The Murmurs of Mitral Regurgitation

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An apical holosystolic murmur with radiation to the axilla and back is the hallmark for the diagnosis of mitral regurgitation.¹,² In these patients, the presence of a third heart sound suggests that the incompetence is significant, and frequently this ventricular filling sound is followed by a short rumbling diastolic murmur owing to increased flow across the mitral valve.³ (Fig 1).

The purpose of this communication is to discuss other murmurs of mitral regurgitation which are less common.

The Early Systolic Murmur

Patients with dominant mitral stenosis and mild or moderate mitral incompetence frequently have early decrescendo systolic murmurs which start with the first sound and terminate in midsystole (Fig 2). In our experience, selective left ventriculography in this type of patient demonstrates mild degree of mitral regurgitation.

We have also noted the early systolic murmur in patients with solitary mitral stenosis (Fig 3). Surawicz et al have reported that these systolic murmurs occur in 48 percent of these patients.⁴

We do not have an appropriate explanation for the systolic murmur appearing in mitral stenosis alone. It has been suggested that in these patients minimal mitral incompetence is present but is not detected by left cine ventriculography.⁴

We conclude that often it is not possible by auscultation only, to differentiate solitary mitral stenosis from dominant mitral stenosis with mild mitral insufficiency.

The Late Systolic Murmur

Most physicians now believe that the late apical systolic murmur sometimes preceded by a “non-ejection click” is indicative of mild or moderate mitral regurgitation.⁵-⁸ (Fig 4,5). This murmur should not be confused with the holosystolic murmur composed of soft vibrations in early systole and a louder murmur at the end of the systole.

Recently, several investigators have established the origin of the click and late systolic murmur to be in the mitral valve and subvalvular structures.⁵-⁸

Barlow et al⁹ believe that a “non-ejection click” denotes uneven distribution of tension in the chordal mechanism and that one or more chordae are

Figure 1. Multichannel phonocardiograms with simultaneous ECG and indirect carotid pulse (CAR) are taken from a patient with severe mitral regurgitation. High (HF), medium (MF) and low frequency (LF) filters are used. The apical systolic murmur (SM) is holosystolic, beginning with the first heart sound (S₁) and ending with the second sound (S₂). A loud third heart sound (S₃) and short diastolic murmur (DM) are noted.

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lengthened or are relatively longer than other fibrosed chordae." In these conditions, sudden tensing of a chorda or the leaflet to which it is attached results in the click.

Similarly, these authors suggest that the presence of late systolic murmurs are due to chordae which are lengthened (anatomically or functionally) or ruptured. In each case, the leaflet to which the affected chorda is attached prolapses abnormally into the left atrium during systole. In most of the cases reported, the posterior leaflet is involved and when the nonejection click is present, it is synchronous with maximal ballooning of the posterior mitral leaflet. This leaflet has chordae of the third order which insert in the central portion of its ventricular surface and provide support to the leaflet. Any process leading to elongation or rupture of these chordae would permit the ballooning of the posterior leaflet as shown experimentally by Stannard et al. Once this process has started, it is probably progressive, producing stretching of the leaflet which becomes voluminous and puts greater strain on the chordae of the first and second order. With progressive stretching of these chordae, mitral regurgitation develops, and it is a self-perpetuating mechanism which may lead to more serious degrees of incompetence. This concept proposed by Barlow et al may explain how several etiologic factors (rheumatic valvulitis, myocardial and papillary muscle dysfunction, trauma leading to rupture of a chorda tendinea, bacterial endocarditis, Marfan's syndrome, idiopathic subaortic stenosis, mitral valve surgery, etc) involving the leaflets, chordae, papillary muscles or their combination could lead to ballooning of the posterior mitral leaflet and mitral regurgitation confined to late systole.

Congenital weakness of the valve could explain, in some patients, the aneurysmal deformity of the posterior leaflet and the prolapse of this leaflet into the left atrium during systole. Patients with the Marfan syndrome and prolapse of the posterior mitral leaflet have been reported.

In our experience, 50 patients with the late systolic murmur had a modest degree of mitral regurgitation (grade 1 or 2 out of 4) with the exception of one patient who had severe mitral incompetence (Fig 5).

Subacute bacterial endocarditis and even sudden death have been reported in patients with the late systolic murmur. These patients should be carefully followed and prophylactic antibiotics administered when indicated.

**Mid-Systolic Murmur**

A mid-systolic murmur, diamond-shaped, has been described by Burch et al and Phillips et al.
in the patient with mechanical dysfunction of the papillary muscles due to myocardial infarction or ischemia. In this condition, the ischemic papillary muscle fails to shorten during ventricular ejection resulting in mitral incompetence. This murmur begins after isovolumetric contraction and is well heard at the apex with radiation to the left axilla and sometimes to the aortic area.\textsuperscript{14}

The crescendo-decrescendo characteristic of this murmur is probably related to the tendency of the valve to increase its incompetence in midsystole reflecting the changes in left ventricular pressure.\textsuperscript{14}

Scarring of a papillary muscle may also be expected to produce a holosystolic murmur if the mitral incompetence begins with the first heart sound (during isovolumetric contraction of the left ventricle). A similar situation would occur if a normally contracting papillary muscle is implanted in an area of ventricular aneurysm.\textsuperscript{13} The murmur of papillary muscle dysfunction may become louder during transient episodes of papillary muscle ischemia.\textsuperscript{14}

Chordal rupture leads to different degrees of mitral incompetence; the apical murmur is usually

\textbf{Figure 4.} The late systolic murmur (SM) begins in midsystole and ends with the second heart sound ($S_2$). A non-ejection click (C) in midsystole introduces this murmur. Mild mitral regurgitation (grade 1) was demonstrated by cine ventriculography.

\textbf{Figure 5.} The late systolic murmur (SM) follows a non-ejection click (C) and ends with the second heart sound ($S_2$). This was the only example of our series of 50 patients with late systolic murmurs who had severe mitral regurgitation (grade 3) demonstrated by left ventricular cineangiography.

\textbf{Figure 6.} Phonocardiograms taken from a patient with congestive failure due to cardiomyopathy. Mitral regurgitation (grade 2) was demonstrated by cineangiography. Systolic murmurs are absent. Note the pulsus alternans in the carotid recording and the alternating intensity of the third heart sound ($S_3$).
holosystolic. This murmur frequently radiates to the basal areas of the precordium and even to the neck vessels, where it assumes a crescendo-decrescendo configuration and is sometimes confused with the murmur of aortic stenosis. The propagation of the murmur to the base of the heart should suggest rupture of a chorda attached to the posterior mitral leaflet. If the ruptured chorda belongs to the anterior mitral leaflet, the radiation of the murmur is generally toward the back.

The transmission of the apical murmur to the base of the heart and neck vessels is due to the regurgitant jet directed to the interatrial septum and adjacent root of the aorta. In the cases with ruptured chordae of the anterior mitral leaflet, the jet is directed toward the posterior wall of the left atrium adjacent to the spine. This favors propagation of the murmur to the cervical and lumbar spine and even to the vertex of the head.

A thrill palpated over the middle thoracic spine and slightly to the left of it has been described in patients with ruptured chordae tendineae of the anterior mitral leaflet.

**Silent Mitral Regurgitation**

Silent mitral incompetence has rarely been reported in rheumatic heart disease. When present, it is usually associated with mitral stenosis.

In the last year, we have studied three patients with congestive cardiomyopathy and no systolic murmur. Two of these patients had ventricular gallops (Fig. 6) and the third patient had a third heart sound followed by a short and loud rumbling murmur at the apex (Fig. 7). The first two patients had moderate mitral regurgitation (grade 2 out of 4), and the third had only mild mitral incompetence. These three patients demonstrated severe congestive failure which may be responsible for attenuation of the murmur. Although intracardiac phonocardiograms were not obtained, it is possible that the systolic murmur was present in the left atrium and absent on the surface of the chest. The flow murmur in the third patient is even more difficult to explain in view of the modest degree of mitral regurgitation and the absence of a systolic murmur.

**Summary**

Not all murmurs of mitral regurgitation are holosystolic. Occasionally, the patient with mitral reflux will demonstrate an early systolic murmur, a late systolic murmur or a mid-systolic murmur. Rarely, a systolic murmur is absent. We have also described a patient with silent mitral regurgitation and a short diastolic rumbling murmur.

**REFERENCES**


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