Editorial Comment

Insights Into the Physiologic Significance of the Mitral Inflow Velocity Pattern*

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The ability to measure mitral inflow velocities by Doppler echocardiography has allowed us to examine left ventricular filling noninvasively (1,2). Initially, the availability of this information generated great enthusiasm for extracting intrinsic diastolic properties of the left ventricle from a simple analysis of these velocities, in particular, the early and late diastolic filling velocities (the E and A waves) and their ratio. This initial enthusiasm has been tempered by the growing realization that inflow patterns result from a complex amalgam of ventricular, atrial and mitral valve properties and are profoundly influenced by superimposed loading conditions (3–5). Because of these multiple determinants, similar mitral inflow patterns could appear in different clinical settings if the time course of the transmitral pressure gradient is similar in those conditions.

The hypertrophic pattern versus decreased preload. One of the earliest characteristic abnormal patterns described (6–8) was the diminished early filling (E) wave and decreased E/A wave ratio in patients with hypertrophic cardiomyopathy. These findings were explained by delayed ventricular relaxation causing relatively high early diastolic left ventricular pressures and, therefore, a relatively low early diastolic gradient across the mitral valve. More recently, however, a similar pattern has been reproduced simply by decreasing left-sided filling pressures without intrinsic myocardial changes, for example, by nitroglycerin infusion: the decreased left atrial pressure lowers the early diastolic gradient and the corresponding E wave velocity (2.9–12). Conversely, the classic pattern in hypertrophic cardiomyopathy can be masked by associated mitral regurgitation, which raises left atrial pressure (7).

The restrictive pattern. Another distinctive filling pattern has been observed in restrictive cardiomyopathy (13–15). The early filling wave rapidly decelerates from its peak as left ventricular pressure rises rapidly in early diastole, and the late filling (A) wave is variably reduced, presumably by the high diastolic ventricular pressures. The E/A wave ratio is consequently increased, and the elevated ventricular pressures cause late diastolic mitral regurgitation in some patients.

The effect of severe aortic regurgitation. This restrictive pattern is mimicked by that now described by Oh et al. (16) in this issue of the Journal in severe aortic insufficiency of relatively acute onset. In 11 patients with this condition, the early filling velocity was higher than in matched normal control subjects and decelerated more rapidly from its peak: the late filling wave tended to be smaller, and the E/A wave ratio was abnormally high—changes that could be reversed by surgical repair. Late diastolic mitral regurgitation also occurred in 8 of the 11 patients.

Understanding these results in aortic regurgitation. Our understanding of these findings is improved by relating them to the fundamental determinants of transmitral flow. We have described (3,4) a mathematical model of ventricular filling that uses atrial compliance, ventricular compliance and relaxation, and mitral valve morphology to predict the time course of atrial and ventricular pressure and mitral velocity. The effect of aortic regurgitation can be studied by including aortic pressure and regurgitant orifice area in this model (giving five coupled nonlinear differential equations that must be solved simultaneously). The advantage of such analysis is that it allows us to isolate primary causes from secondary effects on the transmitral velocity pattern. For instance, Figure 1A shows the isolated effect of increasing aortic regurgitant orifice area with all other parameters held at roughly physiologic values (mitral area = 4 cm², exponential pressure-volume curves for the atrium and ventricle, ventricular relaxation time constant = 40 ms, initial left atrial pressure = 10 mm Hg, aortic pressure = 100 mm Hg). As the aortic regurgitant orifice area increases from 0 to 0.6 cm², the early filling wave undergoes profound changes, with peak velocity falling from 71 to 43 cm/s and deceleration time shortening from 124 to 66 ms. These changes reflect the rising ventricular pressure, which decreases the pressure gradient across the mitral valve. The filling volume for this portion of diastole falls from 37.4 to 15.7 cm³.

Thus, the isolated effect of worsening aortic regurgitation...
Therefore, these features of the inflow pattern must be interpreted in the context of atrial and ventricular loading conditions.

The pattern described by Oh et al. (16), along with findings such as proximal jet width (17,18) and the deceleration half-time of the aortic regurgitant jet (19), can serve as corroborative evidence of severe aortic regurgitation. It must be recognized that this mitral inflow velocity pattern, like the aortic regurgitant half-time, depends on left ventricular compliance, and is therefore most applicable with regurgitation of acute onset, as in the patients studied. Over time and with increasing compliance, diastolic ventricular pressures should fall, causing mitral inflow velocities to assume a more normal pattern. Similarly, Downes et al. (20) described diastolic mitral regurgitation in a series of patients with acute severe aortic regurgitation but not in a group with chronic insufficiency, also severe. Further, as Oh et al. (16) note, their findings were described in a group of patients known to have severe aortic regurgitation by clinical assessment and correlative tests; broader application of these findings will require further prospective study.

Finally, their results emphasize the need to study the effects of aortic insufficiency and left ventricular compliance on the Doppler pressure half-time method for assessing stenotic mitral valve area. The dependence of the half-time on factors other than mitral valve area has already been demonstrated by the inaccuracies in this method immediately after percutaneous mitral valvotomy (21). The potential of aortic insufficiency for decreasing the pressure half-time and causing mitral valve area to be overestimated requires further study as a function of the chronicity of the regurgitant lesion and ventricular compliance, in view of the variable initial clinical results (22-25).

Conclusions. Similar Doppler mitral inflow velocity patterns can be produced by different conditions causing similar changes in the time course of the transmitral pressure difference. Nevertheless, informed interpretation of these
patterns guided by the results of computational models and in vitro simulations can allow us to extract meaningful pathophysiologic insights from them.

We thank Arthur E. Weyman, MD for review of the manuscript.

References


