Concentric adaptation of the left ventricle in response to controlled upper body exercise training

PHILLIP E. GATES, KEITH P. GEORGE, AND IAN G. CAMPBELL

1Department of Exercise and Sport Science, Alsager Faculty, Manchester Metropolitan University, Alsager, Cheshire ST7 2HL; and 2Division of Sport, Health, and Exercise, Staffordshire University, Stoke-on-Trent, Staffordshire ST2 4DF, United Kingdom

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Gates, Phillip E., Keith P. George, and Ian G. Campbell. Concentric adaptation of the left ventricle in response to controlled upper body exercise training. J Appl Physiol 94: 549–554, 2003. First published October 18, 2002; 10.1152/japplphysiol.00263.2002.—Upper body exercise has many applications to the rehabilitation and maintenance of cardiovascular health of individuals who are unable to exercise their lower body. The hemodynamic loads of upper body aerobic exercise are characterized by relatively high blood pressure and relatively low venous return. It is not clear how the left ventricle adapts to the specific hemodynamic loads associated with this form of exercise training. The purpose of this study was to measure left ventricular structure and function in previously sedentary men by using echocardiography before and after 12 wk of aerobic arm-crank exercise training (n = 22) or a time control period (n = 22). Arm-crank peak oxygen consumption (in ml • kg−1 • min−1) increased by 16% (P < 0.05) after training, and significant differences (P < 0.05) were found in wall thickness (from 0.86 to 0.99 cm) but not in left ventricular internal dimension in diastole or systole. This suggested a concentric pattern of left ventricular hypertrophy that persisted after scaling to changes in anthropometric characteristics. No differences (P < 0.05) were found for any measurements of resting left ventricular function. We conclude that upper body aerobic exercise training results in a specific left ventricular adaptation that is characterized by increased left ventricular wall thickness but no change in chamber dimension.

Arm-crank exercise; athletic heart; physiological cardiac hypertrophy

Regular exercise training has been reported to influence left ventricular structure with equivocal data reported regarding resting left ventricular function. The general consensus that endurance- and resistance-trained athletes demonstrate eccentric and concentric left ventricular hypertrophy was reported at the 26th Bethesda Conference (18) and has received empirical support from meta-analysis of cross-sectional (13, 20, 21) and longitudinal studies (20). However, the majority of studies have focused on the effect of lower body aerobic training on left ventricular structure and function. This is despite the fact that upper body aerobic exercise has important applications to the rehabilitation and health of spinal cord-injured individuals, to cardiac rehabilitation programs, and to activities that include a significant upper body exercise component. Characterizing the nature of left ventricular adaptation to specific forms of exercise training, including upper body training, may also be of importance to the clinician for differentiating physiological from pathological left ventricular hypertrophy; wall thickness values compatible with the diagnosis of hypertrophic cardiomyopathy were reported in Olympic athletes from disciplines that had a large upper body component, e.g., canoeing and rowing (19).

Although it might be tempting to extrapolate data regarding left ventricular adaptation to lower body exercise to upper body exercise, this approach has limited theoretical validity. Several studies have reported that physiological responses to upper body aerobic exercise are different from those that occur during lower body aerobic exercise (1, 6, 7). Of particular relevance are studies that have reported hemodynamic differences, especially higher blood pressure, lower stroke volume (1), and higher heart rate (2), when arm-crank exercise was compared with cycling ergometry. Given that the hemodynamic response to exercise has been implicated as a mechanism that may determine the pattern of left ventricular adaptation (15), it is plausible that upper body aerobic exercise will induce a pattern of left ventricular adaptation that is specific to the hemodynamic characteristics of the activity. To date, no data are available to test this hypothesis in humans. Furthermore, many authors have used exercise-specific hemodynamic loads to explain the ventricular morphology reported in a variety of athlete populations (for review, see Perrault and Turcotte (20)). However, this is a hypothetical explanation because no studies have attempted to manipulate exercise hemodynamic loads to induce a specific change in ventricular morphology. In this respect, upper body exercise provides a useful model with which to test the hypothesis that the hemodynamic loads that occur...
during exercise provide the primary stimulus for left ventricular adaptation.

The aim of this study, therefore, was to determine the influence of an upper body aerobic exercise-training program on left ventricular structure and function. In particular, we aimed to test the hypothesis that upper body aerobic exercise training would cause left ventricular remodeling characterized by increased wall thickness. As the majority of echocardiographic data do not support the concept of an alteration to resting left ventricular function after exercise training, we also hypothesized that upper body aerobic exercise would have no effect on resting left ventricular function, as determined by ultrasound echocardiography.

METHODS

Subjects. Twenty-six previously sedentary men, aged between 20 and 40 yr, volunteered to take part in the training program, and four of these subjects withdrew. Twenty-two time-control subjects were recruited and pair matched on the basis of sex and age. Pair matching allowed subjects of similar age and the same sex to be studied, thus avoiding the influence of sex- and age-associated change in left ventricular structure and function. Mean age ± SD was 33.4 ± 5.8 yr in the training group and 33.3 ± 5.6 yr in the control group, and all subjects were within 12 mo of the age of their training group counterparts. Controls were either sedentary or recreationally active, having maintained a regular pattern of activity in the year before participation in the study and doing so through the control period. Subjects were apparently healthy and showed no signs or symptoms of cardiovascular disease during maximal exercise testing. At an initial visit, subjects were familiarized with the laboratory environment and arm-crank ergometry, and written, informed consent was obtained. Ethical approval for the study was granted from Manchester Metropolitan University Ethical Research Committee.

Measurements. All measurements were made under similar conditions before and after training and control periods. The day of the week and time of the day that pre- and posttesting sessions took place were the same.

Echocardiography and cardiovascular measurements. Subjects attended the laboratory in a rested state for an echocardiographic examination that was conducted by using a Hewlett Packard Sonos 100 ultrasound imaging system. Subjects were examined after a 10-min rest using a 2.5-MHz transducer acoustically coupled with ultrasound transmission gel. Examinations were recorded on high-quality videotape for later analysis.

Two-dimensional images were obtained from the left parasternal long-axis window to obtain M-mode images of the left ventricle. The guidelines of the American Society of Echocardiography (25) were followed for M-mode measurements, and care was taken to avoid off-axis measurements. Measurements were taken of left ventricular internal dimension at end-diastole (LVIDd; in cm) and systole (LVIDs; in cm) and of interventricular septal thickness (ST; in cm), posterior wall thickness (PWT; in cm), and aortic dimension at end diastole. Left ventricular mass (LVM; in g) was calculated by using a previously validated regression-corrected cube formula (9). Fractional shortening percent and ejection fraction percent were used as indexes of contractility, and stroke volume was calculated as LVIDd² – LVIDs² × 0.627.

Pulsed-wave Doppler measurements were obtained after accurate placement of the sample gate at the tips of the mitral valve leaflets by using two-dimensional imaging from the apical window, and care was taken to avoid measurements at an angle more than zero degrees. Early diastolic inflow, late ventricular inflow after atrial contraction, and systolic outflow envelopes were analyzed for peak flow velocities (cm/s) and flow integrals (cm). Ratios of early diastolic inflow to late ventricular inflow after atrial contraction peak velocities and integrals were also calculated. To derive the coefficient of variation for each echocardiographic variable, a subsample of the 44 subjects (n = 29) had measurements taken on two separate occasions, exactly 2 wk apart at baseline.

Resting blood pressure was measured manually by using auscultation with subjects in a supine position, and resting heart rate was measured by using 12-lead electrocardiography.

Anthropometric measurements. Body mass (BM) was measured to the nearest 0.1 kg and stature to the nearest 0.1 cm. Body surface area (m²) was estimated from stature and BM (10). Subcutaneous fat measurements were made by using skinfolds obtained with a Harpenden calliper and measured to the nearest 0.02 cm. Subcutaneous fat measurements were corrected by using the regression-corrected cube formula (17). The mean of three measurements at each site was used to calculate percent body fat by using the body density equations of Durnin and Womersley (11). As the majority of subjects were adult British men, the use of this formula seemed most appropriate as the equations were validated on a similar population. Fat-free mass (FFM; in kg) was calculated as follows: FFM = BM – (BM/100 x % body fat).

Determination of aerobic capacity. To assess changes in aerobic fitness and to validate the effectiveness of the program at improving aerobic capacity, peak minute oxygen uptake (VO₂ peak) was measured before and after the training and control periods. Measurement of VO₂ peak also allowed exercise intensities to be set for the training group. To account for changes in aerobic fitness during training, VO₂ peak tests were administered at 4 and 8 wk, and training intensities were adjusted accordingly.

Arm-crank ergometry was used to determine VO₂ peak by using a modified Monark 814E cycle ergometer. This was securely mounted on a table that was bolted to a wall for stability. The pedals were replaced with handles. The protocol adopted for the determination of VO₂ peak was that described by Price and Campbell (22) and outlined in brief here. The test was continuous with 2-min, incremental work stages until the subject reached volitional exhaustion. Subjects were asked to arm crank at 70 revolutions/min with work increments of either 21 or 35 W, depending on ability. After volitional exhaustion, a 5-min recovery period was allowed. Subjects then completed a 2-min verification stage at one work increment higher than that achieved at volitional exhaustion. The purpose of this stage was an attempt to demonstrate a plateau in oxygen uptake.

Expired gas was sampled by using a Sensormedics 2900 metabolic cart, and heart rate was measured throughout the test by using a Polar PE4000 heart rate monitor.

Training program design. The training program lasted 12 wk and involved a progressive increase in training session duration and frequency. Subjects were asked to arm crank continuously for 20 min, three times a week for the first 2 wk. This was increased to three, 30-min sessions for weeks 3 and 4 and to three, 40-min sessions for weeks 5 and 6. For the last 6 wk, subjects exercised for 45 min, four times a week. During training sessions, subjects were asked to exercise at a target heart rate corresponding to 60% VO₂ peak. All 22 subjects completed all training sessions in the 12-wk period.
Statistical analysis. All data were analyzed with two-way analysis of variance by using SPSS statistical software version 7.5.1. Post hoc Tukey's tests were used where significant interactions were found. The statistical significance level was set at \( P < 0.05 \) for all analyses. All data are expressed as means ± SD. Effect sizes were calculated where significant differences were found (mean posttest – mean pretest/mean standard deviation). Effect sizes have been used as an estimate of the meaningfulness of differences in outcome variables where 0.2 would reflect lower meaningfulness and 0.8 would suggest high meaningfulness (8).

Scaling procedures were applied to the structural data measured from the left ventricle. Data for ST, PWT, and LVIDd were scaled to BM raised to the power 0.33 and FFM raised to the power 0.33 (FFM\(^0.33\)). Scaling procedures were also applied to calculated LVM by using BM raised to the power 1.00 and FFM raised to the power 1.00. This allowed changes in body composition over the training or control periods to be taken into account. These scaling procedures have received empirical support from data obtained from untrained and athletic populations (3–5, 14).

RESULTS

Effectiveness of the training program at increasing \( \dot{V}O_2 \) peak. The training group demonstrated a significant increase in mean \( \dot{V}O_2 \) peak from 2.63 ± 0.32 to 2.95 ± 0.37 l/min (effect size > 0.8). In the control group, values were similar before and after the time-control period. \( \dot{V}O_2 \) peak was 2.62 ± 0.52 l/min before the control period and 2.60 ± 0.51 l/min after. These data indicate that the program effectively increased upper body aerobic fitness.

Echocardiographic and cardiovascular measurements. There were no significant differences between the training and control groups in any of the echocardiographic data at baseline. Resting systolic and diastolic blood pressure and resting heart rate did not change in either group (Table 1).

Both ST and PWT increased significantly in the training group by a mean of 0.13 and 0.11 cm, respectively (effect size > 0.8), but no difference was apparent in the control group (Fig. 1, A and B). Left ventricular internal dimension at end diastole and end systole remained similar in both groups (Fig. 1, C and D).

Table 1. Blood pressure and heart rate before and after training and control conditions

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Fig. 1. Both septal (A) and posterior wall (B) thickness increased significantly over the training period but not over the time-control period. The greatest increase was in the septum (change of 0.13 cm). There were no significant changes in left ventricular (LV) chamber dimension in diastole (LVIDd; C) or systole (LVIDs; D). The increase in wall thickness without significant change in LVIDd resulted in a trend for an increase in diastolic wall thickness-to-chamber dimension (h/R) ratio (E; \( P = 0.06 \)) in the training group. LV mass (F) was greater by 37 g after training but did not increase in the time-control group. *Significant difference compared with pretraining (Pre) \( (P < 0.05) \). †Significant difference compared with control group posttraining (Post) time-control period \( (P < 0.05) \).
study period. There are, therefore, independent of changes in body size and thickness meant that LVM was also greater after no change in chamber dimension. The increase in wall characterized by an increase in wall thickness but with no change in chamber dimension. This is a novel finding, because echocardiographic and anthropometric data have not been reported after a controlled arm-crank training program to date. The magnitude of the increase in wall thickness (15% for ST and 13% for PWT) was greater than the respective coefficient of variation determined in a subsample of the study population (4.6 and 3.4%, respectively).

The finding of a concentric pattern of left ventricular hypertrophy after upper body aerobic training does not comply with the consensus statement that aerobic (dynamic) training results in eccentric hypertrophy (18). Furthermore, increases in wall thickness have been reported after aerobic training in cross-sectional studies of other athletic populations (12, 16, 28). The athletic groups that showed significant increases in wall thickness included oarsmen (28), swimmers (12), and cyclists (16). All of these sports have a significant upper body component, in addition to a lower body component, that may have influenced the nature of left ventricular adaptation.

The only study to have made echocardiographic measurements after arm-crank exercise training on five subjects was reported by Thompson et al. (27). No statistically significant changes in left ventricular dimensions were reported, but the authors acknowledged that their training program was moderate in intensity. Unfortunately, data from the arm-crank training group were not reported, and it is not possible to make a comparison with the data presented here. The values obtained after arm-crank training in the current experiment approach, but do not equal, the values reported from highly trained athletes (19). This indicates that some degree of ventricular remodeling takes place rapidly, but ventricular morphologies found in athletes may take longer to achieve or may require a genetic predisposition.

Despite the fact that wall thickness values increased in the training group, they did not increase beyond normal ranges in any subject, and concentric hypertrophy was associated with an increased, rather than decreased, exercise tolerance. From a clinical perspective, therefore, it does not appear that upper body exercise training induces left ventricular hypertrophy beyond normal limits, and this concurs with data from other trained populations. It should be noted, however,

### Table 2. Systolic and diastolic functional characteristics of the left ventricle before and after training and control periods

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<tbody>
<tr>
<td>Stroke volume, ml</td>
<td>101 ± 18</td>
<td>102 ± 21</td>
<td>100 ± 20</td>
<td>107 ± 21</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>75 ± 8</td>
<td>76 ± 7</td>
<td>75 ± 5</td>
<td>77 ± 6</td>
</tr>
<tr>
<td>Fractional shortening, %</td>
<td>38 ± 6</td>
<td>38 ± 6</td>
<td>37 ± 4</td>
<td>39 ± 5</td>
</tr>
<tr>
<td>E, cm/s</td>
<td>76 ± 10</td>
<td>76 ± 9</td>
<td>67 ± 9</td>
<td>70 ± 8</td>
</tr>
<tr>
<td>A, cm/s</td>
<td>43 ± 9</td>
<td>43 ± 8</td>
<td>39 ± 9</td>
<td>43 ± 9</td>
</tr>
<tr>
<td>E/A velocity ratio, cm/s</td>
<td>1.76</td>
<td>1.76</td>
<td>1.72</td>
<td>1.63</td>
</tr>
<tr>
<td>Systolic velocity, cm/s</td>
<td>89 ± 9</td>
<td>91 ± 12</td>
<td>85 ± 13</td>
<td>86 ± 13</td>
</tr>
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</table>

Values are means ± SD. E, early diastolic inflow velocity; A, late ventricular inflow velocity after atrial contraction.

There was a trend for an increase in diastolic wall thickness-to-chamber dimension ratio in the training group (P = 0.06; Fig. 1E). Due to the increases in wall thickness, calculated LVM increased significantly in the training group by 20% (effect size > 0.8; Fig. 1F). Aortic diameter remained similar in both groups (2.95 ± 0.24 vs. 2.91 ± 0.19 pre- to posttest in the training group; 2.97 ± 0.27 vs. 3.01 ± 0.21 pre- to posttest in the control group). All significant differences were greater than the coefficient of variation determined from two separate baseline echocardiographic examinations obtained from the subsample of subjects (coefficient of variation for ST and PWT ~0.04 cm and ~13 g, respectively, for LVM). When scaled to FFM<sup>0.33</sup>, ST and PWT remained significantly different, as did LVM.

There were no apparent differences in resting fractional shortening, ejection fraction, or stroke volume after the training or control period (see Table 2). Doppler velocity and integral data were not different for either diastolic inflow or systolic outflow.

**Anthropometric measurements.** BM was reduced slightly, but not significantly, in the training group and remained similar in the control group (Table 3). Percentage body fat was significantly lowered after training (effect size = 0.8), and the difference was greater than the coefficient of variation determined at baseline (~0.55%; Table 3). There was no change in percent body fat in the control group. A reduction in percent body fat while maintaining BM resulted in an increase in calculated FFM in the training group but no significant change in the control group. Fat and body surface area were similar between groups and did not change significantly over the study period.

### Table 3. Anthropometric characteristics of subjects before and after training and control periods

<table>
<thead>
<tr>
<th></th>
<th>Before Training</th>
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<th>Before Control</th>
<th>After Control</th>
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<tbody>
<tr>
<td>Body mass, kg</td>
<td>79.7 ± 10.6</td>
<td>78.8 ± 11.0</td>
<td>76.8 ± 8.4</td>
<td>76.9 ± 8.2</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>19.3 ± 3.8†</td>
<td>16.1 ± 4.2&lt;sup&gt;‡&lt;/sup&gt;</td>
<td>16.9 ± 5.4</td>
<td>16.7 ± 5.4</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>64.1 ± 7.1</td>
<td>65.9 ± 7.4</td>
<td>63.5 ± 5.2</td>
<td>63.8 ± 5.5</td>
</tr>
</tbody>
</table>

Values are means ± SD. FFM, fat-free mass. †Significantly different from pretraining value (P < 0.05). ‡Significantly different from control group pre- and posttraining (P < 0.05).

**DISCUSSION**

The main finding of this study is that left ventricular adaptation to aerobic arm-crank exercise training is characterized by an increase in wall thickness but with no change in chamber dimension. The increase in wall thickness meant that LVM was also greater after training. These differences persisted after scaling data to collateral changes in anthropometric variables and are, therefore, independent of changes in body size and composition. It appears that upper body exercise training induces an activity-specific remodeling of left ventricular morphology. This is a novel finding, because echocardiographic and anthropometric data have not been reported after a controlled arm-crank training program to date. The magnitude of the increase in wall thickness (15% for ST and 13% for PWT) was greater than the respective coefficient of variation determined in a subsample of the study population (4.6 and 3.4%, respectively).

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Despite the fact that wall thickness values increased in the training group, they did not increase beyond normal ranges in any subject, and concentric hypertrophy was associated with an increased, rather than decreased, exercise tolerance. From a clinical perspective, therefore, it does not appear that upper body exercise training induces left ventricular hypertrophy beyond normal limits, and this concurs with data from other trained populations. It should be noted, however,
that Pelliccia et al. (19) identified 16 athletes who presented wall thickness values in a range compatible with hypertrophic cardiomyopathy, and canoese featured among this group. Current consensus that ventricular morphology is determined by participation in sports classified as “static,” “dynamic,” and “combined” (18) may need to incorporate more specific information on the mode of exercise.

No significant differences were found for any of the Doppler or M-mode functional indexes from pre- to posttraining or over the control period. Few longitudinal training studies have reported Doppler data before and after completion of a program of exercise training. Consistent with our results, previous studies have reported no change in left ventricular Doppler parameters after lower body aerobic training (23, 24). Although data are limited, it appears that young, previously sedentary subjects do not have altered Doppler indexes of resting left ventricular function after short-term exercise training. This conclusion is supported by a meta-analysis of cross-sectional data on highly trained athletes (21).

Resting heart rate and stroke volume were not different after training. Lower body aerobic training studies have consistently reported reductions in resting heart rate and increases in stroke volume that were accompanied by an increase in LVIDd [for review, see George et al. (15)], and endurance athletes have been reported to have heart rates between 40 and 50 beats/min. Low heart rates are usually attributed to an increase in chamber dimension and stroke volume that is associated with an increase in central blood volume. In this study, LVIDd and indexes of contractility remained the same, indicating that there was no effect on resting stroke volume. Although the increase in wall thickness may have the potential to increase stroke volume through a more forceful contraction, this enhanced inotropic state might only occur during exercise when the myocardium is exposed to elevated levels of circulating catecholamines and greater sympathetic innervation (26). The ability to contract more forcefully may be a useful adaptation to arm-crank training because it would enable the ventricle to overcome the increased afterload imposed by bouts of arm exercise.

**Conclusions.** The data from this study indicate that arm-crank exercise training results in increased left ventricular wall thickness without a significant change in resting left ventricular function. LVM was also increased after training. This concentric pattern of left ventricular hypertrophy was accompanied by increases in aerobic fitness indexes and improved body composition.

Increased left ventricular wall thickness after aerobic training may be an adaptation that is specific to exercise involving the arms. These data have applications to ongoing discussion regarding clinically normal ventricular morphology in athletes, the benefits of exercise training in the reversal of cardiac atrophy in subjects with a spinal cord injury, and the efficacy of upper exercise training for health and fitness. It also has theoretical application to understanding the mechanisms of left ventricular adaptation to exercise.

**REFERENCES**


