Mitral Valvular Prolapse and Systolic Click-Murmur Syndrome

To the Editor:

In the December 1976 issue of *Chest*, Nakhjavan et al. reported that mitral valvular prolapse occurred in 20 of 36 patients with atypical angina and normal coronary arteries. I think that the reason for the high incidence of mitral valvular prolapse in this series of patients lies in the fact that Nakhjavan et al. did not deal with the systolic click-murmur syndrome per se. Mitral valvular prolapse was demonstrated only by angiograms in 16 cases, while only in four patients was a nonejection click noted. Thus, the mitral valvular prolapse reported by Nakhjavan et al. was mostly silent. In my opinion, silent prolapse of the mitral valve has to be differentiated from the systolic click-murmur syndrome and should not be included in the so-called mitral valvular prolapse syndrome. We do not know yet the real incidence of silent prolapse of the mitral valve in the normal population, as the condition can only be diagnosed by angiograms. Even echocardiographic studies seem to be unsensitive in revealing silent prolapse of the mitral valve.

In their discussion, Nakhjavan et al. put forward a hypothesis of the myocardial origin of mitral valvular prolapse. Our echocardiographic experience yields some evidence substantiating their hypothesis. In our echocardiographic laboratory, while routinely evaluating unselected cardiac patients, we found nine cases of the systolic click-murmur syndrome with prolapsing mitral leaflets associated with nonobstructive asymmetric septal hypertrophy (Fig 1). All patients but one were male patients, and all had normal coronary arteriograms. The total prevalence of asymmetric septal hypertrophy was 65 patients, and mitral valvular prolapse occurred in 156 patients (out of about 4,000).

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REFERENCES


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Whether or not silent prolapse of the mitral valve is the same entity as systolic click-murmur syndrome is conjectural, especially since even in the clinically manifest syndrome, the physical findings of a systolic click and murmur may not be present constantly. As mentioned in our article (*Chest* 70:706-710, 1976), because of a lack of pathologic specimens in these patients, a “definite” conclusion cannot be reached in regard to the pathogenesis of this syndrome. We are encouraged by Lengyel’s echocardiographic experience supporting our hypothesis of a myocardial origin for mitral valvular prolapse.

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