

Effects of Physical Deconditioning After Intense Endurance Training on Left Ventricular Dimensions and Stroke Volume

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To determine the role of preload in maintaining the enhanced stroke volume of upright exercise-trained endurance athletes after deconditioning, six highly trained subjects undergoing upright and supine bicycle ergometry were characterized before and after 3, 8 and 12 weeks of inactivity that reduced oxygen uptake by 20%. During exercise, oxygen uptake, cardiac output by carbon dioxide rebreathing, cardiac dimensions by M-mode echocardiography, indirect arterial blood pressure and heart rate were studied simultaneously. Two months of inactivity resulted in a reduction in stroke volume, calculated as cardiac output/heart rate, during upright exercise ($p < 0.005$) without a significant change during supine exercise. A concomitant decrease in the left ventricular end-diastolic dimension from the trained to the deconditioned state was observed in the upright posture (5.1 ± 0.3 versus 4.6 ± 0.3 cm; $p = 0.02$) but not with

recumbency (5.4 ± 0.2 versus 5.1 ± 0.3 cm; $p = \text{NS}$). There was a strong correlation between left ventricular end-diastolic dimension and stroke volume ($r > 0.80$) in all subjects. No significant changes in percent fractional shortening or left ventricular end-systolic dimension occurred in either position after cessation of training. Estimated left ventricular mass was 20% lower after 3 and 8 weeks of inactivity than when the subjects were conditioned ($p < 0.05$ for both).

Thus, the endurance-trained state for upright exercise is associated with a greater stroke volume during upright exercise because of augmented preload. Despite many years of intense training, inactivity for only a few weeks results in loss of this adaptation in conjunction with regression of left ventricular hypertrophy.

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Endurance exercise training increases maximal cardiac output and stroke volume during exercise (1-4). Both decrease with subsequent bed rest (3). The mechanisms responsible for these changes are controversial but include improvement in left ventricular contractile function, augmented preload, reduced afterload or a combination of these factors. Some investigators have reported trained human subjects (5-7) to have higher cardiac filling pressures and a larger left ventricular end-diastolic volume due to eccentric cardiac hy-

pertrophy, whereas studies in conditioned animals (8,9) have indicated that improved diastolic function may partially account for their higher stroke volume. A few investigators (10,11) have not observed cardiac enlargement after training. Variability in loading conditions attributable to differences in posture is one explanation for this apparent discrepancy. Thus, in previous studies (6,12) some subjects trained or were tested in the upright posture and others during recumbency. Analysis was often cross-sectional, and differences among subjects may have been inherited rather than acquired.

To our knowledge, left ventricular size and function during exercise under different preloads have not been characterized at comparable heart rate and mean blood pressure in the same subjects in the trained and untrained states. Nor has the effect of deconditioning been delineated in subjects who have been training intensely for many years. Our objectives were to define the role of cardiac loading conditions and left ventricular performance in mediating the enhanced exercise stroke volume of endurance athletes and to characterize the rate and extent to which these adaptations are lost during inactivity. Using two independent noninvasive

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techniques, we assessed cardiac output and cardiac dimensions simultaneously during upright and supine bicycle ergometry in six highly trained subjects before and at selected intervals during a detraining regimen that resulted in a 20% decrease in maximal oxygen uptake.

Methods

Subjects, training and detraining regimens. Six highly trained healthy subjects, aged 26 ± 1 years (mean \pm SE), participated in the study. Five were men and one was a woman. There were two bicyclists and four runners. Three of the latter group were former members of a collegiate track team and, at the time of the study, were regarded as quite successful in large local competitive events. Electrocardiograms at rest demonstrated typical findings observed in endurance athletes (13) and included wandering atrial pacemaker in two subjects, ST segment elevation (≥ 1 mm) in three and sinus bradycardia (< 60 beats/min) with pronounced sinus arrhythmia in all six. Voluntary written informed consent was obtained from all subjects after approval of the study by the Washington University Human Studies Committee.

Before cessation of regular activity, all subjects trained approximately 1 hour a day, 5 to 7 days a week, for an average of 10 ± 3 years. This included high intensity interval training, 2 to 3 times a week, in the 6 months immediately preceding inactivity. During deconditioning, subjects were frequently reminded to restrict exertion to the minimum compatible with sedentary daily living. Bed rest was limited to normal nighttime hours.

Maximal oxygen uptake. Maximal oxygen uptake was measured in all subjects on the last day of training and after 3 and 8 weeks of inactivity. In five subjects, additional measurements were made at 12 weeks. Subjects were studied in the upright posture on both a Quinton Instruments electronically braked bicycle ergometer and a motor-driven treadmill. Expired air was collected in meteorologic balloons while the subjects breathed through a mouthpiece connected to a Daniel valve. Oxygen and carbon dioxide concentrations in expired air were measured with a Perkin Elmer 1100 mass spectrometer, and ventilatory volumes with a Parkinson-Cowan dry gas meter calibrated with a Tissot spirometer. An incremental protocol resulting in exhaustion after 5 to 10 minutes was selected. Maximal oxygen uptake was defined as the highest oxygen uptake value observed on either the bicycle or the treadmill. The leveling off criterion (that is, no further rise in oxygen uptake with an increased work rate) was satisfied in all subjects.

Cardiac output, heart rate, stroke volume and blood pressure. Cardiac output (\dot{Q}) was determined as previously described (14), using the carbon dioxide (CO_2) rebreathing technique of Defares (15) as modified by Jerneus et al. (16). This method is based on the equation $\dot{Q} =$

$\dot{V}\text{CO}_2/\text{CO}_{2v} - \text{CO}_{2a}$, where $\dot{V}\text{CO}_2 = \text{CO}_2$ production, $\text{CO}_{2v} =$ venous CO_2 content and $\text{CO}_{2a} =$ arterial CO_2 content. Carbon dioxide production was calculated from mass spectrometric and dry gas meter measurements on expired air. Arterial carbon dioxide content was estimated from the corrected end tidal partial pressure of carbon dioxide as described by Paterson et al. (17). Venous carbon dioxide content was calculated with a computer system (18) by extrapolation to equilibrium of the exponential rise of the end tidal partial pressure of carbon dioxide from breaths 3, 4 and 5 during rebreathing of carbon dioxide from a closed system anesthesia bag containing initial concentrations of 5% carbon dioxide and 95% oxygen (19). With subjects performing both upright and supine submaximal bicycle exercise, two to four nearly simultaneous determinations of cardiac output and oxygen uptake were made at each of two to three steady state work rates lasting approximately 10 minutes each and interspersed with short rest intervals (10 minutes). Work rate progressed in intensity but never beyond the point of hyperventilation, at which time measurement of cardiac output with the technique used might be inaccurate because of lack of equilibrium between arterial and end tidal carbon dioxide (20).

Heart rate was calculated from at least three consecutive RR intervals of the electrocardiogram taken during maximal or submaximal exercise. Cardiac output and electrocardiographic heart rate were consistently determined simultaneously. Stroke volume was calculated as cardiac output/heart rate.

Blood pressure was obtained by the cuff method simultaneously with measurement of cardiac output. Systolic blood pressure was defined by the onset of Korotkoff sounds and diastolic blood pressure by their complete disappearance. Mean blood pressure was calculated from the equation: $1/3$ (systolic blood pressure + $2 \times$ diastolic blood pressure). Total peripheral resistance was determined as: total peripheral resistance ($\text{dynes}\cdot\text{cm}^{-5}$) = (mean blood pressure $\times 80$)/cardiac output.

To avoid the occurrence of hyperventilation that might preclude accurate measurement of cardiac output, higher work rates were reduced in some subjects at 2 and 3 months of inactivity. In young normal subjects, stroke volume during upright exercise is nearly constant over a wide range of heart rates (that is, between approximately 40 and 100% of maximal effort) (21). Therefore, stroke volume values from all work rates were averaged and the mean value was reported for a given posture and state of training. Stroke volume data at a similar heart rate and mean blood pressure were also reported. Mean blood pressure was used as an index of afterload to facilitate interpretation of these results (22). Oxygen uptake, cardiac output, heart rate, mean blood pressure and total peripheral resistance data were reported at two equivalent submaximal work rates during upright (800 ± 86 and $1,033 \pm 71$ kilopond-meter \times min $^{-1}$) and

supine exercise (763 ± 99 and $1,000 \pm 89$ kilopond-meter $\times \text{min}^{-1}$).

Echocardiography. M-mode echocardiograms were recorded during both upright and supine bicycle exercise in the trained state and after 3 and 8 weeks of inactivity. An Ekoline-20A ultrasonoscope (Smith-Kline) with a 2.25 MHz unfocused transducer, interfaced to a Cambridge multi-channel strip chart recorder, was used to obtain these tracings as previously described (23). Two to four tracings were obtained during each period of submaximal exercise; all were taken during determination of cardiac output. The position of the transducer was maintained perpendicular to the chest wall so that the left ventricle was visualized just below the tips of the mitral valve leaflets. Subjects were assisted in restricting movement of the torso. To minimize bias, echocardiographic data were coded and selected for inclusion in blinded fashion on the basis of anatomic landmark clarity and technical quality, before measurement of cardiac dimensions. For enhancement of reliability, many more beats were measured than is customary. The mean number of measured beats/subject ranged from 16 ± 5 to 34 ± 7 in the upright posture and 18 ± 5 to 28 ± 5 in the supine posture at various stages of training. Tracings from one subject were judged technically inadequate and not included in the results.

The left ventricular end-diastolic dimension was defined as the distance between the left side of the interventricular septum and the posterior endocardial wall at the onset of the QRS complex of the electrocardiogram. The left ventricular end-systolic dimension was taken to be the smallest perpendicular distance from the posterior endocardial wall endocardium to the left side of the interventricular septum. Left ventricular fractional shortening percent, which cor-

relates closely with ejection fraction, was calculated as: fractional shortening percent ($[\text{left ventricular end-diastolic dimension} - \text{left ventricular end-systolic dimension}] \times 100$)/left ventricular end-diastolic dimension. Systolic blood pressure/left ventricular end-systolic dimension was used as an index of left ventricular contractile function (24). Left ventricular mass was estimated from echocardiograms taken at rest as described by Troy et al. (25) with the use of measurements of left ventricular end-diastolic dimension and left ventricular posterior wall thickness at end-diastole. Left ventricular posterior wall thickness was defined as the distance between the electrically damped posterior wall epicardial signal and the undamped endocardial signal.

Poliner et al. (26) showed that there was little difference in left ventricular dimensions or contractility over the range of submaximal exercise heart rates achieved by our subjects. We also found no consistent change in either of these variables at the exercise intensities selected. Therefore, average left ventricular end-diastolic and end-systolic dimensions and other derived values from all work rates in each posture and activity state were reported. However, to control for the effect of heart rate on preload and contractility, the values for left ventricular end-diastolic dimension and systolic blood pressure/left ventricular end-systolic dimension were compared at equivalent heart rates. To define the role of preload, the subjects were studied in the upright and supine postures. The variability of echocardiographic measurements was characterized by determination of the mean coefficient of variation for the echocardiographic variables during steady state exercise. Coefficient of variation was defined as standard deviation/mean $\times 100\%$. The mean coefficient of variation was determined for each posture and for each state of training.

Table 1. Oxygen Uptake and Hemodynamic Values at Two Constant Submaximal Work Rates in Each Posture for Subjects in the Trained State and at Selected Interval Times During Detraining

Physiologic State	Work Rate I					Work Rate II				
	$\dot{V}O_2$ (liters/min)	HR (beats/min)	Mean BP (mm Hg)	\dot{Q} (liters/min)	TPR (dynes \cdot s \cdot cm $^{-5}$)	$\dot{V}O_2$ (liters/min)	HR (beats/min)	Mean BP (mm Hg)	\dot{Q} (liters/min)	TPR (dynes \cdot s \cdot cm $^{-5}$)
A. Upright Exercise										
Trained	1.52 ± 0.12	111 ± 6	86 ± 3	16.3 ± 0.8	424 ± 14	1.87 ± 0.10	122 ± 6	91 ± 2	17.5 ± 1.0	418 ± 14
Detrained										
3 weeks	1.51 ± 0.12	115 ± 7	93 ± 5	16.1 ± 1.2	464 ± 16	1.87 ± 0.13	$133 \pm 6^*$	95 ± 2	17.9 ± 0.9	427 ± 10
8 weeks	1.49 ± 0.14	$124 \pm 5^*$	92 ± 2	15.1 ± 1.0	496 ± 28	1.82 ± 0.11	$142 \pm 4^*$	95 ± 2	17.1 ± 1.0	450 ± 26
12 weeks	1.52 ± 0.14	$123 \pm 8^*$	$96 \pm 3^*$	16.7 ± 0.7	466 ± 13	1.95 ± 0.10	$138 \pm 8^*$	99 ± 4	17.4 ± 0.8	451 ± 14
B. Supine Exercise										
Trained	1.40 ± 0.16	107 ± 6	$92 \pm 3^\dagger$	16.4 ± 1.0	454 ± 21	1.81 ± 0.15	121 ± 6	$98 \pm 4^\dagger$	18.3 ± 1.0	434 ± 22
Detrained										
3 weeks	1.38 ± 0.17	112 ± 6	94 ± 1	16.9 ± 1.2	454 ± 33	1.74 ± 0.18	$126 \pm 5^*$	101 ± 3	18.1 ± 1.1	454 ± 23
8 weeks	1.40 ± 0.18	$123 \pm 7^*$	91 ± 2	18.1 ± 1.9	425 ± 46	1.80 ± 0.17	$134 \pm 4^*$	98 ± 3	$19.9 \pm 1.5^{\dagger*}$	405 ± 26
12 weeks	1.51 ± 0.18	$119 \pm 7^*$	93 ± 2	18.2 ± 1.4	413 ± 32	1.84 ± 0.17	$135 \pm 5^*$	98 ± 3	19.3 ± 1.1	415 ± 22

* $p < 0.05$ vs. trained; $^\dagger p < 0.05$ for supine compared with upright posture. BP = blood pressure; HR = heart rate. \dot{Q} = cardiac output; TPR = total peripheral resistance; $\dot{V}O_2$ = maximal oxygen uptake.

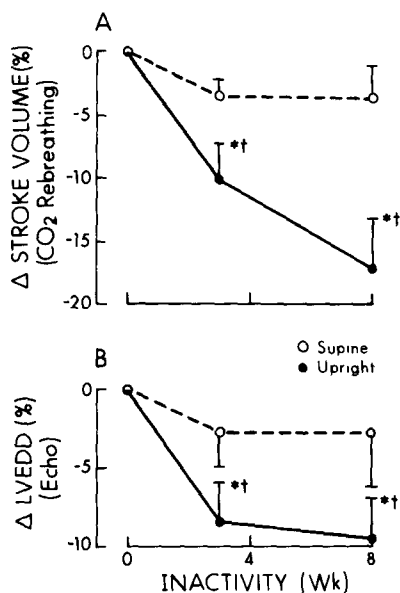


Figure 1. A, Percent change in exercise stroke volume in the upright and supine postures as a function of duration of inactivity. B, Percent change in left ventricular end-diastolic dimension (LVEDD) during exercise in the upright and supine postures as a function of duration of inactivity. * $p < 0.005$ compared with results for subjects in the trained state; † $p < 0.05$ upright compared with supine posture. Echo = echocardiogram.

Statistics. Results were expressed as means \pm SE. One-way analysis of variance followed by Newman-Keuls multiple comparison post hoc tests was used to assess the statistical significance of differences in oxygen uptake, maximal oxygen uptake, heart rate, blood pressure, cardiac output, stroke volume, left ventricular end-diastolic dimension, left ventricular end-systolic dimension, fractional shortening percent and left ventricular mass with time. Student's *t* test for paired observations was used to evaluate statistical differences between upright and supine exercise. The significance of the relation between stroke volume and left ventricular end-diastolic dimension was determined by least squares linear regression analysis.

Results

Maximal oxygen uptake. Initial maximal oxygen uptake was quite high (4.47 ± 0.21 liters/min and 62.7 ± 4.0 ml/min per kg). Deconditioning for 21 days resulted in a significant decline to 4.18 ± 0.21 liters/min ($p < 0.01$). At 2 months it had fallen 20% to 3.56 ± 0.15 liters/min ($p < 0.01$ compared with the value during training). No significant further reduction in maximal oxygen uptake was evident at 12 weeks. There were no changes in oxygen uptake at the same absolute work rates at any time during the study (Table 1).

Exercise cardiac output, heart rate and stroke volume (Table 1). Results of preliminary studies from our labo-

ratory demonstrated a mean coefficient of variation of 6.5% for triplicate determinations of exercise cardiac output during 100 exercise periods. The test-retest correlation in 10 subjects studied on 2 separate days in the same state of training was 0.98. There was no significant effect of exercise intensity on stroke volume. In the trained state, upright and supine exercise cardiac output and heart rate were similar during submaximal exercise. Hence, mean stroke volume at all work rates was not significantly different for the two postures (151 ± 10 ml upright versus 155 ± 12 ml supine). During inactivity, there was a tendency for exercise cardiac output at the same absolute work rate to increase in the supine posture resulting in a slightly higher exercise cardiac output in the supine versus the upright position after 2 months of deconditioning. Exercise heart rate at the same work rate rose progressively with duration of inactivity. However, the downward adjustment in the highest and lowest work rates as inactivity progressed resulted in only a slight rise in mean heart rate when results from all work rates were averaged.

The effect of inactivity on exercise stroke volume was posture dependent (Fig. 1A). Three weeks after cessation of training, mean upright stroke volume had declined significantly (136 ± 10 versus 151 ± 10 ml for subjects in the trained state; $p < 0.005$). This decrease was even more pronounced at 8 and 12 weeks (125 ± 6 and 129 ± 9 ml, respectively; both $p < 0.005$). A statistically significant fall in mean stroke volume did not occur in the supine posture (149 ± 12 ml at 3 weeks and at 8 weeks and 144 ± 12 ml at 12 weeks of inactivity). Hence, with inactivity, stroke volume was lower and its percent reduction greater with upright compared with supine posture.

Left ventricular dimensions, mass and fractional shortening. Paralleling the inactivity-induced decrease in stroke volume that occurred only with upright exercise, there

Figure 2. The left ventricular end-diastolic dimension (LVEDD) during upright and supine exercise as a function of heart rate in the trained and detrained states. * $p < 0.005$ compared with results for subjects in the trained state.

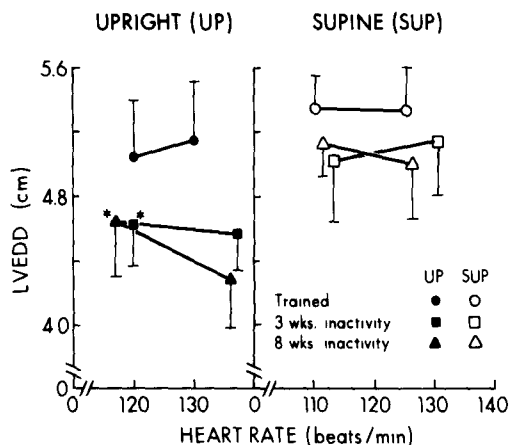
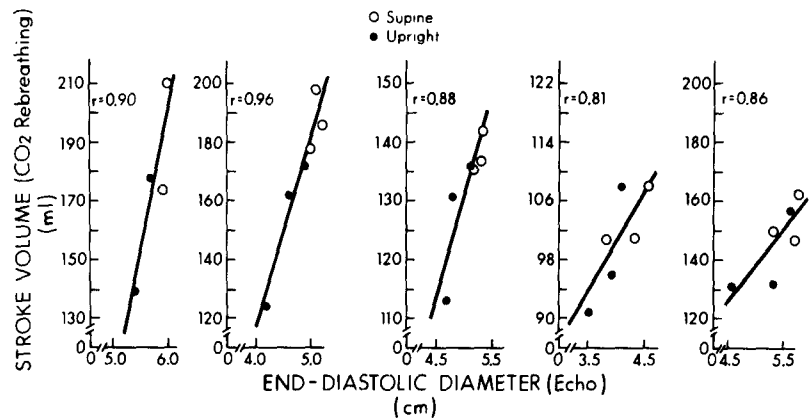


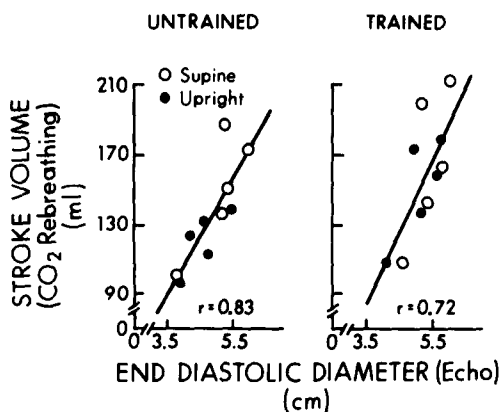
Figure 3. Individual data showing correlations between the left ventricular end-diastolic dimension on echocardiogram (Echo) and stroke volume during upright and supine exercise in the trained and 3 and 8 week detrained states.



was a decrease in mean upright left ventricular end-diastolic dimension that appeared after 3 weeks of detraining and was slightly more pronounced after 8 weeks of inactivity (4.6 ± 0.3 and 4.5 ± 0.3 cm, respectively, compared with 5.1 ± 0.3 cm for the trained state; $p = 0.03$ and 0.02 , respectively). Cessation of training had no significant effect on mean left ventricular end-diastolic dimension with supine exercise after either 3 or 8 weeks (both 5.1 ± 0.3 cm compared with 5.4 ± 0.2 cm in the trained state). Throughout inactivity, the percent change of mean left ventricular end-diastolic dimension was significant for upright but not supine exercise (Fig. 1B). Inactivity was associated with a decrement in left ventricular end-diastolic dimension for upright posture even when heart rate was similar (Fig. 2).

Despite differences in posture and training state, stroke volume was highly correlated with left ventricular end-diastolic dimension in each individual subject (Fig. 3). The correlation coefficient ranged from 0.80 to 0.95. The overall correlation between stroke volume and left ventricular end-diastolic dimension was statistically significant with subjects in both the untrained and the trained state (both $p < 0.02$) (Fig. 4).

Figure 4. Overall correlations between the left ventricular end-diastolic dimension and stroke volume during upright and supine exercise in the trained and 8 week detrained states. Echo = echocardiogram.



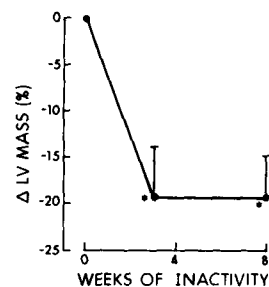
The changes in left ventricular end-systolic dimension induced by inactivity were not statistically significant. Three weeks after cessation of training, upright end-systolic dimension was 2.7 ± 0.2 versus 2.9 ± 0.3 cm initially. Supine end-systolic dimension was identical under both conditions (3.1 ± 0.3 cm). Eight weeks after the subjects became sedentary, left ventricular end-systolic dimension was 2.6 ± 0.1 cm in the upright and 3.0 ± 0.5 cm in the supine posture. Fractional shortening percent remained similar for both postures throughout the study.

Deconditioning was associated with a regression of left ventricular hypertrophy. Left ventricular posterior wall thickness declined progressively from 0.8 ± 0.04 cm in the trained state to 0.7 ± 0.1 cm after 3 weeks and 0.6 ± 0.1 cm after 8 weeks ($p < 0.05$) of inactivity. However, the reduction in estimated left ventricular mass appeared to be complete after 3 weeks (124 ± 14 g at 3 weeks and 124 ± 15 g at 8 weeks, both $p < 0.05$ compared with 154 ± 12 g for the trained state) (Fig. 5).

Reproducibility of echocardiographic results. Echocardiographic variability for the left ventricular end-diastolic dimension was only slightly greater with exercise than at rest (23). Coefficients of variation ranged from 3.6 to 4.7% at a given exercise intensity for the supine posture and from 4.3 to 5.5% for upright exercise.

Mean blood pressure, total peripheral resistance and systolic blood pressure/left ventricular end-systolic di-

Figure 5. Percent change in left ventricular (LV) mass as a function of duration of inactivity. * $p < 0.05$ compared with results for subjects in the trained state.



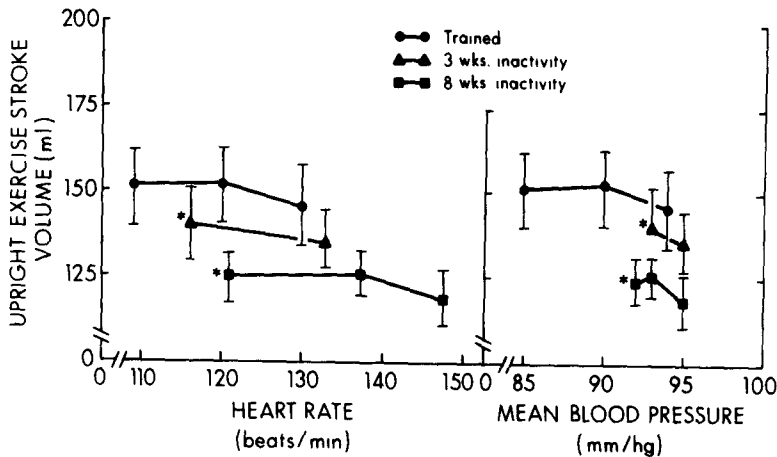


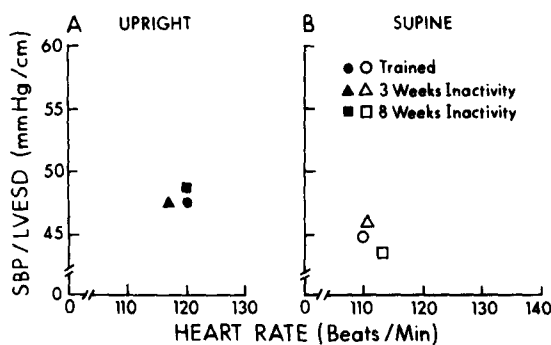
Figure 6. Upright exercise stroke volume as a function of heart rate and mean blood pressure in the trained state and after 3 and 8 weeks of inactivity. * $p < 0.005$ compared with results for subjects in the trained state.

mension (Table 1). For the trained state, mean exercise blood pressure was slightly higher in the supine than in the upright posture. Deconditioning resulted in a significant elevation of mean blood pressure with upright ($p = 0.04$) but not with supine exercise at the lowest work rate. A trend for similar results was evident at the higher work rate ($p = 0.15$). Therefore, for the detrained state, blood pressure in the upright and supine postures was nearly equal. Total peripheral resistance during upright exercise also tended to increase with detraining ($p = 0.06$ and 0.17 at work rates I and II, respectively). However, stroke volume during upright exercise was lower with subjects in the detrained state even when heart rate and mean blood pressure were similar (Fig. 6). The decline of upright exercise stroke volume with detraining was not associated with significant changes in systolic blood pressure/left ventricular end-systolic dimension at a similar submaximal heart rate (Fig. 7).

Discussion

Our results indicate that detraining even without increased bed rest leads to reductions in stroke volume and

Figure 7. The relation between the systolic blood pressure-left ventricular end-systolic dimension ratio (SBP/LVESD) and heart rate during upright and supine exercise in the trained and 8 week detrained states.



left ventricular end-diastolic dimension with upright but not supine exercise in young healthy subjects previously well conditioned by many years of endurance training in the upright posture. The linear relation between left ventricular end-diastolic dimension and stroke volume, independent of posture or training status, and the absence of a significant decrease in stroke volume or end-diastolic dimension with supine exercise at comparable work rates even after 2 months of inactivity suggest that augmented preload is a critical determinant of the adaptive increase of stroke volume in response to regularly performed upright endurance exercise. In this context, a linear relation between preload (end-diastolic volume) and left ventricular stroke work has been reported recently (27).

Effects of heart rate, afterload and contractility. Other factors besides preload that could affect stroke volume include changes in heart rate, afterload and myocardial contractility. The relative tachycardia induced by deconditioning does not explain the observed fall of upright exercise stroke volume. After inactivity, stroke volume and left ventricular end-diastolic dimension were greater with supine than with upright exercise at any given heart rate. Changes in afterload are also not likely to explain these findings. Afterload can be approximated by estimates of left ventricular systolic wall stress. Wall stress is directly proportional to left ventricular dimensions and pressure and indirectly related to left ventricular posterior wall thickness (28). In our subjects, there was no significant effect of inactivity on left ventricular end-systolic dimension during upright exercise. Although we did not measure left ventricular pressure, stroke volume at a given mean blood pressure during exercise was lower after detraining. Mean blood pressure is considered to be a major determinant of afterload (22). Inactivity resulted in a somewhat higher mean blood pressure with upright exercise of the same absolute intensity. However, an acute increase in afterload results in a decline in stroke volume because of greater end-systolic volume which, in the normal heart, is generally followed by a secondary rise in end-diastolic volume (29). In our subjects the

decrease in upright stroke volume with detraining was associated with a reduced left ventricular end-diastolic dimension. Therefore, even at the same absolute work rate, it is unlikely that the lower stroke volume was mediated by increased afterload. We did not directly estimate left ventricular systolic wall stress because of limitations and difficulties in making accurate simultaneous measurements of blood pressure and left ventricular posterior wall thickness during exercise. To our knowledge, noninvasive ultrasound techniques for estimating left ventricular systolic wall stress have been validated only at rest (30).

There was no evidence that the lower stroke volume was caused by deterioration of myocardial contractility. Both fractional shortening and the systolic blood pressure/left ventricular end-systolic dimension ratio at a given heart rate during upright exercise were unchanged after inactivity.

Mechanisms. Although endurance training is known to enhance exercise stroke volume in humans (1-4), the mechanisms responsible for this adaptation are controversial. Bar-Shlomo et al. (12) reported that ventricular performance during maximal supine exercise is enhanced in athletes. Rerych et al. (6) found no change in ejection fraction but a substantially larger end-diastolic volume during upright exercise after training. Both left ventricular performance and end-diastolic volume were found to be greater in athletes undergoing semirecumbent exercise (31). Such disparities may be attributable in part to the differential effects of posture on preload during exercise, as suggested by our results. However, increased cardiac filling during upright exercise appears to be one of the important mechanisms accounting for the greater upright exercise stroke volume seen in athletes. Volume overload left ventricular hypertrophy may play a role in maintaining relatively constant left ventricular wall stress under increased loading conditions (13).

Factors that potentially affect cardiac filling during exercise include total blood or plasma volume, pericardial and myocardial compliance, active diastolic relaxation and arteriolar and venomotor tone. Rerych et al. (6) found that swimming training increased total body blood volume by 35%. However, others (3,32) observed much smaller changes after physical conditioning. The effect of acute expansion of blood volume on maximal cardiac output or exercise capacity is variable (33,34), possibly because of differences in the level of training. Thus, we are reluctant to attribute our results solely to a decrease in blood or plasma volume with detraining. It appears possible that endurance training in the upright posture may result in specific adaptations such as increased venomotor tone that facilitate cardiac filling during upright exercise. In addition, exercise-induced cardiac dilation could produce distention of the pericardium such that pericardial compliance remains greater after detraining than in the untrained state. In this context, Stray-Gundersen et al. (35) recently observed that pericardiectomy

resulted in a rise in exercise stroke volume in the untrained dog.

Previous studies. Our results are consistent with those of Bevegård et al. (5), who showed that, compared with untrained subjects, well trained cyclists had higher cardiac filling pressures and more modest relative differences between exercise stroke volume in the supine versus the upright position. Furthermore, Saltin et al. (3) reported no effect of training on average stroke volume at similar absolute work rates in the supine posture, but an increase in stroke volume of more than 10% with upright exercise. Convertino et al. (36) found that bed rest was associated with a greater decline in peak oxygen uptake with upright compared with supine posture.

Cardiac enlargement has been reported (37,38) to persist for many years after previous training and athletic competition. However, this conclusion was based largely on radiographic criteria that may be insensitive for detecting modest changes in left ventricular dimensions. The current results confirm and extend previous observations from our laboratory (7) and indicate that physiologic cardiac hypertrophy is rapidly and perhaps completely reversible even after many years of strenuous endurance training. In previous studies in experimental animals (39-41) a similar rapid regression of cardiac hypertrophy of diverse etiology (for example, in response to aortic banding, hyperthyroidism, chronic exercise) was observed after removal of the inciting stimulus.

Limitations. The limitations of M-mode echocardiograms are well recognized. However, the variability of the indexes measured in our study was small. Furthermore, the parallel changes of left ventricular end-diastolic dimension and stroke volume determined by independent techniques support the validity of the M-mode echocardiographic results. Beat to beat variability during upright exercise, as assessed by the coefficient of variation, was only 2 to 3% greater than that seen during supine rest. Some of the variability is likely to be biologic and associated with effects of the increased depth and frequency of respiration during exercise, although left ventricular dimensions vary less with respiration than do those of the right ventricle (42). We recognize that the number of subjects in our study was relatively small. However, it is difficult to recruit highly trained athletes who are willing to stop exercising for even a few weeks.

Conclusions. The results of this study obtained with two independent noninvasive methods indicate that augmentation of exercise stroke volume in endurance athletes who have trained in the upright posture reflects enhanced preload, and that the decrease of stroke volume with inactivity becomes detectable with upright posture as a consequence of decreased cardiac filling. Deconditioning, even after many years of previous intense training, leads to the rapid loss of augmented preload and concomitant regression of left ventricular hypertrophy.

References

1. Ekblom B, Åstrand P-O, Saltin B, Stenberg J, Wallstrom B. Effect of training on circulatory response to exercise. *J Appl Physiol* 1968;24:518-28.
2. Scheuer J, Tipton CM. Cardiovascular adaptations to physical training. *Annu Rev Physiol* 1977;39:221-51.
3. Saltin B, Blomqvist CG, Mitchell JH, Johnson RL, Wildenthal K, Chapman CB. Response to exercise after bed rest and after training. A longitudinal study of adaptive changes in oxygen transport and body composition. *Circulation* 1968;38(suppl VII):VII-1-78.
4. Clausen JP. Effect of physical training on cardiovascular adjustments to exercise in man. *Physiol Rev* 1977;57:779-815.
5. Bevegård S, Holmgren A, Jansson B. Circulatory studies in well trained athletes at rest and during heavy exercise with special reference to stroke volume and the influence of body position. *Acta Physiol Scand* 1963;57:26-50.
6. Rerych SK, Scholz PM, Sabiston DC, Jones RH. Effects of exercise training on left ventricular function in normal subjects: a longitudinal study by radionuclide angiography. *Am J Cardiol* 1980;45:244-52.
7. Ehsani AA, Hagberg JM, Hickson RC. Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. *Am J Cardiol* 1978;42:52-6.
8. Penpargkul S, Scheuer J. The effect of physical training upon the mechanical and metabolic performance of the rat heart. *J Clin Invest* 1970;49:1859-68.
9. Bersohn M, Scheuer J. Effects of physical training on end-diastolic volume and myocardial performance of isolated rat hearts. *Circ Res* 1977;40:510-6.
10. Peronnet F, Perrault H, Cleroux J, et al. Electro- and echocardiographic study of the left ventricle in man after training. *Eur J Applied Physiol* 1980;45:125-30.
11. Wolfe LA, Cunningham DA, Rechnitzer PA, Nichol PM. Effects of endurance training on left ventricular dimensions in healthy men. *J Appl Physiol* 1979;47:207-12.
12. Bar-Shlomo B-Z, Druck MN, Morch JE, et al. Left ventricular function in trained and untrained healthy subjects. *Circulation* 1982;65:484-8.
13. Huston TP, Puffer JC, Rodney WM. The athletic heart syndrome. *N Engl J Med* 1985;313:24-32.
14. Coyle EF, Martin WH, Ehsani AA, et al. Blood lactate threshold in some well-trained ischemic heart disease patients. *J Appl Physiol* 1983;54:18-23.
15. Defares JG. Determination of $P_{50}CO_2$ from the exponential CO_2 rise during rebreathing. *J Appl Physiol* 1958;13:159-64.
16. Jernerus R, Lundin G, Thompson D. Cardiac output in healthy subjects determined with a CO_2 rebreathing method. *Acta Physiol Scand* 1963;59:390-9.
17. Paterson DH, Cunningham DH. Comparison of methods to calculate cardiac output using the CO_2 rebreathing methods. *Eur J Applied Physiol* 1976;35:223-30.
18. McHardy GJR. Relationship between the difference in pressure and content of carbon dioxide in arterial and venous blood. *Clin Sci* 1967;32:299-309.
19. Jones NL, Campbell EJM, Edwards RHT, Wilkoff WG. Alveolar to blood CO_2 difference during rebreathing in exercise. *J Appl Physiol* 1969;27:356-60.
20. Jones NL, Campbell EJM, Edwards RHT, Robertson DG, eds. Conduct of stage 2, 3 and 4 exercise tests. In: *Clinical Exercise Testing*. Philadelphia: WB Saunders, 1975:130-51.
21. Åstrand P-O, Cuddy TE, Saltin B, Stenberg J. Cardiac output during maximal and submaximal work. *J Appl Physiol* 1964;19:268-74.
22. Jaski BE, Fifer MA, Wright RF, Braunwald E, Colucci WS. Positive inotropic and vasodilator actions of milrinone in patients with severe congestive heart failure. *J Clin Invest* 1985;75:643-9.
23. Ehsani AA, Martin WH, Heath GW, Coyle EF. Cardiac effects of prolonged and intense exercise training in patients with coronary artery disease. *Am J Cardiol* 1982;50:246-54.
24. Sagawa K, Suga H, Shoukas AA, Bakalar DM. End-systolic pressure/volume ratio—a new index of ventricular contractility. *Am J Cardiol* 1977;40:748-53.
25. Troy BL, Pombo J, Rackley CE. Measurement of left ventricular wall thickness and mass by echocardiography. *Circulation* 1972;45:602-11.
26. Poliner LR, Dehmer GJ, Lewis SE, Parkey RW, Blomqvist CG, Willerson JT. A comparison of the responses to exercise in the upright and supine positions. *Circulation* 1980;62:528-34.
27. Glower DD, Spratt MD, Snow ND, et al. Linearity of the Frank-Starling relationship in the intact heart: the concept of preload recruitable stroke work. *Circulation* 1985;71:994-1009.
28. Grossman W, Jones D, McLaurin LP. Wall stress and patterns of hypertrophy in the human left ventricle. *J Clin Invest* 1975;56:56-64.
29. Braunwald E, Sonnenblick EH, Ross J. Contraction of the normal heart. In: Braunwald E, ed. *Heart Disease. A Textbook of Cardiovascular Medicine*. Philadelphia: W.B. Saunders, 1984:409-46.
30. Reichel N, Wilson J, St. John Sutton M, Plappert T, Goldberg S, Hirshfeld JW. Noninvasive determination of left ventricular end-systolic stress: validation of the method and initial application. *Circulation* 1982;65:99-108.
31. Osbakken M, Locko R. Scintigraphic determination of ventricular function and coronary perfusion in long-distance runners. *Am Heart J* 1984;108:296-304.
32. Oscai LB, Williams BT, Hertig BA. Effect of exercise on blood volume. *J Appl Physiol* 1968;24:622-4.
33. Robinson BF, Epstein SE, Kahler RL, Braunwald E. Circulatory effects of acute expansion of blood volume: studies during maximal exercise and at rest. *Circ Res* 1966;19:26-32.
34. Kanstrup I-L, Ekblom B. Acute hypervolemia, cardiac performance, and aerobic power during exercise. *J Appl Physiol* 1982;52:1186-91.
35. Stray-Gundersen J, Haidet GC, Musch TI, Orday GA, Mitchell JH. The effect of pericardiectomy on stroke volume during maximal exercise in the untrained dog (abstr). *Physiologist* 1983;26:7.6.
36. Convertino V, Hung J, Goldwater D, DeBusk RF. Cardiovascular responses to exercise in middle-aged men after 10 days of bedrest. *Circulation* 1982;65:134-40.
37. Pyorala K, Karvonen MJ, Taskinen P, Takkunen J, Kyronseppä H, Peltokallio P. Cardiovascular studies on former endurance athletes. *Am J Cardiol* 1967;20:191-205.
38. Saltin B, Grimby G. Physiological analysis of middle-aged and old former athletes. Comparison with still active athletes of the same ages. *Circulation* 1968;38:1104-15.
39. Leon AS, Bloor CM. Effects of exercise and its cessation on the heart and its blood supply. *J Appl Physiol* 1968;24:485-90.
40. Beznak M, Korecky B, Thomas G. Regression of cardiac hypertrophies of various origin. *Can J Physiol Pharmacol* 1969;47:579-86.
41. Cutilletta AF, Dowell RT, Rudnik M, Arcilla RA, Zak R. Regression of myocardial hypertrophy. I. Experimental model, changes in heart weight, nucleic acids, and collagen. *J Mol Cell Cardiol* 1975;7:767-81.
42. Goldblatt A, Harrison DC, Glick G, Braunwald E. Studies on cardiac dimensions in intact, unanesthetized man. II. Effect of respiration. *Circ Res* 1963;13:455-60.