Can prolonged exercise-induced myocardial ischaemia be innocuous? reply

I read with interest the study by Nöel et al.1 aimed at evaluating the innocuousness of intense and prolonged exercise training above the threshold for myocardial ischaemia in patients with coronary artery disease. Following the training sessions, the authors evaluated cardiac troponin T and ventricular diameters and ejection fraction, which were found not significantly different from baseline (data are not reported). The authors conclude that ‘prolonged and repeated ischemic training sessions up to 60 min can be well tolerated without evidence of myocardial injury, significant arrhythmias or left ventricular dysfunction’. However, systolic function evaluation is not the most sensitive method to definitely exclude myocardial damage. Maximal exercise in patients with coronary disease has been shown to cause sustained diastolic dysfunction2 in the presence of unchanged systolic function. Considering that the evaluation of left ventricular response was the most important target of Nöel’s study, the adoption of more accurate tests in order to objectively exclude progressive left ventricular deterioration in their patients would had been advisable. Among the others, evaluation of natriuretic peptides and diastolic function index was determined by Doppler techniques or radionuclide ventriculography.3 Despite the study patients did not show significant changes in ejection fraction at follow-up, we cannot exclude that they had developed minor degrees of ventricular dysfunction. In fact, myocardial function and metabolism often remain abnormal for as long as 1 week after short periods of ischaemia and brief repetitive bouts of ischaemia may have a cumulative effect and cause myocardial necrosis.4 Considering the context, I would prefer to see further studies before considering prolonged exercise-induced myocardial ischaemia as ‘innocuous’.

References

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We appreciate Dr Fragasso’s interest in our recent work.1 Fragasso et al.2 showed in patients with coronary disease a lowered left ventricular filling rate 2 days after exercise that was no longer significantly different from baseline at 7 days. Whether this is sufficient to support the notion that exercise-induced myocardial ischaemia in patients with stable coronary artery disease causes sustained clinically significant diastolic dysfunction cannot be certain. Importantly, the 15 patients in Dr Fragasso’s study had severe coronary disease as evidenced by their poor exercise capacity (70 ± 30 W) that was less than half that of our patients (152 ± 56 W), their development of myocardial ischaemia at 217 ± 161 s of exercise compared with 442 ± 85 s for our patients and the lower rate-pressure product attained by their patients compared with ours (22 697 ± 5315 vs. 27 308 ± 7445 b.p.m. mmHg).

During a structured exercise training program above their myocardial ischaemic threshold, throughout serial evaluations, our patients had no troponin rises or significant arrhythmias and unchanged left ventricular systolic function. Nor did we observe any alteration of VO2max, a physiological variable closely related to cardiac function and most powerful predictor of mortality and morbidity.3 Because of the need for brevity, we did not report the spectral tissue Doppler echocardiography E/e’ ratio that was within normal range in the experimental group (15 ± 6). This ratio, derived from the septal annulus velocity, is known to have similar...