Impaired Longitudinal Function in Aortic Stenosis: Abnormal Contractility or Afterload?

Deterioro de la función longitudinal en la estenosis aórtica: contractilidad o poscarga anormal?

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Aortic stenosis is the most common single valvular lesion seen in the adult population. Patients with aortic stenosis may remain asymptomatic for many years. As the disease progresses symptoms associated with physical activities will appear. The classic triad of angina, syncope or heart failure and its prognostic significance (1, 2) highlight the relationship between increased afterload, LV remodeling and impaired contractility in patients with severe aortic stenosis. Worsening aortic stenosis leads to progressive increments in the aortic transvalvular gradient and left ventricular pressure overload. The LV adapts to the systolic pressure overload developing concentric hypertrophy resulting in increased wall thickness with normal chamber volume. (3) The increased wall thickness allows normalization of wall stress. However, the development of hypertrophy may have negative consequences, including subendocardial ischemia leading to increased interstitial fibrosis, (4) and systolic and diastolic dysfunction.

Echocardiography is the most useful diagnostic tool for the evaluation of patients with suspected or known aortic stenosis. In addition to evaluating valvular stenosis severity, echocardiography is of paramount importance to evaluate LV function. Echocardiography may be used to define circumferential shortening at the LV endocardial surface or at the mid-wall, given its high spatial resolution. Tissue Doppler velocities and speckle tracking have been evaluated in patients with aortic stenosis. In patients with severe aortic stenosis and normal ejection fraction, septal annular A’ velocity <9.6 cm/s appears to be associated with reduced event-free survival (5). In aortic stenosis patients, diastolic untwisting is prolonged. (6, 7) To be interpreted appropriately, these measurements must be normalized to produce regional strain and strain rate. The spiral architecture of the myocardial fiber bundles determines strain deformation in multiple directions. Thus, changes in LV geometry during LV systole relate primarily to radial (short axis), longitudinal (long axis) and meridional (LV torsion) strain. Although myocardial velocities and strain have been used as a surrogate of LV contractility, it is important to consider that the systolic performance of the LV is best described by its ability to eject a volume of blood (stroke volume) against resistance (afterload). The LV stroke work is defined as the product of developed pressure and total stroke volume. Urheim et al first used strain Doppler echocardiography and invasively-determined pressure for the calculation of myocardial work from pressure-strain loop areas. More recently, Russell et al validated and proved that a noninvasive method of regional LV pressure-strain area corresponded well with invasive methods and it measured myocardial work which correlated well with myocardial glucose metabolism on positron emission tomography (PET) studies. (8) We have more recently utilized these principles to differentiate amyloid (impaired contractility) from hypertensive cardiomyopathy (increased afterload) by estimating myocardial work as the product of peak systolic blood pressure and peak systolic longitudinal strain.

Abnormalities in LV regional deformation have been well described in patients with aortic stenosis. Further, the sensitivity of longitudinal strain in particular to changes in afterload has been previously established, suggesting that longitudinal strain is impaired in patients with LV hypertrophy, being reduced when compared to controls, whereas radial and circumferential strain are not. (9)

In this issue of La Revista Argentina de Cardiología, Migliore et al studied 101 patients with severe AS and 63 normal control subjects to determine the relationship between LV longitudinal systolic function, afterload and myocardial contractility. They demonstrated that lateral mitral annulus systolic displacement and tissue Doppler systolic (S’) velocity correlated directly with shortening indexes as ejection fraction (EF) and mid-wall fractional shortening (mFS) and inversely
with afterload indexes such as end-systolic wall stress (ESS). However, they observed no significant correlation between systolic longitudinal function and LV contractility indices. Their findings support that the observed alterations in longitudinal function may be a response to an afterload mismatch and not necessarily indicate impaired LV contractility. Indeed, their conclusions are supported by the previously published study by Carasso et al, who demonstrated that impaired longitudinal strain in patients with aortic stenosis improves immediately after aortic valve replacement (11). The findings of this study are quite important, as they not only provide an insight into the pathophysiology of aortic stenosis, but they also allow us to interpret correctly the findings observed on echocardiographic studies. Furthermore, the implications from this study may help us in the near future to refine the criteria for determining the appropriate time for aortic valve replacement in asymptomatic subjects, a topic of continuing debate. Thus, a disproportionate alteration in LV longitudinal function to the degree of afterload mismatch may be indicative of true myocardial dysfunction and may lead us to recommend valve replacement early. Finally, once again Migliore et al remind us that ingenuity is more important than technology when it comes to advancing our knowledge. Not having the latest technology available should never discourage young investigators from conducting important clinical research in countries like Argentina.

Conflicts of interest
None declared.

(See authors’ conflicts of interest forms in the website/Supplementary material).

REFERENCES