
The authors reply:
To the Editor:

As Dr. Mirsky points out, clinical determinations of cardiac contractility are complicated by errors inherent in obtaining data and difficulties in interpretation. We attempted in the manuscript to discuss these problems in detail. It does not seem fruitful to repeat the discussion; however two points deserve further comment.

Dr. Mirsky states: "In numerous studies with children it has been observed that analysis of data obtained from Statham SF-1 and P23Db pressure transducers yield significantly different results." We are not aware of any data to document that statement. The only published studies in children which we have seen are from the laboratory of Dr. Mirsky and his associates.1, 2 They utilized both catheter-tip transducers and fluid-filled catheter systems and imply that similar results were obtained. Our conclusion, based on detailed estimates of the errors introduced by the limited dynamic response of fluid-filled catheters presented in our paper, is that well-designed systems can be used successfully to obtain V_{max}.

In our initial studies,3 we obtained results in a small group of 12 patients which were similar to those of Gault et al.4 in 15 patients. As we expanded the series and included more patient groups, we found that the correlations based on V_{CG} at peak stress and peak V_{CF} were not useful in separating patients.

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Ultrasonic Echoes
To the Editor:

Ultrasonic echoes show motion of the mitral ring, not the opening of the valve. Ultrasonic echoes from the pericardium, the walls of the heart chambers and the valves have given useful diagnostic information. But we must realize that the echo labeled "anterior mitral leaflet" only shows motion of the ring, and tells us nothing about separation and approximation of the leaflets, for there rarely is any echo from the posterior leaflet.

Siggers' suggests that the motion observed is a composite of motion of the leaflet and that of the ring. Actually it has only the pattern of ring motion, familiar to us for a quarter of a century in the studies of calcific rings by fluoroscopy,2 slit kymography,3 and cinefluorography.4 It moves toward the apex during systole as the papillary muscles, through tension on the chordae and leaflets, pull the base of the heart down. It moves toward the atrium during diastole when muscular relaxation, the filling of the ventricle, and emptying of the atrium allow the ring to move back up.

The leaflet echo shows only the motion of the ring, whose septal edge has the largest excursion of any part of the heart,5 and a separation of a posterior leaflet from the conventional echo in diastole is observed only in markedly dilated hearts.6 The diastolic aperture is a crescentic slit and motion of the leaflets relative to the ring or to each other is "surprisingly" small.7 The actual motion of the leaflets, as described in dogs, has been confirmed in man by cineangiography.8 It has been best analyzed by ultrasonic echoes and cineangiograms of the mitral area in patients whose chordae and papillary muscles have been removed and who have an aortic homograft with semilunar cusps in the mitral orifice.9 These valve leaflets bulge toward the atrium throughout systole and remain wide open, pushed toward the apex, during diastole. Their pattern of motion matches that of normal mitral leaflets in studies which exclude annular motion, and is quite different from the conventional "anterior leaflet" echogram.

It is desirable to name an echo which moves like a ring as though it originated in the ring, and
V\textsubscript{max} as an Index of Contractile State in Man: The authors reply:
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