Evidence of impaired left ventricular performance after an uninterrupted competitive 24 hour run


ABSTRACT The effect of extremely exhaustive exercise on left ventricular performance was studied echocardiographically in 13 experienced male ultramarathon runners who took part in a competitive 24 hr run, completing distances of 114 to 227 km. Although the left ventricular end-diastolic dimension (EDD) was reduced by 7% (54 ± 5 to 50 ± 7 mm; p < .005), the end-systolic dimension (ESD) increased slightly (33 ± 5 to 34 ± 6 mm; NS). As a consequence, the stroke dimension (21 ± 2 to 16 ± 2 mm; p < .005) and fractional shortening (38 ± 5% to 32 ± 5%; p < .005) declined by 24% and 16%, respectively. The reduction in fractional shortening was related to ΔESD (r = −.66; p < .05) but not to ΔEDD (r = .22; NS). In spite of reduced afterload, the mean velocity of circumferential fiber shortening also decreased by an average of 9% (p < .01) in proportion to the distance completed (r = −.69; p < .01). The systolic blood pressure/ESD ratio was 21% lower after the race (4.2 ± 0.9 to 3.3 ± 0.6; p < .005). Body weight loss was not related to any alterations in left ventricular dimensions or ejection phase indexes. The stroke dimension and ejection phase indexes continued to decline within the last 6 hr of the race but returned to the prerace level 2 to 3 days after the race. Total serum creatine kinase peaked at 3917 to 64740 U/liter (mean 27427) and its MB percentage peaked at 2% to 6%. No electrocardiographic evidence of myocardial injury was seen immediately after the race. Highly strenuous prolonged exercise thus appears to result in impaired resting left ventricular function, in part because of a reversible depression in the contractile state. The possibility of cardiac fatigue suggests a need for strict selection of ultramarathon participants.


ALTHOUGH the response of the human heart to exercise has been studied extensively, little information is available on the effects of utterly exhaustive exercise on cardiac performance in man. Marathon running is growing in popularity, however, and thousands of runners, more or less well trained, are taking part in various ultramarathon races (i.e., distances greater than the marathon) all over the world each year. It is well recognized that ultramarathon running is not without risk. In addition to casualties caused by exhaustion, heat stroke, renal failure, and sudden cardiac death, repeated hemoptysis and pulmonary edema due to left ventricular failure has been reported in two well-trained athletes during a 90 km run. However, subsequent clinical investigations in these athletes, including cardiac catheterization, revealed no apparent cardiac abnormalities, and it was postulated that some as yet unknown factor (or factors) could lie behind these observations.

We had a unique opportunity to make echocardiographic and biochemical measurements on well-trained ultramarathon runners in connection with a competitive 24 hr run. Our results indicate a deleterious effect of such severe prolonged exercise on left ventricular performance.

Methods

Subjects. Thirteen healthy male marathon runners 23 to 54 years old who took part in a competitive 24 hr run in August 1983 volunteered as subjects for this study. All of them were experienced endurance athletes, having participated in many marathon and ultramarathon races. The subjects were placed among the best 15 runners in the race (table 1). Each athlete had been training for several years. The training program involved mainly aerobic endurance running (“slow long distance”), ranging from 400 to 1000 km monthly.

A high-quality echocardiogram was obtained from each subject before the race and on its completion. The interval between the end of the race and recording of data varied to some extent because we used only one set of echocardiographic equipment. It was ≤5 min in eight athletes (three echocardiograms were recorded during the last half hour of the race, after which the men kept running to complete the race) and 6 to 25 min in five (Nos. 4, 9, 6, 12, and 1). Seven athletes volunteered for addi-
tional echocardiographic measurements, which were made after about 16 to 18 hr of running and during recovery 2 to 3 days afterwards. During the breaks the interval between stopping the run and data collection was <5 min, whereas it was somewhat longer afterwards (subjects 1, 4, 9, and 12). Blood samples were obtained from those seven athletes volunteering for additional echocardiographic studies plus one before and during the race (at 6 hr intervals) and during recovery (1, 2, and 6 days afterwards). Each athlete was weighed without clothing before and immediately after the race, blood pressure was taken with a sphygmomanometer simultaneously with the echocardiogram, and a standard 12-lead electrocardiogram was recorded with the athlete in a supine position. A continuous Holter recording was obtained in one athlete (No. 1) during the 24 hr of the race.

**Conditions.** The athletes ran for 24 hr on a 400 m Novotan track in cool weather, starting at 6 p.m. The temperature ranged from 7° to 15° C, the sky was clear, humidity was from 50% to 60%, and the wind speed was from 1 to 7 m/sec. The athletes were allowed to choose their own speed and the number and duration of their breaks during the race. They usually kept running at a speed of 8 to 12 km/hr. Liberal fluid intake facilities were provided. The athletes drank mainly water and glucose solution (25 g/liter) containing sodium (9.9 mmol/liter), calcium (9.9 mmol/liter), potassium (0.3 mmol/liter), citrate (16.7 mmol/liter), phosphate (2.2 mmol/liter), and chloride (1.2 mmol/liter).

**Echocardiography.** The echocardiograms were recorded at the stadium (at a distance of 20 m from the track) with two-dimensional echocardiographic equipment (ATL MK 600). The serial M mode recordings were made with the athlete in a supine right anterior oblique position (30 degrees) with standard techniques used at our laboratory. The images were derived from the real-time two-dimensional image by locating a cursor across the regions of interest by means of conventional parasternal long-axis views. Care was taken to obtain the best simultaneous resolution of the septal and posterior wall echoes just below the mitral valve tips. Paper speeds of both 50 and 100 mm/sec were used for each recording. The echocardiograms were calibrated and digitized with a desk-top computer. To minimize the respiratory effect on the measurements, the reported values were expressed as means of at least five representative cardiac cycles or as means of up to 10 consecutive cardiac cycles if any variation in left ventricular dimensions was seen. All measurements were performed by the same investigator.

The left ventricular end-diastolic dimension (EDD), the posterior wall thickness, and the interventricular septal thickness were measured at the onset of the Q wave of the electrocardiogram, whereas the end-systolic dimension (ESD) was defined as the smallest left ventricular diameter. The stroke dimension was defined as the difference between the EDD and ESD. Fractional shortening was defined as (EDD − ESD) × 100/EDD, and mean velocity of circumferential fiber shortening (mean V_c) as (EDD − ESD)/EDD divided by the left ventricular ejection time, which was measured from a simultaneously recorded carotid pulse tracing. The left ventricular relative wall thickness (radius to thickness ratio; R/Th) was determined as (EDD/2)/Th, where Th is the posterior wall thickness. The product of systolic blood pressure and R/Th was used as an index of peak systolic wall stress (P-R/Th). The systolic blood pressure/ESD ratio was used as a noninvasive index of myocardial contractility.

**Blood samples.** Venous blood drawn from the forearm was stored in ice, centrifuged within 15 min and analyzed for hemoglobin, hematocrit, serum electrolytes, creatinine, uric acid and lactate dehydrogenase. Samples were frozen and stored at −20° C for enzyme, triglyceride, and free fatty acid analyses, all of which were performed with standard techniques. Statistical analysis. Analysis of variance for dependent observations (Friedman) was used to test the significance of changes during and after the race, and these were then further evaluated by the Wilcoxon test for paired differences. Standard formulas were used for the calculation of linear correlation coefficients. All group data were expressed as mean ± SD. Differences were considered significant at p < .05.
Results

Although no athlete needed medical treatment during or after the race, the runners complained of increasing muscle pain and fatigue of the lower extremities during the race. Three of them had to drop out because of muscle pain after running for 14 to 22 hr 15 min. The remaining 10 who kept running for 24 hr covered an average of 183 km (range 146 to 227), i.e., they maintained an average speed of about 8 km/hr. The body weight of the runners decreased by 4.2% (66.6 ± 7.1 to 63.8 ± 7.3 kg; p < .005), mainly within the first 6 hr (figure 1), their blood pressure was lower after the race, and resting heart rate was somewhat elevated (table 1). The prerace electrocardiograms showed inverted T waves (four cases), which disappeared after the race, early repolarization of the ST segment (five cases), incomplete right bundle branch block (two cases), a widened QRS complex (≥0.12 sec; three cases), and junctional rhythm (one case). No new electrocardiographic changes were seen after the race. The peak heart rates obtained in the participant with the Holter monitor were in the range of 120 to 140 beats/min during the race (figure 2). After the 24 hr run, most of the athletes had difficulty walking because of muscle pain and stiffness.

Echocardiography. The ultramarathon athletes had relatively large left ventricular EDDs and increases in both the posterior wall and interventricular septal thickness (table 2), but no athlete had evidence of asymmetric septal hypertrophy (defined as an interventricular septal/posterior wall thickness ratio ≥1.5), mitral valve prolapse, or regional wall motion abnormalities.

The EDD was reduced by 7% after the race (p < .005), whereas a more variable response in the ESD resulted in a slight but insignificant net increase (table 3). The stroke dimension and fractional shortening consequently decreased by 24% and 16%, respectively, the latter in proportion to ΔEDS (r = −.66; p < .05) but not to ΔEDDD (r = .22; NS). The decline in mean Vcf by 9% (p < .01) was proportional to the distance completed (r = −.69; p < .01). The systolic blood pressure/EDS ratio was reduced by 21% after the race (p < .005). Reduced wall stress was reflected in decreases in the left ventricular R/Th by 12% (p < .005) and in the product of systolic blood pressure and R/Th by 26% (350 ± 50 to 260 ± 50 mm Hg; p < .005).

The alterations in the left ventricular dimensions and ejection phase indexes became somewhat more marked toward the end of the race (figure 1), while only the decreases in EDD and fractional shortening between 16 to 18 and 24 hr were significant (p < .05). The altered left ventricular performance was not related to the loss in body weight (r = .12 for stroke

(number)
TABLE 2
Prerace echocardiographic data in the ultramarathon athletes (n = 13)

<table>
<thead>
<tr>
<th></th>
<th>EDD (mm)</th>
<th>ESD (mm)</th>
<th>FS (%)</th>
<th>VS (circ./sec)</th>
<th>LVPW (mm)</th>
<th>IVS (mm)</th>
<th>LAD (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ultramarathon athletes</td>
<td>53.9 ± 5.3</td>
<td>33.3 ± 4.5</td>
<td>38.2 ± 4.8</td>
<td>1.28 ± 0.21</td>
<td>10.7 ± 1.0</td>
<td>12.0 ± 2.1</td>
<td>37.7 ± 4.1</td>
</tr>
<tr>
<td>Normal values</td>
<td>48.8 ± 3.2</td>
<td>33.1 ± 3.4</td>
<td>32.2 ± 5.0</td>
<td>1.18 ± 0.24</td>
<td>8.5 ± 1.2</td>
<td>8.6 ± 1.5</td>
<td>31.5 ± 5.7</td>
</tr>
</tbody>
</table>

FS = fractional shortening; LVPW = left ventricular posterior wall thickness; IVS = interventricular septal thickness; LAD = left atrial diameter.

The data represent normal values reported previously from this laboratory in healthy sedentary men (n = 13). Statistical comparisons (Student’s t test): a p < .01 (athletes vs controls); b p < .001 (athletes vs controls).

dimension, .49 for fractional shortening, and .20 for mean VS). The changes in left ventricular dimensions and function were reversible, as measured 2 to 3 days after the race. The difference between the EDD before the race and that recorded 2 to 3 days afterwards was more than 1 mm in only one athlete.

Biochemical findings. The enzyme changes became more marked as the race continued (table 4). Immediately on completion of the race, total creatine kinase and its MB fraction were elevated more than 100-fold above the prerace level, whereas the MB percentage remained on average within normal limits (<4%), ranging from 0.9% to 6.0% during the race and from 1.0% to 2.8% immediately afterwards. Total creatine kinase and lactate dehydrogenase remained elevated for several days during recovery. No significant changes were seen in the hemoglobin, hematocrit, or serum electrolyte levels, whereas the serum creatinine and uric acid values were somewhat elevated on completion of the race (table 5). The elevation in free fatty acids was not related to the alterations observed in left ventricular performance.

Discussion

The distances completed by the best participants (more than 200 km) indicate that ultramarathon runners represent a unique group of athletes capable of exercising for very prolonged periods without rest. The effect of their training was reflected not only in relatively large EDDs and an increase in posterior wall thickness but also in a rapid postexercise decline in heart rate. Although the athletes appeared to have nearly a 60% increase in the left ventricular muscle mass over the control subjects, the R/Th ratio was identical to that reported by Roeske et al. in professional athletes and was within normal limits, suggesting an "appropriate" or physiologic form of cardiac hypertrophy. A primarily aerobic perform-

TABLE 3
Echocardiographic data before (B) and after (A) the 24 hr run

<table>
<thead>
<tr>
<th>Subject</th>
<th>EDD (mm)</th>
<th>ESD (mm)</th>
<th>SD (mm)</th>
<th>FS (%)</th>
<th>VS (circ./sec)</th>
<th>R/Th</th>
<th>SBP/ESD</th>
</tr>
</thead>
<tbody>
<tr>
<td>01</td>
<td>56</td>
<td>36</td>
<td>24</td>
<td>40</td>
<td>1.25</td>
<td>2.7</td>
<td>3.3</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>30</td>
<td>20</td>
<td>40</td>
<td>1.37</td>
<td>2.3</td>
<td>4.7</td>
</tr>
<tr>
<td></td>
<td>52</td>
<td>44</td>
<td>21</td>
<td>41</td>
<td>1.73</td>
<td>2.6</td>
<td>4.0</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>46</td>
<td>24</td>
<td>47</td>
<td>1.43</td>
<td>2.5</td>
<td>6.2</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>43</td>
<td>16</td>
<td>35</td>
<td>1.27</td>
<td>2.1</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>67</td>
<td>67</td>
<td>23</td>
<td>34</td>
<td>1.09</td>
<td>2.8</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>46</td>
<td>17</td>
<td>34</td>
<td>1.25</td>
<td>2.3</td>
<td>4.2</td>
</tr>
<tr>
<td></td>
<td>52</td>
<td>48</td>
<td>22</td>
<td>42</td>
<td>1.23</td>
<td>2.9</td>
<td>5.3</td>
</tr>
<tr>
<td></td>
<td>53</td>
<td>43</td>
<td>21</td>
<td>39</td>
<td>1.31</td>
<td>2.4</td>
<td>3.7</td>
</tr>
<tr>
<td></td>
<td>56</td>
<td>51</td>
<td>23</td>
<td>41</td>
<td>1.40</td>
<td>2.5</td>
<td>3.9</td>
</tr>
<tr>
<td></td>
<td>56</td>
<td>57</td>
<td>23</td>
<td>41</td>
<td>1.42</td>
<td>2.8</td>
<td>3.9</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>49</td>
<td>16</td>
<td>32</td>
<td>1.03</td>
<td>2.3</td>
<td>4.0</td>
</tr>
<tr>
<td></td>
<td>57</td>
<td>55</td>
<td>18</td>
<td>31</td>
<td>0.86</td>
<td>3.6</td>
<td>3.6</td>
</tr>
<tr>
<td>Mean</td>
<td>54</td>
<td>50</td>
<td>21</td>
<td>38</td>
<td>1.28</td>
<td>2.6</td>
<td>4.2</td>
</tr>
<tr>
<td>± SD</td>
<td>±5 ±7</td>
<td>±5 ±6</td>
<td>±2 ±2</td>
<td>±5 ±5</td>
<td>±0.21 ±0.16</td>
<td>±0.4</td>
<td>±0.9 ±0.6</td>
</tr>
</tbody>
</table>

p valueA <.005 NS <.005 <.005 <.01 <.005 <.005
% diff. −7% +3% −24% −16% −9% −12% −21%

SD = left ventricular stroke dimension; FS = left ventricular fractional shortening; SBP/ESD = the ratio of systolic blood pressure to ESD.

A Wilcoxon test for paired differences.
ance in the event was suggested, in accordance with previous findings,\textsuperscript{15} by the moderate running speed (8 km/hr on average) and the peak heart rate levels measured by the Holter monitor (120 to 140 beats/min), while the considerable total energy cost appears to have been met by oxidation of an equivalent of nearly 2 kg of fat.\textsuperscript{15} The reduction in body weight by 4.2% (2.8 kg on average) suggests that hypohydration was moderate, and even less than that reported after a marathon.\textsuperscript{16–18} Since the hemoglobin, hematocrit, and serum electrolyte levels were unchanged after the race, subsequent plasma volume reduction was probably limited.\textsuperscript{18–20}

The decrease in both fractional shortening (p < .005) and mean $V_{cf}$ (p < .01) by 16% and 9%, respectively, does not necessarily imply an altered inotropic state after the race, because these commonly used ejection phase indexes of myocardial contractility are also influenced by preload and afterload.\textsuperscript{21–24} Since the EDD was reduced by 7% after the race (p < .005) and the extent of left ventricular fiber shortening is directly related to the end-diastolic fiber length (preload),\textsuperscript{21, 25, 26} diminished fiber shortening by the Frank-Starling mechanism may have contributed to the decrease in fractional shortening. This conclusion was contradicted, however, by the lack of correlation between $\Delta$EDD (r = .22; NS) and the change in fractional shortening compared with that between the latter and $\Delta$ESD (r = -.66; p < .05). The decrease in EDD is probably directly related to the altered heart rate after the race (56 to 72 beats/min). Regression equations obtained by atrial pacing\textsuperscript{27–29} suggest that the difference in heart rate may account for about half of the $\Delta$EDD in this study.

Mean $V_{cf}$, on the other hand, is relatively insensitive to short-term alterations in preload but varies inversely with afterloading.\textsuperscript{21, 30} The reduction in the product of systolic blood pressure and R/Th by 26% after the race (p < .005) was suggestive of lower peak systolic wall stress.\textsuperscript{8} Although this means that the mean $V_{cf}$ was not useful for evaluating actual changes in the contractile state, the lower postrace $V_{cf}$ despite the decreased afterload suggests indirectly that depressed inotropy may have been responsible for the change in $V_{cf}$.\textsuperscript{30, 31} The lower R/Th observed in this study was most likely a result of lower systolic blood pressure, since this ratio can be altered by changes in systolic blood pressure.\textsuperscript{32}

End-systolic fiber length appears to be a direct function of afterload.\textsuperscript{9, 33, 34} A stronger argument in support of depressed contractility was thus the observation that lower systolic blood pressure (p < .005) did not result in a smaller ESD after the race. The end-systolic pressure-dimension ratio is independent of loading conditions and is primarily affected by contractile state.\textsuperscript{35}

### TABLE 4
Selected biochemical data before, during, and after the 24 hr run

<table>
<thead>
<tr>
<th>Sample (n = 8)</th>
<th>Creatine kinase</th>
<th></th>
<th>LDH</th>
<th>TRG</th>
<th>FFA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total (U/liter)</td>
<td>MB (U/liter)</td>
<td>MB (%)</td>
<td>(U/liter)</td>
<td>(mmol/liter)</td>
</tr>
<tr>
<td>Prerace</td>
<td>248 ± 154</td>
<td>4 ± 3</td>
<td>1.9 ± 2.1</td>
<td>390 ± 67</td>
<td>2.4 ± 1.2</td>
</tr>
<tr>
<td>During 6 hr</td>
<td>1003 ± 456</td>
<td>17 ± 11</td>
<td>1.7 ± 0.5</td>
<td>581 ± 94</td>
<td>1.1 ± 0.7</td>
</tr>
<tr>
<td>12 hr</td>
<td>7038 ± 8680</td>
<td>116 ± 81</td>
<td>1.5 ± 0.7</td>
<td>882 ± 333</td>
<td>0.9 ± 0.9</td>
</tr>
<tr>
<td>24 hr</td>
<td>27427 ± 24898</td>
<td>422 ± 571</td>
<td>1.5 ± 0.8</td>
<td>1732 ± 1128</td>
<td>0.2 ± 0.2</td>
</tr>
<tr>
<td>Recovery 1 day</td>
<td>10389 ± 11784</td>
<td>98 ± 143</td>
<td>0.9 ± 0.6</td>
<td>1319 ± 1243</td>
<td>1.0 ± 0.5</td>
</tr>
<tr>
<td>2 days</td>
<td>2931 ± 2334</td>
<td>39 ± 59</td>
<td>1.4 ± 1.5</td>
<td>1238 ± 1018</td>
<td>1.4 ± 0.5</td>
</tr>
<tr>
<td>6 days</td>
<td>534 ± 500</td>
<td>21 ± 35</td>
<td>2.3 ± 1.9</td>
<td>1029 ± 1040</td>
<td>1.8 ± 0.6</td>
</tr>
<tr>
<td>Normal values</td>
<td>&lt;120</td>
<td>&lt;5</td>
<td>&lt;4</td>
<td>120–400</td>
<td>0.4–1.7</td>
</tr>
</tbody>
</table>

LDH = lactate dehydrogenase; TRG = triglycerides; FFA = free fatty acids.

Values are given as mean ± SD.

### TABLE 5
Selected biochemical parameters before and after the 24 hr run

<table>
<thead>
<tr>
<th>Sample (n = 8)</th>
<th>Hb (g/liter)</th>
<th>Hct (%)</th>
<th>Na\textsuperscript{+} (mmol/liter)</th>
<th>K\textsuperscript{+} (mmol/liter)</th>
<th>Creatinine (µmol/liter)</th>
<th>Uric acid (µmol/liter)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prerace</td>
<td>137 ± 7</td>
<td>41 ± 2</td>
<td>142 ± 2</td>
<td>3.9 ± 0.3</td>
<td>108 ± 15</td>
<td>342 ± 45</td>
</tr>
<tr>
<td>Postrace</td>
<td>135 ± 7</td>
<td>39 ± 3</td>
<td>140 ± 3</td>
<td>4.2 ± 0.4</td>
<td>126 ± 18</td>
<td>407 ± 94</td>
</tr>
</tbody>
</table>

Values are given as mean ± SD. Normal values for creatinine were 60 to 115 µmol/liter and those for uric acid were 150 to 400 µmol/liter.
The reduction in the systolic blood pressure/ESD ratio by 21% (p < .005) was thus strongly suggestive of significantly depressed left ventricular contractile function after the 24 hr run. The potential limitations of this noninvasive ratio have been noted previously.36, 37

The observation that left ventricular function appeared to become somewhat more depressed during the last 6 to 8 hr of the race, together with the negative correlation between mean Vcf and the distance completed (r = −.69; p < .01), may suggest a direct association between altered left ventricular performance and the intensity of exercise. The changes in the ejection phase indexes, however, may well have been influenced by altered preload and afterload, as noted above. Depressed left ventricular function nevertheless appeared to be reversible, as observed during recovery 2 to 3 days after the race. The mechanism behind altered left ventricular function remains to be established. Elevated plasma free fatty acids together with reduced cardiac glycogen subsequent to prolonged exercise38, 39 have been postulated to result in impaired myocardial performance5, 38. However, the fourfold increase in free fatty acids in this study was apparently too low to be responsible for the altered left ventricular function.

To minimize test-to-test variation in the serial echocardiographic studies, strictly standard techniques from our laboratory were used and care was taken to use the same positions for the transducer and athlete in the serial recordings. The minimal variation in EDD before the race and during recovery, despite the different conditions, suggests in part that these findings were probably not influenced by test-to-test variation. In addition, the echocardiographic examination appears to be exceptionally successful in athletes in our experience. Geometrical error was unlikely because of symmetrical wall motion. The inevitable slight variation in the interval between the echocardiographic recording and the end of the race in some athletes did not seem to affect our results.

The more than 100-fold elevation in total creatine kinase, clearly exceeding the values reported after a classic marathon,40, 41 was indicative of skeletal muscle injury. Although the MB fraction was also markedly elevated after the race, except in two athletes, its increase was not associated with an abnormally high MB percentage of total creatine (≥4%). No electrocardiographic evidence of myocardial injury was seen after the race. This, together with previous findings,40, 42 suggests that the rise in the MB fraction is probably noncardiac in origin.

In conclusion, very strenuous prolonged exercise may result in reversible depressed left ventricular contractile function. This raises the possibility of cardiac fatigue. There may be a risk of manifest left ventricular failure under less favorable conditions, such as hot weather, particularly in athletes with latent minor cardiac abnormalities. Although the rationale behind ultramarathon running may be seriously questioned, thousands of runners are probably taking part in such events each year and great care should be taken in selecting the participants.

References
Evidence of impaired left ventricular performance after an uninterrupted competitive 24 hour run.
K O Niemelä, I J Palatsi, M J Ikäheimo, J T Takkunen and J J Vuori

_Circulation_. 1984;70:350-356
doi: 10.1161/01.CIR.70.3.350

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1984 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/70/3/350