tr>
<td>Calleja et al (2010)</td>
<td>Retrospective chart review of patients with AS who underwent noncardiac surgery, 1998–2007; compared patients with severe AS to age- and gender-matched controls with lesser AS</td>
<td>30 patients</td>
<td>Severe AS defined as AVA &lt;1.0, peak velocity &gt;40 m/sec. Most surgeries considered intermediate risk</td>
<td>Intraoperative hypotension more common in patients with severe AS (30% vs 17%). Perioperative MI 3% in severe AS and controls; no deaths in patients with severe AS</td>
<td>80% of cases involved general anesthesia; 80% were elective</td>
</tr>
<tr>
<td>Raymer and Yang (1998)</td>
<td>Retrospective chart audit of patients with AS who underwent noncardiac surgery compared to matching controls</td>
<td>55 patients</td>
<td>Mild (AVA 1.0–1.6 cm²): 18 patients; moderate (AVA 0.8–0.9 cm²): 13; severe (AVA &lt;0.8 cm²): 24</td>
<td>5/55 (9%) AS patients experienced postoperative complications (2 heart failure; 1 ventricular fibrillation; 1 MI and CHF; 1 MI, CHF, and death); 6/55 control patients had cardiac complications</td>
<td>Controls and cases not well-matched. Death occurred in 64-year-old patient, with AVA 0.7 cm², undergoing an abdominal aortic aneurysm repair</td>
</tr>
<tr>
<td>Torsher et al (1998)</td>
<td>Retrospective record review of all patients with severe AS (AVA &lt;0.5 cm²/m² body surface area or mean gradient &gt;50 mmHg), undergoing noncardiac surgery at Mayo Clinic, Rochester, MN, 1988–1992</td>
<td>19 patients (28 surgical procedures)</td>
<td>84% of patients were symptomatic, most with dyspnea. Mean AVA for the group was 0.67 cm² with AVA index 0.37 cm²/m²</td>
<td>Intraoperative hypotension requiring vasopressors occurred in 16 procedures among 14 patients</td>
<td></td>
</tr>
<tr>
<td>Kertai et al (2004)</td>
<td>Retrospective study at Erasmus Medical Center, Rotterdam, the Netherlands, of all patients with moderate (mean gradient 25–29 mmHg) or severe (mean gradient &gt;30 mmHg) AS undergoing noncardiac surgery, 1991–2000; compared to controls from the same database</td>
<td>108 patients</td>
<td>92 patients with moderate AS, 16 with severe AS: 36% vascular, 21% orthopedic, 12% abdominal procedures</td>
<td>15 deaths or nonfatal MI among patients with AS (4.4% event rate); 4 events among 216 controls (1.8%)</td>
<td>Patients had higher cardiac risk indicators prior to surgery and were much older than controls. RCRI was predictive of events among patients with AS; RCRI 0 points = 0% rate, 1 point = 10%, 2 points = 16%, 3 points or more = 29%</td>
</tr>
<tr>
<td>Zahid et al (2005)</td>
<td>National Hospital Discharge Survey Database patients diagnosed with AS who underwent noncardiac surgery compared to matched controls without AS, 1996–2002</td>
<td>5149 patients with diagnosis of AS</td>
<td>59.7% low-risk, 35.4% moderate-risk, 4.9% high-risk surgery; 29.6% patients known to have heart failure, 15.0% coronary artery disease</td>
<td>Acute MI 3.9% patients with AS; 2.0% controls. Death 5.4% AS patients vs 5.7% controls</td>
<td>Large database study that does not afford assessment of severity of AS or even echocardiographic confirmation of the diagnosis</td>
</tr>
</tbody>
</table>

**Abbreviations:** AS, aortic stenosis; AVA, aortic valve area; CHF, congestive heart failure; MI, myocardial infarction; RCRI, Revised Cardiac Risk Index.
TABLE 3. Perioperative Mortality and Nonfatal Myocardial Infarction by Revised Cardiac Risk Index Risk Categories

<table>
<thead>
<tr>
<th>RCRI* Risk Indicators</th>
<th>Patients With Aortic Stenosis</th>
<th>Patients Without Aortic Stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0/16 (6%)</td>
<td>0/108 (0%)</td>
</tr>
<tr>
<td>1</td>
<td>3/31 (10%)</td>
<td>2/64 (3%)</td>
</tr>
<tr>
<td>2</td>
<td>6/38 (16%)</td>
<td>1/33 (3%)</td>
</tr>
<tr>
<td>3 or more</td>
<td>6/21 (29%)</td>
<td>1/18 (6%)</td>
</tr>
</tbody>
</table>

NOTE: Adapted from Kertai et al.18 Abbreviations: RCRI, Revised Cardiac Risk Index. * See Lee et al.20

⁎ High-risk surgery, coronary artery disease, history of heart failure, history of cerebrovascular disease, diabetes mellitus treated with insulin, renal insufficiency defined by preoperative serum creatinine >2.0 mg/dL.

Aortic Stenosis typically progresses in a relatively slow and steady fashion, demonstration of mild aortic stenosis by echocardiogram within the preceding few years is considered reassuring.

Emergent surgery (for example, exploratory laparotomy for a ruptured viscus) typically does not allow time for echocardiography prior to the procedure. If a previous echocardiogram is available, this may be useful in deciding the intensity of intraoperative monitoring. However, the presence of a suspicious systolic murmur should prompt careful hemodynamic monitoring and the anesthesiologist should be made aware of the suspicion of AS.

For patients with AS facing urgent surgery (for example, repair of a hip fracture), there is typically time to review previous echocardiograms and, if there has been no recent echocardiogram, it is reasonable to obtain one. The presence of severe AS by echocardiogram should prompt careful hemodynamic monitoring. Some anesthesiologists advocate the use of intraoperative transesophageal echocardiography (TEE) to monitor ventricular filling in patients with severe AS.24-26 Intraoperative TEE provides real-time assessment of the cause of left ventricular dysfunction and allows the anesthesiologist to manipulate hemodynamics to address the dysfunction. Intraoperative TEE prompted significant changes in therapy for 4 of 7 patients with AS in a larger cohort of noncardiac surgical patients monitored with TEE.27 A retrospective study of 123 intraoperative TEE examinations found an impact on management in 81% of patients undergoing noncardiac surgery, although only a small number of these patients had cardiac valvular abnormalities.28 Recent anesthesiology practice guidelines recommend that TEE be considered in patients who have cardiovascular pathology that might result in severe hemodynamic, pulmonary, or neurologic compromise.29 The anesthesiologist should decide potential utility of intraoperative TEE, but it is important that the consulting hospitalist be aware of this possible approach to hemodynamic monitoring. Intraoperative TEE requires specialized expertise and may not available in many hospitals.

For elective surgery, presence of a murmur suggestive of significant AS mandates echocardiography, unless there are study results available from the preceding year.30 Optimally, symptomatic AS should be addressed by aortic valve replacement prior to noncardiac surgery. For patients requiring semi-urgent surgery but are deteriorating because of severe AS, temporizing percutaneous balloon valvuloplasty can be considered, but there are limited data and serious complication rates can be high.31-33 Among 15 AS patients requiring noncardiac surgery but with a contraindication to valve replacement, 3 experienced ventricular perforation during percutaneous balloon valvuloplasty, with 1 death.31 In another series of 7 patients, there were no complications of the valvuloplasties, and all 7 patients underwent uncomplicated noncardiac surgery under general anesthesia thereafter.33

In the absence of interventions to improve cardiac hemodynamics, patients could proceed to necessary noncardiac surgery, understanding the high risk of mortality and morbidity (Table 2). These patients should have careful perioperative hemodynamic monitoring and could be considered for intraoperative TEE if available.

Patients with asymptomatic but severe AS can proceed to low- or moderate-risk surgical procedures without further intervention, but with appropriate hemodynamic monitoring. Those patients with asymptomatic but severe AS needing high-risk surgery should consider valve replacement prior to surgery. In addition, we believe most patients with severe AS should have a cardiologist involved in their perioperative care.

CONCLUSIONS

In summary, patients with suspected AS who require noncardiac surgery need thoughtful consideration by the medical consultant. Careful cardiac examination should be performed on all patients prior to noncardiac surgery. If there is no precordial murmur radiating to the right carotid artery or right clavicle, and if there are no other signs (eg, delayed or reduced carotid upstroke, or absent or distant second heart sound) or symptoms (eg, history of angina, congestive heart failure, or exertional syncope or presyncope), then echocardiography performed for the purpose of discovering AS is not necessary. The majority of patients with a suggestive systolic murmur should be evaluated with echocardiography to provide more accurate prognostic estimates and to guide hemodynamic management during the operation. Patients with severe symptomatic AS are at particularly high risk of cardiac complications, and aortic valve replacement should take priority if the noncardiac surgery can be delayed.

Acknowledgement

The authors would like to acknowledge Dr. Jason Qu for his advice on intraoperative TEE.

Disclosure: Nothing to report.
REFERENCES


