What is the actual contribution of atrioventricular plane displacement to left ventricular stroke volume?

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TO THE EDITOR: In the recent article by Carlsson et al. (4), the authors found that left atrioventricular plane displacement (AVPD) accounted for 60% of left ventricular (LV) stroke volume. The discrepancy with earlier reports suggesting lower values (3) mostly depends on the use of epicardial short-axis areas in the calculation of the stroke volume generated by AVPD. This approach takes into account that systolic longitudinal shortening, assuming myocardial incompressibility, leads to a redistribution of myocardial volume characterized by radial thickening, thus contributing to LV pumping (6).

A major issue in the interpretation of these findings is that the study included only subjects on opposite sides of the spectrum of LV systolic function, i.e., healthy individuals and athletes with normal systolic performance, and patients with advanced dilated cardiomyopathy and severely depressed systolic performance (ejection fraction, 22 ± 2%). In these subjects, longitudinal and circumferential systolic functions were likely to be either both preserved or both markedly impaired, and this may partially explain the similarity between groups in terms of AVPD contribution to LV stroke volume. However, caution is required in extending the conclusions of the study to patients with intermediate degrees of systolic impairment. We have recently shown that a depression in systolic longitudinal function precedes that in circumferential function even when the latter is evaluated at the midwall, since both M-mode annular excursion and tissue Doppler-derived peak systolic annular velocity are related to midwall indexes by inverse exponential functions (1). Also, according to these relations, the relative decrease in longitudinal indexes over the range of LV systolic function precedes that in circumferential function even when the latter is evaluated at the midwall, since both M-mode annular excursion and tissue Doppler-derived peak systolic annular velocity are related to midwall indexes by inverse exponential functions (1). Also, according to these relations, the relative decrease in longitudinal indexes over the range of mild to moderate systolic dysfunction is higher than that in circumferential indexes. As a consequence, many patients in the early and middle stages of systolic dysfunction may show even considerably reduced long-axis systolic annular excursion, despite a relative preservation of circumferential performance. For instance, based on our nonlinear models, a decrease in annular excursion from 15 to 10 mm (33.3% relative reduction) corresponded to a decrease in midwall shortening from 17.9% to 15.6% and in load-adjusted circumferential contractility from 96.3% to 83.3% (12.7% and 13.5% relative reduction, respectively). When we consider that systolic annular excursion is the primary determinant of LV long-axis excursion volume (5), a consistently lower contribution of AVPD to LV pumping is reasonably expectable in these subjects compared with normal individuals or patients with severe systolic impairment. Moreover, isolated longitudinal systolic dysfunction is often observed in the presence of normal LV diameters and wall thickness (2), so that the compensative mechanism due to increased short-axis epicardial area—allowing the maintenance of long-axis systolic excursion volume in advanced dilated cardiomyopathy—is not present in most of these patients.

Based on these considerations, the intriguing concept of the predominant contribution of AVPD to LV pumping could not be generalized. The nice study by Carlsson et al. (4) provides important insights for the comprehension of complex LV systolic dynamics, but further studies including subjects with progressive degrees of systolic impairment are needed to explore the effective AVPD contribution to LV stroke volume over the entire range of LV systolic function.

REFERENCES