Presystolic Murmur of Mitral Stenosis Revisited*

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Mitral stenosis usually gives rise to a diastolic murmur that is accentuated in the presystolic period, that is, as atrial contraction augments ventricular inflow before the mitral first sound. Numerous investigators (1-9), however, have observed a short sound transient that precedes this portion of the first heart sound even in the presence of atrial fibrillation, thus eliminating the possibility that it could be caused by atrial contraction. Although beginning simultaneously with the onset of left ventricular contraction, this sound component has been labeled "presystolic" by several authors, a term that we consider misleading. We believe the term "early systolic" or "preisovolumic," to be more appropriate.

The origin of these vibrations has been the subject of considerable debate. Three explanations have been offered:
1) Tricuspid closure might provide their origin, for it has long been known that, in mitral stenosis, mitral closure might be delayed sufficiently to follow tricuspid closure (1).
2) Transmirtal blood flow is accelerated transiently as the closing mitral leaflets narrow this orifice before their complete closure in early systole (2-6). The resulting increase in anterograde velocity would cause intensification of the diastolic mitral rumble. This theory has gained considerable support and, if correct, would suggest that, even in atrial fibrillation, the mitral diastolic murmur manifests "presystolic" accentuation. 3) The early systolic vibrations represent a sound transient that results from initial contraction of the ventricle which, in turn, acts to abruptly decelerate the flow of blood into this chamber (7-9). This explanation suggests that the nature of this sound is similar to that of a third heart sound, emanating from the myocardium as active forward flow into this chamber is checked abruptly by the stiffening ventricular walls.

Components of the first heart sound: the early systolic vibrations. Normally, the first heart sound consists of a prolonged mixture of vibrations that is usually dominated by two large, audible components that occur in the middle portion—the sounds of mitral valve closure followed by tricuspid valve closure. One or two small vibrations usually precede the mitral component and synchronize with the onset of ventricular contraction, coinciding with the initial systolic upstroke of the apexcardiogram. This early component is believed to be "muscular" in origin, resulting from myocardial contraction. In previous studies from our laboratory (6,7), we observed that one could identify this initial component in the presence of atrial fibrillation, even when mitral stenosis was lacking. Enhancement of this component was noted in short cardiac cycles, and third heart sounds of preceding cycles appeared to be augmented when the two events coincided. We postulated at that time that these early systolic vibrations in mitral stenosis were in no way unique to this condition and were related to abrupt deceleration of transmirtal blood flow as the ventricles stiffen during initial contraction. We also believed it conceptually unacceptable to postulate an increase in anterograde mitral flow velocity in the face of a rapidly decreasing pressure gradient, because the mitral leaflets close primarily because of this rapidly decreasing gradient in harmony with the blood surrounding them, not in opposition to it. Nevertheless, these vibrations often appear to be of longer duration and more intense in mitral stenosis in comparison with other conditions, and this finding also required an explanation.

Thus, we theorized that this component seemed more intense and longer in mitral stenosis for two reasons: 1) Intensity of this sound is dependent on the presence and velocity of anterograde mitral flow at the time of ventricular contraction. Because forward flow into the left ventricle at a high velocity persists for a relatively long time during diastole in mitral stenosis, there would be a longer opportunity for an abrupt deceleration of this anterograde flow even relatively late in diastole. 2) Because the interval from the onset of left ventricular contraction to mitral valve closure is prolonged in mitral stenosis, any early systolic sounds might be allowed to seem longer and more intense when not obscured by an early mitral first heart sound.

Although lacking direct proof at the time of earlier studies, one could rather confidently exclude a tricuspid origin for the early systolic sound because of its synchronization with the onset of left ventricular contraction. Tricuspid valve closure occurs much later than this. Moreover, Armstrong et al. (9) pointed out that this component is still present even after combined mitral and tricuspid valve replacement.

The study of Hada et al. (10). More definitive proof that this sound is associated with neither tricuspid valve closure nor increased velocity of anterograde mitral flow is

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supplied by Hada et al. (10) in this issue of JACC. Direct interrogation of the mitral orifice with Doppler ultrasound disclosed only abrupt deceleration of flow during this early systolic sound component. Of great interest is the apparent relation between the rate of this deceleration and the intensity of the early systolic vibrations; that is, with short cardiac cycles, mitral flow had a more rapid deceleration coinciding with more intense vibrations. Hada et al. further point out that the presystolic accentuation of the mitral murmur, which synchronizes with atrial systole but precedes the onset of ventricular contraction, does indeed represent an increase of mitral flow velocity. Thus, the time-honored concept of presystolic accentuation, or the so-called atriosystolic murmur, associated with mitral stenosis and regular sinus rhythm remains intact. This should supply some solace to those who have written texts on physical diagnosis or to those who accept new concepts begrudgingly!

Although Hada and his coworkers did provide strong evidence to eliminate certain postulated origins for the early systolic sound in mitral stenosis, two questions remain unresolved. Does this sound truly emanate from the ventricular myocardium as we have postulated, or are there other responsible factors? Is its origin similar to that of the early "muscular" component of the normal first heart sound? If the answer to both of these questions is affirmative, it will prove to be one of those rare occasions in medical science when all of one's original hypotheses eventually prove to be correct!

References