

Authors:

Philippe Meyer, MD
Thibaut Guiraud, MSc
Mathieu Gayda, PhD
Martin Juneau, MD
Laurent Bosquet, PhD
Anil Nigam, MD

Exercise

CASE REPORT

Affiliations:

From the Cardiac Rehabilitation and Prevention Center (Centre ÉPIC) (PM, TG, MG, MJ, AN), Montreal Heart Institute, University of Montreal, Montreal, Canada; and Department of Kinesiology (LB), University of Montreal, Montreal, Canada.

Correspondence:

All correspondence and requests for reprints should be addressed to Anil Nigam, MD, Montreal Heart Institute, 5000 Belanger Street, Montreal, QC, Canada H1T 1C8.

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High-Intensity Aerobic Interval Training in a Patient with Stable Angina Pectoris

ABSTRACT

Meyer P, Guiraud T, Gayda M, Juneau M, Bosquet L, Nigam A: High-intensity aerobic interval training in a patient with stable angina pectoris. *Am J Phys Med Rehabil* 2009;88:000–000.

Recently, high-intensity aerobic interval training was shown to be more effective than continuous moderate-intensity exercise for improving maximal aerobic capacity and endurance in patients with coronary heart disease. However, patients with exercise-induced ischemia were not included in those studies. We present the acute cardiopulmonary responses of a 67-yr-old man with stable angina pectoris during a 34-min session of high-intensity aerobic interval training. Exercise was well tolerated with neither significant arrhythmia nor elevation of cardiac troponin-T. We observed a complete disappearance of symptoms and signs of myocardial ischemia after 24 mins of exercise. This observation is similar to the warm-up angina phenomenon, an adaptation to myocardial ischemia that remains poorly understood. We conclude that high-intensity aerobic interval training is a promising mode of training for patients with stable coronary heart disease that should also be investigated further in patients with exercise-induced ischemia.

Key Words: Exercise Training, Prescription, Cardiac Rehabilitation, Coronary Disease, Myocardial Ischemia

High-intensity interval training (HIT) is a form of aerobic exercise consisting of alternating short periods of high- and low-intensity exercise that has been used for decades by competitive athletes for training purposes. Recent studies suggest that HIT is safe and more effective than traditional moderate-intensity continuous training for improving maximal aerobic capacity and endurance in patients with stable coronary heart disease.^{1,2} Interval training was incorporated into the latest recommendations on cardiac rehabilitation, at lower intensities yet, corresponding to 50%–80% of maximal exercise capacity.³ A significant proportion of patients with coronary heart disease have exercise-induced ischemia, either symptomatic or not, and many are managed noninvasively, notably by a comprehensive cardiac rehabilitation program. Previous reports suggest that training at high intensities may also be beneficial and safe in these patients,^{4–6} but, to our knowledge, the acute response to HIT in humans with stable exertional ischemia has not been described.

CASE DESCRIPTION

As part of an ongoing study investigating the acute cardiopulmonary responses and safety of HIT in coronary heart disease patients,⁷ we recently evaluated a 67-yr-old man, presenting with Canadian Cardiovascular Society (CCS) class I angina, who was recruited by poster announcement in our rehabilitation center. The study protocol was approved by the Ethics Committee of the Montreal Heart Institute, and the patient gave written informed consent for his participation in the study and publication of his data. He was the victim of a myocardial infarction in 1986, which was initially treated medically, followed by angioplasty of the right coronary artery as a result of severe angina 12 yrs later. At that time, a chronic total occlusion of the circumflex artery was identified, and left ventricular ejection fraction on angiography was found to be 70%. Since then, the patient has taken part in a phase III rehabilitation program at our center, including on average three sessions of moderate-intensity continuous training per week. His current medication included aspirin, statin therapy, an angiotensin-converting enzyme inhibitor, and atenolol 75 mg once daily.

A baseline maximal exercise stress test was performed on a bicycle ergometer (Ergoline 800S, Bitz, Germany) with an initial workload at 60 W, which was increased by 15 W/min until exhaustion. The power of the last completed stage was considered as the maximal aerobic workload. Oxygen uptake ($\dot{V}O_2$) was determined continuously on a breath-by-breath basis using an automated cardiopulmonary exercise system (Oxycon Alpha, Jaeg-

ger, Germany). $\dot{V}O_{2max}$ was measured at 28.5 ml/min/kg or 8.1 metabolic equivalents (METs) with a maximal heart rate of 118 bpm and a maximal aerobic workload of 165 W. The electrical ischemic threshold (>1-mm ST-segment depression 80 msec after the J point) was reached at 105 bpm (89% of maximal heart rate) and a $\dot{V}O_2$ of 22.7 ml/min/kg (80% of $\dot{V}O_{2max}$) or 6.5 METs (Fig. 1). One week after baseline exercise testing, the patient performed a 34-min HIT session, consisting of 15 secs of exercise at an intensity of 100% of maximal aerobic workload, alternating with 15 secs of passive rest. Time spent during the baseline ischemic threshold was determined by summing each 5-sec block of $\dot{V}O_2$ mean values over 22.7 ml/kg/min.⁸ The patient's resting and exercise electrocardiograms (ECGs) during the HIT session are displayed in Figure 2. The resting ECG was unremarkable, showing normal sinus rhythm and absence of Q waves or repolarization abnormalities. At 4 mins of exercise, the patient noted the onset of mild angina with evidence of borderline horizontal ST-depression in lead V6. Angina increased slightly until 9 mins, where it peaked at a perceived grade of 2 of 10 with 1.5-mm horizontal or downsloping ST-depression in leads V5 and V6. Symptoms then declined progressively and disappeared completely at 24 mins. ST-segment depression was <1 mm at 24 mins and showed complete normalization at 34 mins (end of HIT protocol). Total exercise time spent during the baseline ischemic threshold was 1565 secs or 26 mins and 5 secs, but the time spent with effective symptoms and signs of myocardial ischemia during the training session was only 20 mins. Importantly, the

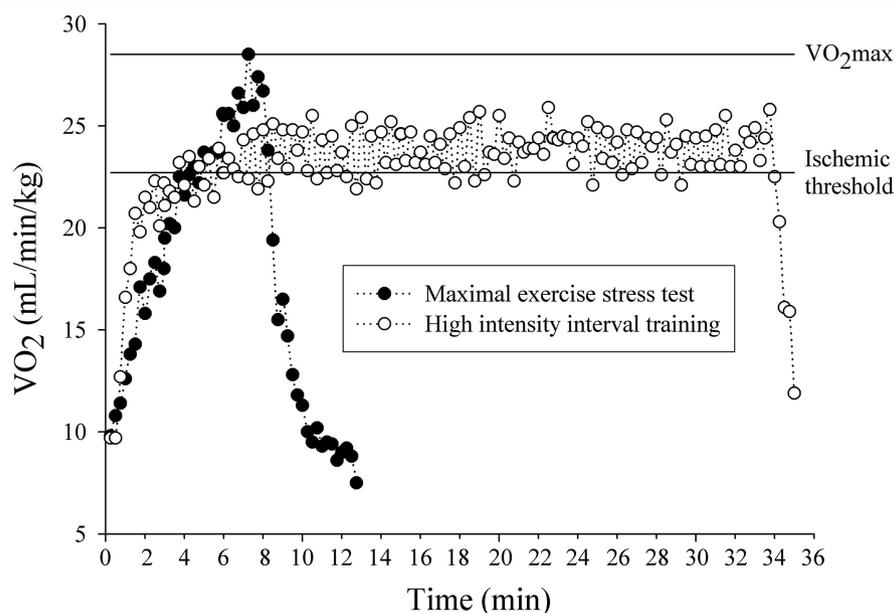


FIGURE 1 Oxygen uptake (averaged every 15 secs) during maximal exercise stress test and high-intensity interval training.

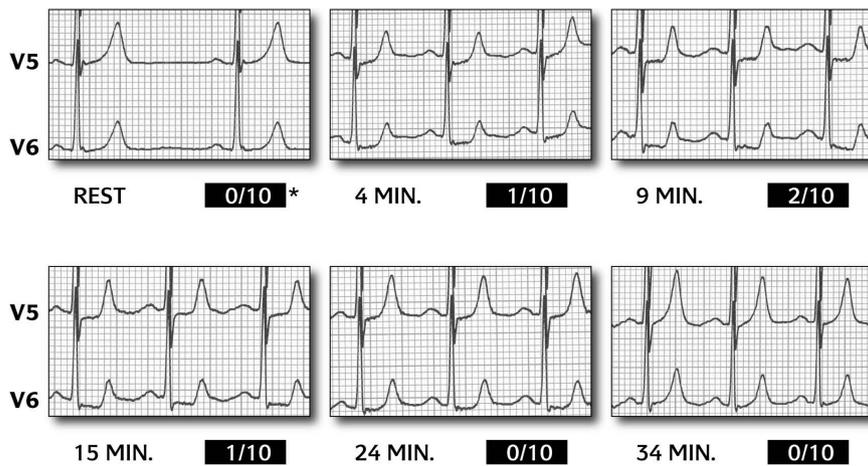


FIGURE 2 ECGs (leads V5 and V6) and perceived angina during a 34-min session of high-intensity interval training. *Rating scale of perceived angina (0–10).

patient experienced no significant arrhythmias or elevation of cardiac troponin-T measured 24 hrs after the HIT session. All tests were conducted in our rehabilitation center by an experienced exercise physiologist and a nurse, and they were supervised by a cardiologist with resources for cardiac resuscitation in immediate proximity to the patient. Patients were monitored using continuous ECG and blood pressure measurements at 2-min intervals.

DISCUSSION

To the best of our knowledge, this is the first report of the acute cardiopulmonary responses to HIT in a patient with chronic exertional angina. This case highlights the fact that HIT can be well tolerated in certain patients with stable angina, allowing for a good control of anginal symptoms and permitting a longer training session than would be possible with a conventional continuous aerobic exercise session.

After 9 mins of exercise, clinical and electrical signs of ischemia declined, with complete resolution of myocardial ischemia from 24 mins until the end of the training session. This observation is similar to the warm-up angina phenomenon, which has been demonstrated in numerous studies, and refers to a significant decrease in ECG signs of myocardial ischemia on the second of the two consecutive exercise tests performed within a short time interval.^{9,10} The intensity of the first exercise test and a short delay between both exercise tests seem to be crucial triggering factors.^{11,12} This may explain why successive exercise phases at high intensities interspersed with short rest phases, as in HIT, may optimally induce an adaptation to myocardial ischemia. The warm-up angina phenomenon remains poorly understood, but several potential underlying mechanisms have been proposed: (1) an improvement in oxygen supply by collateral recruitment or myocardial perfusion redistri-

bution; (2) a reduction in regional myocardial oxygen consumption; and (3) a form of myocardial adaptation to ischemia, which would seem to involve different pathways than ischemic preconditioning.¹³ Based on recent data, it may be postulated that HIT induces an increase in coronary flow by improving endothelial function.^{14,15} With regard to the long-term effects of this mode of training, intermittent ischemia induced by exercise promoted coronary collateral formation in animal models.¹⁶

According to the current guidelines, patients with exercise-induced ischemia should train at a heart rate corresponding to 10 bpm below the clinical or electrical ischemic threshold.¹⁷ However, continuous prolonged training above the ischemic threshold was recently shown to be well tolerated without the evidence of myocardial damage, significant arrhythmias, or left ventricular dysfunction.^{5,6} In this case, HIT was well tolerated allowing for a long exercise session duration at high aerobic intensity, with complete disappearance of symptoms and signs of myocardial ischemia. Further research is needed to demonstrate the safety and potential benefits of this mode of exercise training in active patients with coronary heart disease with chronic exertional angina.

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