Changes in stroke volume with β-blockade before and after 10 days of exercise training in men and women

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Mier, Constance M., Melissa A. Domenick, and Jack H. Wilmore. Changes in stroke volume with β-blockade before and after 10 days of exercise training in men and women. J. Appl. Physiol. 83(5): 1660–1665, 1997. We sought to determine whether 10 days of training would be a sufficient stimulus for cardiac adaptations that would allow a greater compensatory stroke volume during β-blockade. We also sought to determine whether men and women had a similar cardiac reserve capacity for increasing stroke volume with β-blockade during submaximal exercise. Eight men (age 29 ± 2 yr, mean ± SE) and eight women (25 ± 2 yr) cycled at 65% of peak O2 consumption (unblocked) under placebo-control and β-blockade (100 mg atenolol) conditions performed on separate days. These tests were repeated at the same power output after training (10 consecutive days, 1 h of cycling per day). Before training, β-blockade significantly (P < 0.05) decreased heart rate (HR) and cardiac output and increased stroke volume in both men and women. After training, the increase in stroke volume and decrease in HR with β-blockade was significantly less while cardiac output was reduced more. There were no gender differences in the effects of β-blockade on HR, stroke volume, or cardiac output. These data indicate that, during exercise with β-blockade, exercise training for 10 days does not enhance the compensatory increase in stroke volume in untrained men and women. After training, β-blockade during submaximal exercise with β-blockade was significantly less while cardiac output was reduced more. There were no gender differences in the effects of β-blockade on HR, stroke volume, or cardiac output. These data indicate that, during exercise with β-blockade, exercise training for 10 days does not enhance the compensatory increase in stroke volume in untrained men and women. After training, β-blockade during submaximal exercise with β-blockade was significantly less while cardiac output was reduced more. There were no gender differences in the effects of β-blockade on HR, stroke volume, or cardiac output. These data indicate that, during exercise with β-blockade, exercise training for 10 days does not enhance the compensatory increase in stroke volume in untrained men and women.

METHODS

Subjects. Eight men and eight women participated in this study. They were healthy, sedentary nonsmokers and had not participated in regular endurance exercise for at least 1 yr before this study. Age (mean ± SE) did not differ between men (29 ± 2 yr) and women (25 ± 2 yr), and men had a greater body mass (76.9 ± 3.7 kg) than women (63.5 ± 3.6 kg). All subjects completed an activity questionnaire, reviewed the study protocol and associated risks, and signed a consent form that had received prior approval by the University of Texas at Austin Institutional Review Board. According to their responses to the activity questionnaire, men and women had similar activity levels before participation in this study.

Experimental design. The first laboratory visit included a test to exhaustion on an upright electrically braked cycle ergometer (model Ergometrics-800S; SensorMedics, Yorba Linda, CA) to determine peak O2 uptake (cycle VO2peak). Several days after the first visit, single-blinded and randomly ordered tests were performed under placebo-control or β-blockade conditions on 2 separate days. These tests were separated by at least 3 nontest days to allow sufficient washout time for the β-blocker. During these visits, a submaximal test (upright cycling) was performed at 65% of cycle VO2peak that lasted ~20 min. O2 uptake (VO2), HR, blood pressure, cardiac output, and blood hemoglobin (Hb) concentration were determined during the submaximal exercise test.

β-blockade markedly decreases heart rate (HR) during exercise. Despite this large reduction in HR, cardiac output is reduced only 5–14% because of the compensatory increase in stroke volume (16, 21, 22, 27). Because both endurance exercise training and β-blockade independently increase stroke volume in untrained men, and because β-blockade increases stroke volume in endurance-trained men who have a relatively large blood volume (16), it is possible that the combined effects of training and β-blockade on stroke volume would be greater than the single effect of β-blockade if the untrained subject has a sufficient cardiac reserve capacity. Because significant cardiac adaptations occur within several days of endurance exercise training (6), 10 days of training may be a sufficient stimulus for cardiac adaptations that would allow a greater compensatory stroke volume during exercise with β-blockade.

Studies in which stroke volume responses to endurance exercise training in women have been investigated are few, and their results conflicting (4, 5, 17, 24, 25). Although increases in stroke volume during submaximal (4) and maximal (17, 25) exercise have been reported in women after training, others have failed to show these increases (5, 24). Furthermore, gender differences in left ventricular function during acute exercise appear to exist in that the increase in ejection fraction and stroke volume from rest to exercise is less in women (1, 12, 14). In light of these data, it is possible that women do not hold a similar cardiac reserve capacity for increasing stroke volume compared with men. This could place women at a disadvantage during exercise with β-blockade if stroke volume cannot adequately compensate for a reduction in HR.

The purpose of this study was twofold: 1) to determine whether, because of an enhanced compensatory increase in stroke volume, 10 days of endurance exercise training would attenuate the reduction in cardiac output with β-blockade during submaximal exercise in young, sedentary men and women and 2) to determine possible gender differences in the cardiac output and stroke volume responses to β-blockade during submaximal exercise. In addition, the study design allowed us to examine possible interactive effects of gender, training, and β-blockade on the hemodynamic responses to submaximal exercise. We hypothesized that 10 days of endurance exercise training would result in a greater compensatory increase in stroke volume during submaximal exercise with β-blockade. Second, we hypothesized that women would demonstrate less increase in stroke volume during submaximal exercise with β-blockade, resulting in a greater decrease in cardiac output compared with men.

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Three hours before each of the submaximal cycle tests, the subject ingested either a placebo tablet or a 100-mg atenolol tablet (β-blocker). The dose and time of ingestion relative to the test were chosen to ensure a maximum β-blockade effect on HR (2a). The drug atenolol was chosen because, on the basis of previous observations made from this laboratory, less discomfort (i.e., shortness of breath, general fatigue, nausea) would be experienced compared with the use of a nonselective β-blocker such as propranolol.

After completing the tests described above, the subjects began endurance exercise training on a cycle ergometer for 1 h/day on 10 consecutive days. After the training period, the single-blinded, randomly ordered placebo and β-blockade tests were repeated. The power output (watts) during these posttraining submaximal cycle tests was identical to the pretraining power output. Because these tests were separated by 3 nontest days, subjects performed 1 h of training on 1 of the nontest days to maintain the training effects.

Exercise tests. All exercise tests were performed under controlled environmental conditions (21°C, 50% relative humidity). Expired ventilatory volume and concentrations of O2 and CO2 gas were measured continuously during both submaximal and maximal tests by a SensorMedics 2900 metabolic cart, an automated computerized analysis system. During the cycle VO2peak test, subjects warmed up at a low intensity for 2 min. This was followed by progressive increases in work rate of 25 W each minute until voluntary exhaustion. The criteria for achieving cycle VO2peak were a respiratory exchange ratio value >1.10 and a HR within 10% of the age-predicted maximum HR. HR was monitored by using a HR monitor (model XL; Polar USA, Montvale, NJ).

During the submaximal tests, steady-state VO2 was measured after 5–7 min, immediately before initiation of noninvasive CO2 rebreathing maneuvers for determination of cardiac output. The Collier rebreathing method was used to determine CO2 equilibrium (3) from which cardiac output was estimated by using the indirect Fick equation corrected for Hb (15). Three CO2-rebreathing maneuvers were performed within a 15-min period, and the average cardiac output was calculated from these measures. HR was recorded every minute, and before each CO2-rebreathing maneuver, systolic (SBP) and diastolic blood pressures were measured by using an auscultatory cuff attached to a fully automated blood pressure monitor (model STBP-680, Colin Medical Instruments, San Antonio, TX). A 0.5-ml blood sample was drawn without stasis for Hb measures after the last CO2-rebreathing maneuver. Hb was measured in quadruplicate by using the cyanmethemoglobin method.

The three cardiac output measures taken during the placebo-control and β-blockade tests yielded an average coefficient of variation of 4.1 and 3.9%, respectively. SBP and DBP yielded an average coefficient of variation of 3.0 and 5.7%, respectively, for the placebo-control tests and 2.7 and 4.6%, respectively, for the β-blockade tests. The day-to-day reproducibility for cardiac output, SBP, and DBP determined at 60% of VO2peak has been previously reported from this laboratory (28). For cardiac output, SBP, and DBP, the coefficient of variation was 5.9, 5.7 and 8.3%, respectively, and the intraclass correlation coefficients were 0.93, 0.82, and 0.77, respectively.

Cardiac output and HR were used to calculate stroke volume. Mean blood pressure (MBP) was calculated as [(2 × DBP) + SBP]/3. Using a correction factor of 80 (converting mmHg·min⁻¹·l⁻¹ to dyn·s·cm⁻²), total peripheral resistance (TPR) was calculated from MBP and cardiac output.

Training. Training consisted of 1-h cycling bouts performed daily on 10 consecutive days. During each bout, subjects cycled for 30 min at a power output that elicited ~80% of peak HR. During the second 30-min period, subjects cycled at a power output that elicited ~95% of peak HR for 2 min, followed by 1 min of low-intensity pedaling, for a total of 10 intervals. Power output was increased on a daily basis to achieve the appropriate training HR within a range of 5 beats/min.

Statistical analyses. A three-way repeated-measures analysis of variance (1 between, 2 within) was performed to determine the effects of β-blockade, gender, and training on the physiological responses to submaximal exercise. Where a significant interactive effect occurred, a Duncan’s multiple-range test was performed to determine significant differences among groups. Significant differences were established at P < 0.05, and data were expressed as means ± SE.

RESULTS

Effects of training and β-blockade. Ten days of endurance exercise training significantly increased cycle VO2peak similarly in men (3.14 ± 0.13 vs. 3.42 ± 0.13 l/min) and in women (2.11 ± 0.10 vs. 2.37 ± 0.12 l/min). Peak HR did not change with training in either men (190 ± 3 vs. 190 ± 3 beats/min) or women (189 ± 3 vs. 189 ± 2 beats/min). When measurements were determined at 65% of pretraining VO2peak (unblocked), we found that training significantly decreased HR and increased stroke volume in both men and women while having no effect on VO2, cardiac output, SBP, DBP, MBP, or TPR (Table 1).

At 65% of pretraining VO2peak, β-blockade significantly reduced HR, cardiac output, SBP, DBP, and MBP, and increased stroke volume compared with placebo control (Table 2). After subjects were trained, the effect of β-blockade on HR was attenuated (Table 3, column T × B). However, the decrease in cardiac output with β-blockade was greater after training because of the lower increase in stroke volume. Both DBP and MBP were reduced more after training with β-blockade.

Table 1. Physiological responses at 65% of pretraining cycle VO2peak during placebo-control tests before and after 10 days of endurance exercise training

<table>
<thead>
<tr>
<th></th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretraining</td>
<td>Posttraining</td>
</tr>
<tr>
<td>VO2, l/min</td>
<td>1.38 ± 0.06</td>
<td>1.38 ± 0.07</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>152 ± 2</td>
<td>142 ± 2*</td>
</tr>
<tr>
<td>Stroke volume, ml/beat</td>
<td>85 ± 4</td>
<td>96 ± 6*</td>
</tr>
<tr>
<td>Cardiac output, l/min</td>
<td>12.9 ± 0.6</td>
<td>13.5 ± 0.8</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>148 ± 7</td>
<td>156 ± 6</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>68 ± 4</td>
<td>74 ± 3</td>
</tr>
<tr>
<td>MBP, mmHg</td>
<td>95 ± 4</td>
<td>101 ± 3</td>
</tr>
<tr>
<td>TPR, dyn.s.cm⁻²</td>
<td>588 ± 30</td>
<td>605 ± 36</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 8 women, 8 men. VO2, O2 uptake; VO2peak, peak VO2; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure; TPR, total peripheral resistance calculated from cardiac output and MBP. *P < 0.05 vs. pretraining.
ing cycle $\dot{V}O_2_{peak}$ during both the placebo-control and $\beta$-blockade tests in men and women. HR was significantly decreased with training during both the placebo-control and $\beta$-blockade tests. Whereas stroke volume was significantly greater after training during the placebo-control test, there was no difference in stroke volume before and after training during the $\beta$-blockade test. Cardiac output tended to be greater after training during the placebo-control test ($P < 0.053$) but tended to be lower after training during the $\beta$-blockade test ($P < 0.086$).

Interactive effects of training and $\beta$-blockade with gender. At 65% of pretraining $\dot{V}O_2_{peak}$, men had significantly greater $\dot{V}O_2$, stroke volume, cardiac output, SBP, DBP, and MBP. Neither HR nor TPR differed between men and women. There were no significant interactive effects of training and gender or $\beta$-blockade and gender (Table 3, columns $T \times G$ and $B \times G$). There was a tendency for cardiac output to be reduced more in men with $\beta$-blockade ($P < 0.053$); however, the effect of $\beta$-blockade was significant in both men and women. Although SBP and MBP were both significantly reduced with $\beta$-blockade before and after training, the effect after training was greater in women compared with men and compared with women's pretraining responses (Table 3, column $T \times B \times G$). In women, there was a tendency for $\beta$-blockade to increase TPR before training and to decrease it after training ($P < 0.61$). In men, the effects of $\beta$-blockade on SBP, DBP, MBP, and TPR were similar before and after training.

Figure 2 illustrates the effects of training on MBP and TPR during both the placebo-control and $\beta$-blockade tests. In women, MBP was significantly greater after training during the placebo-control tests, whereas during the $\beta$-blockade test, MBP tended to be lower after training ($P < 0.057$). Similarly, in women, SBP was significantly greater during the placebo-control tests after training, whereas during the $\beta$-blockade test, SBP tended to be lower after training ($P < 0.071$). TPR or

### Table 2. Physiological responses at 65% of pretraining cycle $\dot{V}O_2_{peak}$ during $\beta$-blockade tests before and after 10 days of endurance exercise training

<table>
<thead>
<tr>
<th></th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}O_2$, l/min</td>
<td>$1.35 \pm 0.06$</td>
<td>$2.00 \pm 0.07$</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>$116 \pm 2$</td>
<td>$114 \pm 2$</td>
</tr>
<tr>
<td>Stroke volume, ml/beat</td>
<td>$103 \pm 4$</td>
<td>$128 \pm 4$</td>
</tr>
<tr>
<td>Cardiac output, l/min</td>
<td>$11.9 \pm 0.5$</td>
<td>$14.4 \pm 0.4$</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>$129 \pm 5$</td>
<td>$152 \pm 4$</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>$66 \pm 3$</td>
<td>$78 \pm 4$</td>
</tr>
<tr>
<td>MBP, mmHg</td>
<td>$87 \pm 3$</td>
<td>$103 \pm 3$</td>
</tr>
<tr>
<td>TPR, dyn·s·cm$^{-5}$</td>
<td>$596 \pm 33$</td>
<td>$573 \pm 27$</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 8 women, 8 men. *$P < 0.05$ vs. placebo control (Table 1).

### Table 3. Analysis-of-variance significance for interactive effects of gender, training, and $\beta$-blockade effects

<table>
<thead>
<tr>
<th></th>
<th>$T \times B$</th>
<th>$T \times G$</th>
<th>$B \times G$</th>
<th>$T \times B \times G$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}O_2$, l/min</td>
<td>0.11</td>
<td>0.388</td>
<td>0.290</td>
<td>0.772</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>0.006</td>
<td>0.448</td>
<td>0.375</td>
<td>0.240</td>
</tr>
<tr>
<td>Stroke volume, ml/beat</td>
<td>&lt;0.001</td>
<td>0.736</td>
<td>0.416</td>
<td>0.865</td>
</tr>
<tr>
<td>Cardiac output, l/min</td>
<td>0.014</td>
<td>0.434</td>
<td>0.053</td>
<td>0.203</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>0.147</td>
<td>0.595</td>
<td>0.589</td>
<td>0.011</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>0.020</td>
<td>0.245</td>
<td>0.359</td>
<td>0.158</td>
</tr>
<tr>
<td>MBP, mmHg</td>
<td>0.013</td>
<td>0.280</td>
<td>0.711</td>
<td>0.012</td>
</tr>
<tr>
<td>TPR, dyn·s·cm$^{-5}$</td>
<td>0.380</td>
<td>0.864</td>
<td>0.170</td>
<td>0.061</td>
</tr>
</tbody>
</table>

$T$, training; $B$, $\beta$-blockade; $G$, gender.
Previously, stroke volume has been shown to increase the increase before training (20 vs. 9%, respectively) in both men and women after training was exercise, the increase in stroke volume with exercise blockade after training because of an inadequate increase in cardiac output with exercise blockade during submaximal exercise. Instead, there was a greater attenuation of cardiac output with exercise blockade in men and women after 10 days of endurance exercise training under placebo-control conditions. Furthermore, cardiac output during exercise blockade tended to be lower after training because of a lower HR response during exercise.

Differences in stroke volume response to exercise blockade between these subjects, who underwent 10 days of endurance exercise training, and highly trained athletes suggests that there are some myocardial or extramyocardial adaptations associated with long-term training that do not occur within 10 days (13). After 10 days of endurance exercise training, end-diastolic volume may be at or near maximum capacity by having reached the limit of the myocardium and/or pericardium such that preload could increase no further with exercise blockade. These data indicate that 10 days of endurance exercise training is not an adequate training stimulus for cardiac adaptations that would allow a greater compensatory stroke volume during exercise blockade, despite there being a greater stroke volume during placebo control.

Effects of gender. The effects of exercise blockade on HR and stroke volume during submaximal exercise were similar in men and women. Furthermore, 10 days of endurance exercise training increased stroke volume similarly in men and women. A compensatory increase in stroke volume with exercise blockade results from a greater preload facilitated by enhanced diastolic filling time and from a reduced afterload that accommodates left ventricular emptying (2, 21, 23). Our data indicate that the compensatory increase in stroke volume previously demonstrated in men (16, 21, 22, 27) occurs as well in women. This result suggests that women have a similar cardiac reserve capacity for raising stroke volume during submaximal exercise under exercise blockade conditions. Previously, gender differences in left ventricular function were observed during acute exercise, possibly mediated by differences in afterload or the contractile reserve of the left ventricle (1, 12, 14). It is possible that whatever the mechanisms for gender differences in left ventricular function are, they may not become apparent at moderate exercise intensities during exercise blockade.

An unexpected result was that, after 10 days of training, exercise blockade reduced blood pressure more in women than in men, despite having a similar effect on cardiac output in men and women. Reportedly, men respond to isometric exercise and postexercise ischemia with greater muscle sympathetic nervous activity as well as a greater blood pressure response, independent of muscle mass (7). Furthermore, during orthostatic challenge, women demonstrate a similar or greater increase in HR, whereas men demonstrate a greater increase in TPR (8, 10). These data indicate gender differences in cardiovascular control under specific conditions. Unlike the men in this study, women did not increase TPR with exercise blockade. As a result, blood pressure was more greatly compromised under exercise blockade conditions in women after training. Mechanisms for these gender differences are not known. However, because of the potent vasodilatory effects of estrogen (9, 18, 26), it is possible that estrogen potentiates the endothelial-mediated increase in vasodilation that occurs in the skeletal muscle vasculature during exercise.

**DISCUSSION**

Effects of training. Ten days of endurance exercise training did not attenuate the effect of exercise blockade on cardiac output during submaximal exercise. Instead, there was a greater attenuation of cardiac output with exercise blockade after training because of an inadequate increase in stroke volume. During submaximal exercise, the increase in stroke volume with exercise blockade in both men and women after training was ~50% that of the increase before training (20 vs. 9%, respectively). Previously, stroke volume has been shown to increase ~20% with exercise blockade during exercise in highly trained men (16); this suggests that the capacity to raise stroke volume under the effects of exercise blockade is not limited by an already large preload in these athletes. Although 10 days of endurance exercise training resulted in an increase in stroke volume during submaximal exercise under placebo-control conditions, stroke volume was no different before and after training under exercise blockade conditions. Furthermore, cardiac output during exercise blockade tended to be lower after training because of a lower HR response during exercise.

**Fig. 2.** Mean blood pressure (A) and total peripheral resistance (TPR) (B) measured at 65% of pretrained VO$_{2peak}$ under placebo-control (no block) and exercise blockade conditions before and after 10 days of endurance exercise training. Symbols and lines as in Fig. 1. *P < 0.05 vs. pretraining.

DBP did not differ before and after training in women during either the placebo-control or exercise blockade test. In men, there were no differences in SBP, DBP, MBP, and TPR before and after training during either the placebo-control or exercise blockade tests.
Under β-blockade conditions when cardiac output and HR are compromised, an enhanced vasodilatory response from estrogen may not allow TPR to increase adequately, which would in turn result in a lower blood pressure response.

Limitations. Our sample size of eight men and eight women might limit the interpretation of our data. It is possible that significant differences may arise with a larger sample population. We recognize that the blood pressure response to β-blockade was not the primary focus of this study. Therefore, not having measured either sympathetic nerve activity or blood levels of catecholamines limits our interpretation of the blood pressure data. Such measurements might reveal potential mechanisms for gender differences in blood pressure regulation during exercise with β-blockade.

Each subject ingested 100 mg of atenolol before exercise. Because of gender differences in body weight, women received a greater dose relative to their weight; this fact may have resulted in a greater effect in women. However, this did not appear to be the case, because β-blockade reduced HR similarly in men and women. Because of the number of tests and the time necessary to allow sufficient β-blockade washout, we were unable to control for the menstrual cycle. The menstrual cycle may have confounded some of the results, because a higher HR response during exercise has been reported during the luteal phase compared with the follicular phase (11, 20). However, in this study, possible menstrual cycle effects may have been masked by the training and β-blockade effects. Furthermore, there were no differences in HR response during exercise in men and women.

Conclusions. Ten days of endurance exercise training did not attenuate the effect of β-blockade on cardiac output during submaximal exercise because of an inadequate increase in stroke volume. This indicates that although stroke volume increased during placebo-control phases, 10 days of training is not an adequate training stimulus to provide a greater compensatory increase in stroke volume during β-blockade. Second, β-blockade had a similar effect on stroke volume in men and women during submaximal exercise, indicating that men and women have a similar cardiac reserve capacity for increasing stroke volume during β-blockade. In addition, men better maintained their blood pressure response to submaximal exercise during β-blockade after endurance exercise training, indicating gender differences in the mechanisms involved in cardiovascular control during exercise.

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