patients and in no instance did we observe the tumor to remain in the left atrium throughout the cardiac cycle. Interestingly, one patient with documented prolapse of the atrial myxoma demonstrated the so-called Type II left atrial hemodynamics (fig. 1) with a slow Y descent on the pulmonary wedge pulse. There was also a notch on the left ventricular pressure pulse indicating that the tumor moved from the ventricle to the atrium during systole. An obvious misinterpretation of the echocardiogram is evident in Dr. Sung's case 4 (his fig. 6) which clearly shows prolapse of the tumor into the mitral "funnel" when the anterior mitral leaflet is visualized. It is likely that the wavy mass of echoes beneath the mitral valve would have been more clearly identified if the transducer had been angled less superiority. The mechanism for the production of echoes beneath the mitral valve during diastole is prolapse of the tumor across that valve.1,2

I submit that there are probably no distinct hemodynamic features to validly separate prolapsing and nonprolapsing atrial myxomas. An alternative explanation for the slow Y descent on the pulmonary wedge pulse seen in figure 1 and in the so-called Type II atrial hemodynamics would be early obstruction of the mitral orifice by the prolapsing tumor.

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References

The authors reply:

To the Editor:

With regard to Dr. Potts' statement that there was no instance of nonprolapsing left atrial myxoma in his series, we feel that one should not statistically draw a conclusion from such a small number of patients studied. It should be realized that the mobility of the tumor may vary from time to time in the same patient. This phenomenon is known to produce positional variations in auscultatory features, blood pressure, and symptomatology found in patients with left atrial myxoma.1 In case 5 reported in our paper,4 the levophase of pulmonary angiograms clearly demonstrated that the tumor remained within the left atrium during both systole and diastole (fig. 4). In fact, we made an observation that the tumor obstructed the left upper lobe pulmonary vein in left lateral decubitus in the same patient, as reflected by changes in pulmonary artery wedge (PAW) pressure curves.

The echocardiogram presented in figure 6 (case 4) of our paper4 was correctly interpreted. Admittedly, by moving the transducer along the major left ventricular axis from the left ventricle (LV) to the level of the aorta, only two mitral complexes were present with the first one being ample demonstration. As was clearly illustrated, the tumor descended to about the level of the atroventricular canal, but did not enter the LV free cavity. Certainly, these tumor echoes diminished and were absent as the transducer was directed more inferiorly. When the left atrium was visualized by directing the transducer superiority these tumor echoes were present within the left atrium throughout the cardiac cycle. These findings are in contrast to the echocardiographic features of prolapsing left atrial myxoma described in the literature (Dr. Potts' references 2 and 3).

The pulse pressure tracing of a patient with angiographically documented prolapsing type left atrial myxoma presented by Dr. Potts (fig. 1) is most interesting. Dr. Potts is calling attention to the fact that his tracing resembles that of the middle panel in our figure 2 (case 4). However, the Y descent of the PAW pressure curve appears to be steeper, and, as has been pointed out by Dr. Potts, there is an upstroke slurring (not a notch) on the corresponding LV pressure curve. Dr. Potts' proposed mechanism for these features to occur seems acceptable. Alternatively, as compared to the usual type of prolapsing tumor, this may be due to a lesser extent of tumor prolapsing into the LV cavity. We believe that simultaneous recordings of echocardiograms and pulse pressure curves would be helpful in substantiating our hypothesis.

Finally, we wish to emphasize, as we did in our paper,4 that echocardiography has an important role in clinical assessment of patients with mitral valve disease prior to cardiac catheterization, and that, regardless of the pressure curve contours, transesophageal puncture should be avoided, instead using pulmonary angiography to visualize the left atrium, in patients clinically suspected of having left atrial myxoma.

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References

E-F Slope without "a" Wave

To the Editor:

D'Cruz et al. (Circulation 52: 460, 1975) report interesting data on echocardiographic changes with cardiac tamponade. I think, however, that the authors are not in a position to comment on the variation in mitral diastolic slope because they did not measure the E-F slope correctly. The F point of the echocardiogram of the anterior mitral leaflet precedes the mitral "a" wave and it is seen only when there is a distinct "a" wave or when diastole is long enough so the rapid and slow filling phases are evident. With sinus tachycardia there is shortening of diastole and fusion of the "a" wave with the E point, the F point is no longer seen, and the E-F slope is not obtainable. In figures 1, 2, 4 and 5 the "a" wave is intermittently seen, most likely due to slight changes in heart rate, which are not obvious at that paper speed, or in beam angulation. The authors still use the letter F on mitral valve echoes with no F point. This could also account for the alleged increase in E-F slope after pericardiocentesis as clearly seen in figure 7.

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Letter: E-F slope without "a" wave.
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