

# Recovery Pattern of Baroreflex Sensitivity after Exercise

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## ABSTRACT

NIEMELÄ, T. H., A. M. KIVINIEMI, A. J. HAUTALA, J. A. SALMI, V. LINNAMO, and M. P. TULPPO. Recovery Pattern of Baroreflex Sensitivity after Exercise. *Med. Sci. Sports Exerc.*, Vol. 40, No. 5, pp. 864–870, 2008. **Purpose:** To test the association between exercise mode and the recovery pattern of baroreflex sensitivity (BRS) after exercise. **Methods:** The study population included healthy male subjects ( $N = 12$ , age:  $31 \pm 3$  yr). Four different interventions were performed in a randomized order: 1) aerobic exercise session on a bicycle ergometer, 2) light resistance exercise session, 3) heavy resistance exercise session, and 4) control intervention with no exercise. All interventions lasted 40 min.  $R-R$  intervals and continuous blood pressure were measured before (10 min) and 30–180 min after the interventions.  $BRS_{LF}$  was calculated by the transfer function method from the low-frequency band (LF, 0.04–0.15 Hz) of the  $R-R$  intervals and systolic blood pressure spectra. **Results:**  $BRS_{LF}$  had blunted until 30 min after aerobic and light resistance exercise ( $11.1 \pm 4.3$  and  $10.0 \pm 3.6$  vs  $17.5 \pm 7.0$   $ms \cdot mm \text{Hg}^{-1}$ ,  $P = 0.002$  for both, compared with the control intervention, respectively). However,  $BRS_{LF}$  was significantly blunted until 60 min after heavy resistance exercise ( $9.3 \pm 2.3$  vs  $15.1 \pm 4.7$   $ms \cdot mm \text{Hg}^{-1}$ ,  $P = 0.005$ , compared with the control intervention). The high-frequency power of  $R-R$  intervals (0.15–0.4 Hz) was significantly reduced, and the LF power of systolic blood pressure oscillation was significantly augmented 30 min after heavy resistance exercise ( $P < 0.01$  for both), whereas both indices were restored to the control level by 30 min after aerobic and light resistance exercise. **Conclusion:** BRS after acute exercise is associated with exercise intensity, showing relatively rapid recovery after aerobic and light resistance exercise and delayed recovery after heavy resistance exercise. The delayed BRS pattern after heavy resistance exercise is regulated by delicate interplay between the withdrawal of vagal outflow and the probably increased sympathetic vasomotor tone documented by measurements of heart rate and blood pressure variability. **Key Words:** AUTONOMIC NERVOUS SYSTEM, HEART RATE VARIABILITY, BLOOD PRESSURE VARIABILITY, RESISTANCE EXERCISE

Heart rate (HR) recovery after physical exercise is a powerful independent predictor of mortality in healthy subjects and in different patient populations (3,21). A more detailed analysis of cardiovascular signals including analysis of HR and blood pressure variability provides a deeper insight into cardiovascular regulation in different physiological settings. High-frequency (HF, 0.15–0.4 Hz) oscillation of  $R-R$  intervals is a widely used index of vagal activity (38), and low-frequency (LF, 0.04–0.15) oscillation of  $R-R$  intervals and blood pressure may reveal the activity of sympathetic outflow (13). Furthermore, baroreflex action can be measured using the transfer function technique between  $R-R$  interval and systolic blood pressure oscillation spectra (2). The function of baroreflex is generally characterized by a dynamic gain,

namely, baroreflex sensitivity (BRS) (32). BRS has been extensively studied over the last 20 yr, and its prognostic value in several cardiovascular diseases has been widely accepted (16).

BRS is markedly reduced during dynamic exercise due to resetting of the baroreceptors, which allows the baroreflex function curve to move to a new operational point (12,31). BRS is reduced after both aerobic and resistance exercise compared with baseline values (10). However, BRS fluctuates widely over time, even without any intervention in healthy human subjects (7). Therefore, the control measurements without exercise should be included in the study protocol to detect better possible changes after specific interventions. Secondly, the long-term recovery pattern of BRS between exercise modes and intensities are not well known at present. Therefore, the purpose of the present study was to examine the recovery pattern of BRS after aerobic exercise, light resistance exercise, and heavy resistance exercise, compared with control measurements.

## METHODS

**Subjects and study protocol.** Twelve healthy, nonsmoking male volunteers were recruited (Table 1). All subjects were free of cardiovascular diseases, and none of

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TABLE 1. Characteristics of the test subjects (mean  $\pm$  SD).

	<i>N</i> = 12
Age (yr)	31 $\pm$ 3
Range (yr)	27–40
Height (cm)	178 $\pm$ 5
Weight (kg)	81 $\pm$ 12
BMI	26 $\pm$ 4
Range (kg·m <sup>-2</sup> )	22–32
Body fat (%)	18 $\pm$ 4
$\dot{V}O_{2max}$ (L·min <sup>-1</sup> )	4.4 $\pm$ 0.3
$\dot{V}O_{2max}$ (mL·kg <sup>-1</sup> ·min <sup>-1</sup> )	54 $\pm$ 7

them were taking any cardiovascular-acting medications. All subjects gave written informed consent. The laboratory measurements were performed in the department of exercise and medical physiology at Verve (Oulu, Finland). The subjects were not allowed to eat for 2 h or to drink coffee for 4 h before the tests. Since the study protocol lasted over 4 h, the subjects were instructed to have small and similar meal 2 h before each intervention. The subjects were asked not to perform physical exercise and avoid alcohol drinking for 48 h before the testing. All subjects first performed a test of peak oxygen consumption followed by four interventions in a randomized order to assess baroreflex sensitivity recovery: 1) an aerobic exercise session on a bicycle ergometer, 2) a light resistance exercise session, 3) a heavy resistance exercise, and 4) a control intervention (no exercise). At least one resting day was required between the interventions. Continuous blood pressure and *R–R* intervals were measured at rest (10 min) and 180 min after

the interventions. During the recovery phase, signals were measured 10–30, 40–60, 70–90, 100–120, 130–150, and 160–180 min after the cessation of the interventions. For the final analysis of autonomic function, the last 10 min of every portion were used for the calculations (Figs. 1–3). The protocol was approved by the ethical committee of the Northern Ostrobothnia Hospital District, Oulu, Finland.

**Maximal O<sub>2</sub> consumption.** The subjects performed a ramp maximal exercise test on a bicycle ergometer (839E Monark, Stockholm, Sweden). The test was started at 25 W, and the work rate was increased by 1 W every 5 s until voluntary exhaustion. Ventilation, gas exchange (M909 Ergospirometer, Medikro, Kuopio, Finland), and heart rate (Cardiolife TEC-7721K, Nihon Kohden, Tokyo, Japan) were monitored continuously during the protocol. The highest value of oxygen uptake measured during the test (1-min collection) was taken as the peak oxygen uptake ( $\dot{V}O_{2peak}$ ). All subjects fulfilled the criteria for  $\dot{V}O_{2peak}$  given in the literature (i.e., respiratory exchange ratio >1.1 or maximal HR within  $\pm$  10 beats of the age-appropriate reference value) (5). In addition, 1RM and personal adjustments to machines (leg press, chest curl, leg extension and biceps) were measured at least 2 d before first intervention (HUR machines, HUR Oy, Kokkola, Finland).

**Aerobic exercise session on a bicycle ergometer.** The exercise session on a bicycle ergometer lasted for 40 min. Work rate was 50% of the maximal load. Pedaling rate was 70 rpm. Blood pressure and RPE (Borg rating of

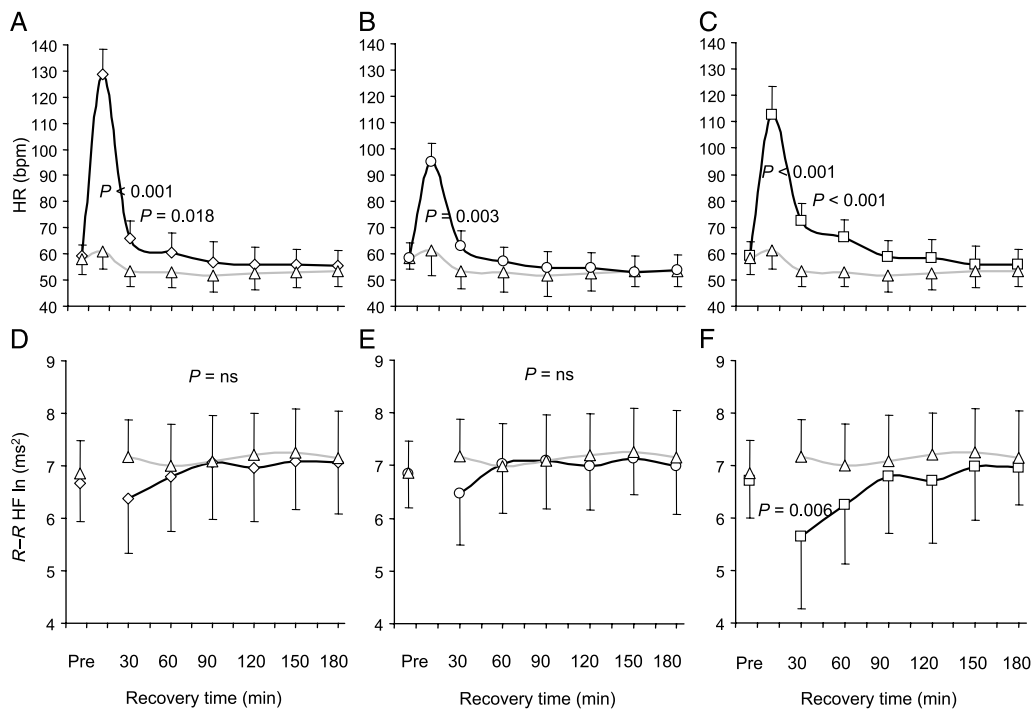
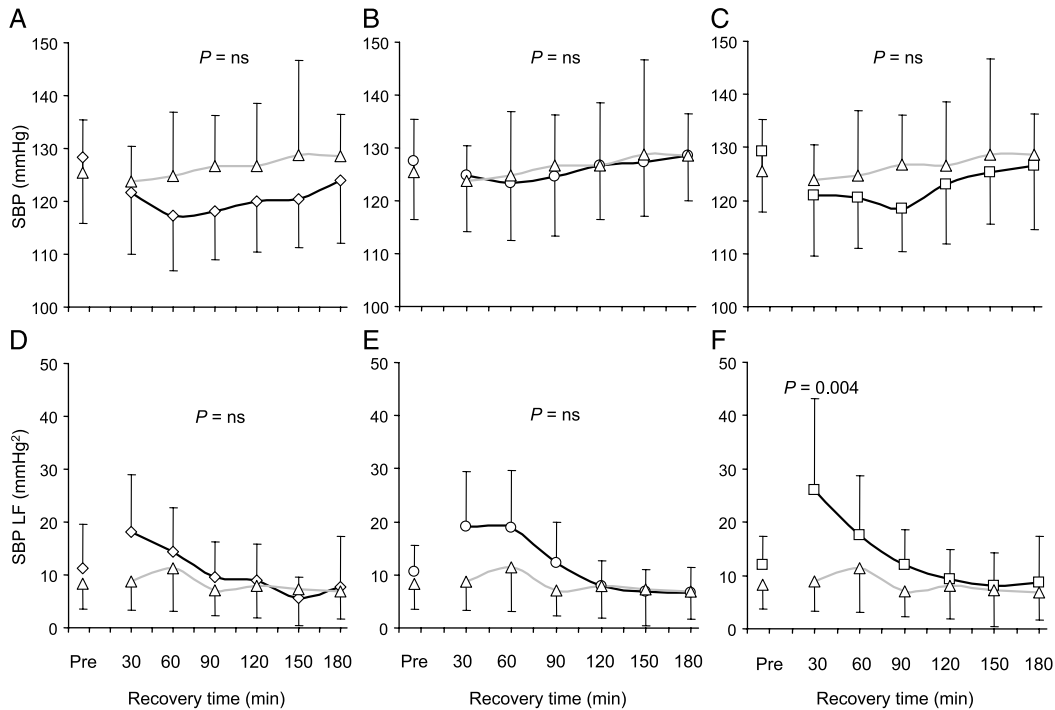


FIGURE 1—Heart rate and heart rate variability indices after aerobic, light resistance, and heavy resistance exercise sessions compared with control measurements (gray line with triangles). A, Heart rate after aerobic exercise; B, heart rate after light resistance exercise; C, heart rate after heavy resistance exercise; D, high-frequency (HF, 0.15–0.4 Hz) spectral power of *R–R* intervals after aerobic exercise; E, HF power of *R–R* intervals after light resistance exercise; F, HF power of *R–R* intervals after heavy resistance exercise.



**FIGURE 2**—Systolic blood pressure (SBP) and SBP variability indices after aerobic, light resistance, and heavy resistance exercise compared with control measurements (gray line with triangles). *A*, SBP after aerobic exercise; *B*, SBP after light resistance exercise; *C*, SBP after heavy resistance exercise; *D*, low-frequency (LF, 0.04–0.15 Hz) spectral power of SBP values after aerobic exercise; *E*, LF power of SBP values after light resistance exercise; *F*, LF power of SBP values after heavy resistance exercise.

perceived exertion scale) were measured after every 10 min of exercise. Subjects drank 20 cL of water immediately after the exercise. The test was performed in standard laboratory conditions. None of the subjects had any difficulties to perform the required exercise.

#### Light and heavy resistance exercise session.

Light and heavy resistance exercise sessions were performed at a gym next to the laboratory. Sessions lasted for 40 min. After 5 min of light warm-up on a bicycle ergometer (100 W), the subjects performed three sets of contractions on four different machines (leg press, chest curl, leg extension, and biceps). Sets of contractions started at every 2 min. At the heavy resistance exercise, subjects were instructed to perform 12 contractions at 80% 1RM. If the subject was not able to perform 12 contractions, resistance was lowered from 80% enough to achieve 12 contractions and maximal effort. When performing light resistance exercise subjects were asked to make 20 contractions at 30% 1RM. Blood pressure and RPE were measured after each set of the contractions. The exercise sessions was followed by 5 min of light cycling (100 W) on a bicycle ergometer to start recovering. Finally, the subjects were allowed to drink 20 cL of water. The intensity of heavy (80% 1RM) and light (30% 1RM) exercise are based on lower and upper limits of ACSM recommendations to improve muscle strength in healthy subjects (15).

**Control intervention.** The control intervention was performed to measure the individual responses to the protocol. The subjects sat in a laboratory environment for

40 min, reading newspapers, followed by a similar measuring protocol as after the exercise interventions (from 30 to 180 min).

**Assessment of BRS during recovery.** The subjects sat in a supine position in a quiet room for 10 min as a baseline measurement. The baseline measurement was followed by an intervention for 40 min. BRS recovery was then measured for 30–180 min after the intervention, with the subject sitting in a quiet room. The subjects breathed spontaneously. No moving or speaking was allowed during the measurements. The interventions were started at the same time of day for each subject. Six subjects performed their exercises in the morning and the other six in the afternoon.

**Measurements.** ECG was recorded by standard methods (Nihon Kohden TEC-7700). Blood pressure was recorded noninvasively on a beat-by-beat basis by Finapres (Ohmeda; Louisville, CO). The blood pressure measurements by Finapres were used only for systolic blood pressure fluctuation and BRS analysis since the absolute blood pressure levels measured by Finapres may not be correct during a long-term measurements. Therefore, blood pressure was also measured with an automatic blood pressure recorder at every 2 min at baseline and at every 5 min during the recovery phase (Tango, SunTech, Raleigh, NC). If the finapres values differed more than 15 mm Hg from brachial values, the servo mechanism was used before starting measurements. The average of five measurements (Tango) was used as baseline blood pressure values, and

the average of two measurements were used at the different recovery phases.

**Signal processing.** The signals were recorded and digitized ( $f_s = 1000$  Hz) with the Power Lab (AD Instruments) recording system.  $R$ -peaks were detected automatically from the ECG based on thresholds for amplitude and the first derivative. The ECG was manually corrected in the case of false alarms and missed peaks. After the correction, a tachogram was calculated from the  $R$ - $R$  intervals. A systogram was calculated from the continuous blood pressure signal by detecting the maximum values between the corresponding adjacent  $R$ -peaks. The systogram was inspected visually to correct possible artifacts. After extracting the  $R$ - $R$  intervals and beat-to-beat systolic blood pressure values, the power spectral analysis of HR and systolic blood pressure variability was performed using an autoregressive model (order 15). The power spectrum densities of the LF (0.04–0.15 Hz) and HF (0.15–0.4 Hz) oscillation of  $R$ - $R$  interval and systolic blood pressure oscillation were calculated (35).

**BRS estimation.** BRS was estimated by using LF spectra ( $BRS_{LF}$ , 0.04–0.15 Hz) of the  $R$ - $R$  interval and systolic blood pressure spectra. The transfer function (TF) technique (30) was used to calculate BRS in milliseconds per millimeters of mercury when coherence between the signals was  $> 0.50$  (37). We similarly calculated BRS in the HF and LF band (0.04–0.4 Hz) as BRS mean when coherence was  $> 0.50$ .

**Statistical methods.** Standard statistical methods were used for the calculation of means and standard deviations. Kolmogorov–Smirnov’s goodness-of-fit test was used to verify normal Gaussian distribution of the data ( $z$  value  $> 1.0$ ). HR variability parameters were not normally distributed, and logarithmic transformation to the natural base was therefore performed. The effects of the interventions on the measured variables were studied by two-way ANOVA for repeated measures with time and intervention (main effect for interaction). One-way ANOVA was used to study differences between endurance, light resistance, heavy resistance, and control conditions at different time points followed by Bonferroni’s *post hoc* test. All statistical analyses were performed using SPSS for Windows (version 14.0, Chicago, IL).

## RESULTS

**Heart rate and heart rate variability.** The changes in HR and HR variability from HF band during the interventions are shown in Figure 1. HR was restored back to the control level at 90 min after aerobic exercise (Fig. 1A), at 60 min after light resistance exercise (Fig. 1B), and at 90 min after heavy resistance exercise (Fig. 1C). The vagally mediated HF power of  $R$ - $R$  intervals did not change after aerobic or light resistance exercise (Fig. 1D and E), but it was significantly reduced ( $5.6 \pm 1.3$  vs  $7.2 \pm 0.7$   $\ln$   $\text{ms}^2$ ,  $P = 0.006$  compared with control measurement) at 30 min

after heavy resistance exercise (Fig. 1F). The mean LF power of  $R$ - $R$  intervals did not change in the recovery phase after any interventions compared with the control level ( $7.0 \pm 1.1$  vs  $7.2 \pm 0.6$   $\ln$   $\text{ms}^2$ ) after 30 min of control intervention and heavy resistance exercise intervention, respectively ( $P = \text{ns}$ ). LF/HF ratio was significantly elevated 30 min after heavy resistance exercise compared with control measurements ( $4.5 \pm 2.9$  vs  $1.3 \pm 1.0$ ,  $P = 0.006$ ) but not after aerobic ( $3.1 \pm 2.9$ ) or light resistance exercise ( $2.3 \pm 1.5$ ,  $P = \text{ns}$  for both).

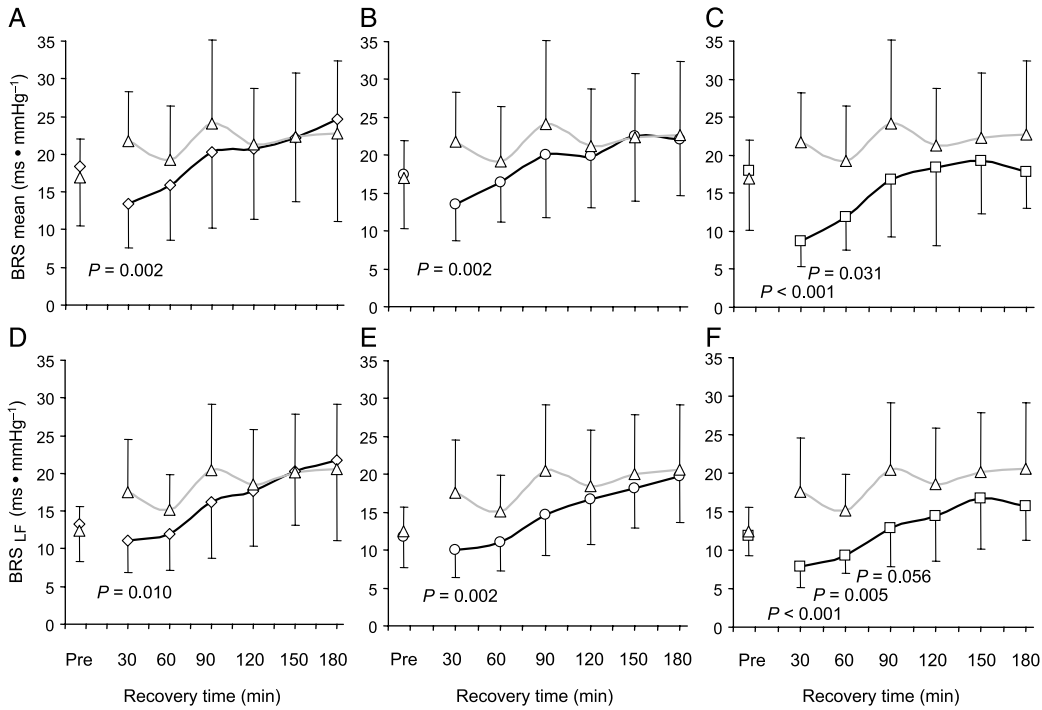
**Blood pressure and blood pressure variability.** Systolic blood pressure did not change after any interventions when compared with control values (Fig. 2A–C). Similarly, the HF power of systolic blood pressure oscillation did not change after any exercise. After aerobic and light resistance exercise, the LF power of systolic blood pressure oscillation did not change compared with the control measurements (Fig. 2D and E). However, the LF oscillation of systolic blood pressure was significantly augmented (Fig. 2F) at 30 min after heavy resistance exercise compared with control measurements ( $26.1 \pm 16.9$  vs  $8.8 \pm 5.5$  mm Hg,  $P = 0.004$ ).

**Baroreflex sensitivity.** After aerobic and light resistance exercise, BRS mean was reduced for 30 min compared with control measurements (Fig. 3A and B). However, BRS mean showed a prolonged reduction of 60 min after heavy resistance exercise ( $11.8 \pm 4.4$  vs  $19.2 \pm 7.2$ ,  $P = 0.031$ , compared with control level, Fig. 3C). Similarly,  $BRS_{LF}$  was reduced for 30 min after aerobic and light resistance exercise (Fig. 3D and E) but for 60 min after heavy resistance exercise ( $9.3 \pm 2.3$  vs  $15.1 \pm 4.7$   $\text{ms} \cdot \text{mm Hg}^{-1}$ ,  $P = 0.005$ , Fig. 3F) when compared with control.

## DISCUSSION

The main finding of the present study is that exercise intensity had an effect on the pattern of autonomic function recovery. First, the vagally mediated HF power of  $R$ - $R$  intervals was significantly reduced and LF/HF ratio augmented after heavy resistance exercise compared with control measurements. Secondly, the LF power of systolic blood pressure oscillation, which reflects sympathetic mediated vasomotor tone, was significantly augmented only after heavy resistance exercise. Thirdly, heavy resistance exercise resulted in long-term reduced BRS values as evidence of altered autonomic regulation after heavy resistance exercise. In contrast, autonomic function was restored to the control level already 30 min after aerobic and light resistance exercise, as documented by HR and BP variability techniques. Together, these findings may provide insight into the long-term recovery pattern of autonomic regulation after aerobic, light-, and heavy-intensity resistance exercise.

**Blood pressure and HR after aerobic and resistance exercise.** Postexercise hypotension after aerobic



**FIGURE 3—Baroreflex sensitivity (BRS) indices after aerobic, light resistance, and heavy resistance exercise compared with control measurements (gray line with triangles). A, Mean BRS values (0.04–0.4 Hz) after aerobic exercise; B, mean BRS after light resistance exercise; C, mean BRS after heavy resistance exercise; D, BRS from low-frequency spectral band (BRS<sub>LF</sub>, 0.04–0.15 Hz) after aerobic exercise; E, BRS<sub>LF</sub> after light resistance exercise; F, BRS<sub>LF</sub> after heavy resistance exercise.**

exercise is a common phenomenon in hypertensive subjects. Postexercise reduction of catecholamines and peripheral resistance in association with vasodilator and vasoconstrictor factors have been suggested to explain postexercise hypotension (17,24). However, the prevalence of postexercise hypotension in normotensive subjects is under debate. Blood pressure assessments after resistance exercise have shown unchanged or increased values compared with preexercise values (8,29). In the present study, postexercise blood pressure after three different exercise interventions did not differ from the control values in normotensive subjects.

Previous studies have reported that HR remains elevated for 60 min after moderate-intensity aerobic exercise (20 min of exercise at 80% of the individual's anaerobic threshold). Comparing the effects of exercise mode, HR recovery has been shown to be slower after resistance exercise than after aerobic exercise (11). In the present study, HR was elevated at 60 min after aerobic and heavy resistance exercise and at 30 min after light resistance exercise compared with the control level. In other words, only HR itself would inform similar recovery time after aerobic and heavy resistance exercise. However, the more detailed analysis of autonomic regulation by HR and blood pressure variability techniques revealed clear differences among exercise types.

**Autonomic regulation after exercise.** HF power of heart rate variability (HF<sub>R-R</sub>) has been accepted as a marker of vagal activity (6). The association between HF

power of R-R intervals and vagal outflow has been documented by pharmacological interventions in human and animal studies (9). Furthermore, during dynamic exercise, the HF power of R-R intervals decreases from rest until 50–60% of the maximal work load, as evidence of the withdrawal of vagal outflow (39,40). Previous studies have shown the HF power of R-R intervals to be blunted until 30 min after moderate-intensity aerobic exercise compared with the baseline level (11,26). In the present study, with the assessment of control measurements, HF power was already back to the control level after 30 min of aerobic and light resistance exercise. The intensity of aerobic exercise was markedly lower in the present study than in the earlier studies, which may well explain the difference in the pattern of HF power recovery. Similarly, the LF power of R-R intervals as well as blood pressure oscillations, analyzed from both spectral bands (LF and HF), returned to the control level 30 min after aerobic and light resistance exercise. Overall, the neural control of the autonomic nervous system, expressed as blood pressure and HR variability, is back to the control level 30 min after low-intensity aerobic exercise and after light resistance exercise. The physiological background for augmented HR until 60 min after aerobic exercise, without any changes in noninvasive markers of autonomic regulation, is not known. It is possible that the HR or blood pressure variability techniques are not sufficiently sensitive methods to detect subtle changes in autonomic control in the present conditions.

There were more evident changes in autonomic regulation after heavy resistance than after aerobic and light resistance exercise. First, vagal outflow was significantly blunted after heavy resistance exercise compared with the control measurement, as documented by the changes in the HF power of  $R$ - $R$  interval fluctuation. Secondly, sympathovagal balance was shifted towards a state of sympathetic predominance after heavy resistance exercise assessed by LF/HF ratio. Thirdly, sympathetic vasomotor tone, expressed as LF oscillation of systolic blood pressure, was augmented after heavy resistance exercise. This is in line with a recent study reporting similar changes 30 min after heavy resistance exercise (11). Based on the changes in LF/HF ratio of  $R$ - $R$  intervals and LF oscillation of blood pressure augmented sympathetic activation was directed to the heart and blood vessels after heavy resistance exercise (17).

**Baroreflex sensitivity after exercise.** It is well documented that, immediately after the termination of aerobic exercise, BRS is markedly reduced (31,34). In the post-exercise period, BRS gradually increases back to the baseline level. Somers et al. (33) reported that BRS is decreased only for 20 min, but some studies have shown the recovery period to be longer (10,34). In earlier studies, BRS has been shown to exceed the preexercise values at 60 min after aerobic exercise (17), but this finding has not been reported by all (34). In our present study,  $BRS_{LF}$  had reduced until 30 min after aerobic and light resistance exercise. Interestingly,  $BRS_{LF}$  values tended to rise above the baseline for 60–180 min after both aerobic and light resistance exercise, but the same trend was seen in the control measurement. In summary, all of the autonomic markers measured in the present study, that is, HR and blood pressure variability, as well as all BRS indicators, revealed relatively rapid and similar recovery after both aerobic and light resistance exercise.

BRS values are reported to be reduced significantly at 30 min after resistance exercise (10) and to be restored back to the preexercise level at 180 min after exercise. However, the time course of the BRS recovery among heavy

and light resistance exercise and aerobic exercise has not been reported before. We recorded a steady and more prolonged  $BRS_{LF}$  reduction after heavy resistance exercise than after other exercise types. The physiological mechanisms for more reduced BRS after heavy resistance exercise than after other interventions are not clear, and several mechanisms may explain these differences. Heffernan et al. showed recently that central arterial stiffness increases after a single bout of heavy resistance exercise, whereas it decreases after aerobic exercise (10). However, BRS decreased after both exercise types, and, therefore, arterial stiffness may not be the major contributor for the more clearly reduced BRS after heavy resistance exercise as opposed to aerobic or light resistance exercise.

**Limitations of the study.** The major limitation of the present study is the lack of a mechanistic explanation for the increased sympathetic activation and more reduced BRS after heavy resistance exercise. Measurements of cardiac output, peripheral resistance, and direct sympathetic nervous activity by microneurography technique would be needed to solve this question. Secondly, the results cannot be extrapolated to exercise training programs, but they should be regarded as pertinent to a single bout of exercise. Thirdly, the relatively low subject number may limit the usefulness of the present study. Finally, we estimated sympathetic activation by a noninvasive method from the LF oscillation of beat-to-beat systolic blood pressure and LF/HF ratio from  $R$ - $R$  interval variability. These methods are still debatable as a marker of sympathetic activation (19).

In summary, the recovery pattern of cardiovascular autonomic regulation after acute exercise is associated with exercise intensity, showing relatively rapid recovery after aerobic and light resistance exercise, but significantly delayed recovery after heavy resistance exercise in healthy males. From the autonomic regulation point of view, light resistance exercise may be as safe as aerobic exercise.

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