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## Stress Echocardiography in Prosthetic Heart Valves

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## Dear Editor,

Echocardiography is the best method for assessing the function of a prosthetic valve (PV) and yields data as valuable as those obtained in the evaluation of a native cardiac valve (1). However, there may be a considerable difference in the assessment of occluders, trans- and paravalvular flows and gradients, and fluid dynamics between a PV and a native cardiac valve.

Transvalvular flow is eccentric in monoleaflet valves and also is formed of 3 separate jets in bileaflet valves, with flow velocity being greater in the central orifice jet than in the other 2 lateral orifice jets.

As most PVs are innately and inherently stenotic, the effective orifice area (EOA) of a PV is frequently small in relation to the patient's body size, an important phenomenon known as prosthesis/patient mismatch (PPM). In aortic valves, PPM is defined1 as moderate when the indexed EOA is  $\leq$  0.85 cm<sup>2</sup>/m<sup>2</sup> and is defined as severe when the indexed EOA is  $< 0.65 \text{ cm}^2/\text{ m}^2$ . In the mitral valve, the cutoff points are 1.2 and 0.9 cm<sup>2</sup>/m<sup>2</sup>, correspondingly. Importantly, PPM has been linked to suboptimal symptomatic improvement, weakened exercise capacity, pulmonary artery hypertension, incomplete regression of left ventricular hypertrophy, increased heart events, and even mortality rates after valve replacement (2-4). According to the literature, PPM is the most common cause of an increased transprosthetic gradient; however, it is essential to distinguish this state from other acquired PV stenoses, which may result from significant leaflet calcification on bioprosthetic valves and pannus overgrowth or thrombus creation on mechanical PVs.

The presence of a high mean transprosthetic gradient (increase of 15 - 20 mmHg for the aortic PV and 5 - 7 mmHg for the mitral PV) with/without symptoms necessitates quick assessment. It is essential to determine whether the high gradient is due to PPM, a real intrinsic stenosis of the PV, or a localized increased gradient (a phenomenon that happens only in bileaflet mechanical PVs). Infrequently, an unusually increased jet velocity secondary to a localized gradient may indeed be documented by continuous-wave Doppler through the small central and slit-like orifice of the bileaflet mechanical PV. This phenomenon results in an abnormally low EOA and a high gradient, thereby mimicking the signs of real intrinsic prosthetic dysfunction (1, 4).

Because abnormally and normally functioning PVs can yield similar estimated resting gradients by transthoracic echocardiography, it might be difficult to differentiate between high gradients due to factitious reasons or PPM. Importantly, in these conditions, stress echocardiography can be valuable and useful in excluding or confirming hemodynamically important PV stenosis or PPM, particularly when there is discordance between the hemodynamics of the prosthesis assessed by echocardiography at rest and the patient's symptomatic position (1, 5, 6). In contrast to a well-matched and normally functioning PV (containing a bileaflet PV with a localized elevated resting gradient), a stenotic mechanical PV or PPM is commonly correlated with a noticeable increase in gradient with exercise, pulmonary artery hypertension, development of signs and symptoms, and impaired exercise capacity on stress echocardiography (7, 8).

A disproportionate rise in transvalvular gradient (> 20 mmHg for the aortic PV or > 12 mmHg for the mitral PV) commonly shows severe PV dysfunction or PPM. Interestingly, elevated resting and also stress gradients happen more frequently in biological rather than in mechanical PVs, in stented rather than in stentless bio-PVs, in smaller ( $\leq$  21 for aortic and  $\leq$  25 for mitral prostheses) rather than in larger PVs, and in mismatched rather than in non-mismatched PVs. Indeed, the value of transprosthetic gradient in exercise settings is basically defined and confirmed by the indexed EOA (based on body surface area), prosthesis size and model, and also pathological obstruction of the PV due to thrombus, pannus, and/or significant calcification (1, 5, 8).

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As is the case in a patient with a native aortic valve that has low-flow, low-gradient aortic stenosis, dobutamine stress echocardiography can be helpful in discriminating true PV stenosis from pseudostenosis in cases with PV concomitant with significant low cardiac output. In a patient with pseudostenosis and low cardiac output, at rest, transprosthetic flow rate and therefore the force and power tolerated by the leaflets are significantly low to fully open the PV. However, during the infusion time of dobutamine in these cases, there appears a considerable elevation in the EOA of the prosthesis with the rising of flow rate, with negligible or no increase in PV gradient. Nonetheless, true significant prosthesis stenosis or PPM is allied to no important elevation in the EOA and a noticeable elevation in gradient with the infusion of dobutamine, frequently with additional helpful diagnostic changes like left ventricular dysfunction or discernible increase in pulmonary arterial pressure and presence of some symptoms (5).

It is deserving of note that dobutamine stress echocardiography or exercise does not differentiate between acquired PV stenosis and PPM inasmuch as in both conditions, the gradient elevates significantly while the EOA remains small with stress; and in this state, the EOA data obtained during stress echocardiography should be compared with the standard reference values of the EOA for the size and model of the particular PV inserted for the patient (1). An EOA considerably lower than its normal reference value should arouse suspicion of PV dysfunction. Nevertheless, if the assessed EOA is in the normal values and the measured indexed EOA is low, PPM should be taken into consideration (1, 5, 8).

In patients receiving surgical management for ischemic mitral regurgitation, a restrictive annuloplasty in tandem with coronary artery bypass grafting surgery is the most common management approach. This modality is, however, associated with a moderate rate of reappearance of mitral regurgitation. In addition, restrictive annuloplasty may give rise to functional mitral stenosis in some cases. In patients with the postoperative signs and symptoms of either functional mitral stenosis or residual mitral regurgitation, exercise stress echocardiography may be useful to evaluate exercise capacity and symptoms. Furthermore, assessment of hemodynamics with stress echocardiography can confer additional significant data on the importance of mitral stenosis or/and residual regurgitation (9).

There is a paucity of information in the existing literature on the diagnostic value of stress echocardiography in the management of cases with PVs or mitral annuloplasty rings, with the few studies having been conducted hitherto focusing on a low volume of patients. Indeed, although research has already demonstrated the efficacy of stress echocardiography in the assessment of PPM and PV dysfunction, its importance in risk stratification and clinical prognosis and outcome has yet to be fully elucidated. More studies are, therefore, necessary in this interesting field.

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