Cardiovascular responses to static exercise in distance runners and weight lifters

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A VARIETY OF INVESTIGATIONS into static exercise have demonstrated that significant increases in heart rate, blood pressure, myocardial contractility, and cardiac output occur (14, 16, 28). However, the responses to static exercise are modified in certain states in which the function of the myocardium is depressed. Thus in coronary artery disease or after β-adrenergic blockade, cardiac output increases less while filling pressure and systemic vascular resistance increase more (1, 7, 11, 16). It is not known whether the cardiovascular responses to static exercise are modified by physiological factors such as exercise training. Therefore, three groups of athletes including long-distance runners, competitive and amateur (noncompetitive) weight lifters, and age- and sex-matched controls were studied with hemodynamic and echocardiographic methods to determine if these three types of training programs altered their responses to static exercise.

METHODS

Subject characteristics and resting studies. Sixty white male subjects were studied. This included the following groups: 12 long-distance runners (LDR), 7 competitive weight lifters (CWL), 7 amateur (noncompetitive) weight lifters (AWL), 10 light controls (LC), and 14 heavy controls (HC). For some portions of the study the 24 males in the light and heavy control groups (CNT) were combined. The criteria for selection and characteristics of each group have been detailed previously (15). In brief, the long-distance runners averaged 79 mi/wk for a mean of 6.4 yr. The competitive weight-lifter group was composed of both Olympic and power (AAU) lifters who had competed for an average of 8.9 and 5.8 yr, respectively. The body builders combined repetitive weight lifting with light running and sought to achieve maximal muscle mass rather than maximal strength.

The body surface areas were estimated on each individual by the DuBois nomogram from the weight in kilograms and height in centimeters. In addition, each subject's resting heart rate was determined by an electrocardiographic rhythm strip as an average of 15 beats over a 5-min period of supine rest. The resting systolic, diastolic, and calculated mean arterial blood pressure (= pulse pressure × 0.33 + diastolic arterial pressure) were also obtained with a sphygmomanometer cuff as previously described (15).

Echocardiographic methods and calculations. Echocardiographic measurements were made in the 20° upright or a 20° upright, 30° left lateral decubitus position with an M-mode echocardiograph equipped with a 2.25 MHz, 4-7-cm focused transducer (Unirad series C). Within each group there was no significant difference in hemodynamic response, comparing both positions for echocardiographic recordings. Details of transducer placement and requirements for quality of the echocardiograms for adequate unambiguous interpretation have been stated previously (15). After an echogram was obtained at rest, static exercise was performed during which the transducer was maintained in the same position.
without removal from the chest wall until the completion of exercise.

The left ventricular internal diameters during systole (LVIDs) and diastole (LVIDd) were recorded at rest and during each minute of exercise according to the method of Troy et al. (30) with careful attention to accurate determination of the left ventricular endocardial septal and posterior wall surfaces. In addition, the ejection time (ET) was recorded as the time from the peak of the QRS to the time of maximal anterior excursion of the posterior left ventricular wall minus 50 ms for the time of isovolumic systole. Measurements at each time period were recorded as an average of three cardiac cycles by two independent observers (JCL and ARK) to the nearest 0.5 mm. Discrepancies in the two sets of measurements by more than 5% (generally 0.5-1.0 mm) were carefully reevaluated by both individuals.

From the basic hemodynamic (blood pressure and heart rate) and echocardiographic data during rest and exercise the following calculations were made.

double product (DP) = systolic arterial pressure \times \text{heart rate (HR)}

left ventricular end-diastolic volume index (2) \( \text{LVVID} = \left( \frac{7.0}{2.4} \times \text{LVIDd}^3 \right) \times \text{body surface area (BSA)}^{-1} \) (29)

left ventricular end-systolic volume index (3) \( \text{LVSIs} = \left( \frac{7.0}{2.4} \times \text{LVIDs}^3 \right) \times \text{BSA}^{-1} \) (29)

stroke volume (SV) index (SVI) = \( \text{LVVID} - \text{LVSIs} \) (4)

stroke volume index (SVI) = \( \text{LVVID} - \text{LVSIs} \) (LVIDd - LVIDs)


\( \frac{\text{SVI}}{\text{HR} \times \text{BSA}^{-1}} \) (7)

velocity of circumferential fiber shortening (9) \( \text{VCF} = \left( \text{LVIDd} - \text{LVIDs} \right) \times \text{LVIDd}^{-1} \times \text{ET} \)

Exercise protocol. In the resting supine position maximal voluntary contraction (MVC) was determined with the dominant hand by having each individual briefly (~1 s) grip a hand dynamometer (Stoelting) as forcefully as possible with the arm extended and supported. This procedure was repeated twice, and the strongest contraction was recorded as the MVC. The subject then rested 5 min with the intermittent blood pressure and heart rate recordings to ensure a steady state. Exercise at 40% MVC was then begun and continued until fatigue, defined as an inability to further grip to 40% MVC. During exercise the electrical signal from a strain gauge attached to the hand grip dynamometer was put into an oscilloscope providing the subject with a constant visual feedback to precisely maintain the force of contraction. Before and during exercise the individual was cautioned against doing a Valsalva maneuver or contracting any other muscles of his body. One observer constantly monitored the oscilloscope screen to encourage precise grip maintenance. A second person constantly observed the exercising subject to encourage muscle relaxation other than the exercising forearm and to caution against a Valsalva maneuver. Blood pressure, heart rate, and echocardiographic measurements were made at 30-s intervals throughout exercise stopping at the time of fatigue.

Statistical analysis. The hemodynamic variables, echocardiographic measurements, and the derived volumes, outputs, resistances, and indices of contractile state were analyzed between all the groups at rest \( t_0 \), at half time of fatigue \( t_{1/2} \), and at the time of fatigue \( t_{\text{max}} \) by a two-way analysis of variance followed by an appropriate repeated measures test (Duncan's Rank Sum Test) to assess for nonrandom variation. A similar type of analysis was performed on each group individually over time. Differences were considered to be statistically significant at \( P \leq 0.05 \).

RESULTS

Group characteristics. The age and anthropometric data for these athletes and control subjects have been previously published (15). Briefly, the ages (yr) of all the groups were similar (CWL: 26.9 ± 1.2, AWL: 28.1 ± 2.7, HC: 26.9 ± 1.0, LC: 24.9 ± 0.9, LDR: 28.5 ± 1.6). The body weights (kg) were similar for the competitive lifters (89.8 ± 3.4), the amateur lifters (82.6 ± 4.7), and the heavy controls (89.1 ± 2.6). The weights of the runners (67.7 ± 2.1) and the light controls (68.4 ± 1.4) were similar. The body surface area (m²) were similar for the competitive lifters (2.11 ± 0.05), the amateur lifters (2.03 ± 0.03), and the heavy controls (2.11 ± 0.03). Likewise the body surface areas were the same for the light controls (1.85 ± 0.03) and the long-distance runners (1.83 ± 0.04).

Static exercise strength and endurance times. Although the weight lifters demonstrated slightly greater maximal voluntary contractions, there were no significant differences between any of the groups studied (Table 1). Likewise, at 40% MVC there were no significant differences between any of the groups studied (Table 1). The endurance times were 2.5 ± 0.2 min for the runners, 2.1 ± 0.1 min for the combined control group, 1.9 ± 0.3 min for the amateur lifters, and 1.7 ± 0.1 min for the competitive lifters. These small differences were not significantly different between the control group and either the runners or the lifters.

| TABLE 1. Maximum voluntary contraction and 40% maximum voluntary contraction |
|------------------|---|---|---|---|---|
|                  | CWL| AWL| HC | LC | LDR |
| MVC, kg          | 52 ± 1.5 | 52 ± 2.5 | 50 ± 2.0 | 45 ± 2.5 | 46 ± 2.3 |
| 40% MVC, kg      | 21 ± 0.55 | 21 ± 0.92 | 20 ± 0.80 | 18 ± 1.1 | 18 ± 0.96 |

Values are means ± SE. CWL, competitive weight lifters; AWL, amateur weight lifters; HC, heavy controls; LC, light controls; LDR, long distance runners. MVC, maximum voluntary contraction. Values are not significantly different at \( P < 0.05 \).
TABLE 2. Blood pressures, heart rates, and double product at rest and during static exercise

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<th>Controls</th>
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Values are means ±SE. CWL: competitive weight lifters; AWL: amateur weight lifters; LDR: long-distance runners; $t_0$: time at rest; $t_{1/2}$: half time to fatigue; $t_{max}$: fatigue time. Values outside each set of brackets are significantly different at $P \leq 0.05$ from values contained within the brackets.

significant differences in the blood pressures between any of the athletes or the combined control group at rest, halfway to fatigue, or at fatigue. Comparing the blood pressures from rest ($t_0$) to half exercise time ($t_{1/2}$) to maximal exercise time ($t_{max}$) for each group individually revealed that significant increases occurred throughout exercise. On the other hand, compared to the control group, the heart rate was lower in the runners at rest and during half and maximal exercise times. The competitive and amateur weight lifters maintained similar heart rates as the controls at rest, half-fatigue time, and at the time of fatigue. Unlike the smooth response of blood pressure during exercise, the greatest and significant increase in heart rate in all groups occurred between $t_0$ and $t_{1/2}$ with little further increase occurring from $t_{1/2}$ to $t_{max}$. The double product, an index of myocardial oxygen demand, remained lower in the runners than in the control group at rest and at all times during static exercise. The double product of the weight lifters, however, did not differ from the control group at rest or during exercise. The increases in the double product of all groups were greatest during the first half of exercise although they all showed further significant increases during the second half of exercise.

Left ventricular volumes. The left ventricular end-diastolic and end-systolic volume indexes of the runners were greater than the light controls at rest, significantly greater at half-maximal exercise time and again greater at the time of fatigue (Figs. 1 and 2, top). The weight lifters had end-diastolic volume indexes similar to the heavy control group at rest and throughout handgrip exercise. During exercise the competitive weight lifters, heavy controls, and long-distance runners did not significantly change their end-diastolic volumes, whereas the amateur weight lifters and the light controls showed significant increases (Fig. 1, bottom). The end-systolic volumes remained unaltered during exercise for all groups except the long-distance runners who increased their volume significantly at half time to fatigue (Fig. 2, bottom).

The calculated stroke volume index in the runners was greatest at rest but was similar to the light control group.
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during static exercise (Fig. 3, top). The weight lifters had stroke volume indexes similar to the heavy controls at rest and during exercise. The weight lifters and the light controls all increased their stroke volume during exercise although only the amateur weight lifters had a significant increase (Fig. 3, bottom). The ejection fraction was similar at rest and during exercise in all of the athletes and their respective control groups (Fig. 4, top). Ejection fraction did not change during exercise in any of the groups (Fig. 4, bottom).

**Cardiac indexes and systemic vascular resistances.** Cardiac index at rest and during exercise was calculated to be similar in the runners, the light controls, and the weight lifters compared to the heavy controls (Fig. 5, top). An increase in cardiac index occurred from rest to half-maximal exercise time in all groups (Fig. 5, bottom). All the groups had small, insignificant, further increases in cardiac index except for the light controls who significantly decreased their cardiac index.

Systemic vascular resistance was similar among all the groups at rest. However, during exercise the runners maintained a higher vascular resistance than all other groups (Fig. 6, top). The weight lifters demonstrated systemic vascular resistances similar to the combined control group during exercise. Comparing the values at rest to the half-maximal exercise time to the time of fatigue, none of the groups significantly changed their vascular resistance although all of them showed slight decreases at the half-maximal time of exercise.

**Stroke work indexes and velocity of circumferential fiber shortening.** At rest the long-distance runners performed more stroke work than the combined control group whereas during exercise these groups performed a similar amount of stroke work (Fig. 7, top). The weight lifters performed a degree of stroke work similar to the control group at rest and during exercise. There were large increases in the stroke work index during the first half of exercise for all groups and smaller, mostly insignificant increases during the second half of exercise (Fig. 7, bottom).

The mean normalized velocity of circumferential fiber shortening was similar for all groups at rest. During exercise the runners demonstrated a lower velocity than the combined control groups (Fig. 8, top). The two groups of weight lifters, however, demonstrated velocities similar to the control group during exercise. The competitive weight lifters and the long-distance runners did not change their velocities of circumferential fiber shortening.
In this study, the pressor response was similar throughout exercise whereas most of the tachycardia response occurred during the first half of exercise. The larger initial increase in heart rate may be due to central nervous system irradiation proposed by Krogh and Lindhard (13). Lind et al. (14) demonstrated similar blood pressure and heart rate transients during exercise in a small number of untrained individuals.

A significant finding in this study was the lower heart rate and double product in the runners throughout the static exercise. The double product correlates closely with myocardial oxygen demands (10). Two other factors related to myocardial oxygen consumption are the stroke volume index and the adjusted stroke work index (2). These factors were similar in the runners and the control groups throughout exercise. The lower heart rate in the runners suggests that the minute myocardial work was less in these endurance athletes compared to the controls. However, the relative bradycardia in the runners was the principal factor decreasing the double product and minute stroke work index.

The mean normalized velocity of circumferential fiber shortening ($V_{CF}$) during static exercise was significantly lower in the endurance athletes than the control group. This difference was caused by the increase in the $V_{CF}$ in the control group, confirming the work of other investigators (28), and the lack of a change of $V_{CF}$ in the runners. The mean normalized $V_{CF}$ reflects alterations in myocardial performance (3). However, it is influenced by acute heart rate and blood pressure changes (19). The heart rate of the runners at half time to fatigue was equivalent to the resting controls' heart rate. However, the $V_{CF}$ for the two groups at those times was still significantly different (LDR: 0.94, CNT: 1.1 circumferences/s) although it was expected that the runners' contractility during exercise, whereas the amateur weight lifters and the combined control group significantly increased their velocities during the first half of exercise (Fig. 8, bottom).

**DISCUSSION**

The increase in heart rate, systolic, mean, and diastolic arterial blood pressures and cardiac index, and the lack of increase in systemic vascular resistance and stroke volume (except for the amateur weight lifters) confirm many other available studies (14, 16, 27). Few studies, however, have looked at the midpoint responses to exercise. The point of fatigue is a reproducible end point that allows comparisons between different groups of individuals, trained or untrained, or within the groups over time. The half time to fatigue is a similar relative time that is comparable between individuals or between groups. It is desirable to make comparisons in this manner because the cardiovascular response to static exercise continuously changes at levels greater than 15% MVC (14). Cardiovascular variables at rest compared to those at half time to fatigue and at the time of fatigue during static exercise provide an analysis at similar relative points on the fatigue curve. Analysis at the same relative time during exercise helped to normalize for these variations in endurance. Other factors that influence the cardiovascular response to static exercise are the absolute (21) and relative tensions exerted (14). Although it was expected that training by weight lifting would increase the grip strength, these athletes demonstrated similar absolute and relative strengths of contraction compared to other groups. This suggests that the work performed and the cardiovascular responses should have been similar.
would increase during exercise. The blood pressure increased equivalently in the runner and control groups during exercise. Thus, although part of the V_{CR} difference between runners and controls may have been caused by the lower heart rate, part of the difference during exercise was likely due to lower contractile state of the myocardium. A relatively lower left ventricular performance in the runners compared to the control group supports the concept that myocardial oxygen demands in endurance athletes is reduced because the contractile state, in part, determines the myocardial oxygen consumption (5, 26). However, both runners and weight lifters have a greater absolute left ventricular mass than untrained subjects, a factor that may increase the total myocardial oxygen consumption. Since wall stress or force per unit cross-sectional area, as determined by the wall thickness-to-radius ratio is unaltered in these athletes (runners or weight lifters), the wall tension probably does not alter this myocardial oxygen consumption (15, 17). Thus there is a complex interplay of the variables that increase with those that decrease myocardial oxygen consumption so the net result cannot presently be accurately determined.

The resting end-diastolic volume index was greater in the runners than all other groups at rest. During exercise the stroke volume index was equalized by the long-distance runners rather than in the control subjects at rest. The larger end-diastolic volume during exercise in runners compared to controls (despite a similar relative tachycardia response) also suggests a basic structural enlargement of the myocardium of runners.

Few studies are available on the changes in the end-diastolic volume occurring with static exercise. End-diastolic volume index has been reported to be little altered by static exercise (27, 28). Studies of the left ventricular end-diastolic pressure, an index of preload changes, have concluded that, under normal physiological conditions, there is either no change (1, 7) or a small increase with exercise (8, 12). Venomotor tone in normal subjects increases in proportion to the percent MVC (24). In the present study the left ventricular end-diastolic volume index increased in the weight lifters, the light controls, and the runners but to a significant extent only in the amateur lifter and the light control groups. It is uncertain why the heavy controls responded differently than the light controls. However, overall the end-diastolic volume tends to increase during static exercise in the supine position presumably as a result of venoconstriction.

The larger end-systolic volume index in the long-distance runners rather than in the control subjects at rest and during exercise may reflect a decrease in the myocardial performance of endurance athletes’ hearts. This suggestion confirms the lower velocity of circumferential fiber shortening in the runners compared to the control group during exercise. The end-systolic volume is thought to both acutely and chronically parallel the inotropic state of the heart (6, 21). Published observations on the end-systolic volumes in endurance athletes at rest are variable. Some have shown a larger volume than control groups (22, 31), while others have demonstrated a decrease in volume after training (4). The first two studies did not carefully match the control groups and the volumes were not related to the body surface area. Two preliminary communications have examined the response of end-systolic dimension to static exercise in athletes. One cross-sectional study suggested that the end-systolic volume decreased more in runners than controls (25). Another longitudinal study concluded that the end-systolic volume at rest and during exercise responded similarly before and after training (18). Both studies did not normalize the volume for the body size, a factor that varies between athletes and control subjects and was considered in the present study.

Although the end-diastolic and the end-systolic volume indexes were increased in the runners at rest and during static exercise, the stroke volume index was greater only at rest. During exercise the stroke volume of the light controls and long-distance runners was equalized by the greater increase in end-diastolic volume index in the former group (L.C.: 18, L.D.R.: 7 ml/m²), while the end-

![Graph](image-url)
systolic volume increase was similar in both groups (6 ml/m²). Thus the insignificantly greater ejection fraction in the light controls than the runners resulted in similar stroke volumes in both groups during static exercise. The slightly greater stroke volume index at rest and during exercise in the long distance runners compared to the light controls offset the lower heart rate in the former group, resulting in similar calculated cardiac indexes. The insignificantly smaller cardiac output and similar mean arterial blood pressure in the runners during exercise yielded a significantly greater calculated systemic vascular resistance compared to the combined control group. The increased vascular resistance in runners probably resulted from the lower heart rate that limited the cardiac output during exercise relative to the control group. However, a primary increase in vasoconstriction in the runners cannot be excluded. Other studies have also shown that vascular resistance does not change during lower levels (>50% MVC) of static exercise (7, 14, 16, 28).

At rest the weight lifters, both amateur and competitive, demonstrated similar end-diastolic and end-systolic volume indexes to the heavy control subjects. This resulted from a similar total left ventricular volume in both groups (15).

Both groups of weight lifters responded to exercise in a manner similar to the heavy controls or the combined control group. These athletes increased their blood pressures, heart rates, double product, cardiac index, mean normalized \( V_\text{cF} \), and adjusted stroke work index. Likewise, the competitive weight lifters responded like the heavy control group without any significant change in end-diastolic, end-systolic, and stroke volume indexes, and ejection fraction during static exercise. The amateur weight lifters, on the other hand, significantly increased their end-diastolic and stroke volume indexes during static exercise perhaps due to a greater augmentation in venous return.

In conclusion, substantial evidence has been presented suggesting that long-distance runners react differently than control subjects to the stress of static exercise. The endurance athletes maintain a lower double product, a lower minute stroke work index, and a relatively lower state of contractile function all of which could lower the myocardial oxygen demands for these individuals, although the greater myocardial mass could offset these increases. The larger end-diastolic and end-systolic volume indexes at rest and during static exercise were thought to be related more to a structural difference in their hearts rather than a difference in heart rates. The stroke volume index was similar in the runners and the light controls due to a slightly larger ejection fraction in the latter group. And, although there were only slight differences in cardiac index between these two groups, the calculated systemic vascular resistance was greater in the runners. The weight lifters in contrast to the endurance athletes, at rest and during exercise, responded to static exercise in a manner very similar to the control subjects.

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