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Cardiovascular Imaging

A handbook for clinical practice

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A handbook for clinical practice

EDITED BY
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Christopher M. Kramer
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Preface

As part of The European Society of Cardiology Education Series, this book is focused on the use of non-invasive imaging in clinical cardiology. Currently, the main non-invasive imaging modalities include echocardiography, nuclear imaging, cardiac magnetic resonance (CMR), and (multi-slice) computed tomography (MSCT). Rather than providing another textbook on imaging techniques, the central theme in this book is how to use these different imaging modalities to solve clinical problems that physicians encounter on a regular basis. A variety of clinical syndromes are discussed, including valvular disease, coronary artery disease, and myocardial and pericardial disease. In these various pathologies, the incremental value of echocardiography, nuclear imaging, CMR and MSCT are highlighted. Timely issues are discussed, for example the use of all imaging modalities in the assessment of myocardial viability in ischemic heart failure, the use of tissue Doppler echocardiography in cardiac resynchronization therapy, non-invasive angiography using MSCT in the evaluation of coronary artery disease, and the use of CMR in the evaluation of adult congenital heart disease.

All the chapters are clinically oriented, illustrating the contribution of different imaging techniques to the management of these clinical issues. The chapters reflect the expertise of the authors in managing the clinical problems, and can serve as a guide to physicians as to how these clinical issues can be addressed. The majority of the chapters are also illustrated with representative case histories and the moving images are available on the accompanying CD-Rom. The cases in particular offer excellent examples of how to use the imaging modalities in clinical cardiology.

The authors were selected based on their knowledge and experience in the field, and represent a broad panel of expertise both from a scientific and clinical point-of-view. Contributors are active members of the various Working Groups and Association of the European Society of Cardiology, including the Working Groups on CMR and Nuclear Cardiology respectively and the Association of Echocardiography. Besides contributors from Europe, additional authors from the United States and Asia have been included to provide a global perspective on the use of non-invasive imaging in clinical cardiology. Not necessarily all imaging modalities are discussed in each chapter, since different imaging modalities are more or less useful in the clinical scenarios discussed. The contributors have provided their own view on how to approach the different clinical problems and which techniques to use. It is possible that other imaging modalities will emerge to be as useful in the future; yet we trust that the current state of the art is adequately described.

The editors (each representing different imaging modalities) are grateful to all the authors for their excellent contributions. With this goal in mind, we sincerely hope that this book will be seen by clinicians as a useful handbook and help them to make the best usage of cardiovascular imaging modalities.

Jeroen J. Bax
Christopher M. Kramer
Thomas Marwick
William Wijns

Foreword

Over the last decade, we have witnessed an exponential development in imaging technology. Today, imaging plays a pivotal role in clinical management and decision making in patients with nearly every disease of the cardiovascular system. Accurate information on anatomy, perfusion, function, tissue viability, and even on molecular mechanisms of the disease process, can be obtained non-invasively through various techniques, all contributing to refined diagnosis and prognosis, and to better understanding of the pathophysiology.

However, the large volume of information can be overwhelming for the clinician who finds it increasingly difficult to select the most appropriate technique to be used in a specific disease. As a result, patients are often submitted to multiple imaging modalities, which may provide redundant information, contributing to the rapidly increasing costs of health care.

This new book in the "The ESC Education Series" intends to provide the reader with the answer to the most critical question that we ask ourselves every day: "Which imaging modality should I use for this particular patient with this specific clinical presentation?". Thus, it is not another technique-driven textbook, but rather a practical guide on optimal use of non-invasive cardiovascular imaging. We trust that this practical, case-based approach, presented by the leading experts in imaging, will make this book an interesting and useful tool to most clinical cardiologists.

Michal Tendera, FESC
President, European Society of Cardiology, 2004–2006

Section one
Valve disease

CHAPTER 1
Mitral stenosis

Kewal Krishan Talwar and Manojkumar Rohit

Introduction

Mitral stenosis (MS) is a progressive disease that can result in serious complications which may be fatal unless an intervention enlarges the mitral valve orifice enough to permit adequate cardiac output. The predominant cause of MS is rheumatic heart disease. Approximately 25% of all patients with rheumatic heart disease have pure MS, and an additional 40% have combined MS and mitral regurgitation.[1]

When MS is symptomatic, the anatomic features consist of thickened mitral cusps, fusion of the valve commissures, shortening and fusion of the chordae tendineae, or a combination of these features. Characteristically, mitral valve cusps fuse at their edge, and fusion of the chordae tendineae results in thickening and shortening of these structures. Although the major obstruction in patients with MS is usually caused by fusion of commissures, it may be below the valve itself, secondary to fusion of the chordae, and this assessment is important because significant subvalvular involvement leads to suboptimal results with mitral commissurotomy or balloon dilatation.

Other rare cause of MS include congenital mitral stenosis (e.g. supramitral ring, cor triatriatum), mitral annular calcification, systemic lupus erythematosus, rheumatoid arthritis, and mucopolysaccharidoses.

Although there are multiple clues to the presence of MS by physical examination, they are often subtle and likely to be overlooked during a routine physical examination of an asymptomatic patient. The diagnosis of MS is often made when the patient presents with a complication (e.g. atrial fibrillation, embolism, acute pulmonary edema, or massive hemoptysis).

The various imaging modalities that are useful in confirming the diagnosis and assessing the severity of MS are discussed in this chapter.

Case Presentation

A 25-year-old woman was referred to our Institute with progressive shortness of breath for 6 months, with chest X-ray as shown in Fig. 1.1. This chest X-ray shows straightening of left heart border with pulmonary venous hypertension. How consistent is this with a diagnosis of MS?

Figure 1.1 Chest X-ray posteroanterior (PA) view showing straightening of left heart border, cephalization of pulmonary veins, and double atrial shadow.

The most frequent roentgenographic findings in MS include left atrial enlargement, redistribution of blood flow to the upper lobes of the lung, Kerley B lines, and enlarged pulmonary artery. Although their cardiac silhouette may be normal in the frontal projection, patients with hemodynamically significant MS almost invariably have evidence of left atrial enlargement.

Left atrial enlargement is one of the earliest signs of mitral stenosis; however, its presentation may be subtle and limited to enlargement of the left atrial appendage, causing a straightening of the left heart border. In more advanced cases, the left atrium is recognized as a double density and elevation of main stem bronchus on the postanterior film. Radiologic changes in the lung fields indirectly reflect the severity of MS. Redistribution of blood flow to the upper lobes correlates best with the degree of mitral valve obstruction. The presence of Kerley B lines is an important finding in patients with MS. These are fine parallel densities in the peripheral lung fields which are perpendicular to a pleural surface and are most frequently seen in the costophrenic sulci. The lines are caused by thickened interlobar septa and signify chronic pulmonary venous hypertension.

This finding is present in 30% of patients with resting pulmonary arterial wedge pressures less than 20 mmHg and in 70% of patients with pressures greater than 20 mmHg.

Case Presentation (Continued)

Although the chest X-ray is strongly suggestive of MS, management decisions need to be informed by details of anatomy and severity, so echocardiography remains the examination of choice for evaluating MS. Indeed, the chest X-ray may be unnecessary, and of course should be avoided in a pregnant woman. The patient's M-mode showed a decreased E-F slope and the posterior mitral leaflet moved anteriorly during diastole, indicative of MS (Fig. 1.2). Is the M-mode still useful?

Although a decreased E-F slope is almost always present in severe MS, it is not diagnostic of MS and it is an unreliable indicator of its severity. The specificity of the diagnosis of MS by M-mode echocardiography is greatly improved by visualizing the initial diastolic movement of the posterior mitral leaflet.[2] In the normal mitral valve, the posterior leaflet moves away from the anterior leaflet during early diastole. In MS, the posterior leaflet moves anteriorly during early diastole.

Case Presentation (Continued)

The patient's two-dimensional (2D) imaging in the short axis view showed a typical "fishmouth" appearance of severely stenotic mitral valve with a mitral valve area of 0.6 cm^2 (Fig. 1.3). Does this confirm the diagnosis of MS on 2D imaging, and how should we evaluate severity of MS and suitability for percutaneous mitral commissurotomy?

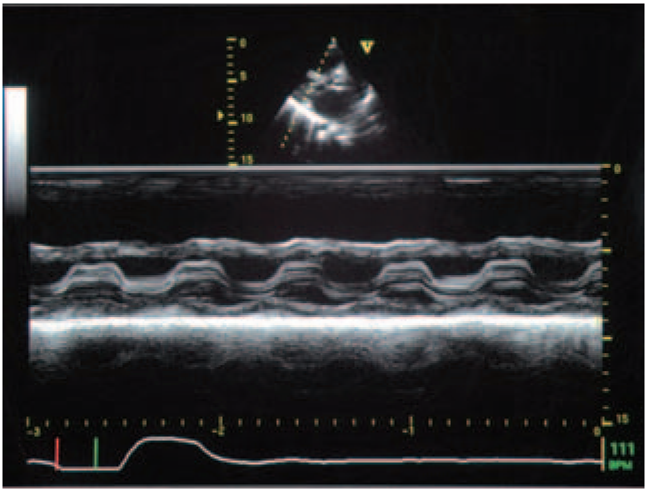

Figure 1.2 M-mode echo showing paradoxical posterior mitral leaflet (PML).

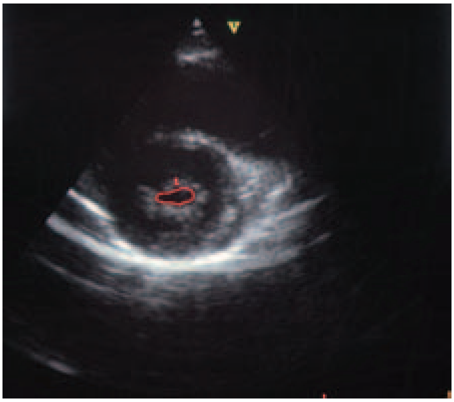

Figure 1.3 Parasternal short axis view showing typical "fishmouth" appearance of severely stenotic mitral valve (mitral valve area [MVA] 0.6 cm^2).

The short axis view allows the mitral valve area (MVA) to be measured by planimetry, although technical factors may compromise the accuracy of this method—not least the difficulty in ensuring that imaging is being performed at the tips of the leaflets. Heavily calcified leaflets may have indistinct borders that are difficult to trace, and there may also be dropout of echoes, leaving gaps in the area to be traced. The hallmark of MS on 2D echocardiography is thickening and restriction of motion of both mitral valve leaflets, with the predominant pathologic process being at the tips of the leaflets and proximal chordae. The abnormal motion of the leaflets is apparent in early diastole. Fusion of the commissures causes restriction in the motion of the tip of the anterior leaflet. The commissural fusion usually causes the posterior leaflet to move anteriorly during diastole with the larger anterior leaflet rather than moving posteriorly.

Doppler echocardiography assesses the severity of the stenotic lesion and color flow imaging is instrumental in determining associated mitral regurgitation. This is important because moderate mitral regurgitation (more than 2+) would be a contraindication to perform a closed procedure. MS produces characteristic changes in the mitral flow velocity pattern, involving an increase in the early diastolic peak velocity of flow and slower than normal rate of fall in velocity. The transvalvular pressure gradient can be measured continuously throughout diastole and correlates well with mean pressure gradient measured by cardiac catheterization. However, the pressure gradient is affected by heart rate, cardiac output, and valvular regurgitation in addition to orifice area, and hence it provides only a rough estimate of severity.

The pressure half-time is the time required for the instantaneous gradient across the valve to fall to half of the peak value (Fig. 1.4). This means of assessing MVA is usually sufficiently accurate for clinical use. The pressure half-time method is not valid for several days after mitral balloon valvuloplasty, probably because of a decrease in left atrial pressure without a commensurate improvement in left atrial compliance.

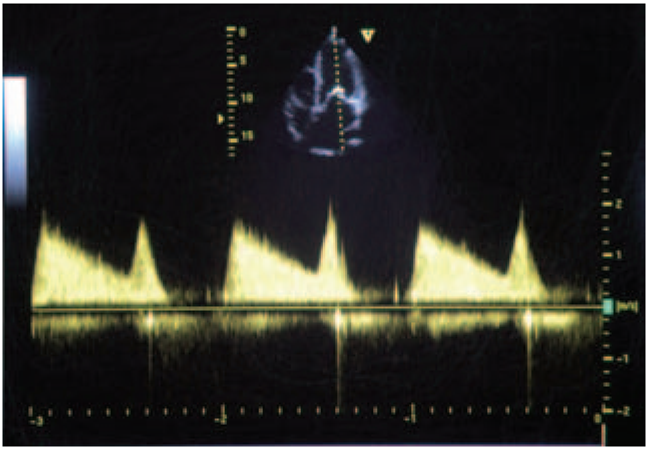

Figure 1.4 Continuous wave Doppler showing gradient across severely stenotic mitral valve.

Case Presentation (Continued)

Pressure half-time measurement in this case showed a mean gradient of 16 mmHg at the heart rate of 66 b/min (Fig. 1.4). Color Doppler performed on our index patient confirmed the stenotic mitral valve and fortunately showed no mitral regurgitation. Is the patient suitable for percutaneous intervention?

An echocardiographic scoring system developed by Wilkins *et al.*[3] has been used widely for assessment of suitability for percutaneous mitral commissurotomy. Leaflet rigidity, thickening, valvular calcification, and subvalvular involvement are each scored from 0 to 4. A score of 8 or less is usually associated with excellent immediate and long-term results, whereas scores exceeding 8 are associated with less impressive results. In our experience, slight commissural calcium is not a contraindication for percutaneous mitral commissurotomy, but when the calcium score is more than 2+, the incidence of restenosis is higher and thus surgical repair of the mitral valve is preferable. Significant subvalvular pathology is a more important determinant of suboptimal results following percutaneous mitral commissurotomy.

In addition to determining the presence and severity of MS, it is important to evaluate the heart for secondary effects of MS. These include left atrial enlargement, stasis, thrombus formation, and secondary pulmonary hypertension. The aortic, tricuspid, and pulmonic valves can likewise be directly evaluated for evidence of rheumatic involvement.

Case Presentation (Continued)

According to the Wilkins scoring system, our patient's mitral valve score was 6. There was no calcium on the mitral valve. This is an ideal candidate for percutaneous transmitral commissurotomy (PTMC) and a good result could be anticipated. What other steps are required before PTMC?

Transesophageal echocardiography

Transesophageal echocardiography (TEE) provides excellent images of the mitral valve leaflets, the left atrium, and the left atrial appendage (Fig. 1.5). Transthoracic imaging is usually diagnostic of MS and can accurately assess the severity of the stenosis. However, in some cases the transthoracic accoustic window may be inadequate. Multiplane TEE visualizes most of this anatomically complex structure and thrombus in the appendage can be accurately diagnosed, although experience is needed to avoid mistaking the pectinate muscle for thrombus.

TEE is well established as the gold standard for detecting thrombi in the left atrium (LA) and LA appendage, with a sensitivity and specificity of 100% and 99%, respectively.[4] The semi-invasive nature and safety of the test make it ideal for serial follow-up of thrombi in the LA body and appendage. TEE is also indicated if there is doubt regarding the presence or severity of mitral regurgitation and assessment of subvalvular pathology, if this is unclear on transthoracic echocardiography (Fig. 1.6). In our practice, all patients undergoing balloon valvuloplasty with atrial fibrillation and suspicion of clot on transthoracic echocardiography undergo TEE before the procedure. Finally, TEE may be used to guide the atrial trans-septal puncture during the balloon valvuloplasty procedure,[5] although we do not use TEE during the procedure at our institution.

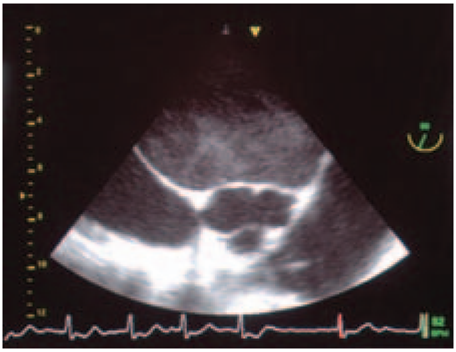

Figure 1.5 Transesophageal echocardiography (TEE) showing large left atrium with dense spontaneous contrast and amputated left atrial appendage.

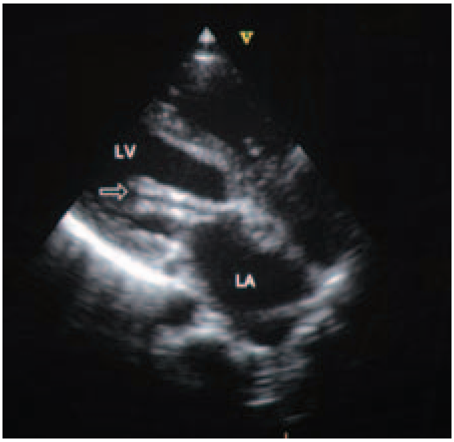

Figure 1.6 Parasternal long axis showing stenotic mitral valve and significant subvalvular thickening.

Case Presentation (Continued)

In this case, TEE was performed before PTMC. TEE showed dense spontaneous contrast but there was no clot in the left atrium (Fig. 1.5).

At cardiac catheterization before PTMC, left heart pressure was measured by retrograde catheterization of the left ventricle. Left atrial pressure was initially measured by pulmonary artery (which is accurate) and later by direct entry into the left atrium through trans-septal puncture. The mean gradient across the mitral valve was 15 mmHg before the PTMC (Fig. 1.7). The mitral valve was successfully dilated with a 26-mm Inoue balloon and the mean gradient across the mitral valve after the procedure was 4 mmHg with mitral valve area of 1.7 cm^2 and trivial mitral regurgitation.

Recently, there has been much interest in cardiac magnetic resonance imaging (MRI) and three-dimensional echocardiography in the assessment of valve lesions. We do not believe that MRI gives any additional information over echocardiography for MS, and MRI is expensive, time-consuming, and may be compromised by atrial fibrillation, which is not uncommon in MS. Three-dimensional echocardiography is a newer imaging modality and, in selected cases, will be useful, especially to assess the subvalvular apparatus.

Three-dimensional echocardiography

Real-time three-dimensional (R3D) echocardiography is a novel technique that permits visualization of mitral valvular anatomy in any desired plane of orientation. The use of R3D echocardiography in evaluation of mitral stenosis has been studied by Zamorano *et al.*[6] In 76 patients with significant MS, these

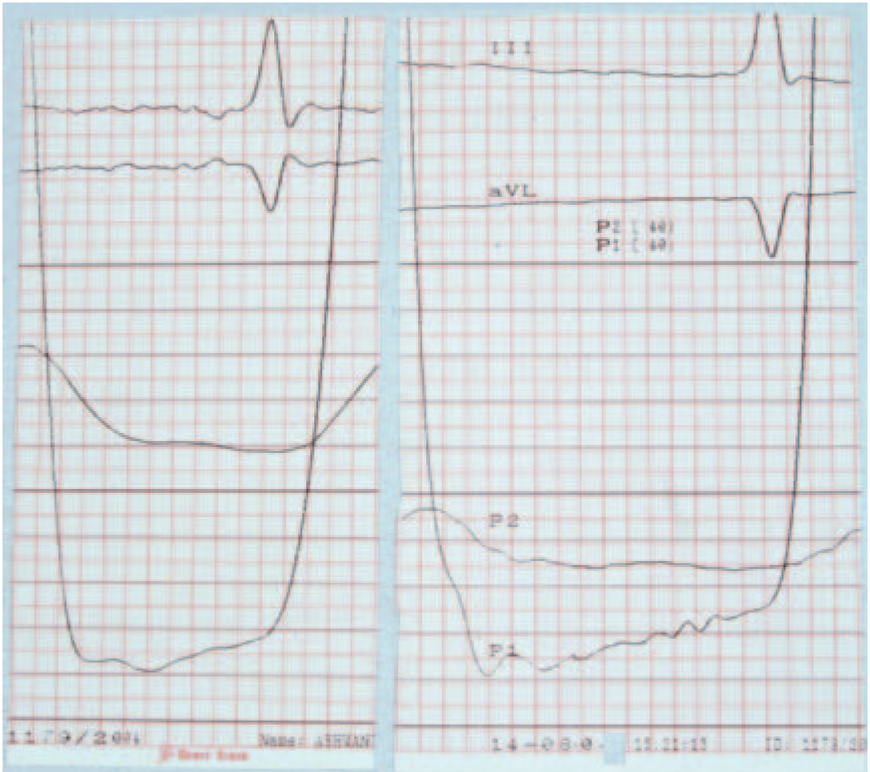

Figure 1.7 Left heart catheterization of left ventricle (LV) and left atrium (LA) tracing showing gradient across mitral valve before and after percutaneous transmitral commissurotomy (PTMC).

authors demonstrated that R3D echocardiography is a feasible, accurate, and highly reproducible technique for assessing MVA. MVA calculation with R3D echocardiography has the best agreement with invasive methods (average difference between both methods: 0.08cm^2). R3D echocardiography may improve the assessment of MS severity in patients with discordant results between different methods and in clinical scenarios where these methods have limitations, particularly after balloon valvoplasty.[7]

Magnetic resonance imaging

Magnetic resonance imaging can be used to identify the presence of valvular stenosis. The high-velocity flow across the narrow valvular orifice may be recognized as a signal void on cine MRI. Imaging may be performed with steady-state free precession (SSFP) imaging for semi-quantitative assessment of valvular dysfunction or with a standard breath-hold segmented gradient–recalled echoplanar imaging sequence (GE-EPI) (Fig. 1.8). A study by

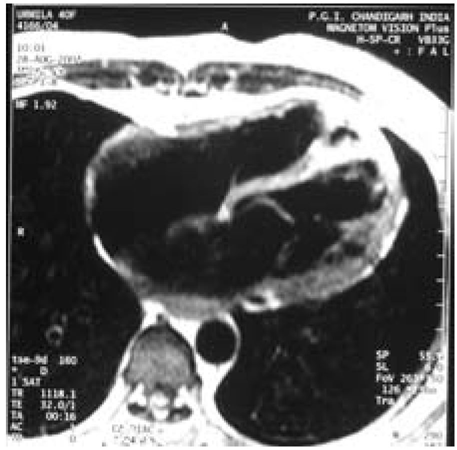

Figure 1.8 Magnetic resonance image (MRI) showing typical thickened doming mitral valve.

Krombach *et al*.[8] demonstrated both SSFP and GE-EPI sequences to share a high sensitivity (100%), although the image quality of SSFP was rated higher than GE-EPI. Peak flow velocity across MS can be quantified using velocity-encoded cine MRI (VE-MRI), analogous to echocardiography.[9] Lin *et al*.[9] reported excellent correlation of MVA by VE-MRI and Doppler echocardiography in 17 patients with MS ($r = 0.86$). An important strength of MRI for evaluation of mitral stenosis is that visualization of the spatial configuration of the mitral valve is excellent and quantification of transvalvular flow jet is unrestricted by echo windows. None the less, the MRI measurement is subject to several potential inaccuracies related to marginal temporal resolution, slice thickness, and signal loss.

Conclusions

In summary, 2D Doppler echocardiography gives sufficient information for imaging of MS. TEE has additional value for the evaluation of LA clot, and in some cases for assessment of mitral regurgitation and subvalvular pathology that is not clear on 2D echo. Of the newer imaging modalities, MRI and 3D echocardiography appear promising, and in selected cases they will avoid invasive (transesophageal or catheterization) studies.

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CHAPTER 2
Mitral regurgitation

Frank A. Flachskampf and Fausto Pinto

Introduction

Mitral regurgitation (MR) is the most frequent adult valvular lesion and the second most frequent reason for valvular surgery (after aortic stenosis). This chapter illustrates the evaluation of the patient with an asymptomatic pansystolic murmur. However, other typical clinical scenarios in which evaluation of MR is critical are the following:

1 The patient presenting with chronic dyspnea and a murmur suggesting MR.
2 The patient with severe dyspnea of abrupt new onset and a new murmur suggesting MR, with or without fever and signs of infection.
3 The patient with acute or chronic dyspnea, chronic coronary artery disease, an impaired left ventricle, and clinical or invasive evidence of MR.
4 The patient with acute severe dyspnea in the context of an acute coronary syndrome.

As in all forms of valvular regurgitation, assessment of severity is difficult, and no single parameter exists that is both easy to obtain and reliable for grading MR. Clinical assessment of MR therefore relies on gathering information on several characteristics and parameters, including mechanism, severity, duration, impact on the left ventricle, cardiac rhythm, amenability to repair, and other clinical data. These issues can almost always be resolved by careful clinical evaluation and application of modern imaging techniques (Table 2.1). Additionally, clinical and echocardiographic signs of the underlying etiology of MR should be sought systematically (Table 2.2).

Case Presentation

A 55-year-old man presents to his family physician for a routine check-up examination. He is physically active, completely asymptomatic, and in regular heart rhythm. A pansystolic murmur is heard over the apex, radiating to the axillary region. MR is suspected on clinical grounds and he is referred to a cardiologist for further evaluation.

Table 2.1 Imaging goals in mitral regurgitation.

Assessment of severity: mild, moderate, severe, and intermediary degrees, based on qualitative (color jet configuration, Doppler saturation, pulmonary venous flow) or quantitative (effective regurgitant orifice, regurgitant fraction, regurgitant volume) evaluation
Assessment of mechanism: normal/excessive/restricted mobility, degenerative changes (thickening, calcification, shortening), signs of endocarditis (vegetations), dilated annulus, structural damage, hypertrophic obstructive cardiomyopathy with systolic anterior motion, congenital anomaly
Assessment of location of regurgitant lesion (Fig. 2.4): anterior/posterior leaflet, anterolateral, central, posteromedial scallops (Carpentier nomenclature: P1–3, A1–3)
Assessment of left ventricular function: ejection fraction, left ventricular end-diastolic and end-systolic diameter (or volume), contractile reserve during stress
Other echocardiographic signs of underlying etiology: wall motion abnormalities for ischemic MR, leaflet thickening for mitral valve prolapse and systolic displacement, doming and thickening for rheumatic valvular disease

Table 2.2 Etiology of mitral regurgitation.

Degenerative chordal rupture with consecutive flail leaflet , typically in mitral valve prolapse
Degenerative fixation of a leaflet (in particular the posterior leaflet)
Ischemic:
- Impaired left ventricular function with dilatation causing eccentric pull of papillary muscles, restricted leaflet motion, and incomplete closure, together with mitral annular dilatation
- Papillary muscle rupture following myocardial infarction

Dilated cardiomyopathy (similar mechanism as in ischemic impaired left ventricular function)
Infective endocarditis with valvular destruction
Rheumatic valve disease
Hypertrophic obstructive cardiomyopathy
Congenital disease, e.g. mitral valve cleft
Mitral prosthetic dysfunction:
- Postoperative suture dehiscence/paravalvular leak
- Bioprosthetic degeneration
- Bioprosthetic endocarditis with leaflet destruction
- Ring abscess with large paraprosthetic leak or prosthetic dehiscence
- Prosthetic thrombosis with fixed position of occluder
- Fracture of mechanical prosthetic valve with occluder embolization

Rare causes: trauma, postvalvotomy regurgitation, Libman–Sacks endocarditis (systemic lupus erythematosus)

Chest X-ray

In a patient with dyspnea, a chest X-ray usually is performed as part of the basic work-up. Although heart and heart chamber enlargement resulting from MR (e.g. left ventricular and left atrial enlargement) can be diagnosed on chest X-ray, echocardiography is much more specific and accurate for these findings. Thus, nowadays the main information from a chest X-ray in MR is assessment of pulmonary congestion, ranging from mild pulmonary hypervolemia to frank pulmonary edema and pleural effusion. The absence of at least some degree of pulmonary congestion makes severe MR an unlikely cause for symptoms.

Case Presentation (Continued)

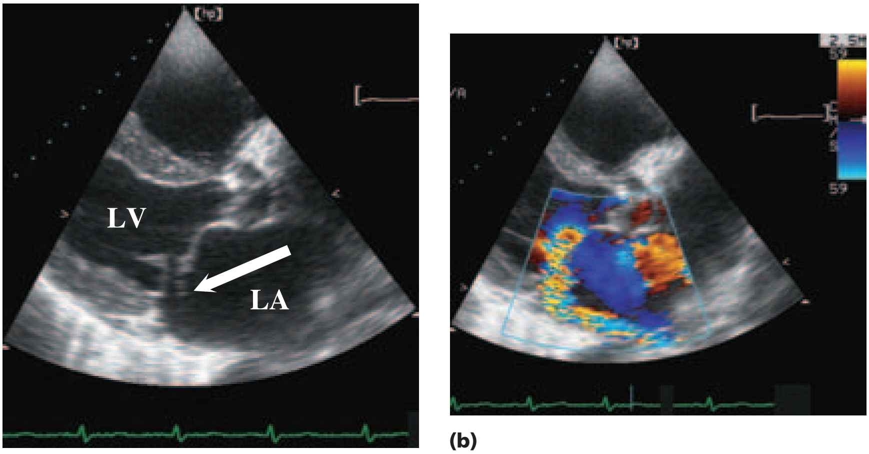
A chest X-ray demonstrates cardiac enlargement and mild pulmonary congestion. Kerley lines and pleural effusions are absent.

Which imaging modality for definite diagnosis?

In clinical practice, two imaging modalities are used to evaluate MR: echocardiography and contrast ventriculography by cardiac catheterization. Contrast ventriculography is invasive, costly, carries a small risk, is operator-dependent, and subject to large interobserver variabilities in interpretation.[1] Nevertheless, a well-performed ventriculogram showing no or minimal MR excludes substantial regurgitation. With higher degrees of regurgitation, the influence of injection technique, amount of contrast, catheter position, left ventricular function, and premature ventricular beats become more pronounced. It is particularly difficult to separate moderate from moderate-to-severe or severe degrees of regurgitation. Ventriculography should not be considered a "gold standard," and evaluation of MR per se is only very rarely the indication for cardiac catheterization. Nonetheless, a left ventriculogram is usually part of coronary angiography.

Echocardiography, while operator-dependent and having substantial interobserver variability, is now the dominant imaging technique to evaluate severity of MR, identify its mechanism, and devise therapy. Every patient with substantial MR should have an echocardiogram, and the decision to send a patient to surgery (especially if mitral repair is considered) mandates a thorough echocardiographic work-up, often with transesophageal echocardiography (Figs 2.1 & 2.2). Transesophageal echocardiography also is essential intraoperatively during repair surgery to assess results before the chest is closed.[2]

The use of three-dimensional (3D) echocardiography can improve the anatomic visualization of the different mitral valve scallops. For example, the location of prolapsing or flail leaflet segments is often immediately recognizable on 3D echocardiography, but requires expertise to pinpoint by conventional 2D echocardiography (Fig. 2.3). Communication with the surgeon may be aided by displaying "surgeon's views" from 3D data sets. Other benefits that may prove extremely useful, not only for diagnostic purposes, but also to help the surgeon in defining the feasibility and type of valve repair include a better appreciation



(a) (b)

Figure 2.1 (a) Severe mitral regurgitation caused by a flail anterior mitral leaflet. Echocardiographic parasternal long axis view. The arrow points at the systolic position of the tip of the anterior mitral leaflet within the left atrium, thus creating a large regurgitant opening between anterior and posterior leaflet. (b) Color Doppler echocardiography, same patient and view as in (a). A large eccentric, posteriorly directed (away from the flail leaflet) turbulent, high-velocity jet is visible in the left atrium. LA, left atrium; LV, left ventricle.

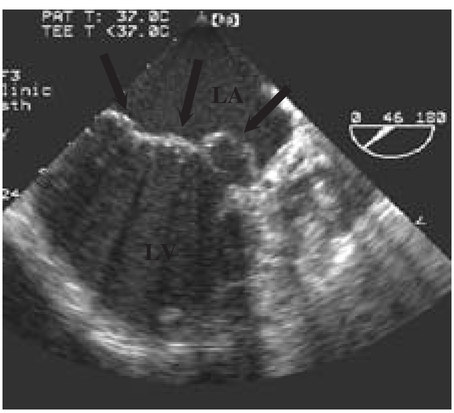


Figure 2.2 Mitral valve prolapse of both leaflets (arrows). Transesophageal view.

of the extension of the prolapse above the mitral annulus plane, the precise location of the diseased portion of the leaflets, its relation to important anatomic landmarks such as the valve commissures, and even the quantification of the volume of prolapsing tissue.[3] Furthermore, 3D echo is superior to 2D echo in calculating left ventricular volumes and ejection fraction.[4]