Functional Classification of Mitral Regurgitation

A functional classification for MR has also been proposed. This classification was originally described by Carpentier as a functional rather than aetiological classification based on the surgical aim of mitral valve reconstruction which is to restore normal valve function rather than normal valve anatomy[8.6]. This functional classification is categorised by the opening and closing motions of the mitral leaflets such that type I refers to normal leaflet motion, type II refers to increased leaflet motion and type III refers to diminished or restricted leaflet motion (Fig. 8.37).

In the type I classification there is normal leaflet motion and MR occurs when there is a annular dilatation; leaflet perforation and cleft leaflets also fall into this classification. Type II refers to MR that occurs due to increased leaflet motion; this type therefore includes MVP, cordal elongation or rupture, and/or papillary muscle elongation or rupture.

Type III refers to MR that occurs due to restricted leaflet motion. This type can be further subcategorised into:

- type IIIa: restricted mitral leaflet motion during both diastole and systole that is caused by leaflet thickening and/or retraction, cordal thickening and/or shortening, and/or commissural fusion. This classification of MR is most often associated with rheumatic mitral valve disease.
- Type IIIb: restricted leaflet motion during systole only. The most common causes for this type of MR include LV dilatation with apical papillary muscle displacement as seen in ischaemic heart disease.

Importantly, it should be noted that more than one Carpentier type may coexist in any given individual. Furthermore, the Carpentier classification does not describe all mechanisms of MR. For example, MR due to SAM of the anterior mitral leaflet as seen with hypertrophic obstructive cardiomyopathy is not included in this classification.

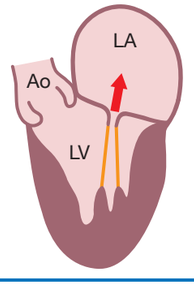
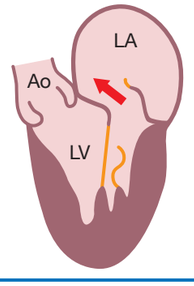
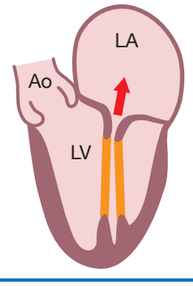
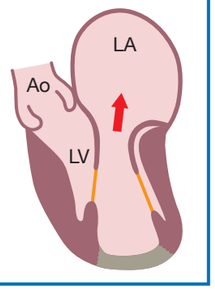
Functional Type	Type I	Type II	Type III	
			IIIa	IIIb
Cusp Motion	Normal	Increased	Restricted during systole & diastole	Restricted during systole only
Example lesion	• Annular dilatation • Leaflet perforation • Cleft valve	• Prolapse • Cordal elongation • Cordal rupture • Papillary muscle elongation • Papillary muscle rupture	• Leaflet thickening and/or retraction • Cordal thickening and/or shortening • Commissural fusion	• Papillary muscle displacement and/or leaflet tethering
Illustrative example				

Figure 8.37 This figure illustrates the functional classification for mitral regurgitation (MR). See text for further details. Ao = aorta; LA = left atrium; LV = left ventricle.

Table 8.7 Principal Mechanisms of Ischaemic Mitral Regurgitation

Mechanism	Cause of Mitral Regurgitation
Left ventricular (LV) remodelling	Decreased closing forces due to LV global or regional dysfunction and LV geometric distortion (LV dilatation) can lead to MR. LV geometrical changes result in displacement of papillary muscles which pulls the leaflets away from each other preventing adequate systolic leaflet coaptation.
Increased systolic tethering of the mitral leaflets	Papillary muscle displacement restricts systolic leaflet motion resulting in valve tenting or tethering which prevents complete leaflet coaptation during systole. Leaflet tethering may be symmetric (global LV remodelling) or asymmetric (regional LV remodelling).
Reduced leaflet closing forces	LV systolic dysfunction prevents the production of sufficient ventricular closing forces to adequately close the valve.
Annular dilatation	Annular dilatation pulls the leaflets further from one another preventing them from coapting during systole.

[8.6] Carpentier A. Cardiac valve surgery--the "French correction". *J Thorac Cardiovasc Surg.* 1983 Sep;86(3):323-37.

Pathophysiology of Mitral Regurgitation

The pathophysiology of MR can be discussed in terms of acute and chronic MR.

Acute, severe MR may occur due to PMR following an AMI or acute cardiac trauma. In the acute situation, the mitral regurgitant volume (RV) fills a normal sized LA as the LA has not had time to compensate for increased systolic volume by dilating. This results in a marked elevation in the LA pressure. In addition, as the pulmonary capillaries are not 'protected' by a dilated LA and are subjected to the markedly elevated LA pressure, the pulmonary capillary pressures also rise markedly and acute pulmonary oedema and PHTN ensue. Furthermore, the LV end-diastolic volume (LVEDV) is increased because the LV receives the mitral RV along with the normal forward flow returning from the lungs. At the same time, LV afterload is reduced because the LV is essentially ejecting into the LA. As a result, the LV is able to eject more completely so the LV end-systolic volume (LVESV) is decreased. Therefore, as the LVEDV is increased and the LVESV is decreased, the LV ejection fraction (LVEF) is usually greater than normal or "supernormal". However, because the majority of the SV is being ejected into the LA, the total forward SV to the body is significantly decreased resulting in a low cardiac output.

In the case of chronic 'compensated' MR, the heart has had time to develop compensatory mechanisms. In particular, chronically increased LA pressure results in LA dilatation and when the LA is dilated and still compliant, it can accommodate the mitral RV at a lower pressure, so that LA pressure is only mildly elevated. Over time, the increased LV volume causes increasing LV dilatation and hypertrophy. This allows for a larger increase in total SV so that forward SV is normal (see the Frank-Starling principle in Chapter 2). The LVEF also remains increased. However, as the severity of MR increases over time, the ability of the LV to augment its systolic function reaches its limit and LV systolic function then decreases. This is referred to as chronic 'decompensated' MR. In this situation, LV contractile function is reduced due to muscle damage caused by prolonged and severe LV volume overload. As a result, the weakened LV can no longer shorten adequately so the LVESV increases. This leads to a decrease in total and forward SV with a subsequent increase in LA and LV filling pressures. In addition, there is a further increase in LVEDV, without a concomitant decrease in LVESV; therefore, both the total and forward SV are decreased. The LVEF also declines but often remains within the "normal" range but this LVEF does not reflect the true contractile function of the ventricle. Furthermore, in chronic MR pulmonary hypertension is also common due to the backwards transmission of increased LA pressures to the pulmonary capillary bed.

Clinical Manifestations of Mitral Regurgitation

The clinical signs of MR are caused by backward flow of blood across the mitral valve, leading to increased LA pressures. The clinical manifestations of MR depend on the severity of MR as well as whether MR is acute or chronic.

Patients with acute MR typically present with frank pulmonary oedema, hypotension, and signs and symptoms of cardiogenic shock.

Patients with chronic MR may be relatively asymptomatic if the LV systolic function remains normal. In patients with chronic, symptomatic MR, the most common symptoms include generalised weakness, fatigue, and exercise intolerance. In more severe cases of chronic MR, signs of congestive heart failure, evidence of pulmonary congestion and pulmonary oedema may be present.

Role of Echocardiography in Mitral Regurgitation

The aims and objectives of echocardiography in the assessment of MR are to:

- Determine the aetiology of the lesion,
- Assess LA size,
- Assess LV size and systolic function,
- Estimate the severity of the regurgitation,
- Estimate RVSP.

Aetiology of Mitral Regurgitation

As discussed previously, the anatomy of the mitral valve complex consists of many components and disorders of any of these components can result in MR (see Table 8.5). Two important conditions that warrant special consideration are DMVR (MVP) and MR due to ischaemia.

'Physiological' or a mild degree of MR is commonly encountered in the normal population with the incidence of MR increasing with advancing age. MR may also be described in terms of functional or organic regurgitation. Organic or primary regurgitation refers to diseases that involve the leaflets and their supporting apparatus; that is, MR occurs due to intrinsic valvular disease. Functional or secondary regurgitation refers to diseases that affect the LV and LA resulting in altered ventricular geometry; so MR occurs due to annular dilatation of an otherwise normal mitral complex.

Mitral Valve Prolapse

As stated above, DMVR refers to a spectrum of conditions that ultimately result in MVP. MVP may be defined as the slipping of one or more mitral leaflets and/or scallops beyond the mitral annulus during systole.

In considering the echocardiographic appearance of MVP, it is important to be aware of a number of factors. Firstly, the orientation of the echocardiographic short axis view of the mitral scallops varies compared to the surgical or pathological short axis view of the scallops. For example, the surgical or pathological inspection of the mitral valve is typically viewed from above; that is, from the LA aspect of the valve. From the 2D parasternal short axis view of the mitral valve, the valve is viewed from below; that is, the valve is viewed from the LV aspect of the valve. As a result, from the 2D short axis view the lateral aspect of the valve appears to the right of the image while the medial aspect appears to the left of the image. Therefore, on 2D echocardiography, the mitral scallops are seen as a mirror image of the surgical or pathological view of the scallops (Fig. 8.38).

Secondly, while there may be prolapse of the whole leaflet, prolapse may be isolated to one or more mitral scallops. Therefore, an awareness of the various mitral scallops with respect to the various echocardiographic views is also required (Fig. 8.39).