Left Ventricular Dynamics During Exercise in Elite Marathon Runners

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To assess left ventricular structure and function at rest and during exercise in endurance athletes, 10 elite marathon runners, aged 28 to 37 years, and 10 matched nonathletes were studied by echocardiography and supine bicycle ergometry. Each athlete’s best marathon time was <2 h 16 min. Echocardiography was performed at rest, at a 60 W work load and at an individually adjusted work load, at which heart rate was 110 beats/min (physical working capacity 110 [PWC110]). Oxygen uptake at PWC110 averaged (±SD) 1.14 ± 0.2 liters/min in the nonathletes and 2.0 ± 0.2 liters/min in the runners (p < 0.001).

The left ventricular internal diameter at end-diastole was similar at the three activity levels in the control subjects but increased significantly from rest to exercise in the runners (p < 0.001). Left ventricular systolic meridional wall stress remained unchanged during exercise in the nonathletes but was significantly higher at PWC110 in the athletes (p < 0.05). Both the systolic peak velocity of posterior wall endocardial displacement and fractional shortening of the left ventricular internal diameter increased with exercise; at PWC110 the endocardial peak velocity was higher in the runners than in the control subjects (p < 0.01). The endocardial peak velocity during relaxation was comparable in athletes and control subjects at rest, increased similarly at a 60 W work load, but was higher in the runners at PWC110 (p < 0.05).

In conclusion, left ventricular dynamics during supine bicycle exercise differ between elite marathon runners and nonathletes; ventricular dilation, with preserved fractional shortening, and a faster relaxation may contribute to the development of a high stroke volume in the exercising athlete.

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The capacity to develop a high cardiac output is an indispensable factor for excellence in endurance sports, such as long-distance running and cycling. Because maximal heart rate is limited, endurance athletes must be able to develop a high stroke volume during exercise. This could be achieved either by a more complete emptying of the left ventricle than in nonathletes, or by a larger left ventricular end-diastolic dimension with normal or increased ejection fraction. The shortening of the diastolic period during exercise could impede left ventricular filling so that a faster relaxation of the ventricle would be beneficial to the athlete.

At rest, endurance athletes, in comparison with nonathletes, do have a larger left ventricular internal dimension with similar systolic and diastolic cardiac function (1–11). There are, however, limited and conflicting data on left ventricular dynamics in the exercising athlete. Some investigators (12–15) observed that exercise induces ventricular dilation in the athlete, whereas others (16–18) found that the larger left ventricular internal diameter at rest was simply maintained during exercise. Divergent results may be due to the selection of athletes and to methodologic differences. Systolic left ventricular function appears to be similar in the exercising athlete and nonathlete (13,15–18). In the only report (17) on diastolic function, left ventricular filling was enhanced in exercising runners as compared with control subjects.

We examined 10 elite marathon runners and 10 matched control subjects at rest, at a fixed submaximal work load and at a similar relative work load corresponding to a heart rate of 110 beats/min. Echocardiography was applied in subjects in the supine position, to study cardiac structure and systolic and diastolic left ventricular function.

Methods

Study subjects. Ten male marathon runners, aged 28 to 37 years, were studied. They had participated in athletic com-
petition for a mean 14 ± 5 (±SD) years. At the time of the investigation they ran 154 ± 12 km/week. Their best marathon time averaged 133 ± 1.3 min and was <2 h 16 min in all. Ten male nonathletes matched for age, height and weight were also studied. They were not involved in competitive sports, but some engaged in moderate recreational sports activities (0 h in four subjects, 1 to 2 h in five and 3 h in one subject). The study protocol was approved by the ethical committee of the Faculty of Medicine and the subjects gave informed consent.

**Preliminary study.** All subjects underwent a preliminary investigation, always in the morning, comprising a clinical examination with measurements of heart rate and blood pressure and a 12 lead electrocardiogram (ECG). They completed a questionnaire on past and present health status and on their sports activities. If there was no evidence of disease, echocardiography was performed with use of an Ultra-Imager (Honeywell) with photographic paper, followed by a symptom-limited bicycle exercise test in the supine position. After a 5 min rest period, work load was increased by 20 W every minute with measurements of heart rate from the ECG, blood pressure by the cuff method, and expiratory gas analysis.

**Experimental test.** At least 1 week after the preliminary test, the subjects came to the air-conditioned laboratory at 9 AM after a light breakfast at home. Height, weight and skinfold thicknesses (triceps, biceps, subscapular and suprailliac) were measured. Supine and standing blood pressures were taken in triplicate after the subjects had been resting in the recumbent position for 5 min in a quiet room. They then assumed the supine position on a bicycle ergometer table with shoulder support to limit torso motion and rested for 5 min. Heart rate, blood pressure (in triplicate) and respiratory variables were recorded during the next 3 min. Echocardiography of the left ventricle was then performed. The subjects then exercised at a work level of 60 W and, after 30 min rest, at the work load corresponding to a heart rate of 110 beats/min (physical working capacity 110; [PWC110]). This work load was determined from the preliminary test but, during the first 5 min of exercise, adjustments of work load were allowed to approximate the target heart rate as closely as possible. At both exercise levels, heart rate, blood pressure (in triplicate) and respiratory variables were measured between 5 and 8 min of exercise; echocardiography was then performed.

**Echocardiography.** For the definitive test, all echocardiograms were performed by the same investigator. The subjects were studied in a slight left lateral decubitus position, by tilting the ergometer table to 15°; further adjustments could be made using pillows. M-mode echocardiograms were obtained under two-dimensional guidance, and were recorded at end-expiration. The hand-held 3.5 MHz transducer was placed at the left sternal border. To study the left ventricle, the transducer was angled to ensure simultaneous visualization of the left-sided septal and posterior wall endocardium and epicardium, just below the mitral valve apparatus at the level of the posterior chordae tendineae. At each level of activity and for each measurement, two tracings with separate transducer placements were recorded. Good quality echocardiograms could be obtained at all three levels of activity in nine athletes and nine control subjects.

The echocardiograms were processed with use of a Houston Instrument HIPAD digitizing tablet connected to a Hewlett-Packard 1000 computer system (11). On each tracing three to five heartbeats were analyzed and averaged. The criteria of the American Society of Echocardiography (19) were applied: wall and cavity measurements were made by using the leading edge method, the beginning of the QRS complex was taken as end-diastole and the nadir of septal motion as end-systole. The left ventricular internal diameter and posterior wall thickness were measured at end-diastole (LVIDd;PWTd) and during systole (LVIDs; PWTs) and were expressed in millimeters. From these dimensions the fractional shortening of the left ventricular internal diameter (%) was calculated as 100 × (LVIDd - LVIDs)/LVIDd, left ventricular mass (g) as 0.0008 × 1.04 × (LVTDD3 - LVIDD3) + 0.6 g (20) and systolic left ventricular meridional wall stress (21) (mm Hg) as 0.334 × P × LVIDs/PWTs × (1 + PWTs/LVIDs), where P is the systolic blood pressure (average of three readings). In addition, the peak velocity (mm/s) of the posterior wall endocardial displacement was obtained during systole and during relaxation.

**Respiratory gas analysis.** With use of an open circuit method, expired air passed through a mixing box and was continuously analyzed for percent oxygen and carbon dioxide with an OM-11 oxygen analyzer and an LB-2 carbon dioxide analyzer (Beckman Instruments). Pulmonary ventilation was measured with a pneumotachograph (body temperature, pressure and saturated conditions). The pneumotachograph and the gas analyzers were calibrated before each experimental period. An on-line computer (Hewlett-Packard 1000) was used to calculate continuously oxygen uptake and carbon dioxide production (standard temperature, pressure and dry conditions) and the respiratory exchange ratio (carbon dioxide output/oxygen uptake). Data were stored for subsequent analysis.

**Statistical analysis.** Data are reported as mean values ± SD. The triplicate measurements of blood pressure and duplicate measurements of echocardiographic data were averaged. The characteristics of athletes and nonathletes were compared with use of the unpaired Student’s t test. Data at rest and during exercise were analyzed by multivariate repeated measures analysis of variance (22); between-subjects effects (athletes versus nonathletes) and within-subjects effects (level of activity and level by group interaction) are reported. With use of the contrast transformation, between-group differences at each level of effort were compared with the differences at rest. When of inter-
Table 1. General Characteristics of the Subjects

<table>
<thead>
<tr>
<th></th>
<th>Control Subjects (n = 10)</th>
<th>Marathon Runners (n = 10)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>29.4 ± 3.9</td>
<td>32.0 ± 2.9</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>64.4 ± 5.6</td>
<td>61.6 ± 4.1</td>
<td>NS</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.74 ± 0.06</td>
<td>1.73 ± 0.06</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Skinfold thickness</td>
<td>28 ± 7</td>
<td>18 ± 3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>(sum of 4; mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>65 ± 10</td>
<td>50 ± 9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Blood pressure (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>123 ± 11</td>
<td>121 ± 12</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic</td>
<td>76 ± 9</td>
<td>73 ± 7</td>
<td>NS</td>
</tr>
<tr>
<td>Physical working</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>capacity 130 (PWC130)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Watts</td>
<td>122 ± 29</td>
<td>211 ± 33</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Oxygen uptake (liters/min)</td>
<td>1.61 ± 0.39</td>
<td>2.72 ± 0.41</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are expressed as mean values ± SD. NS = not significant. Physical working capacity 130 = work load corresponding to a heart rate of 130 beats/min.

Results

General characteristics of the subjects (Table 1). The runners did not differ from the control subjects in age, height or weight. Skinfold thickness, however, was significantly less in the athletes (p < 0.001). Heart rate at rest was lower in the runners (p < 0.01); blood pressure was similar in both groups. At the preliminary supine bicycle exercise test, work load and oxygen uptake at a heart rate of 130 beats/min were significantly (p < 0.001) higher in the athletes.

Heart rate, blood pressure and oxygen uptake (Table 2). At the definitive test, heart rate was lower (p < 0.001) in the runners than in the control subjects at rest (46 ± 7 versus 60 ± 9 beats/min) and at 60 W work load (81 ± 8 versus 106 ± 9 beats/min). At the second work load, heart rate was close to the target heart rate of 110 beats/min in both groups and averaged 109 ± 9 beats/min in the nonathletes and 107 ± 4 beats/min in the athletes. This work load averaged 132 ± 18 W in the athletes and 62 ± 18 W in the control subjects (p < 0.001). Systolic and diastolic blood pressures were similar at rest, lower in the athletes at a 60 W work load and lower in the control subjects at physical working capacity 110 (PWC110). Oxygen uptake was not different between the groups at rest and at a 60 W work load. At PWC110, when the respiratory exchange ratio was similar in athletes (0.90 ± 0.05) and nonathletes (0.90 ± 0.06), oxygen uptake was significantly higher in the athletes.

Left ventricular structure. On the rest echocardiogram, left ventricular mass averaged 214 ± 45 g in the athletes and 146 ± 37 g in control subjects (p < 0.001). Figure 1 illustrates the significant difference in end-diastolic left ventricular internal diameter between the groups (F[1,16] = 8.1; p < 0.05) and the divergent response to exercise (F[2,15] = 8.8; p < 0.01); the between-group difference at PWC110 was significantly larger than the difference at rest (F[1,16] = 13; p < 0.01) and the same tendency was seen at a 60 W work load (F[1,16] = 3.6; p = 0.08). When the analysis was performed within each group, the end-diastolic dimension did not change in the control subjects (F[2,7] = 0.0; p = NS) but was significantly higher during exercise than at rest in the runners, both at a 60 W work load (F[1,8] = 5.8; p < 0.05) and at PWC110 (F[1,8] = 24; p = 0.001).

The end-systolic left ventricular internal dimension was also significantly different between the groups (F[1,16] = 11;

Table 2. Blood Pressure and Oxygen Uptake at Rest and Exercise

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>At Rest</th>
<th>60 W Work Load</th>
<th>PWC110</th>
<th>Between-Group Effects</th>
<th>Within-Subjects Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control subjects</td>
<td>122 ± 10</td>
<td>153 ± 16</td>
<td>144 ± 16</td>
<td>F_4(1,17) = 0.7, NS</td>
<td>F_4(2,16) = 71*</td>
</tr>
<tr>
<td>Athletes</td>
<td>116 ± 11</td>
<td>132 ± 15*</td>
<td>158 ± 16</td>
<td>F_4(2,16) = 11*</td>
<td></td>
</tr>
<tr>
<td>Diastolic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control subjects</td>
<td>77 ± 7</td>
<td>86 ± 10</td>
<td>81 ± 9</td>
<td>F_4(1,17) = 0.3, NS</td>
<td>F_4(2,16) = 9.04</td>
</tr>
<tr>
<td>Athletes</td>
<td>75 ± 6</td>
<td>79 ± 7</td>
<td>87 ± 6</td>
<td>F_4(2,16) = 8.1§</td>
<td>F_4(2,16) = 8.1§</td>
</tr>
<tr>
<td>Oxygen uptake (liters/min)</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control subjects</td>
<td>0.42 ± 0.07</td>
<td>1.16 ± 0.10</td>
<td>1.14 ± 0.20</td>
<td>F_4(1,15) = 32*</td>
<td>F_4(2,14) = 363*</td>
</tr>
<tr>
<td>Athletes</td>
<td>0.37 ± 0.08</td>
<td>1.14 ± 0.11</td>
<td>2.00 ± 0.24</td>
<td>F_4(2,14) = 33§</td>
<td></td>
</tr>
</tbody>
</table>

Values are expressed as mean values ± SD. F_4 = F ratio for the effect of group (athletes versus control subjects); F_4 = F ratio for the effect of level of activity; F_4 = F ratio for the interaction between level of activity and group. NS = not significant; PWC110 = physical working capacity at a heart rate of 110 beats/min. Significance of the F ratios: *p < 0.01; **p < 0.001. Significance of the between-group difference at 60 W work load or at PWC 110 with between-group difference at rest: *p < 0.05; **p < 0.01; ***p < 0.001.
The interactions between group and activity level was not significant (F[2,15] = 2.6; p = 0.10) but within-group analysis showed that the systolic diameter remained unchanged in the exercising athlete (F[2,7] = 0.7; p = NS) and was lower than at rest at a 60 W work load (F[1,8] = 41; p < 0.001) and at PWC$_{110}$ (F[1,8] = 13; p < 0.01) in the exercising nonathlete. In the control subjects left ventricular posterior wall thickness was similar at the three activity levels (9.0 ± 1.5; 8.8 ± 1.9; 8.2 ± 1.9 mm; F[2,7] = 1.2; p = NS), but wall thickness decreased significantly with exercise in the runners (11.1 ± 1.7; 10.2 ± 1.4; 9.9 ± 1.7; F[2,7] = 8.5, p = 0.01).

**Left ventricular function.** Systolic left ventricular meridional wall stress was slightly lower in the athletes than in the control subjects at rest. The between-group difference at a 60 W work load was not different from the between-group difference at rest (F[1,15] = 0.5; p = NS), but at physical working capacity 110 (PWC$_{110}$) a significant contrast with data obtained at rest was observed (F[1,15] = 6.8; p < 0.05) (Fig. 2). Within-group analysis showed that wall stress was not affected by exercise in the nonathletes (F[2,7] = 0.1; p = NS) but was significantly higher at PWC$_{110}$ than at rest in the runners (F[1,7] = 9.3; p < 0.05).

The fractional shortening of the left ventricular internal diameter was not significantly different between athletes and control subjects (F[1,16] = 0.8; p = NS), and it increased similarly with exercise in both groups (Fig. 3). To further explore the determinants of fractional shortening the changes from rest to exercise ($\Delta$FS) were related to the changes in left ventricular internal diameter, heartrate and wall stress in (stepwise) multiple regression analysis, including a dummy variable for group and the interactions of the independent variables with group. The following equations were obtained: a) at 60 W work load: $\Delta$FS = $-0.9 + 1.3 \Delta$ LVId (p < 0.001) − 1.31 $\Delta$ stress (p < 0.001) + 0.14 $\Delta$ HR (p < 0.05); b) at PWC$_{110}$: $\Delta$FS = 5.5 + 1.6 $\Delta$ LVId (p < 0.001) − 0.31 $\Delta$ stress (p < 0.001). An athletic history did not contribute significantly to the change of fractional shortening with exercise at any level of activity.

The peak velocity of displacement of posterior wall endocardium was significantly higher during exercise than at
The purpose of the present study was to assess left ventricular dynamics of elite endurance athletes during exercise, in comparison with such dynamics in nonathletic control subjects. Because of the higher exercise capacity of athletes, it is necessary to study athletes and nonathletes not only at the same absolute work load but also at a similar relative work load; therefore studies were performed at a 60 W work load and at a work load corresponding to a heart rate of 110 beats/min (PWC110).

**Figure 4.** Peak velocity of the posterior wall (PW) endocardial displacement during systole (left panel) and during diastole (right panel) in athletes (solid circle) and control subjects (open circle) at rest, at a 60 W work load and work load at a heart rate of 110 beats/min (PWC110). See text and Figure 1 for details on the statistical analysis.

rest both during systole and during diastole (Fig. 4). Overall there were no significant differences between the groups, but during systole there was a significant interaction between group and level of activity (F[2,14] = 6.7; p < 0.01). During diastole, the interaction between group and activity level was of borderline significance (F[2,14] = 3.3; p = 0.07). Comparison of the between-group differences at rest and exercise revealed significant contrasts at PWC110 both during systole (F[1,15] = 11; p < 0.01) and during diastole (F[1,15] = 5.8; p < 0.05).

**Discussion**

The purpose of the present study was to assess left ventricular dynamics of elite endurance athletes during exercise, in comparison with such dynamics in nonathletic control subjects. Because of the higher exercise capacity of athletes, it is necessary to study athletes and nonathletes not only at the same absolute work load but also at a similar relative work load; therefore studies were performed at a 60 W work load and at a work load corresponding to a heart rate of 110 beats/min (physical working capacity 110 [PWC110]).

**Left ventricular dimensions during exercise.** During moderate supine bicycle exercise, left ventricular internal dimension remained essentially unchanged in the control subjects. This is in agreement with other reports in which the cardiac response to supine or semisupine exercise of nonathletes was studied by echocardiography. Indeed, at heart rates between 100 and 125 beats/min the average change in left ventricular internal diameter as compared with values at rest was not significant in any report (17,18,23–30). During severe exercise, at heart rates >150 beats/min, left ventricular diameter increased in one study (+16%) (27) but not in others (29,30).

In the marathon runners, the echocardiographic end-diastolic left ventricular internal diameter was slightly higher than in control subjects at rest, and increased further during supine submaximal exercise. Both at a work load of 60 W and work load at a heart rate of 110 beats/min, end-diastolic dimension was larger than at rest. Similar results have been obtained when radionuclide angiography was applied during upright or semierect bicycle exercise. End-diastolic volume increased from rest to exercise in runners (13,14) and pentathletes (15), whereas it remained unchanged in control subjects (13,15) or increased less in less trained than in highly trained runners (14); however, other studies do not agree. In university level distance runners (17,18), left ventricular dimension on M-mode echocardiography at rest was larger than in control subjects, but the difference at rest simply persisted during supine exercise at one or more absolute work loads (18) or at a heart rate of 100 beats/min (17). Bar-Shlomo et al. (16) also observed that radionuclide end-diastolic volume remained unchanged when rowers of both genders exercised in the supine position; athletes, however, behaved differently from control subjects whose diastolic volume increased by 25% during exercise.

Divergent results of the various studies may be due to differences in selection of the athletes and to methodologic differences such as echocardiography versus radionuclide techniques, supine versus sitting exercise and differences in work load. We have demonstrated that elite marathon runners, whose best performance was <2 h 16 min, increased the echocardiographic left ventricular internal diameter during moderate supine bicycle exercise. This may at least partly reflect an augmented venous return and left ventricular filling volume per cardiac cycle, in view of the 24% lower heart rate in athletes than in control subjects at a 60 W work load and the 75% higher oxygen uptake, and presumably cardiac output, at a heart rate of 110 beats/min. The heart of these athletes has the capacity to utilize the Frank-Starling mechanism during effort.

**Comparison of athletes and nonathletes cannot distinguish between genetic differences and adaptations to physical training.** Studies in twins at rest (31) suggest that the heritability of cardiac size is limited but there are no data on the inheritance of the left ventricular response to exercise. Longitudinal studies (28,32,33) have shown that left ventricular structure at rest varies with training. Two studies (28,33) assessed left ventricular dynamics during exercise in nonathletes, before and after training, and found that the training-induced increase in left ventricular internal dimension at rest was simply maintained during exercise, with no further ventricular dilation. However, the characteristics of the training program of these sedentary subjects cannot be compared with those of the training regimens of competitive athletes. White et al. (34) trained pigs intensively, resulting...
in an increase of peak oxygen uptake from 49 to 67 ml/kg per
min, and found that physical training could alter the re-
sponse of the left ventricular internal dimension to exercise.

**Left ventricular systolic function during exercise.** In
agreement with previous reports, peak velocity of the pos-
terior wall endocardial displacement (17,27,30,35) and frac-
tional shortening of the left ventricular internal diameter (17,18,23-25,27-30) increased significantly with exercise.
This is mainly attributed to enhanced sympathetic stimula-
tion and increased contractile performance of cardiac muscle.
Whereas fractional shortening was not different between
runners and control subjects, both at rest and during exer-
cise, as previously observed (13,15-18), peak velocity of the
posterior wall endocardium responded differently to exercise
in the two groups. Indeed, at a heart rate of 110 beats/min,
endocardial peak velocity was higher in the athletes, which
can probably be explained by different loading conditions
of the heart in the exercising athlete and nonathlete. The
exercise-induced enlargement of the left ventricular end-
 diastolic internal diameter in the athlete increases fiber
length and therefore preload, which does not occur in the
nonathlete. This difference in preload may explain the higher
peak velocity of posterior wall endocardial displacement in
the exercising runner as compared with the nonathlete.
Fractional shortening of the left ventricular internal diameter
is preload- and afterload-dependent.

**Left ventricular systolic medional wall stress,** a measure
of afterload, did not change with exercise in the control
subjects but clearly increased when the runners exercised at
a heart rate of 110 beats/min. The higher systolic wall stress
may explain why the higher preload was not translated to a
greater fractional shortening in the athletes during effort as
compared with that in the nonathletes. After adjusting for
changes in left ventricular internal diameter, wall stress and
heart rate, the increase of fractional shortening from rest to
exercise was not different between athletes and control
subjects, suggesting that exercise-induced changes in intrin-
sic myocardial contractility are not different between ath-
letes and nonathletes.

**Left ventricular diastolic function during exercise.** At
a heart rate of 110 beats/min, the peak velocity of posterior
wall endocardial displacement during diastole was signifi-
cantly higher in the athletes than in nonathletes. This higher
rate level suggests that the left ventricle of runners relaxes
faster at an elevated heart rate, when the short diastolic
period may hamper left ventricular filling. This view is
compatible with the observation of improved relaxation in
conditioned rats and may be related to an increased capacity
for calcium transport in the sarcoplasmic reticulum (36).
This finding may also be related to a higher left ventricular
filling volume per heartbeat in the athletes. Matsuda et al.
(17) demonstrated, with digitized M-mode echocardiog-
raphy, that the contribution of early diastolic filling of the left
ventricle to global filling was greater in runners than in
nonathletes at a heart rate of 100 beats/min; in contrast to
our findings, they also observed enhanced relaxation in
runners at rest. Most studies, however, found normal left
ventricular diastolic function at rest in athletes (5,7,10,11).

**Conclusions.** Left ventricular dynamics during supine
bicycle exercise differ between elite marathon runners and
nonathletes; ventricular dilation, with preserved fractional
shortening and a faster relaxation, may contribute to the
development of a high stroke volume in the exercising
athlete.

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