Assessment of the patient with valvular heart disease: An integrative approach

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INTRODUCTION

In managing the patient with valvular heart disease (VHD), three major issues must be addressed: (1) assessment of the severity of disease, (2) the effect the disease is having or is likely to have on the patient and his/her cardiovascular system, and (3) the timing and type of intervention to be used to correct the lesion. Existing guidelines are helpful in addressing these tenets in many cases, often using quantifiable parameters to aid the clinician in making key clinical decisions. Many times these guidelines allow the physician and patient to arrive easily at a management strategy. However, in other cases it may take every piece of available data to develop a management plan that is still only a best guess at the proper course to take.

Overall, the indications for intervention in VHD are straightforward: valvotomy (in mitral stenosis), or valve repair or valve replacement is indicated when severe VHD causes symptoms or cardiac dysfunction. In some cases, low-risk intervention such as mitral valve repair may be undertaken in the absence of symptoms or dysfunction when it seems inevitable that deterioration will occur because of markedly severe disease. These assessments are rarely made using one test and usually require the integration of all the clinical acumen that can be summoned to address the issues above.

The following will summarize the general approach to the assessment of VHD severity, impact, and timing of intervention, with attention paid to the specifics of each individual disease.

SEVERITY OF DISEASE

The AHA/ACC guidelines for assessing severity of disease are displayed in Table 1 [1]. The distinction between mild, moderate and severe disease is thought crucial since it is believed that, in most cases, mild and moderate disease are tolerated indefinitely (unless severity worsens) and only severe disease (as defined) causes symptoms and cardiac dysfunction. It is critical to understand that these definitions have been developed from consensus of opinion and are not from the result of any large, randomized trials. Rather they are built upon the general experience of experts, and expert opinion is not monolithic. For instance, the mean transvalvular gradient consistent with severe aortic stenosis (AS) was deemed to be 50 mm Hg in the guidelines published in 1998 [2] but was revised to be 40 mm Hg in the 2006 guidelines [1]. This change was not derived from new data acquired between the writing of the two sets of guidelines; rather, it reflected differences of opinion due to changes to the committee make-up from one writing committee to the next. In addition, the 2006 writing committee removed the adjective “critical” as a descriptor of AS to indicate that while a manmade definition of “severe” was a matter of consensus, the definition of “critical” (a valve area certain to cause morbidity or death) was unknown.

OBJECTIVE ASSESSMENT OF DISEASE SEVERITY

The physical examination

In this age of high-tech diagnostic modalities, the physical examination, and the skill applied when performing it, seem to be diminishing. However, the importance of the physical exam cannot be
Table 1. Taken from Ref. [1] with permission.

A. Left-sided valve disease

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aortic Stenosis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jet velocity (m per second)</td>
<td>Less than 3.0</td>
<td>3.0–4.0</td>
<td>Greater than 4.0</td>
</tr>
<tr>
<td>Mean gradient (mm HG)</td>
<td>Less than 25</td>
<td>25–40</td>
<td>Greater than 40</td>
</tr>
<tr>
<td>Valve area (cm²)</td>
<td>Greater than 1.5</td>
<td>1.0–1.5</td>
<td>Less than 1.0</td>
</tr>
<tr>
<td>Valve area index (cm² per m²)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mitral Stenosis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean gradient (mm Hg)</td>
<td>Less than 5</td>
<td>5–10</td>
<td>Greater than 10</td>
</tr>
<tr>
<td>Pulmonary artery systolic pressure (mm Hg)</td>
<td>Less than 30</td>
<td>30–50</td>
<td>Greater than 50</td>
</tr>
<tr>
<td>Valve area (cm²)</td>
<td>Greater than 1.5</td>
<td>1.0–1.5</td>
<td>Less than 1.0</td>
</tr>
<tr>
<td><strong>Aortic Regurgitation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Qualitative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angiographic grade</td>
<td>1+ Central jet, width</td>
<td>2+ Greater than mild</td>
<td>3–4+ Central jet, width</td>
</tr>
<tr>
<td>Color Doppler jet width</td>
<td>less than 25% of LVOT</td>
<td>but no signs of severe AR</td>
<td>greater than 65% LVOT</td>
</tr>
<tr>
<td>Doppler vena contracta width (cm)</td>
<td>Less than 0.3</td>
<td>0.3–0.6</td>
<td>Greater than 0.6</td>
</tr>
<tr>
<td>Quantitative (cath or echo)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regurgitant volume (ml per beat)</td>
<td>Less than 30</td>
<td>30–59</td>
<td>Greater than or equal to 60</td>
</tr>
<tr>
<td>Regurgitant fraction (%)</td>
<td>Less than 30</td>
<td>30–49</td>
<td>Greater than or equal to 50</td>
</tr>
<tr>
<td>Regurgitant orifice area (cm²)</td>
<td>Less than 0.10</td>
<td>0.10–0.29</td>
<td>Greater than or equal to 0.30</td>
</tr>
<tr>
<td>Additional essential criteria</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular size</td>
<td>Increased</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mitral Regurgitation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Qualitative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angiographic grade</td>
<td>1+ Small, central jet</td>
<td>2+ Signs of MR</td>
<td>3–4+ Vena contracta width greater</td>
</tr>
<tr>
<td>Color Doppler jet area</td>
<td>less than 4 cm² or less than 20% LA area</td>
<td>greater than mild present but no criteria for severe MR</td>
<td>than 0.7 cm with large central MR jet (area greater than 40% of LA area) or with a wall-impinging jet of any size swirling in LA</td>
</tr>
<tr>
<td>Doppler vena contracta width (cm)</td>
<td>Less than 0.3</td>
<td>0.3–0.6</td>
<td>Greater than or equal to 0.70</td>
</tr>
<tr>
<td>Quantitative (cath or echo)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regurgitant volume (ml per beat)</td>
<td>Less than 30</td>
<td>30–59</td>
<td>Greater than or equal to 60</td>
</tr>
<tr>
<td>Regurgitant fraction (%)</td>
<td>Less than 30</td>
<td>30–49</td>
<td>Greater than or equal to 50</td>
</tr>
<tr>
<td>Regurgitant orifice area (cm²)</td>
<td>Less than 0.20</td>
<td>0.2–0.39</td>
<td>Greater than or equal to 0.40</td>
</tr>
<tr>
<td>Additional essential criteria</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left atrial size</td>
<td>Enlarged</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular size</td>
<td>Enlarged</td>
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</tbody>
</table>

(continued on next page)

overemphasized because it lays the Bayesian foundation for all tests that follow. Bayes’ theory states that the accuracy of any test is determined by the pretest probability that the condition being tested for is present. Since no test in VHD is 100 percent accurate, the physical exam lays the basis for subsequent tests, directing them toward the condition hypothesized from the exam data.

**IMAGING**

**Echocardiography**

Echocardiography forms the mainstay of laboratory diagnosis in VHD. Echo’s low-cost, accuracy, portability and reproducibility make it ideal for both the initial assessment of the patient with VHD as
Table 1 (continued)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>B. Right-sided valve disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve less than 1.0 cm²</td>
<td>Severe tricuspid stenosis:</td>
</tr>
<tr>
<td>Vena contracta width greater than 0.7 cm and systolic flow reversal in hepatic veins</td>
<td>Severe tricuspid regurgitation:</td>
</tr>
<tr>
<td>Jet velocity greater than 4 m per second or maximum gradient greater than 60 mm Hg</td>
<td>Severe pulmonic stenosis:</td>
</tr>
<tr>
<td>Color jet fills outflow tract; dense continuous wave Doppler signal with a steep deceleration slope</td>
<td>Severe pulmonic regurgitation:</td>
</tr>
</tbody>
</table>

1 Valve gradients are flow dependent and when used as estimates of severity of valve stenosis should be assessed with knowledge of cardiac output or forward flow across the valve. Modified with permission from Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr 2003;16:777–802 (27).

AR indicates aortic regurgitation; cath, catheterization, echo, echocardiography; LA, left atrial/atruim, LVOT, left ventricular outflow tract; and MR, mitral regurgitation.

well as for the repeated studies necessary to follow the progression of the disease. Provided that the patient’s sonographic window is adequate, the echocardiogram gives information about valve pathoanatomy, transvalvular pressure gradients, severity of valvular regurgitation and the impact of the pathophysiology of a patient's VHD on cardiac chamber geometry and function. Quantitative measures of valvular stenosis are quite accurate when compared with other methods of assessment. Although quantitative methods of the assessment of valvular regurgitation have been introduced, they are generally less precise and less applied than those for assessing valvular stenosis and thus are more subjective.

With regards to pathoanatomy, transesophogal echocardiography produces excellent images of the mitral valve, and 3-D echocardiography produces a view akin to what the surgeon sees at the operating table.

**Magnetic resonance imaging (MRI)**

The strength of MRI lies in its ability to very precisely measure left ventricle (LV) volumes allowing for accurate evaluation of the effect of valvular regurgitation on LV remodeling, LV ejection fraction and quantification of regurgitant flow and regurgitant fraction [3].

**Invasive hemodynamics**

If after a careful history, physical examination and non-invasive imaging, the diagnosis and/or degree of disease severity is still unclear, invasive hemodynamic investigation is performed to arrive at a final diagnosis. Invasive investigation affords direct measurement of intracardiac pressures, and cardiac output, which are in fact the essentials of cardiac function. It must be noted that invasive evaluation has its own pitfalls and these have been magnified more recently by lack of specific training in this discipline in many training programs. Invasive evaluation relies upon careful pressure measurement using meticulously calibrated manometers connected to properly-placed and flushed catheters, as well as careful measurement of cardiac output [4].

**IMPACT OF VHD ON THE PATIENT**

**Symptoms**

For every type of severe VHD lesion, the presence of symptoms referable to that lesion are indications for mechanical therapy because prognosis worsens with their presence [5–8]. While symptoms are obviously subjective and not as elegant a measurement as transvalvular gradient, for instance, symptoms do offer a measurement of cardiovascular integrity that other techniques of assessment cannot avail. Symptoms develop from integrated abnormalities in left and right ventricular systolic function, diastolic function, atrial compliance, filling pressures, coronary blood flow and cardiac output. No objective measure of cardiovascular function has this capability. Thus it is not surprising that the presence or absence of symptoms has prognostic implication for the management of every valvular lesion. Unfortunately history-taking skills may be waning as medicine concentrates on more high-tech methods of evaluation. In obtaining a good history of a patient’s symptomatic state, it is important to get the information not only from the patient but also from the spouse or close associate because the patient may fail to recognize his/her symptoms or may simply deny that they are present.
Exercise tolerance
Because of the subjective nature of the symptomatic state and because of its importance in decision making, it is almost always advisable to observe a formal (or even informal) episode of exercise. Carefully monitored exercise testing of the asymptomatic patient with VHD is safe and also quite logical since the patient who feels he is asymptomatic might exercise with impunity. If indeed such a patient is at risk because he/she fails to recognize symptoms, it is better to detect them under the watchful eye of the physician (such studies should not be left to the care of technical personnel). If the patient has a perfectly normal test, achieving age-predicted exercise tolerance and normal hemodynamics, it is reassuring to both the physician and patient that continued “watchful waiting” is safe and appropriate [9–12]. However if the patient has unexpectedly poor exercise tolerance, develops hemodynamic instability, or has frank symptoms during exercise, these are usually indications to move to therapeutic intervention.

Ventricular dysfunction
As noted above, severe VHD imposes a volume or pressure overload (or both) on the left and/or right ventricles (LV or RV). If prolonged, this hemodynamic burden damages the myocardium, and leads to heart failure and death. Obviously, early detection of myocardial damage as it leads to LV dysfunction presages the need for intervention before damage becomes irreversible. Unfortunately ejection fraction (EF) remains the primary benchmark for assessing systolic function. Ejection fraction is determined by contractility (the property describing myocardial function), preload and afterload. Because both preload and afterload may be extraordinarily abnormal in VHD, EF becomes a potentially very confounded measure of LV function. Thus high afterload in aortic stenosis may cause depressed EF despite relatively well-preserved contractility that could mislead the clinician into believing his patient has a falsely poor prognosis (Fig. 1) [13]. Conversely increased preload in mitral regurgitation enhances EF and causes it to overestimate myocardial contractility [14]. While literally dozens of other measures of ventricular function have been developed, those more accurate than EF have proved cumbersome and have never received widespread clinical usage. Instead, a number of caveats have been developed to tailor EF according to the effect that a given lesion has upon it.

One index that is clinically useful is end systolic dimension or (volume). Because end systolic dimension is dependent upon contractility, afterload and LV remodeling but independent of preload [15], this measure has been favored as an indicator of systolic function in volume overload VHD where increased preload most affects EF [16,17].

More advanced measures of LV function including strain rate imaging have been investigated as potentially more sensitive to early changes in both systolic and diastolic function [18]. While showing promise, none have become routine in clinical decision making in VHD.

Figure. 1 Ejection fraction (EF) is plotted against systolic wall stress ($\sigma$) (afterload) for patients with AS and heart failure. For some patients (circles) EF is reduced almost entirely due to excess afterload. In others, (x) EF is reduced primarily due to myocardial damage and contractile dysfunction. Taken with permission from Ref. [13].
Biomarkers
The imprecision of symptoms and the measures of ventricular function noted above have led to a search for better indicators to help time intervention for VHD. B-type natriuretic peptide (BNP) and its precursor NT proBNP are thought to be secreted by the ventricles as a result of sarcomere stretch. Thus as the hemodynamic overloads of VHD cause the ventricles to rely more on preload reserve for compensation, these biomarkers are released in greater quantities. Indeed BNP is increased in severe VHD, while many cutoff values have been suggested as indicating decompensation in VHD [19–21], none has been widely employed as a surrogate marker indicating the need for intervention. On the other hand, low BNP offers some reassurance of ventricular compensation and the presence of normal filling pressures.

SPECIFIC DISEASES
Aortic stenosis
The asymptomatic patient with severe disease. Management of the patient with symptomatic severe aortic stenosis constitutes one of the most straightforward decisions in Cardiology because of the remarkable lethality of the disease. Either the patient undergoes aortic valve replacement (AVR) or suffers a 75 percent chance of death in 3 years [5,22,23]. The advent of transcatheter aortic valve implantation (TAVI) offers an alternative to surgical AVR for patients with risk factors that make surgery untenable [24].

Conversely the asymptomatic patient with severe disease presents much more of a challenge. The risk of sudden death is small but palpable (about 0.5 percent/year) [25,26], but the risk of AVR is also minimal, making it hard to weigh the clinical options in such patients. A high jet velocity (exceeding 4.0 m/sec), heavy valve calcification, poor exercise tolerance (or a fall in blood pressure during exercise), severe LVH and/or a rising BNP are indicators of higher than average risk and that AVR will be required soon because of the onset of symptoms or LV dysfunction [9,19,26–28]. In such cases elective AVR may be advisable, especially for patients with few comorbidities and for those wishing to pursue an active life style or whose occupations might increase the risk of exercise-induced sudden death.

In most cases, echocardiographic imaging with Doppler interrogation of the aortic valve is adequate to establish AS severity. However, in some cases obtaining invasive hemodynamics is necessary to fully evaluate the disease. In such instances exact transducer balancing and calibration, and exact catheter placement together with a carefully obtained cardiac output are needed to insure that an aortic valve area can be calculated accurately [29]. Because there is often a pressure gradient between the body of the LV and the LV outflow tract, it is important to place the LV catheter well within the body of the LV. Because there is substantial pressure recovery distal to where flow exits the valve, the distal catheter should be placed in the ascending aorta never in the femoral artery where the pressure gradient can be severely underestimated (Fig. 2) [30].

The patient with far advanced LV dysfunction, low gradient and low EF. Left ventricular hypertrophy is thought to occur in AS in response to the pressure overload that AS engenders. Afterload can be quantified as wall stress (\(\sigma\)) where \(\sigma = P r / 2 th\) and \(P = LV\) pressure, \(r =\) ventricular radius and \(th =\) wall thickness. Grossman et al. postulated that as pressure increases in the stress equation numerator it causes concentric LVH to develop whereby increased pressure is offset by increased wall thickness in the equation’s denominator [31]. In this manner, wall stress (afterload) is normalized, a compensatory function since LV ejection is inversely related to afterload. However, in some patients, LVH is inadequate to normalize stress, afterload increases and EF falls [13]. Such patients have a high transvalvular gradient and an excellent prognosis following surgery [32].

In other patients EF and LV stroke volume are reduced not by exceptionally high afterload but by severely compromised myocardial contractility. Such patients typically have a low gradient (LV pressure and afterload are only modestly increased) and a poor prognosis [33–35]. Valve area may be very misleading in this group of patients because valve area can be flow dependent, increasing as flow increases until cardiac output reaches about 5 l/min [36]. In low flow, low gradient, low EF patients, a dobutamine challenge either in the echocardiographic or catheterization laboratories may be very useful [35]. Three different responses to dobutamine may be seen (Table 2). First there may be an substantial (> 20 percent) increase in stroke volume with a concomitant increase in gradient and only a small increase in AVA. Such patients have severe AS, inotropic reserve and a relatively
good prognosis at surgery (Fig. 3) [35]. A second response is an increase in stroke volume, little increase in gradient and a large increase in AVA. It is thought that in these patients there is only moderate AS and that increasing flow increases valve area by physically opening the valve to a greater orifice area. Since the AS was in fact not truly severe despite an initially calculated AVA that suggested severe AS (pseudo-AS) [37], it is believed that AVR will not lead to much improvement in the patient’s condition. A third response is that stroke volume fails to increase with dobutamine (lack of inotropic reserve). Such patients have a poor prognosis at AVR although EF may improve substantially if they survive the surgery [38]. Prognosis is especially poor for patients with severe coronary disease, with a mean gradient of $<20$ mm Hg, who also lack inotropic reserve [39].

The patient with low flow, low gradient and normal EF. In still other patients there is concentric LV remodeling without LVH (LV weight is not increased) leading to a small thick chamber with reduced end diastolic volume. Even though EF is normal, stroke volume is reduced and thus so is transvalvular gradient although is usually exceeds 25 mm Hg. Symptomatic patients with this condition have severe symptomatic AS, a good prognosis with AVR but a poor prognosis if untreated [40,41].

Thus management decisions in the AS patient may be very straightforward in the symptomatic patient with a large transvalvular gradient, or conversely they may require the integration of data from every modality available in the case of the patient with low flow and low gradient. In these difficult cases the clinician cannot rely upon a single number, whether AVA, jet velocity or gradient to make the ultimate decision regarding mechanical intervention. He/she must integrate all the data available and combine it with clinical judgment and experience to arrive at the best course of action.

### Chronic aortic regurgitation

Assessment of lesion severity and its impact on the heart. Severe aortic regurgitation (AR) is generally well tolerated for many years, progressing to symptom onset or asymptomatic LV dysfunction at a rate

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**Figure 2** Left ventricular (LV) and femoral artery (FA) tracings (left panel) are compared with pressure recordings from LV and aorta (right panel) in the same patient with AS. Time delay and pressure recovery cause the LV-FA recording to severely underestimate the true transvalvular gradient.

**Table 2.** Potential responses to dobutamine in aortic stenosis patients with low gradient, low ejection fraction.

<table>
<thead>
<tr>
<th></th>
<th>CO</th>
<th>GRAD</th>
<th>AVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>TRUE AS</td>
<td>R</td>
<td>3.5</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>5.0</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>42</td>
<td>0.7</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>0.75</td>
<td>0.7</td>
</tr>
<tr>
<td>PSEUDO AS</td>
<td>3.5</td>
<td>5.0</td>
<td>25</td>
</tr>
<tr>
<td>Negative Response</td>
<td>3.5</td>
<td>3.6</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>26</td>
<td>0.7</td>
<td>1.0</td>
</tr>
</tbody>
</table>

AS = Aortic Stenosis, AVA = Aortic Valve Area cm², CO = Cardiac Output /min, D = Dobutamine, GRAD = transvalvular gradient mm Hg, R = Rest
of about four percent per year [42,43]. Severe disease can be defined as that amount of AR that leads to the sequelae noted above. More precisely a regurgitant fraction exceeding 50 percent is generally thought to be the threshold for the amount of AR that leads to adverse outcomes. Echocardiographic jet area, effective regurgitant orifice area (ERO) pressure half time and regurgitant flow also aid in establishing AR severity [44]. Regurgitant flow can also be estimated using cardiac magnetic resonance imaging [45]. If the severity of AR is still uncertain after non-invasive imaging, cardiac catheterization is useful to assess both severity of the lesion and its impact on the LV. Contrast aortography provides direct imaging of the regurgitant flow and a good estimation of AR severity provided that enough contrast is injected to opacify both the dilated LV and aorta. Assessment of LV filling pressure at rest and during static or active exercise can help determine whether a patient’s symptoms have a hemodynamic basis or whether hemodynamics are significantly deranged despite the absence of symptoms (Fig. 4).

Sudden death in AR is rare but does occur sporadically in patients with very dilated left ventricles, those exceeding an end diastolic dimension of 75 mm [42]. As with aortic stenosis, the onset of symptoms marks a worsening prognosis although without the immediate dire consequences seen in AS [6]. Because the patient may deny symptoms or fail to recognize them, formal exercise testing is helpful in defining both the presence of symptoms and also is of prognostic importance [46]. Prognosis is also reduced even in the absence of symptoms if asymptomatic LV dysfunction intervenes as documented by an EF falling toward 50% or an end systolic dimension approaching 50–55 mm [16,47].

Aortopathy and bicuspid aortic valve. Although controversial [48] many experts believe that some patients with a congenitally bicuspid aortic valve also have disease of the aorta making it more prone to dilate and eventually dissect and rupture. Thus assessment of the patient with AR (and AS) should always include delineation of valve morphology and an assessment of the aortic root, its dimensions and whether it should be addressed at the time of surgery. Following careful assessment, outcome can be excellent even in the face of aortic dilatation [49].

Organic mitral regurgitation (MR)

Assessing severity of MR. The ability to easily visualize the MR jet as it enters the left atrium has led to many approaches for assessing MR severity as defined in Table 1. A common cause of misinterpretation is failure to look at LV and left atrial (LA) volume in the context of estimated MR severity. If MR is both chronic and severe it must lead to LV and LA dilatation in so that the LV generates adequate forward stroke volume to compensate for that lost to MR, and for the LA to

![Kaplan–Meier survival estimates by group and treatment.](image-url)
accommodate the MR at a tolerable filling pressure. Thus, if the diagnosis of chronic severe MR is made, the left-sided chambers should be dilated. These and all other clues, including ERO, systolic pulmonary vein flow reversal and estimated pulmonary pressure, should be assessed to establish whether or not the patient’s MR is severe [50–54]. For the patient complaining of exercise-induced symptoms, exercise echocardiography may be very revealing [12]. In some cases, exercise induces a substantial increase in the amount of MR present, and in other cases, there may be a large increase in pulmonary artery pressure. Both conditions help to explain why symptoms develop during exercise despite a more benign resting echocardiogram.

If after non-invasive evaluation, MR severity is still unclear, cardiac catheterization may clarify the issue. Direct measurement of LV filling pressure at rest or with exercise can help establish whether there is a hemodynamic basis for the patient’s symptoms. Although fallen into disuse, a carefully performed left ventriculogram that avoids LV ectopy and uses enough contrast to opacify the enlarged LA and LV can directly visualize regurgitant flow and help evaluate MR severity.

The impact of mitral valve repair. For patients with AS, the aortic valve must virtually always be replaced, and it must usually be replaced for patients with AR. However, in most cases of non-rheumatic MR the mitral valve is repairable, a fact which has far-reaching implications for patient management. A durable repair carries none of the risks inherent to the implantation of a prosthetic valve. Operative risk for repair is approximately one-fourth that of valve replacement, and following repair there is no need for prolonged anticoagulation and obviously no risk of prosthetic valve

Figure 4 Pulmonary capillary wedge pressure (PCW) at rest (left panel, 4A) and during handgrip exercise (right panel, 4B) is shown for a patient with AR. Afterload increased by handgrip causes a substantial increase in PCW.
structural deterioration [55–57]. These factors allow consideration of mitral repair for severe MR even before symptoms develop and before there is evidence of LV deterioration [1]. However, the patient, and his/her referring physician and cardiac surgeon must have a very high level of confidence that the valve can be repaired to engage in this management strategy. Replacement (and its attendant risks) instead of a repair in an asymptomatic patient with normal LV function constitutes a serious disservice to the patient. In making this decision, excellent preoperative imaging is key. High-grade 2D or 3D echo images may permit the surgeon to predict whether or not the valve pathoanatomy is consistent with his/her ability to repair the valve and thus whether or not to embark upon early surgery.

**Timing of surgery if valve reparability is in question.** If it is uncertain that the mitral valve can be repaired and might need to be replaced as in rheumatic MR or in highly complex myxomatous valves, surgery should be performed at the onset of symptoms or when there is evidence of LV dysfunction [7,17,58]. Many consider the advent of pulmonary hypertension at rest or with exercise and/or the onset of atrial fibrillation also to be indications for proceeding to mitral valve surgery. Because the increased preload in MR together with normal afterload facilitate ejection, an EF of 0.60 has been established as a threshold for surgical intervention, bolstered by evidence that preoperative deterioration to a lower EF results in poor postoperative outcomes [58]. Mitral valve surgery should also occur before the LV is unable to contract to an end systolic dimension of 40 mm [17]. As demonstrated in Fig. 5, it is safe to observe the patient until these thresholds are reached but the time until they are reached may be quite short [59]. Thus, very close follow-up is indicated to avoid missing the optimum time for surgical intervention.

**Functional MR**

In dilated and ischemic cardiomyopathies, severe LV dysfunction, associated wall motion abnormalities, and mitral annular dilatation may act in concert to cause MR. Unlike in organic MR, where valvular abnormalities pose a hemodynamic burden on the heart leading to LV dysfunction, in functional MR the reverse is true: the LV dysfunction causes MR. While the presence of MR in cardiomyopathy worsens prognosis [60], it is not clear whether the MR itself is the culprit or whether the presence of MR simply implies worse LV function, which is the actual cause of poorer outcome. In support of this latter concept is the difficulty in demonstrating that repair of functional MR prolongs life or that it leads to long-term improvement in the quality of life [61–65] as would be expected if the MR itself were a key determinant of outcome. For this reason, mechanical correction of functional MR is reserved for those patients who are very symptomatic after institution of a maximum medical regimen for heart failure [1].
Mitral stenosis (MS)

Assessing lesion severity. Facile imaging of the mitral valve makes assessment of MS relatively straightforward. Valve area can be directly planimetered or established by Doppler interrogation of the valve [66–68]. Pulmonary pressure is easily estimated if any tricuspid regurgitation is present.

If severity is still in question, catheterization can establish the transvalvular gradient. Because an error of only a few mm Hg can make a significant difference in the calculated mitral valve area, careful attention to transducer zeroing, balancing and calibration is mandatory. If pulmonary capillary wedge pressure is substituted for LA pressure, confirmation that the pulmonary catheter is truly wedged by demonstrating that highly oxygenated LA blood can be withdrawn from it is essential [4]. Valve area is then established using a carefully measured transvalvular pressure gradient and cardiac output in concert with the Gorlin formula. Hemodynamic assessment during exercise can be especially revealing in MS. The patient with nearly normal LA and pulmonary pressures at rest may develop striking increases in both during exercise, helping to establish a cause for the patient’s symptoms and support that mechanical intervention will be beneficial.

Timing and selection of mechanical therapy. Mitral stenosis should be corrected when more than mild symptoms develop or when asymptomatic pulmonary hypertension develops [1,69,70]. In many cases balloon mitral valvotomy (BMV) offers a durable correction by performing a satisfactory commissurotomy in patients with severe MS and less than moderate MR [71–73]. A scoring system that allot 1–4 points each for the severity of valve calcification, leaflet mobility, leaflet thickening and disease of the subvalvular apparatus (4–16 points) helps establish the feasibility of BMV [74]. A valve score of <9 is favorable for a successful BMV. However many patients with higher scores still may undergo a successful BMV while not everyone with a low score enjoys success.

If BMV is thought inadvisable because of significant MR (that often worsens post BMV) or because of a high valve score, mitral valve replacement is then undertaken.

Tricuspid regurgitation

Tricuspid regurgitation (TR) frequently accompanies mitral valve disease. TR is often thought to be secondary to mitral disease’s attendant pulmonary hypertension. Accordingly, TR may be expected to improve following successful mitral valve intervention [75]. While such improvement often occurs, it is unfortunately unpredictable [76]. Therefore it is advisable to address TR during mitral surgery by simply installing a ring annuloplasty that helps prevent TR from persisting or worsening following mitral surgery.

SUMMARY

Unlike most other fields in Cardiology where data from clinical trials help prescribe therapy, the lack of large, randomized trials in VHD means that decision-making is often based upon guidelines developed from consensus of opinion. Given the uncertainty to which this leads in many cases, integration of the very best bedside skills, imaging, and hemodynamic data must be brought to bear to arrive at the best management strategy. Assessments of lesion severity, the effect of the lesion on the patient and his heart and the method of lesion correction require a careful analysis of all the data available and rarely if ever can be left to a single diagnostic modality.

References


Trichon BH, Felker GM and Shaw LK et al. Relation of frequency and severity of mitral regurgitation to survival among patients with left ventricular systolic dysfunction and heart failure. Am J Cardiol. 2003;91:538–543.


