Noninvasive Evaluation of Exercise Training in College-age Men

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SUMMARY The purpose of this study was to assess noninvasively the effects of intense aerobic training on cardiac structure and function in a group of healthy, college-age men (25 experimental and 11 control, mean age 22 years). Echocardiographic, electrocardiographic (ECG), and fitness measurements were obtained before and after a 3-month endurance training program and compared with similar measurements obtained in nonexercising subjects. The supervised training program consisted of 50-minute jogging sessions 5 days a week at 85% of maximal heart rate. Compared with the control group, echocardiography after training showed an increase in left ventricular (LV) end-diastolic dimension (p < 0.05). LV posterior wall thickness, septal wall thickness and ejection fraction did not change significantly. ECG measurements revealed a decrease in resting heart rate (p < 0.05) and an increase in R-wave voltage in leads V₅ and V₆ (p < 0.01). The measured maximal oxygen consumption increased by 16% (p < 0.001). These data indicate that intense aerobic training in college-age men results in a significant increase in resting LV end-diastolic dimension and volume. The increase in maximal stroke volume associated with exercise training may be partially explained by these changes in cardiac dimensions.

RECENT STUDIES of the physiologic effects of exercise training indicate important differences between endurance athletes and untrained normal subjects.¹ Eight of the more noticeable differences, especially in younger individuals, is an increase in heart size — the so-called athlete's heart. A physiologically enlarged heart enables endurance athletes to generate a stroke volume nearly twice that of sedentary normal subjects of comparable age.¹ The increased stroke volume in turn enables the athlete to transport more oxygen to the exercising muscle cells and results in higher levels of aerobic work.

The cause of this difference in heart size is not known. Do endurance athletes inherit their large, strong hearts, or does the stress of aerobic training over months and years lead to an increase in cardiac size? Until noninvasive techniques were developed to study cardiac size and function, these questions were difficult to answer. Echocardiography, however, allows quantitative assessment of cardiac structure and function without risk or discomfort to the patient. As a result, echocardiographic studies of subjects undergoing exercise training are being published.¹¹⁻¹⁴

The purpose of this study was to assess noninvasively the effects of intense aerobic training on cardiac structure and function in healthy, college-age men. Echocardiographic, electrocardiographic and fitness measurements were obtained before and after a 3-month endurance training program and compared with similar measurements in control subjects.

Methods

College students who were 18–25 years old were solicited to participate in an endurance training program. Participation in the study required that the subject had not engaged in an organized endurance training program for an extended period of time (greater than 3 months) during the past 5 years. Names of all volunteers who met these requirements were pooled and randomly assigned to two groups. Twenty-five subjects in the experimental group participated in the supervised training program and 11 subjects in the control group did not. The mean age for both groups was 22 years (range 19–24 years).

Each subject in the experimental group underwent a maximal oxygen consumption (VO₂ max) treadmill test, a resting 12-lead ECG, an M-mode echocardiogram and a hydrostatic weighing test before and after an 11-week training program. The control subjects received similar tests 11 weeks apart. The supervised endurance training program consisted of 50-minute jogging sessions 5 days per week at 85% of maximal heart rate. Subjects who developed lower-extremity problems while running continued to train on a bicycle ergometer adjusted in tension to maintain the prescribed heart rate. A daily log was kept of the exercise subject's training progress in terms of exercise heart rate, duration and distance covered during the exercise training sessions. Attendance at 85% of the exercise sessions was required for inclusion in the experimental group. Logs were also kept for each control subject that indicated the type and duration of any exercise engaged in during the 11-week period between tests.

The treadmill test protocol called for a constant speed of 6 mph with increasing grades of 4% every 2 minutes for the first 4 minutes, and 2% every 2 minutes thereafter until the subject reached exhaustion. Subjects were not allowed to use handrails.Expired air was continuously analyzed for percentages of oxygen and carbon dioxide using a Beckman OM-11 O₂ analyzer and a Beckman LB-2 CO₂ analyzer. Expired volume was measured using a Parkinson Cowan

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high-speed gasometer (Dynascience Corp.) and corrected to STPD. Expired volumes and percentages of oxygen and carbon dioxide were recorded on a strip-chart recorder during the treadmill test. These data were used to compute oxygen consumption, carbon dioxide production and respiratory quotients (R) at each level of work. The maximal heart rate was determined from the ECG using a CM-5 electrode position and a Quinton Model 740 ECG computer. Subjects were hydrostatically weighed as described by Luft to determine body density and percentage of body fat.

A standard 12-lead resting ECG was recorded on a Siemens 3170 four-channel electrocardiograph. The chest electrodes were placed with great care to ensure consistency of the precordial lead locations. ECG recordings were taken at end-tidal volume at paper speeds of 25 and 100 mm/sec for each lead. Intervals, durations, QRS voltages and axes were carefully measured with calipers.

Echocardiography was performed in a supine and slight right anterior oblique position with a commercially available ultrasonic unit (Primus, Rohe Scientific Corporation) using a 3.5-MHz transducer with a long-interval focus. Recordings were made using a fiberoptic recorder (Honeywell, LS-6). All measurements were recorded at end-tidal volume using standards developed by the American Society for Echocardiography. A lead II ECG was simultaneously recorded. The protocol followed during the echocardiographic examination has been described. Pulse waves and heart sounds were monitored and recorded at the beginning of each echocardiographic study using a Hewlett Packard 2150D pulse-wave pickup and a Hewlett Packard 2150 A/B contact sensor. After the echocardiographic examination, a well-defined segment (at least four cardiac cycles) of the left ventricular (LV) recording, with the superimposed ECG, was placed on a Graf/Pen (Science Accessory Corporation) sonic digitizer table for manual tracing. The Graf/Pen was interfaced to a Control Data 3300 computer for on-line analysis of the echocardiographic data. A Beehive Electrotech Alpha (BEA) 20 computer terminal provided the interface between the operator and the computer. A Tektronix 601 storage monitor was interfaced to the computer to display alphanumeric messages and graphs. The computer program written for retrieving data from the Graf/Pen could be run by the operator through the BEA terminal. Using the Graf/Pen cursor, the technician entered calibration points from the echocardiogram with regard to time, distance, placement, and onset of the QRS complex (fiducial point). Echocardiographic curves and electrocardiographic signals were then traced by moving the cursor along the leading edge of each relevant echocardiographic interface. Data analysis from this point was totally automatic.

The computer analysis of the echocardiograms was done on each successive heart beat, and the values were then averaged. Traced echocardiographic data were retrieved from the computer disk to be plotted on an Omnigraphic X-Y recorder (Houston Instruments, model 6650) and stored on magnetic tape. Using the digitizer interfaced to the computer made the M-mode echocardiographic analysis a totally automated process and allowed for continuous measurement and calculation of LV variables and dimensional changes over several cardiac cycles. Figure 1 shows an echocardiogram and the computer-plotted tracing.

The following echocardiographic measurements were obtained using this automated system: (1) LV end-diastolic (LVED) and end-systolic (LVES) dimensions, measured at the onset of the QRS complex (F1) and at the point of maximum excursion of the LV posterior wall (F2), respectively; (2) the point of greatest and least dimension between the endocardial surfaces of the interventricular septum and the LV posterobasal wall, measured for maximal and minimal LV dimensions; (3) LV posterobasal wall (LVPW) thickness, measured as the distance between LV endocardium and epicardium at F1 and F2. Based on the
above measurements, the percent shortening of the LV internal diameter was calculated as \((LVDd - LVDs)/LVDd \times 100\).  

Two methods were used to calculate LV volumes.\(^{(18,19)}\) Gibson's regression equation \((LAd = 0.98 \times LVDd + 5.90)\), and \(LAs = 1.14 \times LVDs + 4.18\), where \(d\) and \(s\) are end-diastole and end-systole, respectively) and the hyperbolic equation of Teichholz et al. \((LAd \text{ or } LAs = (7.0/2.4 + D) D^2\), where \(D\) is the dimension measured at LVDd or LVDs) were used to determine the LV long axis. The calculated value of the LV long axis was then substituted into the ellipsoid formula\(^{(20)}\) to derive LV volumes, including:

1. stroke volume (SV), calculated as the difference between end-diastolic and end-systolic LV volumes;
2. LV wall volume (WV), calculated at \(d\) and \(s\); and ejection fraction (EF), measured as SV divided by LV volume at end-diastole. Equations used for LV WV determination were:

\[
WVd = \frac{\pi}{6} (LVDd + LVPWd + IVSd)^2 \\
(0.98 (LVDd + IVSd) + 5.90) - Vd. \quad (1)
\]

\[
WVs = \frac{\pi}{6} (LVDs + LVPWs + IVSs)^2 \\
(1.14 [LVDs + IVSs] - 4.18) - Vs. \quad (2)
\]

\[
WV(d \text{ or } s) = \frac{\pi}{6} (LVD + LVPW + IVS)^2 \\
(13.33 [LVD + IVS]/LVD + IVS + 2.4) - V. \quad (3)
\]

Equations 1 and 2 were described by Gibson\(^{(18)}\) and equation 3 was described by Teichholz et al.\(^{(19)}\) where \(V = LV\) chamber volume. The Teichholz formula is used for both end-systolic and end-diastolic measurements.

All ECGs were analyzed by one cardiologist and echocardiographic tracings were made by one experienced technician. Random numbers were assigned to each subject's chart to ensure that the cardiologist and technician would have no knowledge of subject status or test sequence. All measurements of pre- and post-test data were performed after the training program.

Echocardiographic, ECG, VO\(_2\) max and hydrostatic weighing tests before and after training were compared to determine cardiac and fitness changes. These data were statistically analyzed using analysis of variance.

**Results**

Twenty-two of the 25 exercise subjects successfully completed the 11-week training program. One subject chose to discontinue participation in the study 3 weeks into the program and two subjects did not attain the required 85% attendance at the supervised exercise sessions. Two of the 11 control subjects did not maintain their normal lifestyle: One subject engaged in a heavy weight lifting program and the other began a regular bicycle exercise program. The three exercise subjects and the two control subjects who did not satisfactorily complete the study were not considered in the statistical analysis.

No subject had evidence of any cardiac disease, based on an evaluation that included a resting ECG and a treadmill exercise test. Echocardiography, however, showed that two subjects (one experimental and one control) had bicuspid aortic valves without clinical aortic stenosis. Both subjects successfully completed the study without complication. No subject had mitral valve prolapse.

**Fitness Changes**

Figure 2 shows weekly averages of the distance run by the exercise group each training session, the pace during the session, and the average cumulative distance during the exercise training program. The total average distance covered by the exercise participants during the 11 weeks of training was 243.1 miles. The distance represented an average of 22.1 miles per week and 4.4 miles per session. The average attendance of the experimental group at the supervised exercise sessions was 92.2%. The daily recorded exercise heart rates of the experimental group averaged 90% of their maximal heart rates. Table 1 represents the changes in VO\(_2\) max and body composition for both the experimental and control groups. Compared with the controls, the exercise group had a 16% increase in VO\(_2\) max (48.57 to 56.19 ml/kg/min\(^{-1}\)) \((p < 0.001)\) and a significant decrease in body weight (176.5 to 170.6 lbs) \((p < 0.01)\) and percentage of body fat (17.22% to 13.7%) \((p < 0.001)\). The percentage of body fat in the exercise group decreased by 20% after the 11 weeks of exercise training. Figure 3 shows VO\(_2\) max for the control and experimental groups before and after training.

![Figure 2. Average distance of exercise group per training session (circles), average pace per session (arrows), and the average cumulative distance (stars) during the 11-week exercise training program.](image-url)
Table 1. Body Composition Measurements and Maximal Oxygen Consumption Values Before and After Exercise

<table>
<thead>
<tr>
<th>Exercise group</th>
<th>Post</th>
<th>Gain</th>
<th>Control group</th>
<th>Post</th>
<th>Gain</th>
<th>Anova</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (lbs)</td>
<td>176.50</td>
<td>-6.90</td>
<td>167.20</td>
<td>0.70</td>
<td>p &lt; 0.01</td>
<td></td>
</tr>
<tr>
<td>± 27.90</td>
<td>± 5.80</td>
<td>3.85</td>
<td>± 5.60</td>
<td>± 2.30</td>
<td>± 4.90</td>
<td>p &lt; 0.01</td>
</tr>
<tr>
<td>± 5.80</td>
<td>± 3.50</td>
<td>± 0.55</td>
<td>± 0.54</td>
<td>± 0.31</td>
<td>± 0.21</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>± 6.02</td>
<td>± 3.78</td>
<td>4.30</td>
<td>± 4.50</td>
<td>± 2.10</td>
<td>± 2.76</td>
<td>p &lt; 0.0001</td>
</tr>
</tbody>
</table>

Values are mean ± SD.
Abbreviations: VO₂ max = maximal oxygen consumption; Anova = analysis of variance.

Cardiac Adaptation

The echocardiographic measurements, echocardiographic volume calculations and electrocardiograph measurements before and after training are listed in tables 2, 3 and 4. One control subject had an echocardiogram that was not adequate for measurement using the protocol previously described, and one experimental subject had a lead error in his pretraining ECG.

In the experimental group, the echocardiographic LVDd measured at the onset of the QRS complex increased 3.86 mm during the 11-week training period (p < 0.001). Compared with the controls, the training group showed a significant increase in LVDd (p < 0.05); the control group had no significant increases in LVDd (fig. 4). There was no significant change in LVDs (32.29 to 33.53 mm for the training group and 35.20 to 35.99 mm for the control group). LV posterobasal wall thickness measured at end-diastole (QRS onset) and at end-systole (maximal excursion of the posterior wall) showed no change among the experimental group compared with the control group. The septal wall thickness measured at end-diastole and end-systole and the percentage of fiber shortening did not change significantly in either group.

The two methods used for volume calculations varied considerably in absolute values, but the significance of LV volume change was identical for both methods (table 3). After the 11-week exercise program, there was a significant increase in LV end-diastolic volume (p < 0.05) and stroke volume (p < 0.05) in the experimental subjects compared with controls. However, LV end-systolic volumes, LV wall volumes at end-systole and end-diastole, and LV ejection fraction did not change significantly.

Table 4 lists several electrocardiographic variables that changed significantly in the exercise group compared with the controls. As expected, there was a significant decrease in resting heart rate in the experimental group. In addition, the precordial lead R-wave amplitudes were significantly increased in the exercise subjects compared with the controls. This was seen in leads V₅ and V₆ and in the ECG lead that showed the maximal R-wave amplitude. No significant changes in either group were noted in the PR interval, QRS duration, corrected QT interval, or frontal-plane QRS axis.

Discussion

Changes in Maximal Oxygen Consumption

The VO₂ max is widely accepted as the index of cardiovascular functional capacity. Endurance training has been shown to result in significant increases in VO₂ max. Aside from variations in the training...
program (intensity, duration and frequency), the degree of change in \( \dot{V}O_2 \) max depends largely on the initial functional capacity. In subjects who were seden-tary before training, daily endurance training for 3–6 months has resulted in approximately 33% increase in \( \dot{V}O_2 \) max. When normally active subjects were trained, however, increases in \( \dot{V}O_2 \) max of only 15% were found; little or no increase in \( \dot{V}O_2 \) max was found when subjects were already very active before formal training. With one exception, all of the subjects in this study were considered normally active (healthy but uncon-

<table>
<thead>
<tr>
<th>Exercise group</th>
<th>Control group</th>
<th>Anova</th>
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</thead>
<tbody>
<tr>
<td>LV end-diastolic dimension (mm)</td>
<td>45.75 ± 4.14</td>
<td>48.45 ± 4.80</td>
</tr>
<tr>
<td>LV end-systolic dimension (mm)</td>
<td>32.29 ± 3.98</td>
<td>35.20 ± 4.48</td>
</tr>
<tr>
<td>Septal wall thickness at ED (mm)</td>
<td>10.66 ± 1.83</td>
<td>10.65 ± 1.40</td>
</tr>
<tr>
<td>Septal wall thickness at ES (mm)</td>
<td>13.82 ± 1.88</td>
<td>13.41 ± 1.41</td>
</tr>
<tr>
<td>LVPW thickness at ED (mm)</td>
<td>10.88 ± 1.90</td>
<td>9.70 ± 2.67</td>
</tr>
<tr>
<td>LVPW thickness at ES (mm)</td>
<td>16.35 ± 2.22</td>
<td>14.91 ± 2.75</td>
</tr>
<tr>
<td>Fiber shortening (%)</td>
<td>29.50 ± 5.1</td>
<td>27.39 ± 4.48</td>
</tr>
</tbody>
</table>

Values are mean ± sd.

Abbreviations: LV = left ventricular; ED = end-diastole; ES = end-systole; LVPW = LV posterobasal wall; Anova = analysis of variance.

### TABLE 3. Left Ventricular Volumes Before and After Exercise Training

<table>
<thead>
<tr>
<th>Exercise group</th>
<th>Control group</th>
<th>Anova</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV wall vol at ED (ml)</td>
<td>158.94 ± 32.41</td>
<td>157.96 ± 30.25</td>
</tr>
<tr>
<td>LV vol at ED (ml)</td>
<td>115.59 ± 25.07</td>
<td>133.11 ± 32.42</td>
</tr>
<tr>
<td>LV wall vol at ES (ml)</td>
<td>149.10 ± 26.72</td>
<td>150.61 ± 27.21</td>
</tr>
<tr>
<td>LV vol at ES (ml)</td>
<td>44.19 ± 12.60</td>
<td>54.69 ± 17.81</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>74.60 ± 17.02</td>
<td>78.40 ± 18.93</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>61.95 ± 6.59</td>
<td>59.13 ± 6.33</td>
</tr>
<tr>
<td>Teichholz volumes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV wall vol at ED (ml)</td>
<td>137.70 ± 27.82</td>
<td>136.21 ± 26.42</td>
</tr>
<tr>
<td>LV vol at ED (ml)</td>
<td>97.04 ± 20.29</td>
<td>111.01 ± 25.75</td>
</tr>
<tr>
<td>LV wall vol at ES (ml)</td>
<td>145.45 ± 26.05</td>
<td>146.33 ± 26.12</td>
</tr>
<tr>
<td>LV vol at ES (ml)</td>
<td>42.80 ± 11.98</td>
<td>52.61 ± 16.44</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>54.24 ± 13.03</td>
<td>58.40 ± 13.81</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>56.14 ± 7.57</td>
<td>52.89 ± 7.05</td>
</tr>
</tbody>
</table>

Values are mean ± sd.

Abbreviations: see table 2.
Table 4. ECG Measurements Before and After Exercise Training

<table>
<thead>
<tr>
<th>Exercise group</th>
<th>Control group</th>
<th>Anova</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>63</td>
<td>54</td>
</tr>
<tr>
<td>$\pm 10$</td>
<td>$\pm 8$</td>
<td>$\pm 8$</td>
</tr>
<tr>
<td>$R_{\text{vO}_2}$ (mV)</td>
<td>1.72</td>
<td>1.99</td>
</tr>
<tr>
<td>$\pm 0.52$</td>
<td>$\pm 0.48$</td>
<td>$\pm 0.21$</td>
</tr>
<tr>
<td>$R_{\text{vO}_2}$ (mV)</td>
<td>1.31</td>
<td>1.48</td>
</tr>
<tr>
<td>$\pm 0.34$</td>
<td>$\pm 0.33$</td>
<td>$\pm 0.17$</td>
</tr>
<tr>
<td>$R_{\text{max}}$ (mV)</td>
<td>1.78</td>
<td>2.06</td>
</tr>
<tr>
<td>$\pm 0.56$</td>
<td>$\pm 0.48$</td>
<td>$\pm 0.27$</td>
</tr>
</tbody>
</table>

Values are mean ± SD.
Abbreviation: Anova = analysis of variance.

ditioned) young men before training. An average increase in $\text{VO}_2$ max of 15% would be expected in such subjects. In the present study, the average increase in $\text{VO}_2$ max was 16%. The one exercise subject who was quite sedentary before training increased his $\text{VO}_2$ max by 36%.

As expected, the control subjects showed no significant changes in $\text{VO}_2$ max during the 11-week period of the study (a decrease from 55.67 to 54.60 ml/kg/min). The control group had a significantly higher initial $\text{VO}_2$ max. Consequently, their opportunity for increase in $\text{VO}_2$ max would be less than that of the exercise group. However, the control group had a decrease in their measured $\text{VO}_2$ max at the end of the study. The most likely reasons for a higher pretraining $\text{VO}_2$ max among the control group were that there were only half as many control subjects as experimental, and subjects were randomly assigned to groups before initial exercise testing.

Mechanisms of Increased Maximal Oxygen Consumption

In terms of the Fick equation, $\text{VO}_2$ can be expressed as:

$$(\text{SV} \times \text{HR}) \times (\text{A-V O}_2)$$

where SV = stroke volume, HR = heart rate, and A-V O$_2$ is systemic arteriovenous oxygen difference. Increases in $\text{VO}_2$ max after endurance training, as seen in this study, can be due to increases in maximal cardiac output (maximal SV × maximal HR), widening of the systemic A-V O$_2$ or a combination of these factors. Previous studies in young male subjects have shown that the increase in $\text{VO}_2$ max with training is produced by equal increases in both the maximal cardiac output and the maximal A-V O$_2$.²⁷,²⁸

Although A-V O$_2$ was not measured in our study, a significant portion of the increase in $\text{VO}_2$ max was probably a result of an increase in A-V O$_2$. The most probable mechanism for the A-V O$_2$ increase is an increased extraction of oxygen by the working muscles. Holloszy²⁸ showed that endurance training can increase the oxidative enzymes of the Krebs cycle and the mitochondrial respiratory chain, resulting in an increased ability for the muscle to use oxygen. Electron microscopy of human skeletal muscle has shown an increase in the number and size of mitochondria after physical training.²⁷,²⁸

Since maximal heart rate is primarily determined by age and is unlikely to change during an 11-week training program, the increase in cardiac output is due entirely to an increase in stroke volume. Increases in maximal stroke volume may be accomplished by increases in end-diastolic volume or ejection fraction or both. Although stroke volume and related cardiac variables were measured at rest in this study, it is likely that the observed increase in LVDd at rest in the exercise training group would persist at maximal exer-

Figure 4. Left ventricular end-diastolic dimensions before and after training for exercise and control groups. Dotted line equals the mean value.
cise and contribute to the increases in maximal stroke volume.

Weight loss also contributes to the increase in \( \text{VO}_2 \) max. The exercise group lost an average of 5.86 pounds during the 11-week exercise program. This weight loss alone accounts for a 1.6-ml/kg/min increase in \( \text{VO}_2 \) max and represents 21% of the total 7.6 ml/kg/min gain in \( \text{VO}_2 \) max after training.

**Echocardiographic Changes After Exercise Training**

Advances in echocardiography and other noninvasive techniques have facilitated study of the effects of endurance training on cardiac structure and function. The automated method used to measure echocardiographic variables in this study is more sophisticated than the manual methods used in other exercise training studies.\(^{12-14}\) Our automated process allows continuous measurement of the M-mode echocardiogram. The Teichholz method used for volume calculation in this study was recently concluded to be the best formula for calculating stroke volume.\(^{29}\)

In the present study, echocardiographic measurements before and after training showed a significant increase in LVDd. Similar changes in LVDd have been described in several other studies.\(^{12-14}\) The increased end-diastolic dimension is responsible for the calculated increases in LV end-diastolic volume and stroke volume. Unlike DeMaria and associates,\(^{12}\) we could not detect significant training effects on end-systolic dimension and volume or ejection fraction. The changes in these variables were in the same direction as reported by DeMaria, but did not reach statistical significance relative to our control group. The study of DeMaria et al. was limited by the lack of a separate control group that did not exercise. Although there were no changes in LV posterobasal wall thickness, the significant increase in end-diastolic dimensions would indicate a total increase in LV mass.

The average change in end-diastolic dimension reported in our exercise group was somewhat greater than that reported by DeMaria (3.9 vs 2.0 mm). The DeMaria study involved 12 men and 10 women (mean age 26 years) who participated in a 22-week “walk-jog-run” program 1 hour 4 days a week at 70% of maximal heart rate. The greater intensity of training in our exercise group, averaging 90% of maximal heart rate, may have accounted for the greater change in end-diastolic dimension and volume.

The possibility that decreases in resting heart rate after the training program (62 to 54 beats/min) contributed to changes in LV size was considered. To evaluate this possibility, we plotted individual heart rate changes against end-diastolic dimensional changes and found no correlation. DeMaria et al.\(^{30}\) also found a linear relationship between end-diastolic dimension and heart rate. Their data indicated that a 10-beat/min change in heart rate induced by cardiac pacing would only be associated with a 2.7% change in end-diastolic dimension. Based on these results, the decrease of 8 beats/min in resting heart rate should only have accounted for 1 mm of the 3.9-mm increase in end-diastolic dimension in our exercise group.

**Electrocardiographic Changes After Exercise Training**

Electrocardiographic changes in athletes have been well described.\(^{5,9,31}\) Common findings include sinus bradycardia, atrioventricular conduction disturbances, ST-T-wave changes, and voltage changes of ventricular hypertrophy. These ECG changes are believed to be the result of physiologic changes of the cardiovascular system caused by prolonged physical activity.

Fewer studies that describe the serial changes in normal individuals undergoing exercise training have been reported. DeMaria et al.\(^{32}\) examined ECG variables and indicated that precordial lead QRS voltage increased after training. Although we considered many more electrocardiographic variables before and after training, the only significant changes were increases in R-wave voltage in leads V\(_3\) and V\(_6\), as well as the maximal R-wave voltage in the precordial leads. These ECG changes support the echocardiographic findings of increased LV mass. Bonoris et al.\(^{32}\) speculated that the QRS voltage changes may reflect increased chamber size rather than ventricular mass. We could not correlate R-wave gains in the experimental group with changes in end-diastolic dimension; therefore, the most probable cause for voltage increase is increased LV muscle mass. More studies are needed to determine what causes the ECG changes with exercise training.

In conclusion, the physiologic mechanism for the changes in cardiac structure were not specifically studied. However, the increase in LVDd and LV volume that occurred during the 11-week endurance training program allowed the heart to generate a larger stroke volume during exercise, which accounts for the increased \( \text{VO}_2 \) max associated with endurance training.

The results of this study do not answer the question of the effect that age might have on cardiac adaptations, nor do they discredit the possibility that endurance athletes have a genetic predisposition for larger cardiac volumes. We do not know whether the LV volumes in the training group would have continued to increase had the training period been extended.

**Acknowledgment**

We thank Arnold Nelson, Bill Varley, Monica Noble, and Janet Jones for their technical assistance in this study.

**References**

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