EDITORIAL

Aortic Stenosis: Beyond the Aortic Valve...

Estenosis aórtica: más allá de la válvula ….

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The left ventricle (LV), the aortic valve, and the aorta form a functional unit that is responsible for delivering blood flow to the organs. Any abnormality in one or more components of this unit hinders systemic perfusion and has an impact on morbidity and mortality. Aortic stenosis (AS) is the most common valvular heart disease as age increases, and it creates an imbalance in this functional unit. In patients with AS, the LV often faces a double load: a valvular load imposed by AS and an arterial load caused by a decrease in systemic arterial distensibility (or an increase in systemic vascular resistance) in the context of comorbidities (e.g., age, smoking, hypertension, diabetes). (1) In patients with AS, the LV hemodynamic load is not solely determined by the severity of the stenosis, but is also influenced by systemic vascular resistance, volume flow rate, and body size. Therefore, valvuloarterial impedance (Zva) represents the pressure cost in mm Hg for each systemic mL of blood indexed for body size pumped by the left ventricle during systole, considering the valvular load and the arterial load. Ventricular-arterial coupling (VAC) is calculated as the effective arterial elastance (Ea) to LV end-systolic elastance (Ees) ratio measured in the LV pressure-volume loop. (1) Effective arterial elastance is calculated as the mean LV systolic pressure to stroke volume ratio and is often considered a measure of the arterial hemodynamic load imposed on the LV. End-systolic elastance describes the maximum pressure that the ventricle can develop at any given LV volume and is an index of myocardial contractility, relatively insensitive to changes in preload, afterload, and heart rate. The Ea/Ees ratio is useful to assess the mechanical efficiency of the cardiovascular system and the interaction between cardiac performance and systemic vascular function. (1) Effective arterial elastance is calculated using LV end-systolic pressure and is therefore influenced by arterial load but does not consider valvular load.

In the study "Relationship Between Ventricular-arterial Coupling and Stage of Extravalvular Damage in Aortic Stenosis", Migliore et al. evaluated the relationship between VAC and the different stages of extravalvular damage in AS. (2)

The authors analyzed 205 patients. Mean age was 70 ± 11 years, 59 % had hypertension (HTN), 50 % were in FC III-IV and 40 % had a left ventricular ejection fraction (LVEF) < 60 % (mean LVEF 52± 19%). Baseline peak and mean aortic valve gradients were 70 mm Hg and 41 mm Hg, respectively. Baseline systolic pulmonary artery pressure was 46 ± 17 mm Hg. (2)

Patients were divided into five groups according to extravalvular cardiac damage: 0, no cardiac damage; 1, LV systolic or diastolic dysfunction or LVEF < 60%; 2, left atrial dilation, ≥ moderate mitral regurgitation or atrial fibrillation; 3, signs of pulmonary hypertension (≥ 60 mm Hg), ≥ moderate tricuspid regurgitation; and 4, right ventricular damage or stroke volume index < 30 mL/m^2. As the stage of myocardial damage progressed, VAC impairment increased. The authors concluded that the alteration of VAC that occurs from stage 2 to 4 is due to an increase in Ea without significant changes in the level of contractility (Ees). The progression of extravalvular myocardial damage appears to be associated not only with valvular disease but also with the characteristics of the arterial vasculature. (2)

Measurement of Zva showed differences in stage 4 versus the other stages as an expression of greater valvular heart disease progression.

Some considerations of the study arise from the study population and the above-mentioned results:

This population has long-standing severe AS,

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which is critical for a significant proportion of the population (as indicated by the fact that 107 patients, 52%, had an advanced stage). This assertion is supported by the authors, who note that there are no patients in stage 0, with elevated gradients and reduced left ventricular ejection fraction (LVEF), suggesting that they have an increased valvular load and reduced LV compliance (although their contractility, as measured by Ees, was not significantly different between the groups with structural damage) as the average E/e' ratio is 16.

In turn, 82% of stage 4 patients presented low aortic flow which is associated with higher Ea and Zva.

Hachica et al. analyzed Zva and observed that a cut-off value ≥ 3.5 mm Hg identified a population with excessive aortic impedance (hemodynamic load) that was associated with a more severe disease independently of the events. (3)

However, other studies have not identified Ea as a clinical predictor because it depends on aortic stiffness and heart rate (insensitive to pulsatile flow) and does not take into account valve flow or valve load. (4)

The Ea/Ees ratio was an independent predictor of mid-term outcomes after transcatheter aortic valve implantation (TAVI). (5)

Migliore et al. found increased Ea/Ees ratio in severe AS patients with symptoms or heart failure. (6)

Hypertension and stiffness may also alter VAC and thus accelerate the development of symptoms of AS. (7) In a series of 193 patients with AS, those with HTN developed symptoms earlier despite their valve areas were larger. (7)

Furthermore, in patients with low-flow, low-gradient AS, Zva was associated with lower mean aortic gradient. (8) Higher Zva is associated with impaired longitudinal LV systolic function, (9,10) and lower survival. (3,10) As it occurs with Ea, Zva has limitations because complex pulsatile afterload cannot be represented by a single parameter.

From a clinical perspective, adapting to these measurements would present a challenge and they would continue to be regarded as interesting physiological concepts. The measurement of blood pressure and keeping its values within normal limits are simple measures that can be taken to avoid further impairment of valvular afterload and ventricular function.

Does this limit the value of this work? If one considers that physiology is what allows us to understand biological phenomena in order to better understand diseases, it is always welcome to express pathophysiological bonds, and this is the merit of this research.

New methods (4D flow phase-contrast magnetic resonance imaging, algorithm-based P-V loops) are likely to give us a broader perspective on how to more accurately assess aortic stenosis beyond the valve. (11)

Conflicts of interest
None declared
(See authors conflicts of interest forms on the website).

Ethical considerations
Not applicable.

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