Left ventricular adaptations following short-term endurance training

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Goodman, Jack M., Peter P. Liu, and Howard J. Green. Left ventricular adaptations following short-term endurance training. J Appl Physiol 98: 454–460, 2005. First published September 24, 2004; doi:10.1152/japplphysiol.00258.2004.—This study examined the effects of short-term endurance training (ET) on the left ventricular (LV) adaptation and functional response to a series of exercise challenges with increasing intensity. Eight untrained men, with a mean age of 19.4 ± 0.5 (SE) yr, were studied before and after 6 days of ET consisting of cycling 2 h/day at 65% peak aerobic power (V̇O₂ max). LV ejection fraction and LV volumes were assessed by radionuclide angiography at rest and during exercise at three uninterrupted successive work rates corresponding to 53, 68, and 83% of V̇O₂ max, each lasting 20 min. ET produced a calculated plasma volume expansion of 11.4 ± 2.2% (P < 0.05). The increase in plasma volume was accompanied by an increase in V̇O₂ max from 45.9 ± 1.9 to 49.0 ± 1.0 ml·kg⁻¹·min⁻¹ (P < 0.01) and a decrease in maximal heart rate (197 ± 2.3 to 188 ± 1.0 beats/min; P < 0.01). Resting LV function was not changed, although there was a trend for higher stroke volumes (SVs) and improvement in the rapid filling phase of diastole (P = 0.08). Training induced an increase in exercise SV by 10.4, 10.2, and 7% at 53, 68, and 83% V̇O₂ max, respectively (P < 0.01). These changes were secondary to increases in end-diastolic volume, which increased significantly at each exercise work rate following training (139 ± 6 to 154 ± 6 ml at 53% V̇O₂ max and from 136 ± 5 to 156 ± 5 ml at 83% V̇O₂ max, P < 0.01). End-systolic volumes were unchanged after ET. A significant bradycardia was observed both at rest (decreasing 7%) and exercise (decreasing 10.4%). LV ejection fraction during exercise was increased slightly by training, reaching significance at the highest work rate, after 60 min of exercise. (P < 0.05). Cardiac output was higher following training at the highest workload (20.8 ± 2.2 vs. 22.9 ± 3.1 l/min; P < 0.01). These data indicate that short-term training elicits rapid adaptation to the LV functional response exercise, with increases in SV being secondary to a Frank-Starling effect with minor changes in contractile performance. This produced a volume-induced bradycardia and increase in LV filling, which may be of benefit during prolonged exercise. left ventricle; exercise; endurance training; radionuclide imaging

THE ACUTE RESPONSE TO BRIEF exercise includes an increase in both heart rate (HR) and cardiac output, with the latter increasing during low-to-moderate intensities of exercise due to increased HR and stroke volume (SV). The increase in SV during upright submaximal exercise is largely due to increased left ventricular (LV) end-diastolic volume (EDV) (17, 36). Earlier studies suggested that, in untrained subjects, exercise progresses to maximal effort. LV filling is attenuated, and SV may actually decline (42). However, more recent data (46, 49) suggest that SV in trained athletes may continue to rise, albeit to a smaller extent, throughout graded exercise, and in some cases up to maximal effort. Further increases in cardiac output during intensive exercise are facilitated by a continued rise in HR until the age-limited maximal heart is achieved. However, during prolonged efforts exceeding 40–60 min, and in particular when the cardiovascular system is challenged at high work rates, there can be a gradual loss in ventricular filling and systemic blood pressure (i.e., cardiovascular drift), which can greatly limit exercise performance (11, 38). It is now believed that hypovolemia during exercise explains ~50% of the decline in SV (7), and the rise in HR contributes significantly to the reduction in SV (13). Dehydration can greatly exacerbate cardiovascular function in hyperthermic athletes (16); however, it is unclear if exercise training-induced hypervolemia can improve LV filling.

Whereas most of the data regarding changes in cardiac function after exercise training were drawn from models employing long-term training (e.g., >6 mo) (34), there are limited data on the cardiac adaptations that occur early in the training process (22). The expansion of blood volume, which has been observed during training (22, 28, 41), appears to peak within ~1 wk of training and is explained almost completely by an expansion of plasma volume (26). However, the significance of an expanded plasma volume (PV) on the cardiovascular response to exercise during this early phase of training remains unclear. The evidence that inotropic function is improved with training is equivocal (1), and it appears that enhanced diastolic compliance (31) contributes to a more pronounced utilization of the Frank-Starling mechanism during exercise following training. This is, in turn, reflected by an increase in SV and bradycardia during exercise following sustained aerobic training (3, 15).

We have previously shown that short-term training has been shown to induce changes in cardiovascular hemodynamics (19–22), but measures of LV volumes or measures of systolic performance during exercise have not been studied following this type of training intervention. A recent study has shown that short-term training can increase early diastolic filling at rest (24). However, it remains unclear whether short-term training can alter the LV response to exercise and contribute to improved exercise performance. Accordingly, this study investigated the effects of short-term endurance training on cardiovascular function during exercise in previously untrained men. We hypothesized that short-term training would elicit a hypervolemic response leading to Frank-Starling-mediated improvement in LV function during a series of exercise challenges and an increase in maximal exercise performance.
METHODS

Subjects. Eight untrained but active university students participated in the study. Written consent was obtained from all subjects following approval of the study by the Office of Human Research. Physical characteristics for the subjects included a mean (±SE) age of 19.4 ± 0.5 yr and a mean weight of 70.1 ± 3.8 kg. Peak oxygen consumption (V\textsubscript{O\textsubscript{2}}\text{peak}) was determined from a progressive cycle ergometer test to fatigue before and following training. Unlike the submaximal tests, these measures were carried out at a separate facility employing techniques previously described (21). Briefly, following a 3-min warm-up at 50 W, the work rate was increased by 50 W every 2 min until 200 W, with further increases of 25 W every minute until maximal effort was obtained. Unless a plateau in oxygen consumption (V\textsubscript{O\textsubscript{2}}), despite a rise in work rate, was obtained, the end points used to define V\textsubscript{O\textsubscript{2}}\text{peak} included a respiratory exchange ratio >1.15 and the attainment of the age-predicted HR. Changes in PV following the training period were calculated from the changes in hematocrit, measured in triplicate pre- and posttraining (44).

Experimental design. To investigate the effects of training on cardiac function, subjects were evaluated both before and following training at rest and during 60 min of cycling exercise at three intensities that were increased in a stepwise fashion, designed to elicit work rates that corresponded to ~50, 70, and 80% of the pretraining V\textsubscript{O\textsubscript{2}}\text{max} (these work rates were repeated during postraining assessment), each lasting for a period of 20 min. Respiratory gas measurements and radionuclide angiography (RNA) were performed during the final 2 min of each work period. Fluids were not consumed during the exercise period.

Training consisted of 6 consecutive days of uninterrupted cycling at 65% V\textsubscript{O\textsubscript{2}}\text{max} for 2 h/day. Brief periods of rest were permitted during the training sessions, if necessary; however, these were only required during the first 2–3 days of training. A full 2 h of exercise were required during each training session. All exercise training was performed in an environmentally controlled room, with temperature and humidity maintained between 22 and 24°C and <50%, respectively. Subjects consumed water ad libitum. All subjects completed the training regimen.

Assessment of LV function. Exercise was performed in an upright position, by using a cardiopulmonary exercise protocol, which enables concurrent assessment of LV function using equilibrium RNA and measurement of respiratory gases, as described by our group previously (17). Briefly, subjects were positioned on a cycle ergometer table rotated to a fully upright sitting position. A portable gamma camera (Elscint APEX 215) fitted with a high-sensitivity parallel-hole collimator was used for cardiac imaging and was positioned in the left anterior oblique position, assisted by a dedicated computer using a 64 × 64 matrix at 16 frames per cardiac cycle. Cardiac images were analyzed with the use of a semiautomated procedure, with images gated to the R-wave-R-wave interval of the electrocardiogram. A master LV region of interest was manually drawn, incorporating the LV chamber area, by using a cursor pen by a blinded experienced operator. This process delineated the chamber area from the ventricular walls, producing a cine display of the rest and exercise acquisitions. End-diastolic counts (EDC) and end-systolic counts (ESC), corrected for background activity, were determined from an automated second-derivative edge-detection algorithm. LV ejection fraction (EF) was then calculated by using commercially available software, where EF = (EDC − ESC/EDC). End-diastolic volumes (EDV) and end-systolic volumes (ESV) were determined by using a count-based technique (32) by imaging a syringe containing 8 ml of peripheral blood with the same gamma camera to obtain the reference count activity per unit of blood.

Resting diastolic filling. Resting diastolic filing rates (DFRs) were determined before and after training by using a 4-min equilibrium acquisition, as described above; however, for DFR measures, a high-resolution time activity curve was generated from a minimum of 300 cardiac cycles (vs. 200 cardiac cycles used for standard assessment) recorded at rest before exercise began. Time to peak filling (TPF), the rapid filing phase, peak filling rate, and the percentage of filling contributed by atrial contraction (atrial kick phase) were determined by using a packaged software program and standard techniques (18, 43).

Cardiorespiratory and blood measures. A metabolic cart (Sensormedics 4400) was used to collect expired samples averaged over 30 s, yielding data for V\textsubscript{O\textsubscript{2}} and ventilation. Expired gas sampling was carried out concurrent with the RNA assessment and was reported as means averaged over the final minute of each acquisition during rest and steady-state exercise. HR was monitored continually by using a 12-lead ECG (Hewlett Packard), and blood pressures were monitored by using an automatic monitor (Infrasonde D4000).

Statistics. Student’s t-test was used to test for differences in resting cardiac function, maximal exercise data, and diastolic filing characteristics before and following training. ANOVA with repeated measures was employed to test for differences in all submaximal exercise data. All comparisons were based on a 95% confidence limit (probability level < 0.05).

RESULTS

Subject characteristics and PV. Following training, there was an increase in the calculated PV by 11.4 ± 2.2% (P < 0.05). No changes were observed for body mass.

Maximal exercise. Progressive exercise testing resulted in an increase (P < 0.05) in V\textsubscript{O\textsubscript{2}}\text{max} from 3.35 ± 0.18 to 3.60 ± 0.20 l/min and from 45.9 ± 1.9 to 49.0 ± 1.6 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}. There was a 5.6% reduction in maximal HR (197 ± 2 vs. 188 ± 1 beats/min; P < 0.05) and no change in maximal ventilation.

Resting and submaximal exercise. The 60-min exercise challenge elicited a progressive rise in V\textsubscript{O\textsubscript{2}} at each work rate, with no change in the response observed after training (Table 1). There were no significant training-induced changes in cardiac function or V\textsubscript{O\textsubscript{2}} at rest (Table 2), and we could not detect a significant change in resting HR (74.0 ± 3.5 vs. 67.1 ± 2.9 beats/min) or SV (78.4 ± 2.9 vs. 84.8 ± 6.5 ml). Resting systolic blood pressure was unchanged after training (122 ± 5 vs. 120 ± 3 mmHg), as was the response to the exercise challenge. Resting diastolic blood pressure was lower following training (77 ± 1 vs. 68 ± 2 mmHg) and remained significantly lower during exercise after 20 min (80 ± 4 vs. 68 ± 3 mmHg), 40 min (80 ± 4 vs. 66 ± 3 mmHg), and 60 min of exercise (81 ± 4 vs. 70 ± 3 mmHg) (P < 0.01).

Resting diastolic filing characteristics. See Table 3. The TPF was not changed following training (P < 0.08), nor was

Table 1. Oxygen consumption during submaximal exercise before and following training

<table>
<thead>
<tr>
<th>Exercise Time, min</th>
<th>0</th>
<th>20</th>
<th>40</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise intensity, %pretraining V\textsubscript{O\textsubscript{2}}\text{max}</td>
<td>53</td>
<td>68</td>
<td>83</td>
<td></td>
</tr>
<tr>
<td>Pretraining</td>
<td>0.258 ± 0.02</td>
<td>1.78 ± 0.92*</td>
<td>2.28 ± 0.13†</td>
<td>2.77 ± 0.13†‡</td>
</tr>
<tr>
<td>Posttraining</td>
<td>0.270 ± 0.02</td>
<td>1.78 ± 0.11*</td>
<td>2.13 ± 0.16†</td>
<td>2.85 ± 0.14†‡</td>
</tr>
<tr>
<td>P value</td>
<td>0.48</td>
<td>0.96</td>
<td>0.15</td>
<td>0.46</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 8. All subjects performed 20 min of exercise at each time point (total = 60 min). V\textsubscript{O\textsubscript{2}}\text{max}, maximum oxygen consumption. Significantly different than *rest, †20 min, and ‡40 min: P < 0.05.

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the peak filling rate ($P > 0.1$). There was no change in the contribution of atrial contraction (e.g., atrial kick) to diastolic filling after training.

**Submaximal cardiac function.** In contrast to the resting data, changes in various indexes of cardiac function were observed during submaximal exercise. HR after 20, 30, and 60 min of exercise was reduced after training, amounting to decreases of 10.4, 10.2, and 6.7%, respectively ($P < 0.01$), at each work rate (Fig. 1). There was a trend for an increased LV EF during the first 40 min of exercise after training; however, only at the 60-min work rate was the difference significant ($P < 0.05$) (Fig. 2). Values ranged from 80 to 88% throughout the 60-min exercise session during both sessions. SV (Fig. 3) was elevated posttraining at each work rate by 10.5% (20 min) to 14.8% (after 60 min) above pretraining values ($P < 0.01$). The changes in exercise SV following training were due primarily to an increase in LV EDV, which was sustained through the 60-min exercise challenge (e.g., at all work intensities; Fig. 4), with no change ($P > 0.1$) in ESV volume (Fig. 5) following training. Before training, the EDV declined between 40 and 60 min of exercise ($P < 0.01$); however, following training, it remained constant during this time frame. Cardiac output (Fig. 6) was unchanged at rest and during the first work rate (20 min) following training, but was slightly higher during the second work rate (40 min), becoming significantly higher at the highest intensity ($P < 0.05$).

**DISCUSSION**

This study has demonstrated rapid changes in the LV functional response to exercise following a training period of only 6 days, which also produced a PV expansion of $\sim$11%. The findings were associated with an increase in VO$_2$ max. These observations are consistent with prior reports of improved aerobic power and cardiodynamic responses to exercise after short periods of training (10, 20, 21, 28). Although changes in resting diastolic filling characteristics failed to reach statistical significance, there was evidence that the rapid filling phase of diastole was enhanced after training. In addition, increases in LV EF were detected at the highest exercise intensities. Of particular interest was the finding of an elevated cardiac output at the same high exercise intensity level following training. We believe that this may reflect a hemodilution effect that is secondary to the PV expansion.

The triggers that lead to cardiovascular adaptations following training are not well understood, particularly in the earliest stages of the training stimuli. It is likely that early and rapid

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**Table 2. Resting cardiac function before and following training**

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Posttraining</th>
<th>$P$ Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, beats/min</td>
<td>74.0±3.5</td>
<td>67.1±2.9</td>
<td>0.14</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>62±1</td>
<td>63±2</td>
<td>0.89</td>
</tr>
<tr>
<td>Stroke volume, ml</td>
<td>78.4±2.9</td>
<td>84.8±5.6</td>
<td>0.34</td>
</tr>
<tr>
<td>End-diastolic volume, ml</td>
<td>127±3</td>
<td>133±9</td>
<td>0.53</td>
</tr>
<tr>
<td>End-systolic volume, ml</td>
<td>48±2</td>
<td>49±4</td>
<td>0.93</td>
</tr>
<tr>
<td>Cardiac output, l/min</td>
<td>5.0±0.3</td>
<td>4.9±0.4</td>
<td>0.79</td>
</tr>
</tbody>
</table>

Values are means ± SE; $n = 8$.

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**Table 3. Resting diastolic filling characteristics before and following training**

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Posttraining</th>
<th>$P$ Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to peak filling, counts/s</td>
<td>0.173±0.005</td>
<td>0.169±0.005</td>
<td>0.08</td>
</tr>
<tr>
<td>Peak filling rate, counts/s</td>
<td>2.71±0.06</td>
<td>2.28±0.18</td>
<td>0.10</td>
</tr>
<tr>
<td>Rapid filling phase, %</td>
<td>82.9±2.3</td>
<td>81.4±1.8</td>
<td>0.3</td>
</tr>
<tr>
<td>Atrial kick phase, %</td>
<td>17.1±2.3</td>
<td>18.4±1.8</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Values are means ± SE.
changes in PV during training are coupled to the improvement in cardiodynamic performance during exercise, similar to how acute plasma expansion is tightly linked to exercise SV (44). The changes in PV seen in the present study are similar to those reported previously and are thought to be the result of acute compartmental shifts in plasma protein content, resulting from exercise and allowing the binding of addition water, which are often evident after a single exercise session (5, 14). The specifics of exercise-induced LV remodeling remain unclear.

The significance of these early changes in ventricular function during exercise is intriguing, considering the sequence of more general adaptations that follow in the training process. As Green et al. (21) suggested in an earlier study, the initial stage of training may well be triggered by an expansion of PV, with a shift in the control of cardiac function following an initial and brief training stimulus. This may be due to a resetting of the

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**Fig. 3.** Changes in stroke volume during 60 min of exercise before (●) and following (○) training. Work rates of 53, 68, and 83% $\dot{V}O_2_{\text{max}}$ were performed after 20, 40, and 60 min of cycling, respectively. Values are means ± SE. *Significantly different than pretraining values ($P < 0.05$). All exercise data are significantly different than resting data ($P < 0.01$).

**Fig. 4.** Changes in end-diastolic volume during 60 min of exercise before (●) and following (○) training. Work rates of 53, 68, and 83% $\dot{V}O_2_{\text{max}}$ were performed after 20, 40, and 60 min of cycling, respectively. Values are means ± SE. *Significantly different than pretraining values ($P < 0.05$). All exercise data are significantly different than resting data ($P < 0.01$).

**Fig. 5.** Changes in end-systolic volume during 60 min of exercise before (●) and following (○) training. Values are means ± SE. All exercise data are significantly different than resting data ($P < 0.01$).

**Fig. 6.** Changes in cardiac output during 60 min of exercise before (●) and following (○) training. Values are means ± SE. *Significantly different than pretraining values ($P < 0.05$). All exercise data are significantly different than resting data ($P < 0.01$).
reflexes controlling blood volume (5) secondary to the previously described attenuation of baroreceptor sensitivity seen following exercise-induced exercise hypervolemia (14). The expanded PV may contribute to genomic changes in myocardial structure and function specific to volume overload, which are, in turn, secondary to amplified signaling of stretch-activated ion channels and other multiple signals thought to activate protooncogenes specific to serial growth of the myofibrils (8).

**Systolic performance.** An intrinsic change in cardiac function during a short-term training model has not been previously reported. It remains possible that changes in LV systolic performance have contributed to the rise in SV in the present study. However, given the lack of direct measures of contractility, it remains speculative, and indirect data do not support this. EF increased modestly, suggesting a small improvement in systolic performance following training; however, our interpretations are limited because EF is load dependent and is, therefore, a limited index of myocardial contractility. Given the improvements in EDV concomitant with no change in ESV, ascribing changes to systolic function per se would be spurious. When the pressure-to-volume ratio (SBP/ESV), a more sensitive index of contractility, was calculated as a percent change (rest to exercise) for each subject, the results mirrored the EF data. There is not wide support of an improvement in myocardial contractility following chronic training in young subjects (1). Increases in ventricular volume and fractional fiber shortening have been observed following long-term training (9); however, more recent studies using similar indexes of contractility have failed to document changes following 2–4 wk of training, and improvements in SV are likely secondary to changes in ventricular preload (2, 42) via the Frank-Starling mechanism.

Our observations of an increased EF and increased EDV throughout exercise may be important in light of reports describing LV dysfunction during prolonged effort (27, 45, 47). Although Goodman et al. (18) recently failed to detect systolic impairment during 150 min of steady-state exercise, earlier and recent studies by others conducted in field conditions have reported declines in filling and contractile performance (12, 27, 45, 47).

**Role of the Frank-Starling mechanism.** Our study demonstrated a bradycardia at all levels of submaximal exercise, coincident with increases in SV. These changes appear secondary to a Frank-Starling effect, as previous studies have identified a close inverse relationship between PV and exercise HR, with a 1% increase in PV producing an equal degree of bradycardia (4). It is well known that endurance training has been associated with increased LV chamber volume (38), with small changes in wall thickness (40). In addition, chronic training increases the capacity to utilize the Frank-Starling relation without a change in blood volume (30, 37), possibly by increasing LV compliance (2, 31). This would enhance ventricular volume at a given filling pressure and may explain how SV could be increased following training. The exercise-induced increases in EDV and SV observed in the present study and those described elsewhere (34, 48) support the hypothesis that training elicits a volume-mediated change in ventricular performance. This adaptation may be proportional to the intensity of training, independent of the duration of the stimulus (33, 42).

Changes in DFRs, including the TPF, after training failed to reach statistical significance ($P = 0.08$). Although others have reported changes in diastolic filling times using a similar training intervention (24), we could not replicate these findings. However, interpretation of ventricular filling characteristics remains limited, because both HR and EF can alter the rapid phase of filling, both of which changed slightly at rest, the former significantly during exercise. The diastolic pressure-volume relation is typically shifted to the right in athletes, allowing a larger change in SV for a given filling pressure (30). Rapid changes in PV, such as observed in this study, would increase central venous pressure at rest and exercise (5, 6), producing a rise in EDV without a change in chamber compliance typically seen with long-term training (2, 30), although the present data and those of Harris et al. (24) suggest that compliance may well be enhanced after training, and this may contribute to increased EDV. Interestingly, in either trained or untrained subjects, blood volume expansion alone increases maximal SV and cardiac output secondary to improved diastolic filling (29), yet those with higher blood volumes elicit the greatest SV during exercise.

The change in SV reported here is similar to that of Spina et al. (42), who observed a change in SV at submaximal and maximal exercise following 12 wk of training. They did not measure ventricular volumes; however, they reported an increase in posterior wall thickness after training. Gillen et al. (14) suggested that an acute postexercise attenuation of baroreceptor activity precipitates a PV expansion without a change in peripheral vascular compliance. Although a training-induced bradycardia is known to have some neural (vagal) origins, the longer period of training required for neurally mediated changes in HR (40) are not compatible with the present model of training.

**Possible implications for prolonged exercise.** A decline in SV during prolonged exercise is a well-established observation (38) and is also associated directly with the rise in HR (11). Acute PV expansion has been shown to increase cardiovascular performance during exercise (increased SV and cardiac output, reduced HR), without improving thermal regulation (39), and others have shown that acute blood volume expansion improves exercise hemodynamics during short bouts of intensive exercise (4, 23, 25, 26, 35, 42). Conversely, HR can be prevented from drifting upward if the SV is arrested, as has been shown with $\beta$-blockade (13). The exercise challenge used in this study was unique in that it increased, in a stepwise fashion, exercise intensity over 60 min of exercise, with three step increments in work intensity. This had the effect of further challenging diastolic filling as exercise time extended (due to a rise in HR). Despite a reduced time for filling, the EDV and SV response following training was improved and may confer some benefit during prolonged exercise.

The elevated cardiac output after 60 min of exercise following training (Fig. 6) was an unexpected outcome of this study, yet a similar finding has been observed previously (19). It is our belief that this hyperkinetic circulatory response and “pseudoanemia” may reflect an optimization of $O_2$-carrying capacity. Green et al. (19) observed a decrease in red cell count in their earlier model of short-term training and concluded that the only method available to account for a maintenance of oxygen transport would be a commensurate increase in cardiac output. Our data support the hypothesis that, in the face of...
diminished arterial $O_2$ content, given an increase in cardiac volume reserve, a hyperkinetic cardiac output is compensatory in nature, resulting in an unchanged $V_O_2$ at the corresponding work rate.

Conclusions. Short-term exercise training, which elicits a expansion of PV, produces an enhanced SV and ventricular filling secondary to a Frank-Starling effect, with minimal contribution arising from improved systolic performance. The early change in cardiac function may reflect an immediate contribution arising from improved systolic performance. The technical assistance of Margaret Burnett is gratefully acknowledged.

REFERENCES


