Can prolonged exercise-induced myocardial ischaemia be innocuous?

Martin Noël, Jean Jobin, Audrey Marcoux, Paul Poirier, Gilles R. Dagenais, and Peter Bogaty*

Quebec Heart Institute, Laval Hospital, Laval University, 2725 Chemin Ste-Foy, Ste-Foy, Quebec, Canada G1V 4G5

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Aims To evaluate the innocuousness of intense and prolonged exercise training above the threshold for myocardial ischaemia (1 mm ST-segment depression).

Methods and results Twenty-two patients with ischaemic heart disease (IHD) were randomized to exercise training either at a target intensity that induced myocardial ischaemia (ischaemic group) or that adhered to current guidelines (control group). Training was progressively increased to 60 min under continuous electrocardiographic (ECG) monitoring. Cardiac troponin T (cTnT) was measured at various intervals. Ambulatory ECG monitoring was performed before and after 6 weeks of training and left ventricular function was evaluated in the ischaemic group after at least 6 weeks of training. The ischaemic group had myocardial ischaemia during the first 20, 40, and 60 min exercise sessions for 12.3 \pm 6.8, 29.0 \pm 12.9, and 49.8 \pm 2.2 min, respectively, with ST-segment depression ranging from 1.0 to 2.1 mm. No patient in either group demonstrated significant arrhythmias or increased cTnT. The ischaemic group had preserved left ventricular function.

Conclusion In patients with IHD, prolonged and repeated ischaemic training sessions up to 60 min can be well tolerated without evidence of myocardial injury, significant arrhythmias, or left ventricular dysfunction.

Introduction

It is generally accepted that exercise training intensity in patients with ischaemic heart disease (IHD) should correspond to a heart rate that remains 10 b.p.m. below the threshold for myocardial ischaemia (1 mm ST-segment depression). This recommendation is enshrined in current guidelines of exercise prescription1–4 and acknowledged in textbooks of cardiology.5,6

A limitation of these guidelines is the restriction of exercise training intensity, especially in patients who may manifest myocardial ischaemia at a relatively low level of exercise intensity. Such patients may be deprived of the potential benefits of more intense exercise.7 Higher compared with lower exercise training intensity decreases chronic heart disease risk factors,8 improves insulin sensitivity,9 increases cardiopulmonary fitness to a greater degree,8,10 and imparts superior cardioprotective benefits.7

In a study over 25 years ago, high intensity exercise training in patients who developed exercise-induced myocardial ischaemia was associated with increases in maximal oxygen uptake (\( \dot{V}O_2 \) peak) and myocardial oxygen consumption (\( \dot{MVO}_2 \)).11 At about the same time, studies also suggested that exercise training could raise the heart rate at which electrocardiographic (ECG) ischaemia occurred and improve myocardial perfusion.12,13 Although these findings appeared promising in their implications for cardiopulmonary rehabilitation, myocardial ischaemia was not monitored during this high-intensity exercise.

More recently, in a study of the warm-up angina phenomenon, we found that a first exercise was needed to induce myocardial ischaemia of more than moderate intensity in order to significantly attenuate myocardial ischaemia of a second exercise that closely followed the first.14 Indeed, exercise may also have a delayed attenuating effect on myocardial ischaemia in patients with stable angina.15 Thus, previous studies provide an appropriate rationale to evaluate the potential benefit of prolonged and repeated periods of exercise-induced myocardial ischaemia as part of a training programme in subjects with coronary artery disease.

However, safety had not been specifically addressed in these older high intensity exercise-training studies.11–13 Before evaluating the physiological effects of a prolonged more intensive exercise training protocol that would induce myocardial ischaemia in patients with stable IHD, the innocuousness of repeated ischaemic exercise needed to be assessed in terms of myocardial injury, arrhythmia, and effect on left ventricular function.
Methods
Twenty-two patients with stable IHD documented by (i) a recent (≥6 months) standard Bruce protocol treadmill test that was positive for myocardial ischaemia (≥1 mm horizontal or downsloping ST-segment depression) and (ii) coronary angiography (≥70% arterial diameter narrowing of at least one major coronary artery) and/or stress myocardial scintigraphic imaging (significant reversible perfusion defect) were recruited for this trial. Patients were screened from summaries of outpatient cardiology visits and consecutive reports from the hospital exercise testing laboratory. Patients were excluded if they had significant resting ECG abnormalities such as voltage criteria for left ventricular hypertrophy or resting ST-segment depression >0.5 mm. The other main exclusion criteria were recent acute coronary disease, significant arrhythmias, significant heart valve disease, heart failure, uncontrolled hypertension, locomotion disability, and digoxin medication. The hospital Ethics Review Board approved the study and an independent safety committee was mandated to monitor it. All patients gave informed written consent.

Exercise testing
Before initiation of the study, the 35 potentially eligible patients with a previous positive Bruce treadmill exercise test completed an individualized ergocycle symptom-limited exercise test during which \( V_{\text{O}}_2 \text{peak} \) was evaluated as previously described.\(^6\) \( MV_{\text{O}}_2 \) was estimated by the rate-pressure product (heart rate x systolic blood pressure) (RPP) at peak exercise. Oxygen pulse, a value dependent on stroke volume and the difference between the arterial and mixed venous blood \( O_2 \) content was calculated by dividing \( V_{\text{O}}_2 \) by the simultaneously measured heart rate and was expressed as mL \( O_2 \)/beat.\(^7\) One patient declined participation after initial testing, 12 patients were excluded because either myocardial ischaemia was absent on the ergocycle test (nine patients) or occurred too close to the end of exercise (>90% of maximal workload; three patients). The latter two exclusion criteria were deemed necessary to ensure that ischaemic exercise would be feasible and tolerable. This exercise test was repeated after the first three sessions of 60 min of continuous endurance exercise training that corresponded to 6 weeks of training.

Exercise programme
The 22 patients of the study exercised three times per week under the continuous supervision of at least one of the two study exercise specialists. The exercise sessions consisted of a brief warm-up period of light intensity (5-10 min) followed by endurance exercise (20–60 min) on a stationary ergocycle (Monark 878, Vansbro, Sweden) under continuous ECG monitoring (discussed subsequently). After the endurance portion of the session, strength training (15 min) of the major muscle groups was individually prescribed and followed by a cool-down phase (10 min) that included flexibility exercises. Over a period of 6 weeks, the endurance exercise portion was progressively increased from 20 to 60 min and then maintained for the remainder of the training programme, according to each patient’s response and tolerance as per protocol design.

Endurance exercise portions of >40 min were separated by walks of moderate intensity (5–10 min) in which patients were momentarily not monitored. This physically active pause favoured patients’ comfort that would have been compromised by too lengthy uninterrupted cycling activity. The total exercise sessions lasted between 50 and 95 min.

Patients were randomly divided into two groups that differed in the intensity of the endurance exercise. The randomization sequence was furnished by the research center statistician. The control group exercised at an intensity that is currently recommended by the guidelines (10 b.p.m. below the heart rate at which these patients had 1 mm ST-segment depression on the ergocycle exercise test).\(^3\) The ischaemic group trained at an intensity that induced at least 1 mm but no more than 3 mm of ST-segment depression, provided that there were at most tolerable symptoms of myocardial ischaemia (‘ischaemic zone’). In order to target and remain within this ischaemic zone during endurance exercise, the ECG was continuously monitored and recorded in all patients during all exercise sessions using the MP 150 telemetry system with the AcqKnowledge Software interface version 3.5.7 (Biopac Systems, Santa Barbara, CA, USA). This monitoring system allowed us to dynamically adjust exercise intensity in such a way that the patients in the ischaemic group were constantly kept in their ischaemic zone, whereas patients in the control group were kept outside it. The ECG lead that had shown the greatest ST-segment depression on the ergocycle exercise test performed before initiation of the study was used for continuous monitoring during the endurance exercise portions. Blood pressure was manually measured using a sphygmomanometer (Tycos 767, San Diego, CA, USA) half way and at the end of each endurance exercise portion.

Marker of myocardial injury (troponin T)
Cardiac isoform troponin T (cTnT) was measured 18–24 h after the first 20, 40, and 60 min of continuous endurance exercise which corresponded to the first, 12th, and 21st sessions. cTnT was again measured after the first 3 sessions that included 60 min of continuous endurance exercise. Measurements were performed in the hospital clinical laboratory using the only available commercial assay (Roche Inc., Mannheim, Germany). The decision limit for myocardial injury was set at 0.1 μg/L.\(^18\)

Arrhythmias and ST-segment depression
ECG telemetry was used to monitor for arrhythmias and to characterize the ST-segment depression pattern of the endurance exercise portion of each training session. Figure 1 illustrates the endurance exercise portion of one subject. Each dot represents a quantitative ST-segment depression of one beat in time compared with the average ST-segment measured just before the start of endurance exercise while the patient was sitting on the ergocycle. A mean regression line was calculated to determine the ischaemic time, i.e. the amount of time in which the ST-segment depression was ≥1 mm, and the average ST-segment depression during that time. The gap of missing data represents the active pause taken by the patient in which the ECG was briefly not recorded.

Ambulatory electrocardiographic monitoring
In order to determine the incidence of daily arrhythmias that could be related to an ischaemic training programme, ambulatory 24 h ECG monitoring (Holter) was performed before initiation of the study and following the first three sessions that included 60 min of endurance exercise. The ambulatory recordings were acquired

Figure 1 Example of a computerized analysis of the ST-segment depression (in mV) for an entire duration of a specific 60 min endurance exercise period of one patient.
using a Marquette monitoring system (Marquette Electronics Inc., Milwaukee, WI, USA) and categorized into three periods: (i) 24 h, (ii) daytime (8 a.m. to 6 p.m.), (iii) night-time (midnight to 6 a.m.). The recordings were divided into diurnal and nocturnal periods because daytime activities often included exercising, which may predispose to arrhythmias. An experienced technician blinded to randomization edited the recordings. The analysis of arrhythmias was computer-assisted (HRV Marquette Electronics Inc.) and visually double-checked. Ectopic ventricular and supraventricular beats were classified as isolated premature contractions, bigeminy, and salvos. Premature ventricular contractions were subdivided into episodes of $\geq 10$, 1–9, and 0 b.p.h. Ventricular salvos were also subdivided into episodes of $\leq 5$ beats, 6–10 beats, $>10$ beats and characterized with corresponding heart rate.

Ventricular function

After at least 6 weeks of exercise training, all patients in the ischaemic group underwent transthoracic echocardiographic evaluation by a cardiologist unaware of study details. Left ventricular systolic and diastolic diameters were measured and left ventricular ejection fraction was calculated.19

Statistical analysis

We estimated from our own and previous work that a sample size of 11 patients in the ischaemic exercise group and 11 patients in the control group had 90% power (alpha error of 0.05) for detecting a 20% increase in ischaemic threshold following an ischaemic training programme in the ischaemic group and assuming no change in ischaemic threshold in the control group.11,14 We anticipated a standard deviation (SD) of 20–25% for the ischaemic thresholds.11,14 Data are expressed as mean $\pm$ SD unless otherwise stated. Comparisons of patient data between visits and between patient groups were performed using two-way repeated ANOVA with group and time as fixed factors. Group-specific variances were investigated and all variables suggested a compound symmetry covariance structure based on the likelihood ratios test as well as on Akaike’s and Schwarz’s criteria. The Holter parameters were log transformed to stabilize variances. The univariate normality assumptions were verified with the Shapiro–Wilk test and multivariate normality was verified with the Mardia test. The results were considered significant with $P < 0.05$. All analyses were performed with the statistical package SAS, version 9.1.3 (SAS Institute Inc., Cary, NC, USA).

Results

Twenty men and two women, aged 49–73, participated and completed the study. The histories and clinical characteristics of the patients are summarized in Table 1. Most patients had previously undergone surgical and/or percutaneous revascularization. Although half the patients had a previous myocardial infarction, all of them had a left ventricular ejection fraction greater than 50%. Nine patients experienced anginal symptoms on a regular basis (at least once a week), whereas the others were generally asymptomatic on medical therapy. Patients were treated with standard medications that had neither been recently modified before initiation of the study nor changed during the study. Patients in each group were slightly overweight by body mass index criteria.20 There were no significant imbalances in clinical characteristics in the ischaemic and control groups.

Exercise training

The exercise training programme was well tolerated by all patients. As per study protocol, all patients in the ischaemic training arm showed exercise-induced ECG myocardial ischaemia and four patients regularly reported angina during the training sessions. No session was discontinued because of angina. For the ischaemic exercise group, the mean times spent in the ischaemic zone during the first 20, 40 and 60 min of continuous endurance exercise which also preceded cTnT measurements were 12.3 $\pm$ 2.2 min, respectively ($P = 0.04$), 40 min ($16 329 \pm 5407$ vs. $12 452 \pm 2330$ b.p.m. mmHg, respectively; $P = 0.04$), and 60 min training sessions (18 750 $\pm$ 5 698 vs. $13 352 \pm 2947$ b.p.m. mmHg, respectively; $P = 0.02$) (Figure 2). This difference appeared to be driven by the heart rate that was lower in the control group because systolic blood pressure was comparable in both groups (Table 2).

Arrhythmias and ST-segment depression

Mean ST-segment depression ranged from 1.0 to 2.1 mm during the first 20, 40, and 60 min sessions in the ischaemic

<table>
<thead>
<tr>
<th>Table 1 Clinical characteristics of the study subjects</th>
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<tbody>
<tr>
<td>Controls (n = 11)</td>
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<tr>
<td><strong>Anthropometric data</strong></td>
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<tr>
<td><strong>Men</strong></td>
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<tr>
<td><strong>Age (years)</strong></td>
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<tr>
<td><strong>Weight (kg)</strong></td>
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<tr>
<td><strong>Body mass index (kg/m²)</strong></td>
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<tr>
<td><strong>Clinical history</strong></td>
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<tr>
<td><strong>Diabetes/impaired fasting glucose</strong></td>
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<td><strong>Arterial hypertension</strong></td>
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<tr>
<td><strong>Dyslipidaemia</strong></td>
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<tr>
<td><strong>Cardiac history</strong></td>
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<tr>
<td><strong>Previous myocardial infarction</strong></td>
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<tr>
<td><strong>Left ventricular ejection fraction (%)</strong></td>
</tr>
<tr>
<td><strong>Percutaneous coronary intervention</strong></td>
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<tr>
<td><strong>Coronary bypass surgery</strong></td>
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<tr>
<td><strong>Drug therapy</strong></td>
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<tr>
<td><strong>Aspirin</strong></td>
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<td><strong>Lipid-lowering agent</strong></td>
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<td><strong>Beta-blocker</strong></td>
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<td><strong>Nitrate</strong></td>
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<td><strong>ACE-inhibitor</strong></td>
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<tr>
<td><strong>Calcium antagonist</strong></td>
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<tr>
<td><strong>Diuretic</strong></td>
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<td><strong>Oral hypoglycaemic</strong></td>
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Mean $\pm$ SD (%). ACE, angiotensin-converting-enzyme.
In the ischaemic group, tolerable angina was experienced by one patient during the 20 min session, by three patients in the 40 min, and by three patients in the 60 min training session. Only occasional isolated premature ventricular contractions were observed during exercise and to a similar degree in both groups.

Marker of myocardial injury (troponin T)

None of the patients in either group at any time had a positive elevation of cTnT. All measurements of cTnT remained below the detection limit of the assay (<0.01 μg/L).

Ambulatory electrocardiographic monitoring

Twenty-four-hour Holter monitoring analysis showed no statistically significant difference in the occurrence of supraventricular (Table 3) and ventricular arrhythmias (Table 4) before and after three sessions of 60 min of endurance training were completed, which corresponded to 6 weeks of training. Nor were there any significant differences when the data were further examined in diurnal and nocturnal periods. One four-beat ventricular salve at a rate of 92 b.p.m. occurred at night following a non-exercising day in one patient in the ischaemic group at the 6 weeks’ analysis.

Ventricular function

Following at least 6 weeks of training, all patients in the ischaemic group had a resting left ventricular ejection fraction that was within normal limits (67 ± 7%) and not significantly changed compared with the pre-exercise training value (Table 1). Diastolic and systolic left ventricular dimensions remained within normal limits (51 ± 5 and 29 ± 5 mm, respectively).

Exercise tests

The ergocycle ECG test, performed after 6 weeks of training, remained positive for myocardial ischaemia (≥1 mm ST-segment depression) in all patients. The same nine patients (36%) experienced angina during both exercise tests. The maximal workload (watts) achieved increased in both groups but maximum RPP did not increase (Table 5). There were no significant differences in either group between VO₂ and O₂ pulse at baseline and at 6 weeks (Table 5).

Discussion

To our knowledge, this is the first reported study demonstrating that, in patients with stable IHD, prolonged and repeated exercise, which induces myocardial ischaemia under controlled conditions, is not deleterious and can be well tolerated. Progressively introduced, repeated and prolonged ischaemic exercise was not associated with malignant or significant arrhythmias either while exercising or during Holter monitoring, did not cause myocardial injury.
as evaluated by cTnT levels, and did not result in sustained left ventricular systolic dysfunction or change in left ventricular systolic and diastolic dimensions.

The guidelines on exercise prescription in patients with IHD recommend that training intensity should stay below the threshold for myocardial ischaemia. To the best of our knowledge, this important recommendation appears essentially based on a single study performed over a decade ago by Hoberg et al., which suggested that ischaemic exercise confers an arrhythmic risk. In this latter study, patients with coronary artery disease exercised on an ergocycle for 10 min twice a week at 75% of maximal heart rate for 12 months. Twenty-four-hour Holter monitoring was performed on the first day of training and repeated on a training-free day. The authors identified 10 ischaemic episodes that were associated with ventricular arrhythmias in five patients. These arrhythmias consisted of increased premature ventricular contractions, three ventricular couplets, and one episode of non-sustained ventricular flutter. The significance of these results is unclear given the benign or uncertain prognostic nature of these arrhythmias, the controversial relationship between exercise-induced myocardial ischaemia and ventricular arrhythmias, the absence of a baseline recording of arrhythmic status, and the limited arrhythmic sampling period. Importantly, the conclusions of this and previous studies may not be applicable to ischaemic exercise experienced by the patients in our study because these previous studies based their findings on maximal symptom-limited exercise testing, whereas our patients were enrolled in a structured and progressive training programme. Indeed, in the study by Hoberg et al., patients reached their prescribed training heart rate within 2 min, which corresponded to 75% of maximal heart rate. They abruptly started and ended a 10 min training session, whereas in our study, patients began exercising with 5–10 min of warm-up before starting endurance ischaemic exercise. Warm-up exercise improves endurance performance and favours a better adaptation to metabolic demand, particularly in patients with coronary artery disease. Warm-up exercise

### Table 3: Supraventricular arrhythmias during 24 h Holter monitoring

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<th>Control group</th>
<th>Ischaemic group</th>
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<tr>
<td></td>
<td>Baseline</td>
<td>6 weeks</td>
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<tr>
<td>Premature contractions</td>
<td>18 (4–45)</td>
<td>17 (4–71)</td>
</tr>
<tr>
<td>Bigeminy</td>
<td>1 (0–3)</td>
<td>0 (0–2)</td>
</tr>
<tr>
<td>Salves</td>
<td>1 (0–3)</td>
<td>1 (0–3)</td>
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Data are expressed as median (inter-quartile range 25–75%).

### Table 4: Ventricular arrhythmias during 24 h Holter monitoring

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<th>Control group</th>
<th>Ischaemic group</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>6 weeks</td>
</tr>
<tr>
<td>Premature contractions</td>
<td>4 (1–17)</td>
<td>6 (3–19)</td>
</tr>
<tr>
<td>Episodes ≥10 b.p.h.</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Episodes 0 b.p.h.</td>
<td>20 (12–23)</td>
<td>20 (12–23)</td>
</tr>
<tr>
<td>Bigeminy</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Salves</td>
<td>0</td>
<td>0</td>
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Data are expressed as median (inter-quartile range 25–75%).

**a**One four-beat ventricular salve at a rate of 92 b.p.m. occurred at night following a non-exercising day in one patient.

### Table 5: Exercise test parameters at baseline and after 6 weeks of exercise training

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>Ischaemic Group</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>6 weeks</td>
</tr>
<tr>
<td>VO₂ (mL O₂/kg/min)</td>
<td>19.1 ± 5.1</td>
<td>19.6 ± 5.1</td>
</tr>
<tr>
<td>O₂ pulse (mL/beat)</td>
<td>12.8 ± 4.6</td>
<td>12.9 ± 4.2</td>
</tr>
<tr>
<td>Workload (W)</td>
<td>127 ± 49</td>
<td>138 ± 50*</td>
</tr>
<tr>
<td>Heart rate (b.p.m.)</td>
<td>124 ± 22</td>
<td>125 ± 26</td>
</tr>
<tr>
<td>RPP (b.p.m. mmHg)</td>
<td>25 167 ± 6 263</td>
<td>25 404 ± 6 962</td>
</tr>
</tbody>
</table>

Values are mean ± SD. RPP, rate pressure product (heart rate × systolic blood pressure).

*P < 0.05 between visits.
also attenuates myocardial ischaemia and could well be protective against deleterious arrhythmias. We performed extensive baseline and training arrhythmia monitoring representing 308 patient-hours that included 154 patient-hours of ischaemic training compared with only 14 patient-hours in the study by Hoberg et al. We found no differences in the occurrence of arrhythmias between the control and ischaemic training groups and we observed no malignant arrhythmias during near-continuous monitoring during all training sessions in all subjects as well as during 24 h Holter monitoring. Our results suggest that progressive exposure to repeated ischaemic exercise periods is not pro-arrhythmic.

We measured serum cTnT to evaluate whether repetitive exposure to exercise-induced myocardial ischaemia leads to myocardial injury. Serum cTnT is considered sufficiently sensitive to detect even microscopic myocardial necrosis and sufficiently specific so as not to be confounded by skeletal muscle injury that can occur at higher exercise intensity. We found no evidence of myocardial injury in these patients exposed to repeated bouts of ischaemic exercise. Serum cTnT was not only negative in all patients at all times, but also remained consistently below the detection limit (<0.01 µg/L) of the assay for all measurements.

Limitations
Although these findings are relatively robust and detailed, they must be considered within the context of a carefully selected and limited cohort of stable motivated patients with preserved left ventricular function, training in a controlled environment. Although we did not find sustained left ventricular dysfunction with ischaemic exercise, we cannot rule out transient ischaemia-induced left ventricular dysfunction because we did not study left ventricular function immediately after each ischaemic exercise. However, this appears unlikely since VO2peak and O2 pulse, both physiological variables closely related to cardiac function, were comparable between groups at all times and did not decrease in the ischaemic group after 6 weeks of ischaemic exercise. More study of ischaemic exercise is needed to fully evaluate its physiological and possible cardioprotective effects compared with traditional training. It would be premature to generalize these results to a broader population of patients with IHD.

Conclusions
In controlled conditions, ischaemic exercise training does not cause myocardial injury, does not appear arrhythmogenic, and does not result in sustained left ventricular dysfunction. This study encouragingly suggests that ischaemic exercise training can be safely investigated in an appropriate environment to determine whether it may confer greater cardioprotective and physiological benefits than more standard less-intensive exercise programmes in patients with coronary artery disease.

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Conflict of interest: none declared.

References


Clinical vignette

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Unruptured congenital aneurysms of the right and left sinuses of Valsalva

Konstantinos Zannis1*, Boyan Tzvetkov1, Jean-François Deux2, and Ernest Wilhelheim Matthias Kirsch1

1Department of Cardiothoracic Surgery, Henri Mondor Hospital, 51 avenue du Maréchal de Tassigny, 94000 Creteil Cedex, France and 2Department of Medical Imaging, Henri Mondor Hospital, 51 avenue du Maréchal de Tassigny, 94000 Creteil Cedex, France

* Corresponding author. Tel: +33149812172; fax: +3349812152. E-mail address: konstantinoszannis@gmail.com

Congenital unruptured aneurysms affecting both the right and left sinuses of Valsalva are extremely uncommon. We report the case of a 24-year-old African male admitted to our institution with a 1-year history of chest pain, palpitations, and progressive exertional intolerance. During the past 10 days, he had experienced an acute exacerbation of these symptoms. The chest radiograph delineated an abnormal cardiac thoracic ratio. Electrocardiogram showed no ischaemic changes and a first degree atrioventricular block associated with a complete right bundle branch block and an incomplete left bundle branch block. Transthoracic echocardiography showed two large aneurysms of the left and the right coronary sinuses of Valsalva. Transoesophageal echocardiography (TOE) was performed showing extension of the right sinus of Valsalva aneurysm into the interventricular septum and the left sinus of Valsalva aneurysm appearing as an extracardiac saccular protrusion (Panel A). A multi-slice computed tomography (CT) confirmed the diagnosis (Panel B). Considering the large size of the right and the extracardiac extension of the left coronary sinus aneurysms, operative repair was indicated. The operation was performed through median sternotomy under cardiopulmonary bypass (Panel C). Both aneurysms were repaired through the ascending aorta by closing their orifices with circular Dacron patches. The native aortic valve was preserved. Per-operative TOE confirmed complete aneurysm exclusion without increase in the known aortic regurgitation (Panel D). The patient was discharged from hospital on day 8 and is doing well 11 months after operation.

Panel A. TOE before surgical repair.
Panel B. Multi-slice CT-scan reconstruction.
Panel C. Intra-operative view of the aortic valve with the orifices of the right and left coronary sinus aneurysms.
Panel D. Post-operative TOE with left and right coronary sinuses aneurysms obstructed. LA, left atrium; RA, right atrium; L CSA, left coronary aneurysm; RCS A, right coronary aneurysm, Ao, aorta; LV, left ventricle, S, interventriculaire septum; RV, right ventricle.