EDITORIAL

Exercise testing in patients with aortic stenosis: clinically useful

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by Orłowska-Baranowska et al, see p. 332

Exercise testing (ET) is recommended by both European and American guidelines[1,2] for patients with asymptomatic severe aortic stenosis (AS). Revealed symptoms are a class Ia indication for aortic valve replacement, and a fall in systolic blood pressure a class IIa indication.[1,2]

These recommendations are based on consistent findings showing that ET is useful. Approximately 40% of patients with asymptomatic severe AS develop symptoms during exercise,[3] and between 27% and 65% of patients with moderate and severe AS have a positive test result defined as symptoms or blood pressure and electrocardiography changes.[4-7] A positive ET, compared with a negative one, is associated with approximately 8-fold increased risk of developing spontaneous symptoms, sudden death, or heart failure within a year.[3,8] We showed a 2-year event-free survival of 46% with revealed symptoms versus 70% without revealed symptoms.[9]

Exercise testing in AS is important because the risk of death rises sharply as soon as symptoms develop. However, symptoms may be disguised because patients slow down to avoid them or attribute them to old age or simply forget when being questioned at a clinic. It is not unusual for a patient to claim to be asymptomatic with normal exercise capacity and to develop pulmonary edema on ET. Therefore, ET is a way of revealing patients with early or suppressed symptoms who need surgery to avoid unnecessary complications of AS.

It is thus interesting that a study by Orłowska-Baranowska et al[10] in the current issue of *Polish Archives of Internal Medicine* (*Pol Arch Intern Med*), challenges this received wisdom. In their study, 89 consecutive patients (mean age, 59.5 years; 53 men) with apparently asymptomatic AS had bicycle exercise at baseline and then every 6 months. The findings were: 1) all 244 ETs were negative; 2) despite this, 39 patients (44%) developed spontaneous symptoms before the next test; 3) 45 (51%) were taking β-blockers and were more likely not to achieve 85% target heart rate during ET and to develop symptoms requiring aortic valve replacement; 4) ET was safe.

How can these results be explained? The authors excluded 24 of 120 patients (20%) because of spontaneous symptoms. Highly accurate history taking could potentially ensure that all patients who might have had revealed symptoms were already excluded. However, it is still surprising that patients could develop spontaneous symptoms within 6 months of a negative ET. A striking 45 patients were taking β-blockers of whom 28 (62%) developed symptoms compared with only 11 (25%) not taking β-blockers. It is not stated whether the β-blocker was stopped before the ET and if so, for how long. The resting heart rate was 73 in those developing symptoms compared with 77 in those who were asymptomatic, but it remains possible that the blunting of heart rate response on ET was related to a residual β-blocker effect. The test started at 50 W and increased every 3 minutes by 50 W increments which is steep and may have made stopping for leg fatigue rather than central effects more likely, particularly if the patients were unfamiliar with bicycle exercise. The energy expended was only 6.2 metabolic equivalents (METs) in those developing symptoms and 6.9 METs in those who remained asymptomatic. Therefore, it seems likely that the ET was insufficient to stress the heart as a result of the β-blocker use. Further evidence for this is that the patient excluded during the study because he failed to report spontaneous symptoms did not have symptoms during the bicycle exercise. This patient subsequently died of heart failure secondary to AS.

Another question is what were the reasons for the β-blocker use, which was not stated. This could have been due to hypertension which was present in 58 (65%). However, if it was for coronary disease, then this might have introduced

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uncertainty in the natural history. β-Blockers used to be controversial antihypertensive agents in severe AS, but recent reviews suggest that they may be used safely according to international guidelines.[11-12] Follow-up fell far short of the maximum 36 months. Since 39 became symptomatic, there should have been 50 at the end if follow-up was complete, but only 7 had a final ET. This makes calculations of the predictive ability over this period relatively less secure.

This study suggests that β-blockade disguises the results of ET and that aggressive cycle exercise may not be appropriate for people with AS. Either a more gentle protocol or a treadmill test with 2 warm-up stages as conventionally recommended may be better. We agree with the authors that ET is underused, but ET alone might not be sufficient. The literature is now exploring the use of global longitudinal strain or midwall left ventricular fibrosis either by cardiac magnetic resonance imaging or indirectly reflected by left ventricular strain on electrocardiography as markers indicating a need for early surgery.[13] However, we suggest that the simple ET should be used initially to exclude "presymptomatic" patients who are not aware of the gradual onset of early symptoms. Exercise testing is cheap, widely available, and safe, and should be used routinely to complement history taking when assessing patients with severe AS at heart clinics.

ARTICLE INFORMATION

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