Fractal scaling properties of heart rate dynamics following resistance exercise training

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Heffernan KS, Sosnoff JJ, Fahs CA, Shinsako KK, Jae SY, Fernhall B. Fractal scaling properties of heart rate dynamics following resistance exercise training. J Appl Physiol 105: 109–113, 2008.—With aging and disease, there is a breakdown of the natural fractal-like organization of heart rate (HR). Fractal-like correlation properties of HR can be assessed with detrended fluctuation analysis (DFA). A short-time scaling exponent (αs) value of 1 is associated with healthy HR dynamics, whereas values that deviate away from 1, in either direction, indicate fractal collapse. The purpose of this study was to examine the effect of resistance exercise training (RT) on fractal correlation properties of HR dynamics. Resting ECG was collected at baseline, following a 4-wk time control period and 6 wk of RT (3 days per wk) in 34 men (23 ± 1 yr of age). Fractal properties of HR were assessed with DFA. There was no change in αs following either the time control period or RT (1.01 ± 0.60 to 0.98 ± 0.06 to 0.93 ± 0.04, P > 0.05). Given the potential bidirectional nature of fractal collapse, subjects were retrospectively separated into two groups (higher αs and lower αs) on the basis of the initial αs by using cluster analysis. An interaction was detected for the αs following RT (P < 0.05). There was no change in αs in either group following the time control, but αs increased following RT in the lower αs group (n = 18; 0.73 ± 0.04 to 0.69 ± 0.04 to 0.88 ± 0.04) and αs decreased following RT in the higher αs group (n = 16; 1.20 ± 0.04 to 1.24 ± 0.04 to 0.98 ± 0.04). In conclusion, RT improves fractal properties of HR dynamics.

nonlinear; autonomic nervous system

IN A HEALTHY STATE, heart rate (HR) intervals fluctuate with deterministic chaos (i.e., patterns of fluctuations recur in larger fluctuations over time and are sensitive to the initial state) (30). With aging and disease, there is a breakdown of fractal-like organization of HR dynamics (10, 27). In some pathologies loss of short-term fractal dynamics manifests as uncorrelated randomness in HR variability. This can be seen in congestive heart failure (22), Chagas disease (1, 2), preceding the onset of atrial fibrillation/ventricular tachycardia (19, 35, 36), and following acute myocardial infarction (26). Loss of fractal dynamics may also be associated with excessive order (pathological periodicity whereby the HR signal is mode locked into a single or very few dominant attractors) as seen with aging and stable angina pectoris (10, 20, 27). The unifying theme underlying both routes to pathology is the degradation of correlated, multiscale dynamics (3). Loss of fractal scaling has been shown to predict mortality in patients with left ventricular dysfunction (9, 16), with heart failure (17), following acute myocardial infarction (31), and in healthy elderly (8). Thus interventions that restore fractal scaling properties to HR dynamics may have important clinical implications.

There is little information available on interventions aimed at improving fractal properties of HR. Resistance training (RT) is presently recommended by several medical organizations for health promotion and primary/secondary disease prevention. RT has recently been shown to improve cardiac autonomic modulation (5, 6), a strong determinant of fractal correlation properties of HR (30). Thus it is reasonable to speculate that resistance training may improve fractal properties of HR dynamics, although this has yet to be examined.

The purpose of this study was to examine the fractal correlation properties of HR in young men following RT. We hypothesized that RT would improve fractal-like correlation properties of HR dynamics.

MATERIALS AND METHODS

Subjects. Thirty-four healthy men between 18 and 30 yr of age participated in this study. All subjects were normotensive [systolic blood pressure (SBP): 127 ± 1 mmHg, diastolic blood pressure (DBP): 73 ± 1 mmHg] and had normal fasting glucose and blood lipids (Table 2). According to health history questionnaire, all were apparently healthy without any history of chronic disease that could alter cardiovascular function. Subjects did not smoke, nor did they take any medications. None had previously participated in regular exercise training. Before participation in this project, all subjects gave written informed consent. This study was approved by the Institutional Review Board of the University of Illinois at Urbana-Champaign.

Study design. Measurements were made in each subject at baseline (Pre1), after a 4-wk time control period (Pre2), and following a 6-wk RT intervention (Post). During the time control portion of the study, subjects were instructed to maintain their lifestyles. During the RT component of the study, subjects were instructed not to participate in any structured aerobic/endurance exercise. At baseline, subjects completed a blood test following a 12-h overnight fast. Immediately following the blood test, subjects underwent body composition assessment. During a second visit (24–48 h after the blood draw), subjects underwent resting ECG measurement, blood pressure measurement, maximal aerobic exercise testing, and one-repetition maximum (1-RM) bench press testing (in that order). All within-subject sessions were conducted at the same time of day to reduce possible diurnal variation. Subjects were tested in the postprandial state (~3 h) and asked to refrain from caffeine and alcohol ingestion for 24 h before testing. Postmeasures were conducted ~48 h after the last exercise session.

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Anthropometrics. Body composition was determined using whole body air displacement plethysmography (Bod Pod; Life Measurement, Concord, CA). Height was measured using a stadiometer (to the nearest 0.5 cm). Weight was measured using an electronic scale, which was calibrated before each measurement (Bod Pod). Body mass index was calculated as weight (kg) divided by height (m) squared.

Brachial artery blood pressure assessment. Baseline blood pressure was measured, and this was also used for screening to ensure that all participants were normotensive (<140/90 mmHg). Subjects rested quietly in the supine position in a dimly lit, temperature-controlled room for 15 min. Resting SBP and DBP were measured with an automated oscillometric cuff, in duplicate. For analysis, the average of the two values was used. If values were not within 5 mmHg, a third measure was taken and the two closest values were averaged and used in the analysis (38).

ECG signal acquisition. HR was recorded continuously with a single-lead CM5 configuration ECG (Biopac Systems, Santa Barbara, CA) after a 15-min quiet rest period. Real-time ECG was collected online at a 1,000-Hz sample rate stored on a computer and subsequently analyzed. The offline signal processing was conducted using commercially available software (WinCPRS, Turku, Finland). Following visual and automated data inspection for ectopic beats, the time series was interpolated in accordance with previous suggestions (50) to provide a continuous data stream. Exactly 600 R-R intervals were used for analysis.

Detrended fluctuation analysis. Detrended fluctuation analysis (DFA) was used to quantify the short-term fractal-like scaling properties of R-R interval data. The short-term scaling exponent (α) obtained from this technique provides insight into the embedded scale-invariant characteristics of a nonstationary time series. Unlike traditional methods of assessing variability, DFA is robust to trends and nonstationary data. A detailed description of this technique has previously been described by Peng et al. (22). Briefly, the root-mean-square fluctuations of 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 (9). The scaling exponent α represents the slope of this line, which relates (log) fluctuation to (log) window size. The short-term (from 4 to 11 beats) scaling exponent (α) was calculated on the basis of previous experiments (9). An α value of 0.5 reflects white noise (i.e., no correlations), whereas values <0.5 imply the data are anticoordinated. A value of 1.5 reflects Brownian noise (i.e., random walk). An α value near 1 reflects pink noise (i.e., fractal scaling behavior) and is associated with healthy HR dynamics. It has been suggested that the maximum time scale should be <N/6, where N is the length of the signal (28). With 600 total beats in the signal, we can reliably quantify α up to time scales n = 100. Therefore, our time scale selection of 4–11 beats is sufficient. The intraclass correlation coefficient (ICC) attained on our Pre1 and Pre2 time points was very high (ICC = 0.95), indicating good test-retest repeatability in our laboratory.

Maximal oxygen uptake. Cycle ergometry was used to measure peak oxygen consumption. Following a brief warm-up of unloaded cycling, subjects started pedaling at 50 W, with incremental workload up to time scales of 30 W every 2 min until test termination. HR was measured with a Polar Heart Rate Monitor (Polar Electro, Woodbury, NY). Ratings of perceived exertion were obtained during each exercise stage. Expired air was analyzed with a Quark b2 breath-by-breath metabolic system (Cosmed, Rome, Italy). The test was terminated when subjects met three of the following five criteria: 1) a final rating of perceived exertion score of 17 or greater on the Borg scale (scale 6–20), 2) a respiratory exchange ratio greater than 1.1, 3) no change in HR with a change in workload, 4) a “plateau” (increase of no more than 150 ml) in oxygen uptake with an increase in workload, and/or 5) volitional fatigue.

Fasting blood chemistries. Fasting glucose was assessed via an oxygen rate method by using a Beckman Coulter oxygen electrode (Beckman Coulter, Villepoinate, France). Total cholesterol, HDL cholesterol, and triglycerides were measured enzymatically. LDL was calculated with the use of the Friedewald formula. White blood cell (WBC) count was measured with the use of a quantitative automated hematology analyzer (Sysmex XE-2100; Sysmex, Kobe, Japan).

**1-RM bench press.** Using a bench press exercise, the 1-RM was measured and was used to document the effectiveness of the RT intervention.

**RT.** Subjects exercise trained 3 days per wk (~60 min per session), using a two-way body part split (legs, back, and biceps were exercised on one day; chest, shoulders, and triceps were exercised on a second day), with five exercises in each session. All major muscle groups were included, both upper and lower body, using multijoint and single-joint exercises. All sessions were supervised by personal trainers/strength and conditioning specialists. Each session began with a warm-up consisting of one set of 15 repetitions of the first exercise to be performed during that session using a submaximal load. Three sets of each exercise were then performed with 2–3 min of rest between each set. For the first 2 wk, the exercise weight was selected to ensure fatigue was reached between 12 and 15 repetitions. For the final 4 wk, the exercise weight was selected to induce fatigue between 8 and 12 repetitions. As strength increased, load was progressively increased to ensure fatigue occurred within the desired range of repetitions using proper form.

**Statistical analysis.** ANOVA with repeated measures was used to assess variables over three time points (Pre1 × Pre2 × Post). 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. All results are presented as means ± SE. Data analyses were carried out with the use of the Statistical Package for the Social Sciences (SPSS, v. 12.0.1; SPSS, Chicago, IL).

**RESULTS**

There was no change in αs following either the time control period or RT (1.01 ± 0.06 to 0.98 ± 0.06 to 0.93 ± 0.04, P > 0.05). However, given the bidirectional nature of fractal collapse, subjects were retrospectively divided into two groups: those subjects initially presenting with lower αs and those subjects initially presenting with higher αs, using K-means cluster analysis. Cluster analysis placed 18 men with lower αs in one group (0.73 ± 0.04) and 16 men with higher αs in a second group (1.2 ± 0.04, P < 0.05; Fig. 1). A 3 × 2 ANOVA with repeated measures was then used to assess variables over three time points (Pre1 × Pre2 × Post) in two groups (low αs).
× high \( \alpha_s \). As seen in Fig. 1, a group × time interaction was detected \( (P < 0.05) \), as there was a significant time effect in \( \alpha_s \) in the low-\( \alpha_s \) group \( (P < 0.05) \) concomitant with a significant time effect in \( \alpha_s \) in the high-\( \alpha_s \) group \( (P < 0.05) \).

Groups were not different in age (low \( \alpha_s = 23 ± 1 \) yr vs. high \( \alpha_s = 24 ± 1 \) yr, \( P > 0.05) \). Groups were also not different in baseline body weight, body fat, aerobic fitness, strength, WBC, or blood lipid profile (Table 1, Table 2, \( P > 0.05) \). There was no change in blood weight, body fat, aerobic fitness, WBC, or blood lipid profile following RT (Table 1, Table 2, \( P > 0.05) \). There was a similar increase in strength (absolute and relative) in both groups following RT (Table 1, \( P < 0.05) \). There was no change in mean R-R interval in low \( \alpha_s \) \((1,101.7 \pm 34.5 \) to \( 1,108.3 \pm 29.1 \) to \( 1,103.3 \pm 39.3, P > 0.05) \) or high \( \alpha_s \) \((960.9 ± 36.6 \) to 923.3 ± 30.8 to 992.6 ± 41.7, \( P > 0.05) \).

**DISCUSSION**

This is the first study to prospectively examine the effect of RT on fractal scaling of HR dynamics. The novel finding of the present investigation was that, when partitioning subjects based in initial scaling exponent, RT improved fractal scaling properties of HR in young men, irrespective of starting point. Whether HR dynamics approached Brownian noise or white noise at baseline, both suggestive of low fractal dynamics and less favorable cardiac health, HR fluctuations converged on pink noise following RT. This positive cardiac adaptation occurred despite no change in conventional risk factors such as blood lipids, WBC count, fasting glucose, cardiorespiratory fitness, or body fat.

One of the hallmark features of a fractal is its self-similar scaling; upon magnification with greater resolution, successive smaller subdivisions of the structure resemble the previously undivided structure (i.e., the parts resemble the whole). This property holds on multiple scales. Fractal geometric patterns are frequently found in human structure and function. Examples include branching of dendrites, bronchi, Purkinje fibers, DNA replication, respiration, and movement tremor (3, 4, 23, 29). The most well-investigated fractal process in cardiovascular physiology is oscillations in HR.

Lipsitz and Goldberger (15) proposed the “loss of complexity” hypothesis whereby disease and disability may stem from and/or contribute to a decrease in the complexity of physiological system output. In stark contrast to the traditional view of homeostasis as health, this tenet holds that living systems strive for homeokinesis and operate far from a set equilibrium state. Dynamic health reflects the ability of physiological systems to constantly adapt to internal and external stimuli via the interplay of numerous regulatory mechanisms operating across multiple time scales (14). Being in a chaotic state allows neural regulatory centers to dynamically seek out any one among a host of available periodic orbits in space and time and adequately switch between attractors when necessary (11). The breakdown of this scale-invariant organization in HR dynamics has been shown in numerous investigations to be associated with increased mortality risk and a predecessor to the development of life-threatening arrhythmias and sudden cardiac death (7, 18, 21). This fractal collapse can be bidirectional. In some instances, the presence of a single, dominant characteristic scale results in periodic and predictable behavior. This rigid mode locking does not allow for appropriate adaptation to changing stimuli and is evident with chronic heart failure (i.e., the Cheyne-Stokes frequency), sleep apnea, and psychological tremor (a value approximating 1.5 or Brownian noise) (3). On the opposite end of the gamut is a scale-free system. This system does not gravitate toward any orbit and produces physiological output resembling white noise. Examples include irregular ventricular activity seen during atrial fibrillation and stride interval variability in Huntington’s disease (a value approximating 0.5 or white noise) (3). Overall, a HR signal dominated by a single attractor originating from a strong neural oscillator would correspond to a high degree of regularity (i.e., low fractal organization). Conversely, a HR signal that arises from a network of multiple neural oscillators will be highly irregular (i.e., fractal).

Given the potential bidirectional response in fractal breakdown, analyzing all subjects as one group may not be appropriate. Indeed, when performing the analysis on the group as a whole.

### Table 1. Aerobic fitness, muscular strength, and body composition before and after RT

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre1</th>
<th>Pre2</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, kg</td>
<td>81.5 ± 3.8</td>
<td>82.0 ± 3.9</td>
<td>83.0 ± 3.9</td>
</tr>
<tr>
<td>High ( \alpha_s )</td>
<td>83.2 ± 3.7</td>
<td>83.2 ± 3.7</td>
<td>83.2 ± 3.7</td>
</tr>
<tr>
<td>Low ( \alpha_s )</td>
<td>19.9 ± 2.7</td>
<td>19.3 ± 2.8</td>
<td>19.5 ± 2.6</td>
</tr>
<tr>
<td>% Body fat</td>
<td>16.7 ± 2.5</td>
<td>17.3 ± 2.4</td>
<td>17.9 ± 2.5</td>
</tr>
<tr>
<td>1-RM bench press, kg</td>
<td>73.6 ± 4.9</td>
<td>73.9 ± 4.7</td>
<td>80.2 ± 4.4*</td>
</tr>
<tr>
<td>High ( \alpha_s )</td>
<td>85.1 ± 5.6</td>
<td>86.2 ± 5.6</td>
<td>91.3 ± 5.6*</td>
</tr>
<tr>
<td>Low ( \alpha_s )</td>
<td>0.93 ± 0.07</td>
<td>0.93 ± 0.07</td>
<td>0.99 ± 0.06*</td>
</tr>
<tr>
<td>VO2peak, ml·kg⁻¹·min⁻¹</td>
<td>1.04 ± 0.08</td>
<td>1.06 ± 0.08</td>
<td>1.12 ± 0.07*</td>
</tr>
<tr>
<td>High ( \alpha_s )</td>
<td>32.2 ± 1.4</td>
<td>32.8 ± 1.6</td>
<td>32.7 ± 1.5</td>
</tr>
<tr>
<td>Low ( \alpha_s )</td>
<td>31.3 ± 1.3</td>
<td>31.8 ± 1.5</td>
<td>31.8 ± 1.6</td>
</tr>
</tbody>
</table>

Values are means ± SE. RT, resistance exercise training; 1-RM, one-repetition maximum; Pre1, taken at baseline; Pre2, taken after a 4-wk time control period; taken following a 6-wk RT intervention; \( \alpha_s \), short-term scaling component. *Significant time effect \( (P < 0.05) \).
whole, there was a wash in overall results and no change in $\alpha_s$ following RT was detected. Therefore, we retrospectively separated subjects into two groups on the basis of the initial scaling exponent. Upon doing this, very interesting results were seen. In those young men with HR dynamics initially approximating uncorrelated randomness or white noise (i.e., lower $\alpha_s$), HR dynamics converged on pink noise following RT. Similarly, in those young men with HR dynamics initially approximating a random walk or Brownian noise (i.e., higher $\alpha_s$), HR dynamics also converged on pink noise following RT. Thus, in both groups, HR dynamics became more fractal upon completion of the exercise intervention. Alterations in $\alpha_s$ in the present investigation occurred independent of changes in cardiorespiratory fitness, blood lipids, glucose, body fat, and WBC count, implying that improvements in fractal properties were not secondary to improvements in general cardiovascular health.

Despite the clear clinical implications, there is little information on interventions aimed at improving fractal properties of HR. Lin et al. (13) have shown that short-term beta-blocker therapy (3 mo) increased $\alpha_s$ in patients with congestive heart failure initially presenting with low values. Similarly, much more invasive procedures have also shown temporary utility. Laitio et al. (12) have shown that $\alpha_s$ is reduced to values approximating 1.0 6 wk after coronary artery bypass graft surgery. However, values rebound to preoperative baseline values of 1.22 6 mo following surgery. Finally, Tulppo et al. (32) have shown that aerobic exercise training can reduce $\alpha_s$ in young healthy men with high baseline $\alpha_s$. Our findings add to these previous interventions, and note that another nonpharmacological/noninvasive approach, RT, improves fractal scaling of HR dynamics.

Improvements in fractal properties of HR dynamics following RT may be mediated through the autonomic nervous system (ANS). Sympathoexcitation via infusion of norepinephrine has been shown to reduce $\alpha_s$ in a dose-responsive manner (34), whereas sympathetic blockade does little to effect fractal HR dynamics (37). Conversely, vagal blockade with glycopyrrole or high dose atropine increases $\alpha_s$, whereas vagal stimulation with low dose atropine has negligible effects on $\alpha_s$ (24, 25, 34). Conditions associated with simultaneous sympathetic and parasympathetic activation have also been shown to reduce $\alpha_s$ (33). Findings from these studies suggest that loss of parasympathetic and/or increased sympathetic modulation will result in fractal collapse and movement of HR dynamics toward either white noise or Brownian noise. Paradoxically, vagal stimulation or sympathetic blockade is insufficient in altering fractal HR dynamics. It would appear that for healthy fractal HR dynamics (i.e., a $\alpha_s$ value of 1.0), a sympat-ho-vagal accord must be stricken, and a precise admixture of both branches of the ANS is required (30).

In conclusion, RT improves fractal scaling properties of HR dynamics in young men. Future research is warranted to determine whether improving fractal properties via RT can improve clinical outcome in select patient populations.

ACKNOWLEDGMENTS

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