Echocardiography as a Noninvasive Swan-Ganz Catheter

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Diwan and co-investigators report in this issue of Circulation that the interval between onset of mitral E and annular early diastolic velocity (Ea) by tissue Doppler, T

E-Ea, can be used to estimate left ventricular (LV) filling pressure in patients with mitral valve disease. Garcia and colleagues were the first to report that the onset of Ea occurred 7.5 ± 3.5 ms after peak mitral inflow velocity in 7 patients with restrictive cardiomyopathy, whereas Ea started 22 ± 19 ms earlier than did E in the normal group. Subsequently, T

E-Ea has been shown to correlate with the time constant of LV relaxation (τ) demonstrated by Hasegawa and associates in their elegant animal experiment. With worsening of heart failure by rapid pacing, Ea progressively decreased in velocity and delayed in onset. Mitral E occurred coincidently with the termination of the early diastolic left arterial (LA) and LV pressure gradient at baseline and all stages of heart failure. In contrast, with increasing heart failure, Ea was progressively delayed after LA to LV pressure crossover, and T

E-Ea was related to τ. Rivas-Gotz, Nagueh, and their associates also demonstrated that T

E-Ea was prolonged after constriction of the circumflex coronary artery in dogs. In patients with heart failure, LV myocardial relaxation and compliance are decreased.

Although LV filling is initiated and enhanced by augmentable myocardial relaxation in healthy individuals, it is driven by high filling pressure in patients with heart failure because myocardial relaxation remains reduced, not being sensitive to preload. Myocardial relaxation also affects the isovolumic relaxation time (IVRT). As relaxation becomes abnormal, LV pressure falls more slowly and the opening of the mitral valve is delayed, resulting in a longer IVRT. IVRT, however, becomes shorter as LA pressure increases; hence, IVRT/τ LA pressure. Because T

E-Ea/τ, IVRT×T

E-Ea/LA pressure or LA pressure×T

E-Ea/IVRT. The same investigators demonstrated that pulmonary capillary wedge pressure (PCWP) is closely related to IVRT/T

E-Ea. In fact, IVRT/T

E-Ea<2 was found to have a sensitivity of 91% and a specificity of 89% for detecting PCWP >15 mm Hg. Patients with mitral valve disease or atrial fibrillation were not included in the first study, however, and the present report confirmed that both the ratio of IVRT to T

E-Ea and to τ correlate well with PCWP in patients with mitral valve disease or atrial fibrillation. These authors proposed different IVRT/T

E-Ea ratio cutoff values for different patient populations to predict PCWP >15 mm Hg: <3 for patients with mitral regurgitation, <4.16 for patients with mitral stenosis, and <5.59 for patients with mitral regurgitation who were evaluated prospectively or with atrial fibrillation. On repeat studies after the mitral valve procedure, an increase in the ratio by ≥1.5 identified most patients who had a decrease in mean PCWP ≥5 mm Hg.

Tajik and I wrote an editorial for the article by Rivas-Gotz and associates, in which history and clinical applications of various cardiac time intervals were reviewed, and our views on T

E-Ea as a surrogate for filling pressure and τ were expressed. My views for the article by Diwan et al are similar in that the concept of T

E-Ea is clever and based on the relationship between myocardial relaxation and LV filling in the healthy and the diseased heart. A potential limitation of applying this ratio clinically is a necessity for measuring cardiac time intervals from 4 different locations and 2 different cardiac cycles. We suggested an approach to record the onset of E and Ea simultaneously, which makes this approach more attractive for clinical use. Another drawback of IVRT/T

E-Ea is different cutoff values for different patient populations: 2 for patients with sinus rhythm without mitral valve disease, 3 for patients with mitral regurgitation, 4.16 for patients with mitral stenosis, and 5.59 for patients with atrial fibrillation. It would have been appropriate to provide an explanation for these different cutoff values. Despite these limitations, the findings by Diwan and colleagues clearly define the mechanism of diastolic filling in the healthy and the diseased heart. Their article also provides an opportunity to review the current status of the noninvasive assessment of diastolic filling pressure or PCWP in patients with or without mitral valve disease.

Reliable estimation of filling pressure is the most useful information from the echocardiographic assessment of diastole. Hemodynamically, elevation of filling pressure is a unifying feature for heart failure regardless of underlying cause. Because filling pressure cannot be directly measured noninvasively, we need to use all of the available indirect hemodynamic and functional information to achieve a reliable estimate of filling pressures. Almost always, if not always, patients who have increased filling pressures do have a structural or functional abnormality of the heart. It is unlikely that a patient will have increased filling pressure when cardiac structure and function are normal (ie, normal ejection fraction [EF], normal wall thickness and motion, normal annulus motion, normal LA size, normal pericardium, and no structural abnormalities). Therefore, echocardiographic assessment of filling pressure starts with 2D imaging.
which also provides an explanation when filling pressure is found to be elevated.

Doppler recording of mitral inflow velocities reflecting transmural pressure relationship is the first step after 2D imaging in echocardiographic assessment of filling pressure. Early diastolic mitral inflow velocity (E) is predominantly determined by myocardial relaxation and left atrial pressure. If LV is dilated and EF is markedly reduced (<35%), recording mitral inflow velocities alone is often sufficient.

Because almost all patients with systolic dysfunction have abnormal myocardial relaxation, mitral inflow velocity pattern demonstrates a characteristic filling pattern if filling pressure is not elevated; early diastolic velocity (E) decreases and deceleration time (DT) of E lengthens (>240 ms). As filling pressure increases, E increases (E and late diastolic velocity ratio, E/A, is >1) and DT shortens. Filling pressure is usually increased if DT is <160 ms. Even in patients with significant mitral regurgitation, DT correlates well with PCWP. Estimation of filling pressure by mitral inflow can be supported by other Doppler parameters such as pulmonary venous velocities and comparison of flow durations, but they may not be necessary in patients with reduced EF; however, in patients with normal EF, it is often difficult to estimate filling pressure by mitral inflow velocity alone.

When EF is normal, the status of myocardial relaxation needs to be assessed by visual assessment, M-mode recording of LV longitudinal motion, or recording of early diastolic velocity (Ea) of the mitral annulus. The normal heart meets a demand of increased LV filling without causing pulmonary venous congestion by augmenting LV suction with improved myocardial relaxation, or increased Ea velocity. If diastolic function is abnormal at rest, however, the heart may not be able to increase LV filling without causing high LA pressure because myocardial relaxation does not improve as well, even with exercise or increased demand for more filling. When relaxation is abnormal, not only is its magnitude (Ea) reduced but its onset also is delayed in reference to the onset of mitral valve opening or mitral inflow. From this observation, new indices for increased filling pressure have been identified. As LV filling pressure or PCWP increases, E/Ea ratio rises. When Ea is obtained from the medial mitral annulus, E/Ea >15 usually indicates PCWP >20 mm Hg. The ratio should be reduced to ≥10 if the lateral mitral annulus is used. Ea was found to correlate better than B-type natriuretic peptide with PCWP and is an easy index to obtain and possible in almost all patients. The correlation of E/Ea with PCWP has been found in patients with atrial fibrillation or sinus tachycardia as well as in sinus rhythm. The ratio was found to correlate well with PCWP in patients with significant functional mitral regurgitation and reduced EF. In patients with primary or organic mitral regurgitation, filling pressures could not be predicted by E/Ea. Therefore, the noninvasive estimation of filling pressure in patients with severe mitral regurgitation and normal EF has been problematic.

T_{E-Ea} is the latest of the echocardiographic parameters to correlate with τ, and IVRT/T_{E-Ea} appears to have a good correlation with PCWP even in the setting of a mitral valve disease, as reported in this issue of Circulation. Our group measured T_{E-Ea} in 44 patients with acute myocardial infarction and compared the interval with E/Ea. We were able to measure the time intervals in 40 patients, and T_{E-Ea} increased as E/Ea increased. (T_{E-Ea} was 6.2 ± 22, 22 ± 30, and 47 ± 24 ms for the groups with E/Ea <8, between 8 and 15, and >15, respectively; P <0.03.) Sohn and associates, who were the first to demonstrate that Ea was relatively insensitive to preload, could not find a delay in the onset of mitral annulus velocity compared with the onset of mitral inflow over the wide range of τ (31 to 70 ms), and therefore, no correlation between T_{E-Ea} and τ

References
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