Eur J Appl Physiol (2005) 93: 719 725
DOI 10.1007/s00421 004 1243 x

ORIGINAL ARTICLE

William H. Cooke · Jason R. Carter

Strength training does not affect vagal–cardiac control or cardiovagal baroreflex sensitivity in young healthy subjects

Accepted: 19 September 2004 / Published online: 29 October 2004

Abstract The purpose of this study was to test the hypothesis that high-intensity strength training increases vagal–cardiac control and cardiovagal baroreflex sensitivity. Twenty-two subjects [age 22 (0.8) years] were assigned to either strength training (ST, $n = 12$) or control (CON, $n = 10$) groups. Subjects in the ST group trained each major muscle group 3 days per week for 8 weeks with three sets of 10 repetitions and 2-min rest periods. Resistance was increased progressively when possible. Subjects in the CON group were active recreationally but did not lift weights. Before and after training, resting arterial pressure was measured with an automated sphygmomanometer, and ECG, respiratory rate, and finger photoplethysmographic arterial pressures were recorded with subjects supine and breathing at a set rate (0.25 Hz) for 5 min. Data were analyzed in both time and frequency domains. Arterial baroreflex sensitivity was estimated with the sequence method and cross-spectral transfer function analysis of systolic pressures and R-R intervals. Training increased whole-body muscular strength and decreased arterial blood pressure at rest (the exercise and pressure data have been published elsewhere). R-R intervals and standard deviations, and R-R interval spectral power at the respiratory frequency were unaffected by training. Similarly, training did not affect respiratory or low-frequency systolic pressure spectral power or cardiovagal baroreflex sensitivity. Although evidence supports beneficial cardiovascular adaptations to resistance training, our results demonstrate that resistance training does not affect vagal–cardiac control or cardiovagal baroreflex sensitivity in young healthy subjects.

Keywords Weight training · Arterial baroreflex · Heart rate variability · Frequency domain analysis

The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army of the Department of Defense.

W. H. Cooke (✉)
U.S. Army Institute of Surgical Research,
3400 Rawley E. Chambers Ave. Bld. 3611,
Fort Sam Houston, Texas 78234 6315, USA
E mail: william.cooke@amedd.army.mil
Tel.: +1 210 9161972
Fax: +1 210 9165992

W. H. Cooke · J. R. Carter
Department of Biological Sciences,
Michigan Technological University, Houghton,
Michigan 49931, USA

Introduction

Whole-body strength training increases muscular strength and power, helps maintain skeletal mass and density, and contributes to maintenance of normal body weight (Kraemer et al. 2002). In addition to performance benefits for athletes and recreational sports enthusiasts, strength training has been shown to help control or lower arterial blood pressure (Carter JR et al. 2003; Kelley and Kelley 2000), and has been recommended as therapy for patients with heart disease (McCartney 1998). Although strength training may also affect other factors associated with cardiovascular health (Hurley et al. 1988), some important associations have not been studied.

For example, elevated vagal autonomic neural activity is associated with better prognosis and lower mortality associated with cardiovascular disease (Kleiger et al. 1987). Chronic endurance exercise training has been shown to increase vagal–cardiac control (Carter JB et al. 2003) and cardiovagal baroreflex sensitivity (Iwasaki et al. 2003), and limit reductions of vagal control and baroreflex sensitivity associated with aging (Monahan et al. 2000). Less is known of the effects of strength training on autonomic cardiovascular regulation. Tatro et al. (1992) reported that high-intensity lower-body strength training

Report Documentation Page

Form Approved
OMB No. 0704-0188

1. REPORT DATE	2. REPORT TYPE	3. DATES COVERED
01 MAR 2005	**N/A**	**-**

4. TITLE AND SUBTITLE	5a. CONTRACT NUMBER
Strength training does not affect vagal-cardiac control or cardiovagal baroreflex sensitivity in young healthy subjects	5b. GRANT NUMBER
	5c. PROGRAM ELEMENT NUMBER
6. AUTHOR(S)	5d. PROJECT NUMBER
Cooke W. H., Carter J. R.,	5e. TASK NUMBER
	5f. WORK UNIT NUMBER
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)	8. PERFORMING ORGANIZATION REPORT NUMBER
United States Army Institute of Surgical Research, JBSA Fort Sam Houston, TX 78234	
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)	10. SPONSOR/MONITOR'S ACRONYM(S)
	11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for public release, distribution unlimited

13. SUPPLEMENTARY NOTES

14. ABSTRACT

15. SUBJECT TERMS

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a REPORT	b ABSTRACT	c THIS PAGE			
unclassified	**unclassified**	**unclassified**	**SAR**	**8**	

increased carotid–cardiac baroreflex sensitivity, and one recent study demonstrated that isometric handgrip training increased heart rate variability in elderly patients with hypertension (Taylor et al. 2003). However, whole-body strength training programs are recommended as an important component of a holistic fitness regime (Kraemer et al. 2002), and longitudinal studies investigating the influence of whole-body strength training on autonomic cardiovascular regulatory mechanisms do not exist.

We have shown previously that whole-body strength training has no effect on resting sympathetic neural traffic (Carter JR et al. 2003), but we have not explored the effects of whole-body strength training on autonomic vagal activity. Because strength training is associated with reduction of cardiovascular disease risk factors (Hurley et al. 1988; Kelley and Kelley 2000), and because reduced risk factors are associated with elevated autonomic vagal activity, we tested the hypothesis that strength training increases vagal–cardiac control and cardiovagal baroreflex sensitivity.

Methods

Subjects

A total of 22 subjects participated in this study. Based on pilot work with similar outcome variables and assuming comparable variances (Cooke et al. 2002), we estimated a sample size of nine subjects per group as being the minimum number of subjects required to test our hypothesis ($1-\beta=0.8$, $\alpha=0.05$). Prior to inclusion, all potential subjects were screened and excluded if they were currently participating in, or had within the last month participated in, a structured weight-training program. All subjects were normotensive nonsmokers with no prior history of autonomic dysfunction. In addition, subjects were certified as apparently healthy and free from contraindications to exercise training, as determined by a physician during a routine physical examination. The experimental protocol was approved by the Human Research Committee of Michigan Technological University and all subjects gave written informed consent before participating. The experiments outlined in this report comply with the current laws of the United States of America.

Due to the large number of subjects studied, and due to time constraints of both subjects and investigators, subjects were enrolled on a staggered basis, with only five to eight subjects being studied during any given 8-week period over the course of approximately 1 year. For this reason, the experimental design was not strictly randomized (i.e., subjects were not all recruited prior to the beginning of the study). Subject characteristics are shown in Table 1.

Table 1 Subject characteristics. Values are means (SE). *ST* Strength training group ($n=12$; 11 men, 1 woman), *CON* control group ($n=10$; 7 men, 3 women). Subject characteristics did not differ statistically between groups

Group	Age (years)	Height (cm)	Body mass (kg)
ST	21 (0.3)	180 (2.0)	82 (4)
CON	22 (0.7)	176 (4.3)	78 (6.0)

Exercise testing

The strength training group (ST) comprised subjects required to perform resistance training specifically designed to increase whole-body muscular strength, and the control group (CON) comprised subjects who were active recreationally but did not follow a structured exercise program during the study period. Subjects from both groups reported to the exercise facility (Portage Wellness Center, Houghton, Mich.) at least 1 h postprandial. Maximal whole-body muscular strength was assessed before and after 8 weeks of heavy resistance strength training or 8 weeks of relative inactivity using the following seven isotonic exercises in the order listed (with the primary muscle groups trained shown in parentheses): leg press (quadriceps), leg curls (hamstrings), chest press (pectoralis major and minor, deltoid, triceps), latissimus pull downs (trapezius, latissimus dorsi, rear deltoid), shoulder press (deltoid), bicep curls (biceps), and triceps press (triceps) (Cybex Strength Systems, Medway, Mass, and Ground Zero, Colorado Springs, Colo.). Subjects first completed ten warm-up repetitions, followed by two or three additional sets with increasing resistance until only four to six repetitions could be completed with proper exercise form. One-repetition maximum (1 RM) values were calculated from the last set according to the following equation (Fleck and Kraemer, 1997): 1 RM = weight lifted/[0.95−(number of repetitions−2)(0.025)]. Proper exercise form was taught and enforced by personal trainers who supervised and documented each testing session.

Autonomic testing

Subjects arrived at the laboratory after having abstained from exercise, alcohol, and caffeine for at least 12 h. All tests were done with subjects in the supine position. After resting blood pressures were measured with an automated sphygmomanometer, subjects were instrumented with a three-lead ECG, a finger photoplethysmography cuff (model 2300, Finapres, Ohmeda, Englewood, Colo.) to record beat-by-beat arterial pressure, and a pneumobelt to record respiratory excursions (uncalibrated strain-gauge pressure transducer) [we also measured muscle sympathetic nerve activity (MSNA) by inserting a tungsten microelectrode (Frederick Haer, Bowdingam, ME) into the peroneal nerve in the popliteal region behind the left knee. These data have been published (Carter JR et al. 2003)]. After instrumenta-

tion, subjects were undisturbed for 5 min and were allowed to breathe spontaneously. Subjects then were instructed to control their breathing frequency by breathing in time to a metronome set at a pace of 15 breaths min^{-1} (0.25 Hz) for 5 min. The autonomic function test was repeated after either 8 weeks of strength training (ST) or relative inactivity (CON).

Exercise training

We used both single- and multiple-joint exercises, starting with large muscle groups and finishing with smaller muscle groups, incorporating repetitions and rest intervals conducive to increasing whole-body muscular strength. The subjects assigned to ST underwent three supervised resistance-training sessions per week for 8 weeks. Each subject performed three sets of the same exercises in the same order: leg press, leg curls, chest press, latissimus pulldown, shoulder press, biceps curls, and triceps press. Subjects performed ten repetitions during sets one and two, and as many repetitions as possible during the third set with resistance equaling approximately 75–80% 1 RM. Resistance was increased for the next exercise session if the subject could perform ten complete repetitions during the third and final set for each exercise. Subjects assigned to CON were asked to maintain their normal recreational activity and not to add any form of regular exercise training during the 8-week study period.

Data acquisition and time domain analysis

During the autonomic function tests, data were sampled at 500 Hz and recorded directly to computer with commercial hardware and software (WINDAQ, Dataq Instruments, Akron, Ohio). Data then were imported into a commercial analysis program (WinCPRS, Absolute Aliens, Turku, Finland). R waves generated from the ECG signal were detected and marked at their occurrence in time. Systolic pressures were subsequently marked from the arterial pressure waveform. R-R intervals and R-R interval standard deviations were calculated from the 5-min period of controlled breathing. Within this same time range, we analyzed sequences of three or more increasing systolic pressures (increasing by at least 1 mmHg) and R-R intervals (increasing by at least 4 ms) with linear regression analysis. Only sequences with correlation coefficients of at least 0.8 were included for statistical comparison (Rothlisberger et al. 2003).

Frequency domain analysis

R-R intervals and systolic pressures were made equidistant by spline interpolating and resampling at 5 Hz. Data were then passed through a low-pass impulse response filter with a cutoff frequency of 0.5 Hz. Five-minute data sets were fast Fourier transformed with a Hanning window to obtain power spectrums. Spectral power was expressed as the integrated areas within the low-frequency (0.05–0.15 Hz) and high-frequency (0.15–0.4 Hz) bands. In order to compare power spectra more accurately from different individuals who may vary widely in total power, we also normalized our data in the low- and high-frequency ranges by dividing integrated low- and high-frequency spectra by the total power (minus oscillations occurring below 0.05 Hz) and then multiplying this value by 100.

The squared coherence reveals the strength of the linear association between two signals, and we calculated the squared coherence by dividing the cross-spectral densities of systolic pressure and R-R interval by the product of each individual autospectrum. If the squared coherence value was at least 0.5 at frequencies between 0.05 and 0.15 Hz, then we considered the transfer function magnitude to be an accurate representation of arterial baroreflex sensitivity within these coherent frequency ranges. Transfer function magnitude was calculated by dividing the cross-spectrum of systolic pressure and R-R interval by the autospectrum of systolic pressure (Cooke et al. 1999).

Statistical analysis

A two by two [group (ST vs CON) by time (before vs after the 8-week study period)] ANOVA with repeated measurements on time was used to compare dependent variables. We tested for differences in subject characteristics with paired *t*-tests. Data were analyzed with commercially available statistical software (SAS Institute, Cary, N.C.). Means were considered to be significantly different if $P \leq 0.05$. Data are presented as means (SE) unless otherwise specified.

Results

Exercise responses

The strength training program was successfully completed by all participants. Due to holiday interruptions during the 8-week study period, six subjects completed the required 24 training sessions within 9 weeks. During the week subjects were unavailable for training, they performed the required exercise to the best of their ability with machines that were matched as closely as possible, but these sessions were not monitored or recorded.

We found that the resistance training program increased whole-body muscular strength (by about 22%) and decreased arterial pressure (by about 8 mmHg) at rest. These data were published in a previous report (Carter JR et al. 2003). For our prior publication (Carter JR et al. 2003), five subjects from the control group

($n = 13$) were studied prospectively and did not perform controlled breathing with appropriate instrumentation. For this reason, and for purposes of the present study, we recruited an additional two subjects for the CON group to increase our sample of control subjects performing the autonomic function test from eight to ten.

Autonomic responses

For both ST and CON, R-R intervals averaged approximately 900 ms, R-R interval standard deviations averaged approximately 61 ms, and these values were not significantly different between groups or changed over the course of the 8-week study period (Table 2). Heart rates were similarly unaffected for ST [65 (3) pre- vs 63 (2) beats min^{-1} post-training] and CON [66 (4) pre- vs 65 (4) beats min^{-1} post-training]. Power spectral and cross-spectral analyses revealed oscillatory components of R-R intervals, systolic arterial pressures, and systolic pressure and R-R interval associations. R-R interval and systolic pressure spectral power, and coherence and transfer function magnitude between systolic pressure and R-R interval are shown in Fig. 1; in the subject depicted (and all other subjects), breathing at a controlled rate of 0.25 Hz effectively separated respiratory (high) from lower frequency oscillations. Strong coherence (i.e., above 0.5) was apparent between the two signals at both high and low frequencies for all subjects.

As shown in Table 2, strength training did not significantly alter the magnitude of high- or low-frequency R-R interval or systolic pressure oscillations regardless of whether data were presented as absolute values or as normalized, transformed data. Prior to exercise training, arterial baroreflex sensitivity (assessed from systolic pressure and R-R interval sequences and low frequency transfer function magnitude) averaged approximately 15 ms $mmHg^{-1}$. After training, baroreflex sensitivity averaged 16 ms $mmHg^{-1}$ and the magnitude of change was not different between groups or between analysis techniques.

Discussion

We investigated the influence of strength training on autonomic cardiovascular control. Although this particular strength-training program increases whole-body muscular strength and lowers arterial blood pressure (Carter JR et al. 2003), it does not increase autonomic vagal activity. These results are in contrast to those reported for traditional endurance training programs.

Autonomic variability

Athletes in general, and endurance athletes in particular, display low resting heart rates. This resting bradycardia has been attributed to elevated vagal neural activity (Ekblom et al. 1973) and small reductions of intrinsic heart rates (Smith et al. 1989). R-R intervals change as direct linear functions of vagal–cardiac nerve activity (Katona and Jih 1975), and oscillations of R-R intervals at the respiratory frequency (respiratory sinus arrhythmia) are mediated predominantly by fluctuations of vagal–cardiac nerve traffic. Because direct measurements of vagal nerve traffic have not been made in humans, investigators have relied on time and frequency-domain measures of heart rate variability to estimate vagal activity noninvasively. Resting heart rates were lower after 8 weeks of lower-body strength training (Kanakis

Table 2 Autonomic cardiovascular responses to whole body strength training. Values are means (SE) (ST, $n = 12$; CON, $n = 10$). *RRI*, R wave to R wave, *RRISD* R wave to R wave standard deviation, *BRS* baroreflex sensitivity sequence method, *RRILF* R R interval spectral power at the low frequency, *RRIHF* R R interval spectral power at the high frequency, *RRILFnu* normalized units for R R interval spectral power at the low frequency, *RRIHFnu* normalized units for R R interval spectral power at the high frequency, *SAPLF* systolic pressure spectral power at the low frequency, *SAPHF* systolic pressure spectral power at the high frequency, *SAPLFnu* normalized units for systolic pressure spectral power at the low frequency, *SAPHFnu* normalized units for systolic pressure spectral power at the high frequency, *TF* transfer function magnitude between systolic pressure and R R interval. All *P* values for main and interaction effects were > 0.05

Variable	ST		CON	
	Pre training	Post training	Pre training	Post training
RRI (ms)	902 (32)	915 (57)	896 (35)	917 (29)
RRISD (ms)	59 (9.5)	63 (9.8)	62 (15)	64 (12)
BRS (ms $mmHg^{-1}$)	15 (1.2)	17 (3.3)	14 (2.9)	14 (3.1)
RRILF (ms^2)	852 (342)	891 (243)	875 (223)	902 (301)
RRIHF (ms^2)	1,338 (346)	1,460 (398)	1,412 (340)	1,397 (305)
RRILFnu (ms^2)	47 (6.9)	48 (6.9)	44 (3.6)	46 (6.7)
RRIHFnu (ms^2)	52 (6.9)	53 (7.1)	59 (5.6)	55 (5.9)
SAPLF ($mmHg^2$)	3.9 (3.2)	4.9 (1.3)	5.4 (1.7)	5.2 (2.3)
SAPHF ($mmHg^2$)	3.4 (0.9)	4.1 (0.9)	3.4 (2.1)	3.7 (1.4)
SAPLFnu ($mmHg^2$)	48 (4.9)	46 (6.3)	45 (4.4)	50 (9.5)
SAPHFnu ($mmHg^2$)	52 (4.9)	53 (6.3)	54 (5.2)	54 (9.1)
TF (ms $mmHg^{-1}$)	16 (2.9)	18 (3.8)	16 (3.6)	17 (4.9)

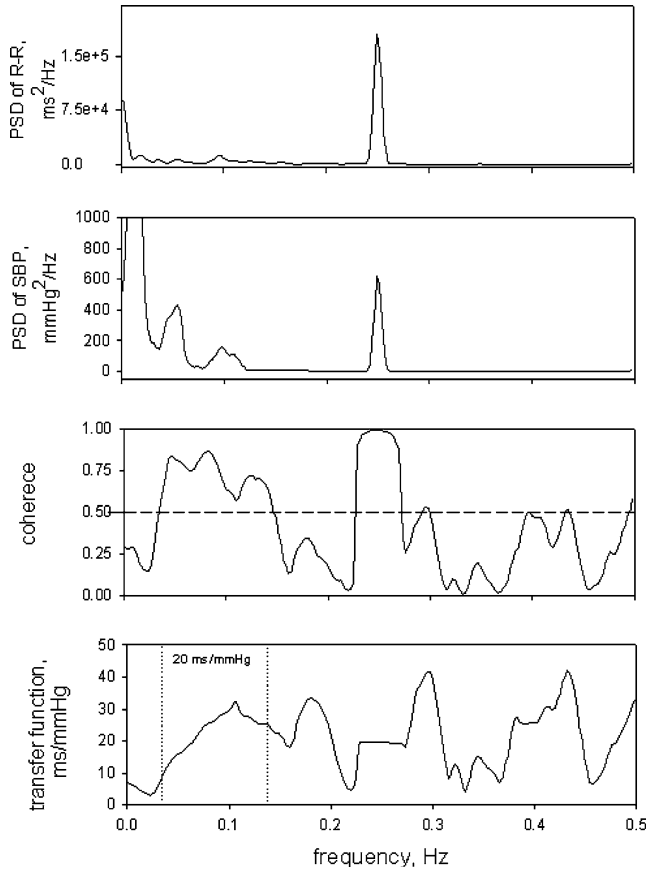

Fig. 1 Power spectral density (*PSD*) representation of R wave to R wave interval (*R R*) and systolic pressure (*SBP*) is shown with coherence and transfer function calculations. The *horizontal dashed line* in the coherence window delineates frequency ranges where systolic pressures and R R intervals showed significant coherence as defined by values above 0.5. The *vertical dotted lines* in the transfer function window encompass coherent values within the low frequency band (0.05 0.15 Hz) that are averaged for statistical comparison. This analysis revealed a cardiovagal baroreflex sensitivity of 20 ms $mmHg^{-1}$ for the subject depicted

and Hickson 1980) and Olympic-style weight lifting (Stone et al. 1983), but heart rate variability was not specifically assessed in these two studies (Kanakis and Hickson 1980; Stone et al. 1983). In the present study, R-R intervals averaged about 900 ms and R-R interval standard deviations (a global measure of heart rate variability) averaged about 61 ms; these values were not affected by strength training (Table 2). Integrated R-R interval spectral power at the high frequency similarly was unaffected by strength training, suggesting that in contrast to endurance training (Carter JB et al. 2003), strength training does not affect the overall magnitude of vagal–cardiac control at rest.

Arterial pressures oscillate at the respiratory frequency primarily through mechanical influences of respiration (Badra et al. 2001), and we did not expect changes in high frequency arterial pressure spectral power. On the other hand, arterial pressure oscillations at the low-frequency conceivably could be affected by strength training, although these associations have not been reported. At least two general hypotheses have been advanced to explain low-frequency arterial pressure rhythms. First, evidence suggests the presence of a central pacemaker (Guzzetti et al. 1994) or intrinsic vasomotor mechanisms (Rizzoni et al. 1995) that modulates arterial pressure rhythms independent of autonomic control loops. Second, low-frequency arterial pressure oscillations have been linked to sympathetic vascular responses to arterial baroreceptor stimulation and inhibition (Guyton and Harris 1951).

As to the first hypothesis, we are unaware of any studies (including the present study) that have investigated the influence of resistance training on arterial pressure oscillations initiated and modulated by the spinal cord or the vasculature. As to the second hypothesis, increases and decreases in sympathetic traffic have been linked directly to increases and decreases in low-frequency systolic pressure oscillations (Malliani 1999), supporting the original suggestion of Guyton and Harris (1951) that 'vasomotor waves' are mediated (at least in part) by sympathetic baroreflex responses. In the present study, strength training did not affect low-frequency systolic pressure spectral power, which suggests that strength training does not affect sympathetic vascular control [these results are in agreement with our observation that muscle sympathetic neural activity was unchanged in these subjects after the 8-week program (Carter JR et al. 2003)]. We conclude that resistance training does not affect the magnitude of vasomotor waves, and speculate that this is due to unchanged resting sympathetic tone.

Cardiovagal baroreflex sensitivity

After Stegemann et al. (1974) first reported impaired arterial pressure control mechanisms in exercise-trained subjects, the notion that endurance exercise training decreases arterial baroreflex sensitivity and compromises orthostatic stability has been both supported (Raven and Pawelczyk 1993) and refuted (Convertino 1993). Both interpretations are probably correct, in that highly trained athletes with aerobic capacities of approximately 65 ml kg^{-1} min^{-1} and above may be susceptible to orthostatic intolerance (Levine et al. 1991), and recreational athletes with 'normal' aerobic capacities of approximately 40–50 ml kg^{-1} min^{-1} may improve tolerance to orthostatic stress with exercise (Convertino et al. 1990). The effects of resistance training on arterial pressure control mechanisms are less clear, but some evidence suggests that resistance athletes may possess greater orthostatic stability due to decreased leg compliance, increased blood volumes, and/or increased carotid–cardiac baroreflex sensitivity (Convertino 1993).

Using a cross-sectional experimental design, Smith et al. (1988) infused phenylephrine to study integrated arterial baroreflex sensitivity and found that slopes

relating mean arterial pressures and heart rates were similar in resistance-trained and untrained subjects. Unfortunately, cross-sectional studies do not reveal training-specific adaptations. Tatro et al. (1992) used neck pressure and suction to study carotid–cardiac baroreflex responses before and after a 12-week program of intense lower-body resistance training. Training lowered arterial blood pressure and increased baroreflex sensitivity by about 20% (Tatro et al. 1992). Others have reported no change in carotid–cardiac baroreflex sensitivity after 12 weeks of whole-body resistance training (Lightfoot et al. 1994). Ours is the first longitudinal study to investigate integrated (cardiopulmonary, aortic, and carotid) cardiovagal baroreflex sensitivity before and after a program of whole-body strength training. Both the sequence method and transfer function analysis revealed that 8 weeks of whole-body strength training does not alter cardiovagal baroreflex sensitivity.

The present study differs from that of Tatro et al. (1992) in at least two important ways. First, Tatro et al. (1992) used the neck pressure and suction technique to test responsiveness specifically of the carotid–cardiac baroreflex and we did not attempt to isolate specifically carotid baroreceptors. Second, the neck suction technique provides stimuli over a wide range of carotid distending pressures (i.e., from ~75 to 160 mmHg), whereas the sequence and transfer function methods analyze dynamic baroreflexes within ranges of naturally occurring arterial pressure rhythms. However, our results are similar conceptually to those presented by Tatro et al. (1992) in at least one respect. Tatro et al. (1992) reported a leftward shift on the response relation describing changes of R-R intervals as functions of changes in estimated carotid distending pressures, showing that carotid baroreflex responses were reset to lower arterial pressures after resistance training. In that study (Tatro et al. 1992), strength training lowered systolic blood pressure by about 6 mmHg, which is consistent with the reduction of about 9 mmHg previously reported for our subjects (Carter JR et al. 2003). Reduction of resting blood pressure with no change of resting R-R interval in the present study suggests that resistance training may reset cardiovagal baroreflexes to lower operating pressures without necessarily changing the sensitivity of the arterial pressure/R-R interval stimulus response relationship.

Limitations

The results of the present study should be considered in light of several limitations. First, during subject recruitment, it was difficult to obtain equal numbers of males and females willing to participate in high-intensity strength training. This is a concern given that autonomic regulatory mechanisms may differ in men and women at rest (Evans et al. 2001) or during hemodynamic challenges (Convertino 1998). However, we are unaware of any data suggesting that such differences between males and females are either negated or enhanced with exercise training. Second, we made no effort to classify or describe our subjects based on their baseline aerobic fitness levels. Although we screened our subjects and stressed the importance that they be active recreationally but not highly trained, it is possible that some subjects from both ST and CON groups may have been aerobically fit, and therefore may have differed in their baseline autonomic responses or their autonomic responses to exercise training (Iwasaki et al. 2003).

Summary

Cardiovascular adaptations with aerobic endurance exercise training are associated with decreased cardiovascular risk factors, and reduced risk factors are associated with elevated autonomic vagal activity. Resistance training also imparts cardiovascular benefits, but the effects of whole-body resistance training on autonomic control mechanisms have not been studied thoroughly. Our results show that high-intensity strength training resulting in significant increases in whole-body muscular strength and decreases in arterial blood pressure (Carter JR et al. 2003) has no effect on vagal–cardiac control or cardiovagal baroreflex sensitivity in young healthy subjects. However, our results, taken together with data presented elsewhere (Tatro et al. 1992), and in combination with data presented in our previous report (Carter JR et al. 2003), support the suggestion that strength training resets cardiovagal baroreflex responses to lower arterial pressures without affecting reflex sensitivity.

Acknowledgements The authors greatly appreciate the hard work and dedication exhibited by all of the subjects who volunteered to participate in this study. Additional thanks are extended to the students who contributed to the success of this study by assisting in the laboratory and serving as personal trainers in the gym: Brooke Anderson, Scott Anderson, Matt Barron, Tanya Brabec, Emily Downs, John Durocher, Mark Jentoft, Tiffany Karkanen, Nathan Leatherman, Sara Mantila, Nick Smith, Aaron Tarnow, Jarett Weinberger, and Mike Yandl. This study was supported by a grant from the American Heart Association (0030203 N to W.H. Cooke).

References

Badra LJ, Cooke WH, Hoag JB, Crossman A, Kuusela T, Tah vanainen K, Eckberg DL (2001) Respiratory modulation of human autonomic rhythms. Am J Physiol 280:H2674 H2688

Carter JB, Bannister EW, Blaber AP (2003) Effect of endurance exercise on autonomic control of heart rate. Sports Med 33:33 46

Carter JR, Ray CA, Downs EM, Cooke WH (2003) Strength training reduces arterial blood pressure but not sympathetic neural activity in young normotensive subjects. J Appl Physiol 94:2212 2216

Convertino VA (1993) Endurance exercise training: conditions of enhanced hemodynamic responses and tolerance to LBNP. Med Sci Sports Exerc 25:705 712

Convertino VA (1998) Gender differences in autonomic functions associated with blood pressure regulation. Am J Physiol 275:R1909 R1920

Convertino VA, Thompson CA, Eckberg DL, Fritsch JM, Mack GW, Nadel ER (1990) Baroreflex responses and LBNP toler ance following exercise training. Physiologist 33:S40 S41

Cooke WH, Hoag JB, Crossman AA, Kuusela TA, Tahvanainen KUO, Eckberg DL (1999) Human responses to upright tilt: a window on central autonomic integration. J Physiol (Lond) 517:617 628

Cooke WH, Reynolds BV, Yandl MG, Carter JR, Tahvanainen KUO, Kuusela TA (2002) Effects of exercise training on car diovagal and sympathetic responses to Valsalva's maneuver. Med Sci Sports Exerc 34:928 935

Ekblom B, Kilbom ASA, Soltysiak J (1973) Physical training, bradycardia and autonomic nervous system. Scand J Clin Lab Invest 32:251 256

Evans JM, Ziegler MG, Patwardhan AR, Ott JB, Kim CS, Leonelli FM, Knapp CF (2001) Gender differences in autonomic car diovascular regulation: spectral, hormonal, and hemodynamic indexes. J Appl Physiol 91:2611 2618

Fleck SJ, Kraemer WJ (1997) Designing resistance training pro grams. Human Kinetics, Champaign, Ill, pp 62 65

Guyton AC, Harris JW (1951) Pressoreceptor autonomic oscilla tion: a probable cause of vasomotor waves. Am J Physiol 165:158 166

Guzzetti S, Cogliati C, Broggi C, Carozzi C, Caldiroli D, Lombardi F, Malliani A (1994) Influences of neural mechanisms on heart period and arterial pressure variabilities in quadriplegic pa tients. Am J Physiol 266:H1112 H1120

Hurley BF, Hagberg JM, Goldberg AP, Seals DR, Ehsani AA, Brennan RE, Holloszy JO (1988) Resistive training can reduce coronary risk factors without altering $\dot{V}O_{2max}$ or percent body fat. Med Sci Sports Exerc 20:150 154

Iwasaki K, Zhang R, Zuckerman JH, Levine BD (2003) Dose response relationship of the cardiovascular adaptation to endurance training in healthy adults: how much training for what benefit? J Appl Physiol 95:1575 1583

Kanakis C, Hickson RC (1980) Left ventricular responses to a program of lower limb strength training. Chest 78:618 621

Katona PG, Jih F (1975) Respiratory sinus arrhythmia: noninva sive measure of parasympathetic cardiac control. J Appl Physiol 39:801 805

Kelley GA, Kelley KS (2000) Progressive resistance exercise and resting blood pressure: A meta analysis of randomized con trolled trials. Hypertension 35:838 843

Kleiger RE, Miller JP, Bigger JT, Moss AJ (1987) Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. Am J Cardiol 59:256 262

Kraemer WJ, Adams K, Cafarelli E, Dudley GA, Dooly C, Fei genbaum MS, Fleck SJ, Franklin B, Fry AC, Hoffman JR, Newton RU, Potteiger J, Stone MH, Ratamess NA, Triplett McBride T, American College of Sports Medicine (2002) American College of Sports Medicine position stand. Progres sion models in resistance training for healthy adults. Med Sci Sports Exerc 34:364 380

Levine BD, Buckey JC, Fritsch JM, Yancy CW, Watenpaugh DE, Snell PG, Lane LD, Eckberg DL (1991) Physical fitness and cardiovascular regulation: mechanisms of orthostatic intoler ance. J Appl Physiol 70:112 122

Lightfoot TJ, Torok DJ, Journell TW, Turner MJ, Claytor RP (1994) Resistance training increases lower body negative pres sure tolerance. Med Sci Sports Exerc 26:1003 1011

Malliani A (1999) The pattern of sympathovagal balance explored in the frequency domain. News Physiol Sci 14:111 117

McCartney N (1998). Role of resistance training in heart disease. Med Sci Sports Exerc 10(Suppl):S396 S402

Monahan KD, Dinenno FA, Tanaka H, Clevenger CM, DeSouza CA, Seals DR (2000) Regular aerobic exercise modulates age associated declines in cardiovagal baroreflex sensitivity in healthy men. J Physiol (Lond) 529:263 271

Raven PB, Pawelczyk JA (1993) Chronic endurance exercise training: a condition of inadequate blood pressure regulation and reduced tolerance to LBNP. Med Sci Sports Exerc 25:713 721

Rizzoni D, Castellano M, Porteri E, Bettoni G, Muiesan P, Mui esan ML, Giulini SM, Cinelli A, Salvetti M, Agabiti Roseri E (1995) Arterial spontaneous rhythmic contractile activity in humans and rats: spectral analysis and regulatory mechanisms. J Hypertens 13:1043 1052

Rothlisberger BW, Badra LJ, Hoag JB, Cooke WH, Kuusela TA, Tahvanainen KUO, Eckberg DL (2003) Spontaneous 'barore flex sequences' occur as deterministic functions of breathing phase. Clin Physiol Funct Imaging 23:307 313

Smith ML, Graitzer HM, Hudson DL, Raven PB (1988) Baroreflex function in endurance and static exercise trained men. J Appl Physiol 62:585 591

Smith ML, Hudson DL, Graitzer HM, Raven PB (1989) Exercise training bradycardia: the role of autonomic balance. Med Sci Sports Exerc 21:40 44

Stegemann J, Busert A, Brock D (1974) Influence of fitness on the blood pressure control system in man. Aerospace Med 45:45 48

Stone MH, Wilson GD, Blessing D, Rozenek R (1983) Cardio vascular responses to short term Olympic style weight training in young men. Can J Appl Sport Sci 8:134 139

Tatro DL, Dudley GA, Convertino VA (1992) Carotid cardiac baroreflex response and LBNP tolerance following resistance training. Med Sci Sports Exerc 24:789 796

Taylor AC, McCartney N, Kamath MV, Wiley RL (2003) Iso metric training lowers resting blood pressure and modulates autonomic control. Med Sci Sports Exerc 35:251 256