Editorial

How to relate diastolic left ventricular dysfunction to the results of stress echocardiography in aortic stenosis?

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Abstract: In aortic stenosis (AS), altered functional capacity may be not only a result of impaired systolic performance but likewise of diastolic left ventricular (LV) dysfunction. In asymptomatic severe AS, in borderline severe AS and in low flow, low gradient AS with reduced ejection fraction (EF), assessment of systolic and diastolic LV function should be completed by evaluation of functional capacity using low dose dobutamine stress echocardiography (SE).

Keywords: Aortic stenosis (AS); stress echocardiography (SE); diastolic left ventricular dysfunction

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Stress echocardiography (SE) is known to be expedient and well tolerated for patients in order to assess aortic stenosis (AS) prior to surgical or percutaneous therapies. Beside assessment of severity, adequate evaluation requires complete functional assessment and discrimination of low flow, low gradient AS with and without preserved ejection fraction (EF) as well as pseudo-severe AS. The clinical scenarios where SE aids diagnosis and has prognostic value include also the evaluation of asymptomatic severe AS and moderate AS with symptoms. Up to now, evaluation of the relation between AS and systolic left ventricular (LV) function is to the fore. In contrast, estimation of diastolic LV function has been less of an issue prior to aortic valve replacement (AVR), although LV compliance is well known to be impaired. As a result, diastolic LV dysfunction is tightly associated with AS what is also of utmost importance for postoperative management (1).

Low dose dobutamine SE starting at 5 μg/kg per minute and increasing in 5 μg/kg per minute increments every three to five minutes up to a maximum of 20 μg/kg per minute is known to be the diagnostic approach of choice in patients with low flow, low gradient severe AS with reduced EF, to distinguish truly severe AS from pseudo-severe AS (2) and to determine contractile reserve. Truly severe AS shows only small changes in valve area with increasing flow rate, but a significant increase in gradients, whereas pseudo-severe AS shows a marked increase in valve area but only minor changes in gradients. AVR should be considered in symptomatic patients with low flow, low gradient with reduced EF, and evidence of contractile (or flow) reserve (class of recommendation IIa, level of evidence C according to current ESC Guidelines), and may be considered in those without flow reserve (class IIb, evidence C) (3). Benefits of surgery are highest in those individuals presenting with preserved contractile reserve defined as increase in stroke volume by at least 20%. Transcatheter AVR may provide a therapeutic approach with lower periprocedural risk in patients without contractile reserve (4). However, the use of SE, either by use of exercise, vasodilators or dobutamin must be considered under-utilized in detailed diagnostics of valvular heard disease (5). In particular this is true for its use in asymptomatic severe AS which is slowly emerging (6). Although the prognosis for those who are asymptomatic is favorable with a <1% per year mortality (7), one third of patients who claim to be asymptomatic actually develop symptoms on exertion. Thus, an increase in mean aortic pressure gradient by ≥18 mmHg during exercise has been reported to be an independent predictor of cardiac events as are symptoms, death, AVR, and acute

hospital admission (8). More recently, an increase in mean gradient of >20 mmHg from a resting mean gradient of about 35 mmHg was demonstrated to be associated with a 9-fold increase in event rate (AVR because of symptom development or death) during the next two years (9).

Although Rassi *et al*. could not directly associate diastolic LV dysfunction with functional capacity, they demonstrated diastolic dysfunction of stage II and higher to be as predictive as functional capacity regarding adverse events in patients with AS. Dyspnea is known to be mainly associated with AS severity, but is also influenced by diastolic dysfunction (10). Diastolic dysfunction representing LV backward failure and impaired functional capacity primarily representing forward failure are apparently two sides of the same coin complementing each other. LV diastolic dysfunction (11) and exercise intolerance (12) often persist even after corrective surgery suggesting the presence of irreversible myocardial damage before surgery. These disappointing results are partly due to poor sensitivity of LVEF as a marker of myocardial damage and thus some investigators support early surgery in asymptomatic patients with normal LVEF (13). Especially in low flow, low gradient AS with preserved EF, impairment of mitral annulus displacement (14) due to concentric LV hypertrophy (15) favors diastolic LV dysfunction to be the main reason for the lowered stroke volume index (<35 mL/m^2) in this challenging subset of patients. Prospective and randomized trials are desirable to clearly associate severe diastolic dysfunction with low output and lacking functional capacity, since prognosis of patients presenting with such a scenario is dismal.

Even if patients present with normal EF, a process of subclinical malfunction ensues in those subjects with structural and functional derangements and diastolic dysfunction as demonstrated by gate gadolinium enhancement on cardiovascular magnetic resonance (16), Doppler echocardiography and increased levels of B-type natriuretic peptide (1). As a consequence, diastolic LV dysfunction may be integrated as an adjunct criterion for surgical intervention in this group of patients. Symptoms and reduced functional capacity are often linked to specific hemodynamic patterns associated with AS: smaller LV cavity and reduced output for syncope and missing increase in stroke volume index during low dose SE versus more advanced diastolic dysfunction for dyspnea and as well reduced functional capacity due to impaired LV filling. Hence, at least in some subsets of patients, comprehensive intracardiac hemodynamics including stroke volume at rest and on exertion as well as diastolic function need to be evaluated in addition to aortic valve area and pressure gradient for entire assessment of AS (17).

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