Left Ventricular Function in Trained and Untrained Healthy Subjects

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SUMMARY Left ventricular function was compared in 18 normal sedentary controls (mean age 28 years, range 22–34 years) and nine endurance-trained athletes (mean age 19 years, range 15–25 years) at rest and during supine bicycle exercise. Gated radionuclide angiograms were performed at rest and at each level of graded maximal supine bicycle exercise. Heart rate, blood pressure, left ventricular ejection fraction and the relative changes in left ventricular end-diastolic and end-systolic volumes were assessed. Athletes attained a much greater work load than controls (mean 22.1 kpm/kg body weight vs 13 kpm/kg body weight). Both groups achieved similar increases in heart rate, blood pressure and ejection fractions. In the controls, the mean end-diastolic volume increased to 124% of that at rest (p < 0.02) during exercise and the mean end-systolic volume decreased to 81% of the rest level (p < 0.02). In contrast, the mean end-diastolic volume did not significantly change during exercise in the athletes, and the mean end-systolic volume decreased to 64% of rest (p < 0.05). Thus, although trained and untrained healthy subjects had similar increases in the left ventricular ejection fraction during exercise, different mechanisms were used to achieve these increases. Untrained subjects increased end-diastolic volumes, whereas trained subjects decreased the end-systolic volumes. The ability of athletes to exercise without increasing preload may be an effect of training and might have important implications in reducing myocardial oxygen demand during exercise.

ALTHOUGH the human left ventricular response to exercise has been the focus of many studies in the last several decades, the data on some aspects of this subject conflict. Until computer-assisted gated radionuclide cardiac angiography became available, accurate serial assessment of left ventricular volume at rest and during exercise was not technically possible.

To address the question of normal left ventricular volume and ejection fraction response to exercise, we studied 18 healthy untrained subjects at rest and during supine bicycle exercise. We also studied nine endurance-trained athletes to determine whether exercise training alters the left ventricular response to exercise.

Materials and Methods

Subjects

The study population consisted of 18 normal sedentary control subjects and nine endurance-trained athletes. The control group was made up of 14 males, mean age of 28 years (range 22–34 years). The athletes, seven males and two females, were scullers who had represented Canada in various international competitions and were involved in rigorous daily training. The daily training schedule included at least 4 hours of rowing, cycling and jogging, and each athlete had been in training for at least 3 years before the study. The mean age of the athletes was 19 years (range 15–25 years). No subject had a history of medical problems, and all subjects were normal by physical examination and standard resting ECG.

Exercise Protocol

After patients were placed supine on the exercise table, with feet positioned on the ergometer pedals, the baseline measurements of heart rate, blood pressure and multiple-lead ECG were taken. A gated blood pool radionuclide angiogram was then done as described below. The subjects performed graded supine bicycle exercise to the point of exhaustion, using a Quinton 845 ergometer, with increments of 300 kpm every 3 minutes. Blood pressure, heart rate, multilead ECG and radionuclide angiograms were repeated serially during each level of exercise.

Imaging Technique

Radionuclide angiography was done with a Ohio Nuclear Sigma Series 420 scintillation camera, using
a parallel-hole, general-purpose collimator and an Ohio Nuclear ECG gate. Data collection and storage were handled with a Digital Equipment Corporation 11/40 computer.

Subjects received 25 mg of Sn-PYP intravenously, followed by 25 mCi of \(^{99m}\text{Tc}\) for in vivo labeling of red blood cells. At rest, counts were collected in a histogram mode in a \(64 \times 64\) matrix, from 300 cardiac cycles divided into 20 equal frames per cycle. The 2-minute collection periods during exercise were divided into 16 equal frames per cycle. During exercise, counts were collected only during the last 2 minutes of each period to allow the heart rate and blood pressure to reach a steady state during the first minute.

Data Analysis
With user-written programs, left ventricular end-diastolic and end-systolic silhouettes and background region were manually outlined by an experienced operator. The ejection fraction was then calculated as

\[
\frac{\text{EDC(bc)} - \text{ESC(bc)}}{\text{EDC(bc)}}
\]

where \(\text{EDC(bc)}\) = background-corrected end-diastolic counts and \(\text{ESC(bc)}\) = background-corrected end-systolic counts.

As volume is proportional to the counts, relative changes in left ventricular end-diastolic and end-diastolic volumes from rest to exercise were calculated by comparing left ventricular count rates, after correction for background activity.

Statistical analysis of changes from rest to exercise was done with the two-tailed paired \(t\) test. Comparisons between the two groups were made with the two-tailed unpaired \(t\) test. Results are expressed as mean ± SD.

Results
The athletes achieved a greater maximal work load than the sedentary control subjects (22.1 ± 3 vs 13 ± 2 kpm/kg body weight, \(p \leq 0.001\)). The limiting symptom in most subjects was leg fatigue, although in three athletes the limiting factor was dyspnea at maximal work load. Table 1 shows the heart rate, blood pressure, left ventricular ejection fraction and left ventricular volume changes from rest to exercise for both groups. There was no significant difference in the mean resting heart rate between athletes and controls. Athletes achieved a mean maximal heart rate of 175 ± 11 beats/min; the control subjects achieved a mean maximal heart rate of 154 ± 19 beats/min (\(p < 0.01\)). The athletes reached 88 ± 6% of their predicted maximal heart rate, compared with only 80 ± 10% in control subjects (NS) (fig. 1).

The mean resting systolic blood pressure for the athletes was 113 ± 13 mm Hg and at peak exercise increased to 205 ± 22 mm Hg (\(p < 0.001\)). For the controls, the mean resting systolic blood pressure was 122 ± 15 mm Hg, which increased to 192 ± 34 mm Hg with exercise (\(p < 0.001\)). There was no significant difference in blood pressure between the athletes and the controls, either at rest or maximal work load (fig. 2).

The mean resting left ventricular ejection fraction was similar for both athletes and controls. The changes from rest to exercise in both groups were highly significant (\(p < 0.001\)), but no significant differences were observed between the athletes and controls at all levels of exercise. In the controls, the ejection fraction increased gradually, peaking at maximal work load; in the athletes, there were similar increments in ejection fraction to a work load of approximately 12 kpm/kg, at which point it plateaued to the peak work load (fig. 3).

The relative changes in left ventricular end-diastolic and end-systolic volumes from rest to each level of exercise are shown in figure 4. The athletes had no significant change in mean left ventricular end-diastolic volumes. However, the mean end-systolic volume decreased significantly during the first stage of exercise and continued to decrease linearly with exercise, reaching a plateau of 64 ± 15% at 12 kpm/kg body weight. In contrast, the mean left ventricular end-diastolic volume for the control group increased abruptly during the first stage of exercise and remained at this level throughout exercise. There was a slight decrease in left ventricular end-systolic volume for the control group, which became statistically significant only at the maximal work load, when the mean end-systolic volume had decreased to 81 ± 7% of that at rest (\(p < 0.02\)).

The differences in end-diastolic volume between athletes and controls were significant at each level of exercise (\(p \leq 0.001\) at 3 kpm/kg and \(p \leq 0.002\) at 12 kpm/kg). Differences in end-systolic volumes between the two groups were not statistically significant.

<table>
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<tr>
<th>Table 1. Summary of Results</th>
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<tr>
<td><strong>Heart rate</strong></td>
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<td>Controls vs athletes</td>
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Abbreviations: EF = left ventricular ejection fraction; EDV = left ventricular end-diastolic volume; ESV = left ventricular end-systolic volume.
Discussion

The main objectives of this study were to determine the left ventricular response to exercise in normal healthy subjects and to compare responses of trained and untrained subjects. The vigorous training schedule of the athletes was evidenced by the achievement of a considerably greater maximal work load than that attained by the untrained subjects.

Despite differences in training, there was no difference in the mean resting heart rate between the two groups. This finding probably reflects differences in anticipatory factors for the two groups. The control group was made up of hospital staff, who were familiar with the laboratory setup and were probably more relaxed, whereas the athletes regarded these tests as a challenge competition against other members of their group. During peak exercise, the athletes achieved a greater mean maximal heart rate ($p < 0.01$); this difference was probably related to differences in age and in motivation between the two groups, and is consistent with previous circulatory changes during exercise for athletes\(^2\) and for untrained subjects.\(^3\)\(^,\)\(^4\)

There was no difference in the left ventricular ejection fraction between athletes and controls at rest or during exercise. In both groups, the resting and exercise left ventricular ejection fractions were similar to those reported for normal subjects.\(^5\)\(^,\)\(^7\) Thus, exercise training in healthy subjects does not appear to alter the left ventricular ejection fraction response to exercise.

Of particular interest are the ventricular volumes during exercise. Although the exercise increase in cardiac output is related to increased heart rate and stroke volume,\(^2\)\(^,\)\(^8\)\(^-\)\(^10\) the actual mechanism responsible for the increase in stroke volume remains controversial. There are conflicting data in the literature regarding the validity of the Frank-Starling mechanism in the intact human heart and several studies have been conducted to clarify this point. Early studies designed to assess left ventricular dimensions at rest and during exercise used methods based on the geometric calculation of ventricular volumes. These included biplanar chest x-ray with ECG-triggered exposure,\(^11\) epicardial and midmyocardial silver-tantalum markers,\(^12\)\(^,\)\(^13\) dye dilution,\(^14\) contrast ventriculography,\(^15\) echocardiographic measurements\(^16\)\(^,\)\(^17\) and geometric calculation of left ventricular volume from ventricular silhouettes obtained during first-pass nuclear angiography.\(^18\) The conflicting data obtained

\[\text{BPM}\]

\[\text{Controls}\]

\[\text{Athletes}\]

\[\text{rest}\]

\[\text{exercise}\]

**Figure 1.** Heart rate at rest and during maximal work load (mean ± sd). BPM = beats per minute.

\[\text{rest}\]

\[\text{exercise}\]

**Figure 2.** Systolic blood pressure at rest and during maximal work load (mean ± sd).
LV FUNCTION IN HEALTHY SUBJECTS/Bar-Shlomo et al.

From these studies very likely reflect the limitations of geometric assessment of ventricular volume as well as patient population differences, type and intensity of exercise, and methods used to outline the ventricular silhouette.

The use of nongeometric methods to assess ventricular volume would eliminate the need for certain major assumptions and circumvent potential errors. Several attempts to assess this, using gated nuclear angiography, have been made. This method is based on the assumption that the left ventricular count rate is linearly related to left ventricular volume, and that if one can accurately assess background activity and tracer decay, accurate rest-to-exercise comparisons can be made. Several investigators have calculated absolute ventricular volume by comparing ventricular activity with that of a known volume of peripheral venous blood, either ignoring or correcting for tissue attenuation. However, the potential error in attenuation correction makes comparisons between subjects difficult. In this study, we compared rest and exercise left ventricular count rates without attempting to calculate absolute volumes. Each patient was used as his own control, which eliminates the potential error of tissue attenuation. A similar approach was taken by Sorensen et al., who validated their calculation of background-corrected stroke counts against a Fick cardiac output determination, both at rest and during exercise. They found good correlation between these methods at rest and at all levels of exercise.

In this study, left ventricular end-diastolic volume increased during exercise in normal untrained subjects. This finding conflicts with studies that have shown no change or a decrease in left ventricular end-diastolic volumes in normal healthy subjects during supine exercise. However, our results are in accord with those of Erickson et al., who studied dogs by echocardiography, and with results from healthy human volunteers studied by echocardiography, first-pass nuclear angiography and gated nuclear angiography.

Left ventricular end-systolic volume decreased in the untrained subjects in the present study, but the decrease became statistically significant only at peak exercise. Most studies show that end-systolic volume decreases with exercise in normal subjects.

The trained subjects had a different volume response to supine exercise. They did not increase ventricular volume during exercise, but rather emptied their ventricles more completely at end-systole, to achieve similar increases in the left ventricular ejection fraction. These results are consistent with earlier studies that used x-ray measurement of total heart volume in trained athletes during exercise. However, these were not measurements of left ventricular volume. In a study of 18 athletes before and after intensive training, Rerych et al. measured left ventricular end-diastolic volume using a geometric calculation from the ventricular silhouette obtained by first-pass radionuclide angiography. Left ventricular end-diastolic volume was increased with exercise, as in our normal controls. This discrepancy may reflect the differences between our count-related method for assessing ventricular volume and the silhouette method. The subjects of Rerych et al. were studied in the upright position, which may have had a postural effect in reducing left ventricular volumes more than during supine exercise.

The differences between athletes and controls in the

![Figure 3](http://circ.ahajournals.org/doi/figure-pdf/10.1161/01.CIR.82.1.487)

**Figure 3.** Left ventricular ejection fraction (EF) changes during exercise (mean ± sd). The solid line represents the athletes and the broken line the controls.

![Figure 4](http://circ.ahajournals.org/doi/figure-pdf/10.1161/01.CIR.82.1.487)

**Figure 4.** Left ventricular end-systolic (ESV) and end-diastolic volume (EDV) changes during exercise (mean ± sd). The solid line represents the athletes and the broken line the controls.
left ventricular volume response to exercise may reflect the effect of training, but it is difficult to separate the effect of training on the heart from its effect on the peripheral circulation. For example, one effect may be to reduce peripheral vascular resistance during exercise, allowing greater left ventricular systolic emptying.

There may also be important central effects, and several reports document the fact that the hearts of athletes are relatively large. It is possible that the left ventricles of athletes are maximally dilated in the supine position at rest, whereas those of the controls are not. The hypertrophied hearts of athletes may also have decreased diastolic compliance, which prevents left ventricular end-diastolic dilatation. Finally, athletes may have increased left ventricular contractility, which could account for the lower end-systolic left ventricular volumes during exercise.

Whatever the mechanism, trained subjects do not rely on the Frank-Starling mechanism during exercise. This has important implications in that the mechanism of increasing stroke volume without increasing left ventricular volume at end-diastole is less expensive in terms of energy consumption, and probably represents an effect of training.

Further studies are needed to clarify this effect by assessing left ventricular performance before and after rigorous training in the same subjects, especially patients with coronary artery disease, in whom reduction of myocardial oxygen consumption during exercise would be crucial.

References

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