pressures to catheter position is only applicable for a catheter-tipped transducer.

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Systolic Click Syndrome

To the Editor:

I have read with interest the article by Liedtke et al. (Circulation 47: 27, 1973) who have demonstrated a reduction in the extent of shortening of the proximal ventricular segment and the mitral valve ring, with an increase in the diameter of the ring in some of their patients. It is probable, however, that this angiographic appearance may have been due to the superimposition of the "doughnut-like" prolapsed leaflets4 over the mitral ring and the proximal ventricular segment. In our experience, the right anterior oblique view is the projection of choice for demonstration of mitral valve prolapse;1 the lateral view used by the authors may have made it difficult for them to demonstrate the prolapse and may have resulted in the superimposition which I postulate.

Assuming that a rigidity of the proximal segment of the left ventricle was proved, this should not be construed as pointing to a primary myocardial etiology of this syndrome. Indeed, similar rigidity of the postero-basal segment of the left ventricle due to secondary fibrosis has been found in rheumatic mitral stenosis, a primary valvular disorder.5 The angiographic picture described by Liedtke et al. may be due to the fibrotic friction lesions of the left ventricular endocardium associated with "hooding of the mitral valve";3 adhesions of the chordae to the endocardium as well as collagenous prolongation into the subjacent myocardium have also been described. Friction by the elongated chordae, which are abnormally close to the endocardium, was considered as the mechanism of these lesions.

In summary, the data presented by the authors, even if confirmed, do not necessarily indicate primary myocardial involvement in this syndrome which is probably secondary to myxomatous degeneration of the mitral leaflets and their chordae.4

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References

The authors reply:

To the Editor:

Dr. Jeresaty's view that the altered left ventricular angiographic silhouette during systole in patients with the systolic click syndrome is the result of prolapse of the posterior mitral leaflet is one that has been shared by a number of previous investigators. Identification of this abnormality in our study as reduced contraction of the left ventricular inflow tract was based, first, on frame by frame drawings of the silhouette which demonstrated clearly that contraction (a characteristic of myocardium and not valve leaflets) actually occurred, but was reduced in extent in this segment. Second, the portion of the wall which exhibited reduced systolic contraction usually extended up to the head of the inferior papillary muscle, well beyond the mitral valve ring. As the angiograms published in our report illustrate, only minimal foreshortening of the ventricular chamber is seen in the lateral projection, so that superimposition of prolapsing leaflets on the inflow region of the chamber, creating the false impression of noncontraction of this region, as suggested by Dr. Jeresaty, does not appear to be a likely alternative to our interpretation. Indeed, it is our impression that the right anterior oblique and lateral projections are quite similar, and in fact these views have yielded virtually identical findings in patients with the systolic click syndrome in our laboratory. That relatively minor degrees of leaflet prolapse, if any, must have been present in our patients is suggested further by the infrequent angiographic demonstration of mitral incompetence. Finally, the silhouette abnormality during contraction in our patients was shown to be identical to that observed in several patients with occlusive coronary artery disease and proven diaphragmatic myocardial infarction, in whom no leaflet prolapse could be demonstrated.

Our hypothesis that a primary myocardial abnormality may be an important component of the systolic click syndrome was based on the observation that this ventricular wall motion abnormality could be demonstrated consistently in patients with minimal or no mitral valve incompetence or evidence of leaflet prolapse. The frequency of electrocardiographic abnormalities (usually T wave abnormalities of diaphragmatic origin) which are often out of proportion to minimal evidences of mitral valve dysfunction, as well as postexercise ventricular arrhythmias and the high incidence of sudden, presumably arrhythmia-related death in the familial disorder, also support the presence and even predominance of a primary myocardial abnormality in this syndrome. Documentation of the presence and nature of the myocardial abnormality, of course, must await more detailed pathologic information than is presently available.

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