TRANSOESOPHAGEAL ECHOCARDIOGRAPHIC ASSESSMENT OF THE
MITRAL VALVE

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INTRODUCTION

The mitral valve is ideally suited to assessment by trans-oesophageal echocardiography (TOE) by virtue of its posterior location close to the oesophagus, separated from the probe by the blood filled left atrium. In comparison to transthoracic echocardiography (TTE), TOE provides superior resolution of mitral valve anatomy and allows more detailed evaluation of individual leaflet scallops and subvalvar chordal structures. This improves our understanding of the pathology and mechanism underlying the mitral regurgitation (MR), in addition to providing the surgeon with information regarding suitability for repair, together with choice of surgical technique. TOE also provides a systematic assessment of commissural fusion and calcification allowing detailed evaluation of rheumatic mitral stenosis and improving the selection of patients for balloon mitral valvuloplasty.

MITRAL VALVE ANATOMY

The mitral valvular complex comprises the annulus, leaflets, chordae tendinae, papillary muscles and the left ventricle. Also important is the left atrial musculature inserting to the leaflets and the walls of the myocardium to which the papillary muscles are inserted. Failure of the normal function of any of these components may lead to regurgitation.
The mitral annulus forms part of the fibrous skeleton of the heart and has a complex three-dimensional saddle-shaped structure. (Figure 1).

**Figure 1 . A: Fibrous skeleton of the heart. B: Saddle shaped mitral annulus**

During TOE, annular dimensions should be assessed at end-systole in both the mid-oesophageal commissural view (low, longer axis) and mid-oesophageal long axis view (high, shorter axis) [1]
The mitral valve is bi-leaflet. The Carpentier system of classification of the mitral valve leaflet segments is based on an anatomical view of the valve and has been adopted by the ASE/SCA guidelines and many cardiac surgeons. (Figure 2)[2]

**Figure 2 Carpentier nomenclature for mitral valve segments**

![Carpentier nomenclature for mitral valve segments](image)

The anterior (aortic) leaflet is in fibrous continuity with the aortic valve and has a base which attaches to one third of the annular circumference. The posterior (mural) leaflet is long and narrow, with a more extensive area of insertion to the annulus and is further divided by clefts into 3 scallops: anterolateral, middle and posteromedial or P1, P2 and P3. The middle or P2 scallop of the posterior leaflet is most commonly involved in prolapse of a floppy mitral valve. The opposing segments of the anterior leaflet are labelled A1, A2 and A3. The curved line of coaptation between the anterior and posterior mitral leaflets is generally regarded as having an anterolateral and posteromedial commissure at either end.
Two papillary muscles (anterolateral and posteromedial) support the mitral valve and each attaches to both leaflets via thin fibrous chordae tendinae (Figure 3)

**Figure 3 Chordal relationships (ASE/SCA terminology as per Carpentier) [3]**

First order chordae attach to the fibrous band running along the free edge of both leaflet tips ensuring coaptation of the contact surfaces (rough zone) of the leaflets. Second order chordae attach to the ventricular surface of both leaflets and contribute to ventricular function. Third order chordae arise from the ventricular wall and attach only to the posterior leaflet base as it inserts into the annulus. The anterolateral papillary muscle has dual blood supply from the LAD and circumflex coronary arteries and supports the anterolateral segments of each leaflet (A1, P1 and part of A2, P2). The posteromedial PM supports A3, P3 and part of A2, P2, it is usually supplied entirely by the right coronary artery and is therefore more susceptible to infarction and rupture with resultant severe MR.
MITRAL REGURGITATION

Carpentier’s functional classification of mitral regurgitation is widely used and describes the mechanism of MR according to whether leaflet motion is normal, excessive or restricted (Figure 4) [4].

**Figure 4. Carpentier’s functional classification of leaflet and chordal motion showing the direction of the MR jet [3]**

*Type I*
MR associated with normal leaflet motion is caused by annular dilatation resulting in failure of coaptation, or leaflet perforation complicating endocarditis.
Type II
Myxomatous degeneration is the commonest cause of MR. There is leaflet thickening and redundancy, chordal elongation with possible rupture and annular dilatation. (Figure 5).

Figure 5. Myxomatous mitral valve disease with a flail segment of the posterior leaflet

This prevents normal apposition of the leaflet tips resulting in a spectrum of pathology including billowing, mitral valve prolapse (MVP) and flail mitral valve with variable degrees of mitral regurgitation. (Figure 6)

Figure 6 Excessive leaflet motion (Carpentier type II) [5]

Billowing occurs when part of the body of the mitral leaflet rises above the annulus in systole, but the coaptation point remains below the plane of the annulus.

Prolapse occurs when the leaflet tip protrudes above the level of the mitral annulus in systole and is the leading cause of isolated mitral regurgitation and regurgitation requiring surgery. Early findings of a 3-15% prevalence of MVP have been overestimated because of non-uniform echo criteria. Due to the non-planar nature of the annulus, the 4-chamber view tends to over-diagnose the presence of prolapse. Analysis in the transthoracic long-axis parasternal or apical views are more specific and using this technique classic MVP, defined as superior displacement of the mitral leaflets >2mm during systole and maximal leaflet thickness of at least 5mm during diastasis, occurred in 1.3% of an unselected community based population [6].

Flail leaflet is a condition in which the edge of the leaflet is free flowing in the left atrium throughout systole. The aetiology is usually ruptured chordae associated with myxomatous
change or rarely papillary muscle rupture following myocardial infarction. The regurgitation generally develops more rapidly, and with increased severity compared with MVP.

Where a single scallop is involved, both prolapse and flail leaflets give rise to an eccentric jet of mitral regurgitation directed away from the affected leaflet, often hugging the wall of the left atrium. Jet eccentricity may, however, be misleading where more than one leaflet of the mitral valve is involved.

**Type III**

Leaflet restriction during systole and diastole (IIIa) is seen with leaflet thickening and calcification and subvalvular tethering from rheumatic valve disease. Leaflet restriction during systole (IIIb) occurs in cases of LV dysfunction where there is an altered geometric relationship between the papillary / chordal apparatus and mitral leaflets. With ventricular dilatation, the more globular shape of the LV results in lateral and apical displacement of the papillary muscles with resultant tethering or restriction of leaflet motion (Figure 4D). The leaflets are morphologically normal but become “tented” with loss of the usual systolic leaflet overlap or with a visible coaptation defect (figure 7).

**Figure 7. Apical displacement (“tenting”) of the mitral valve leaflet tips**

![Figure 7](image)

In dilated cardiomyopathy the MR is usually central. In the case of MR associated with ischaemic heart disease, there may be localised dilatation of the myocardium causing asymmetrical leaflet tethering and papillary muscle dysfunction resulting in an eccentric regurgitant jet which is directed towards the side of the lesion. (Figure 4C)
MITRAL VALVE REPAIR

The advantages of surgical repair over replacement include anatomical restoration of the mitral subvalvar apparatus with preservation of left ventricular function, reduced thromboembolic risk avoiding the need for long-term warfarin and excellent durability. Standard mitral valve repair involves firstly the correction of abnormalities of the leaflets and chordal apparatus, and may also include stabilisation of the annulus by implantation of an annular ring. Pre-operative assessment should involve detailed and systematic analysis of each part of the valve and sub-valvar apparatus (as described below) to determine the mechanism(s) of regurgitation. In addition, the surgeon should be alerted to the presence of annular calcification. Following mitral valve repair, echocardiography should be directed towards (i) assessment of the anatomical surgical result and (ii) assessment of the degree of mitral regurgitation. Therefore some knowledge of the surgical techniques employed is essential for accurate interpretation.

Isolated posterior leaflet prolapse is the most frequent cause of mitral regurgitation accounting for up to 60% of cases in most surgical series, and most commonly involving the middle scallop (P2). Repair of isolated P2 prolapse is highly successful and involves resecting a segment of leaflet with direct suturing of the defect. The traditional quadrangular resection has recently been replaced by triangular resection of the P2 scallop which allows better re-approximation and suturing of the resected margins. If the posterior leaflet is very wide and redundant, systolic anterior motion (SAM) can result, causing left ventricular outflow obstruction and mitral regurgitation. On occasion this necessitates a sliding plasty which consists of detaching the posterior leaflet from the annulus on both sides of the quadrangular resection and resuturing it closer to the opposite segment; this not only decreases its surface area but also redistributes the stress in the suture lines and annulus. [7]

Anterior leaflet prolapse is usually the result of elongation or rupture of the anterior leaflet chordae and repair is more challenging, involving the more demanding techniques of chordal shortening, chordal transfer, and implantation of artificial Gortex chordae. Repair can also be performed in bileaflet prolapse and rheumatic disease.

In mitral regurgitation post-myocardial infarction, over-reduction of the mitral annulus to promote coaptation of the restricted leaflets may be achieved using an annuloplasty ring. This decreases the radius of this cavity and the distance between the papillary muscles, consequently decreasing also the tethering effect on the mitral leaflets [7].
MITRAL STENOSIS

Mitral stenosis is almost always the result of a previous attack of rheumatic fever, although 50% of cases are subclinical. In symptomatic patients with severe mitral stenosis, percutaneous balloon mitral valvotomy (BMV) offers an alternative to surgical commissurotomy with equivalent results and has become the treatment of choice in young patients with pliant, non-calcified valves with predominant commissural fusion. With the disappearance of rheumatic fever in the Western world, most cases of mitral stenosis encountered in centres such as ours are elderly patients with more severe degenerative valve disease which is less suitable for BMV and echocardiography has a key role in the case selection. Occasionally, mitral stenosis is due to a congenital abnormality of the mitral valve. Here, it is important to exclude other cardiac abnormalities (especially stenotic lesions affecting the left heart and aorta).

Pre-valvuloplasty assessment.

The diagnosis and haemodynamic assessment of mitral stenosis can be made adequately from transthoracic echo. The leaflets are thickened initially at the tips with reduced mobility and a bowing motion in diastole. The subvalvar chordal structures are thickened, fibrosed and shortened. The Wilkins Score is the most widely used transthoracic scoring system to guide case selection for BMV and examines four aspects of valve morphology: leaflet thickening, leaflet mobility, leaflet calcification and subvalvar disease. Each is graded 1-4, higher scores (>8) indicating more severe degenerative disease and predicting a worse outcome from valvuloplasty [8].

The dominant mechanism of BMV is commissural splitting. Patients with extensive symmetrical commissural fusion gain an optimal result but calcified commissures resist splitting and result in leaflet tearing and mitral regurgitation. However, the Wilkins Score does not include commissural assessment and many have found this to be a weak predictor of outcome. Although both commissures can be visualised in the parasternal short axis view, in some patients resolution is poor, there is shielding from calcification and distinguishing between leaflet and commissural calcification is difficult. All these limitations are overcome by TOE which allows systematic scanning of each commissure for both the extent of fusion and localisation of calcification (figure 8). A Commissure Score based on this assessment ranging from 0-4 is a more powerful predictor of outcome and is of additional predictive value in patients with a low Wilkins Scores (<8). [8] In patients who are acceptable surgical candidates, a careful TOE assessment will guide appropriate selection of cases for either BMV or MVR. In those elderly patients with severely degenerative valves but prohibitive
surgical risk, BMV can offer a short term palliative option if there is any prospect of commissural splitting.

The presence of moderate or severe MR is generally a contraindication to BMV and TOE is useful for assessment of MR severity. Furthermore, TOE is valuable to confirm a thin interatrial septum suitable for transeptal puncture and to exclude left atrial appendage thrombus (Figure 9) since these structures cannot be reliably assessed from the transthoracic approach.

**Figure 8**
Calcified anterolateral commissure

**Figure 9**
Mobile LA appendage thrombus
With the probe in the midoesophagus, the four chamber view (0°) visualises the anterolateral part of the commissure and the coaptation line is scanned by advancing and withdrawing the probe. Conversely, the 2 chamber view (90°) visualises the posteromedial end of the commissure and the coaptation line is scanned by rotating the probe clockwise and anticlockwise.

One method for systematic examination of the mitral valve scallops begins with the probe in the mid oesophagus at 0° and withdrawn or flexed to a five chamber view visualising the most anterolateral part of the commissure (nearest the LA appendage) between A1, P1. Advancing the probe from the annulus to the centre of the valve visualises coaptation between A2 and P2 with further advancement and retroflexion revealing A3, P3.

At 60°-70° in the mid oesophagus the scan plane cuts the curved mitral commissure twice (commissural or “seagull” view) and visualises from left to right P3, A2, P1 with P3/A3 commissure on the left, A1/P1 on the right. (Figure 11) This view is useful for determining
which part of the commissure is regurgitant and confirms the presence of P2 prolapse by visualising the prolapsing P2 segment in the centre of the annulus above A2. (Figure 12D). Where valve regurgitation is due to annular dilatation, multiple regurgitant jets may be seen.

The mid oesophageal 2 chamber view is obtained at 90° with the LA appendage visible to the right. The scan plane cuts through P3 on the left and all 3 segments of the anterior leaflet on the right. This view shows coaptation between the posteromedial segments P3/A3 and visualises the posteromedial papillary muscle. (Figure 13A)

The midoesophageal long axis view (120°-150°) cuts the coaptation line perpendicularly through A2/P2 and the left ventricular outflow tract (LVOT) is in view. The papillary muscles are excluded. This is a useful view for evaluating SAM following valve repair and measurement of the vena contracta. In this view, clockwise (right) rotation sweeps the plane towards the posteromedial commissure and anticlockwise (left) rotation sweeps towards the anterolateral commissure.

The transgastric basal short axis view can allow visualisation of all mitral valve segments and the entire line coaptation line (Figure 13B). Colour Doppler in this view is helpful in localising the origin of the regurgitant jet and prolapsing segments can often be seen. This is especially useful in confirming the localisation of regurgitant jets seen in the mid-oesophageal views. The transgastric 90° plane provides a long axis view of the LV and mitral valve and is useful for assessing the subvalvular chordal anatomy, particularly in the presence of leaflet calcification where the mid-oesophageal views may have been unhelpful.
Figure 11  Inter-commissural view. Two jets of MR arising from P1-A2 and P3-A2 commissure

Figure 12.  Patient with severe MR due to prolapse P2 scallop
A: 4 chamber view showing A2/P2 coaptation point
B: 4 chamber view showing eccentric jet of MR directed away (anteriorly) from the lesion with large PISA
C: Systolic flow reversal in the left upper pulmonary vein
D: Inter-commissural view showing flail P2 scallop above A2
E: Long-axis view showing A2/P2 coaptation point and LVOT
Figure 13. Severe MR due to flail A3 segment
A: 2 chamber view showing the posteromedial part of the valve with A3/P3 coaptation point and eccentric jet of MR directed away from the lesion (posteriorly)
B: Transgastric view confirming the origin of the MR from the posteromedial part of the commissure
In mitral stenosis, the anterolateral and posteromedial comissures should be scanned systematically in the 0° and 90° mid oesophageal planes respectively from the annulus to the leaflet tips to determine the extent of fusion and presence of commissural calcification. Each comissure can be assigned a score to reflect the likelihood of splitting. A score of 0 is given if there is no fusion, or if commissural calcification, expected to resist splitting, is present. When non-calcified fusion is present, a score of 1 is given if the fusion is partial and a score of 2 given when such fusion is extensive. Each valve therefore has an overall ‘Commissure Score’ ranging from 0 to 4, a high score indicating extensively fused, non calcified commissures which are therefore more likely to split, whereas a low score reflects either minimal fusion or the presence of resistant commissural calcification [9].

**INTRA-OPERATIVE TOE**

The effects of general anaesthesia (after-load and pre-load reduction) will significantly reduce the severity of MR and therefore grading the MR in theatre pre-bypass can be unreliable. It is more important to focus on the mechanism of MR and suitability for repair. Echocardiographic predictors of an unsuccessful repair (or need for MVR) include annular diameter >5cm, mitral annular calcification, central MR jet and extensive bi-leaflet disease (3 or more scallops prolapse or flail)[11].

Immediately after coming off bypass, interpretation of colour Doppler and a definitive assessment of residual MR should be delayed until the left ventricle is adequately filled and appropriate vasoconstrictor therapy has been instituted to recover the awake haemodynamic state. Initial assessment should be directed towards systematic examination of the anatomy of the repair, with no residual prolapse, and a good area of coaptation between the leaflets. This should be made with knowledge of the details of the surgery undertaken. For example, where quadrangular resection has been performed, the posterior leaflet may appear short and less mobile. Second, it is important to assess the severity of any residual MR which may require a second bypass run. Significant MR could result from SAM, incomplete repair with residual prolapse, residual annular dilatation, suture dehiscence which may manifest as a leaflet perforation with the origin of the jet displaced from the coaptation line. Where the repair is borderline, the echocardiographer should assess other aspects of cardiac function that may affect the surgeon’s decision-making. (ie right and left ventricular function) Significant iatrogenic mitral stenosis is uncommon after repair but may be suggested by an elevated E wave velocity >2 m/s and shallow E wave deceleration slope and mean gradients of more than
5 or 6 mmHg. It is important, however, to remember that the alternative (mitral valve replacement) will have a significant residual gradient.

Following mitral valve repair, SAM of the anterior leaflet can cause LVOT obstruction (Figure 14) with significant posteriorly directed MR.

**Figure 14. SAM of anterior leaflet into the LVOT [1].**

SAM is best visualised in the 5 chamber or long–axis planes and the transgastric view can allow alignment of CW Doppler across the LVOT to demonstrate high velocities (3-5 m/s). Factors increasing the risk of SAM include a small, non-dilated LV, use of a small annuloplasty ring or excessive posterior mitral leaflet tissue. Severe SAM may require surgical revision as described above. However, most cases respond to medical therapy with volume loading and discontinuing inotropes/vasodilators [1].

New lateral or inferoposterior regional wall motion abnormality should alert the team to the possibility of circumflex coronary artery injury which is a rare but recognised complication of MV repair and replacement. The finding of severe aortic regurgitation post repair raises the possibility of aortic cusp injury during deep suture placement in the anterior annulus which may require AVR.
Assessment of MR severity and deciding when to operate

A number of ultrasound techniques are available for grading MR severity. Data from these should be considered collectively along with clinical information to decide whether the patient has significant MR to warrant intervention [12] (Table 1)

**Table 1. Echocardiographic parameters indicating severe MR**

<table>
<thead>
<tr>
<th>Echo parameter</th>
<th>Severe MR</th>
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<tbody>
<tr>
<td>LA / LV Size</td>
<td>Markedly increased (except in acute MR)</td>
</tr>
<tr>
<td>Jet area (%LA)</td>
<td>&gt;50</td>
</tr>
<tr>
<td>Spectral CW Doppler</td>
<td>Dense triangular regurgitation signal with early peak and late systolic reduction in velocity. E wave dominant with velocity &gt;1.5 m/s.</td>
</tr>
<tr>
<td>Vena contracta</td>
<td>&gt;7mm</td>
</tr>
<tr>
<td>Pulmonary vein flow</td>
<td>Systolic flow reversal</td>
</tr>
<tr>
<td>Regurgitant volume (ml/beat)</td>
<td>&gt;60</td>
</tr>
<tr>
<td>Regurgitant fraction</td>
<td>&gt;50%</td>
</tr>
<tr>
<td>EROA (cm²)</td>
<td>&gt;0.4</td>
</tr>
<tr>
<td>PISA</td>
<td>Large (radius &gt;1 cm at aliasing velocity 40cm/s) but less accurate in eccentric jets</td>
</tr>
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PISA = Proximal Isovelocity Surface Area (radius r)
EROA = Effective Regurgitant Orifice Area = \(2\pi r^2 \times \text{Alias v} \div \text{MR Vmax}\)

Regurgitant volume = EROA x MR TVI

In the setting of severe non-ischaemic MR, the ACC/AHA task force assign class I recommendations for surgery in patients with symptoms (NYHA II, III or IV), or in asymptomatic patients with evidence of LV disease (EF <60%, ESD >45mm) [13]. The guidelines stress the importance of using mitral valve repair when possible and in general suggest a lower threshold to intervene surgically when successful mitral valve repair appears likely. A recent study from the Mayo clinic showed that in asymptomatic patients with organic mitral regurgitation treated medically, an EROA >0.4 cm² was a powerful predictor of adverse events and should prompt early referral for surgery if valve repair is feasible. [14].
Patients with poor LV function require careful consideration. Severe LV impairment will underestimate the severity of MR. Additionally, in the setting of severe MR, afterload is falsely low as the left ventricle ejects into the left atrium and following restoration of a competent mitral valve the LV may struggle severely to overcome the true afterload and systemic vascular resistance.

**PROSTHETIC VALVES**

TOE has a key diagnostic role in patients with prosthetic valves and a systematic examination should confirm normal leaflet or disk motion, proper seating in the native annulus, normal blood flow profile with absence of paravalvular or transvalvular regurgitation in addition to estimation of transvalvular pressure gradients. A basic knowledge of commonly used bioprosthetic and mechanical valves, their flow dynamics and echocardiographic features is therefore essential. This is beyond the scope of our article and further reading is advised. The most commonly implanted mechanical valve is the bileaflet prosthesis (St Jude, Carbomedic). This is constructed of two semicircular leaflets suspended from four hinge points in a circular annulus surrounded by a sewing ring. Normal and abnormal function is shown in figures 15 and 16.

![Figure 15](image1.png)

**Figure 15 [15]**

Normal functioning bileaflet mechanical mitral valve

A&B: systolic frames showing normal small convergent jets of regurgitation (cleansing jets)

C&D: diastolic frames showing the sewing ring and two parallel open leaflets. Colour Doppler shows acceleration of flow through two lateral orifices and a small central orifice
Figure 16 [5] Bileaflet MVR
Paravalvular regurgitation originating outside the sewing ring is always pathological
REFERENCES (Essential reading highlighted in bold)


5. Perrino A, Reeves S. A practical approach to transesophageal echocardiography. Lippincott Williams & Wilkins. 2003


