4 Assessment of Functional Mitral Regurgitation by Cardiovascular Magnetic Resonance

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Abstract

In functional mitral regurgitation the leaflets of the mitral valve appear morphologically normal but do not close adequately due to left ventricular disease, either ischaemic or cardiomyopathic. While echocardiography generally remains the first line modality for investigation of mitral regurgitation, cardiovascular magnetic resonance (CMR) has complementary and additional roles. It enables assessment of the regurgitant fraction, mitral annular dimensions, left and right ventricular volumes and function, the effects of MR on the left atrium and pulmonary arteries, any pathology in other heart valves and the extent and distribution of any scarring and hence the viability of the left ventricle. CMR also contributes to assessment of the reparability of the mitral valve. This chapter covers the CMR acquisition methods used, some relevant approaches to image analysis and, finally, the limitations and strengths of CMR relative to echocardiography.

Keywords

Functional mitral regurgitation • Dilated cardiomyopathy • Chronic ischaemic mitral regurgitation • Viability • Cardiac MRI • Tricuspid regurgitation

Introduction

Mitral regurgitation (MR) is generally classified as organic (or primary) and functional (or secondary).

In organic MR, the main pathology is intrinsic disease in the mitral valve apparatus and the leaflets often look abnormal. The most common causes of organic mitral regurgitation are degenerative disease (e.g. Barlow's disease, fibroelastic deficiency, Marfan's syndrome,

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Ehlers- Danlos syndrome), rheumatic disease, endocarditis, ruptured papillary muscle, and congenital abnormalities such as cleft mitral valve.

In functional mitral regurgitation, the leaflets appear morphologically normal and open well in diastole, but cannot close (coapt) adequately in systole. This is caused by disease in the left ventricle which affects the function of the papillary muscles and also dilates the mitral annulus. The most common causes of functional mitral regurgitation are ischaemia and cardiomyopathies particularly dilated cardiomyopathy (DCM). It is therefore important to remember that in functional mitral regurgitation the underlying problem is in the left ventricle not the mitral valve.

While echocardiography remains the gold standard for the diagnosis and quantification of mitral regurgitation, CMR is considered to be the gold standard for the assessment of left ventricular size and systolic function. CMR has the additional ability to detect scar or fibrosis in the myocardium, which helps to assess myocardial viability in the case of ischaemic MR and to identify the underlying aetiology of disease in the left ventricle in the case of cardiomyopathies. The presence of myocardial fibrosis on preoperative CMR (in both ischaemic and non-ischaemic patterns; see below) is associated with increased postoperative risk of significant arrhythmia, cardiac pacing, and readmission to intensive care unit [1].

A thorough assessment of functional mitral regurgitation is feasible with CMR. The aims of the investigation are to:

1. Confirm the presence and mechanism of MR.
2. Quantify the severity of MR.
3. Assess mitral annulus.
4. Assess the left ventricular (LV) structure and function.
5. Assess the right ventricular (RV) structure and function and the tricuspid valve.
6. Look for consequences of MR on the left atrium and pulmonary arteries.
7. Look for significant pathology in other valves.
8. Comment on the reparability of the mitral valve. Assess the risk of repair failure if mitral valve repair is to be considered.
9. Assess left ventricular myocardial viability

In this chapter we will first discuss the CMR protocol for the assessment of mitral regurgitation and then explain how to analyse the images to provide answers to the above questions. The same protocol can be used for the assessment of organic mitral regurgitation by CMR.

Imaging Protocol

Scout Imaging

Transaxial, coronal, sagittal.

Thoracic Structures

Acquire transaxial set of steady state free precession (SSFP) or fast spin echo images through the chest. Use the still images to plan the cine images.

Standard Cine Images of the Heart

Acquire end expiratory breath hold steady state free precession (bSSFP) cine images of 2-chamber, 3-chamber (also known as left ventricular outflow tract view), 4-chamber, and short axis stack. Slice thickness 6–8 mm, with 2–4 mm interslice gaps to equal 10 mm.

Mitral Valve Stack

The mitral valve stack allows assessment of the scallops of both mitral leaflets for tethering, prolapse or regurgitation.

(a) Choose a basal short axis slice where mitral valve can be seen well (Fig. 4.1a).
(b) Acquire contiguous stack of oblique slices, 5 mm thickness, aligned orthogonal to the central part of the line of coaptation (Fig. 4.1b). Start from the superior (i.e., anterolateral) commissure adjacent to A1-P1 and progress towards the inferior (i.e., posteromedial) commissure adjacent

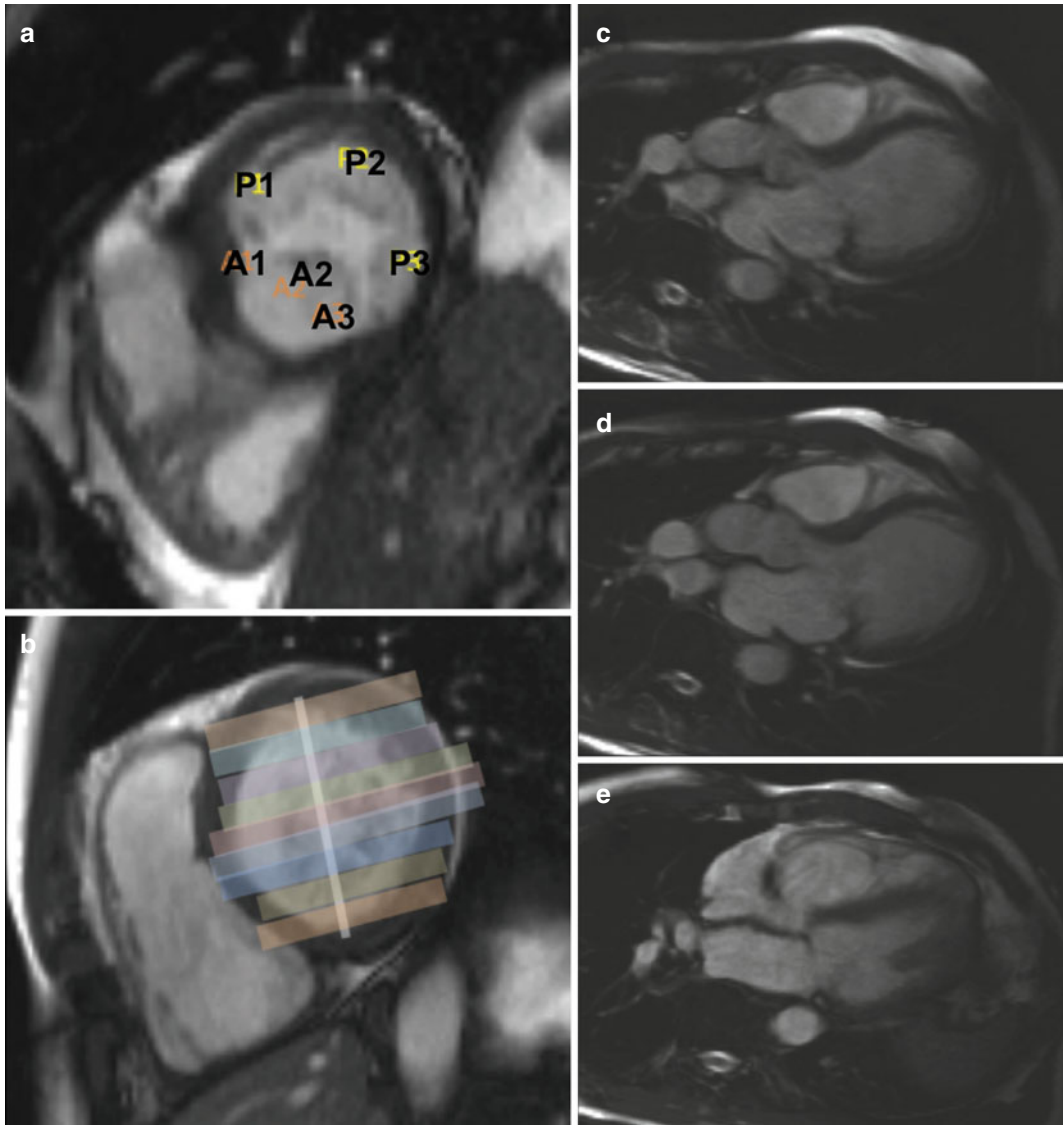

Fig. 4.1 (**a**) A basal short axis slice showing the half open leaflets of the mitral valve, with the P1, P2 and P3 scallops of the posterior leaflet. (**b**) A contiguous stack of oblique slices of 5 mm thickness is aligned orthogonal to the central part of the line of coaptation, covering the extent of the valve. (**c**–**e**) Three of the slices are shown which show the A1-P1, A2-P2 and A3-P3 regions of the valve, respectively

to A3-P3. Slices should be 5 mm in thickness with no gap. Typically 8–10 slices cover the length of the valve. Three are illustrated (Fig. 4.1c–e).

(c) Acquire further pair of oblique slices orthogonal to the oblique line of leaflet coaptation at each end of the valve adjacent to the commissures (across A1-P1 and A3-P3)

Aortic and Pulmonary Flow Study

Phase contrast through-plane velocity mapping of aortic and main pulmonary artery flow is performed. The velocity encoding (VENC) should be adapted to actual velocity using the lowest velocity without aliasing. The flow velocity and volume should be measured perpendicular to the

vessel distal to valve leaflet tips. A useful anatomical landmark for the aortic flow is to measure the flow just above the sinotubular junction at end systole. The recorded net forward volumes are used to calculate regurgitant volume and regurgitant fraction (see below). Because coronary flow does not pass though this slice, the aortic flow measured is typically about 5 % less than main pulmonary artery flow in the absence of a shunt.

In-plane/Through-Plane Fast Low Angle Shot (FLASH) of Mitral Valve (Optional)

FLASH cine acquisitions can have higher sensitivity than bSSFP for the visualisation of the regurgitant jets, depending on their echo time and other aspects of sequence design. A specific FLASH sequence may therefore be found useful for identification of the number, origin, and direction of the regurgitation jet (s), with possible qualitative assessment of the severity of regurgitation.

Phase Velocity Mapping of MR (Optional)

Through-plane phase velocity mapping of the mitral inflow gives a measurement of the anterograde velocity of the mitral inflow which is equal to mitral E and A velocities on pulsed wave Doppler echocardiography. Similar to echo, the measurement should be performed just distal to the tips of the mitral leaflets in diastole. The velocity encoding (VENC) should be adapted to actual velocity using the lowest velocity without aliasing. In addition, as an alternative to the FLASH approach, through-plane breath hold velocity mapping can be used, placed immediately on the atrial side of the closed MV, orthogonal to the jet(s), to map the locations and number of regurgitant lesions. Although MR jet velocity is expected to be higher, a VENC of 250 cm/s is usually adequate because of partial volume averaging, and can result in more effective visualisation than too high a VENC.

Viability

Assessment of myocardial viability and any scar or fibrosis is based on the late enhancement of the myocardium following gadolinium contrast injection. Gadolinium contrast is injected intravenously at a dose of 0.1–0.2 mmol/kg. After 8–10 min wait, the late gadolinium enhanced (LGE) images are acquired. Same views as for cine imaging (except for the mitral valve stack images) should be acquired. Slice thickness is the same as for cine imaging. In-plane resolution is about 1.4–1.8 mm. The acquisition duration per R-R interval should be below 200 ms but should be less in the setting of tachycardia to avoid image blurring. Inversion time is set to null myocardium. We recommend routine use of phase sensitive inversion recovery (PSIR) sequence in addition to magnitude images. The PSIR sequence requires less frequent adjustment of the inversion time (TI) and is particularly useful when the TI used to acquire the magnitude images was not optimal. Read out is usually every other heartbeat. It should be modified to every heartbeat in bradycardic patients and to every third heartbeat in tachycardic patients.

Analysis of the Images

Assess Mitral Valve Structure

Mitral leaflets and the mitral apparatus should be assessed on the 2-chamber, 3-chamber (i.e., LVOT view), 4-chamber, basal short axis, and MV stack cine images for any evidence of thickening, calcification, prolapse, restriction or tethering of the leaflets. The mitral valve stack images help to localise the pathology to mitral leaflet scallops (A1-A3, P1-P3) according to Carpentier's nomenclature. In functional mitral regurgitation, the leaflets appear structurally normal but the mitral annulus is often dilated. Annular dilatation is present when the anteroposterior (AP) diameter is more than 35 mm or the AP diameter/anterior leaflet length ratio is more than 1.3.

The following measurements can be readily performed on CMR (similar to echocardiography)

and help to quantify the extent of LV remodelling and the severity of altered geometry of the mitral valve, as well as the risk of postoperative failure of mitral valve repair.

– **In LVOT view**:
  (a) The length of the anterior and posterior leaflets and the anterior-posterior (AP) diameter of the mitral annulus (Fig. 4.2a). Severe annular dilatation (annular diameter >50 mm) is a predictor of operation failure.
  (b) Basal anterior septum in diastole (Fig. 4.2b). A septal bulge of more than 15 mm in thickness is a predictor of SAM and LVOT obstruction after repair.
  (c) C-Sept: C-Sept is the shortest distance between the basal septal bulge and the coaptation point in systole (Fig. 4.2c). Coaptation point is the point where the mitral leaflets coapt in systole. C-sept <2.5 cm is a predictor of SAM and LVOT obstruction after mitral valve repair. This is because the mitral valve repair procedure usually includes ring annuloplasty. The implantation of the ring moves the whole mitral valve anteriorly towards the LVOT and increases the risk of SAM and LVOT obstruction.
  (d) Aorto-mitral angle in systole and diastole. The aorto-mitral angle is the angle between the mitral annular plane and the aortic annular plane. When the aorto-mitral angle is more than 120°, the risk of SAM and LVOT obstruction following repair would be low. The sharper the aorto-mitral angle, the higher the risk of SAM and LVOT obstruction following repair.
– **In 4 chamber view**:
  (a) Tenting area: This is the area between the mitral annular plane and the mitral leaflets in mid systole in the 4 chamber view (Fig. 4.3a). A tenting area of more than 2.5 cm^2 is a predictor of unsuccessful repair. This is because a large tenting area implies that the leaflets are pulled down severely by the papillary muscles which itself means the LV is severely dilated and remodelled, and if the remodelling continues after the operation the patient will develop severe MR again.

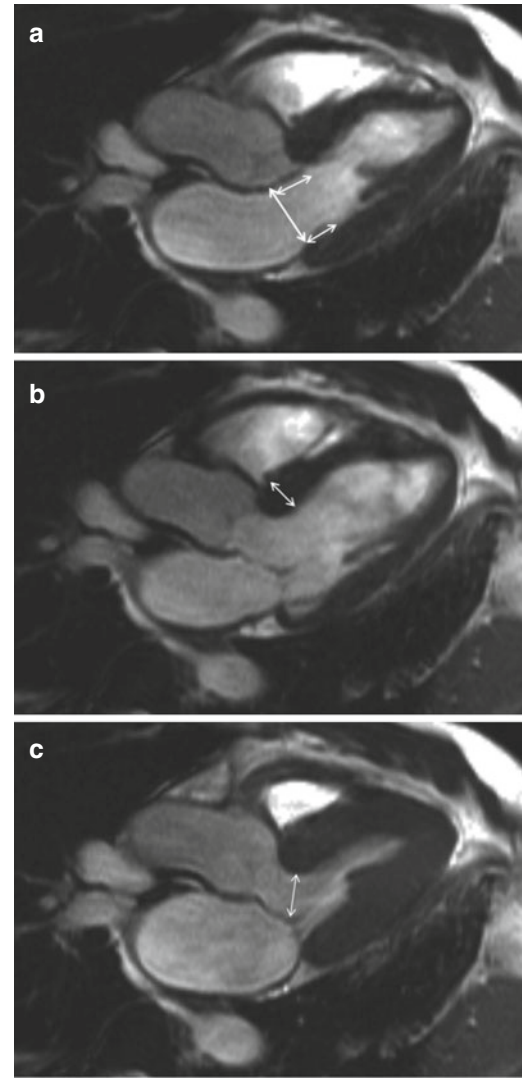

Fig. 4.2 The panels each show a frame of 3-chamber cines in which functionally relevant measurements can be made. (**a**) The lengths of the anterior and posterior leaflets of the mitral annulus and its anterior-posterior diameter. (**b**) The thickness of the basal septum at end diastole. (**c**) The shortest distance between the basal septal bulge and the coaptation point. The aorto-mitral angle, being the angle between the mitral annular plane and the aortic annular plane, can also be measured in systole and diastole. When the aorto-mitral angle is more than 120°, the risk of SAM and LVOT obstruction following repair would be low. The more acute the aorto-mitral angle, the higher the risk of SAM and LVOT obstruction following repair

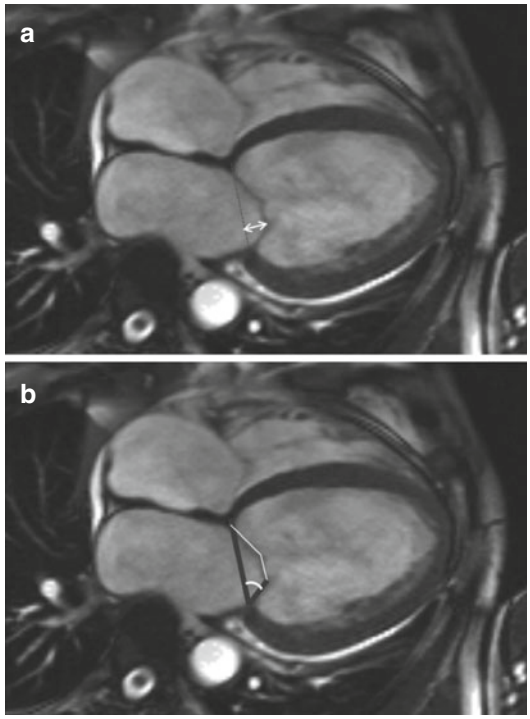

Fig. 4.3 In a four chamber view (**a**), the tenting area is the area between the mitral annular plane and the mitral leaflets in mid systole. The coaptation distance is the longest distance between the coaptation point and the mitral annular plane in systole. The posterior leaflet angle (**b**) is the angle which is the largest angle between the mitral annular plane and the posterior leaflet in mid-systole

(b) Coaptation distance: This is the longest distance between the coaptation point and the mitral annular plane in systole. A coaptation distance more than 1 cm predicts unsuccessful surgery and post-operative MR (Fig. 4.3b)

(c) Posterior leaflet angle: The posterior leaflet angle which is the largest angle between the mitral annular plane and the posterior leaflet in mid-systole is another indicator of LV remodelling and displacement of the papillary muscles. A posterior leaflet angle of more than 45° predicts unsuccessful operation (Fig. 4.3b).

– **In short axis view**:

The intercommissural diameter of the mitral annulus can be measured from the basal LV short axis view of the mitral valve (Fig. 4.4).

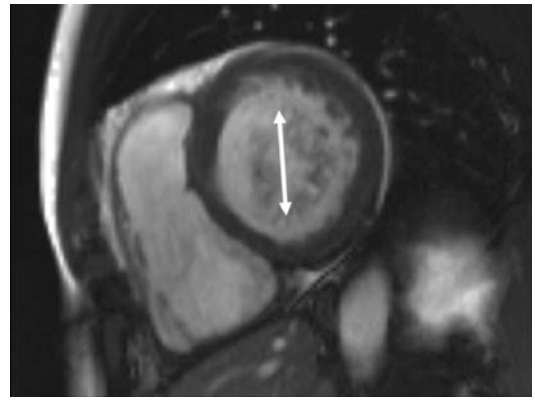

Fig. 4.4 The intercommissural diameter of the mitral annulus as measured from the basal LV short axis view of the mitral valve

There are two papillary muscles in the left ventricle: the anterolateral papillary muscle and the posteromedial papillary muscle. Both papillary muscles should be assessed for rupture, infarction, fibrotic elongation, and displacement. Due to the limited spatial resolution of MRI, chordae tendineae are usually not seen unless they are thickened.

Confirm the Presence and Mechanism of MR

The MR jet can be seen readily on the bSSFP and FLASH cine sequences.

Functional MR in DCM In functional MR secondary to DCM, both leaflets are symmetrically tethered which leads to a symmetrical tenting pattern of the mitral valve in systole (Fig. 4.3c). The same pattern can be seen in patients with ischaemic cardiomyopathy due to both anterior and inferoposterior infarction (see below). In dilated cardiomyopathy, functional MR is a consequence of:

(a) Apical displacement of the papillary muscles. This leads to tethering of both mitral leaflets towards the apex (Carpentier type IIIb).

(b) Dilatation of the mitral annulus so that the leaflets cannot reach each other to coapt in systole (Carpentier type I).

(c) Reduced contraction of the mitral annulus in systole. The normal contraction of the mitral annulus in systole (decrease in annular area in systole) is 25 % [2].
(d) Dysfunction of the LV and papillary muscles as part of the cardiomyopathic process.
(e) Dyssynchronous contraction of the papillary muscles and the left ventricle especially in the presence of left bundle branch block.

Functional MR in chronic ischaemic MR Chronic ischaemic MR is a consequence of previous myocardial infarction (MI) which has led to focal adverse remodelling of the left ventricle and the papillary muscle(s). Chronic ischaemic MR is commonly seen in one of the following patterns:

1. Following inferolateral MI: The inferoposterolateral left ventricular wall and the posteromedial papillary muscle (Carpentier type IIIb) are affected. In these patients the posterior leaflet appears tethered to the infarcted wall and the tenting pattern is asymmetrical. The MR jet is eccentric and posteriorly directed.
2. Following MI in more than one coronary territory: Chronic ischaemic MR can also be seen in ischaemic cardiomyopathy due to previous myocardial infarction in more than one coronary territory. In these patients there is global LV adverse remodelling and both papillary muscles are displaced and dysfunctional. This leads to tethering of both mitral leaflets and therefore the regurgitation jet is centrally directed.
3. It is also possible to see fibrotic elongation of an infarcted papillary muscle which will lead to prolapse of the affected mitral leaflet (Carpentier type 2).

Acute myocardial infarction can be complicated by rupture of a papillary muscle and lead to flail mitral leaflet and acute severe mitral regurgitation. This form of "acute ischaemic MR" which is a cardiac emergency is best classified under "organic MR". The affected patient is usually unwell and in pulmonary oedema and unlikely to be able to lie flat for 30–40 min for a CMR study. These patients are best assessed with echocardiography.

Quantify the Severity of MR

Similar to echocardiography, the severity of mitral regurgitation is initially assessed visually. The presence of an eccentric wall-hugging jet or the presence of a jet core indicates severe mitral regurgitation. To quantify the severity of mitral regurgitation with CMR, regurgitant volume and regurgitant fraction are calculated. The best and the most reproducible way of calculating mitral regurgitant volume (MRV) with CMR is to subtract the aortic forward stroke volume (AoSV) from LV stroke volume (LVSV), (Eq. 4.1). Care must be taken to perform these measurements meticulously. This formula can be used even in the presence of aortic regurgitation:

$$MRV\left(ml\right) = LVSV - AoSV \tag{4.1}$$

The regurgitant fraction (RF) is the ratio of the MRV divided by the LVSV multiplied by 100 (Eq. 4.2):

$$RF\left(\%\right) = \frac{MRV}{LVSV} \times 100 \tag{4.2}$$

Another way of calculating the mitral regurgitant volume is by subtracting the RV stroke volume (RVSV) from the LV stroke volume (Eq. 4.3). This is a less reliable method because RVSV is less reproducible compared to LVSV. Moreover, associated tricuspid regurgitation which often accompanies severe MR, or the presence of aortic or pulmonary regurgitation, invalidates the use of RVSV to determine MRV. Therefore, this formula can only be used in the absence of significant regurgitation in the other valves.

$$MRV\left(ml\right) = LVSV - RVSV \tag{4.3}$$

There are not yet established criteria for grading by CMR. However, regurgitant fractions (RF) calculated from CMR acquisitions have been correlated with echocardiographic grading in 83

patients with mitral regurgitation [3], although relatively few of these had more than moderate regurgitation. In the absence of established criteria for CMR, the findings of this study, derived from LV volume and ascending aortic flow measurements, can be noted: mild = RF ≤ 15 %, moderate = RF 16–24 %, moderate-severe = RF 25–42 %, severe = RF >42 %. If the regurgitant volume is more than 60 ml, the mitral regurgitation is severe.

Assess the Left Ventricular Structure and Function

From the bSSFP cine images, the left ventricle should be assessed for its shape, size, and systolic function. Spherical remodelling of left ventricular shape, dilatation of the left ventricle with displacement of the papillary muscles, and impaired left ventricular systolic function are all important contributors to the development of functional mitral regurgitation.

(a) Left ventricular shape: The normal left ventricle has a bullet-shaped geometry. In advanced stages of dilated and ischaemic cardiomyopathies, adverse myocardial remodelling leads to spherical remodelling of the left ventricular shape and the left ventricle looks more like a balloon than a bullet. Spherical remodelling is associated with adverse outcomes and worse prognosis because a spherically remodelled left ventricle is less likely to improve in response to medical or surgical treatment. The degree of left ventricular sphericity can be assessed visually. It can also be quantified with the measurement of "sphericity index" which is the ratio of maximum cavity diameter and cavity length in systole and diastole (Eq. 4.4):

$$Sphericity\ index = LV\ diameter\ /\ LV\ length \tag{4.4}$$

The normal ranges for sphericity index are given in Table 4.1 [4]. As it can be seen from the figures, women have more spherical ventricles than men.

Table 4.1 Normal left ventricular sphericity index range in the adult

	Men	Women
Sphericity index, diastole	0.35 ± 0.06 (0.22, 0.48)	0.4 ± 0.07 (0.27, 0.53)
Sphericity index, systole	0.20 ± 0.05 (0.10, 0.29)	0.23 ± 0.068 (0.09, 0.36)

(b) Left ventricular size: left ventricular end-diastolic and end-systolic volumes are calculated from the stack of short axis cine images using a computer-aided analysis package and are indexed for body surface area.

Left ventricular internal diameters in end systole and end diastole should also be measured and reported. This is because the current guidelines for the timing of mitral valve surgery in mitral regurgitation refer to the left ventricular internal diameters rather than volumes. Also most cardiologists and cardiac surgeons are more familiar with left ventricular diameter values than left ventricular volumes. Left ventricular internal diameters should be measured from the basal short axis slice immediately basal to the tips of the papillary muscles. Alternatively, the measurements can be performed from the 3-chamber (LVOT) view similar to parasternal long axis view in echocardiography.

(c) Left ventricular function: Assessment of the left ventricular systolic function begins with visual analysis of left ventricular global and segmental function. Wall motion is described as hyperkinetic, normal, hypokinetic, akinetic, and dyskinetic.

Unlike organic severe mitral regurgitation where the left ventricle is initially hyperdynamic, in functional mitral regurgitation the left ventricular function is either regionally or globally impaired. Regional wall motion abnormalities and abnormal myocardial wall thinning in a coronary territory suggest previous myocardial infarction. Inferoposterior myocardial infarctions in the RCA or LCx territories commonly affect the posterolateral papillary muscle and lead to chronic ischaemic mitral regurgitation. In these patients, the posterior mitral leaflet appears teth-

ered to the infarcted segment and the regurgitation jet is posteriorly directed.

Quantitative analysis of the LV systolic function is based on the measurement of the LV end-diastolic and end-systolic volumes by the computer-aided analysis package as described above. LV ejection fraction, stroke volume, and cardiac output are calculated and reported. It is of utmost importance that these measurements are performed meticulously because the calculated LV stroke volume is used to quantify mitral regurgitant volume and fraction.

Assess the Right Ventricular Structure and Function and the Tricuspid Valve

Assessment of the RV begins with visual analysis of RV structure and function.

In normal subjects, RV looks smaller than LV in the 4-chamber view. When the RV looks larger than LV, it is dilated. When the RV looks larger than LV and forms the apex of the heart, it is severely dilated.

It is important to take note of any RV hypertrophy as it can be a sign of pulmonary hypertension. RV hypertrophy is defined as RV free wall thickness >5 mm.

It is also important t to look for systolic and/or diastolic flattening of the ventricular septum. Flattening of the septum makes the LV cavity look like the letter "D". Systolic flattening of the ventricular septum is a sign of RV pressure overload e.g., due to pulmonary hypertension and in the absence of primary disease in the lungs or pulmonary vasculature, indicates significant left heart disease e.g., severe mitral regurgitation. Diastolic flattening of the ventricular septum is a sign of RV volume overload and is commonly seen in severe tricuspid regurgitation, severe pulmonary regurgitation, and severe left to right intracardiac shunt e.g., large atrial septal defect. Depending on the chronicity of the underlying pathology, the RV systolic function may be hyperdynamic, normal, or globally impaired.

Pathological regional wall motion abnormalities are not common in RV and if present are usually due to either previous myocardial infarction (right coronary artery territory) or arrhythmogenic right ventricular cardiomyopathy. Regional wall motion abnormality in the RV free wall is common around the insertion point of the moderator band and is considered to be a normal variant.

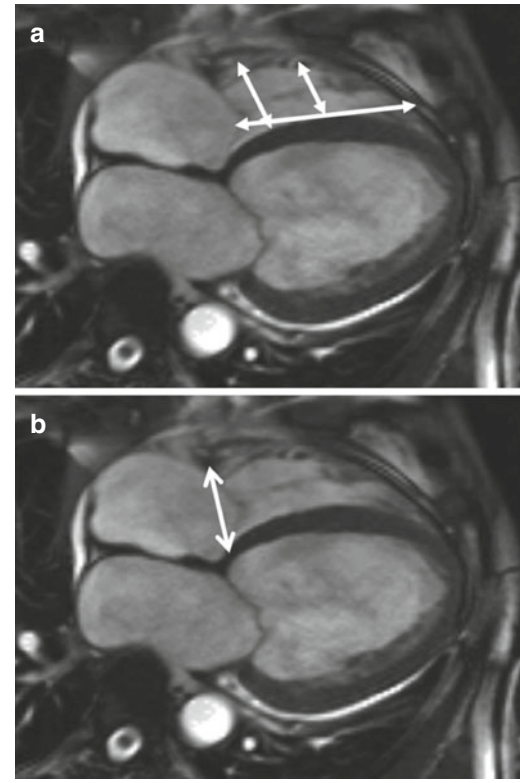

Fig. 4.5 (**a**) in a four chamber view, internal diameters of the RV (base, mid, length) can be measured, as in echocardiography. (**b**) The antero-posterior diameter of the annulus can also be measured

For the quantitative analysis of the RV size and systolic function, RV end-diastolic and end-systolic volumes, stroke volume, and ejection fraction are calculated from the stack of short axis cine images using a computer-aided analysis package. All measurements except for the ejection fraction are indexed for body surface area. RV volumes can also be measured from a transaxial stack of the heart. RV internal diameters (base, mid, length) can be measured from the 4-chamber view in a similar fashion to echocardiography (Fig. 4.5a).

Severe tricuspid regurgitation is most commonly functional and due to annular dilatation secondary to RV and/or right atrial dilatation.

Severe tricuspid regurgitation can gradually make itself worse by causing RV volume overload which leads to further dilatation of RV and tricuspid annulus. It is important to report tricuspid regurgitation and assess its severity. The severity of tricuspid regurgitation can be assessed visually. It can also be quantified by subtracting the pulmonary stroke volume from RV stroke volume (Eqs. 4.5 and 4.6):

$$\textit{Tricuspid regurgitant volume} = \textit{RV stroke volume} - \textit{pulmonary stroke volume} \tag{4.5}$$

$$\textit{Tricuspid regurgitant fraction} = \frac{\textit{Tricuspid regurgitant volume}}{\textit{RV stroke volume}} \tag{4.6}$$

Tricuspid annulus diameter can be measured in 4-chamber view and should be mentioned in the report (Fig. 4.5b). In patients who are candidates for mitral valve surgery, most surgeons consider concomitant restrictive ring annuloplasty of the tricuspid valve if there is more than mild tricuspid regurgitation and the tricuspid annular diameter is more than 4 cm.

Look for Consequences of MR on the Left Atrium and Pulmonary Arteries

Severe MR is usually associated with dilatation of the left atrium. Severe MR can also be associated with pulmonary hypertension which manifests itself with dilatation of the pulmonary arteries on transaxial images.

Look for Significant Pathology in Aortic and Pulmonary Valves

It is important to exclude significant aortic stenosis, aortic regurgitation, and significant disease of the pulmonary valve because it may change the management plan. Both aortic and pulmonary valves should be assessed visually on the cine images of the LVOT and RVOT. If the cine images indicate pathology, the valve should be thoroughly assessed with short axis cine image of the valve to assess opening valve area (in the case of valvular stenosis) and/or coaptation failure (in the case of valvular regurgitation). Appropriate flow studies should be performed with phase contrast velocity mapping to quantify the severity of valve disease.

Assess Left Ventricular Myocardial Viability and Contractile Reserve

Myocardial fibrosis and viability is assessed using LGE studies. Assessment of myocardial viability is particularly important in patients with ischaemic MR and can affect the clinician's decision regarding revascularisation or conservative treatment. The aim of viability testing is to make a distinction between reversible and irreversible myocardial injury. If the damage to the myocardium is reversible, revascularisation can improve LV and papillary muscle function and lead to improvement in the severity of MR. Remember that in the context of LGE studies, the term "non-viable" is used to predict low likelihood of improvement in contractility following revascularisation and the so-called "non-viable" myocardial segments often have an epicardial rim of non-infarcted myocardium.

Further assessment of myocardial viability and LV contractile reserve is possible with low dose dobutamine infusion (see below). In patients with impaired left ventricular systolic function, this information helps to predict the likelihood of reverse LV remodelling after mitral valve repair.

In non-ischaemic cardiomyopathies, assessment of the pattern of myocardial fibrosis can be helpful in determining the underlying aetiology.

(a) LGE studies: The pattern and extent of LGE should be assessed. For most clinical indications, visual assessment is sufficient. The LGE pattern may be ischaemic or non-ischaemic:
   (a) Ischaemic pattern: is characterised by subendocardial hyperenhancement in a coronary artery perfusion territory. The location and extent of subendocardial scar is reported using the American Heart Association 17-segment model. Comparison of the LGE images with the corresponding cine images is recommended for correct interpretation of viability. The average transmural extent of the LGE is estimated within each myocardial segment and represents the transmural extent of the non-viable myocardium. LGE transmurality is reported as 0%, 1–25%, 26–50%, 51–75%, 76–100%. There is an inverse relation between the transmural extent of LGE and the likelihood of improvement in contractility after revascularisation. The smaller the % thickness of LGE, the higher the likelihood of increased contractility after revascularisation (Table 4.2).
   (b) Non-ischaemic pattern: usually spares the subendocardium and is limited to the mid- wall or epicardium. If the subendocardial hyperenhancement is global, then a non-ischaemic pathology such as amyloidosis or endomyocardial fibrosis is more likely. In dilated cardiomyopathy, mid-wall fibrosis is seen in 40–50% of patients and is more common in the ventricular septum.

(b) Contractile reserve:
   The left ventricular contractile reserve can be assessed using low dose dobutamine stress at 5 and 10 mcg/kg/min.

Table 4.2 Relationships between the % LGE transmurality and the likelihood of functional recovery after revascularisation

% LGE transmurality	Likelihood of functional recovery post-revascularisation (%)
0	80
1–25	60
26–50	40
51–75	10
>75	~0

The Relative Strengths and Limitations of CMR

Among its several strengths, magnetic resonance is arguably the most versatile of the imaging modalities. This is by virtue of the control afforded at tissue level, by magnetic field gradient applications, over the interactions of radio signals with the spins of protons in relation to their surroundings. The unpaired protons of hydrogen occur mainly in the water of blood and tissues, and in fat. The radio signals that convey energy and information to and from them through the body are non-destructive and non-ionising. As long as precautions are taken to avoid the possible dangers that might be associated with metal objects or wires in the magnet, CMR is safe and non-invasive. However, the versatility, complex physics and elaborate technology of CMR mean that it is best performed in specialised units, with significant costs associated.

The large, unrestricted fields of view of CMR give relatively comprehensive access to the several structures and flow features relevant to functional MR. Of note, the mitral annulus, leaflets, chordae, papillary muscles, their insertions in the LV wall, and the tissue characteristics and mobility of the myocardial wall itself are all relevant to adequate assessment, as well as visualisation of the regurgitant jets. All these, except typically for more linear chordae, can be visualised and interrogated by CMR. However, the spatial resolution of typical acquisitions may be suboptimal. Although the pixel size (typically about

1.2 × 1.5 mm) can give clear images with good blood-tissue contrast, the thickness of the image slice (typically 5–8 mm for cine imaging) needs to be borne in mind, particularly when imaging thin structures such as valve leaflets or narrow jets. These can nevertheless be seen well if the imaging plane lies perpendicular to the plane or line of the structure, which then minimises the effects of partial volume averaging.

CMR allows arguably the most accurate and reproducible measurements of LV cavity volumes and stroke volume, LV mass and aortic flow, which together enable quantification of mitral regurgitation. However, such quantification is indirect and dependent on more than one type of measurement, so it is important to recognise possible sources of error.

Unfortunately, CMR image quality tends to be compromised by irregularity of heart rhythm, notably atrial fibrillation. This is because the cine images are typically acquired, with ECG triggering, over the 10–25 heartbeats of a single breathhold. Beat-to-beat variations of structural position and flow degrade image quality, particularly of the finer structures and features. This includes any vegetations of endocarditis, which are rarely seen adequately by CMR.

The heavy equipment and magnetic field of CMR mean that it is not available for bedside or intra-operative investigation.

In conclusion, while echocardiography generally remains the favoured modality for imaging and assessing mitral valve disease, CMR has complementary strengths. Not only does it offer an alternative in patients with limited ultrasonic access, but it can also add information, particularly on ventricular volumetric assessment and myocardial tissue characterisation.

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