labelling all these patients as having heart disease with all the consequences attendant thereto?

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The authors reply:

To the Editor:

Dr. Greenwald raises an important question and one to which we would like to respond. Our study was initially prompted by the observation of pansystolic posterior bowing of the mitral valve echogram in the presence of marked mitral prolapse on cineangiogram in two consecutive patients during a one week period. We then catalogued the abnormalities described in our report by both retrospective and prospective analysis of the echograms of all patients with mitral prolapse documented by catheterization in our laboratory. The validity of these observations has been substantiated by other investigators.1 Subsequently, we, like many others, have observed an increasing number of echograms which manifest this pansystolic hammer-like posterior motion and collapse in patients both with and without other clinical evidence of mitral prolapse syndromes. Thus, it is clear that a need exists for further clarification of the exact significance of this echographic phenomenon.

It is important to emphasize from the start that the ultrasound findings which we reported typically occurred in combination. Thus pansystolic bowing reached its nadir in midstole, and characteristically there occurred an increased diastolic excursion of the anterior mitral leaflet (usually abutting the septum), increased motion of the left atrial posterior wall, and multiple systolic echoes. The absence of more than one of these ancillary findings should therefore cast suspicion on the diagnosis of mitral prolapse syndrome. Analyzing echograms in this fashion, we have only infrequently encountered patients without other clinical evidence of mitral prolapse.

Echocardiography would appear to offer certain advantages over cineangiography in the evaluation of mitral valve motion. Nevertheless, left ventricular angiography remains the gold standard for the evaluation of mitral prolapse and it is likely that the recently described echographic findings will be found to be “falsely positive” in relation to this standard in some patients, and we have documented this finding in one patient. However, rigid adherence to the requirement for mid-systolic buckling has yielded falsely negative results in the past. It thus appears that, as with so many tests in medicine, the various echocardiographic manifestations of mitral prolapse will have a sensitivity and specificity. Although the exact sensitivity and specificity remain to be established, and will undoubtedly vary for each echographic manifestation, ultrasound-angiographic correlations thus far available to us have demonstrated all the echographic findings described to be highly specific.

The natural history of the mitral prolapse syndromes remains uncertain. Nevertheless, the relatively benign course thus far exhibited by most patients with mitral prolapse diagnosed in recent years by cineangiogram or echocardiogram, coupled with the similarly innocuous effects of “old healed pericarditis,” manifested by a mid-systolic click and late systolic murmur diagnosed in earlier years, lends support to an optimistic prognosis for this entity. Since these patients exhibited the most overt manifestations of the syndrome, it would be expected that the patients with more subtle signs, such as isolated doming of the mitral leaflets on echogram, would demonstrate an even more benign course. However recent studies in our laboratory have revealed serious cardiac arrhythmias to be present in a surprisingly high percentage of mitral prolapse patients when they were subjected to ambulatory Holter monitoring.2 Thus, further work utilizing more sophisticated methods of cardiovascular evaluation may provide an answer to the practical clinical significance of mitral prolapse for any individual patient.

As a result of the above uncertainties, an approach to “silent” mitral prolapse has evolved in our laboratory. All patients with echographic abnormalities suggestive of prolapse undergo exercise stress testing and Holter monitoring. If the echogram is the solitary abnormality and it is not of the mid-systolic buckling variety, the patients are advised that they have an atypical cardiac finding of uncertain significance and no medications are prescribed. Any of the described echographic abnormalities when accompanied by systolic clicks, murmurs consistent with mitral regurgitation, or frequent arrhythmias, or mid-systolic mitral movement on echogram even when unaccompanied by these findings is sufficient to make the diagnosis of mitral prolapse. Such patients are then treated with prophylactic antibiotics at the time of minor surgery and antiarrhythmic agents as required.

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References

Passive Elasticity of the Human Left Ventricle

To the Editor:

The authors are to be commended on their recent article1 relating to the assessment of passive elastic stiffness of the left ventricle in the clinical setting. Not only have they presented a simplified method for calculating stiffness constants, but they also confirm the fact that these stiffness constants relate to the muscle per se. Furthermore, if their pressure-volume data (obtained from fluid-filled catheters and single-plane cine) are shown to be reliable, the global values obtained for the K constants in patients with coronary artery disease may indeed be sufficient to quantitate the degree of segmental disease. In our own studies,2,3 we were unable to obtain such wide disparities in the values of the stiffness constants and this may be due to the employment of simplified P-V relations. On the other hand, our recent study4 indicated that stiffness constants were independent of the geometry of the left ventricle or stress formulae employed, lending support to the notion that these stiffness constants reflect the elastic properties of the ventricular wall material.

Other comments pertinent to their article are:
1. It has been demonstrated in papillary muscle studies by
"Silent" Mitral Prolapse: The authors reply:  
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Circulation. 1974;50:1285  
doi: 10.1161/01.CIR.50.6.1285  
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231  
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:  
http://circ.ahajournals.org/content/50/6/1285.1.citation

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