Effects of aerobic training on heart rate dynamics in sedentary subjects

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1Merikoski Rehabilitation and Research Center, 90100 Oulu; 2Department of Medicine, Division of Cardiology, University of Oulu, 90220 Oulu; 3Polar Electro, 90440 Kempele, Finland; and 4Department of Kinesiology, University of Waterloo, Waterloo, Ontario, Canada N2L 3G1

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Tulppo, Mikko P., Arto J. Hautala, Timo H. Mäkkialio, Raija T. Laukkanen, Seppo Nissilä, Richard L. Hughson, and Heikki V. Huikuri. Effects of aerobic training on heart rate dynamics in sedentary subjects. J Appl Physiol 95: 364–372, 2003. First published March 21, 2003; 10.1152/japplphysiol.00751.2002.—This study was designed to assess the effects of moderate- and high-volume aerobic training on the time domain and on spectral and fractal heart rate (HR) variability indexes. Sedentary subjects were randomized into groups with moderate-volume training (n = 20), high-volume training (n = 20), and controls (n = 15). The training period was 8 wk, including 6 sessions/wk at an intensity of 70–80% of the maximum HR, lasting for 30 min/session in the moderate-volume group and 60 min/session in the high-volume group. Time domain, frequency domain, and short-term fractal scaling measures of HR variability were analyzed over a 24-h period. Mean HR decreased from 70 ± 7 to 64 ± 8 beats/min and from 67 ± 5 to 60 ± 6 beats/min (P < 0.001 for both) for the moderate- and high-volume training groups, respectively. The normalized high-frequency spectral component increased in both groups (P < 0.05). The normalized low-frequency component decreased from 70 to 60 (P = 0.001 for both). There were no significant differences in the changes of HR variability indexes between groups. Aerobic training in sedentary subjects results in altered autonomic regulation of HR toward vagal dominance. A moderate training volume is a sufficient intervention to induce these beneficial effects.

Methods

Subjects. Subjects were recruited by advertising in a newspaper, which attracted 85 replies. Subjects were interviewed with a standardized scheme to ascertain their medical histories and levels of physical activity. All smokers, subjects with high body mass index (>30), subjects who did regular physical training more than twice a week, and those with diabetes mellitus, asthma, or cardiovascular disorders were excluded. We invited 60 male subjects to our laboratory (Merikoski, Oulu, Finland) for a more specific assessment of physical status and excluded two subjects on account of various relative contraindications for a maximal exercise test. We tested 58 subjects and excluded 3 from the final analysis because of the number of ectopic beats during data acquisition. Finally, 55 male subjects were included in the training study (Table 1). Subjects were randomized into a moderate-volume training group (n = 20), a high-volume training group (n = 20), and a control group (n = 15). The protocol was approved by the ethics committee of the Merikoski Rehabilitation and

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Table 1. Effects of training on diastolic and systolic blood pressure at rest, \( V_{O2peak} \), \( HR_{max} \), and \( Pacemax \)

<table>
<thead>
<tr>
<th></th>
<th>Moderate Training (n = 19)</th>
<th></th>
<th></th>
<th>High Training (n = 16)</th>
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<th></th>
<th>Control (n = 11)</th>
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<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
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<td>Pre</td>
<td>Post</td>
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<tr>
<td>Age, yr</td>
<td>35 ± 10</td>
<td>35 ± 10</td>
<td>36 ± 11</td>
<td>36 ± 11</td>
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<tr>
<td>Min–max, yr</td>
<td>23–52</td>
<td>24–50</td>
<td>25–52</td>
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<td>Height, m</td>
<td>1.81 ± 0.05</td>
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<td>1.82 ± 0.07</td>
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<tr>
<td>Weight, kg</td>
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<td>81 ± 11</td>
<td>79 ± 9</td>
<td>81 ± 9</td>
<td>79 ± 9</td>
<td>81 ± 9</td>
<td></td>
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<tr>
<td>BMI</td>
<td>25 ± 3</td>
<td>25 ± 3</td>
<td>24 ± 2</td>
<td>24 ± 2</td>
<td>25 ± 3</td>
<td>25 ± 3</td>
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<tr>
<td>Fat, %</td>
<td>16 ± 4</td>
<td>16 ± 3</td>
<td>17 ± 4</td>
<td>16 ± 4</td>
<td>16 ± 3</td>
<td>16 ± 4</td>
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</tr>
<tr>
<td>BP_{min}, mmHg</td>
<td>78 ± 7</td>
<td>79 ± 6</td>
<td>77 ± 8</td>
<td>79 ± 5*</td>
<td>79 ± 7</td>
<td>78 ± 4</td>
<td></td>
</tr>
<tr>
<td>BP_{max}, mmHg</td>
<td>127 ± 13</td>
<td>124 ± 9</td>
<td>126 ± 9</td>
<td>128 ± 11</td>
<td>128 ± 10</td>
<td>125 ± 8</td>
<td></td>
</tr>
<tr>
<td>( V_{O2peak} )</td>
<td>( l/min ) 3.3 ± 0.4‡</td>
<td>( 3.6 ± 0.4\‡ )</td>
<td>3.4 ± 0.4‡</td>
<td>3.7 ± 0.5‡</td>
<td>3.4 ± 0.3‡</td>
<td>3.4 ± 0.4‡</td>
<td></td>
</tr>
<tr>
<td>( ml·kg^{-1}·min^{-1} )</td>
<td>41 ± 4</td>
<td>45 ± 4</td>
<td>42 ± 5</td>
<td>46 ± 6\‡</td>
<td>41 ± 4</td>
<td>41 ± 4</td>
<td></td>
</tr>
<tr>
<td>( HR_{max} ), beats/min</td>
<td>189 ± 9</td>
<td>184 ± 8†</td>
<td>187 ± 8</td>
<td>180 ± 10†</td>
<td>185 ± 15</td>
<td>184 ± 17</td>
<td></td>
</tr>
<tr>
<td>( Pacemax ), km/h</td>
<td>11.5 ± 1.3‡</td>
<td>12.9 ± 1.1‡</td>
<td>12.3 ± 1.4</td>
<td>13.6 ± 1.3‡</td>
<td>11.6 ± 0.9</td>
<td>11.6 ± 0.8</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD; \( V_{O2peak} \), peak \( O_2 \) consumption; \( HR_{max} \), maximal heart rate; \( Pacemax \), maximal pace in treadmill; min–max, minimum to maximum; BMI, body mass index; BP_{min}, diastolic blood pressure; BP_{max}, systolic blood pressure. * \( P < 0.05 \); † \( P < 0.01 \); ‡ \( P < 0.001 \): comparisons are between pretraining (Pre) and posttraining (Post) values.

Research Center, and all subjects gave their written, informed consent.

**Protocol.** Subjects were not allowed to eat or drink coffee for 2 h before the exercise test, and all physical exercise and alcohol were forbidden for 48 h before the day of testing. A 24-h ambulatory R-R interval recording was started 25 h before the exercise test. Before the exercise test, subjects remained in a supine position in a quiet room for 10 min, after which blood pressure was measured in a supine position by manual auscultation. Fat percentage was calculated from four skinfolds (sites: biceps, triceps, subscapular, and sub- scapuliac) according to the equation described by Durnin and Womersley (11). All tests were performed at the same time of day before and after the training period.

**Measurement of peak \( O_2 \) consumption.** Subjects performed a graded maximal exercise test on a treadmill (Telineyhtymä, Kotka, Finland), starting at 4.5 km/h followed by a work rate increase at a rate of 0.5 km/h every 2 min until voluntary exhaustion. Minute ventilation and gas exchange (M909 ergospirometer, Medikro, Kuopio, Finland) were monitored continuously during the ramp protocol. Minute ventilation and gas exchange were calculated on a beat-by-beat basis and reported as mean values for 1 min. The highest value of \( O_2 \) consumption measured during the test was used as the peak \( O_2 \) consumption.

**Measurement of R-R intervals.** The R-R intervals were recorded over 24 h with Polar R-R Recorder (Polar Electro, Kempele, Finland) at an accuracy of 1 ms (41) and saved in a computer for further analysis of HR variability with Heart software (Heart Signal, Kempele, Finland). All R-R intervals were edited by visual inspection based on electrocardiogram (ECG) portions, to exclude all the undesirable beats, which accounted for <2% in every subject. The measures of R-R interval dynamics were calculated from the entire 24-h recording and also separately for the hours representing nighttime and daytime (9 AM to 6 PM) hours to detect possible diurnal differences (21, 40). Subjects were asked to go to bed before midnight and to stay in bed until 6 AM on the R-R interval recording days. The R-R intervals were recorded during a nonexercise day before and after training intervention. At the end of training intervention, the R-R intervals were recorded after a 48-h nonexercise period.

**Time and frequency domain analysis of HR variability.** The mean length of R-R intervals and the standard deviation of all R-R intervals were used as time-domain measures of HR variability. An autoregressive model (order 20) was used to estimate the power spectrum densities of R-R interval variability. Ultralow-frequency power (<0.0033 Hz) and very low-frequency (LF) power (0.0033–0.04 Hz) were calculated from the entire 24-h segment. LF and high-frequency (HF) power values were calculated from the segments of 512 R-R intervals over 24-h recording (48). LF and HF were also calculated from 1-h segments of the 24-h recording (by using segments of 512 R-R intervals), and the mean values of these segments were used to detect day and night HR variability values (40). The spectral values are expressed as absolute values (ln; \( ms^2/Hz \)) and in normalized units (nu), which were obtained by dividing the power of each component by total variance from which the very LF component had been subtracted and multiplying this value by 100 (34). The CCV% were calculated as in the following equation: \( CCV\% = 100 \cdot (power \ of \ component)^{1/2}/(mean \ R-R \ interval) \) (16). The frequency at the HF power peak was analyzed for every hour (from the segments of 512 R-R intervals), and the mean values were calculated separately for day and night to evaluate the effects of training on average breathing frequency.

**Fractal analysis of R-R intervals.** DFA quantifies the fractal correlation properties of the R-R interval data (38). The root-mean-square fluctuations of the integrated and detrended data are measured in observation windows of different sizes and then plotted against the size of the window on a log-log scale (Fig. 1). The scaling exponent \( \alpha \) represents the slope of this line, which relates (log)/fluctuation to (log)window size. In this study, the short-term (from 4 to 11 beats) scaling exponent \( \alpha_1 \) was used on the basis of previous experiments (32). The scaling exponent was calculated from 1-h segments of the 24-h recording, and the mean value of these segments was used (40). The power-law relationship of R-R interval variability (\( \beta \)) was also calculated from the frequency range of \( 10^{-4} \) to \( 10^{-2} \) by a previously described method to assess the longer-term correlation of HR (4).

**Training program.** The training period was 8 wk, including six 30-min sessions a week for the moderate-volume training group and six 60-min sessions a week for the high-volume training group at an intensity of 70–80% of maximum HR. The training mode was walking and jogging. All subjects wore a telemetric HR monitor (Polar Smart Edge, Polar Electro) during all training sessions to reach and stay at the correct training intensity. Subjects also recorded the
duration and average training HR of each session. An extra resting day once a week was allowed if they felt exhausted.

**Statistical methods.** Results are expressed as means ± SD. The normal Gaussian distribution of the data was verified by the Kolmogorov-Smirnov goodness-of-fit test. The spectral values of 24-h HR variability were skewed. Therefore, these data were transformed by taking the natural logarithms of the absolute values. The differences within the groups after training were analyzed by a two-factor ANOVA with time and interventions followed by post hoc analysis (Student’s paired t-test). Pearson’s correlation analysis was performed on changes in the different HR variability parameters from baseline to the end of intervention. A P value of <0.05 was considered significant.

**RESULTS**

**Training.** One subject in the moderate-volume training group and four subjects in the high-volume group dropped out from the training because of a lack of motivation or leg problems. The final analysis included 19 subjects in the moderate-volume, 16 subjects in the high-volume, and 12 subjects in the control group. The average amount of training was 5.6 ± 0.4 and 5.7 ± 0.3 sessions/wk at an intensity of 76 ± 2 and 75 ± 1% of maximal HR for the moderate- and high-volume groups, respectively (P = not significant for both). The duration of training was 32 ± 3 and 61 ± 4 min/session for the moderate- and high-volume groups, respectively (P < 0.001). Eight weeks of both moderate- and high-volume training caused an increase in peak O2 consumption as well as a reduction in maximal HR. The diastolic blood pressure changed significantly in the high-volume training group (Table 1). None of the measured variables changed within the control group during the study.

**Time and frequency domain analysis over a 24-h period.** Representative examples of the effects of training on 24-h spectral HR variability are shown in Fig. 1B, and all mean values are shown in Tables 2 and 3. Mean HR decreased significantly for the moderate- and high-volume groups (P < 0.001; Fig. 2A). The mean value of the standard deviation of all R-R intervals increased for both training groups (P < 0.001). The absolute spectral values of HF, LF, and very LF power showed significant (P < 0.01) and similar increases for both training groups after the interventions. The mean value of ultralow-frequency power did not change after the training.

HF power normalized by the average R-R interval (CCV%) increased in both training groups after the interventions (P < 0.01; Fig. 2B), but LF power (CCV%) only increased significantly in the moderate-volume training group (P < 0.05). The LF/HF ratio decreased in both training groups (P < 0.05; Fig. 2C). The mean value of normalized HF (HFnu) power increased in both training groups (P < 0.05). Similarly, the normalized HF (LFnu) power decreased significantly (P < 0.05).

**Scaling analysis of R-R intervals over a 24-h period.** A representative example of the effects of training on 24-h short-term fractal HR behavior is shown in Fig. 1C, and all the mean values are shown in Table 3. The mean value of the short-term scaling exponent (α1)
Table 2. Effects of training on time and frequency domain HR variability measures over 24 h

<table>
<thead>
<tr>
<th></th>
<th>Moderate Training (n = 19)</th>
<th>High Training (n = 16)</th>
<th>Control (n = 11)</th>
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<tbody>
<tr>
<td>HR, beats/min</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>24 h</td>
<td>70 ± 7</td>
<td>64 ± 8‡</td>
<td>67 ± 5</td>
</tr>
<tr>
<td>Midnight–6 AM</td>
<td>55 ± 7</td>
<td>52 ± 6§</td>
<td>54 ± 4</td>
</tr>
<tr>
<td>9 AM–6 PM</td>
<td>78 ± 8</td>
<td>71 ± 8§</td>
<td>72 ± 7</td>
</tr>
<tr>
<td>SDNN, ms</td>
<td></td>
<td></td>
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<tr>
<td>24 h</td>
<td>183 ± 44</td>
<td>202 ± 46†</td>
<td>194 ± 38</td>
</tr>
<tr>
<td>HF, ln ms²</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>24 h</td>
<td>6.19 ± 1.02</td>
<td>6.76 ± 0.96§</td>
<td>6.61 ± 1.01</td>
</tr>
<tr>
<td>Midnight–6 AM</td>
<td>6.68 ± 1.20</td>
<td>7.21 ± 0.97‡</td>
<td>6.96 ± 1.22</td>
</tr>
<tr>
<td>9 AM–6 PM</td>
<td>5.77 ± 0.95</td>
<td>6.35 ± 0.93§</td>
<td>6.18 ± 1.01</td>
</tr>
<tr>
<td>LF, ln ms²</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>24 h</td>
<td>7.42 ± 0.56</td>
<td>7.75 ± 0.59§</td>
<td>7.62 ± 0.57</td>
</tr>
<tr>
<td>Midnight–6 AM</td>
<td>7.61 ± 0.67</td>
<td>7.93 ± 0.67†</td>
<td>7.78 ± 0.79</td>
</tr>
<tr>
<td>9 AM–6 PM</td>
<td>7.30 ± 0.52</td>
<td>7.60 ± 0.51‡</td>
<td>7.48 ± 0.61</td>
</tr>
<tr>
<td>VLF, ln ms²</td>
<td></td>
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<tr>
<td>24 h</td>
<td>7.79 ± 0.59</td>
<td>8.14 ± 0.53§</td>
<td>7.92 ± 0.55</td>
</tr>
<tr>
<td>ULF, ln ms²</td>
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<tr>
<td>24 h</td>
<td>10.0 ± 0.69</td>
<td>10.1 ± 0.55</td>
<td>10.0 ± 0.33</td>
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<tr>
<td>LF/HF ratio</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>24 h</td>
<td>4.1 ± 2.4</td>
<td>3.1 ± 1.5†</td>
<td>3.1 ± 1.6</td>
</tr>
<tr>
<td>Midnight–6 AM</td>
<td>3.3 ± 2.1</td>
<td>2.5 ± 1.2*</td>
<td>2.5 ± 1.3</td>
</tr>
<tr>
<td>9 AM–6 PM</td>
<td>5.5 ± 3.0</td>
<td>4.1 ± 2.3*</td>
<td>4.2 ± 2.0</td>
</tr>
</tbody>
</table>

Values are means ± SD. HR, heart rate; SDNN, standard deviation of R-wave-R-wave intervals; HF, high frequency; LF, low frequency; VLF, very LF; ULF, ultralow frequency. *P < 0.05; †P < 0.01; ‡P < 0.001: comparisons are between Pre and Post values.

decreased for both training groups (P < 0.001; Fig. 2D). The mean value of β, the power law scaling exponent, did not change during the intervention.

Effects of training on the HF spectral peak. The frequency at the HF spectral peak did not change after the intervention (moderate-volume group: from 0.25 ± 0.02 to 0.25 ± 0.02 Hz at night and from 0.32 ± 0.03 to 0.31 ± 0.03 Hz during daytime; high-volume group: from 0.23 ± 0.02 to 0.23 ± 0.03 Hz at night and from 0.30 ± 0.03 to 0.30 ± 0.03 Hz during daytime; P = not significant).

Correlation between the α₁ and normalized spectral components. The change in the scaling exponent α₁ from the baseline to the end of intervention correlate

Table 3. Effects of training on normalized frequency domain HR variability measures and scaling exponents over 24 h

<table>
<thead>
<tr>
<th></th>
<th>Moderate Training (n = 19)</th>
<th>High Training (n = 16)</th>
<th>Control (n = 11)</th>
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</thead>
<tbody>
<tr>
<td>HF, nu</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24 h</td>
<td>24 ± 12</td>
<td>28 ± 11†</td>
<td>28 ± 11</td>
</tr>
<tr>
<td>Midnight–6 AM</td>
<td>30 ± 15</td>
<td>33 ± 12</td>
<td>31 ± 10</td>
</tr>
<tr>
<td>9 AM–6 PM</td>
<td>19 ± 10</td>
<td>24 ± 11†</td>
<td>23 ± 10</td>
</tr>
<tr>
<td>LF, CV%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24 h</td>
<td>2.82 ± 1.17</td>
<td>3.40 ± 1.40†</td>
<td>3.31 ± 1.70</td>
</tr>
<tr>
<td>Midnight–6 AM</td>
<td>2.81 ± 1.31</td>
<td>3.40 ± 1.41†</td>
<td>3.32 ± 1.88</td>
</tr>
<tr>
<td>9 AM–6 PM</td>
<td>2.44 ± 1.06</td>
<td>3.00 ± 1.18†</td>
<td>2.85 ± 1.36</td>
</tr>
<tr>
<td>LF, CV%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24 h</td>
<td>4.87 ± 1.00</td>
<td>5.25 ± 1.11†</td>
<td>5.10 ± 1.48</td>
</tr>
<tr>
<td>Midnight–6 AM</td>
<td>4.18 ± 0.97</td>
<td>4.73 ± 1.27*</td>
<td>4.61 ± 1.78</td>
</tr>
<tr>
<td>9 AM–6 PM</td>
<td>5.05 ± 0.98</td>
<td>5.30 ± 0.97</td>
<td>5.16 ± 1.14</td>
</tr>
<tr>
<td>α₁</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24 h</td>
<td>1.36 ± 0.16</td>
<td>1.27 ± 0.15§</td>
<td>1.31 ± 0.15</td>
</tr>
<tr>
<td>Midnight–6 AM</td>
<td>1.25 ± 0.20</td>
<td>1.18 ± 0.17*</td>
<td>1.22 ± 0.17</td>
</tr>
<tr>
<td>9 AM–6 PM</td>
<td>1.44 ± 0.14</td>
<td>1.33 ± 0.17</td>
<td>1.36 ± 0.15</td>
</tr>
<tr>
<td>β</td>
<td></td>
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</tr>
<tr>
<td>24 h</td>
<td>-1.13 ± 0.11</td>
<td>-1.12 ± 0.13</td>
<td>-1.14 ± 0.16</td>
</tr>
</tbody>
</table>

Values are means ± SD. nu, Normalized units; CV%, coefficient of component variance; α₁, fractal scaling exponent; β, power law scaling exponent. *P < 0.05; †P < 0.01; ‡P < 0.001: comparisons are between Pre and Post values.
moderately with the changes in HF

\( r = -0.76, P < 0.001 \), LF

\( r = 0.76, P < 0.001 \), and the LF/HF ratio

\( r = 0.69, P < 0.001 \) as analyzed over the 24-h recording. The correlation between the change in \( \alpha_1 \) and the change in the LF/HF ratio became weaker during the daytime hours (\( r = 0.57, P < 0.001 \)) compared with the nighttime correlation (\( r = 0.68, P < 0.001 \)).

**DISCUSSION**

There were two main findings in this study. First, moderate-volume training results in similar changes in the average HR and HR variability indexes as in high-volume training. Second, the autonomic regulation, analyzed as nu by spectral analysis of HR variability, changed toward increased vagal dominance after aerobic training in both high- and moderate-volume training groups. Similarly, aerobic training changed the short-term HR behavior (\(<11 \text{ beats}\)) toward less correlated short-term of R-R interval dynamics.

**Methodological considerations for HR variability analysis.** A decreased mean HR is normally associated with an increased total variance of R-R interval fluctuation and increases the absolute HF and LF spectral components (16). Therefore, the time domain HR variability indexes and the absolute spectral components may not be ideal methods for detecting changes in autonomic regulation after physical training interventions, where a decrease in basal HR occurs. In the present study, too, the time domain and absolute spectral calculations of HR variability are influenced by decreased HR because of the long-term aerobic training. The absolute spectral components are not able to indicate which physiological mechanisms, the enhancement in vagal tone and/or the decreased sympathetic activity, are responsible for the altered HR after training.

The normalized spectral values and LH/HF ratio represent the relative changes in the characteristics of the HR fluctuation pattern rather than the magnitude of HR variability (34). Therefore, the nu were used in the present study to assess the effects of training on the autonomic regulation of HR. Spectral components normalized by the average R-R interval length (CCV%) have also been used to evaluate the altered autonomic regulation in various pharmacological and clinical settings, where differences in the HR levels disturb the comparison of the absolute spectral values (16–18). Specifically, the HF power (CCV%) showed a good linear correlation with pharmacologically assessed cardiac vagal tone (16).

The DFA technique is a method to detect qualitative rather than quantitative changes in HR dynamics. From the mathematical point of view, spectral measures are associated with fractal scaling measures when analyzed as nu during strictly controlled external conditions, because both describe relative changes in the characteristics of HR fluctuations rather than the magnitude of HR variability, and, therefore, these indexes are not related to changes in the average HR. Indeed, our laboratory has recently reported (49, 52) a relationship between \( \alpha_1 \) values and the LF/HF ratio in controlled external situations with a fixed respiratory rate. However, the relationship is weak during “free-running” ambulatory ECG recordings (31, 40, 49) because measurement of scaling exponents by the DFA method provide information on the scaling properties of HR fluctuations over several segmented time windows, whereas conventionally computed spectral ratios describe HR fluctuations only in two predetermined time windows. Therefore, both normalized spectral units of HR variability and fractal scaling exponent were used in this study to describe changes in HR dynamics caused by exercise training.

From the physiological point of view, we have described the altered short-term fractal HR behavior in various pharmacological and physiological interventions (49, 52). Briefly, a change in the R-R interval dynamics from fractal behavior (\( \alpha_1 \) of \(<1.0 \)) toward a stronger
correlation in short-term R-R interval dynamics (α1 of ~1.5) was observed during a withdrawal of vagal activity by graded atropine infusion and dynamic low-intensity exercise (49, 52). Similarly, in the present study, the reduced α1 values correspond to increased vagal activity of the heart after aerobic training.

Effects of training on HR dynamics. Bradycardia is a well-known consequence of aerobic training and has been attributed to changes in the autonomic nervous system, i.e., either an increase in parasympathetic activity or a decrease in sympathetic activity, or else to a reduced intrinsic HR. Good aerobic fitness has been suggested to be associated with increased HR variability, especially with vagally mediated respiratory sinus arrhythmia, which is expressed as various time-domain indexes or as absolute spectral HF power (8–10, 13, 14). However, some studies have failed to show any association between good aerobic fitness and enhanced HR variability (7, 26). The results of prospective, well-controlled interventions are also controversial. All of the studies have indicated decreased HR, and most have revealed increased vagal activity measured by absolute HF power after aerobic training (1, 28, 42–44, 46). Contrariwise, some controlled studies have failed to show any association between aerobic training and HR variability (5, 29, 30). The results of these previous contradictory studies are partly limited by either the small sample size (26, 30) and subject characteristics, e.g., a narrow range in aerobic fitness (7). Also, the duration of interventions (from 6 to 36 wk), training frequency (from 3 to 7 sessions/wk), and the intensity of exercise (from ~60 to 90% of maximal HR) varies between the previous studies, and thus the results of different studies are difficult to compare. However, long-duration and/or intense training may not necessarily lead to greater enhancement in HR variability as prolonged (12 mo) and intense training (from walking to running) may return these changes in HR variability back to the control level (22). The training sessions performed during the previous days also affect HR variability and autonomic function (2, 12, 15). In our laboratory's recent study (15), the autonomic function recovered back to the baseline level at the second day after strenuous aerobic exercise. Therefore, 2 days of noneexercise were used in the present study before R-R intervals were recorded.

Several other methodological issues may also explain the controversial results. First, in our population-based cross-sectional study, good aerobic fitness was associated with an augmentation of vagally mediated beat-to-beat R-R interval fluctuation during a controlled exercise but not at controlled rest (51). This suggests that short-term HR variability recording at rest (~10 min) is not an ideal method for evaluating changes in autonomic regulation. Although the spectral HR variability methods with short-term recording at rest may not be sensitive enough to detect subtle changes in autonomic regulation after training, standard spectral methods over 24 h also failed to show any association between aerobic training and spectral HR variability indexes (26, 29). Second, sampling frequency is important for an accurate detection of beat-to-beat fluctuations in R-R intervals, as the precision of sampling of 8 vs. 1 ms resulted in a significant difference in the values of HR variability indexes, e.g., the difference was 14% for the LF/HF ratio in a 24-h recording (47). In the present study, the R-R intervals were recorded with a real-time microprocessor QRS detector system with 1-ms timing accuracy (41).

In the present study, the increased HFnu power and the decreased LFnu power as well as the decreased LF/HF ratio showed an alteration in the autonomic regulation of HR toward increased vagal dominance after aerobic training. The LF/HF ratio decreased also during the night hours after moderate-volume training intervention. This is an important finding because the night hours may reflect a more standardized condition and a subject’s behavior pattern does not disturb the R-R interval recordings. Concurrently, the present data also showed a decreased short-term scaling exponent α1 in R-R interval dynamics during the free-running daytime hours as well as during the night hours after aerobic training. The normalized spectral components remained unaltered during the night hours after a high-volume training period, suggesting that the scaling indexes detect subtle alterations in HR more effectively than simple spectral ratios.

In the previous pharmacological studies, both HFnu power indexes (CCV% and nu) are associated with vagal activity (16, 39). Also in the present study, both HFnu power indexes increased similarly as evidence of increased vagal activity after aerobic training. The opposite changes occur in the LF power normalized by average R-R interval (CCV%) and LFnu after aerobic training. These changes are concurrent with pharmacological studies where LF (CCV%) power decreased significantly during atropine infusion, revealing that this index is partly related to vagal activity (16). On the
contrary, LF\textsubscript{nu} is related to sympathetic activity studied by muscle sympathetic activity recordings from the peroneal nerve (33, 37), and decreased LF\textsubscript{nu} in the present study may therefore indicate a decreased sympathetic activity after aerobic training.

Although not a focus of this study, it was observed that both moderate- and high-volume training groups had reductions of 5–7 beats/min in maximal HR. This could have been a consequence of altered autonomic balance, although vagal activity is elevated only at lower HR. Alternatively, a reduction in intrinsic HR and altered sinoatrial node responses to norepinephrine have been identified as potential contributors to reduced maximal HR after training (19). This might suggest that altered autonomic balance and reduced intrinsic HR contribute to the changes that we observed in HR after training. Another interesting finding is an increased diastolic blood pressure after intervention for the high-volume training group (Fig. 3). This may be caused by an early stage of over-training rather than overreaching syndrome. How-ever, specific and sensitive parameters are not available to identify overreaching syndrome, and we did not focus on that issue in the present study (25).

**Study limitations.** Endurance training produces well-known functional improvements in cardiac performance. An enlarged blood volume, together with greater ventricular compliance and distensibility (27), may influence HR variability with changes of blood pressure variability as the input signal of the baroreflex system. In conscious dogs, changes in cardiac mechanics affect blood pressure variability, especially in HF by influencing stroke volume (36). Furthermore, the tidal volume becomes larger, and breathing frequency is considerably reduced after training interventions, at least in submaximal exercise intensity levels (23, 53). The reduced breathing frequency is known to increase the absolute spectral values of HF power of R-R intervals (39). Therefore, changes in the respiratory components of HR variability, reflecting cardiac outflow, may also be due to altered respiratory patterns caused by physical training. Consequently, the HF power of the R-R interval may not be an ideal index to estimate vagal activity if the cardiac mechanical properties and breathing pattern are changed. The effects of altered cardiac mechanics and respiration on fractal scaling properties of HR fluctuation are not well known. Therefore, more studies of the physiological background, particularly of the effects of respiratory patterns, on the fractal HR dynamics are warranted.

Analysis of cardiac autonomic regulation from 24-h recordings by frequency domain as well as by other measures may also have potential biases. Most importantly, many indexes may be influenced by physical activity during recordings (3). However, several studies have shown good short-term and long-term reproducibility of 24-h recordings when measured from surface ECGs (20, 24, 35). Furthermore, 24-h recordings have appeared to be a reliable and practical method that is not dependent on a subject’s cooperation and does not involve any placebo effect on the results (45, 48). This may result from a rather equal level of physical activity in individual subjects between the different 24-h periods. Therefore, 24-h HR variability measurement can be considered as an ideal method for assessing temporal changes in the autonomic regulation in individual subjects. In fact, reproducibility of 24-h HR variability measures may be even better than assessment of HR dynamics from short-term recordings during controlled situations (6, 50).

In conclusion, the reduction of HR after aerobic training is associated with increased indexes of cardiac vagal activity in healthy sedentary males. An 8-wk aerobic training intervention, including six 30-min sessions a week at an intensity of 70–80% of maximum HR, is a sufficient intervention to induce these effects on cardiac autonomic regulation.

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