Year 2003 Paper two: Questions supplied by Tricia

**Question 46**

A 45-year-old asymptomatic man returns for follow-up. He was diagnosed 10 years ago with aortic regurgitation due to a congenital bicuspid aortic valve. He has never had endocarditis.

Which one of the following echocardiographic profiles most strongly indicates the need for aortic valve replacement?

<table>
<thead>
<tr>
<th></th>
<th>LVEDD (mm)</th>
<th>FS</th>
<th>LA size (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.</td>
<td>70</td>
<td>0.30</td>
<td>60</td>
</tr>
<tr>
<td>B.</td>
<td>75</td>
<td>0.40</td>
<td>40</td>
</tr>
<tr>
<td>C.</td>
<td>70</td>
<td>0.25</td>
<td>45</td>
</tr>
<tr>
<td>D.</td>
<td>65</td>
<td>0.45</td>
<td>50</td>
</tr>
<tr>
<td>E.</td>
<td>75</td>
<td>0.35</td>
<td>55</td>
</tr>
</tbody>
</table>

LVEDD Left ventricular end-diastolic diameter  
LVESD Left ventricular end-systolic diameter  
FS Fractional shortening = (LVEDD-LVESD) / LVEDD  
LA Left atrial

Answer: C as it fulfills the criteria for class I AVR replacement guidelines EF 0.5 or less

What you need to know

1. Ejection fraction and how to calculate  
2. Aortic regurgitation and indications for surgery

Stroke volume (SV) = Left ventricular end-diastolic volume – left ventricular end-systolic volume  
Left ventricular ejection fraction % = SV / LV end-diastolic volume * 100

So fractional shortening is the equivalent of ejection fraction and the lower the greater indication for surgery as below

From: ACC/AHA 2006 guidelines for the management of patients with valvular heart disease  

These are the current recommendations for AVR

**Class I**

1. **AVR is indicated for symptomatic patients with severe AR irrespective of LV systolic function. (Level of Evidence: B)**

2. **AVR is indicated for asymptomatic patients with chronic severe AR and LV systolic dysfunction (ejection fraction 0.50 or less) at rest. (Level of Evidence: B)**

3. **AVR is indicated for patients with chronic severe AR while undergoing CABG or surgery on the aorta or other heart valves. (Level of Evidence: C)**

**Class IIa**

AVR is reasonable for asymptomatic patients with severe AR with normal LV systolic function (ejection fraction greater than 0.50) but with severe LV dilatation (end-diastolic dimension greater than 75 mm or end-systolic dimension greater than 55 mm).* (Level of Evidence: B)

**Class IIb**
Year 2003 Paper two: Questions supplied by Tricia

1 AVR may be considered in patients with moderate AR while undergoing surgery on the ascending aorta. *(Level of Evidence: C)*

2 AVR may be considered in patients with moderate AR while undergoing CABG. *(Level of Evidence: C)*

3 AVR may be considered for asymptomatic patients with severe AR and normal LV systolic function at rest (ejection fraction greater than 0.50) when the degree of LV dilatation exceeds an end-diastolic dimension of 70 mm or end-systolic dimension of 50 mm, when there is evidence of progressive LV dilatation, declining exercise tolerance, or abnormal hemodynamic responses to exercise.* *(Level of Evidence: C)*

Class III

AVR is not indicated for asymptomatic patients with mild, moderate, or severe AR and normal LV systolic function at rest (ejection fraction greater than 0.50) when degree of dilatation is not moderate or severe (end-diastolic dimension less than 70 mm, end-systolic dimension less than 50 mm).* *(Level of Evidence: B)*

Here are the rest of the guidelines

Aortic Regurgitation

Etiology

Common causes of AR

- idiopathic dilatation of the aorta
- congenital abnormalities of the aortic valve (most notably bicuspid valves)
- calcific degeneration,
- rheumatic disease
- infective endocarditis
- systemic hypertension
- myxomatous degeneration
- dissection of the ascending aorta
- Marfan syndrome.

Less common causes include

- traumatic injuries to the aortic valve
- ankylosing spondylitis
- syphilitic aortitis
- rheumatoid arthritis
- osteogenesis imperfect
- giant cell aortitis
- Ehlers-Danlos syndrome
- Reiter’s syndrome
- discrete subaortic stenosis
- ventricular septal defects with prolapse of an aortic cusp
- anorectic drugs have also been reported to cause AR

The majority of these lesions produce chronic AR with slow, insidious LV dilatation and a prolonged asymptomatic phase

Acute causes
Acute Aortic Regurgitation

Pathophysiology

In acute severe AR, the sudden large regurgitant volume is imposed on a left ventricle of normal size that has not had time to accommodate the volume overload. With an abrupt increase in end-diastolic volume, the ventricle operates on the steep portion of a normal diastolic pressure-volume relationship, and LV end-diastolic and left atrial pressures may increase rapidly and dramatically. The Frank-Starling mechanism is used, but the inability of the ventricle to develop compensatory chamber dilatation acutely results in a decrease in forward stroke volume. Although tachycardia develops as a compensatory mechanism to maintain cardiac output, this is often insufficient. Hence, patients frequently present with pulmonary edema or cardiogenic shock.

Acute AR creates especially marked hemodynamic changes in patients with pre-existing pressure overload hypertrophy, in whom the small, noncompliant LV cavity is set on an even steeper diastolic pressure-volume relationship and has reduced preload reserve.

Examples of this latter situation include aortic dissection in patients with systemic hypertension, infective endocarditis in patients with pre-existing AS, and acute regurgitation after balloon valvotomy or surgical commissurotomy for congenital AS.

Patients may also present with signs and symptoms of myocardial ischemia.

As the LV end-diastolic pressure approaches the diastolic aortic and coronary artery pressures, myocardial perfusion pressure in the subendocardium is diminished. LV dilation and thinning of the LV wall result in increased afterload, and this combines with tachycardia to increase myocardial oxygen demand. Therefore, ischemia and its consequences, including sudden death, occur commonly in acute severe AR.

Diagnosis

Many of the characteristic physical findings of chronic AR are modified or absent when valvular regurgitation is acute, which can lead to underestimation of its severity.

- LV size may be normal on physical examination
- cardiomegaly may be absent on chest X-ray
- Pulse pressure may not be increased because systolic pressure is reduced and the aortic diastolic pressure equilibrates with the elevated LV diastolic pressure
- Because this diastolic pressure equilibration between aorta and ventricle can occur before the end of diastole, the diastolic murmur may be short and/or soft and therefore poorly heard
- The elevated LV diastolic pressure can close the MV prematurely, reducing the intensity of the first heart sound. An apical diastolic rumble can be present, but it is usually brief and without presystolic accentuation. Tachycardia is invariably present.

Echocardiography is indispensable in confirming the presence and severity of the valvular regurgitation, determining its cause, estimating the degree of pulmonary hypertension (if TR is present), and determining whether there is rapid equilibration of aortic and LV diastolic pressure. Evidence for rapid pressure equilibration includes a short AR diastolic half-time (less than 300 ms), a short mitral deceleration time (less than 150 ms), or premature closure of the MV.
Acute AR caused by aortic root dissection is a surgical emergency that requires particularly prompt identification and management.

- Transesophageal echocardiography is indicated when aortic dissection is suspected
- Computed tomographic imaging or magnetic resonance imaging should be performed if this will lead to a more rapid diagnosis than can be achieved by transesophageal echocardiography
- Cardiac catheterization, aortography, and coronary angiography are rarely required, are associated with increased risk, and might delay urgent surgery unnecessarily
- Angiography should be considered only when the diagnosis cannot be determined by noninvasive imaging and when patients have known CAD, especially those with previous CABG (see Section 10.2).

### Treatment

Death due to pulmonary edema, ventricular arrhythmias, electromechanical dissociation, or circulatory collapse is common in acute severe AR, even with intensive medical management. Urgent surgical intervention is recommended. Nitroprusside, and possibly inotropic agents such as dopamine or dobutamine to augment forward flow and reduce LV end-diastolic pressure, may be helpful to manage the patient temporarily before surgery. Intra-aortic balloon counterpulsation is contraindicated. Although beta blockers are often used in treating aortic dissection, these agents should be used very cautiously, if at all, in the setting of acute AR because they will block the compensatory tachycardia. In patients with acute severe AR resulting from infective endocarditis, surgery should not be delayed, especially if there is hypotension, pulmonary edema, or evidence of low output. In patients with mild acute AR, antibiotic treatment may be all that is necessary if the patient is hemodynamically stable. Exceptions to this latter recommendation are discussed in Section 4.6.1.

### Chronic Aortic Regurgitation

#### Pathophysiology

The left ventricle responds to the volume load of chronic AR with a series of compensatory mechanisms, including an increase in end-diastolic volume, an increase in chamber compliance that accommodates the increased volume without an increase in filling pressures, and a combination of eccentric and concentric hypertrophy. The greater diastolic volume permits the ventricle to eject a large total stroke volume to maintain forward stroke volume in the normal range. This is accomplished through rearrangement of myocardial fibers with the addition of new sarcomeres and development of eccentric LV hypertrophy.

As a result, preload at the sarcomere level remains normal or near normal, and the ventricle retains its preload reserve. The enhanced total stroke volume is achieved through normal performance of each contractile unit along the enlarged circumference. Thus, LV ejection performance is normal, and ejection phase indexes such as ejection fraction and fractional shortening remain in the normal range.

However, the enlarged chamber size, with the associated increase in systolic wall stress, also results in an increase in LV afterload and is a stimulus for further hypertrophy.

Thus, AR represents a condition of combined volume overload and pressure overload. As the disease progresses, recruitment of preload reserve and compensatory hypertrophy permit the ventricle to maintain normal ejection performance despite the elevated afterload.

The majority of patients remain asymptomatic throughout this compensated phase, which may last for decades. Vasodilator therapy has the potential to reduce the hemodynamic burden in such patients.

In a large subset of patients, the balance between afterload excess, preload reserve, and hypertrophy cannot be maintained indefinitely. Preload reserve may be exhausted and/or the hypertrophic response may be inadequate so that further increases in afterload result in a reduction in ejection fraction, first into the low normal range and then below normal. Impaired myocardial contractility may also contribute to this process. Patients often develop
dyspnea at this point in the natural history. In addition, diminished coronary flow reserve in the hypertrophied myocardium may result in exertional angina.

LV systolic dysfunction (defined as an ejection fraction below normal at rest) is initially a reversible phenomenon related predominantly to afterload excess, and full recovery of LV size and function is possible with AVR. With time, during which the ventricle develops progressive chamber enlargement and a more spherical geometry, depressed myocardial contractility predominates over excessive loading as the cause of progressive systolic dysfunction. This can progress to the extent that the full benefit of surgical correction of the regurgitant lesion, in terms of recovery of LV function and improved survival, can no longer be achieved.

A large number of studies have identified LV systolic function and end-systolic size as the most important determinants of survival and postoperative LV function in patients undergoing AVR for chronic AR.

**Diagnosis and Initial Evaluation**

Class I

1. Echocardiography is indicated to confirm the presence and severity of acute or chronic AR. *(Level of Evidence: B)*

2. Echocardiography is indicated for diagnosis and assessment of the cause of chronic AR (including valve morphology and aortic root size and morphology) and for assessment of LV hypertrophy, dimension (or volume), and systolic function. *(Level of Evidence: B)*

3. Echocardiography is indicated in patients with an enlarged aortic root to assess regurgitation and the severity of aortic dilatation. *(Level of Evidence: B)*

4. Echocardiography is indicated for the periodic re-evaluation of LV size and function in asymptomatic patients with severe AR. *(Level of Evidence: B)*

5. Radionuclide angiography or magnetic resonance imaging is indicated for the initial and serial assessment of LV volume and function at rest in patients with AR and suboptimal echocardiograms. *(Level of Evidence: B)*

6. Echocardiography is indicated to re-evaluate mild, moderate, or severe AR in patients with new or changing symptoms. *(Level of Evidence: B)*

Class IIa

1. Exercise stress testing for chronic AR is reasonable for assessment of functional capacity and symptomatic response in patients with a history of equivocal symptoms. *(Level of Evidence: B)*

2. Exercise stress testing for patients with chronic AR is reasonable for the evaluation of symptoms and functional capacity before participation in athletic activities. *(Level of Evidence: C)*

3. Magnetic resonance imaging is reasonable for the estimation of AR severity in patients with unsatisfactory echocardiograms. *(Level of Evidence: B)*

Class IIb

Exercise stress testing in patients with radionuclide angiography may be considered for assessment of LV function in asymptomatic or symptomatic patients with chronic AR. *(Level of Evidence: B)*
The diagnosis of chronic severe AR can usually be made on the basis of the diastolic murmur, displaced LV impulse, wide pulse pressure, and characteristic peripheral findings that reflect wide pulse pressure. A third heart sound is often heard as a manifestation of the volume load and is not necessarily an indication of heart failure. An Austin-Flint rumble is a specific finding for severe AR.

In many patients with more mild to moderate AR, the physical examination will identify the regurgitant lesion but will be less accurate in determining its severity. When the diastolic murmur of AR is louder in the third and fourth right intercostal spaces than in the third and fourth left intercostal spaces, the AR likely results from aortic root dilatation rather than from a deformity of the leaflets alone. The chest X-ray and ECG are helpful in evaluating overall heart size and rhythm, evidence of LV hypertrophy, and evidence of conduction disorders.

Echocardiography is indicated

- to confirm the diagnosis of AR if there is an equivocal diagnosis based on physical examination
- to assess the cause of AR and to assess valve morphology
- to provide a semiquantitative estimate of the severity of AR
- to assess LV dimension, mass, and systolic function
- to assess aortic root size.

In asymptomatic patients with preserved systolic function, these initial measurements represent the baseline information with which future serial measurements can be compared.

LV wall stress may also be estimated from blood pressure and echocardiographic measurements. However, such wall stress measurements are difficult to reproduce, have methodological and conceptual problems, and should not be used for diagnosis or management decision making in clinical practice.
Medical Therapy

Class I

Vasodilator therapy is indicated for chronic therapy in patients with severe AR who have symptoms or LV dysfunction when surgery is not recommended because of additional cardiac or noncardiac factors. (Level of Evidence: B)

Class IIa

Vasodilator therapy is reasonable for short-term therapy to improve the hemodynamic profile of patients with severe heart failure symptoms and severe LV dysfunction before proceeding with AVR. (Level of Evidence: C)

Class IIb
Vasodilator therapy may be considered for long-term therapy in asymptomatic patients with severe AR who have LV dilatation but normal systolic function. *(Level of Evidence: B)*

**Class III**

1. **Vasodilator therapy is not indicated for long-term therapy in asymptomatic patients with mild to moderate AR and normal LV systolic function. ***(Level of Evidence: B)***

2. **Vasodilator therapy is not indicated for long-term therapy in asymptomatic patients with LV systolic dysfunction who are otherwise candidates for AVR. ***(Level of Evidence: C)***

3. **Vasodilator therapy is not indicated for long-term therapy in symptomatic patients with either normal LV function or mild to moderate LV systolic dysfunction who are otherwise candidates for AVR. ***(Level of Evidence: C)***

Therapy with vasodilating agents is designed to improve forward stroke volume and reduce regurgitant volume. These effects should translate into reductions in LV end-diastolic volume, wall stress, and afterload, resulting in preservation of LV systolic function and reduction in LV mass.

There are 3 potential uses of vasodilating agents in chronic AR.

1. long-term treatment of patients with severe AR who have symptoms and/or LV dysfunction who are considered poor candidates for surgery because of additional cardiac or noncardiac factors.
2. improvement in the hemodynamic profile of patients with severe heart failure symptoms and severe LV dysfunction with short-term vasodilator therapy before proceeding with AVR. In such patients, vasodilating agents with negative inotropic effects should be avoided.
3. prolongation of the compensated phase of asymptomatic patients who have volume-loaded left ventricles but normal systolic function.

If vasodilator therapy is used, the goal is to reduce systolic blood pressure, and drug dosage should be increased until there is a measurable decrease in systolic blood pressure or the patient develops side effects.

Vasodilator therapy is not recommended for asymptomatic patients with mild or moderate AR and normal LV function in the absence of systemic hypertension, because these patients have an excellent outcome with no therapy. In patients with severe AR, vasodilator therapy is not an alternative to surgery in asymptomatic or symptomatic patients with LV systolic dysfunction; such patients should be considered surgical candidates rather than candidates for long-term medical therapy unless AVR is not recommended because of additional cardiac or noncardiac factors. Whether symptomatic patients who have preserved systolic function can be treated safely with aggressive medical management and whether aggressive medical management is as good or better than AVR have not been determined. It is recommended that symptomatic patients undergo surgery rather than long-term medical therapy.

**Physical Activity and Exercise**

There are no data suggesting that exercise, particularly strenuous periodic exercise, will contribute to or accelerate the progression of LV dysfunction in AR. Asymptomatic patients with normal LV systolic function may participate in all forms of normal daily physical activity, including mild forms of exercise and in some cases competitive athletics.

**Serial Testing**

The aim of serial evaluation of asymptomatic patients with chronic AR is to detect the onset of symptoms and objectively assess changes in LV size and function that can occur in the absence of symptoms. In general, the stability and chronicity of the regurgitant lesion and the LV response to volume load need to be established when the patient first presents to the physician, especially if AR is moderate to severe. If the chronic nature of the
lesion is uncertain and the patient does not present initially with one of the indications for surgery, repeat physical examination and echocardiography should be performed within 2 to 3 months after the initial evaluation to ensure that a subacute process with rapid progression is not under way. Once the chronicity and stability of the process has been established, the frequency of clinical re-evaluation and repeat noninvasive testing depends on the severity of the valvular regurgitation, the degree of LV dilatation, the level of systolic function, and whether previous serial studies have revealed progressive changes in LV size or function.

In most patients, serial testing during the long-term follow-up period should include a detailed history, physical examination, and echocardiography. Serial chest X-rays and ECGs have less value but are helpful in selected patients.

Asymptomatic patients with mild AR, little or no LV dilatation, and normal LV systolic function can be seen on a yearly basis, with instructions to alert the physician if symptoms develop in the interim. Yearly echocardiography is not necessary unless there is clinical evidence that regurgitation has worsened. Routine echocardiography can be performed every 2 to 3 years in such patients.

Asymptomatic patients with normal systolic function but severe AR and significant LV dilatation (end-diastolic dimension greater than 60 mm) require more frequent and careful re-evaluation, with a history and physical examination every 6 months and echocardiography every 6 to 12 months, depending on the severity of dilatation and stability of measurements. If patients are stable, echocardiographic measurements are not required more frequently than every 12 months.

Chronic AR may develop from disease processes that involve the proximal ascending aorta. In patients with aortic root dilatation, serial echocardiograms are indicated to evaluate aortic root size, as well as LV size and function.

Repeat echocardiograms are also recommended when the patient has onset of symptoms, there is an equivocal history of changing symptoms or changing exercise tolerance, or there are clinical findings that suggest worsening regurgitation or progressive LV dilatation.

In some centers with expertise in nuclear cardiology, serial radionuclide ventriculograms to assess LV volume and function at rest may be an accurate and cost-effective alternative to serial echocardiograms.

**Indications for Cardiac Catheterization**

**Class I**

1. Cardiac catheterization with aortic root angiography and measurement of LV pressure is indicated for assessment of severity of regurgitation, LV function, or aortic root size when noninvasive tests are inconclusive or discordant with clinical findings in patients with AR. *(Level of Evidence: B)*

2. Coronary angiography is indicated before AVR in patients at risk for CAD. *(Level of Evidence: C)*

**Class III**

1. Cardiac catheterization with aortic root angiography and measurement of LV pressure is not indicated for assessment of LV function, aortic root size, or severity of regurgitation before AVR when noninvasive tests are adequate and concordant with clinical findings and coronary angiography is not needed. *(Level of Evidence: C)*

2. Cardiac catheterization with aortic root angiography and measurement of LV pressure is not indicated for assessment of LV function and severity of regurgitation in asymptomatic patients when noninvasive tests are adequate. *(Level of Evidence: C)*
Cardiac catheterization is not required in patients with chronic AR unless there are questions about the severity of AR, hemodynamic abnormalities, or LV systolic dysfunction that persist despite physical examination and noninvasive testing, or unless AVR is contemplated and there is a need to assess coronary anatomy. The indications for coronary arteriography are discussed in Section 10.2. In some patients undergoing left-heart catheterization for coronary angiography, additional aortic root angiography and hemodynamic measurements may provide useful supplementary data.

Hemodynamic and angiographic assessment of the severity of AR and LV function may be necessary in some patients being considered for surgery when there are conflicting data between clinical assessment and noninvasive tests. Less commonly, asymptomatic patients who are not being considered for surgery may also require invasive measurement of hemodynamics and/or determination of severity of AR when this information cannot be obtained accurately from noninvasive tests.

Hemodynamic measurements during exercise are occasionally helpful for determining the effect of AR on LV function or making decisions regarding medical or surgical therapy. In selected patients with severe AR, borderline or normal LV systolic function, and LV chamber enlargement that is approaching the threshold for surgery (defined below), measurement of cardiac output and LV filling pressures at rest and during exercise with a right-heart catheter may be valuable for identifying patients with severe hemodynamic abnormalities in whom surgery is warranted.

**Indications for Aortic Valve Replacement or Aortic Valve Repair**

The majority of patients with severe AR requiring surgery undergo valve replacement. However, in several surgical centers, there is increasing experience in performing aortic valve replacement in selected patients.

In patients with pure, chronic AR, AVR should be considered only if AR is severe.

**Class I**

1. AVR is indicated for symptomatic patients with severe AR irrespective of LV systolic function. *(Level of Evidence: B)*

2. AVR is indicated for asymptomatic patients with chronic severe AR and LV systolic dysfunction (ejection fraction 0.50 or less) at rest. *(Level of Evidence: B)*

3. AVR is indicated for patients with chronic severe AR while undergoing CABG or surgery on the aorta or other heart valves. *(Level of Evidence: C)*

**Class IIa**

AVR is reasonable for asymptomatic patients with severe AR with normal LV systolic function (ejection fraction greater than 0.50) but with severe LV dilatation (end-diastolic dimension greater than 75 mm or end-systolic dimension greater than 55 mm).* *(Level of Evidence: B)*

**Class IIb**

1. AVR may be considered in patients with moderate AR while undergoing surgery on the ascending aorta. *(Level of Evidence: C)*

2. AVR may be considered in patients with moderate AR while undergoing CABG. *(Level of Evidence: C)*

3. AVR may be considered for asymptomatic patients with severe AR and normal LV systolic function at rest (ejection fraction greater than 0.50) when the degree of LV dilatation exceeds an end-diastolic dimension of 70 mm or end-systolic dimension of 50 mm, when there is evidence of progressive LV
dilatation, declining exercise tolerance, or abnormal hemodynamic responses to exercise.* (Level of Evidence: C)

Class III

AVR is not indicated for asymptomatic patients with mild, moderate, or severe AR and normal LV systolic function at rest (ejection fraction greater than 0.50) when degree of dilatation is not moderate or severe (end-diastolic dimension less than 70 mm, end-systolic dimension less than 50 mm).* (Level of Evidence: B)

*Consider lower threshold values for patients of small stature of either gender.

Symptomatic Patients With Normal Left Ventricular Systolic Function

AVR is indicated in patients with normal LV systolic function (defined as ejection fraction greater than 0.50 at rest) who have NYHA functional class III or IV symptoms. Patients with Canadian Heart Association functional class II to IV angina pectoris should also be considered for surgery. In many patients with NYHA functional class II dyspnea, the cause of symptoms is often unclear, and clinical judgment is required. Patients with well-compensated AR often have chronic mild dyspnea or fatigue, and it may be difficult to differentiate the effects of deconditioning or aging from true cardiac symptoms. In such patients, exercise testing may be valuable. If the cause of these mild symptoms is uncertain and they are not severe enough to interfere with the patient’s lifestyle, a period of observation may be reasonable. However, new onset of mild dyspnea has different implications in severe AR, especially in patients with increasing LV chamber size or evidence of declining LV systolic function into the low normal range. Thus, even if patients have not achieved the threshold values of LV size and function recommended for surgery in asymptomatic patients, development of mild symptoms is an indication for AVR in a patient who is nearing these values.

Symptomatic Patients With Left Ventricular Dysfunction

Patients with NYHA functional class II, III, or IV symptoms and with mild to moderate LV systolic dysfunction (ejection fraction 0.25 to 0.50) should undergo AVR. Patients with NYHA functional class IV symptoms have worse postoperative survival rates and lower likelihood of recovery of systolic function than patients with less severe symptoms but AVR will improve ventricular loading conditions and expedite subsequent management of LV dysfunction

Severely symptomatic patients (NYHA functional class IV) with advanced LV dysfunction (ejection fraction less than 0.25 and/or end-systolic dimension greater than 60 mm) present difficult management issues. Some patients will manifest meaningful recovery of LV function after AVR, but many will have developed irreversible myocardial changes. The mortality associated with valve replacement approaches 10%, and postoperative mortality over the subsequent few years is high. Valve replacement should be considered more strongly in patients with NYHA functional class II and III symptoms, especially if

• symptoms and evidence of LV dysfunction are of recent onset;

• intensive short-term therapy with vasodilators and diuretics results in symptomatic improvement;

• intravenous positive inotropic agents result in substantial improvement in hemodynamics or systolic function.

However, even in patients with NYHA functional class IV symptoms and ejection fraction less than 0.25, the high risks associated with AVR and subsequent medical management of LV dysfunction are usually a better alternative than the higher risks of long-term medical management alone (328).

Asymptomatic Patients
AVR in asymptomatic patients remains a controversial topic, but it is generally agreed that AVR is indicated in patients with LV systolic dysfunction.

AVR is also recommended in patients with severe LV dilatation (end-diastolic dimension greater than 75 mm or end-systolic dimension greater than 55 mm), even if ejection fraction is normal.

Anthropometric normalization of LV end-diastolic dimension (or volume) should be considered, but unfortunately, there is lack of agreement as to whether or not normalization based on body surface area or body mass index is predictive of outcome.

Patients with severe AR in whom the degree of LV dilatation has not reached but is approaching these threshold values (e.g., LV end-diastolic dimension of 70 to 75 mm or end-systolic dimension of 50 to 55 mm) should be followed with frequent echocardiograms every 4 to 6 months. In addition, AVR may be considered in such patients if there is evidence of declining exercise tolerance or abnormal hemodynamic responses to exercise, for example, an increase in pulmonary artery wedge pressure greater than 25 mm Hg with exercise.

The surgical options for treating AR are expanding, with growing experience in aortic homografts, pulmonary autografts, unstented tissue valves, and aortic valve repair. If these techniques are ultimately shown to improve long-term survival or reduce postoperative valve complications, it is conceivable that the thresholds for recommending AVR may be reduced. Until such data are available, the indications for surgery for AR should not vary with the operative technique to be used.