Cardiovascular adaptations to 10 days of cycle exercise

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We hypothesized that 10 days of training would enhance cardiac output (CO) and stroke volume (SV) during peak exercise and increase the inotropic response to β-adrenergic stimulation. Ten subjects [age 26 ± 2 (SE) yr] trained on a cycle ergometer for 10 days. At peak exercise, training increased O2 uptake, CO, and SV (P < 0.001). Left ventricular (LV) size and function at rest were assessed with two-dimensional echocardiography before (baseline) and after atropine injection (1.0 mg) and during four graded doses of dobutamine. LV end-diastolic diameter increased with training (P < 0.02), whereas LV wall thickness was unchanged. LV contractile performance was assessed by relating fractional shortening (FS) to the estimated end-systolic wall stress (σES). Training increased the slope of the FS-σES relationship (P < 0.05), indicating enhanced systolic function. The increase in slope was correlated with increases in CO (r = −0.71, P < 0.05) and SV (r = −0.70, P < 0.05). The increase in blood volume also correlated with increases in CO (r = 0.80, P < 0.01) and SV (r = 0.85, P < 0.004). These data show that 10 days of training enhance the inotropic response to β-adrenergic stimulation, associated with increases in CO and SV during peak exercise.

short-term training; left ventricular function; cardiac output; stroke volume

ENDURANCE EXERCISE TRAINING lasting several weeks increases heart size and improves cardiac reserve capacity manifested as increases in cardiac output and stroke volume during maximal exercise (18, 20). This adaptive increase in pump performance of the heart contributes to the increase in maximal O2 uptake (VO2max) in the trained state (1, 8, 18, 20, 23). The increase in stroke volume in response to training is mediated by increases in left ventricular end-diastolic volume and diastolic filling (6, 13), which may likely be associated with a greater plasma volume (4), and an augmented contractile response to β-adrenergic stimulation (12, 22).

Although these adaptations occur with endurance exercise training lasting several weeks, it is not known whether short-term endurance exercise training (7–10 consecutive days of training) can elicit comparable improvements. Similar to endurance exercise training of several weeks' duration, short-term training induces a significant increase in stroke volume both at rest and during submaximal-intensity exercise, as well as significant increases in VO2max or peak O2 uptake (VO2peak), blood volume, and left ventricular end-diastolic diameter (4, 6, 15, 21). Surprisingly, however, it is not known to what degree stroke volume and cardiac output increase with short-term training during maximal or peak exercise. Furthermore, it is not known whether short-term training enhances the inotropic response to β-adrenergic stimulation and whether the changes in β-adrenergic-mediated contractile function are associated with increases in stroke volume and cardiac output during maximal or peak exercise.

Given the significant cardiac adaptations to short-term endurance exercise training (6), we hypothesized that 10 days of training would induce cardiovascular adaptations with increases in cardiac output and stroke volume during peak exercise and enhance the inotropic response to a β-adrenergic agonist. Furthermore, we sought to determine whether the increased inotropic response to β-adrenergic stimulation would be related to the increases in cardiac output and stroke volume during peak exercise.

METHODS
Subjects. Ten healthy sedentary young subjects, five men and five women [age 26 ± 2 (SE) yr] participated in this study. The experimental procedures were approved by the Human Subjects Committee at Washington University School of Medicine, and subjects gave their written informed consent to participate in the study. None of the subjects had either symptoms or history of cardiovascular disease, and they all had normal physical examinations and resting and exercise 12-lead electrocardiograms.

VO2peak. VO2peak was determined on an electrically braked cycle ergometer (Bosch) before and after training by using a continuous exercise protocol, in which power output was increased 25–50 W every 2 min. O2 uptake (VO2) was measured continuously by open-circuit spirometry and was averaged every 30 s with the use of an automated on-line system. Inspiratory volume was measured with a Parkinson-Cowan CD-4 dry-gas meter. Fractional concentrations of O2 and CO2 were sampled from a mixing chamber and quantified with the use of electronic O2 (Applied Electrochemistry S3-A) and CO2 (Beckman LB-2) analyzers. VO2peak was defined as the mean of the two highest consecutive 30-s VO2 measurements, corresponding with a respiratory exchange ratio value ≥1.10 and a heart rate within 10 beats of predicted maximum heart rate.

Plasma volume. Plasma volume was determined before and after training with the Evans blue dye technique, as previously described (9, 15). Subjects rested supine 30 min before a known volume (4–5 ml) of a sterile dye solution (New World Trading, DeBary, FL) was injected into an antecubital vein via a Teflon catheter. Ten minutes later, a blood sample was taken for subsequent determination of plasma dye concentration by using spectrophotometry at 615 nm. Samples were measured in triplicate, and the average coefficient of variance was 2.2%. Plasma volume was calculated from the plasma dye concentration and a known standard dye concentration. Blood volume was calculated by dividing plasma volume by (1 – hematocrit). Hematocrit was measured in quadruplicate and corrected for trapped plasma and venous sampling.

Cardiac output. Cardiac output was measured during peak cycle exercise before and after training on separate days from

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the \( \dot{V}O_{2\text{peak}} \) tests by using the acetylene (\( C_2H_2 \)) rebreathing method (26). For cardiac output measurements, subjects rebreathed a mixture of 10% \( He \)-45% \( O_2 \)–0.5% \( C_2H_2 \)-44.5% \( N_2 \) from a dosed-system anesthesia bag. Concentrations of \( C_2H_2 \) and \( He \) were continuously monitored by a Perkin-Elmer mass spectrometer (MGA-1100) interfaced with a PDP-11 computer for storage and processing of the data. Cardiac output was calculated from the exponential disappearance rate of \( C_2H_2 \) relative to \( He \) in several sequential end expirations. Reproducibility of this method performed during maximal exercise has been reported previously by this laboratory (12, 23). The intraclass correlation coefficient for repeated measurements of cardiac output was calculated to be \( r = 0.86 \). Heart rate and blood pressure (sphygmomanometer) were measured during the test. Blood pressure was measured at least twice during exercise by the same person before and after training.

Mean blood pressure (MBP) was calculated as \[ \text{MBP} = \frac{\text{SBP} + 3 \times \text{DBP}}{4} \] (mmHg). The arterial and mixed venous \( O_2 \) content difference \([\text{a}-\text{v}]O_2\) at peak exercise was calculated as \( \dot{V}O_{2\text{peak}}/\text{cardiac output} \times 100 \) (ml/100 ml). Total peripheral resistance (TPR) was estimated as \( \text{MBP}/\text{cardiac output} \times 80 \) (dyn·s·cm\(^{-5} \)).

Left ventricular size and performance. Left ventricular size and systolic function were evaluated before and after training by using two-dimensional guided M-mode echocardiography with a 2.5-MHz transducer (model 77020A, Hewlett-Packard). Left ventricular end-diastolic dimension (LVDD), end-systolic dimension (LVESD), posterior wall thickness (LVPWT), and septal wall thickness (LWSW) were measured by using standard guidelines recommended by The American Society of Echocardiography (19). Fractional shortening (FS) was estimated as \[ \text{FS} = (\text{LVDD} - \text{LVESD})/\text{LVDD} \times 100 \% \]. Left ventricular end-systolic wall stress (\( \sigma_{\text{es}} \)) was estimated as described by Grossman et al. (10), \[ \sigma_{\text{es}} = P_{\text{es}} h/(2 h + r_5) \], where \( P \) is SBP, expressed as grams per square centimeter, \( r \) is end-systolic radius (ESD/2), and \( h \) is posterior wall thickness at end systole. Diastolic-filling dynamics were evaluated by using the pulsed Doppler transmitral diastolic flow-velocity profile. Early (E), late (A), and the ratio of early-to-late (E/A) diastolic flow velocities were used as measurements of left ventricular filling (17). Reproducibility of echocardiographic measurements from this laboratory have been reported previously (7, 14). Calculated from repeated measures, intraclass correlation coefficients were \( r = 0.85, 0.80, \) and 0.87, and coefficients of variation were 3.6, 5.5, and 5.9% for LVDD, LVESD, and LVPWT, respectively.

Response to \( \beta \)-adrenoreceptor agonist. After acquisition of baseline echocardiographic, Doppler, and blood pressure data, 1.0 mg of atropine was administered intravenously. The rationale for the use of atropine was to facilitate detection of enhanced \( \beta \)-adrenergic-mediated left ventricular contractile function that might otherwise have been blunted by increased vagal tone after training (25). Echocardiographic, Doppler, and blood pressure measurements were repeated 2 min after atropine injection. Dobutamine was then continuously infused at successive doses of 3.0, 6.0, 9.0, and 12.0 \( \mu g \cdot kg^{-1} \cdot min^{-1} \) by using an infusion pump (model 122, Harvard Apparatus, South Natick, MA). Echocardiographic, Doppler, and blood pressure measurements were taken beginning 2 min after each dose. Blood pressure was measured three times at baseline, after atropine injection, and during each dose of dobutamine. Each dose lasted 5–6 min. Left ventricular contractile performance was assessed by using the FS-\( \sigma_{\text{es}} \) relationship by plotting FS as a function of \( \sigma_{\text{es}} \), with a steeper slope being suggestive of enhanced contractile function (2).

Measurement of plasma catecholamine concentrations. A 1-ml blood sample was taken at baseline, after atropine injection, and during each dose of dobutamine. In addition, blood samples were obtained during the cycle ergometer test for peak cardiac output. The plasma was separated and stored at \(-80^\circ\)C for later analysis. Plasma catecholamines were assayed by using a single-isotope derivative method (5).

Exercise training. Exercise training consisted of 1-h cycling bouts performed daily on 10 consecutive days. During each bout, subjects initially cycled for 10 min at 65% \( \dot{V}O_{2\text{peak}} \), followed by 25 min at 75% \( \dot{V}O_{2\text{peak}} \). During the last 25 min of each bout, subjects cycled for 3 min at 95% \( \dot{V}O_{2\text{peak}} \), followed by 2 min of low-intensity pedaling, a pattern that was repeated for a total of five intervals. Work rates were increased daily to maintain the established target heart rate. Target heart rates at each intensity were established on day 1 concurrently with \( \dot{V}O_{2\text{peak}} \) measurements.

Statistics. Physiological variables during peak exercise and \( \beta \)-adrenergic stimulation were compared with a repeated-measures analysis of variance design. Differences among responses during the dobutamine infusion were determined by using Duncan’s multiple-range post hoc test when a significant training or dobutamine effect, or a significant interaction between training and dobutamine, was evident. Linear regression was used to determine the slope and intercept of the FS-\( \sigma_{\text{es}} \) relationship and the \( \sigma_{\text{es}}-\text{ESD} \) relationship for each individual, and a paired Student’s t-test was used to compare the mean of the individual slopes and intercepts before and after training. Linear regression was used to determine independent variables relating to the training-induced changes in stroke volume, cardiac output, and \( \dot{V}O_{2\text{peak}} \) during peak cycle exercise. Significant differences and significant linear relationships were established at \( P \leq 0.05 \), and all data were expressed as means ± SE.

RESULTS

Cycle \( \dot{V}O_{2\text{peak}} \). All subjects completed the 10 days of training. Body weight did not change significantly with training (70.4 ± 4.5 vs. 70.9 ± 4.7 kg after training) in either men (78.5 ± 7.1 vs. 79.4 ± 7.3 kg) or women (62.3 ± 3.1 vs. 62.5 ± 3.1 kg). Cycle ergometer \( \dot{V}O_{2\text{peak}} \) increased 10% from 2.54 ± 0.29 l/min (35.4 ± 2.5 ml·kg\(^{-1} \)·min\(^{-1} \)) to 2.80 ± 0.32 l/min (38.9 ± 2.9 ml·kg\(^{-1} \)·min\(^{-1} \)) \((P < 0.0001)\). In men, \( \dot{V}O_{2\text{peak}} \) increased from 3.32 ± 0.27 to 3.64 ± 0.30 l/min (42.6 ± 1.5 ml·kg\(^{-1} \)·min\(^{-1} \)) in women it increased from 1.75 ± 0.09 to 1.96 ± 0.11 l/min (28.2 ± 1.0 to 31.5 ± 1.9 ml·kg\(^{-1} \)·min\(^{-1} \)). There was no difference between men and women in the \( \dot{V}O_{2\text{peak}} \) response to training.

Hemodynamic and hormonal responses to peak exercise (Table 1). The 10% increase in cycle \( \dot{V}O_{2\text{peak}} \) was associated with a 12% increase in cardiac output during peak exercise \((P < 0.001)\). This increase in cardiac output was solely the result of an increase in stroke volume, which was 15% higher after training \((P < 0.060)\) because peak heart rate did not change, although there was a tendency for it to be lower after training \((P < 0.060)\). The change in cardiac output was greater in men \((3.3 \pm 1.2 \text{l/min in women, } P < 0.03)\). Similarly, the absolute increase in stroke volume was greater in men \((22.3 \text{ vs. } 8.3 \text{ ml/beat in women, } P < 0.02)\). Training did not affect peak exercise SBP or
change significantly with training. Although LVEDD increased (P < 0.02), and LVESD tended to increase (P < 0.060) in response to training, LVPWT, LVSWT, and left ventricular wall thickness-to-radius ratio (h/r) did not change significantly. Neither FS nor estimated $\sigma_{ES}$ changed significantly with training. Neither plasma norepinephrine (pretraining: 255 ± 55 pg/ml; posttraining: 186 ± 10 pg/ml) nor epinephrine (pretraining: 16.7 ± 3.6 pg/ml; posttraining: 18.2 ± 2.7 pg/ml) concentrations changed significantly with training at baseline.

Effects of atropine. Atropine increased heart rate from the baseline by 26 beats/min both before and after training (P < 0.0001). There was no difference between the pre- and posttraining heart rate after atropine (94 ± 4 vs. 90 ± 6 beats/min). Atropine increased DBP both before and after training (P < 0.004) but had no effect on SBP. LVEDD, LVESD, and FS were not affected by atropine either before or after training. Estimated $\sigma_{ES}$ increased with atropine both before and after training (P < 0.03). Neither plasma norepinephrine nor epinephrine concentrations were changed by atropine. There were no differences between men and women in response to atropine.

Effects of β-adrenergic stimulation (Table 3). Throughout the infusion of dobutamine, LVEDD was greater after training (P < 0.004). However, the increasing doses of dobutamine had no effect on LVEDD from postatropine values either before or after training. In contrast, LVESD decreased during dobutamine infusion (P < 0.0001), reaching a plateau at 9.0 µg·kg⁻¹·min⁻¹ both before and after training. LVESD was greater in response to training after atropine and during dobutamine infusion (except at 3.0 µg·kg⁻¹·min⁻¹; P < 0.05). SBP increased in response to dobutamine both before and after training (P < 0.0001), reaching a plateau at 9.0 µg·kg⁻¹·min⁻¹. Training, however, had no effect on SBP response to dobutamine. DBP was not affected significantly by either dobutamine or training. The changes in plasma epinephrine and norepinephrine concentrations during dobutamine infusion were not significant, nor did training affect catecholamine concentrations significantly.

Heart rate increased progressively during dobutamine infusion without reaching a plateau, both before and after training (P < 0.0001) (Fig. 1A). However, the increase in heart rate from atropine injection to the highest dose of dobutamine (12.0 µg·kg⁻¹·min⁻¹) was less after than before training (P < 0.01). Heart rate was lower after training at each dose of dobutamine (P < 0.004), even though postatropine heart rate values were similar. FS increased during dobutamine infusion (P < 0.0001), reaching a plateau at 9.0 µg·kg⁻¹·min⁻¹, both before and after training (Fig. 1B). Training, however, did not influence FS responses to β-adrenergic stimulation. Estimated $\sigma_{ES}$ decreased progressively in response to dobutamine before training (P < 0.0001) (Fig. 1C). After training, however, $\sigma_{ES}$ decreased initially and then reached a plateau at the dobutamine dose of 6.0 µg·kg⁻¹·min⁻¹ with no further decrease. At the highest dobutamine dose (12.0 µg·kg⁻¹·min⁻¹), $\sigma_{ES}$ was higher after training (P < 0.004). There were no

### Table 1. Physiological responses during peak cycle ergometer exercise

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretraining</th>
<th>Posttraining</th>
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<tbody>
<tr>
<td>$V_O_2$, l/min</td>
<td>2.54 ± 0.29</td>
<td>2.80 ± 0.32*</td>
</tr>
<tr>
<td>RER</td>
<td>1.30 ± 0.02</td>
<td>1.28 ± 0.02</td>
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<tr>
<td>Cardiac output, l/min</td>
<td>18.3 ± 1.3</td>
<td>20.5 ± 1.7*</td>
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<tr>
<td>Heart rate, beats/min</td>
<td>189 ± 2</td>
<td>184 ± 2†</td>
</tr>
<tr>
<td>Stroke volume, ml/beat</td>
<td>97 ± 7</td>
<td>112 ± 9*</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>188 ± 4</td>
<td>186 ± 6</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>87 ± 2</td>
<td>81 ± 2*</td>
</tr>
<tr>
<td>MBP, mmHg</td>
<td>120 ± 2</td>
<td>117 ± 2*</td>
</tr>
<tr>
<td>(a-V)_O_2, ml/100 ml</td>
<td>13.6 ± 0.8</td>
<td>13.4 ± 0.6</td>
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<tr>
<td>TPR, dyn·s·cm⁻²</td>
<td>549 ± 35</td>
<td>481 ± 38*</td>
</tr>
<tr>
<td>Epi, pg/ml</td>
<td>498 ± 108</td>
<td>502 ± 102</td>
</tr>
<tr>
<td>NE, pg/ml</td>
<td>2.631 ± 271</td>
<td>3.611 ± 603†</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 10 subjects. $V_O_2$, O₂ uptake; RER, respiratory exchange ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure; (a-V)_O_2, arterial and mixed venous O₂ content difference calculated from $V_O_2$ and cardiac output; TPR, total peripheral resistance calculated from cardiac output and MBP; Epi, plasma epinephrine; NE, plasma norepinephrine. *P < 0.05 vs. pretraining. †P < 0.06 vs. pretraining.

### Table 2. Physiological adaptations at baseline

<table>
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<tr>
<th>Variable</th>
<th>Pretraining</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP, mmHg</td>
<td>110 ± 3</td>
<td>111 ± 3</td>
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<tr>
<td>DBP, mmHg</td>
<td>68 ± 2</td>
<td>69 ± 3</td>
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<tr>
<td>Heart rate, beats/min</td>
<td>67 ± 4</td>
<td>64 ± 4</td>
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<td>LVEDD, mm</td>
<td>48.5 ± 2.1</td>
<td>50.6 ± 1.5*</td>
</tr>
<tr>
<td>LVESD, mm</td>
<td>32.2 ± 16</td>
<td>33.8 ± 0.9†</td>
</tr>
<tr>
<td>LVPWT, mm</td>
<td>9.0 ± 0.7</td>
<td>8.6 ± 0.6</td>
</tr>
<tr>
<td>LVSWT, mm</td>
<td>8.5 ± 0.9</td>
<td>8.4 ± 0.7</td>
</tr>
<tr>
<td>h/r</td>
<td>0.38 ± 0.03</td>
<td>0.34 ± 0.02</td>
</tr>
<tr>
<td>Fractional shortening, %</td>
<td>33.8 ± 1.1</td>
<td>33.8 ± 0.9</td>
</tr>
<tr>
<td>End-systolic wall stress, g/cm²</td>
<td>50.4 ± 4.4</td>
<td>52.3 ± 3.0</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 10 subjects. Baseline, before atropine injection; LV, left ventricular; EDD, end-diastolic dimension; ESD, end-systolic dimension; PWWT, posterior wall thickness; SWWT, septal wall thickness; h/r, LV wall thickness-to-radius ratio. *P < 0.05 vs. pretraining. †P < 0.06 vs. pretraining.
Left ventricular filling dynamics. To evaluate the effects of training on left ventricular filling dynamics, comparisons were made at comparable heart rates before and after training during dobutamine infusion. Six subjects whose heart rates were similar before and after training (92 ± 8 beats/min both pre- and posttraining) were included in this comparison. Although there was a trend for the early (E) transmitral flow velocity to increase from 80.8 ± 11.0 to 96.0 ± 11.1 cm/s (P < 0.07), neither the late (A) transmitral flow velocity (pretraining: 55.5 ± 8.6 cm/s; posttraining: 57.5 ± 4.5 cm/s) nor the E/A (pretraining: 1.50 ± 0.13; posttraining: 1.66 ± 0.10) was changed significantly with training.

Relationships between physiological variables. The training-induced increase in blood volume correlated with training-induced increases in cardiac output (r = 0.80, P < 0.01) and stroke volume (r = 0.85, P < 0.004) during peak exercise. The increase in blood volume with training also tended to correlate with the increase in VO$_{2\text{peak}}$ (r = 0.65, P < 0.08). The change in the slope of the FS-$\Delta$ES relationship with training correlated significantly with the training-induced increases in cardiac output (r = −0.71, P < 0.05) and stroke volume (r = −0.70, P < 0.05) but not with the increase in VO$_{2\text{peak}}$ (r = −0.50, P = 0.20). Both before and after training, resting baseline LVEDD correlated significantly with stroke volume (pretraining: r = 0.79, P < 0.01; posttraining: r = 0.83, P < 0.005) and blood volume (pretraining: r = 0.85, P < 0.003; posttraining: r = 0.85, P < 0.004). However, the change in resting baseline LVEDD induced by training did not correlate with the change in either stroke volume (r = −0.36, P = 0.37) or blood volume (r = −0.47, P = 0.24).

**DISCUSSION**

The results of this study show that 10 days of training induces adaptations suggestive of an increased inotropic response to β-adrenergic stimulation,
and these adaptations are associated with increases in cardiac output and stroke volume during peak exercise. The adaptive enhancement of the inotropic response to \( \beta \)-adrenergic receptor stimulation appears to be similar to those attained with long-term training (12, 22). In the present study, the training-induced improvement in the inotropic response to \( \beta \)-adrenergic stimulation was reflected by a steeper slope of the FS-\( \sigma_{ES} \) relationship such that, for a given \( \beta \)-adrenergic-mediated decrease in \( \sigma_{ES} \), there was a greater increase in FS observed at similar plasma catecholamine concentrations and without a higher heart rate response.

Improved contractile function in response to \( \beta \)-adrenergic activation after several weeks of training has been demonstrated in isolated ventricular papillary muscle in animals (16, 27). In these animal models, training enhanced the contractile response to \( \beta \)-adrenergic stimulation by increasing the maximum rate of tension development and decreasing the time to peak tension in isolated heart muscle preparations. In humans, 12 wk of training resulted in an increase in the inotropic response to \( \beta \)-adrenergic stimulation at comparable changes in preload, reflected by a greater FS response (22). In addition, in a cross-sectional study, a steeper slope in the FS-\( \sigma_{ES} \) relationship was demonstrated in response to dobutamine in endurance-trained athletes compared with untrained individuals (12).

In this study, the training-induced larger preload after 10 days of training was evidenced by an increase in LVEDD, associated with a larger blood volume, even though the training-induced changes in LVEDD did not correlate with those in blood volume. The trend observed for enhanced early diastolic-filling velocity was probably due to a larger LVEDD and also to increased sensitivity to catecholamines. The increase in stroke volume during peak exercise can result from increased

![Fig. 1](image1.png)

**Fig. 1.** Heart rate (A), fractional shortening (B), and estimated left ventricular end-systolic (LVEDD) wall stress (C) beginning with atropine injection (0) and throughout continuous infusion of dobutamine. Values are means ± SE; \( n = 10 \) subjects. \(*P < 0.05\) vs. pretraining (Pre) values. \( \dagger P < 0.05 \) interactive effect between training and dobutamine. Post, posttraining.

![Fig. 2](image2.png)

**Fig. 2.** Plot of mean of individual slopes and intercept of fractional shortening-LVES wall stress relationship (A) and LVES wall stress-LVESD (LVESD) relationship (B). Mean values were calculated in 10 subjects in response to dobutamine. Solid lines, Pre; dashed lines, Post. Shift in slopes and intercepts with training suggests enhanced contractile response to \( \beta \)-adrenergic stimulation, i.e., for a given decrease in LVES wall stress there is a larger increase in fractional shortening and a greater decrease in LVESD after training.
preload, reduced afterload, and/or enhanced inotropic response to β-adrenergic stimulation (1). Our data suggest that both a larger preload and increased inotropic response to β-adrenergic stimulation are associated with the greater stroke volume during peak exercise after 10 days of training.

In contrast to an enhanced inotropic response, the chronotropic response to β-adrenergic stimulation was significantly reduced in response to training. Similar results have also been observed in animals (11) and in humans (12, 24) after training. Evidence for this uncoupling of inotropic and chronotropic responses comes from a previous animal study that reported a selective downregulation in β-adrenergic receptors located in the right atrium, associated with a lower chronotropic response to β-adrenergic stimulation, whereas no change in β-receptor number was observed in the left ventricle of pigs (11). In the present study, the attenuated heart rate response after training was not accompanied by a smaller increase in plasma catecholamine concentrations during dobutamine infusion. This suggests that the increase in sympathetic activity in response to dobutamine was probably similar before and after training. The attenuated heart rate response suggests that enhanced LV systolic function was due to a direct effect of dobutamine on β-adrenergic receptors rather than to enhancement of the force-frequency relationship (11).

Although the purpose of this study was not to study gender differences, the fact that both men and women were included in this study warranted attention. There were no differences between men and women in the response to β-adrenergic stimulation, and each subject demonstrated an increase in the FS-ES relationship, indicating that both men and women responded to training with an increased contractile response to β-adrenergic stimulation. Furthermore, there were no gender differences in VO2peak response to training. However, training increased peak exercise cardiac output and stroke volume and also increased blood volume more in men. Given the small number of men and women in the present study, these data should be interpreted with caution.

A limitation of this study is that measurement of ES is only an estimate. Second, the FS-ES relationship is not totally independent of preload because preload is known to increase FS (2, 3). However, our data suggest that the observed increase in FS in the trained state was at least partially independent of an increase in LVEDD. First, the changes in LVEDD during dobutamine infusion were similar before and after training. Second, there was no significant correlation between the change in FS and the change in LVEDD (r = 0.23). Furthermore, in those subjects whose LVEDD were decreased or had changed insignificantly after training (n = 6), we found that FS response to dobutamine was greater after training in four of these subjects during the dobutamine infusion. We also evaluated the ES-ES relationship, which is probably less dependent on preload, and found a greater decrease in ES for a given decrease in ES after training during the dobutamine infusion.

In conclusion, our findings suggest that 10 days of training, similar to training lasting several weeks, can result in increases in cardiac output and stroke volume during peak exercise in the trained state in young subjects. Furthermore, both a training-induced enhancement of inotropic response to β-adrenergic stimulation and a greater blood volume at rest are associated with increases in cardiac output and stroke volume during peak exercise. These results indicate that significant cardiac adaptations can occur in response to short-term exercise training and provide insight into the mechanisms for cardiac adaptations associated with increased exercise capacity after short-term training.

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