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EDITORIAL

Left ventricular diastolic dysfunction: An old, known entity in a technologically modern era

KEYWORDS
Left ventricular diastolic dysfunction;
Arterial hypertension;
Preserved left ventricular ejection fraction;
Left ventricular hypertrophy

Left ventricular diastolic dysfunction (LVDD) manifests early and is a common pathophysiological pathway leading to many cardiovascular diseases.[1] Diastolic dysfunction can present in a spectrum of diverse patterns, ranging from a simple delay of ventricular relaxation without significant hemodynamic changes, to pulmonary venous congestion due to ventricular diastolic pressure elevation with displacement of the upper, left pressure-volume loop (Figure 1). Cardiac catheterization provides data on the left side of the heart, the right side of the heart, systemic and pulmonary arterial pressures, vascular resistances, cardiac output, and ejection fraction. These data are often used as markers of cardiac preload, afterload, and global function, although each of these parameters reflects additional complex interactions between the heart and loading conditions. Hemodynamic evaluation has long been considered to be the "gold-standard" method for characterizing load, contractility, diastole, and, ventricular-arterial interactions, but has not been adopted for clinical practice. Attaining a working understanding of each of these elements remains paramount to properly interpreting hemodynamic changes in patients who have heart failure with preserved ejection fraction (HF-PEF). However, it is worth noting that recently, conventional and newer echocardiographic methods[2–6] have been used to accurately evaluate patients with HF-PEF (Table 1). Recently, Kasner et al.[4] reported that the quotient of simultaneously measured E/E' and LV end-diastolic volumes using TDI and 3DE allows a simple determination of LV stiffness in the clinical routine. The combination of left ventricular dysfunction and coronary artery disease in hypertensive patients usually aggravates LV diastolic parameters, as Vlasseros et al.[5] have published.

Any type of heart disease that leads to myocardial structural alteration and/or pericardial effusion can cause LVDD. Sometimes this structural abnormality is macroscopically evident as LV hypertrophy, whereas in other cases, diastolic dysfunction is linked to abnormalities of the cellular mechanisms of myocyte relaxation and is caused by hypoxia and/or ischemia. Diastolic dysfunction is a dominant feature in many HF-PEF patients, and many factors contribute to diastolic dysfunction, including both vascular and myocardial stiffening. Diffuse stiffening that occurs throughout the cardiovascular system because of aging or comorbidities interferes with the forces that normally develop during systole to produce ventricular suction and thus reduces early diastolic filling. Left ventricular diastolic dysfunction may be related to changes in the extracellular matrix or changes that are intrinsic to myocyte stiffness, microvascular dysfunction, and metabolic abnormalities.

Left ventricular hypertrophy is the most common pathological condition that induces LVDD and is associated with a poor cardiovascular prognosis. Furthermore there has been no direct comparison of the LVDD characteristics between patients with and without LVH. Therefore, Kattel et al.[7] hypothesized that mild to moderate LVDD with LVH carries a higher risk for developing severe LVDD and diastolic heart failure than LVDD without LVH. The authors studied 2 LVDD groups, a group with mild to moderate LVDD with LVH (LVH group) and a group with mild to moderate LVDD without LVH (non-LVH group), to determine the echocardiographic differences between the two groups. The authors compared two major echocardiographic characteristics, the left ventricular filling pressure (FP) and the

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Tei-index (also known as the myocardial performance index), between those 2 groups. Furthermore, they assessed whether LVH had any additional effects on LV diastolic function and overall cardiac function in patients with mild to moderate LVDD.

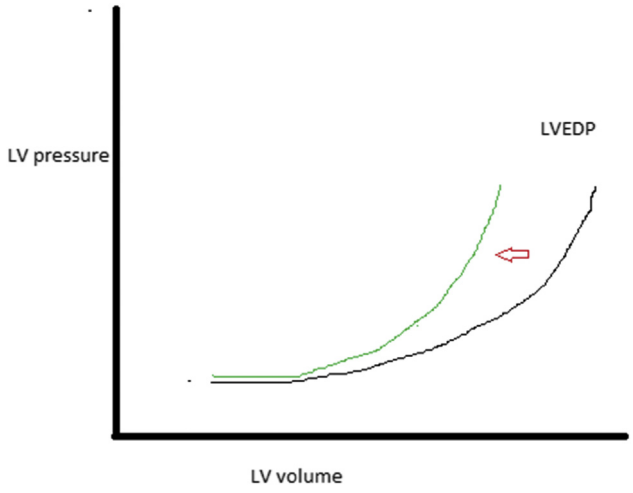

Figure 1 The black line depicts the curve in a normal person. The curve (green) that is shifted up and to the left (red arrow) indicates the effect of increased passive chamber stiffness.

Table 1 Conventional and newer echocardiographic parameters in the evaluation of the LV filling pressure.

Method	
Transmitral inflow (E/A)	Normal>1 Impaired relaxation <1 Restrictive>2
Presence of L wave	Indicates advanced LV dysfunction
DT	Normal 160–240 ms Impaired relaxation >240ms Pseudonormal 160–240 ms Restrictive <160ms
Pulmonary venous flow	S/D Normal >1
Tei index	Normal <0.4
TDI	Normal Lateral E'>10 cm/s Normal Septal E'>8 cm/s Normal Averaged E/E' ratio<8
LA evaluation	Normal Volume index<34 ml/m^2
Speckle tracking imaging	Normal LA strain>18% E/E'SR-ST>93 surrogate marker of elevated LV filling pressure
Impaired vortex generation	Advanced diastolic dysfunction

The study by Kattel et al. demonstrated that FP was significantly higher when LVDD was accompanied by LVH. Higher FP was also reflected by the significantly larger left atrial size and greater left atrial volume index (ml/m^2) in the LVH group. In addition, a higher Tei-index was attributed solely to poorer LV diastolic function in the LVH group.

A limitation of this study is that the authors did not use newer echocardiographic techniques, such as speckle tracking imaging, that could detect earlier and more accurate information regarding LVDD in hypertensive patients without LVH.

Because of the potential for a poorer prognosis for patients with mild to moderate LVDD, it may be beneficial to have closer monitoring and more aggressive hypertension management when LVH co-exists.

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