Exercise Training Improves Left Ventricular Systolic Function in Older Men

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To determine whether endurance exercise training can improve left ventricular systolic function in older men, 10 healthy sedentary men (64±3 years old; mean±SD) were studied. Training consisted of endurance exercise 4±0.3 days per week for 11.8±2.5 months at a progressively increasing intensity of 60–80% of maximal O₂ uptake (VO₂max) with additional brief bouts of exercise equal to 93±13% of VO₂max. VO₂max increased from 29.6±4.1 to 37.2±5.7 ml/kg/min (p<0.001). Percent body fat was decreased (17.8±3.6% versus 15.6±3.6%; p<0.001). Before training, left ventricular ejection fraction, determined by electrocardiographic-gated equilibrium blood pool imaging, increased only modestly during exercise (from 66.3±6.7% at rest to 70.6±6.9% at peak exercise). After training, the increase in ejection fraction during exercise was significantly greater (from 67±4.8% at rest to 77.6±7.5% at peak exercise) than that observed before training and was similar to that in young sedentary men (64±7% at rest versus 74±9% at peak exercise). Although the changes in systolic pressure from rest to exercise were similar, end-systolic volume decreased significantly at peak exercise after (51±12 versus 38±13 ml; p<0.005) but not before (46±18 versus 43±13 ml; p=NS) training with a shift in the end-systolic volume–systolic blood pressure relation to the left compatible with enhanced inotropic state. Exercise training induced proportional increases in left ventricular end-diastolic diameter and posterior wall thickness, measured echocardiographically, with no change in the wall thickness-to-radius ratio (0.27±0.04 versus 0.28±0.03), suggestive of volume-overload hypertrophy. End-diastolic volume at rest was increased (138±11 versus 155±26 ml; p<0.05). Stroke volume was significantly greater at peak exercise in response to training (110±17 ml before versus 132±27 ml after training; p<0.05). The differences in end-diastolic volume and stroke volume at peak exercise between the trained and untrained states correlated strongly (r=0.95). At a given increase in end-diastolic volume during exercise, the increase in stroke volume was more after than before training. Furthermore, at a given change in end-systolic volume from rest to exercise, the subjects attained a higher systolic blood pressure after than before training. These data suggest that endurance exercise training of sufficient intensity can improve left ventricular systolic performance in older men. (Circulation 1991;83:96–103)

Primary aging is characterized by a progressive decline in maximal aerobic exercise capacity generally attributed to the age-related alterations in the cardiovascular system, skeletal muscle, and life-style habits.1–7 Although endurance exercise training increases maximal aerobic power in young and middle-aged subjects,8–10 it was not clear until recently that older subjects can also adapt to exercise training.11 Results from a recent study suggest that if the level of training stimulus in terms of duration, frequency, and intensity is adequate, older individuals can also exhibit improvement in aerobic work capacity.12 The mechanisms underlying the training-induced augmentation of exercise capacity remain obscure. In young and middle-aged subjects, the increase in O₂ uptake capacity after training is generally attributed to both enhanced left ventricular performance resulting in augmented maximal cardiac output (central adaptations) and increased O₂ extraction by working skeletal muscle reflected in widening of the arteriovenous O₂ difference (peripheral adap-
tions). It is not known, however, whether exercise training can enhance left ventricular performance in older subjects who are prone to develop left ventricular dysfunction with advancing age. In a recent study, no discernible improvement in the age-related decline in cardiac performance was reported in response to exercise training. The reported lack of cardiac adaptations in the elderly may be the consequence of either the inability of older subjects to adapt to training or of an inadequate training stimulus. The latter possibility seems more plausible in view of recent evidence suggesting that a relatively high intensity training stimulus is required to induce central adaptations in patients with ischemic heart disease and evidence from studies in rats showing prevention of age-related changes in cardiac contraction by exercise training. Therefore, the present study was designed to test the hypothesis that a program of prolonged and intense endurance exercise training can induce cardiac adaptations in older healthy subjects.

Methods

Subjects

After giving their informed consent, 13 sedentary men were studied who met the criteria of age of 60–70 years, willingness to participate in a 1-year-long program of daily exercise, absence of symptoms, no previous history of cardiopulmonary diseases, no current history of smoking, no history of hypertension, normal cardiovascular examination and resting 12-lead electrocardiogram, no evidence of exercise-induced myocardial ischemia defined as 0.1 mV or greater horizontal or downsloping ST segment depression at maximal exercise, and absence of exercise-induced impairment of regional myocardial perfusion during thallium-201 exercise stress testing in subjects whose ST segment responses to maximal exercise were considered equivocal for ischemia. A total of 19 volunteer men 60–70 years old were screened, and 13 men who fulfilled the above criteria were selected. Three subjects dropped out in the early phase of the study before commencing the exercise training program—one because of lack of motivation, one because of orthopedic problems, and one because of new onset of chest pain and new abnormal exercise ST segment changes suggestive of myocardial ischemia. Therefore, the data on the remaining 10 sedentary men (64±3 years old; mean±SD) who completed the program of training are reported. Two subjects whose electrocardiographic changes were considered equivocal for myocardial ischemia demonstrated no evidence of exercise-induced impairment of myocardial perfusion during 201Tl exercise testing. One subject had a mild regional left ventricular contraction abnormality but showed no evidence of impaired regional myocardial perfusion during 201Tl stress testing. To verify that aging can impair left ventricular ejection fraction response to exercise, as reported by others, we also studied 10 young, healthy, sedentary men (31±4 years old; age range, 24–38 years) at rest and during supine cycle ergometer exercise. The study was approved by the Human Studies Committee of Washington University.

Maximal Aerobic Exercise Capacity  \( \dot{V}O_2 \)max

Each subject had an initial treadmill exercise test using the Bruce protocol to evaluate heart rate, blood pressure, and electrocardiographic changes in response to exercise and to select the appropriate protocol for determination of maximal \( \dot{V}O_2 \) consumption. One week later, another treadmill exercise test was performed for measurement of maximal \( \dot{V}O_2 \) consumption as previously described. Briefly, after 5 minutes of warm-up exercise that generally consisted of walking flat at a speed of 1.7 or 2.5 mph, the subjects began to exercise at a speed of 2.5 mph with 5–10% grade. From this point on, the speeds and grades were increased alternately every 2 minutes. \( \dot{V}O_2 \) and the respiratory-exchange ratio were measured using an open-circuit system. Inspiratory volume was measured by the Parkinson-Cowan CD-4 dry gas meter. Fractional concentrations of expired \( O_2 \) and \( CO_2 \) were measured from a mixing chamber using Applied Electrochemistry S3A \( O_2 \) and the Beckman LB-2 \( CO_2 \) analyzers, respectively. \( \dot{V}O_2 \)max was defined as the attainment of plateau of \( \dot{V}O_2 \) (i.e., <100 ml change in \( \dot{V}O_2 \)) with increasing work rates (leveling off criterion) and/or the respiratory-exchange ratio of 1.15 or greater. Maximal attainable peak \( \dot{V}O_2 \) was also measured during upright and supine cycle ergometer exercise testing. \( \dot{V}O_2 \)max was not determined in young subjects.

Left Ventricular Systolic Function

Left ventricular function at rest and during exercise was assessed using standard electrocardiographic-gated blood pool imaging as previously described. Briefly, after the in vitro labeling of erythrocytes with stannous pyrophosphate and 25 mCi of technetium-99m, images were obtained using a scintillation camera equipped with a low-energy, medium-resolution, parallel hole collimator with the patients supine and the scintillation camera positioned in the left anterior oblique projection (35°) and modest (15°) caudal angulation for optimal separation of left atrial and ventricular images. Data were collected in the frame mode (32 frames per cardiac cycle) in a 64×64-pixel matrix and processed off-line with a minicomputer (VAX 11/750). The left ventricular ejection fraction (LVEF) was calculated using a count-based technique developed in our laboratory that uses manually defined end-diastolic and end-systolic regions of interest: \( EF = \frac{EDC - ESC}{EDC} \times 100/EDC \), where EDC and ESC are end-diastolic and end-systolic counts, respectively, corrected for background activity. The left ventricular end-diastolic volume (LVEDV) was calculated using the standard geometric area–length method of Dodge et al: \( V = \frac{8A^2}{3\pi} \), where \( V \) is volume, \( A \) is
area, and l is long axis of the left ventricular image. Spatial calibration factors for the correction of digital images were obtained using the phantom technique described by Esser et al.\textsuperscript{21} Left ventricular end-systolic volume (LVESV) and stroke volume (SV) were derived from LVEF and LVEDV.\textsuperscript{14} After images were obtained at rest, each subject performed a graded supine exercise using a cycle ergometer (Engineering Dynamics Corp.) with the initial work rate of 50 W followed by 25-W increments every 3 minutes. Images were obtained in the last 2 minutes of each exercise stage in the left anterior oblique projections. Heart rate and blood pressure were measured every minute concurrently with the 2 minutes of data collection. Reported heart rate and blood pressure values are averages of the corresponding values recorded at the second and third minute of each stage of exercise. Blood pressure was measured using a standard sphygmomanometer. The pedalling rate was maintained between 65 and 75 rpm. Peak \textsubscript{O2} consumption was determined in nine of the subjects during supine cycle ergometer exercise by the collection of expired gases in the meteorological balloons, which were analyzed by a mass spectrometer (Perkin-Elmer MA 1100) as previously described.\textsuperscript{14} LVEF and LVEDV were measured by one of the authors who had no knowledge of the subject’s exercise status. The reproducibility of LVEF was ±4\% (95\% confidence limits), and the intraobserver variability was ±2\%. The intraobserver variability for the LVEDV was 6±9 ml at rest and 7±9 ml at peak exercise.\textsuperscript{14}

Variables used to assess left ventricular systolic performance were changes in EF as a function of mean blood pressure used in the present study as a crude index of afterload even though we recognize that blood pressure can not be equated with left ventricular wall stress, changes in the systolic blood pressure-end-systolic volume (SBP-ESV) relation at rest and during exercise, changes in ESV from rest to exercise at a given change in SBP before and after training, the relation between SV and EDV, and the changes in SV from rest to exercise at a given change in EDV before and after training.

To characterize the pattern of the training-induced left ventricular hypertrophy, resting two-dimensional guided M-mode echocardiograms were obtained from five men (three of the subjects and two additional older men who had completed a similar program of exercise training). Recordings were made using the guidelines recommended by the American Society of the Echocardiography.\textsuperscript{22} End-diastolic and end-systolic diameters as well as posterior wall and septal thicknesses were measured as previously described.\textsuperscript{23} The end-systolic wall stress was calculated from the SBP (g/cm\textsuperscript{2}), end-systolic radius (diameter/2), and end-systolic posterior wall thickness using the equation described by Grossman et al.\textsuperscript{24} The reproducibility of echocardiographic measurements in our laboratory has been reported.\textsuperscript{23,25}

**Submaximal Exercise**

To characterize peripheral adaptations to training, changes in heart rate and blood pressure during submaximal exercise at a given absolute work intensity were examined. Each subject performed a steady-state submaximal exercise test on a treadmill with an intensity requiring 77±2\% of initial \textsubscript{Vo2}max for 15 minutes. Heart rate, blood pressure, and \textsubscript{O2} consumption were measured 1 minute before the completion of the exercise.

**Body Fat Determination and Diet**

Percent body fat was estimated by the skinfold thickness measurement using a Lange caliper at six sites.\textsuperscript{26} These measurements were obtained by the same investigator before and after training. No specific dietary intervention was implemented. Daily caloric intake was measured by analysis of the 4-day food recall.

**Training Program**

Before commencing endurance exercise training, each subject completed approximately 3 months of daily stretching and flexibility exercises. This phase of the program was intended to familiarize each subject with endurance exercise training, to identify and correct any possible musculoskeletal problems by appropriate stretching exercises, and to determine the most suitable form of endurance exercise for each subject. During this phase of study, the subjects did not engage in endurance types of exercise, and they were instructed to not alter their routine habitual physical activities. This was verified by determination of maximal aerobic exercise capacity at the completion of the flexibility exercise program.

After completion of the flexibility exercises, subjects began an endurance exercise training program consisting of walking, running, and cycle ergometer and treadmill exercises 5 days per week for 1 hour per session for 12 months. The initial intensity of exercise was adjusted to require 60–70\% of the subject’s \textsubscript{Vo2}max. Thereafter, it was increased progressively to 70–80\% of \textsubscript{Vo2}max supplemented by additional brief intervals of intense exercise requiring 90–100\% of \textsubscript{Vo2}max two or three times per week. \textsubscript{Vo2}max was measured at 3-month intervals throughout the program to maintain the training intensity constant relative to \textsubscript{Vo2}max.

**Study Design**

Each subject performed a maximal multistage treadmill stress (Bruce protocol) test followed in 1 week by determination of \textsubscript{O2} uptake capacity on a treadmill as well as on a cycle ergometer before the program of flexibility exercises. In addition, skinfold-thickness measurements were obtained at that time. On completion of this phase of the program and before endurance exercise training, another \textsubscript{Vo2}max determination, radionuclide ventriculography, and submaximal exercise testing were performed. These procedures were repeated after completion of the
endurance exercise training program. In addition, maximal \( \text{O}_2 \) uptake was measured periodically at selected intervals during the training program.

**Statistics**

The differences between the data obtained before and after training were evaluated using paired Student's \( t \) tests. Repeated-measures two-way analyses of variance were used when appropriate. Data are given as mean±SD.

**Results**

There was a significant improvement in the subjects' exercise capacity and endurance. The duration of flexibility and endurance exercise phases of the program was 3.3±0.6 and 11.8±2.5 months, respectively. The subjects exercised an average of 4±0.3 days per week. The range of intensity of exercise was 60–80% of \( \text{Vo}_2 \max \) with additional short bouts of exercise equal to 93±13% of \( \text{Vo}_2 \max \). In the last 3 months of the training program, six subjects were running 14.5±2.8 miles per week with an average speed of 5.82±2.5 mph. In addition, they were performing exercise on a cycle ergometer with an intensity of 129±25 W for 21±9.5 minutes during each exercise session. Three other subjects exercised predominantly on a cycle ergometer with an average work rate of 136±13 W for 29±28 minutes per exercise session as well as running 7±3.6 miles per week. One subject exercised only on a cycle ergometer. Resting heart rate decreased from 72±10 to 61±6 beats/min (\( p<0.025 \)). However, neither SBP nor DBP at rest changed in response to training. Body weight did not change significantly (80±6 versus 79±4 kg) even though daily caloric intake increased from 2,276±487 to 2,487±490 kcal (\( p<0.05 \)) during training. Estimated percent body fat decreased in response to training (17.8±3.6% versus 15.6±3.6%, \( p<0.001 \)).

**Adaptive Responses During Submaximal Exercise**

Heart rate and rate-pressure product were decreased significantly during steady-state submaximal exercise at a constant exercise intensity (\( \text{Vo}_2 \): 2.00±0.36 before and 1.95±0.36 l/min after training; Figure 1). SBP, however, was not decreased significantly after training.

**Maximal Aerobic Exercise Capacity**

\( \text{Vo}_2 \max \) did not change during the flexibility program (Table 1). However, it increased by 23% (\( p<0.001 \)) in response to 12 months of endurance exercise training (Table 1). \( \text{Vo}_2 \max \) increased from 29.6±4.1 l/min at baseline to 33.8±4.9 l/min after 3 months, 35.3±5.0 l/min after 6 months, and 37.2±5.7 l/min on completion of the training program (Table 1). Peak \( \text{Vo}_2 \) during supine cycle ergometer exercise increased from 1.85±0.2 l/min before to 2.05±0.26 l/min (\( n=9; \ p<0.005 \)) in response to training. Maximal heart rate during treadmill exercise did not change (171±10 before versus 167±10 beats/min after training, \( p=\text{NS} \)).

**Adaptive Responses in Left Ventricular Systolic Function**

EF at rest did not change in response to training (Table 2). Before training, the increase in EF from rest to exercise was modest (from 66.3±6.7% to 70.6±6.9%) and considerably less than that observed in sedentary young men, which is consistent with the age-related decline in cardiac reserve (Figure 2 and Table 2). In contrast, EF increased substantially during exercise after training (from 67±4.8% to 77.6±7.5%; Figure 2 and Table 2) even though values for mean blood pressure at peak exercise, used in the present study as a crude index of afterload, were similar before and after training (Figure 3A). Peak exercise EF after training was significantly higher than that before training (\( p<0.005 \), Figure 3A). The magnitude of increase in EF from rest to exercise in the trained state was comparable to that observed in young sedentary men (Figure 2 and Table 2). Left ventricular exercise reserve (\( \Delta \text{LVEF} \)) was greater after than before training (Table 2). Before training, EF peaked early during exercise and then decreased at peak exercise (Figure 4A). In contrast, it continued to rise progressively during exercise and reached maximum at peak rather than submaximal exercise after training (Figure 4A). The subjects did not show any significant change in ESV from rest to peak exercise before training (46±8 versus 43±13 ml, \( p=\text{NS} \), Figure 3B and Table 2). However, LVESV was decreased markedly at peak

**Table 1. Effect of Exercise Training on \( \text{O}_2 \) Uptake Capacity**

<table>
<thead>
<tr>
<th></th>
<th>Initial</th>
<th>Flexibility exercise</th>
<th>Final (endurance exercise)</th>
</tr>
</thead>
<tbody>
<tr>
<td>l/min</td>
<td>2.36±0.09</td>
<td>2.38±0.09</td>
<td>2.89±0.13*</td>
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<tr>
<td>ml/kg/min</td>
<td>29.6±4.1</td>
<td>29.9±4.6</td>
<td>37.2±5.7*</td>
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</table>

*\( p<0.001 \) compared with initial.
exercise after training (51±12 versus 38±13 ml, p<0.005, Figure 3B and Table 2). The extent of increases in SBP from rest to peak exercise was similar in the trained and untrained states (Figure 3B). Therefore, the SBP–ESV relation at peak exercise was shifted to the left after training compatible with enhancement of left ventricular systolic performance (Figure 3B). Furthermore, when changes in ESV from rest to exercise were matched (Δ ESV: −12±8 before versus −12±8 ml after training), the magnitude of the increase in SBP was larger after than before training (Δ SBP: 54±29 before versus 66±21 mm Hg after training, p<0.05, n=9, Figure 5A). Before training, ESV decreased modestly during submaximal exercise and then increased at peak exercise (Figure 4C). After training, ESV continued to decrease throughout exercise and reached its nadir at peak exercise (Figure 3C). LVEDV at rest was significantly higher in the trained compared with the untrained state (138±11 versus 155±26 ml, p<0.05). LVEDV at peak exercise was 153±9 before and 170±27 ml after training (Figure 4B). EDV increased during early exercise and then reached a plateau with higher exercise intensities both before and after training (Figure 4B). SV at rest did not change significantly (92±15 versus 104±19 ml, p=NS) but was significantly higher at peak exercise (110±17 versus 132±27 ml, p<0.05) in response to training. When changes in EDV from rest to exercise were matched (Δ EDV: 6±14 before versus 7±15 ml after training), the extent of increase in SV was significantly greater after compared with before training (14±11 versus 25±9 ml, p<0.05, n=5, Figure 5B). There was a strong correlation (r=0.95) between changes in peak exercise and EDV and SV from the untrained to the trained state (Figure 6). Training had no appreciable effect on cardiac output at rest.

**TABLE 2. Effects of Exercise Training on Left Ventricular Systolic Performance**

<table>
<thead>
<tr>
<th>Subject</th>
<th>EF&lt;sub&gt;rest&lt;/sub&gt; (%)</th>
<th>EF&lt;sub&gt;ex&lt;/sub&gt; (%)</th>
<th>Δ EF (%)</th>
<th>ESV&lt;sub&gt;rest&lt;/sub&gt; (ml)</th>
<th>ESV&lt;sub&gt;ex&lt;/sub&gt; (ml)</th>
<th>Δ ESV (ml)</th>
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±SD ±6.7 ±4.8 ±6.9 ±7.5* ±6.9 ±6.2† ±8 ±12 ±13 ±13 ±14 ±13‡

EF, ejection fraction; ESV, end-systolic volume; ex, peak exercise.

*p<0.005, †p<0.025, ‡p<0.05 compared with initial.

**Figure 2.** Bar graphs of differences in left ventricular ejection fraction (LVEF) response to exercise between older men (before and after exercise training) and sedentary young men. In the untrained state, the increase in LVEF during exercise in older men was significantly lower (p<0.05) than in sedentary young men. In the trained state, LVEF responses to exercise in older men were similar to those observed in sedentary young men.

**Figure 3.** Plots of effects of exercise training on left ventricular contractile function. Panel A: Left ventricular ejection fraction (LVEF) is plotted as a function of mean blood pressure (BP), used as an estimate of afterload. Training had no effect on LVEF at rest. Peak exercise LVEF was significantly higher after than before training despite comparable levels of mean BP. Panel B: Left ventricular end-systolic volume (LVESV)–systolic blood pressure (SBP) relation was shifted to the left at peak exercise in the trained state with a smaller LVESV at a comparable level of SBP suggestive of enhanced contractility.
(6.4±1.1 versus 6.3±0.9 l/min, p=NS). Peak exercise cardiac output, however, was increased by 18% in response to training (15.7±2.6 versus 18.5±4.1 l/min, p<0.025). Calculated arteriovenous O₂ difference at peak exercise was unchanged after training (12.2±5 versus 12±2.7 vol O₂/100 ml, n=9).

End-diastolic diameter increased from 53±8 to 60±8 mm (p<0.05) in response to training. The posterior wall thickness at end diastole was 7±0.8 before and 8±0.8 mm after training (p<0.025). The left ventricular wall thickness-to-radius ratio did not change in response to exercise training (0.27±0.04 before and 0.28±0.03 after training, p=NS). End-systolic diameter was 37±7 before and 42±8 mm after exercise training (p=NS). Posterior wall thickness at end systole was 13±2 before compared with 14±2 mm after training. SBP at rest in these five men was 120±8 and 120±14 mm Hg in the untrained and trained states, respectively. The end-systolic wall stress was 50±13 before and 52±19 g/cm² after (p=NS) exercise training.

**Left Ventricular Systolic Function in Young Subjects**

LVEF at rest was 64±7% and increased to 72±8% during submaximal exercise at work rate equal to 61±11% of peak exercise and to 74±9% at peak exercise (p<0.01 rest versus peak exercise LVEF) in young sedentary men (Figure 2). The extent of increase in EF from rest to exercise was considerably greater in younger subjects than that observed in older men before training (Figure 2).

**Discussion**

The findings of the present study indicate that elderly subjects can adapt to endurance exercise training if the level of training stimulus is adequate as evidenced by significant improvement of maximal aerobic exercise capacity consistent with recent observations. The increase in maximal O₂ uptake in these men appears to be mediated primarily by central adaptations as reflected by enhancement of left ventricular systolic performance and larger SV at peak exercise. However, the smaller increase in heart rate during the submaximal exercise at a given exercise intensity observed in the trained state is also compatible with concomitant peripheral adaptations in our subjects. The lack of increase in the peak exercise arteriovenous oxygen difference after training is most likely due to the mode of exercise (supine) and possibly inadequate sample size. SV during exercise is regulated by a highly integrated mecha-
nism involving alterations in inotropic state and concomitant changes in cardiac loading conditions. The larger peak exercise SV in response to training in our subjects appears to be mediated by at least two mechanisms. One is the combination of left ventricular enlargement due to volume-overload left ventricular hypertrophy (chronic adaptations) and the Frank-Starling mechanism as evidenced by a strong correlation between EDV and SV at peak exercise. The other potential mechanism is the training-induced enhancement of contractility with or without concomitant reduction of afterload. The enhanced left ventricular systolic performance in response to training is reflected by a larger increase in SV at similar changes in EDV during exercise, significantly higher peak exercise EF at comparable levels of mean blood pressure, a smaller ESV at peak exercise despite similar increase in SBP, and a greater increase in SBP during exercise at a given change in ESV. Because the changes in EDV from rest to exercise were not affected by training, higher LVEF and Δ LVEF observed in the trained state are unlikely to result entirely from the changes in preload.

The possibility that reduction of left ventricular wall stress, due to the training-induced increased wall thickness, might have been responsible for the increase in LVEF cannot be excluded. However, this seems unlikely because the endurance training-induced cardiac hypertrophy is modest in magnitude and characterized by proportional increases in left ventricular wall thickness and end-diastolic diameter, as reflected in no significant change in wall thickness-to-radius ratio, typical of volume-overload hypertrophy. This type of hypertrophy does not generally result in reduced wall stress as suggested by our echocardiographic data. However, it is difficult to extrapolate these adaptations observed under basal conditions to those at peak exercise because both SBP and left ventricular geometry are likely to change during exercise, which may in turn alter left ventricular wall stress profoundly. The limitations of the SBP–ESV relation that we used for the assessment of contractility include use of peak-systolic pressure instead of ESP and recent observations suggesting that this relation may not be totally load independent. Nevertheless, despite these limitations, our findings suggest that enhanced inotropic state at peak exercise may be one of the major adaptations to training in older men. One mechanism underlying increased contractile function is simply the higher sympathetic activity at peak exercise in the trained state because our subjects were able to attain higher peak work rates after training. However, this is unlikely because 1) before training, EF increased early with exercise during which the contribution of sympathetic activity is relatively modest but decreased at higher intensities of exercise during which the sympathetic activity would certainly be higher and 2) at a given exercise intensity, EF was higher after than before training even though the magnitude of adrenergic stimulation is expected to be lower at any given work rate in the trained state. An alternate explanation for the improvement in contractility is the possibility of increased sensitivity to catecholamines in the trained state, particularly in view of the known age-related diminished inotropic sensitivity to catecholamines.

The presence of the regional wall motion abnormality during exercise in one of our subjects raises the possibility of occult coronary artery disease. However, when we excluded this subject from the analysis, our results did not differ from those reported here. True VO₂max can be attained only during treadmill exercise testing. Thus, the relative contributions of central and peripheral adaptations to the training-induced increase in VO₂max should be evaluated during a treadmill maximal exercise. The relatively rigorous criteria we used for our subject selection and the level of the training stimulus used for this study may have resulted in selection of “elite older men” whose adaptations to training may not necessarily be representative of those in average elderly populations. However, because our main objective was to characterize the effect of endurance exercise training on the known age-related changes in cardiac function, we attempted to exclude the inherent problem of chronic diseases commonly associated with increasing age and used a training stimulus that would be expected to produce the desired adaptations. Furthermore, our subjects may not necessarily be considered biologically superior, at least in terms of maximal aerobic exercise capacity, because their VO₂max values were not higher than the values reported for the average elderly population.

The increase in daily caloric intake in our subjects during training is consistent with the well-known observation that athletes’ caloric consumptions are greater than those of sedentary persons. The reduction of body fat content after training as reflected in the changes in the skinfold thickness despite an increase in daily caloric intake is due to enhanced fat oxidation during endurance exercise in the trained state.

The age-related impairment of LVEF response to exercise is characterized by a smaller rise in EF and a lack of decrease in ESV at peak exercise in the elderly compared with young subjects. Higginbotham et al recently reported an inverse relation between age and peak exercise EF only in untrained subjects. Our data confirm these findings, showing a small rise in EF during exercise in older men before training compared with a significantly larger increase in EF in sedentary young men. When we excluded the data obtained on two men whose Δ LVEFs were 10% or greater, we found that the results were similar to those for the entire group. It is unlikely that improvements observed after training were due to spontaneous changes in physiological variables. It is well known that VO₂max does not increase spontaneously in healthy subjects. Furthermore, Schocken et al did not report any significant change in EF, even after mild-intensity exercise training in the elderly.
Our findings are compatible with those of Spurgeon et al., who demonstrated prevention of age-related alterations in cardiac contraction in rats by means of exercise training and suggest that the age-related deterioration in left ventricular systolic function can be improved by relatively prolonged vigorous exercise training in older men. A recent study suggested that elderly subjects do not exhibit enhanced left ventricular function in response to exercise training. The major reason for the discrepancy between our findings and those of others seems to be the nature of the training stimulus used in the present study, which was much longer in duration and higher in intensity.

The results of the present study indicate that endurance exercise training can result in enhanced left ventricular systolic performance at peak exercise in older men. Our findings also suggest that mechanisms responsible for the increase in SV at peak exercise in the trained state are left ventricular enlargement and probably enhanced inotropic state.

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