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Diastolic Leading to Systolic Anterior Motion:

New Technology Reveals Physiology*

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The most effective therapy demands the most complete understanding of the mechanism. Noninvasive imaging has provided key early observations of left ventricular outflow tract (LVOT) narrowing and flow acceleration in hypertrophic cardiomyopathy (HCM) (1,2), resulting from developmental abnormalities (3). On the basis of these observations, the original lift (Venturi) theory for LVOT obstruction by systolic anterior motion (SAM) of the mitral valve in HCM implicated upper septal hypertrophy as the sole culprit, but was therefore unable to explain residual SAM after upper septal reduction or SAM without septal hypertrophy (4–12). Those observations were subsequently explained by a more complete understanding of the interaction of mitral valve structural abnormalities with altered flow, which positions slack leaflet portions into the path of flow diverted by a bulging septum to affect the posterior leaflet surface and generate form-drag forces pushing the leaflet anteriorly; these are compounded by Venturi lift forces later in systole as the LVOT narrows and flow accelerates above the valve (13–23). An important element in understanding the role of mitral valve abnormalities in causing SAM was the observation of Sherrid et al. (24,25) that the distal leaflet (13) is pre-positioned in the outflow tract, such that even low flow rates can move the distal leaflet anteriorly and superiorly to occlude the outflow tract.

Why does this pre-positioning occur (Figure 1)? Certainly, anterior papillary muscle (PM) positioning anteriorly and leaflet elongation may predispose to it; but, because motion of structures is driven by flow-induced forces, this leaflet orientation suggests the influence of as yet unknown flow vectors oriented to push the leaflet anteriorly. Because Doppler color flow mapping can only provide velocity components parallel to the beam, until now, actual

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flow *vectors* relative to cardiac structures, which could provide greater pathophysiologic information, could be estimated but not precisely determined (26–28).

As in the case of noninvasive measurement of pressure gradients from Doppler velocities, Doppler technology has yielded its richest clinical rewards when combined with fluid mechanics, the conservation principles of classical mechanics applied to fluids within ingeniously-selected control volumes. In this issue of the *Journal*, Ro et al. (29) have now applied a novel technology that provides vector maps of intracardiac flows from Doppler velocity components. The vectors are derived using the stream function of flow by applying conservation laws to measured Doppler velocity components within volumes of interest containing noncompressible blood.

The investigators applied this technology to answer our original question of leaflet pre-positioning in HCM: the majority of patients with obstruction have swirling flow vortices (26) that affect the posterior leaflet surfaces from below, moving them anteriorly. These vortices are generated by late-diastolic mitral inflow within the confined upper ventricular cavity, and pre-position the distal leaflets into the oncoming systolic wave, creating what can be termed *diastolic anterior motion* (DAM) as a precursor to SAM. In the remainder of patients, septal geometry redirects even the earliest, low-velocity systolic flows beneath the posterior leaflet, producing drag or pushing forces that drive SAM.

The study confirms that SAM results from anatomically-predisposed leaflets that protrude into these late diastolic/early systolic flows. Once SAM has begun, additional vectors directed posteriorly into the cul-de-sac beneath the posterior leaflet provide insight into its progression, and might conceivably help explain its resolution through a competing “negative or reverse Venturi” (14). Venturi forces act perpendicular to the axis of high-velocity jets. Such forces may act not only on the outflow side of the leaflets in an anterior direction (reinforcing SAM-septal contact), but also on their posterior surface in an opposing direction, initiating detachment of the leaflet from the septum, and thus promoting resolution of SAM.

Clinical implications of these findings include a better understanding of how abnormal valvular structures interact with flows determined by abnormal ventricular geometry, for example, to cause residual SAM with potential for dynamic obstruction in patients following septal reduction. These findings highlight the importance of septal reduction over a sufficient axial length to minimize anteriorly-directed vortex flows and flow redirected to affect the posterior leaflet surface to reduce residual SAM. These results further support the potential of leaflet modification and papillary muscle reorientation to reduce the anatomic substrate for SAM (30–39).

It will, therefore, be of great interest to learn how these flows change in patients with septal reduction, and how those changes correlate with residual SAM, obstruction, and mitral regurgitation (20). An intriguing future direction would combine 3-dimensional assessment of cardiac structure with computational flow modeling to answer the even more basic question of how these structures create abnormal vortices in the first place, and how we

might ultimately “sculpt” LV geometry to ensure the most physiologic outflow without obstruction (26).

Thus, this study uses a new technology to answer a longstanding question, indicating how SAM has a late-diastolic impetus, generated by diastolic anterior motion—DAM leading to SAM—which may be modified therapeutically. On a more general note, these observations emphasize that, due to the cyclic nature of cardiac action, phenomena occurring during one cardiac phase can best be understood by also considering the impact of the preceding one.

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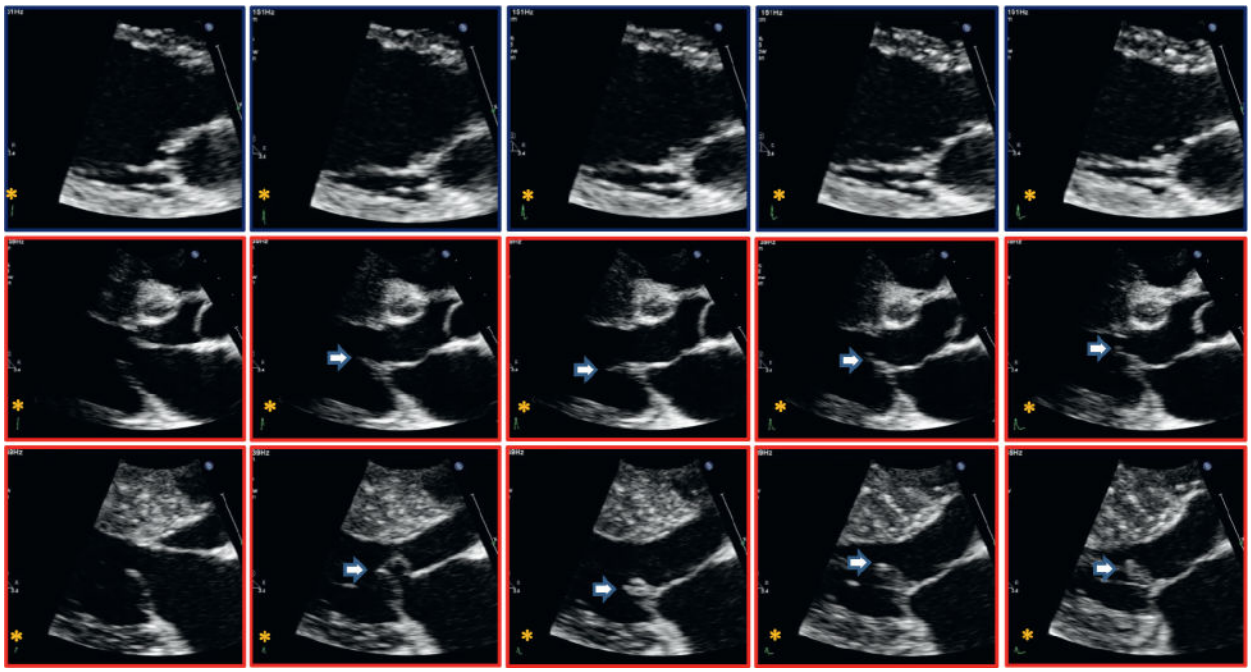

FIGURE 1. Comparison of Positioning and Movement of the Distal Mitral Leaflet From End Diastole to Early Systole in Normal Control and HCM Patients

(**Top**) Normal control patients and (**middle and bottom**) hypertrophic cardiomyopathy (HCM) patients. Anterior motion of the posterior leaflet as the leaflets move toward closure (late diastole and during isovolumic contraction) before onset of systolic anterior motion (**arrows**) is prominent in HCM, normally absent.