Extreme mountain bike challenges may induce sub-clinical myocardial damage

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Aim. The relationship between extreme exercise and coronary artery disease is not well understood, and the information available is contradictory. The aim of the present study was to determine whether strenuous endurance exercise, performed under conditions in which the partial pressure of environmental oxygen varies constantly, can induce myocardial cell injury.

Methods. Plasma urea, creatinine, creatine kinase, myoglobin and cardiac troponin I (cTnl) concentrations were measured the day before and immediately after a mountain bike challenge (distance 95 km, cumulative altitude difference 2 340 m) in eleven amateur male cyclists.

Results. All biochemical markers of muscle cell damage increased significantly after exercise. Although cTnl concentrations also increased significantly, they remained below the threshold (0.5 μg/L) indicative of acute myocardial infarction.

Conclusions. In male, amateur mountain bikers, this kind of strenuous exercise may induce sub-clinical myocardial injury.

Key Words: Cardiac troponin I - Strenuous exercise - Physical endurance - Myocardial injury - Anoxia

Abnormalities in the plasma lipid profile, myocardial infarction, and sudden cardiac death, however, when highly demanding and prolonged exercise is performed, skeletal muscle damage also contributes to an increase in these markers. In the clinical setting, cardiac troponin T and cardiac troponin I (cTnl) provide highly specific (and commonly used) markers of myocardial cell injury. The latter may be the most specific marker for detecting cardiomyocyte necrosis, even when skeletal muscle damage is present. The aim of the present study was to determine whether acute, strenuous, endurance exercise, performed under conditions of continuous variation in the partial pressure of environmental
TABLE I.—Subject characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Age (y)</th>
<th>Weight (kg)</th>
<th>Height (m)</th>
<th>BMI (kg/m²)</th>
<th>Training (h/w)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SD)</td>
<td>34 (8)</td>
<td>74 (9.7)</td>
<td>1.75 (0.78)</td>
<td>24.1 (1.9)</td>
<td>11 (4)</td>
</tr>
<tr>
<td>Range (min-max)</td>
<td>25-46</td>
<td>62-93</td>
<td>1.63-1.87</td>
<td>20.4-27.3</td>
<td>4-18</td>
</tr>
</tbody>
</table>

h/w: hours per week; BMI: body mass index; Min-Max: minimum-maximum.

oxygen (an additional stressor), induces myocardial cell injury, as shown by plasma cTnI concentrations.

Materials and methods

Subjects

The study subjects were eleven healthy, male, amateur mountain bikers. Table I shows the characteristics of these subjects. All volunteered to participate in the Sierra Nevada Limite 2003 race, a highly demanding mountain bike challenge in which the entrants have to complete a distance of 95 km over a highly irregular geographical profile (Figure 1). The cumulative altitude difference over the course is 2 430 m. Their training program involved about 11 h per week. All subjects were informed of the nature and purpose of the study and all gave their written, informed consent to be included. A medical examination was performed the day before the race. All subjects were free of disease and took no medication at the time of the study. None had a history of cardiovascular disease. A questionnaire was administered to obtain demographic, training, dietary and cardiovascular risk factor data. The study was approved by the Review Committee for Research Involving Human Subjects of the University of Granada.

Blood samples

Blood was collected from a vein in the forearm the day before the race (following an overnight fast) and again immediately after the competition. Plasma concentrations of cTnI, myoglobin, creatine kinase (CK), urea and creatinine were measured at Granada University Hospital within 1 h of collection. Cardiac troponin I and myoglobin were determined in a two-step sandwich microparticle enzyme-immunoassay using a Beckman analyzer (Fullerton, USA). The upper reference limits used by this hospital for cTnI and myoglobin are 0.5 μg/L and 70 μg/L, respectively. Serum CK levels (reference range 40-200 U/L), urea (reference range 10-50 mg/dL) and creatinine (reference range 0.6-1.3 mg/dL) were determined by the Synchron LX system using a Beckman analyzer (Fullerton, USA).

To account for the fluid shifts known to occur with exercise and dehydration, all values were corrected for changes in plasma volume.16

Statistical analysis

The results are presented as mean (standard deviation and range). The Shapiro-Wilk test was used to examine the normality of their distribution. Since they were not normally distributed (P<0.1), changes in biochemical marker levels were compared using the non-parametric Wilcoxon test. The α error was set at 0.05.
### Table II.—Biochemical markers analysed before and after the race.

<table>
<thead>
<tr>
<th></th>
<th>Prerace</th>
<th>Postrace</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea (mg/dL)</td>
<td>32.82 (21%)</td>
<td>39.01 (15%)</td>
<td>0.033</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>0.95 (15%)</td>
<td>1.22 (16%)</td>
<td>0.005</td>
</tr>
<tr>
<td>CK (U/L)</td>
<td>136.5 (44%)</td>
<td>200.6 (27%)</td>
<td>0.022</td>
</tr>
<tr>
<td>Myoglobin (μg/L)</td>
<td>38 (53%)</td>
<td>113.45 (31%)</td>
<td>0.003</td>
</tr>
<tr>
<td>cTnI (μg/L)</td>
<td>0.02 (50%)</td>
<td>0.07 (157%)</td>
<td>0.016</td>
</tr>
</tbody>
</table>

CK: creatine kinase; cTnI: cardiac troponin I. Values are expressed as mean (coefficient of variation) and range (minimum value-maximum value). Postrace values were corrected for changes in plasma volume.

### Results

All subjects completed the race satisfactorily in a mean time of 332 min (range 247-501 min). The mean percentage change in plasma volume was -6.7±8.3%, but individual percentage changes ranged from -20.1% to +5.9%. Individual corrections of the biochemical values were made accordingly. The serum levels of all the biochemical markers studied increased significantly after the race (Table II). However, the postrace urea levels did not increase above the upper reference limit. Four subjects (36%) had creatinine levels and 6 (55%) had CK levels above their upper reference limits. In addition, all subjects (100%) had myoglobin levels above the upper normal limit. The mean levels of cTnI also increased significantly, but no subjects showed a cTnI concentration above the limit clinically considered to be indicative of acute myocardial infarction (Figure 2).

### Discussion

The results show that this prolonged, strenuous exercise, performed under conditions of continuous variation in the partial pressure of environmental oxygen (range ~146-122 mmHg), can induce significant increases in the plasma concentration of cTnI, a specific marker of myocardial injury. Although these values did not reach the limit at which myocardial infarction would be diagnosed, the significant increase in cardiac troponin concentrations might be indicative of sub-clinical myocardial damage. Similar results have been reported by other authors.\(^\text{17}\) Several mechanisms have been proposed for subclinical myocardial injury during strenuous exercise. The increase in plasma norepinephrine, increased mechanical stress on the ventricular wall, changes in energy dynamics and calcium homeostasis, and oxygen-derived free radicals have been claimed as causes.\(^\text{18-20}\) Strenuous exercise is associated with a dramatic increase in oxygen uptake.\(^\text{21}\) Fluctuations in oxygen partial pressure associated with strenuous exercise, e.g., during rapid changes from hypoxia to normoxia, may encourage, and then partially explain, the transient cardiac dysfunction after prolonged exercise. Nevertheless, the lack of comparison with a control race with similar degree of exercise duration and intensity but conducted entirely at sea level, preclude us to definitely characterize the influence of altitude. In the present study, only one subject showed postrace cTnI levels of 0.39 μg/L close to those used to diagnose myocardial infarction.

Other, less specific markers of myocardial injury (e.g. myoglobin levels) increased significantly by the end of the race, as previously reported.\(^\text{7-10, 22-25}\) Koller
et al. recorded one case of infarction-positive cTnI values after a marathon. Similar results were found after a strenuous exercise challenge performed at high altitude. In the Hawaii Ironman Triathlon, cTnI levels were raised in 2 of 23 entrants (9%), while 6 of 25 athletes (24%) participating in a triathlon in Scotland had infarction-positive post-race cTnI values.

Other studies have used the cardiac troponin T (cTnT) as a means of detecting myocardial injury in professional road cyclists and in athletes who participated in an Ironman Triathlon, and no evidence of damage was found. However, high cTnT levels were detected in one subject after a 50 mile cycling trial in normobaric, hypoxic conditions. These authors suggested that some people might show minimal cardiac damage after exercise in such environments. Interestingly, in the present study, the subject with the highest postrace cTnI levels finished the race in second position (race time 315 min). This agrees with findings reported by Neumayr et al.

Cardiac troponins are the current markers of choice for evaluating myocardial damage, especially in circumstances where skeletal muscle damage is also likely to occur. Cardiac troponin I is associated with specific amino acid sequences encoded by genes different from those that code for the skeletal muscle isoforms. In addition, cTnI has been shown completely specific for cardiac muscle, while cTnT is present in small amounts in skeletal muscle during human fetal development, and is re-expressed in a number of skeletal muscle diseases.

Strenuous exercise is frequently accompanied by skeletal muscle damage. This was observed in the present study: plasma CK, creatinine and urea levels were all higher after the race (Table II). This has also been reported in other studies. A prospective study suggested that habitual vigorous exercise diminishes the risk of sudden death from cardiac causes during vigorous exertion. But benefits do not come without some risk, and this is greater for untrained or sedentary people who abruptly undertake vigorous exertion. The balance of evidence thus supports the need for regular exercise.

Conclusions

In male, amateur mountain bikers, strenuous endurance exercise performed under conditions of continuous variation in the partial pressure of oxygen may induce sub-clinical myocardial damage.

References


