Heart rate recovery and heart rate complexity following resistance exercise training and detraining in young men

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Heffernan KS, Fahs CA, Shinsako KK, Jae SY, Fernhall B. Heart rate recovery and heart rate complexity following resistance exercise training and detraining in young men. Am J Physiol Heart Circ Physiol 293: H3180–H3186, 2007.—The purpose of this investigation was to assess the effect of resistance training and detraining on cardiac autonomic modulation in young healthy men. We hypothesized that although resistance exercise training increases heart rate complexity and HRR after exercise but has no effect on spectral measures of HRV in young healthy men. These autonomic changes regress shortly after cessation of training. Thus, linear HRV was spectrally decomposed using an autoregressive approach. Nonlinear dynamics of heart rate variability included sample entropy (SampEn) and Lempel-Ziv entropy (LZEn). HRR was calculated from a graded maximal exercise test as maximal heart rate attained during the test minus heart rate at 1 min after exercise (HRR). There was no change in SampEn, LZEn, or HRR after the time-control portion of the study (P > 0.05). SampEn (P < 0.05), LZEn (P < 0.05), and HRR (P < 0.05) increased after resistance training and returned to pretraining values after detraining. There was no change in spectral measures of HRV at any time point (P > 0.05). These findings suggest that resistance exercise training increases heart rate complexity and HRR after exercise but has no effect on spectral measures of HRV in young healthy men. These autonomic changes regress shortly after cessation of training. However, a great deal of information in the HRV signal spectra is not solely harmonic. A certain degree of randomness or irregularity exists in the system (24). As such, use of linear methods alone to analyze beat-to-beat changes in heart rate results in loss of information on cardiovascular autonomic regulation (24).

Nonlinear means of assessing heart rate and its dynamics may provide additional information regarding cardiac autonomic fluctuations that cannot be detected by linear-based methods alone (24). Both branches of the autonomic nervous system (ANS) contribute to nonlinear oscillations in heart rate kinetics (41). Complexity refers to the irregularity of a dynamic process and can be measured quantitatively by assessment of the uncertainty of patterns reoccurring within a time-event series (29). Complexity and variability are not necessarily analogous terms: a periodic sinusoidal signal can be variable but not complex, whereas a random signal may be less variable and highly complex (29). Reduction in heart rate complexity occurs with aging (3, 29) and cardiovascular diseases, such as myocardial infarction and congestive heart failure (57), and is independently associated with mortality after myocardial infarction (48). Reductions in heart rate complexity have also been shown to predict onset of paroxysmal atrial fibrillation (54) and can occur despite no change in traditional HRV parameters (46, 55).

The purpose of this investigation was to assess the effect of resistance training and detraining on cardiac autonomic modulation in young healthy men. We hypothesized that although there would be no change in linear-based measures of HRV after resistance training, there would be increases in nonlinear heart rate complexity [sample entropy (SampEn) and Lempel-Ziv entropy (LZEn)] and faster HRR after exercise consistent with improved cardiac autonomic modulation.

MATERIALS AND METHODS

Subjects. Fourteen healthy men participated in the present study. All were previously sedentary, and none had been participating in regular resistance training. All subjects were normotensive (<140/90 mmHg), had normal fasting glucose and blood lipids, and did not have anemia or any electrolyte imbalances. Descriptive characteristics are presented in Table 1. According to health history questionnaire, none had any other overt chronic disease that could alter cardiovascular autonomic function. Subjects did not smoke or take medications of any kind. Before participation in this project, which was approved by the Institutional Review Board of the University of Illinois at Urbana-Champaign, all subjects gave written informed consent.

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Table 1. Subject characteristics at baseline

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>25.0±1.1</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>26.1±1.3</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>126±3</td>
</tr>
<tr>
<td>Diastolic blood pressure, mmHg</td>
<td>73±2</td>
</tr>
<tr>
<td>Fasting blood lipids, mg/dl</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>165.2±9.8</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>39.4±2.0</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>103.6±7.8</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>111.6±21.1</td>
</tr>
</tbody>
</table>

Values are means ± SE (n = 14).

Study design. Measurements were made in each subject at baseline (Pre1), after a 4-wk time-control period (Pre2), after a 6-wk resistance-training intervention (Post1), and after a 4-wk detraining period (Post2). Length of the resistance-training stimulus and length of the detraining period were based on previous aerobic exercise-training/detraining interventions producing autonomic changes following similar, and sometimes even shorter, time periods (17, 25, 49). During the time-control portion of the study, subjects were instructed to maintain their present sedentary lifestyle. During the resistance-training component of the study, subjects were instructed to refrain from any structured aerobic/endurance exercise. Subjects were asked to resume their previous inactive lifestyle and refrain from all forms of structured exercise during the detraining period. At baseline, subjects completed a blood test after an overnight (≥12-h) fast. Immediately after the blood test, body composition was assessed. During a second visit, 24 – 48 h after the blood draw, resting ECG and blood pressure were measured, and maximal aerobic exercise testing and 1-repetition maximum (RM) bench press testing were performed (in that order). All within-subject sessions were conducted at the same time of day to reduce possible diurnal variations in physiological signals. Subjects were tested in the postprandial state (~3 h) and asked to refrain from caffeine and alcohol ingestion for 24 h before testing.

Anthropometrics. Body composition was determined using whole body air-displacement plethysmography (Bod Pod, Life Measurement, Concord, CA). Height was measured (to the nearest 0.5 cm) using a stadiometer. Weight was measured using an electronic scale that was calibrated before each measurement (Bod Pod). Body mass index was calculated as weight (kg) / height² (m²).

Brachial artery blood pressure assessment. Blood pressure (BP) was measured at baseline and used for screening purposes to ensure that all participants were normotensive. Subjects rested quietly in the supine position in a dimly lit, temperature-controlled room for ≥15 min. Resting systolic BP (SBP) and diastolic BP (DBP) were measured with the subject in the supine position with use of an automated oscillometric cuff. All brachial BP measurements were made in duplicate. The average of the two values was recorded and used for subsequent analysis. If values were not within 5 mmHg, a third measurement was obtained and the two closest values were averaged (38).

ECG signal acquisition. After the 15-min quiet rest period, heart rate was recorded continuously by ECG with a single-lead CM5 configuration with subjects still in the supine position (Biopac Systems, Santa Barbara, CA). Lead site preparation and placement were standardized according to American Heart Association standards (15). Breathing was paced with a metronome at 12 breaths/min. The ECG was collected online at a sampling rate of 1,000 Hz, in real time, and stored on a computer. All data were stored and analyzed offline. Subsequent offline signal processing was performed using commercially available software (WinCPRS, Turku, Finland). Data were visually and automatically inspected for ectopic beats (premature, supraventricular, and ventricular) and interpolated in accordance with previous suggestions (50) to provide a continuous data stream. An R-R interval (RRI) time-event series was generated from successive heart rate peaks. The time series was detrended and resampled at 5 Hz.

Heart rate complexity. After removal of the linear trend, SampEn and LZEn were used to quantify the complexity of the RRI time-event series and calculated using nonlinear dynamics as previously described (20; for a detailed description of these processes, see Refs. 24 and 44).

SampEn, which determines the probability of finding specific patterns or matches in a short time series, ranges from 0 to 2. In a highly predictable (i.e., regular) signal, such as a sine wave, it will have a value close to 0. In contrast, a highly irregular signal will have a value close to 2 (44). The embedding dimension m (length of sequences to be compared) may range from 2 to 10, while the filter parameter r (tolerance for accepting matches) may range from 0.10 to 0.50 (54). In the present investigation, m was fixed at 2, while r was set at 20% of the standard deviation of the time series on the basis of previous suggestions (24). Although SampEn is robust, it has been suggested that data sets with <200 points (N) produce large confidence intervals (24). Therefore, we analyzed >200 consecutive stable and ectopy-free RRI. On average, 220–250 RRI were used for the analysis (based on maximal allowable beats owing to resting heart rate). Given that entropy measures may be sensitive to the absolute number of data points used in the analysis, the same number of beats was used for all within-subject analyses.

LZEn was calculated using the same data points used for SampEn. This measure, based on Kolmogorov estimates, counts the number of different and repeating patterns, from short to long, in the time-event series and generates a string of symbols using binary coding: when a value above the mean is attained, “1” is assigned, and when a value below the mean is attained, “0” is assigned. Thus the binary sequence is constructed by two operations: insertion of symbol(s) to form a subsequence (a sequence of 0s and 1s) and copying of this subsequence (2). With use of this comparison and accumulation method, LZEn is computed on the basis of the number of such insertion and copying operations needed to generate the original sequence (2, 24). Similar to SampEn, an LZEn value near zero means that the signal is deterministic (regular), whereas higher values reflect increased randomness of the signal.

HRV. Power spectral analysis was performed on the same data points used for complexity assessment by a maximum entropy method, as we have previously described (19, 20). The optimum order of the autoregressive model was determined using Akaike’s information criterion. If this method yielded a model order ≥16, we used 16 to avoid possible shifting of the spectral peaks (6). The power was calculated by measuring the area under the peak of the power spectra density curve, and corresponding bandwidths were interpreted as follows: an HF (0.15–0.40 Hz) region, indicative of parasympathetic modulation of heart rate, and a low-frequency (0.04–0.15 Hz) region, mediated by the sympathetic and parasympathetic arms of the ANS (35, 51). The power spectra were calculated in absolute and normalized units to represent the relative value of each power component as a proportion of the total power (35, 51). All data acquisition and postacquisition analyses were carried out in accordance with the standards of the Task Force of the European Society of Cardiology and North American Society of Pacing and Electrophysiology (51). All analyses were carried out using WinCPRS software. Intraclass correlation coefficients attained in our laboratory are high (20) and consistent with previous findings for short-term measures of HRV in the frequency domain (45).

Maximal O₂ uptake and HRR. Peak O₂ consumption (V⁰₂peak) was assessed using a graded cycle ergometry protocol. Subjects completed a brief warm-up consisting of cycling against no resistance (0 W) for 2 min. The first workload was set at 50 W. Workload was increased by 30 W every 2 min until test termination. Heart rate was measured using a heart rate monitor (Polar Electro, Woodbury, NY). Ratings of perceived exertion (RPE) were also assessed once per stage. Analyses of expired gases were carried out with a breath-by-breath metabolic
system (Quark b², Cosmed, Rome, Italy). The test was terminated when subjects met three of the following five criteria: 1) a final RPE score of ≥17 on the Borg scale (scale 6–20), 2) a respiratory exchange ratio >1.1, 3) no change in heart rate with a change in workload, 4) a “plateau” (increase of ≥150 ml) in O₂ uptake with an increase in workload, and/or 5) volitional fatigue.

HRR was calculated as the difference between maximum heart rate during the test and heart rate 1 min after cessation of exercise. The recovery protocol consisted of 2 min of light cycling (50 rpm and 0 W) followed by 1 min of quiet sitting on the cycle. The intraintra exchange correlation coefficient for HRR attained on 2 separate days in our laboratory is 0.85.

**Fasting blood chemistries.** Fasting glucose was assessed by an O₂ rate method using an O₂ electrode (Beckman Coulter, Villepoinat, France). Total cholesterol, HDL cholesterol, and triglycerides were measured using enzymatic techniques. LDL was calculated using the Friedewald formula. Hematocrit (Hct) and hemoglobin (Hb) were measured using an automated hematology analyzer (Sysmex, Kobe, Japan). Na⁺, K⁺, Cl⁻, and Ca²⁺ were measured using indirect potentiometry (Synchron LX ISE, Beckman Coulter).

**1-RM bench press.** One RM (defined as the maximum amount of weight lifted with proper form through a full range of motion for a single repetition) for the bench press was ascertained in accordance with established guidelines (1). Subjects first completed a brief warm-up consisting of 10 repetitions of a submaximal load. An RPE was then attained using a scale of 1–10, and a second warm-up was attained using a scale of 1–10, and a second warm-up was performed (1). The final weight successfully lifted was recorded as the subject’s perceived capacity. Weight was added in 2.3- to 11.4-kg increments until subjects could no longer successfully complete one repetition (1). The final weight successfully lifted was recorded as the 1-RM. Subjects were allowed 3–5 min of rest between sets to ensure that a maximal effort was exerted with each attempt. Maximal values were attained for all subjects in less than five attempts. A spotter was present at all testing sessions to ensure the safety of the subject during bench press testing. The 1-RM value was taken as a measure of upper body strength and used to gauge effectiveness of the resistance-training intervention (i.e., documentation of a training effect).

**Resistance training.** Training sessions were carried out 3 days/wk (~60 min/session). All sessions were supervised by personal trainers/ strength-and-conditioning specialists. The resistance-training protocol was a two-way body part split. Each session consisted of five exercises. Exercises were selected to stress all major muscle groups of the upper and lower body with multiple- and single-joint exercises. The primary muscles of the legs (quadiceps and hamstrings), back (latisimus dorsi, upper, middle, and lower trapezius, erector spinae, and rhomboids), and biceps were trained during one training session. The primary muscles of the chest (pectoralis major/minor), shoulders (deltoide), and triceps were trained during the next training session. Exercises were performed using a combination of free weights (barbells and dumbbells) and machine (Body Solid EXM-3000LPS). Each session began with a brief warm-up consisting of 1 set of 15 repetitions of the first exercise to be performed during that session using a submaximal load. Then three sets of each exercise were performed, with 1–2 min of rest allowed between each set. During the initial 2 wk, load was selected to ensure that fatigue was reached at 12–15 repetitions. During the final 4 wk, load was adjusted to ensure that fatigue was reached at 8–12 repetitions. As strength increased, load was progressively increased to ensure that fatigue occurred within the desired repetition schema with proper form. This program was designed to maximize strength and muscular hypertrophy (1).

**Statistical analysis.** ANOVA with repeated measures was used to assess variables over four time points (Pre1 × Pre2 × Post1 × Post2). When a significant main effect was detected at a significance level of P < 0.05, t-tests were used for post hoc comparisons. Adjustment for multiple comparisons was made with Bonferroni’s correction. Statistical significance was set at P < 0.01 for all pair-wise comparisons. Total power, absolute low-frequency power, and absolute HF power were not normally distributed (determined from Shapiro-Wilk and Kolmogorov-Smirnov tests as well as analysis of Q-Q plots). Therefore, these values were logarithmically transformed to meet assumptions for parametric statistical analysis. Values are means ± SE. Data analyses were carried out using Statistical Package for the Social Sciences (version 12.0.1, SPSS, Chicago, IL).

### RESULTS

All participants completed the time-control, resistance-training intervention, and detraining portions of the study. There was no change in body fat, lean mass, or fasting blood chemistries over the course of the study (Tables 2 and 3; P > 0.05). There was no change in Hb or Hct over time (Table 2; P > 0.05). An overall time effect was detected for relative VO₂peak (Table 2; P < 0.05). VO₂peak at Pre2 was different from VO₂peak at Post1 (Table 2; P < 0.01). A time effect was also detected for 1-RM bench press. Post1 and Post2 1-RM bench press values were significantly greater than Pre1 and Pre2 values (Table 2; P < 0.01). A significant time effect was detected for HRR: HRR Post1 was significantly higher than Pre1 and Pre2 (P < 0.01).

There was no change in any spectral HRV parameter (Table 4; P > 0.05). There was also no change in RRI or standard deviation of RRI (Table 4; P > 0.05). A significant time effect

### Table 2. Body composition, strength, and aerobic capacity before and after resistance training

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre1</th>
<th>Pre2</th>
<th>Post1</th>
<th>Post2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, kg</td>
<td>85.4±4.8</td>
<td>85.9±5.3</td>
<td>86.4±4.9</td>
<td>86.1±4.9</td>
</tr>
<tr>
<td>%Body fat</td>
<td>22.3±2.6</td>
<td>21.9±3.0</td>
<td>22.9±2.6</td>
<td>21.7±2.7</td>
</tr>
<tr>
<td>%Lean mass</td>
<td>77.6±2.6</td>
<td>77.4±2.7</td>
<td>77.1±2.6</td>
<td>78.3±2.7</td>
</tr>
<tr>
<td>1-RM bench press, kg</td>
<td>67.7±4.5</td>
<td>69.2±4.6†</td>
<td>76.8±4.6†</td>
<td>72.8±4.6††</td>
</tr>
<tr>
<td>VO₂peak, ml·kg⁻¹·min⁻¹</td>
<td>29.5±1.1</td>
<td>27.3±1.0†</td>
<td>30.7±1.0</td>
<td>29.5±1.0</td>
</tr>
</tbody>
</table>

Values are means ± SE. Pre1, baseline; Pre2, after 4-wk time-control period; Post1, after 6-wk resistance training intervention; Post2, after 4-wk detraining period. RM, repetition maximum; VO₂peak, peak O₂ consumption. *Significantly different from Pre1 and Pre2 (P < 0.01). †Significantly different from Post1 (P < 0.01).

### Table 3. Blood chemistries before and after resistance training

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre1</th>
<th>Pre2</th>
<th>Post1</th>
<th>Post2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺, mmol/l</td>
<td>140.6±0.5</td>
<td>141.1±0.4</td>
<td>140.4±0.5</td>
<td>140.6±0.4</td>
</tr>
<tr>
<td>K⁺, mmol/l</td>
<td>4.43±0.08</td>
<td>4.48±0.09</td>
<td>4.53±0.10</td>
<td>4.35±0.11</td>
</tr>
<tr>
<td>Cl⁻, mmol/l</td>
<td>102.9±0.6</td>
<td>103.1±0.5</td>
<td>103.0±0.5</td>
<td>103.9±0.6</td>
</tr>
<tr>
<td>Ca²⁺, mg/dl</td>
<td>9.76±0.09</td>
<td>9.73±0.05</td>
<td>9.54±0.06</td>
<td>9.53±0.08</td>
</tr>
<tr>
<td>Glucose, mg/dl</td>
<td>92.8±2.0</td>
<td>90.4±2.6</td>
<td>90.5±1.7</td>
<td>90.4±1.8</td>
</tr>
<tr>
<td>Hb, g/dl</td>
<td>15.5±0.3</td>
<td>15.4±0.2</td>
<td>15.5±0.3</td>
<td>15.5±0.3</td>
</tr>
<tr>
<td>Hct, %</td>
<td>45.5±0.8</td>
<td>44.7±0.6</td>
<td>45.5±0.9</td>
<td>45.5±0.9</td>
</tr>
</tbody>
</table>

Values are means ± SE. Hct, hematocrit.
was detected for SampEn (Fig. 2; \(P < 0.05\)). SampEn Post1 was significantly higher than SampEn Pre2 (\(P < 0.01\)). Although SampEn Post1 was greater than SampEn Pre1 and SampEn Post2, this did not reach statistical significance (\(P = 0.029\) for each). A significant time effect was detected for LZEn (Fig. 3; \(P < 0.05\)). LZEn Post1 was significantly higher than LZEn Pre1, LZEn Pre2, and LZEn Post2 (Fig. 3; \(P < 0.01\)).

**DISCUSSION**

There are several novel findings of the present study. 1) Short-term resistance exercise training increases HRR after exercise and nonlinear entropy measures of heart rate complexity, suggesting improved cardiac autonomic modulation. 2) With no change in HRV after resistance training, heart rate complexity and HRR provided additional information on cardiac autonomic regulation that was not detected by spectral measures alone. On removal of the exercise stimulus (i.e., exercise detraining), cardiac autonomic parameters returned to pretraining values within 4 wk, in conjunction with a partial maintenance of muscular strength.

**HRR.** To our knowledge, this is the first study to prospectively examine the effect of resistance exercise training on HRR after exercise. Consistent with previous cross-sectional observations in resistance-trained men (34), a resistance-training intervention increases HRR after exercise. These findings are also consistent with improvements noted after aerobic/endurance exercise training in cardiac rehabilitation patients (26). Postexercise recovery of heart rate is mediated by both branches of the ANS. The initial decrease in heart rate is mediated via prompt parasympathetic reactivation, with later reductions due to continued parasympathetic reactivation and sympathetic withdrawal (39). Recently, Dewland et al. (14) suggested that HRV and HRR yield information concerning distinct and independent aspects of cardiac autonomic regulation. According to Buchheit et al. (7, 8), although HRV more aptly reflects phasic fluctuations in vagal efferent activity (parasympathetic modulation), HRR is an index of mean cholinergic signaling at the level of the sinoatrial node (vagal tone). In this context, our findings suggest that resistance training increases vagal tone (i.e., increased acetylcholine release from the vagus and increased receptor number/sensitivity) more than parasympathetic modulation.

**HRV and complexity.** Numerous studies acknowledge improvements in spectral measures of HRV after aerobic/endurance exercise training (13, 17, 25, 31). Consistent with prospective findings of Cooke and Carter (12), we noted no change in spectral measures of HRV after resistance training. However, we did note significant increases in entropy measures of heart rate complexity (SampEn and LZEn) after training. Although we previously showed that acute resistance exercise significantly reduces HRV (19, 20) and heart rate complexity (20), these subacute responses do not appear to transpose to a state of permanence. The potentially negative

**Table 4. HRV parameters before and after training**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pre1</th>
<th>Pre2</th>
<th>Post1</th>
<th>Post2</th>
</tr>
</thead>
<tbody>
<tr>
<td>lnTP, ms(^2)</td>
<td>8.3±0.2</td>
<td>8.7±0.2</td>
<td>8.7±0.2</td>
<td>8.4±0.3</td>
</tr>
<tr>
<td>lnLF, ms(^2)</td>
<td>6.6±0.2</td>
<td>6.9±0.2</td>
<td>7.0±0.2</td>
<td>6.8±0.3</td>
</tr>
<tr>
<td>LF, NU</td>
<td>0.28±0.04</td>
<td>0.28±0.04</td>
<td>0.29±0.05</td>
<td>0.31±0.05</td>
</tr>
<tr>
<td>lnHF, ms(^2)</td>
<td>7.7±0.3</td>
<td>7.9±0.3</td>
<td>8.0±0.3</td>
<td>7.6±0.3</td>
</tr>
<tr>
<td>HF, NU</td>
<td>0.71±0.04</td>
<td>0.69±0.04</td>
<td>0.68±0.05</td>
<td>0.67±0.05</td>
</tr>
<tr>
<td>LF/HF, %</td>
<td>49.7±14.0</td>
<td>50.0±11.6</td>
<td>63.1±19.3</td>
<td>63.7±17.6</td>
</tr>
<tr>
<td>RRI, ms</td>
<td>974.7±47.1</td>
<td>920.2±32.5</td>
<td>947.0±43.2</td>
<td>925.7±34.1</td>
</tr>
<tr>
<td>RRI SD, ms</td>
<td>73.0±7.6</td>
<td>87.2±9.5</td>
<td>87.2±8.9</td>
<td>78.9±10.5</td>
</tr>
</tbody>
</table>

Values are means ± SE. HRV, heart rate variability; TP, total power; LF, low frequency; HF, high frequency; NU, normalized units; RRI, R-R interval.
acute response may result in positive adaptations after repeated exposure.

It has been suggested that nonlinear methods of beat-to-beat heart rate assessment provide complementary and additive information beyond traditional spectral measures of HRV (24). In animal models, nonlinear cardiovascular fluctuations appear to be mediated via the vagus and cholinergic system, inasmuch as cholinergic blockade with atropine significantly reduces cardiovascular complexity (4, 36). This is similar in humans: parasympathetic blockade with glycopyrrolate (37) or high-dose atropine reduces heart complexity (42), and parasympathetic stimulation with low-dose atropine increases complexity (42). The β-adrenoceptor system plays little role in the generation of nonlinear cardiovascular dynamics in animal models, inasmuch as β-adrenoceptor blockade with propranolol has negligible effects on cardiovascular complexity (4, 36). It has been suggested that vagal withdrawal is a more important modulator of nonlinear deterministic regulation than sympathetic tone in animals and humans (18). However, sympathetic activation with pharmacological (i.e., isoproterenol) or physiological (i.e., head-up tilt, active standing, dynamic cycling exercise, and isometric handgrip exercise) manipulation has been shown to reduce irregularity/complexity (18, 41, 42, 56, 58), and sympathetic blockade with propranolol increases complexity (18, 27). This supports a role for the sympathetic branch of the ANS in modulating cardiovascular nonlinearities in humans. Because conditions associated with sympathoexcitation may also be linked to vagal withdrawal, the exact contributions of the sympathetic and parasympathetic branches of the ANS to nonlinear fluctuations in heart rate remain difficult to separate. Porta et al. (41) proposed that complexity measures of HRV are a reflection of a general sympathovagal balance. In this context, our findings denote that short-term resistance training increases autonomically mediated heart rate complexity, reflective of increased parasympathetic and/or reduced sympathetic cardiac autonomic control.

Resistance exercise detraining. This is the first study to examine the effect of resistance exercise detraining on cardiac autonomic modulation. Sugawara et al. (49) showed a regression of postexercise vagal reactivation to preendurance training values after 2 wk of detraining. Similarly, we noted a complete reversibility of HRR and LZEn and a near-complete reversibility of SampEn after the detraining component of the study. Thus alterations in autonomic modulation after short-term training may be dependent on continuation of the exercise stimulus. With longer duration of training, more permanent/chronic adaptations may ensue, e.g., cross-sectional findings in endurance- and strength-trained athletes (5, 34).

Potential mechanisms. Numerous complex mechanisms (i.e., multiple attractors) contribute to the genesis of cardiovascular oscillations, including gating of respiration to efferent sympathetic and vagal motoneurons (16), central modulation, negative feedback from peripheral reflexes (chemoreflex and baroreflex), and/or self-sustained peripheral vasomotor oscillations (10). Nonlinear fluctuations in RRI may be related to baroreflex physiology, inasmuch as pharmacological manipulation of this feedback control loop results in concomitant alterations in RRI complexity (24). It is possible that the baroreflex was reset to a new pressure operating range after resistance training (52), thus augmenting cardiac vagal outflow. Resetting with resistance training may occur with no change in baroreflex sensitivity at the new operating point (12, 28, 30). It has been suggested that respiratory gating is the most crucial and prepotent factor governing modulation of autonomic rhythms, and changes in baroreflex physiology may be secondary to changes in respiratory physiology (10, 16). It is possible that resistance training alters regions within the brain (neuroplastic adaptations) responsible for the generation of linear/nonlinear autonomic fluctuations (central modulation of a cardiovascular/respiratory oscillator or central command). Nelson et al. (33) demonstrated that aerobic/endurance training alters dendritic fields of neurons within the posterior hypothalamic area, periaqueductal gray, cuneiform nucleus, and nucleus of the tractus solitarius of rats, crucial regions responsible for neural cardiorespiratory regulation. Nelson and Iwamoto (32) further demonstrated that detraining reverses the neuroplastic adaptations. Resistance training and subsequent detraining may induce similar neuroplastic adaptations in humans.

We noted no change in Na+, K+, Cl−, or Ca2+, suggesting that resistance training did not alter electrolyte balance and, subsequently, alter cardiac autonomic function. There was no change in body composition or blood glucose, suggesting that findings are not secondary to changes in these parameters with training. Although there was a statistical difference in VO2peak from Pre2 to Post1, it is unlikely that this was physiologically relevant. There was no difference in VO2peak from Pre1 to Pre2/Post2, arguing against improvements in aerobic fitness being the determinant of improved autonomic modulation. The changes in VO2peak did not mirror changes in heart rate complexity or HRR. However, we cannot rule out with certainty a modest improvement in aerobic capacity stemming from the resistance exercise training. We noted no change in Hb and Hct with training, arguing against plasma volume alterations underlying the autonomic changes. However, previous research has noted small increases in blood volume with resistance training (30); thus modulation of blood rheological properties contributing to autonomic modulation remains plausible.

Clinical implications. HRR after exercise is a predictor of cardiovascular mortality and is related to arrhythmia and sudden cardiac death (9, 11, 47). Slow HRR after exercise is associated with impaired fibrinolysis, increased inflammation, and increased carotid atherosclerosis (21–23). Loss of cardiac complexity has been shown to be associated with inflammation (43), cardiovascular disease, and arrhythmia development (46, 48, 54, 55). Given that resistance training increases HRR and heart rate complexity, this may have favorable cardiovascular health implications (40).

The strength of the present study resides in its design. With use of a time-control and detraining component, findings strongly suggest that alterations in autonomic modulation were due to the resistance training stimulus and were not secondary to natural/seasonal physiological variability. However, limitations to this study should also be noted. This study had a relatively small sample size. Given potential sex differences in autonomic modulation, our findings may not be extended to women. Our findings may also not be applicable to older adults or clinical populations.

In conclusion, short-term resistance exercise training increases autonomically mediated heart rate complexity and HRR after exercise but does not affect spectral measures of HRV in young healthy men. Resistance exercise detraining is associated with a near-complete return of HRR and heart rate
complexity to pretraining values, suggesting that autonomic adaptations regress soon after the resistance exercise stimulus is removed.

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REFERENCES


