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 have been advisable. Among the others, evaluation of natriuretic peptides and diastolic function indexes determination by Doppler techniques or radionuclide ventriculography. 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. Considering the context, I would prefer to see further studies before considering prolonged exercise-induced myocardial ischaemia as ‘innocuous’.

References

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Can prolonged exercise-induced myocardial ischaemia be innocuous? reply

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 caused 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\(^1\) 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

accuracy to B-type natriuretic peptide as a non-invasive surrogate for the diagnosis of diastolic function.4

Experimental models using brief intermittent coronary occlusion that show deleterious myocardial effects cannot necessarily be extrapolated to the human context of coronary disease and exercise-induced myocardial ischaemia.5 Nor is it characteristic of active patients with uncomplicated chronic stable angina and good ventricular systolic function who frequently have episodes of myocardial ischaemia (most often asymptomatic) in daily life, to evolve towards heart failure.6 Does this not in itself suggest that regular ischaemic episodes in such patients may not be deleterious? Notwithstanding our findings of the excellent tolerance and apparent innocuity of an ischaemic exercise program in our patients, we were careful to state the need for further studies in similar and other patient groups. We agree for the need for caution in this field but also believe that the door should not be shut on such explorations given the possible benefits of more intense exercise in patients with chronic stable ischaemic heart disease.

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