Effects of 3-day bed rest on physiological responses to graded exercise in athletes and sedentary men

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Effects of 3-day bed rest on physiological responses to graded exercise in athletes and sedentary men. J Appl Physiol 91: 249–257, 2001.—To test the hypotheses that short-term bed-rest (BR) deconditioning influences metabolic, cardiorespiratory, and neurohormonal responses to exercise and that these effects depend on the subjects’ training status, 12 sedentary men and 10 endurance- and 10 strength-trained athletes were submitted to 3-day BR. Before and after BR they performed incremental exercise test until volitional exhaustion. Respiratory gas exchange and heart rate (HR) were recorded continuously, and stroke volume (SV) was measured at submaximal loads. Blood was taken for lactate concentration ([LA]), epinephrine concentration ([NE]), human growth hormone concentration ([hGH]), testosterone, and cortisol determination. Reduction in lactate concentration ([LA]), epinephrine concentration ([NE]), human growth hormone concentration ([hGH]), testosterone, and cortisol determination. Reduction of peak oxygen uptake (V\textsuperscript{\textcircled{\small O}_2\textsubscript{peak}}) after BR was greater in the endurance athletes than in the remaining groups (17 vs. 10%). Decrements in V\textsuperscript{\textcircled{\small O}_2\textsubscript{peak}} correlated positively with the initial values (r = 0.73, P < 0.001). Resting and exercise respiratory exchange ratios were increased in athletes. Cardiac output was unchanged by BR in all groups, but exercise HR was increased and SV diminished in the sedentary subjects. The submaximal [LA] and [LA] thresholds were decreased in the endurance athletes from 71 to 60% V\textsuperscript{\textcircled{\small O}_2\textsubscript{peak}} (P < 0.001); they also had an earlier increase in [NE], an attenuated increase in [hGH], and accentuated PRA and cortisol elevations during exercise. These effects were insignificant in the remaining subjects. In conclusion, reduction of exercise performance and modifications in neurohormonal response to exercise after BR depend on the previous level and mode of physical training, being the most pronounced in the endurance athletes.

exercise tolerance; blood lactate threshold; catecholamines; hormones; plasma renin activity

Prolonged bed rest (BR) causes reduction of exercise performance as a result of impairment of oxygen transport (7, 16, 17) and thermoregulation (15, 22), disturbances in intermediary metabolism (3, 30), and adverse changes in musculoskeletal structure and function (4).

Both cardiovascular and metabolic adjustments to exercise are controlled by the autonomic nervous and endocrine systems. In addition, enhanced secretion of growth hormone and testosterone exert a prolonged effect by stimulating anabolic processes in skeletal muscles and other tissues after exercise. Thus, for better understanding of the BR deconditioning mechanisms, it is necessary to elucidate functioning of the neural and hormonal regulatory systems.

Importance of the sympathetic nervous system (SNS) for determining work tolerance and aerobic capacity during deconditioning was emphasized by Sullivan et al. (34), who showed that administration of dobutamine (a synthetic adrenomimetic drug) to BR subjects prevented the decline in peak oxygen uptake (V\textsuperscript{\textcircled{\small O}_2\textsubscript{peak}}) and attenuated the increase in blood lactate concentration ([LA]) during exercise. However, there are few data on the influence of BR deconditioning on the sympathetic nervous system response to exercise. Engelke and Convertino (14) demonstrated a marked increase in the post-maximal-exercise plasma norepinephrine concentration ([NE]) after 16 days of BR without change in plasma epinephrine ([Epi]), whereas Sullivan et al. (34) found, after 21 days of BR, higher arterial [Epi] during submaximal exercise without significant change in [NE] at both submaximal and maximal exercise. Microneurography data during forearm isometric exercise indicated that forearm muscle sympathetic nerve activity (MSNA) during isometric exercise after 14 days of BR was similar to that before BR, but the MSNA response to muscle ischemia, induced by circulatory arrest after exercise, was attenuated (24). These findings indicate that the metaboreflex from skeletal muscles, which activates SNS, can be attenuated by BR deconditioning.

Little is known about the effects of BR on endocrine responses to exercise. McCall et al. (27) utilized a series of isometric plantar flexions after BR and found...
that an increase in plasma bioassayable growth hormone concentration was inhibited, suggesting that lack of activity and/or unloading of skeletal muscles causes disruption of the muscle afferent-pituitary axis, modulating growth hormone release. They did not find any influence of BR on either resting or postexercise levels of plasma immunoassayable growth hormone (hGHI), testosterone, cortisol, or thyroid hormones. There appear to be no data on the effect of BR on the renin-angiotensin system response to exercise, although an increase in resting plasma renin activity (PRA) during BR has been reported consistently (18).

Physiological responses to exercise depend on the subjects’ level of physical fitness; however, except for maximal oxygen uptake (Vo2 max), few data are available on the impact of subjects’ fitness status (initial Vo2 max) on neuroendocrine responses to exercise after BR. Some data indicate that the magnitude of the Vo2 max decline during BR is greater in highly fit subjects than in those with lower working capacity (9, 10, 31, 35). However, a positive correlation between the initial Vo2 max per kilogram body mass and its percent decrease was not confirmed (21) unless the subjects exercised in the supine position, which minimized the influence of orthostatic factors on cardiovascular adjustments (10).

The present study was designed to test the following hypotheses: 1) short-term BR influences neurohormonal responses to exercise, and 2) metabolic, cardiorespiratory, and neurohormonal responses to exercise after BR depend on the level and mode of the subjects’ habitual physical activity and their working capacity. Thus cardiorespiratory parameters, blood lactate [LA], plasma [Epi], [NE], [hGHI], testosterone, cortisol, and PRA responses to graded incremental exercise were compared among sedentary men and endurance-trained and strength-trained athletes after 3 days of BR deconditioning.

METHODS

Subjects. Twelve healthy, untrained male students, 10 endurance-trained athletes (cyclists), and 10 bodybuilders volunteered for this study after giving written informed consent (Table 1). The study protocol was approved by the Ethics Committee of Academy of Physical Education in Poznań, Poland. The endurance-trained athletes were amateur cyclists who had been training regularly for 5.5 ± 2.7 (SD) yr, and their average training distance was 100 km/wk. The bodybuilders’ resistance training experience was 3.6 ± 1.8 (SD) yr, and they were currently training for at least 3 h/wk with a program that included bench press and leg press and squat exercise.

Procedure. The BR was conducted in the students’ hospital in Poznań where the subjects reported 2–3 days after their last training session. Three days of BR were selected because exposure to a few days of inactivity is sufficient to attenuate exercise performance (18). Also, such a short period of confinement is often prescribed for treatment of injury and mild diseases. During BR the subjects were allowed to ambulate no more than 20 min/day (to shower and toilet); for the rest of the day they read books, listened to the radio, and watched television in the supine position. A nursing staff provided 24-h care. The subjects had mineral water ad libitum, and their diet consisted of three meals per day freshly prepared in the hospital kitchen with a total energy intake of 12,000 kJ/day (carbohydrates 50%, fat 35%, protein 15%).

Before and after BR the subjects performed a graded, incremental cycle exercise test in the upright (sitting) position, between 8:30 and 10:00 AM, 2 h after a light breakfast. The load, starting from 50 W, was increased by 50 W each 3 min until volitional exhaustion and the stages were separated by 1-min rest intervals. During exercise the pulmonary minute ventilation (VE) and respiratory gas exchange were measured continuously using the Cardiopulmonary Exercise System (MedGraphics, St. Paul, MN), and heart rate (HR) was recorded by the Sport Tester (PE 3000, Polar Electro, Kempele, Finland). Stroke volume (SV) and cardiac output were measured during submaximal loads up to 100–150 W by impedance cardiography (26) using a monitoring device designed in this laboratory (11). Validity of SV measurements was determined at rest by using echocardiography (r = 0.90, n = 21, P < 0.001) (12) and during exercise by using the CO2-rebreathing method (r = 0.72, n = 10, P < 0.01).

Immediately after each exercise load, 3-ml blood samples were taken through an indwelling catheter inserted 30 min before exercise into the antecubital vein for blood [LA] and plasma [Epi] and [NE] determinations. Additional 5-ml blood samples were taken before and after exercise for determination of PRA, [hGHI], and testosterone and cortisol concentrations.

Analytic methods. Blood [LA] was measured enzymatically by using commercial kits (Boehringer, Mannheim, Germany), whereas plasma [Epi] and [NE] were measured by the radioenzymatic method of DaPrada and Zurcher (13) using the Catechola tests produced by Immunotech (Prague, Czech Republic). Plasma [hGHI] was determined by radioimmunoassay using the HGH-IRMA MI-131 kits (Polatolm, Swierk, Poland), plasma cortisol and testosterone concentrations with the radioimmunoassay kits of Orion Diagnostica (Espoo, Finland), and PRA by radioimmunoassay using Immunotech Angiotensin I kits (Prague, Czech Republic). The intra-assay analytic errors (coefficients of variation) for Epi, NE, hGHI, PRA, cortisol, and testosterone were 10.8, 8.7, 4.2, 4.4, 7.2, and 3.5%, respectively.

Calculations. The exercise loads associated with a rapid increase in blood LA, Epi, and NE concentrations were defined as thresholds of these variables and calculated by using

Table 1. Characteristics of subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Age, yr</th>
<th>Body Mass, kg</th>
<th>Height, cm</th>
<th>Body Mass Index, kg/m²</th>
<th>Peak Vo2peak, ml/kg−1·min−1</th>
<th>Duration of Training, yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untrained</td>
<td>12</td>
<td>23.3 ± 1.4</td>
<td>78.2 ± 8.9</td>
<td>181.6 ± 6.3</td>
<td>23.7 ± 2.2</td>
<td>35.2 ± 7.3</td>
<td>0</td>
</tr>
<tr>
<td>Endurance</td>
<td>10</td>
<td>20.3 ± 1.9</td>
<td>75.1 ± 9.3</td>
<td>179.8 ± 6.6</td>
<td>23.2 ± 2.1</td>
<td>54.8 ± 2.1</td>
<td>5.5 ± 2.7</td>
</tr>
<tr>
<td>Strength</td>
<td>10</td>
<td>21.4 ± 1.3</td>
<td>69.9 ± 8.6†</td>
<td>181.9 ± 7.6</td>
<td>25.4 ± 2.0†</td>
<td>34.4 ± 3.8</td>
<td>3.6 ± 1.8</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of subjects. Vo2peak, peak oxygen uptake. * Significant difference between endurance athletes and untrained subjects or strength athletes, P < 0.001. † Significant difference between strength athletes and endurance athletes, P < 0.05.
the two-segmental linear regression (log exercise load vs. log [LA], [Epi], or [NE]) according to Beaver et al. (2).

Statistics. Statistical evaluation of differences between pre- and post-BR data was made using a two-way analysis of variance for repeated measures. The two factors were the testing condition and repeated measures of cardiorespiratory parameters, blood LA, and hormone concentrations during exercise. When a significant $F$ value was achieved, a paired Student's $t$-test was used to locate the pairwise differences between means. The same test was used for evaluation of differences between the pre- and post-BR maximal exercise load, $\dot{V}O_2$ peak, blood LA, and plasma catecholamine thresholds. A comparison between groups was made using a non-parametric Whitney-Mann test. Linear regression was used to evaluate a relationship between the initial $\dot{V}O_2$ peak and its decrease during BR. As a level of significance $P < 0.05$ was accepted. All results are presented as means ± SE unless otherwise stated.

RESULTS

BR reduced the maximal exercise load and $\dot{V}O_2$ peak in all groups (Table 2) without a change in the subjects' body mass (data not presented). The decreases in $\dot{V}O_2$ peak were more pronounced in the endurance-trained subjects (by 17%; $P < 0.01$) than in strength-trained or sedentary subjects (by 10%; $P < 0.01$). However, the post-BR $\dot{V}O_2$ peak in the endurance athletes was still higher ($P < 0.001$) than the initial value in the

Fig. 1. A relationship between the initial peak oxygen uptake ($\dot{V}O_2$ peak) and its decrement ($\Delta \dot{V}O_2$ peak) after bed rest. ○, Sedentary subjects; ●, endurance-trained athletes; †, strength-trained athletes.

Fig. 2. Respiratory exchange ratios during graded exercise before (○) and after (●) bed rest in 3 groups of subjects. Values are means ± SE. The last points (connected with dashed lines) represent mean values at the maximal exercise load. Significant difference between the pre- and post-bed-rest values: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

Table 2. Maximal exercise load, $\dot{V}O_2$ peak, and blood [LA] attained during the test and blood [LA] threshold before and after bed rest

<table>
<thead>
<tr>
<th>Group</th>
<th>Maximal Load, W</th>
<th>$\dot{V}O_2$ peak, l/min</th>
<th>Maximal [LA], mmol/l</th>
<th>[LA] Threshold, W</th>
<th>% $\dot{V}O_2$ Threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untrained subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>216.6 ± 7.1</td>
<td>2.614 ± 0.079</td>
<td>8.40 ± 0.91</td>
<td>126.8 ± 6.5</td>
<td>65.0 ± 3.4</td>
</tr>
<tr>
<td>After</td>
<td>187.5 ± 6.5*</td>
<td>2.360 ± 0.054*</td>
<td>6.97 ± 0.72</td>
<td>107.3 ± 10.2*</td>
<td>59.8 ± 4.9</td>
</tr>
<tr>
<td>Endurance athletes</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Before</td>
<td>315.0 ± 18.0‡</td>
<td>4.032 ± 0.108‡</td>
<td>6.14 ± 0.67</td>
<td>193.1 ± 10.2‡</td>
<td>71.2 ± 3.9‡</td>
</tr>
<tr>
<td>After</td>
<td>270.0 ± 11.0*</td>
<td>3.367 ± 0.077*</td>
<td>4.86 ± 0.57</td>
<td>145.3 ± 9.1†</td>
<td>59.7 ± 4.6†</td>
</tr>
<tr>
<td>Strength athletes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>250.0 ± 12.9§</td>
<td>2.930 ± 0.152</td>
<td>7.53 ± 0.49</td>
<td>120.1 ± 10.6</td>
<td>58.0 ± 4.5</td>
</tr>
<tr>
<td>After</td>
<td>225.0 ± 11.0*</td>
<td>2.624 ± 0.142*</td>
<td>7.95 ± 0.55</td>
<td>108.0 ± 9.4</td>
<td>56.6 ± 4.0</td>
</tr>
</tbody>
</table>

Values are means ± SE. [LA], lactate concentration. *Significantly different from values before bed rest, $P < 0.01$. †Significantly different from values before bed rest, $P < 0.001$. ‡Significant difference between endurance athletes and untrained subjects or strength athletes, $P < 0.001$. §Significant difference between strength athletes and untrained subjects, $P < 0.05$.
sedentary subjects. The individual values of BR-induced decreases in $V_\text{O}_2$ peak (l/min) correlated positively with the initial $V_\text{O}_2$ peak expressed in liters per minute (Fig. 1; $r = 0.73$, $P < 0.001$). Significant correlations were also present when the decreases in $V_\text{O}_2$ peak were expressed as percentages of the initial values ($r = 0.64$, $P < 0.001$), when $V_\text{O}_2$ peak and its decreases were calculated per kilogram body mass ($r = 0.52$, $P < 0.01$), and when the decreases of $V_\text{O}_2$ peak per kilogram body mass were expressed as percentages of the initial values ($r = 0.40$, $P < 0.01$).

The resting and submaximal $V_\text{E}$ values were similar before and after BR in all groups. Maximal $V_\text{E}$ was lowered significantly by BR (94.7 ± 6.9 vs 74.5 ± 3.8 l/min; $P < 0.05$) in the endurance athletes, but the ratio of $V_\text{E}$ to oxygen uptake ($V_\text{O}_2$) was unchanged. Before BR the respiratory exchange ratio (RER) at rest was significantly higher ($P < 0.01$) in the endurance-trained than in sedentary or strength-trained subjects. After BR the RER values at rest and during submaximal loads were higher than before BR in both groups of athletes but not in the sedentary subjects (Fig. 2).

Before BR the preexercise HR and SV did not differ significantly between the groups. Resting HR was increased and SV was decreased after BR in the sedentary and endurance-trained subjects. During submaximal exercise the HR were increased and SV decreased by BR only in the untrained subjects (Fig. 3). In no group did BR modify cardiac output during exercise.

Maximal [LA] was unchanged in all groups by BR (Table 2, Fig. 4); however, the submaximal [LA] values were higher after BR and the blood [LA] threshold was shifted to lower workloads in the endurance athletes (Table 2).

Similar to blood [LA], the plasma [Epi] and [NE] increased exponentially during exercise (Figs. 5 and 6). Before BR the calculated NE threshold was higher ($P < 0.01$) in both groups of athletes compared with sedentary subjects, with no significant difference between the endurance and strength athletes. The plasma Epi thresholds were similar in all groups. Neither preexercise nor exercise plasma [Epi] and [NE] was affected significantly by BR, but the plasma NE threshold was lowered by BR ($P < 0.05$) in the endurance athletes. The post-BR NE thresholds in athletes were still higher ($P < 0.01$) than in sedentary subjects. There was a significant correlation ($r = 0.48$, $P < 0.01$) between the BR-induced changes in [LA] and [NE] thresholds for all subjects. The correlation coefficient in the endurance athletes was 0.78 ($P < 0.01$). The plasma Epi thresholds were not affected by BR.
either resting or postexercise concentrations of cortisol and testosterone as well as PRA, whereas the postexercise plasma hGH levels were significantly higher ($P < 0.05$) in the athletes than in the sedentary subjects. In all groups, BR elevated significantly ($P < 0.01$) resting PRA; the highest values occurred in the sedentary subjects and differed significantly ($P < 0.01$) from those in both groups of athletes. In the endurance- and strength-trained athletes, the exercise-induced increases in PRA were greater after than before BR ($P < 0.01$ and $P < 0.05$, respectively).

**DISCUSSION**

The present data clearly showed that only 3 days of BR can cause marked reduction in exercise tolerance manifested by a decrease in maximal exercise load and corresponding $V\dot{O}_2$ peak that occurred during incremental exercise. The most pronounced effect of BR deconditioning was found in the endurance-trained athletes with the highest aerobic capacity. The strength-trained athletes, who did not differ significantly from sedentary subjects in their ambulatory $V\dot{O}_2$ peak, also had similar post-BR decreases in exercise tolerance. The percent decline in $V\dot{O}_2$ peak in the latter two groups (by $\sim 10\%$) is comparable to that reported after short-term BR deconditioning in highly fit but not regularly training subjects, whereas the nearly 17% reduction in $V\dot{O}_2$ peak in the endurance-trained athletes in the present study corresponds with levels obtained after 2–3 wk of BR (7). The significant correlation coefficients confirmed a relationship between the ambulatory exercise capacity and the decrease in post-BR $V\dot{O}_2$ peak in a relatively large group of 32 subjects.

BR deconditioning involves effects of changes in body position and usually a decrease in physical activity. The contribution of postural changes to the effect of BR was probably similar in all three groups of subjects. However, the degree of reduction in physical activity was certainly greater in the endurance athletes than in the sedentary subjects. With the bodybuilders, training was directed mainly to enhance their muscle mass and strength but not their aerobic capacity; thus their habitual physical activity was also reduced by BR more than in the sedentary subjects, but the test applied in this study was not specific for their training regime. Thus the more pronounced decrease of aerobic exercise performance in endurance athletes than in strength-trained or sedentary subjects, as well as lack of a major difference between the two latter groups, was expected.

Alterations in cardiac and vascular functions induced by prolonged BR deconditioning have been reported to be the main factors responsible for diminution of exercise performance (7, 16, 17), but the mechanisms of the reduction of exercise performance after 3 days of BR are not clear. In the present study, the maximal cardiac output was not measured; however, in the sedentary subjects, the submaximal SV
values were diminished and HR was elevated after BR. If it is assumed that SV during graded exercise reaches the maximum level at submaximal loads, it might be expected that the maximal cardiac output was reduced in this group. On the other hand, in neither group of athletes was the exercise HR or SV up to 150 W altered by BR. This suggests that reduction of maximal cardiac output is not the main factor responsible for the decline in \( \dot{V}O_2 \) peak. However, SV in the athletes can increase progressively during incremental exercise with no plateau (20). Thus, on the basis of the submaximal SV values, the maximal cardiac output cannot be predicted and the contribution of reduced maximal cardiac output to limitation of performance after BR cannot be excluded.

The reduced exercise performance cannot be attributed to decreased pulmonary gas exchange because the maximal exercise \( \dot{V}E \) did not differ significantly from the pre-BR values in the sedentary subjects and strength athletes. In the endurance-trained subjects, the maximal \( \dot{V}E \) was reduced proportionally to the decrease in \( \dot{V}O_2 \) because \( \dot{V}E/\dot{V}O_2 \) was not affected. Lack of influence of BR deconditioning on the maximal \( \dot{V}E \) was reported previously (8, 9, 31).

It seems likely that inadequate adjustment of the peripheral circulation to exercise or an impairment of muscle aerobic capacity contributes to the limitation of working capacity after short-term BR. This hypothesis is supported by the enhanced blood \( [LA] \) during submaximal exercise and lowered blood \( [LA] \) threshold after BR in the endurance athletes. This is in agreement with previous data (9, 25) that showed greater increases in blood \([LA]\) at submaximal loads after BR lasting 5–10 days. Moreover, Convertino et al. (9) reported that the anaerobic threshold, detected on the basis of pulmonary ventilation during graded exercise, is shifted toward lower exercise intensity after 10 days of BR. Sullivan et al. (34) also found a decrease of anaerobic threshold after 3 wk of BR unless the subjects were treated with dobutamine.

Greater \( [LA] \) production and shifting of its threshold may also result from an increased contribution of carbohydrates to the energy-yielding processes, as occurs after a high-carbohydrate diet (36). After BR, both at rest and during submaximal exercise, the RER was elevated in both groups of athletes. However, despite similar elevation of RER after BR in endurance- and strength-trained subjects, only in the former was the blood \([LA]\) threshold markedly lowered. An increased RER after prolonged BR was reported previously (3, 8, 30), but the mechanism of this effect is still unclear. The diet during BR in the present study did not contain excessive amounts of carbohydrates compared with the subjects’ habitual diet, but avoidance of exercise for 2 days preceding and during the next 3 days of BR could result in muscle glycogen accumulation and/or reduction in activities of muscle enzymes involved in fatty acid oxidation.
BR did not affect the preexercise plasma [Epi] and [NE] in any group, indicating that the previously reported inhibition of basal sympathetic activity after deconditioning (32, 33) is blunted in subjects sitting on a cycle ergometer anticipating exercise. The maximal postexercise plasma catecholamine concentrations were similar before and after 3 days of BR, contrary to results of Engelke and Convertino (14), who found plasma [NE] at volitional exhaustion higher by 64% after BR, but the duration of their BR was longer (16 days) than in the present investigation. In agreement with previous reports (6, 29), our data demonstrated an exponential pattern of changes in the plasma catecholamine concentrations during progressive graded exercise with thresholds similar to that of [LA]. In the sedentary and strength-trained subjects, the plasma catecholamine concentrations at submaximal loads and the catecholamine thresholds were similar before and after BR; however, in the endurance athletes the plasma [NE] at submaximal loads tended to be higher after BR and the NE threshold was significantly lower. Thus the deconditioning effect of 3 day-BR on the SNS response to exercise became evident only in the endurance-trained subjects.

It seems likely that the shift of the [NE] threshold toward lower exercise loads, reflecting earlier activation of the SNS, could explain the lower perceived exertion and higher heart rate observed in endurance-trained subjects after BR (14). In the present investigation, the changes in plasma NE concentrations after 3 days of BR were larger in endurance-trained than in sedentary subjects, suggesting that the deconditioning effect on catecholamine responses to exercise is more evident in endurance-trained subjects. The differences in the NE threshold between groups were no longer significant 2 weeks after BR (34), indicating that the deconditioning effect of short-term BR was reversible in endurance-trained subjects.

Table 3. Plasma renin activity, and human growth hormone, cortisol, and testosterone concentrations at rest and after exercise before and after 3-day bed rest

<table>
<thead>
<tr>
<th>Group</th>
<th>PRA, ng·ml⁻¹·h⁻¹</th>
<th>hGH, nmol/l</th>
<th>Cortisol, nmol/l</th>
<th>Testosterone, nmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Exercise</td>
<td>Rest</td>
<td>Exercise</td>
</tr>
<tr>
<td>Untrained subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>3.3 ± 0.6</td>
<td>4.7 ± 0.9</td>
<td>3.8 ± 1.9</td>
<td>13.1 ± 5.8</td>
</tr>
<tr>
<td>After</td>
<td>7.4 ± 0.9†</td>
<td>9.8 ± 1.6†</td>
<td>5.5 ± 3.5</td>
<td>11.6 ± 7.4</td>
</tr>
<tr>
<td>Endurance athletes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>2.2 ± 0.2</td>
<td>7.4 ± 1.1</td>
<td>3.1 ± 0.8</td>
<td>53.4 ± 9.1</td>
</tr>
<tr>
<td>After</td>
<td>3.8 ± 0.4*</td>
<td>12.1 ± 2.0†</td>
<td>3.1 ± 2.1</td>
<td>27.0 ± 8.4†</td>
</tr>
<tr>
<td>Strength athletes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>2.5 ± 0.3</td>
<td>6.3 ± 0.9</td>
<td>12.1 ± 6.5</td>
<td>49.3 ± 15.8</td>
</tr>
<tr>
<td>After</td>
<td>4.1 ± 0.6†</td>
<td>9.9 ± 1.5†</td>
<td>5.3 ± 3.2</td>
<td>26.3 ± 11.1†</td>
</tr>
</tbody>
</table>

Values are means ± SE. PRA, plasma renin activity; hGH, human growth hormone. *Significantly different from values before bed rest, $P < 0.05$. †Significantly different from values before bed rest, $P < 0.01$. ‡Significantly different from values before bed rest, $P < 0.001$. 

Fig. 6. Plasma epinephrine concentrations during graded exercise and epinephrine thresholds before (○ and open bars) and after (● and solid bars) bed rest in 3 groups of subjects. Values are means ± SE. The last points (connected with dashed lines) represent mean values after the maximal exercise load.
tion of the SNS, contributed to the increased lactate production because the BR-induced changes in the [NE] and [LA] thresholds were significantly correlated. There is an apparent discrepancy between this observation and the findings of Sullivan et al. (34), who reported that dobutamine administration during BR prevented the fall in exercise performance and lowering of the anaerobic threshold. However, application of daily infusions of dobutamine throughout 3 wk of BR probably simulated effects of aerobic exercise training on the cardiovascular system and on oxidative enzymes in skeletal muscle cells.

The decrease in hGH response to exercise after BR is consistent with data reported by McCall et al. (27); they used isometric exercise performed with a small group of muscles, which, before BR, induced an increase in the bioassayable, but not in immunoassayable, form of hGH. The exercise-induced increases in hGH were much greater in both groups of our athletes than in the sedentary subjects. However, in the group of strength-trained athletes there were three subjects with very high levels of hGH both before and after BR. If these subjects were excluded from the calculations, the mean exercise induced increments in this group would have been lower than in the endurance athletes but still higher than in the sedentary subjects. The decrease in the hormone responses to exercise after BR occurred in both athletic groups, but it was more pronounced in the endurance-trained subjects. These data suggest that the magnitude of the hGH response to maximal cycle exercise depends on the absolute exercise intensity, and the BR-induced reduction of this response may be the result of earlier exhaustion. However, disruption of the reflex mechanism activating hGH release, as suggested by McCall et al. (27), cannot be excluded.

Although activation of the renin-angiotensin system after BR is well documented (19), there are no data on the effect of BR deconditioning on the PRA response to exercise. The present results indicated that, apart from elevation of resting and postexercise PRA values in all groups of subjects, BR caused an enhanced PRA response to exercise in the athletes despite their reduced maximal exercise load. β-Adrenergic stimulation of the juxtaglomerular apparatus of the kidney is probably the main mechanism responsible for an increase in renin release during exercise (23). Thus it appears that the increased PRA response to exercise after BR can be attributed to the β-adrenergic-receptor sensitization, as was suggested previously from experiments with β-adrenergic-agonist infusion at rest (1, 28). Earlier activation of the SNS, as indicated by lowering of the [NE] threshold after BR in the endurance athletes, may also have facilitated their greater PRA response to exercise.

In none of the subjects did BR modify resting or postexercise plasma testosterone concentration, in confirmation of results of McCall et al. (27). In contrast, however, our postexercise cortisol levels were significantly elevated in the endurance athletes despite lower workloads after BR.

In summary, these data show that work tolerance, aerobic capacity, and the anaerobic threshold are diminished in endurance-trained men after only 3 days of BR, whereas less pronounced changes occur in sedentary subjects and strength-trained athletes. Three-day BR increased resting PRA in all three groups of subjects. In endurance athletes it modified neuroendocrine responses to graded exercise by an earlier increase in plasma [NE], attenuation of the increase in plasma [hGH], and accentuated elevation of PRA and cortisol. The effect of 3-day BR on the hormonal responses to exercise was negligible in strength-trained athletes and sedentary subjects. It appeared that the initial aerobic capacity determines the magnitude of these exercise effects. Further studies are needed to elucidate whether they depend on the level of activity specific for endurance training preceding BR or the genetic factors associated with aerobic capacity.

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