Presystolic Murmur in Atrial Fibrillation

To the Editor:

Doctors Tavel and Bonner in their recent editorial1 have advanced a detailed and compelling refutation of the concept that a presystolic murmur can occur in mitral stenosis with atrial fibrillation. We welcome their critical comments, and appreciate the opportunity to utilize some newer data to substantiate the validity of the theory. Space will not permit a point-by-point rebuttal, so we will confine our response to three salient features: 1) there is a crescendo murmur, 2) the murmur occurs during mitral inflow (ventricular filling), and before the onset of isovolumetric systole, and thus can be considered presystolic, and 3) mitral valve flow velocity has been demonstrated to increase during valve closure in mitral stenosis.

Figure 1 demonstrates a simultaneous electrocardiogram, apex phonocardiogram, mitral valve echogram, and left atrial and ventricular pressures in a patient with mitral stenosis and atrial fibrillation. There is a crescendo phase of the diastolic murmur seen in the first two full cycles which is absent on the third cycle. We submit that this is indeed a murmur in that it is occurring during presumably turbulent blood flow, and that it is not a transient (or sound) in that it prolongs the murmur by more than 0.08 seconds. If the murmurs in the first two cycles are compared with those seen in the third cycle, they can be seen to be longer, louder, and to possess a crescendo phase leading to the first heart sound. The difference in murmur intensity and duration can be related to the presence of a persistent diastolic gradient at the time of initiation of valve closure (dashed line) as defined simultaneously on the echocardiogram by the abrupt posterior excusion of the anterior leaflet (AML). If this sonic event is not a murmur, it is at least a phenomenon indistinguishable from a murmur.

Admittedly, the limits of the phases of the cardiac cycle (systole and diastole) are imprecise and subject to change with varying hemodynamic states, so that no one definition of the limits could possibly apply to all of the various pathological conditions. For example, in severe mitral regurgitation there is often a regurgitant flow and murmur caused by left ventricular contraction which persists after aortic valve closure (technically diastole), and yet we accept the phenomenon as a systolic event. By the same token, in mitral stenosis blood is driven across the mitral valve after the onset of left ventricular contraction because of the presence of a left atrial/left ventricular pressure gradient. We submit that it is not inappropriate to term this forward flow and the sonic events resulting from it "pre-systolic." The term "presystolic" has been in wide usage since the 19th century to describe the characteristic prefirst heart sound murmur of mitral stenosis, despite the fact that investigators for more than a century have recognized that the murmur is, at least in part, technically systolic.2

Doctors Tavel and Bonner state that it is "highly improbable that the velocity of blood across a valve orifice actually increases in the face of a rapidly falling pressure gradient," and cite the work of Kalmanson3 which they interpret to "not show increases in blood velocity during this period, only smooth deceleration as one would expect." Although Kalmanson's3 illustrations do not provide the simultaneous pressure and flow data necessary for the interpretation of Doctors Tavel and Bonner, they in fact demonstrate steady increase in velocity proceeding through the QRS complex, reaching a peak value at 0.08 to 0.10 seconds after the Q, in their figures 2, 3, 4, 6, 11, 12, and 14 illustrating patients with significant mitral stenosis. In addition, the above referenced figures that have adequate simultaneous phonocardiograms demonstrate a peak mitral flow velocity to halt abruptly at the inscription of S1 on the phonocardiogram. An explanation of this "highly improbable" increase in velocity at the time of mitral valve closure requires an appreciation of the momentum imparted by the atrioventricular pressure gradient to the column of blood traversing the mitral orifice. Kalmanson's data support the fact that a transient

**Figure 1.** Recording from a patient with mitral stenosis and atrial fibrillation.
acceleration occurs at the time of valve closure, apparently as a result of the narrowing of the orifice in the face of this forward flow. In contrast, aortic valve closure occurs after cessation of forward flow in aortic stenosis, and hence no acceleration and no resulting augmentation of the murmur would be expected.

Although the editorial raised a number of other points, with which we disagree, we do not believe them to be germane to the challenge stated in the title: “Presystolic Murmur in Atrial Fibrillation — Fact or Fiction?” We leave it to your readers to answer the question.

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References
3. Omerod EL: Observations of a direct mitral or tricuspid murmur. Med Times 42: 276, 1864

To the Editor:

Perhaps some of the problems relate to terminology, but we consider that there are several points in the editorial of Drs. Tavel and Bonner which warrant comment.

Presystolic accentuation of the murmur of mitral stenosis in the presence of atrial fibrillation was independently recognized by Criley and co-workers, and ourselves. The data in support of this concept, the explanation of such accentuation and the use of the term “presystolic” have been challenged by Drs. Tavel and Bonner.

Since the onset of mechanical systole is identified in the clinical situation by the first heart sound, it is appropriate that accentuation of the murmur, which immediately precedes this sound, should be regarded as “presystolic” although the vibrations have been shown to occur during the pre-isovolumetric phase of left ventricular contraction. In patients with mitral stenosis in sinus rhythm and a relatively short P-R interval, the “presystolic” accentuation does start prior to the onset of ventricular contraction but an increasing intensity of the murmur continues throughout the pre-isovolumetric phase. In patients with mitral stenosis and a relatively long P-R interval, the left atrial “a” wave occurs earlier in diastole with a resultant increase in the left atrial/left ventricular pressure difference at that time. This pressure difference then decreases prior to the onset of left ventricular contraction. For these reasons the atrial systolic murmur may have a crescendo-decrescendo configuration. However, with the onset of left ventricular contraction, the ventricular pressure rises, and as the leaflets move toward closure, the murmur accentuates again. Thus, even with sinus rhythm, this latter part of the “presystolic” murmur is not truly presystolic in that it occurs during the pre-isovolumetric phase of left ventricular systole but because the clinical recognition of systole is determined by the first heart sound this nomenclature must surely be retained.

A principal statement made by Drs. Tavel and Bonner that “It is highly improbable that the velocity of blood across a valve orifice actually increases in the face of a rapidly falling pressure gradient” is, in our view, supported by tenuous argument. Although the work of Kalmanzon and collaborators is quoted in support of the statement, perusal of their illustrations (figs. 2, 3, 6, and 12) does reveal that, in fact, there is an increase in blood velocity ending with the first sound on simultaneously recorded phonocardiograms. We maintain that in mitral stenosis while there is a left atrial/left ventricular pressure difference during diastole blood must be flowing from the left atrium into the ventricle. As the pressure difference decreases during the pre-isovolumetric phase of ventricular systole, the mitral leaflets are forced toward their closed position, which occurs at, or very shortly after the point in time at which the rising left ventricular pressure crosses that of the left atrium. The left atrial pressure is invariably falling until the onset of the pre-isovolumetric phase, when it may be observed to rise again. We consider that this rise in left atrial pressure is explained by the upward movement of the leaflets toward the closed position which must decrease the volume of the high-pressure left atrial chamber. Throughout the pre-isovolumetric period the mitral orifice is progressively narrowing and the situation is, we believe, analogous to closing the front door of a house in the face of a strong wind. As the door is forced toward closure, the volume of air entering the house decreases whereas the velocity of air flow increases, as does the intensity of sound vibrations of the wind passing through the narrowing aperture.

The matter is not entirely an academic problem. “Presystolic” accentuation is readily apparent in mitral stenosis with pliable leaflets. When the leaflets are thickened and rigid, the orifice size cannot change as much and consequently there is less accentuation of the murmur during pre-isovolumetric ventricular systole irrespective of whether sinus rhythm or atrial fibrillation is present.

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References

False Positives on Diagnostic Tests for CHD

To the Editor:

I read with interest the article by Eriksen et al. (Circulation 54: 371, 1976). In the concluding remarks of the authors, it states, “It also establishes that a considerable portion of suggestive cases are false positive cases.” Several additions to the protocol in this very interesting article may have decreased the number of false positive cases: 1) the performance of standing and hyperventilation ECGs, as these two methods are known to produce ST-segment depressions in patients with normal coronary arteries; 2) left ventriculography specifically looking for idiopathic hypertrophic subaortic stenosis and prolapse of the mitral valve, either the anterior or posterior leaflets or both; 3) echocardiography; 4) a detailed history of medications which can produce false positives, particularly diuretics and tranquilizers which are available to the general population through friends and neighbors, although not specifically prescribed to these patients by their private physicians.
Presystolic murmur in atrial fibrillation.
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