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

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/ventricular pressure difference at that time. This pressure difference then decreases prior to the onset of ventricular contraction. For these reasons the atrial systolic murmur may have a crescendo-decrescendo configuration. However, with the closure 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 Kalmanon 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 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 Eriksson 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 sub-aortic 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.
False positives on diagnostic tests for CHD.
J P Liss

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