Presystolic Murmur in Atrial Fibrillation
Fact or Fiction?

FOR MANY YEARS, conventional wisdom has taught that in mitral stenosis, the advent of atrial fibrillation results in the disappearance of the presystolic accentuation of the diastolic rumble. What could be simpler? Without effective atrial systole, one loses the impetus for flow across the stenotic mitral valve immediately prior to ventricular systole; hence, the murmur accentuation is lost. In defiance of these tenets, several workers\(^1,4\) recently have purported to demonstrate a presystolic accentuation of the mitral murmur even in atrial fibrillation. This accentuation has been attributed to increased velocity of forward blood flow as the mitral orifice narrows immediately prior to total valve closure in early ventricular systole. Murmur augmentation is said to be most apparent in beats following short cycles, when the mitral pressure gradient is higher at the onset of ventricular systole. In the presence of sinus rhythm, it is generally conceded that atrial systole may produce an augmentation of the diastolic rumble, but this occurs earlier than the above mentioned augmentation, and it may precede clearly the onset of left ventricular contraction. Because of several facts we have serious reservations about the new concepts explaining the presystolic accentuation in atrial fibrillation.

The so-called presystolic accentuation appears only after the ventricle has begun to contract; therefore, whatever its nature, this sound is not truly presystolic. We believe the record should be clarified and the sound renamed early systolic or pre-isovolumic sound or accentuation. If clinicians desire to continue using the imprecise term presystolic (referring to its location before the first heart sound), they should do so only with the full understanding of these points.

In atrial fibrillation and mitral stenosis, discrete sound vibrations have long been identified during initial ventricular systole before mitral closure. Ongley et al.\(^5\) observed such a sound and, believing it to be a discrete transient, attributed it to "falling of the first sound earlier than usual in diastole because of the rapid heart rate." Hultgren and Leo,\(^6\) in studying cases with mitral stenosis and atrial fibrillation, suggested that tricuspid closure often accounted for vibrations which preceded mitral closure. They were often discrete and clearly resembled a sound transient rather than a murmur. Moreover, augmentation of this sound was observed after short cycles, a finding which appeared to lend support to a tricuspid origin. A tricuspid origin for this sound, although seemingly plausible, appears untenable for the following reasons: 1) similar sounds precede mitral closure in the absence of mitral stenosis,\(^7,8\) even though tricuspid closure would be expected to follow that of the mitral. 2) These initial vibrations coincide with the very onset of ventricular contraction,\(^9\) as indicated by the apexcardiogram, a point which occurs too early for tricuspid closure. 3) Recent echocardiographic study\(^10\) indicates that the tricuspid valve completes its closure an average of 90 msec after the onset of the QRS complex in normals, similarly ranging from 70 to 110 msec in patients with mitral stenosis. The transient described by Hultgren and Leo followed the QRS complex by only 50-70 msec, too early for tricuspid closure. When one scrutinizes the data of workers such as Criley et al.\(^1,2\) even the so-called presystolic murmur accentuations often resemble strikingly these previously observed discrete sounds. Thus, we believe it likely that all such vibrations represent the same phenomenon.

As alluded to above, we have recently observed that early systolic sounds are usually seen in atrial fibrillation without mitral stenosis.\(^11\) They may resemble crescendo murmurs and are strikingly accentuated in beats terminating short cycles. Thus, to have such a presystolic murmur, one need not postulate either mitral stenosis or a significant transvalvar mitral pressure gradient. One obtains a hint of the mechanism of these vibrations by observing that a third heart sound may be accentuated strikingly when followed by an early beat, an event which places it into the period during initial left ventricular contraction of this subsequent beat. Similarly, the initial low-frequency component of the first heart sound is accentuated when this beat follows the preceding one by a short interval.

It is highly improbable that the velocity of blood across a valve orifice actually increases in the face of a rapidly falling pressure gradient. One must bear in mind that the mitral leaflets themselves probably are closing primarily because of this rapidly falling gradient — they do so in conjunction with the blood about them — not in opposition to it. Moreover,
direct studies of mitral flow velocity in normals and in those with mitral stenosis do not show increases of blood velocity during this period, only smooth deceleration as one would expect. If a rapidly falling pressure gradient across a stenotic valve orifice were capable of increasing momentarily flow velocity as the orifice narrows, one might also expect the systolic ejection murmur of aortic or pulmonic stenosis to accentuate immediately prior to the closure of the respective semilunar valve. Thus, we find it difficult conceptually to accept the idea that mitral flow velocity can increase during early ventricular systole.

One additional observation has some importance in searching for a unifying theory to explain early systolic sound vibrations: Most workers have noted that in mitral stenosis, diastolic mitral murmur vibrations are present during periods when the mitral leaflets are either held in one position or are moving in the direction of the incoming blood. Little or no sound is produced during rapid anterior mitral movements, i.e., early in diastole prior to the opening snap. The same is true to a lesser extent late in diastole as the leaflets display limited forward motion with initial atrial contraction. Since the mitral apparatus is moving forward with the atrioventricular blood during these periods, blood traversing the mitral orifice would be minimized and less turbulence expected. This would be analogous to the situation of the aortic ejection click, wherein the aortic valve moves forward in early systole until it is fully open. As this end point is reached, an ejection click is produced and only then does blood begin to traverse the stenotic orifice to produce the initial portion of the systolic ejection murmur.

Even a brief anterior movement of the mitral leaflets would explain why there is an apparent inordinate delay in the onset of the so-called atriosystolic murmur in comparison to other phenomena such as the fourth heart sound. While this observation is not directly pertinent to the situation in atrial fibrillation, it does explain why the late appearing presystolic murmur in normal sinus rhythm may lead one into erroneously believing that all presystolic accentuations are related to early ventricular systole rather than atrial contraction. This idea can be easily refuted by the common observation that the murmur can be clearly demonstrated to precede the QRS complex of the electrocardiogram, especially in varying degrees of heart block.

Finally, the concept that sound can be produced by sudden muscular contraction is an old one. Early experiments by Wiggers demonstrated that small, isolated strips of ventricular muscle from the hearts of cats, when made to contract, produced a sound. He regarded this sound as the result of bringing the muscle fibers to a state of sudden tautness. Most modern authors accept a muscular origin of the initial vibrations of the normal first heart sound.

We can best summarize by propounding a general thesis which will bring into harmony all the various observations concerning the early systolic sound in atrial fibrillation. This sound precedes mitral and tricuspid closure in both mitral stenosis and other states, occurring at the very onset of ventricular contraction. The so-called presystolic accentuation of the diastolic murmur appears most likely to represent a sound transient which is actually early systolic (presimultaneous) and arises from a mechanism apart from that of the diastolic mitral rumble. This sound transient is neither peculiar to nor indicative of mitral stenosis. It most likely results from initial contraction of the ventricle which acts to decelerate abruptly the flow of blood into this chamber. The nature of this sound is probably akin to a third heart sound, i.e., it emanates from the ventricular walls. To produce or enhance this sound one must have two simultaneous events: 1) active flow of blood into the ventricle, as seen in early diastole, and 2) an abrupt change in compliance of the left ventricular walls (brought about by early ventricular contraction) acting to decelerate this blood. Apparently, sound production does not require a great rate of flow; hence, a significant sound can occur even in severe grades of mitral stenosis. On the other hand, mitral stenosis may prolong the potential for this sound’s accentuation, inasmuch as it fosters the continuation of active mitral flow into the left ventricle for longer periods of time, a fact which probably enables prominent early systolic vibrations to become manifest even after fairly long cycles. Moreover, since the interval from the onset of left ventricular contraction to mitral closure is prolonged in mitral stenosis, any such early systolic sounds might be more readily prolonged, in contrast to the situation in normal individuals, in whom this interval is too short to allow for production of sustained vibrations.

Thus, before the medical world accepts the concept that there is truly presystolic accentuation of the diastolic rumble in mitral stenosis despite the presence of atrial fibrillation, we urge careful consideration of the above comments.

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References

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