

Editorial



Diastolic Function Assessment in Atrial Fibrillation Conundrum

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► See the article "Assessment of the Left Ventricular Diastolic Function and Its Association with the Left Atrial Pressure in Patients with Atrial Fibrillation" in volume 2 on page 55.

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Conflict of Interest

No potential conflict of interest relevant to this article was reported.

The authors of "Assessment of the left ventricular diastolic function and its association with the left atrial pressure in patients with atrial fibrillation" examined the usefulness of the American Society of Echocardiography (ASE) guidelines for the evaluation of diastolic function in estimating left atrial pressure (LAP). 1 LAP may be used as a surrogate to left ventricular end-diastolic pressure (LVEDP) and pulmonary capillary wedge pressure (PCWP) in the absence of mitral valve disease. 2-6 Elevated LVEDP negatively impacts left ventricular (LV) filling especially in the early period of diastole. In Doppler echocardiography, E/A ratio, the medial and lateral mitral annulus tissue velocities (e') and the E/e' highly correlate with LAP and LVEDP in the presence of sinus rhythm. 7-9)

Atrial fibrillation (AF) presents a challenging problem in the assessment of LV diastolic function using Doppler echocardiography. Several studies have shown the validity of Doppler parameters, yet contrasting at times, especially in the reliability of E/e' in estimating LAP.⁷⁴³⁾ Could the etiology of AF explain the variances?

The left atrium undergoes structural, metabolic, neurohumoral, and electrical changes in response to chronic external stressors. ¹⁴⁾ Animal experiments have shown that the mechanism for LA remodeling is different between atrial tachycardia-induced LA remodeling and LV pressure/volume overload-induced AF. While myolysis is the dominant structural change in atrial tachycardia, fibrosis is the underlying structural change in heart failure. Additionally, mechanism for AF development in tachycardia-induced atrial myopathy is multiple wavelet re-entry; while delayed afterdepolarization is the underlying mechanism for AF development in heart failure. ¹⁴⁾ The disparity in mechanisms of development of AF may partly explain the authors' findings as they correlate echo-Doppler parameters with LAP.

A direct assessment of LAP can provide a more complex and self-contained assessment of the LA condition. ¹⁵⁾ LAP is an important measure to detect early stages of LA structural and functional remodeling. ⁴⁾ An increase in LAP \geq 15 mmHg denotes LA hypertension, a final common pathway of cardiac diseases producing AF. ¹⁶⁾¹⁷⁾



Tissue Doppler velocities of the medial and septal annulus reflect the combined "pull and push" forces of the LA and LV during diastole. As the LVEDP increases, the LA pressure must increase to effectively fill the LV. As the pressure difference between these two chambers decrease, the tissue velocities decrease while blood flow velocity across the mitral valve may remain preserved with shortened deceleration time. The combined consequential effects to E and e' velocities support the elevated E/e' in diastolic dysfunction.

Doppler echocardiography has its own challenges and limitations in assessing LV diastolic function in the presence of AF by the variability in cycle length, the absence of organized atrial activity, and the frequent occurrence of LA enlargement regardless of filling pressures. ¹⁸⁾ It is critical to adhere to ASE recommendations regarding imaging and measurement of both E and e' if they were to reliably evaluate diastolic function and estimate LAP. ¹⁸⁾

The study by Kim et al.¹⁾ poses an important question. Do the ASE recommended guidelines in assessing diastolic function reliably depict LAP in the presence of AF? ASE prefaced its guidelines with a definition of LV filling pressure.¹⁸⁾ The guidelines distinguished LAP, PCWP and LVEDP to refer to LV filling pressure and also pointed out the correlations of these pressures with known echo-Doppler parameters of diastolic function. Their study aptly addresses this issue.¹⁾

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