Cardiac fatigue after prolonged exercise

PAMELA S. DOUGLAS, M.D., MARY L. O'TOOLE, PH.D., W. DOUGLAS B. HILLER, M.D.,
KEITH HACKNEY, AND NATHANIEL REICHEK, M.D.

ABSTRACT To determine the effects of prolonged exercise on systolic and diastolic left ventricular function, we studied 21 athletes before, at the finish (within 11 ± 5 min), and during recovery (28 ± 9 hr) after the Hawaii Ironman Triathlon (2.4 mile swim, 112 mile bike, 26.2 mile run). Two-dimensionally guided M mode echocardiograms were digitized for wall thickness, cavity dimension, fractional shortening, and peak rates of cavity enlargement and wall thinning. Pulsed Doppler left ventricular inflow recordings were analyzed for peak early and late velocities and their ratio. Left ventricular diastolic dimension was reduced at race finish (5.4 ± 0.6 to 5.1 ± 0.6 cm) and remained reduced after 1 day of recovery (5.2 ± 0.6 cm, p<.05). Fractional shortening fell at race finish (39 ± 5% to 35 ± 5%), although systolic blood pressure was unchanged, and rose to 40 ± 4% after recovery (p<.05). The return to prerace shortening values after recovery occurred despite continued reduction in diastolic size. Peak circumferential shortening did not change significantly. Individual reductions in fractional shortening were correlated with increases in systolic cavity size (r = −.64, p<.01), but not with decreases in diastolic size. The stress-shortening relationship was displaced downward at race finish, but returned toward baseline after 1 day of recovery, despite a persistent reduction in cavity size. This suggests that the decrease in shortening was due to impaired contractility as well as altered preload. The left ventricular filling pattern was altered at race finish, with increased late inflow velocity (38 ± 12 to 51 ± 19 cm/sec) and a reduced ratio of early to late velocities (1.9 ± 0.6 to 1.5 ± 0.6). In contrast, peak rates of cavity enlargement and wall thinning were unchanged. All functional variables returned to prerace values during recovery. We conclude that prolonged exercise may result in alterations in systolic and diastolic left ventricular performance. The rapid reversal of all changes suggests cardiac “fatigue.”


EXTREME EXERCISE is widely known to pose a variety of health hazards, although myocardial dysfunction is not often considered among them. However, with increasing participation in ever-more grueling, ultraendurance racing events, clear delineation of the cardiac response becomes important to athlete safety. Existing data suggest that the effects of prolonged exhaustive exercise on left ventricular systolic performance may be deleterious.1-6 The effects of exercise on diastolic performance have not been examined, although depression of inotropic state could affect active relaxation as well as systolic contraction.

Accordingly, we sought to determine the effects of prolonged, competitive exercise on left ventricular systolic and diastolic performance. Echocardiography, Doppler velocimetry, electrocardiography, and biochemical studies were performed before, immediately upon finishing, and during recovery from the Hawaii Ironman Triathlon, an ultraendurance race consisting of a 2.4 mile swim, 112 mile bike ride, and 26.2 mile run.

Methods

Subjects. The study population consisted of 21 ultraendurance athletes with a mean age of 34 ± 9 years (range 19 to 55 years) and included 13 men and eight women. Environmental conditions during the race included humidity ranging from 40% to 85%, ambient temperatures ranging from 24° to 42° C, and water temperature of 26° C. During the race the athletes chose their own speed and rest periods, and had liberal access to fluids and foods. Subjects were weighed nude and dry immediately before and after the race.

Echocardiograms. All subjects underwent three echocardiographic and Doppler studies: 2 to 4 days before the race, immediately upon finishing the race (average time from finish to recording was 11 ± 5 min, range 3 to 23 min), and after 1 to 2 days of recovery (28 ± 9 hr). Two-dimensionally guided M mode echocardiograms of the left ventricle were recorded at the chordal level with an ATL Ultramark 8 equipped with a 3.0 MHz transducer and strip-chart recorder. Imaging location and gain settings were adjusted to yield optimal definition of endocardial and epicardial borders. Echograms of three to five cardiac cycles were digitized at 10 msec intervals along the left septal and posterior wall endocardium and posterior wall epi-
cardium to determine continuous cavity and wall dimensions. Maximum and minimum cavity and wall thickness dimensions were taken as end-diastolic and end-systolic values. Fractional shortening was calculated as:

\[
\frac{(EDD) - (ESD)}{(EDD)} \times 100
\]

Digitized data were differentiated to yield normalized peak rates of circumferential shortening (peak V_c), cavity enlargement, and posterior wall thinning. Validation of computerized analysis of echograms has been performed by comparison with angiographic data. 7,8

Cuff sphygmomanometer blood pressure was taken simultaneously with performance of echocardiographic studies. Systolic blood pressure was combined with echocardiographic dimensions to provide an index of end-systolic meridional stress: 0.334 P(LVID)/PWT(1 + PWT/LVID), where P = pressure; LVID = internal dimension; PWT = posterior wall thickness.9

**Doppler recordings.** Two-dimensionally guided pulsed Doppler recordings of left ventricular inflow at the level of the mitral anulus were obtained with an ATL Ultramark 8 ultrasonograph equipped with a combined 3.0 MHz imaging and Doppler transducer. All recordings were obtained from the apical two- or four-chamber views parallel to assumed flow and with optimal definition of the spectral envelope. Data from three to five cardiac cycles were digitized to yield peak velocities of left ventricular inflow in early and late diastole and the ratio of early-to-late diastolic flow velocity. Validation of Doppler assessment of left ventricular filling has been performed by comparison with angiographic and nuclear data.10,11

Doppler recordings of ascending aortic flow were obtained with a 3.0 MHz nonimaging Doppler transducer applied to the suprasternal notch. If recordings were inadequate, left ventricular outflow was obtained from the apical two- or four-chamber view under two-dimensional echocardiographic guidance. The same view was used for all three samples (prerace, finish, and recovery) in any given subject. Data from three to five cardiac cycles were digitized to yield peak velocity and flow velocity integrals.

**Electrocardiogram.** Standard 12-lead electrocardiograms obtained using a Marquette MAC 12 electrocardiograph were recorded before the race, at race finish, and after an average of 28 hr of recovery.

**Blood samples.** Venous blood samples were obtained 24 hr before the race, at the finish, and after an average of 22 hr of recovery. Samples were clotted and centrifuged, and the serum was stored on ice and shipped for analysis. Measurement of creatine kinase activity, MB subfraction, and lactate dehydrogenase and its first isoenzyme (LD-1) were performed by photometric immunochemical methods with the use of a Cobas centrifugal analyzer. Finger stick samples were obtained at race finish and analyzed immediately for lactate concentration with a YSI Model 23L enzymatic lactate analyzer.

**Brief exercise control data.** Since any possible changes found in cardiac function after prolonged exercise might be due to exercise itself rather than its exhaustive nature, we also examined a comparable group of athletes after brief exercise. Eight ultraendurance athletes (mean age 37 ± 8 years; five men, three women) were studied before and immediately after a 10 min period of treadmill exercise. The duration of exercise was chosen to correspond to that of a routine stress test. Treadmill speed was individually selected to maintain a heart rate of 140 beats/min since this was the average heart rate maintained by 18 athletes monitored during actual Ironman Triathlon competition.12 Average treadmill speed was 6.74 ± 1.02 mph and ranged from 5.65 to 8.19 mph.

Control athletes underwent similar echocardiographic, Doppler, and electrocardiographic studies at baseline and immediately after brief exercise. Data were analyzed as described above.

To examine the reliability of M mode echographic and Doppler variables in the population of interest, we performed repeat resting studies in eight triathletes 2 to 30 days apart. These athletes were not participants in either the brief or prolonged exercise bouts. Data were analyzed by comparison of each individual’s measurements with those obtained from his previous study. Comparison of echographic measurements, including wall thickness and cavity dimension, showed repeated measurements to be similar, with a close correlation (r = .98) between data obtained at repeat study. The relationship had a slope of 1.03 and intercept of 0.08 cm, and the SEE was 3 mm. Comparison of continuously digitized M mode echocardiographic data (peak rates of wall thinning and cavity enlargement) obtained on different days yielded an adequate correlation (r = .98). The relationship had a slope of 0.997, y intercept of 0.24 cm/sec, and an SEE of 2.24 cm/sec. Comparison of Doppler measurements of peak left ventricular inflow velocities yielded an adequate correlation (r = .87) between data obtained at repeat study. The relationship had a slope of 1.01 and intercept of 7.15, and the SEE was 10.8 cm/sec.

**Statistical analysis.** Comparison of prerace, finish, and recovery values were performed by one-way repeated-measures analysis of variance with post hoc testing performed by the Newman-Keuls test. The relationships of changes in cavity size and heart rate and race duration with changes in functional variables were assessed by linear regression analysis. The stress-shortening regression lines obtained before the race, at the finish, and at recovery were compared with large-sample Z tests for parallelism and a common intercept.13 Data in text and tables are presented as the mean ± SD.

**Results**

**Brief exercise.** Comparison of echographic and Doppler data obtained before and after brief exercise revealed no significant differences in cavity sizes, wall thickness, or systolic and diastolic function (table 1).

**Prolonged exercise.** All athletes undergoing prolonged exercise successfully completed the Hawaii Ironman Triathlon consisting of a 2.4 mile swim, 112 mile bike ride, and 26.2 mile run. Average race duration was 12 hr 30 min and ranged from 10 hr 30 min to 15 hr 50 min. Average weight loss was 6.1 ± 4.2 pounds, or 4% (p<.01). Sodium and potassium concentrations at race finish were unchanged from resting values in all athletes; all lactate values were less than 2 mg/dl. Heart rate was significantly increased at race finish compared with prerace and recovery values, while systolic blood pressure was similar at all three determinations (table 2).

**Echocardiography and prolonged exercise (table 2; figure 1).** At rest, athletes had top normal values for left ventricular cavity dimension, wall thickness, and fractional shortening. At race finish, diastolic dimension was reduced (p<.05) and did not change significantly after 1 day of recovery. In individual athletes, end-systolic dimension was not consistently increased or
TABLE 1

<table>
<thead>
<tr>
<th>Physical characteristics</th>
<th>Baseline</th>
<th>After exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>54 ± 7</td>
<td>65 ± 7*</td>
</tr>
<tr>
<td>(41–61)</td>
<td>(65–78)</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>127 ± 19</td>
<td>120 ± 16</td>
</tr>
<tr>
<td></td>
<td>(94–142)</td>
<td>(98–148)</td>
</tr>
</tbody>
</table>

Echocardiographic variables
- Cavity dimension, diastole (cm)
  5.5 ± 0.5 vs 5.4 ± 0.4 
- Cavity dimension, systole (cm)
  3.5 ± 0.5 vs 3.4 ± 0.4 
- Wall thickness, diastole (cm)
  0.8 ± 0.2 vs 0.9 ± 0.2 
- Wall thickness, systole (cm)
  1.7 ± 0.2 vs 1.7 ± 0.2 
- Fractional shortening (%)
  37 ± 5 vs 37 ± 6 
- Wall stress (×10^6 dynes/cm²)
  64 ± 19 vs 60 ± 21 
- Rate of cavity enlargement (dD/dt/D)
  3.4 ± 1.1 vs 3.9 ± 1.1 
- Rate of wall thinning (−dh/dt/h)
  8.1 ± 3.9 vs 8.8 ± 3.3 

Doppler variables — mitral flow
- Early velocity (cm/sec)
  87 ± 15 vs 87 ± 11 
- Late velocity (cm/sec)
  53 ± 11 vs 51 ± 9 
- Velocity ratio
  1.7 ± 0.3 vs 1.8 ± 0.6 

* p < .01 vs baseline.

The data show that during physical exercise, mean values were similar at all three recordings. Mean fractional shortening was reduced at race finish, despite a lack of change in blood pressure or wall stress. The reduction in shortening tended to be greatest in those with the fastest race times (r = .50, p < .03). Shortening rapidly returned to normal after 1 day of recovery, while diastolic cavity size remained decreased. When examined in individual athletes, the reduction in shortening was correlated with changes in systolic dimension (r = −.64, p < .01), but not with changes in diastolic dimension (r = .18, p = NS) (figure 2).

The linear, inverse correlation between stress and shortening was displaced downward at race finish as measured by comparison of the y intercepts (p = .02), indicating less shortening for a given afterload. The intercept returned toward prerace values during recovery, despite a persistent reduction in left ventricular cavity dimension. The slopes of the three equations were not statistically different (figure 3).

Peak V<sub>cf</sub> (cm/sec<sup>2</sup>/cm) fell from a prerace value of 3.85 ± 0.75 to 3.63 ± 1.09 at finish and rose to 4.21 ± 0.84 after 1 day of recovery. These changes approached statistical significance with a p value of .09. Neither the ratio of end-systolic dimension to end-systolic stress nor mean V<sub>cf</sub> changed significantly at any data recording point. Similarly, wall stress and normalized peak rates of wall thinning and cavity enlargement were unchanged.

TABLE 2

<table>
<thead>
<tr>
<th>Prolonged exercise: physical, echographic, and Doppler results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before race</td>
</tr>
<tr>
<td>-------------</td>
</tr>
<tr>
<td>Physical characteristics</td>
</tr>
<tr>
<td>Body weight (pounds)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
</tr>
<tr>
<td>(38–79)</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

Echocardiographic variables
- Cavity dimension, diastole (cm)
  5.4 ± 0.6* vs 5.1 ± 0.6 vs 5.2 ± 0.6 vs 5.2 ± 0.6
- Cavity dimension, systole (cm)
  3.3 ± 0.5 vs 3.3 ± 0.4 vs 3.2 ± 0.4 vs 3.2 ± 0.4
- Wall thickness, diastole (cm)
  1.0 ± 0.2 vs 1.0 ± 0.2 vs 1.0 ± 0.2 vs 1.0 ± 0.2
- Wall thickness, systole (cm)
  1.6 ± 0.3 vs 1.5 ± 0.2* vs 1.7 ± 0.2 vs 1.7 ± 0.2
- Fractional shortening (%)
  39 ± 5 vs 35 ± 5* vs 40 ± 4 vs 40 ± 4
- Wall stress (×10^6 dynes/cm²)
  61 ± 17 vs 61 ± 16 vs 49 ± 10* vs 49 ± 10*
- Rate of cavity enlargement (dD/dt/D)
  2.5 ± 0.4 vs 2.5 ± 12 vs 2.6 ± 0.5 vs 2.6 ± 0.5
- Rate of wall thinning (−dh/dt/h)
  9.5 ± 2.7 vs 10.1 ± 3.0 vs 8.3 ± 4.1 vs 8.3 ± 4.1

Doppler variables — mitral flow
- Early velocity (cm/sec)
  69 ± 16 vs 68 ± 16 vs 70 ± 14 vs 70 ± 14
- Late velocity (cm/sec)
  38 ± 12 vs 51 ± 19* vs 36 ± 9 vs 36 ± 9
- Velocity ratio
  1.9 ± 0.6 vs 1.5 ± 0.6* vs 2.0 ± 0.4 vs 2.0 ± 0.4

Doppler variables — aortic flow
- Peak velocity (cm/sec)
  101 ± 22 vs 98 ± 25 vs 105 ± 20 vs 105 ± 20
- Flow velocity integral
  20.7 ± 5.2 vs 16.9 ± 6.4 vs 19.3 ± 4.8 vs 19.3 ± 4.8
- Heart rate × flow velocity integral
  1232 ± 414 vs 1565 ± 682* vs 1161 ± 303 vs 1161 ± 303

*p < .05 vs both other determinations.
* *p < .01 vs both other determinations.
Doppler velocimetry and prolonged exercise (table 2; figure 1, bottom). Early diastolic velocities of left ventricular inflow were similar at all three measurement times, while late diastolic velocities were higher at race finish and returned to baseline during recovery. The ratio of early to late velocities reflected these changes, falling an average of 21% at race finish, but returning to normal by recovery sampling. These changes were unrelated to the increased heart rate present at race finish.

Aortic flow recordings showed no significant changes in peak velocity or flow velocity integral. The product of heart rate and flow velocity integral, an estimate of cardiac output, was increased at race finish (p<.05) (table 2).

Results of cardiac enzyme determinations are listed in table 3. All athletes had a marked rise in creatine kinase and its MB fraction, although the percent MB was not increased. Similarly, total lactic dehydroge-

nase was increased at both finish and recovery, while the %LD-1 was decreased or unchanged. Twelve-lead electrocardiograms showed no new ST segment or T wave abnormalities at either race finish or recovery that were suggestive of myocardial ischemia.

Possible relationships between race performance, or the time required to finish the triathlon, and left ventricular functional changes were examined. There were modest correlations between a shorter race duration and the reduction in left ventricular cavity sizes (diastolic, r=.54, p<.02; systolic, r=.58, p<.01) and the reduction in fractional shortening (r=.50, p<.03). There was no significant relationship between race duration and changes in either Doppler or echocardiographic variables of diastolic function.

**Discussion**

In response to upright submaximal exercise, cardiac output increases substantially due to tachycardia and to
increases in stroke volume produced by a higher ejection fraction and a larger end-diastolic volume. The Starling mechanism appears to be operative, with an increase in initial fiber length (diastolic chamber size) contributing to enhanced cardiac function. As exercise is prolonged, however, the heart rate rises and stroke volume falls, so that cardiac output is unchanged or even decreased. These changes have been termed cardiovascular “drift” and have been variously attributed to decreases in blood volume, redistribution of flow to the skin vasculature to aid heat loss, and cardiac fatigue. While the former two factors have been well documented, the possible contribution of a decline in cardiovascular function is unclear.

Systolic performance. In the present study, fractional shortening was found to decrease by an average of 10%. However, because this index of myocardial performance is preload dependent, its significance can only be determined with simultaneous consideration of this factor. The reduction in diastolic cavity size at race finish suggests that decreased preload, via the Starling mechanism, may have contributed to reduced shortening. However, several other factors suggest that impaired cardiac performance may also be contributing to the observed dysfunction. First, the correlation between stress and shortening was displaced downward at race finish, but the slope was unchanged, suggesting that for a given afterload, shortening was reduced (figure 3). More importantly, the stress-shortening regression line returned toward normal after 28 hr, indicating partial recovery, despite a persistent reduction in left ventricular cavity dimension. Although the difference between the prerace and finish correlations may be attributable to altered preload, that between finish and recovery cannot, suggesting that contractility was reduced at race finish.

Similarly, although shortening rapidly returned to normal during recovery, diastolic dimension, an index of preload, did not. The trend toward a reduction in peak V_{EO} is consistent with these data. Finally, in individual athletes, the decrease in shortening was correlated with increases in systolic cavity dimension (r = −.64, p < .01), and not with decreases in diastolic dimension (r = .18, p = NS). This correlation was significant despite the fact that mean end-systolic dimension did not change.

Shortening is also afterload dependent, such that a reduction in shortening in the presence of unchanged load implies a reduction in inotropic state. Thus, the reduction in shortening without a concomitant increase in systolic blood pressure and/or wall stress suggests that cardiac performance may be adversely affected by prolonged exercise.

Our findings regarding systolic function are concordant with those in the literature. Maher et al. noted a decrease in peak isometric tension and velocity of shortening (as a result of glycogen depletion) after prolonged stimulation of isolated rat myocardium.
Because concentrations of ATP, phosphocreatine, and norepinephrine were unchanged, they concluded that exhaustion was associated with a depression of contractile state.

In man, several authors have reported evidence for a deleterious effect of exercise on systolic cardiac function. Upton et al.\(^3\) used blood pool scanning to study the cardiac response to maximal exercise alone or after 2 hr of prolonged submaximal exercise in conditioned man. They found that in response to maximal exercise after prolonged submaximal exercise, the heart was unable to augment stroke volume to levels previously obtained during maximal exercise alone. In fact, ejection fraction actually fell in response to the increased workload in five of nine subjects, suggesting a role for cardiac fatigue. Niemela et al.\(^2\) studied runners during a 24 hr race and found them to have reduced fractional shortening and \(V_{cf}\) at race finish, despite lower blood pressure. These abnormalities were rapidly reversible, returning to normal 2 to 3 days after the race. The amount of dysfunction (fractional shortening fell from 38% to 32%) was analogous to that found in the present study, and also occurred in the setting of reduced diastolic, but unchanged systolic, cavity sizes. However, because in individuals the decrease in shortening was correlated with increases in systolic dimension and not with decreases in diastolic dimension, the authors concluded that prolonged exercise resulted in a depression of contractility.

Another parallel between the study of Niemela et al. and the present study is the finding that reversible cardiac dysfunction may be related to exercise intensity. Niemela et al. observed increasing impairment throughout the race and also found a negative correlation between \(V_{cf}\) and the total distance run. Perrault et al.\(^6\) studied cardiac fatigue before and after a marathon run (2.5 to 4 hr duration) and found that fractional shortening was unchanged despite decreased wall stress. While suggestive of cardiac impairment, perhaps the small magnitude of the observed changes was due to the relatively short exercise duration, as the physiologic stress of exhaustive exercise is likely related to duration of effort as well as its intensity.

Two other preliminary studies found evidence of transient depression of systolic function after a marathon run. Boynton et al.\(^20\) noted an immediate postrace decrease in the rate of end-systolic pressure to dimension that returned to normal in 1.5 to 3 hr. Chan et al.\(^21\) found transient reductions in two-dimensional echocardiographic ejection fraction and stroke volume in runners completing a marathon in less than 3.5 hr.

A similar impairment in systolic performance has been noted in untrained individuals performing prolonged submaximal exercise. Seals et al.\(^5\) exercised healthy men to exhaustion (average duration 170 ± 33 min at 70% of aerobic capacity) and found a marked reduction in fractional shortening (33% to 28%) despite unchanged diastolic left ventricular size and lowered wall stress. It is interesting to note that, in these untrained individuals, the amount of exercise needed to impair cardiac function was less than that needed in highly trained athletes. The “threshold” of cardiac fatigue in the elderly, or in those with known myocardial disease, may be even lower.

**Diastolic performance.** Despite evidence that systolic cardiac function is unfavorably affected by prolonged exercise, previous investigators have not examined diastolic performance. In the present study, assessment of early diastolic function showed no change in peak rates of wall thickening or in cavity enlargement as measured by digitized M mode echocardiography, and no change in peak velocity of early left ventricular inflow as measured by the pulsed Doppler method. In contrast, assessment of left ventricular filling pattern showed an increased velocity of atrial or late diastolic inflow, resulting in a decrease in the ratio of early-to-late flow
velocities. These changes were rapidly reversible and the left ventricular filling pattern returned to normal during the 28 hr of recovery. Neither the increased late diastolic velocity nor the reduced flow velocity ratio were correlated with the increased heart rate or decreased diastolic cavity size seen at race finish.

Similar changes in Doppler inflow patterns have been noted with aging, ischemia, and hypertrophy caused by aortic stenosis and hypertension. Such abnormal filling patterns may be due to reduced ventricular compliance or increased myocardial stiffness. In the athletes studied, a change in either variable is possible, but neither can be assessed noninvasively. An additional possibility is simply augmentation of the force of left atrial contraction resulting in a higher velocity of blood flow during atrial systole. Speculation as to specific causes is difficult since the components of diastole are multiple and their interrelationships are complex. Furthermore, many factors may affect diastolic performance, including loading conditions, inotropic state, myocardial characteristics, and their interactions. While Doppler-derived variables of diastolic function compare favorably with results obtained with contrast and nuclear angiography, and therefore allow evaluation of left ventricular filling, they cannot provide a complete assessment of the components of diastolic performance or identify the cause of the abnormality.

The different results obtained from analysis of wall thinning and cavity enlargement and Doppler velocimetry may be readily explained. M mode measures primarily evaluate ventricular relaxation during early diastole, whereas Doppler-derived variables reflect left atrial to left ventricular inflow and are measured throughout diastole. Others have found rates of cavity enlargement to be normal when Doppler inflow patterns are not, suggesting that Doppler measurements are more sensitive to early diastolic abnormalities than are M mode measurements.

The deleterious effects of exercise we observed in the absence of metabolic derangements (such as acidosis or electrolyte abnormalities) raise the question of cardiac injury. However, the lack of ischemic electrocardiographic changes, and the lack of elevation of cardiac fraction of creatine kinase and lactic dehydrogenase enzymes suggest that irreversible cardiac injury is insignificant, if indeed it is present at all. Although we are unable to absolutely exclude altered preload as a contributor to our findings, rapidly reversible changes in systolic and diastolic function, perhaps due to cardiac fatigue, may assume clinical importance in athletes competing frequently or over many years and in those with underlying cardiac dysfunction or under more extreme race or environmental conditions. These factors must be considered in advising individuals who pursue prolonged, strenuous exercise and in providing medical care to athletes during competition.

We thank Jeannette Forte and Evelyn Robles for their excellent secretarial support and John Fiolkoski for his much appreciated technical expertise.

References
20. Boynton M, Winslow E, Ling R, Mehlman D: Are echocardio-
Cardiac fatigue after prolonged exercise.
P S Douglas, M L O'Toole, W D Hiller, K Hackney and N Reichek

_Circulation_. 1987;76:1206-1213
doi: 10.1161/01.CIR.76.6.1206

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1987 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/76/6/1206

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/