Cardiac fatigue following prolonged endurance exercise of differing distances

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ABSTRACT

WHYTE, G. P., K. GEORGE, S. SHARMA, S. LUMLEY, P. GATES, K. PRASAD, and W. J. McKENNA. Cardiac fatigue following prolonged endurance exercise of differing distances. Med. Sci. Sports Exerc., Vol. 32, No. 6, pp. 1067–1072, 2000. Purpose: Recent echocardiographic studies have reported cardiac dysfunction following ultra-endurance exercise in trained individuals. The duration of exercise required to elicit cardiac dysfunction and the mechanisms underlying this phenomenon have not been fully elucidated. The aim of the present study was to examine the presence of cardiac dysfunction following a half-Ironman and Ironman triathlon in trained individuals. Methods: 14 male triathletes (age: 32 ± 5 yr; height: 180 ± 8 cm; body mass: 75 ± 9 kg) completed a half-Ironman triathlon. Following a 4-wk period, 10 of the original 14 triathletes completed an Ironman triathlon. All triathletes were assessed using ECG, echocardiography, and blood analysis pre-, immediately post-, and 48 h postrace for both distances. Results: Echocardiographic results indicated diastolic and systolic left ventricular dysfunction, for both race distances, which were associated with altered relaxation characteristics and a reduced inotropic contractility, respectively. Following 48-h recovery, all echocardiographic measures were similar to resting values. Creatine kinase MB (CKMB) was significantly elevated immediately postrace for both distances; however, it accounted for less than 5% of the total CK value and in the presence of an elevated total CK and CKMM implied that the elevated CKMB was noncardiac in origin. Troponin-T, however, was significantly elevated immediately postrace for both distances and returned to normal following 48-h recovery indicating myocardial damage. Conclusions: Ironman and half-Ironman competition resulted in reversible abnormalities in resting left ventricular diastolic and systolic function. Results suggest that myocardial damage may be, in part, responsible for cardiac dysfunction, although the mechanisms responsible for this cardiac damage remain to be fully elucidated. Key Words: ECHOCARDIOGRAPHY, LEFT VENTRICLE, CARDIAC DYSFUNCTION, TRIATHLON

Although the cardiovascular benefits of exercise are well established (13), it is recognized that the risk of death is transiently increased during an acute exercise bout in individuals with underlying pathology (1). Recent echocardiographic studies, however, have reported cardiac dysfunction following endurance exercise in the absence of underlying cardiovascular diseases, which has been attributed to “cardiac fatigue” (18). Previous echocardiographic studies have reported left ventricular diastolic and systolic dysfunction following ultra-endurance exercise in trained individuals (6,11). In contrast, Seals et al. (18) demonstrated “cardiac fatigue” following shorter duration endurance exercise (20-km running) in untrained individuals. The duration of exercise required to induce cardiac dysfunction in trained individuals remains to be fully elucidated.

Cardiac myocyte damage has been implicated in the observed cardiac fatigue due to the presence of humoral mark-
Measurement, echocardiographic evaluation, 12-lead ECG, and sessions that took place 2 wk before, immediately post, and of the half-Ironman. In total, there were six data collection in an Ironman triathlon involving exactly twice the distance ing a 4-wk period, 10 of the original 14 athletes competed tinuous 1.9-km swim, 90-km cycle, and 21-km run. Follow-competed in a half-Ironman triathlon race involving a con-
sidered similar for all athletes (20% swimming, 50% cycling, and 30% running). A group of 10 healthy controls (age: 29 ± 3, height: 179 ± 5 cm, body mass: 78 ± 13 kg), participating in less than 2 h of organized physical activity per week were assessed once at the start of the study. All subjects were healthy, free from cardiovascular disease and a family history of cardiovascular disease. None was taking cardiovascular drugs at the time of the study and all were in sinus rhythm. Following ethical approval from the Universities’ ethical committee and before commencing the study, subjects were informed of the testing protocol and signed an informed consent.

Testing schedule. All athletes in the present study competed in a half-Ironman triathlon race involving a continuous 1.9-km swim, 90-km cycle, and 21-km run. Following a 4-wk period, 10 of the original 14 athletes competed in an Ironman triathlon involving exactly twice the distance of the half-Ironman. In total, there were six data collection sessions that took place 2 wk before, immediately post, and 2 d post race for both triathlons (see Fig. 1). Data collection during all testing session consisted of body mass (kg) measurement, echocardiographic evaluation, 12-lead ECG, and venous blood collection. Subjects’ VO$_{2\text{max}}$ was assessed during the first session alone. Although athletes consumed fluid ad libitum during each race, immediate postrace fluid consumption was not allowed until completion of data collection. The range in race times allowed data collection to be completed within 15 min of race completion in all subjects. Measurement of VO$_{2\text{max}}$. A maximal ramping (workload increment started at 0 W and increased by 25 W·min$^{-1}$) cycle-ergometer (Ergo-Metrics 800’ electronically braked cycle-ergometer, Sensor Medics BV, Bilthoven, The Netherlands) exercise test was used to determine VO$_{2\text{max}}$, which was defined as: 1) a plateau in oxygen consumption with increasing workload, 2) a plateau in heart rate with increasing workload, and/or 3) a respiratory quotient (R) greater than 1.5. During rest and exercise, expired gas was collected by an on-line SM2900 metabolic cart (Sensor Medics BV) using the breath-by-breath method. Oxygen concentrations were measured using a zirconium oxygen analyzer, and carbon dioxide concentrations were measured using an infrared absorption method. The SM2900 gas analyzers were calibrated before each test using gases of known quantities. Volume of expired air (V$_E$, L·min$^{-1}$) was measured using a mass flow meter (thermal conductivity). The flow meter was calibrated before each test using a syringe of known volume.

**Echocardiographic measurement.** Echocardiography was performed in all subjects using a Acuson Computed Sonograph 128XP/10c (Hewlett Packard, Andover, MA) with simultaneous ECG recordings. Blood pressure recordings were obtained using a standard sphygmomanometer at the time of echocardiographic interrogation. Subjects were instructed to lie in the left lateral decubitus position and standard two-dimensional guided M-mode echocardiography was used to evaluate cardiac dimensions. M-mode images at the tips of the mitral valve leaflets were used to measure inter-ventricular septal thickness during diastole (IVSd), left ventricular internal diameter during diastole and systole (LVIDd and LVIDs, respectively), and left ventricular posterior free wall during diastole (LVPWd). All measures were taken in accordance with the guidelines set down by the American Society of Echocardiography (ASE). Three to five consecutive measures were made and the average was taken by a single blinded experienced sonographer.

Several derived parameters of left ventricular morphology were calculated. The ratio of mean wall thickness to internal radius (h/R) was calculated using the following formula: h/R = [(IVSd + LVPWd)/2]/(LVIDd/2). The ratio of inter IVSd to LVPWd was also calculated. Left ventricular mass (LVM) was calculated using a previously validated (5) regression-corrected “cube formula”; (LVM (g) = ([(IVSd + LVIDd + LVPWd)/3] - (LVIDd)$^3$) × 1.055) - 13.6 g.

Left ventricular systolic function, evaluated by examination of ejection fraction (EF), velocity of fractional shortening (FS), and stroke volume (SV), were calculated using

![Figure 1—Schematic of data collection and race program.](http://www.msse.org)
the following formulae: \( EF(\%) = \{(LVIDd)^3 - (LVIDs)^3\}/100(LVIDd)^3 \); \( FS(\%) = \{(LVIDd - LVIDs)/LVIDd\} \times 100\); \( SV(\text{mL}) = (LVIDd^3 - LVIDs^3) \). Left ventricular meridional wall stress (LVMWS) was calculated using the formula of Grossman et al., 1975: \( \text{LVMWS} (\text{g-cm}^{-2}) = \text{SBP} \times (LVIDd \times (1.35/4)) \times \text{LVPWs}(1 + \text{LVPWs/LVIDs}) \).

Doppler echocardiography was performed using a 2.5-MHz transducer to assess diastolic function. A two-dimensional apical four-chamber view was imaged, taking care to maximize the diameter of the mitral valve annulus. Pulsed-wave Doppler interrogation of mitral valve inflow velocities were then performed with alignment of the sample volume cursor parallel to flow at the level of the mitral annulus with minor transducer adjustments being made to obtain optimal spectral display (highest velocity with least spectral dispersion). The Doppler velocity curves of three consecutive cardiac cycles were digitized through the darkest gray scale, and the parameters obtained were averaged. Peak early filling (E wave, cm s\(^{-1}\)), and peak late filling (A wave, cm s\(^{-1}\)) velocities were measured and the ratio of early to late diastolic filling (E:A) was calculated.

When interpreting echocardiographic data, it is important to be aware of the sources and magnitude of errors involved in assessment and measurement. Previous studies have stated that the acts of image acquisition and quantitation varied more between than within technicians and readers (9). Therefore, in the present study ultrasound images were obtained using a single experienced sonographer. Intra-observer coefficients of variation for IVSd, LVIDd, LVPWd, LVIDs, and LVM were 3.6%, 2.7%, 5.0%, 2.6%, and 5.4%, respectively. These results are similar to those reported in previous studies and represent approximately 1 mm for all cardiac structures measured (4,9).

**Blood measurements.** Thirty milliliters of venous blood were obtained from an ante-cubital vein. The biochemical parameters assessed were serum sodium, potassium, urea, creatinine, and osmolality. Hematological parameters measured included hemoglobin (Hb), hematocrit (Hct), mean corpuscular hemoglobin (MCH), total and differential counts of leukocytes, platelet count, and RBC count and volume. Total creatine kinase (TCK), creatine kinase isofoms MB (CKMB) and MM (CKMM) fraction, and troponin-T were assessed from each sample as biochemical markers of cardiac myocyte damage.

The isoenzymes for creatine kinase and the isofoms CKMM and CKMB were analyzed by the agarose gel electrophoresis. Bands were visualized using a fluorescent substrate and quantified using a densitometric scanner (16). Serum cardiac troponin-T was analyzed by ELISA using an ES-300 auto-analyzer (11).

**Statistical analysis.** Results were analyzed for each race distance separately via a one-way analysis of variance (ANOVA) with a Tukey post hoc test. Separate analyses for the two distances were completed due to the slightly different sample sizes in each race. A qualitative comparison of data between the two races was undertaken. The alpha level was set at \( P < 0.05 \). Data analysis was completed using the SPSS software package (SPSS Inc., Chicago, IL).

**RESULTS**

All 14 athletes successfully completed the half-Ironman, and all 10 athletes successfully completed the Ironman triathlon. Average race duration for the half-Ironman was 5 h 29 min 29 s ± 20 min 13 s (range: 5:01:12–5:57:20), and for the Ironman 10 h 40 min 28 s (range: 9:23:42–13:27:21). Data were collected within 15 min postrace (8 ± 4 min). Body mass was not significantly altered immediately postrace for both the half-Ironman (76.5 ± 4.9 vs 74.3 ± 6.8 kg) and Ironman (77.2 ± 10.0 vs 75.0 ± 8.5 kg). Resting heart rate was significantly \( P < 0.05 \) increased immediately postrace for both half-Ironman and Ironman (respectively, 57 ± 9 vs 83 ± 7, and 55 ± 10 vs 80 ± 9 bpm), and systolic blood pressure was significantly \( P < 0.05 \) reduced following both race distances (123 ± 7 vs 116 ± 12, and 122 ± 5 vs 114 ± 8 mm Hg, respectively).

**Echocardiographic data.** Compared with controls, triathletes demonstrated a significantly larger IVSd (10.3 ± 1.4 vs 8.5 ± 0.8 cm), LVIDd (54.5 ± 4.1 vs 50.5 ± 2.1 cm), LVIDs (54.9 ± 3.1 vs 50.0 ± 3.2 cm), and LVMWS (31.7 ± 12.7 vs 28.4 ± 12.5 dynes-cm\(^{-2}\)).

**TABLE 4. Ironman functional echocardiographic data.**

<table>
<thead>
<tr>
<th></th>
<th>SV (mL)</th>
<th>EF (%)</th>
<th>FS (%)</th>
<th>E (cm s(^{-1}))</th>
<th>A (cm s(^{-1}))</th>
<th>E:A</th>
<th>LVMWS (dynes-cm(^{-2}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Race</td>
<td>91.3 ± 11.6</td>
<td>77 ± 3</td>
<td>40 ± 3</td>
<td>81 ± 8</td>
<td>43 ± 7</td>
<td>1.9 ± 0.3</td>
<td>31.7 ± 12.7</td>
</tr>
<tr>
<td>Post-Race</td>
<td>80.5 ± 7.5</td>
<td>75 ± 3</td>
<td>37 ± 2*</td>
<td>76 ± 6*</td>
<td>50 ± 5*</td>
<td>1.5 ± 0.2*</td>
<td>27.7 ± 11.1</td>
</tr>
<tr>
<td>48 h Post</td>
<td>88.0 ± 6.5</td>
<td>77 ± 2</td>
<td>40 ± 1</td>
<td>79 ± 9</td>
<td>40 ± 5</td>
<td>2.0 ± 0.4</td>
<td>29.4 ± 12.2</td>
</tr>
</tbody>
</table>

* Significantly different from pre-race and 48 h post data (\( P < 0.05 \)).

SV, stroke volume; EF, ejection fraction; FS, fractional shortening; E, early diastolic filling; A, late diastolic filling; E:A, ratio of early to late diastolic filling; LVMWS, left ventricular meridional wall stress.
LVPWd (10.0 ± 1.0 vs 7.7 ± 0.9 cm), SV (94.7 ± 13.8 vs 92.1 ± 12.0 mL), and VO_{2max} (68.9 ± 6.1 vs 54.0 ± 6.7 mL·kg⁻¹·min⁻¹). Following both the half-Ironman and Ironman IVSd, LVIDs, and LVPWd remained unchanged (see Table 1 and Table 2, respectively). A small decrease in LVIDd was observed following both race distances; however, this was not statistically significant. Stroke volume, fractional shortening, and ejection fraction were significantly \((P < 0.05)\) reduced immediately postrace for the Ironman. Following the half-Ironman, stroke volume was significantly \((P < 0.05)\) reduced, whereas fractional shortening and ejection fraction were unchanged (see Table 3 and Table 4, respectively). Early diastolic filling was significantly \((P < 0.05)\) reduced, late diastolic filling was significantly \((P < 0.05)\) increased, and the resultant early to late diastolic filling ratio was significantly \((P < 0.05)\) reduced immediately post race for both the half-Ironman and Ironman (see Table 3 and Table 4, respectively). Left ventricular meridional wall stress remained unchanged for both race distances.

**Blood data.** White blood cells and neutrophils were significantly \((P < 0.05)\) increased following both race distances and remained elevated above resting levels following 48-h recovery. Lymphocytes and eosinophils were significantly \((P < 0.05)\) reduced immediately postrace and remained below resting values following 48-h recovery for both race distances. Sodium, hemoglobin, Hct, and mean corpuscular hemoglobin remained unchanged following both half-Ironman and Ironman races (see Table 5 and Table 6, respectively). Creatinine and urea were significantly \((P < 0.05)\) elevated immediately postrace demonstrating significant skeletal muscular damage following both half-Ironman (creatinine: 95 ± 13 vs 133 ± 25; urea: 5.5 ± 1.2 vs 7.3 ± 1.2) and Ironman (creatinine: 95 ± 13 vs 112 ± 21; urea: 5.5 ± 1.2 vs 8.9 ± 2.3). Creatinine returned to normal levels following 48-h recovery; however, urea remained significantly \((P < 0.05)\) elevated above resting levels for both race distances (half-Ironman: 5.5 ± 1.2 vs 6.6 ± 1.9; Ironman: 5.5 ± 1.2 vs 6.1 ± 2.2).

Total CK, CKMM, CKMB, and troponin-T were significantly \((P < 0.05)\) increased immediately postrace for both distances. Following 48-h recovery, total CK, CKMM, and CKMB remained significantly \((P < 0.05)\) elevated; however, troponin-T had returned to resting levels for both the half-Ironman and Ironman (see Table 7 and Table 8, respectively).

Resting 12-lead electrocardiograms demonstrated no new ST segment or T-wave changes immediately postrace suggestive of myocardial ischemia for the half-Ironman and Ironman distances.

**DISCUSSION**

The effect of training upon the triathletes was demonstrated by the significantly increased VO\(_{2\text{max}}\), IVSd, LVIDd, and LVPWd. Echocardiographic findings in triathletes were similar to those previously reported (7), and although findings for a number of measures were significantly different from controls, all values were within normal limits, indicating a physiologic cardiac enlargement (10).

Echocardiographically determined left ventricular structure was not significantly altered immediately postrace. However, a smaller LVIDd was observed following both race distances. This difference may be the result of a decreased preload (venous return), or an alteration in the left ventricular relaxation characteristics. In the present study, body mass, Hb, Hct, and serum electrolytes were unaltered immediately post exercise indicative of limited changes in postrace plasma volumes (19). The decreased LVIDd in the present study may, therefore, have occurred as a result of altered relaxation characteristic that was supported by the significant alteration in diastolic filling indices in the present study, a finding previously noted following ultrendurance running (11). The reduced early diastolic filling (E), increased late diastolic filling (A), and resultant reduction in the E:A ratio is associated with left ventricular diastolic stiffness often observed in pathological conditions such as hypertrophic cardiomyopathy (10).

### TABLE 5. Half-Ironman hematological blood analysis data.

<table>
<thead>
<tr>
<th></th>
<th>Hemoglobin</th>
<th>Hematocrit</th>
<th>MCH</th>
<th>WBC</th>
<th>Neutrophils</th>
<th>Lymphocyte</th>
<th>Eosinophils</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Race</td>
<td>14.1 ± 1.0</td>
<td>42 ± 2</td>
<td>30 ± 2</td>
<td>5.2 ± 2.3</td>
<td>36.2 ± 2.7</td>
<td>45.7 ± 16.7</td>
<td>10.9 ± 8.5</td>
</tr>
<tr>
<td>Post-Race</td>
<td>14.8 ± 1.2</td>
<td>43 ± 3</td>
<td>31 ± 1</td>
<td>15.2 ± 3.3</td>
<td>78.9 ± 3.6</td>
<td>8.7 ± 2.8</td>
<td>0.8 ± 0.7</td>
</tr>
<tr>
<td>48 h Post</td>
<td>13.3 ± 0.8</td>
<td>40 ± 2</td>
<td>30 ± 1</td>
<td>5.9 ± 0.9</td>
<td>50.0 ± 6.5</td>
<td>27.4 ± 8.2</td>
<td>5.5 ± 2.8</td>
</tr>
</tbody>
</table>

\(^{a}\)Significantly different from pre-race and 48 h post data \((P < 0.05)\).

\(^{b}\)Significantly different from post-race data \((P < 0.05)\).

\(^{c}\)Significantly different from pre-race data \((P < 0.05)\).

MCH, mean corpuscular hemoglobin; WBC, white blood cells.

### TABLE 6. Ironman hematological blood analysis data.

<table>
<thead>
<tr>
<th></th>
<th>Hemoglobin</th>
<th>Hematocrit (%)</th>
<th>MCH</th>
<th>WBC</th>
<th>Neutrophils</th>
<th>Lymphocyte</th>
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<td>36.2 ± 2.7</td>
<td>45.7 ± 16.7</td>
<td>10.9 ± 8.5</td>
</tr>
<tr>
<td>Post-Race</td>
<td>14.2 ± 0.8</td>
<td>42 ± 2</td>
<td>30 ± 1</td>
<td>16.4 ± 2.9</td>
<td>80.2 ± 6.1</td>
<td>8.5 ± 3.2</td>
<td>1.0 ± 0.8</td>
</tr>
<tr>
<td>48 h Post</td>
<td>13.0 ± 0.8</td>
<td>40 ± 2</td>
<td>30 ± 1</td>
<td>6.2 ± 0.1</td>
<td>57.2 ± 6.7</td>
<td>30.3 ± 4.8</td>
<td>3.5 ± 1.5</td>
</tr>
</tbody>
</table>

\(^{a}\)Significantly different from pre-race and 48 h post data \((P < 0.05)\).

\(^{b}\)Significantly different from post-race data \((P < 0.05)\).

MCH, mean corpuscular hemoglobin; WBC, white blood cells.
Stroke volume was significantly reduced immediately postrace for both the half-Ironman and Ironman. This reduction SV occurred as a result of minor, nonsignificant changes in LVIDd with no change in LVIDs. The significant increase in fractional shortening (FS) and ejection fraction (EF) observed immediately postrace for the Ironman in the present study are in agreement with previous studies (6,11,18). This reduction in FS and EF does not in itself imply a reduced inotropic state, as afterload and preload are strong determinants of these commonly used ejection phase indexes of myocardial contractility. However, in the present study after-load (systolic blood pressure) was significantly reduced and preload (discussed earlier) was unaltered immediately postrace suggesting that the reduction in SV, EF, and FS were related to systolic dysfunction associated with a reduced inotropic state. In support of this argument, LVIDs was unaltered immediately postrace despite a significantly reduced after-load (systolic blood pressure) and an unchanged left ventricular meridional wall stress. These findings are similar to those reported in previous studies following 24 h running (11), Ironman triathlon (6), and prolonged cycling (14). In another study, Perrault et al. (15) reported no effect of marathon running upon left ventricular performance in trained individuals. However, although no change in FS, EF, and Vcf was observed, the authors suggested that the significant decrease in systolic blood pressure concomitant with an unaltered LVIDs was suggestive of a decreased inotropic contractility.

Following both half-Ironman and Ironman races, sodium, hemoglobin, Hct, and mean corpuscular hemoglobin remained unchanged. Hemoglobin, however, was significantly reduced 48 h postrace for both distances. The mechanism underlying this finding is not clearly understood; however, it may be associated with a reduced splanchnic blood flow and a high demand for oxygen during exercise resulting in a reduced rate of lysed red blood cell removal during exercise.

Increases in total CK, CKMM, and CKMB were noted immediately postrace for both distances. These elevated values persisted following 48 h of recovery. The time taken for total CK and CKMB to reach maximum values has been previously reported as 18 h and 17.4 h, respectively (12), thus explaining, in part, the persistent elevation noted in the present study. The significant increase in CKMB may be associated with myocardial cell damage, however, the MB fractions in the present study were not greater than 5% (3.6% and 4.5% for the half-Ironman and Ironman, respectively) of the total CK value (12), and in the presence of elevated CKMM values the elevated CKMB may be noncardiac in origin (6,11). The large increases in CKMM immediately postrace and following 48-h recovery are associated with skeletal muscle breakdown (23), which may account for the increase noted in CKMB. This hypothesis is supported by an earlier study examining muscle biopsies in endurance trained athletes and controls following training (2). The results of Apple et al. (2) demonstrated CKMB accounted for 8.9% of total CK activity compared with 3.3% in controls. The diagnostic significance of an elevated CKMB fraction in the presence of an elevated total CK must be viewed with caution. Indeed an elevated CKMB may be simply indicative of transient rhabdomyolysis of skeletal muscle in the absence of other indices of myocardial damage (23).

In contrast to the relatively nonspecific CKMB fraction, troponin-T is a highly specific marker of myocardial injury (20). The presence of a significant increase in troponin-T and its subsequent reduction to normal values following 48-h recovery in the present study indicates that myocardial damage may have occurred following both race distances. This cardiac myocyte damage may result in the cardiac dysfunction reported in the present and previous echocardiographic studies (6,11,18). The increased troponin-T was small and does not reflect those values observed following myocardial infarction. Indeed troponin-T values return to normal 48 h postrace, indicating the transient effect of prolonged endurance exercise upon the myocardium. Although the elevated CKMB fraction is associated with transient rhabdomyolysis, the presence of an elevated troponin-T may indicate that cardiac myocyte damage contributed to the overall increase in CKMB. The combination of early markers of myocardial injury such as myoglobin together with more specific markers such as CKMB and troponin-T may improve the sensitivity in identifying cardiac myocyte damage following endurance exercise and is an area for future investigation.

The underlying mechanism(s) responsible for myocardial damage and the resultant cardiac dysfunction observed in the present study remains to be elucidated. Cardiac dysfunction in the absence of myocardial necrosis has been previously described as “myocardial stunning” (3). This “stunning” may be the result of transient ischemia during exercise and may be associated with the accumulation of oxygen free radicals (21). Although abnormal ST segment and T-wave alterations immediately post exercise were absent in the present study, it does not rule out the possibility of ischemia during exercise. Further, left ventricular dysfunction may occur before abnormalities are observed in ECG waveforms (8). A number of mechanisms resulting in myocardial ischemia during exercise have been described and include: 1) increased catecholamine concentration resulting in increased vascular tone and a reduced myocardial blood supply, 2) increased shear stress on coronary arteries resulting in damage to the endothelial lining, 3) magnesium ion deficiency resulting in coronary vasospasm, and 4)
increased thrombogenesis as a result of magnesium deficiency and increased catecholamines (15). The underlying mechanisms responsible for the myocardial damage observed in the present study remain to be fully elucidated and warrant further investigation.

Conclusions. Ironman and half-Ironman competition result in reversible abnormalities in resting left ventricular diastolic and systolic function. The significant increase in troponin-T in the present study suggests that myocardial damage occurred as a result of prolonged endurance exercise in trained individuals. The mechanisms responsible for this cardiac damage remain to be fully elucidated.

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