Afterload mismatch revealed by an exercise biphasic response in aortic stenosis

Sténose aortique hémodynamique sévère révélée par une réponse biphasique à l’effort

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A 71-year-old man with asymptomatic severe aortic stenosis was referred to our stress echocardiography laboratory for risk stratification. He had no coronary risk factors but led a sedentary life. A symptom-limited bicycle exercise was performed in the supine position. The patient reached the target heart rate (128 beats/min) and developed moderate dyspnoea at a workload of 100 W. The systolic blood pressure initially increased from 130 to 150 mmHg whereas no further changes occurred at a higher exercise level. Baseline transthoracic echocardiography (rest) confirmed the presence of severe valvular aortic stenosis, with an aortic valve area of 0.78 cm² (outflow tract time velocity integral 20 cm) and a mean transaortic pressure gradient of 40 mmHg. At low-level exercise, the increase in longitudinal long-axis function (peak negative strain rate obtained off-line by tissue Doppler reconstruction at the level of the septal and lateral walls) (Fig. Top panel) was accompanied by a significant rise in transaortic pressure gradients (Fig. Bottom panel). The calculated aortic valve area was 0.71 cm² (outflow tract time velocity integral 19 cm) indicating absence of valve compliance. At peak exercise, the transaortic pressure gradient decreased as a result of a significant impairment of left ventricular function (decrease in peak negative strain rate). The aortic valve area remained fairly unchanged (0.69 cm²) whereas the left ventricular outflow tract time velocity integral decreased (15 cm), indicating reduced flow across the aortic valve. No inducible ischaemia was observed during test and no concomitant coronary artery stenosis was found at angiography.
Comment

In this patient, the inadequate left ventricular adaptation to exercise is due to exercise-induced afterload mismatch. Initially, the heart adapted appropriately to the increased afterload. The recruited inotropic reserve — increase in longitudinal myocardial deformation — was accompanied by a significant rise in transaortic pressure gradient. At high-level exercise, the aortic valve compliance recruitment was fully exhausted and a mismatch between afterload and contractility occurred as expressed by reduced long-axis function and aortic flow.

Conflicts of Interests

No conflict of interests.