ELSEVIER

International Journal of Psychophysiology 37 (2000) 121–133

INTERNATIONAL JOURNAL OF PSYCHOPHYSIOLOGY

www.elsevier.nl/locate/ijpsycho

Heart rate variability, trait anxiety, and perceived stress among physically fit men and women

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Received 20 July 1999; received in revised form 14 November 1999; accepted 16 November 1999

Abstract

Background: It is unclear from prior reports whether the relationships between self-ratings of anxiety or emotional stress and parasympathetic nervous system components of heart rate variability are independent of personality and cardiorespiratory fitness. We examined those relationships in a clinical setting prior to a standardized exercise test. *Methods and results:* Heart rate variability (HRV) was measured during 5 min of supine rest among 92 healthy men ($N = 52$) and women ($N = 40$) who had above-average cardiorespiratory fitness as indicated by peak oxygen uptake measured during grade-incremented treadmill exercise. HRV datasets were decomposed into low-frequency (LF; 0.05–0.15 Hz) and high-frequency (HF; 0.15–0.5 Hz) components using spectral analysis. Self-ratings of trait anxiety and perceived emotional stress during the past week were also assessed. *Conclusions:* There was an inverse relationship between perceived emotional stress during the past week and the normalized HF component of HRV ($P = 0.038$). This indicates a lower cardiac vagal component of HRV among men and women who perceived more stress. That relationship was independent of age, gender, trait anxiety, and cardiorespiratory fitness. It was also independent of heart rate; mean arterial blood pressure; and respiration rate, factors which can influence HRV and might be elevated among people reporting anxiety and perceived stress. We conclude that vagal modulation of heart period appears to be sensitive to the recent experience of persistent emotional stress, regardless of a person's level of physical fitness and disposition toward experiencing anxiety.

Keywords: Nervous system; Autonomic; Heart rate; Cardiac-vagal

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0167-8760/00/$ - see front matter
PII: S0167-8760(00)00085-4

1. Introduction

Heart rate variability (HRV) provides non-invasive, unobtrusive information about modulation of heart rate by the autonomic nervous system in a variety of dynamic circumstances (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), including evoked emotions (McCraty et al., 1995) and exercise (Saito and Nakamura, 1995). Heart rate variability is commonly described by the standard deviation of intervals between successive R waves (SDRR) of the cardiac cycle. Short-term variation (e.g. measured during periods of several minutes) can be decomposed mathematically into spectral components which estimate autonomic modulation of heart rate. The high frequency (HF) spectrum (0.15–0.5 Hz) corresponds to vagally-mediated modulation of HVR associated with respiration (i.e. respiratory sinus arrthymia). The low frequency (LF) spectrum (0.05–0.15 Hz) corresponds to baroreflex control of heart rate and reflects mixed sympathetic and parasympathetic modulation of HRV, depending upon the circumstances of the assessment. When measured during slow, deep breathing or in the supine position (Pomeranz et al., 1985; Grasso et al., 1997), LF activity is believed to be vagally controlled. Activity in the very low frequency (VLF) spectrum (0.0033–0.05 Hz) can provide another index of sympathetic influence on heart rate during apnea (Shiomi et al., 1996). To estimate autonomic balance during short-term fluctuations in heart rate, the HF and LF spectra are commonly normalized to their total power (e.g. $[HF/(HF + LF) \times 100]$) in order to remove influences of VLF. Long-term (e.g. 24 h) monitoring of HRV permits assessment of the ultra low frequency (ULF) spectrum (< 0.0033 Hz) which is strongly correlated with the 24-h SDRR (i.e. total HRV).

Interest in the relationship between HRV and persistent emotional stress has increased after findings that perceived stress is predictive of transient myocardial ischemia (TMI) (Jiang et al., 1996; Gullette et al., 1997) and that HRV is associated with perceived stress (Sloan et al., 1994) and a reduced threshold for TMI (Goseki et al., 1994). Also, low HRV is associated with cardiac arrhythmia, cardiac mortality, and all cause mortality after myocardial infarction (Bigger et al., 1992; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Therefore, HRV may be a population risk factor for cardiac events (Tsuji et al., 1996).

Alterations in HRV have been reported in people with clinical anxiety disorders (Friedman and Thayer, 1998). In one report, patients with generalized anxiety disorder had a lower HF component of HRV during a resting baseline and during worry, consistent with reduced cardiac-vagal modulation, when compared with a non-anxious control group (Thayer et al., 1996). Yeragani et al. (1993) reported lower LF and ULF components among panic disorder patients compared with controls. In contrast, Rechlin et al. (1994) found a higher VLF component, consistent with elevated sympathetic modulation of HRV, in panic disorder patients compared with controls who were matched on age and gender. A study of patients with Posttraumatic Stress Disorder (PTSD) (Cohen et al., 1997) reported elevated power in normalized LF and HF components. However, PTSD patients had similar total HRV power, compared with case-controls matched on age, gender, and smoking habit.

Aside from studies of patients with clinical anxiety disorders, it is unclear whether HRV is altered among otherwise healthy people who have personality dispositions toward high anxiety or who experience persistent emotional stress. Fuller (1992) reported that HRV in the high frequency spectrum measured the day before a comprehensive examination was lower among female graduate students having high trait anxiety when compared with peers having low trait anxiety. Those findings are difficult to interpret, because the two groups did not differ on state anxiety the day of the exam. Because of this, it was not possible to conclude that the difference in the HF spectrum resulted from different emotional responses in anticipation of the examination or from other attributes associated with autonomic balance that could have affected HRV. Also, the high and low HRV spectra examined in that study were not

reported in normalized (i.e. High Frequency/Total Power [HF/TF]) or relativized (i.e. LF/HF) forms. These indexes of cardiac autonomic regulation provide better estimates of parasympathetic and sympathetic modulation of HRV in several circumstances (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Grasso et al., 1997). Finally, potential influences on the HF spectrum by heart rate, blood pressure, and respiration rate were not controlled in the Fuller study. Each of those factors can influence HRV (Brown et al., 1993; Grossman and Kollai, 1993; Huikuri et al., 1996a) and might be elevated among people who report anxiety or perceived stress.

To address these issues, we examined whether the relationship between trait anxiety and HRV reported by Fuller (1992) is independent of cardiorespiratory fitness and perceived stress during the past week. Cardiorespiratory fitness is associated with a resting bradycardia (Berntson et al., 1993) and the high frequency component of HRV (Coats, 1992; Gallagher et al., 1992; Demeersman, 1993; Goldsmith et al., 1997; Rossy and Thayer, 1998; Jackson et al., in press). Hence, the lower HF power among the high anxious women in the Fuller (1992) study could have resulted from a lower fitness level among those women. Furthermore, increased cardiorespiratory fitness after exercise training has been accompanied by a reduction in trait anxiety and perceived stress (King et al., 1993). By measuring perceived stress during the past week, we planned to determine whether a lower HF component of HRV would be related to trait anxiety or to contemporary perceptions of persistent stress which might alter more directly the autonomic regulation of HRV. We also examined whether effects differed according to age and gender, each of which is associated with HRV (Huikuri et al., 1996b; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

We report an inverse relationship between perceived stress during the past week and the normalized HF component of HRV that was independent of age, gender, trait anxiety, and cardiorespiratory fitness level. Our results suggest a reduced cardiac vagal tone among men and women who report that they recently have been experiencing stress.

2. Materials and methods

2.1. Participants

One hundred and three apparently healthy male ($N = 59$) and female ($N = 44$) normotensive volunteers aged 20–59 years of age were recruited from the Dallas–Fort Worth area by newspaper advertisement, flyers posted on the campus of the Cooper Clinic and Institute for Aerobics Research, and by word-of-mouth. Potential participants were first screened by a telephone call and, if eligible, were sent a medical history questionnaire. All were screened for medications and medical conditions known to affect HRV or contraindications for maximal treadmill testing as specified by the American College of Sports Medicine (American College of Sports Medicine, 1995). Eligible volunteers arrived at the laboratory at least 3 h post-prandial and after being instructed not to consume caffeineated beverages for at least 12 h prior to testing. Participants were allowed a light workout 48 h prior, but no exercise 24 h prior, to testing. Individuals having a resting blood pressure greater than 140 mmHg systolic and 90 mmHg diastolic or taking anti-hypertension, other cardiovascular, or psychotropic drugs were excluded. All individuals gave written informed consent prior to testing, which was approved by the Institutional Review Board for the Cooper Institute. The final data analysis included 92 participants (40 women and 52 men) who met the study's inclusion criteria and had complete data for the HRV analysis and the self-ratings of anxiety and perceived stress.

Table 1 gives mean ± S.D. for select characteristics of the participants. Men and women did not differ on age (40 ± 11 years), anxiety (5.08 ± 2.6), or perceived stress (16.26 ± 6.69). As expected, percent body fat was higher in women (25.5 ±

Table 1
Select characteristics (mean ± S.D.) of participants ($N = 92$)

Characteristic	Women ($n = 40$)	Men ($n = 52$)	P-value (two-tailed)
Age (years)	40.3 ± 12.5	39.7 ± 9.9	0.826
Height (cm)	163.4 ± 6	176.6 ± 6.3	0.001
Weight (kg)	62.5 ± 8.8	76.4 ± 8.5	0.001
Body Fat (% of mass)	25.5 ± 6.4	15.2 ± 5.2	0.001
Standing heart rate (b.p.m.)	70 ± 11	76 ± 11	0.008
Systolic blood pressure (mmHg)	118 ± 12	123 ± 10	0.061
Diastolic blood pressure (mmHg)	80 ± 8	82 ± 7	0.108
Supine heart rate (b.p.m.)	61 ± 9	54 ± 7	0.011
Systolic blood pressure (mmHg)	119 ± 11	122 ± 8	0.155
Diastolic blood pressure (mmHg)	75 ± 6	75 ± 7	0.946
Peak heart rate (b.p.m.)	184 ± 10	185 ± 12	0.826
Peak $\dot{V}O_2$ (ml kg^{-1} min^{-1})	37.4 ± 9.4	51.2 ± 9.7	0.001
Peak R–R power (Hz)	0.26 ± 0.04	0.26 ± 0.05	0.655
Respiratory rate (breaths min^{-1})	18.4 ± 6.1	18.67 ± 4.8	0.816
Ventilation (L min^{-1})	8 ± 2.6	10 ± 2.9	0.002
Cohen's Perceived Stress Scale	16.6 ± 6.3	16 ± 7	0.707
Taylor Manifest Anxiety Scale	4.95 ± 2.6	5.04 ± 2.6	0.872

6.4%) than men (15.2% ± 5.2) and $\dot{V}O_{2peak}$ was higher in men (51.2 ± 9.7 ml kg^{-1} min$^{-1)}$ than women (37.4 ± 9.4 ml kg^{-1} min^{-1}). Forty-five percent of the participants were classified as highly fit based on age, gender, and $\dot{V}O_{2\,peak}$ criteria. The remaining 55% were classified as fit, with no participants classified as having below average cardiorespiratory fitness. Ninety percent reported they exercised on a regular basis, defined as at least 20 min, three times per week.

2.2. Psychological instruments

Standardized self-ratings were obtained in a quiet room in the laboratory prior to administration of a clinical exercise test. To permit comparisons with related studies (Fuller, 1992; King et al., 1993), trait anxiety was measured by the Bendig short form of the Taylor Manifest Anxiety Scale (TMAS) (Taylor, 1953), which assesses the disposition to experience signs and symptoms of anxiety often and in various settings. Stress was measured by Cohen's Perceived Stress Scale (CPSS) (Cohen et al., 1983), which asks about perceptions during the past week. High scores on each scale indicate more anxiety or stress, respectively.

2.3. Measurement of heart rate variability

Each participant wore a chest belt hard-wired to a digital R–R recorder (Polar Electro Oy, Kempele, Finland) where the QRS-signal waveform (R–R signal) was sampled at the resolution of 1 ms. Participants rested supine in a quiet, semi-dark laboratory, temperature controlled between 18 and 21°C. After 10 min of rest, a 5-min recording of R–R intervals was obtained while the participant rested in a supine position. Respiration rate was not controlled. There were a few abnormal R–R intervals caused by artificial noise which were corrected by either omitting or interpolating beats. The number of beats edited manually was less than 1% of the total beats analyzed for every person. The standard deviation of all R-R intervals (SDRR) was used to estimate long-term components of heart rate variability. A 5-min R–R interval sequence was divided into three blocks. The linear trend was eliminated for each block by linear regression, and a cosine-tapered window in the time domain was applied to the data set. Fast Fourier transformation was applied to each block, and the spectral power was estimated as the ensemble average on the frequency domain. After converting the frequency

unit of cycle/beat to hertz based on the average R–R interval, the integrated spectral power densities of 0.05–0.15 Hz (LF) and 0.15–0.5 Hz (HF) were calculated, as well as the total power (TF) ranging from 0.017 to 0.5 Hz. When measured in the supine position, the HF and LF spectra primarily estimate cardiac vagal-mediated modulation of the heart period during breathing (Brown et al., 1993; Grossman and Kollai, 1993) and baroreflex activation (Pomeranz et al., 1985; Watkins et al., 1996; Grasso et al., 1997), respectively. Normalized HF (i.e. HF/TF) and the LF/HF ratio were calculated as indexes of cardiac parasympathetic nervous activity according to recommendations by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996).

2.4. Blood pressure and respiration rate

While each participant remained in a supine posture, auscultatory systolic and diastolic blood pressures were measured at Korotcoff sounds I and IV using a mercury sphygmomanometer. After each participant stood for 5 min, respiration rate and minute ventilation were measured for 1 min using a flow meter interfaced with a Sensor Medics (Yorba Linda, CA) model 2900 metabolic cart. Respiration rate during R–R assessments also was estimated by FFT as the frequency in the HF spectrum where peak power was observed (peak R–R power) (Watkins et al., 1998).

2.5. Measurement of cardiorespiratory fitness and body composition

Cardiorespiratory fitness was assessed by a grade-incremented treadmill exercise test to determine peak oxygen uptake ($\dot{V}O_{2\,peak}$). To more closely standardize relative exercise intensity, we developed six exercise protocols for this project. Each protocol consisted of continuous 4-min stages and was designed to last 16–24 min. Exercise intensity was gradually increased by using increments of speed and/or elevation equivalent to 2 METs or less for each stage. The protocols were selected based on each person's predicted $\dot{V}O_{2peak}$ (Jackson et al., 1990). A 12-lead ECG configuration was monitored during exercise.

Metabolic variables were assessed using a Sensor Medics 2900 metabolic cart. Expired gas was collected into a mixing chamber and sampled every 20 s. Metabolic variables were reported each 20 s using a rolling 1-min average. The exercise test was regarded as a maximal effort if the participant demonstrated a plateau in oxygen uptake, despite an increase in work rate, or

Table 2
Descriptive statistics (mean ± S.D.) for measures of heart rate variability ($N = 92$)[a]

	Women ($n = 40$)		Men ($n = 52$)		P-value (two-tailed)
RR interval (ms)	1002	162	1135	159	0.001
SDRR (ms)	56.2	25	73.5	31.8	0.006
LF (ms^2)	376	308	746	757	0.002
LF/HF	1.08	1.00	1.64	1.37	0.026
HF (ms^2)	600	668	737	920	0.428
HF/TF	0.32	0.17	0.23	0.13	0.012
TF (ms^2)	1806	1624	3033	3334	0.035
LnSDRR	3.9	0.44	4.2	0.42	0.003
LnLF	5.5	1.0	6.2	0.97	0.002
LnLF/HF	−0.25	0.81	0.18	0.82	0.015
LnHF	5.8	1.2	6.0	1.1	0.335
LnHF/TF	−1.3	0.67	−1.6	0.58	0.029
LnTF	7.1	0.92	7.6	0.87	0.006

[a] *Abbreviations:* LF, low frequency; HF, high frequency; LF/HF, index of cardiac sympathetic activity; LF/TF, low frequency/total power — normalized HF — index of cardiac parasympathetic activity; Ln, natural log.

achieved a respiratory exchange ratio (RER) ≥ 1.05 and a HR within 15 beats of predicted peak HR (HR_{peak}). Peak oxygen uptake ($\dot{V}O_{2\,peak}$) represents the highest average value recorded during the last 2 min of exercise.

Body composition was determined for all participants using a seven-site skinfold technique administered by the same experienced technician (Jackson and Pollock, 1978; Jackson et al., 1980). Percent fat mass was calculated as the average of the Brozek et al. (1963)) and Siri (1961)) equations.

2.6. Statistical analysis

Anxiety and the HRV measures were not normally distributed in this sample and were, therefore, natural log transformed before statistical analysis. The Pearson product-moment correlation coefficient was used to describe the relationships among the variables. To determine the independent effects of age, gender $\dot{V}O_{2\,peak}$, anxiety, and perceived stress on HRV, those variables were entered in a multiple linear regression model which was used in separate analyses to predict RR and the HRV variables: SDRR, HF/TF, and LF/HF. Supine HR and mean arterial pressure (MAP) also were included in the regression models to control for possible influences on HRV by baroreflex control of blood pressure. HR was excluded from the RR model. Probability values less than 0.05 were considered significant. Analyses were done with SPSS Windows 9.0 (SPSS Inc., Chicago, IL).

3. Results

3.1. Descriptive findings

The HRV variables were within normal levels for this age group (see Table 2), and their interrelationships were as expected (see Table 3). Pearson product-moment correlations indicated that HF/TF was inversely related to age ($r = -0.35$, $P < 0.001$) and was lower in men than women ($r = -0.25$, $P = 0.008$) regardless of age. LF/TF was positively related to age ($r = 0.33$, $P < 0.001$) and was higher in men than women ($r = 0.28$, $P = 0.004$) regardless of age. With the exception of HF/TF, dependent variables did not differ according to self-ratings of anxiety and perceived stress (see Tables 4 and 5).

Consistent with past studies, $\dot{V}O_{2\,peak}$ was inversely related to age ($r = -0.57$), regardless of gender, was higher in men than women ($r = 0.65$), regardless of age, and was positively related to RR ($r = 0.50$) and SDRR ($r = 0.44$), as well as HF ($r = 0.36$) and LF ($r = 0.36$) (P for all < 0.001). After controlling for age and gender, there was no relationship between $\dot{V}O_{2\,peak}$ and HF/TF ($P > 0.711$) or LF/HF ($P > 0.572$); correlations with SDRR ($r = 0.22$, $P = 0.04$) and HF ($r = 0.19$, $P = 0.07$) were attenuated and the correlation with LF was extinguished, after controlling for age and gender. A multiple linear regression model containing age (partial $r = 0.43$), $\dot{V}O_{2\,peak}$ (partial $r = 0.53$), SDRR (partial $r = 0.31$), and HF/TF (partial $r = 0.36$) predicted RR ($F_{4,87} = 17.6$, adj. $R^2 = 0.42$, $P < 0.001$). Self-rated anxiety and perceived stress were related ($r = 0.57$, $P < 0.001$), but each was unrelated to all other variables (P for all > 0.198). Peak R–R power in the HF spectrum and standing respiration rate were related ($r = 0.34$, $P = 0.01$), and each were higher among the participants who scored high on anxiety and perceived stress (see Table 4). However, neither measure of respiration rate was correlated with HF/TF ($P > 0.570$) or the other HRV variables (all $P > 0.13$).

3.2. Hypothesis testing

The linear regression models were significant for RR, $F_{6,85} = 5.5$, $P < 0.001$, and SDRR, HF/TF, and LF/HF, $F_{7,84} = 5.18$, 4.00, 3.13, and 2.79, respectively (P for all < 0.01). Participants who were older ($t = 2.4$, $P = 0.01$), had higher ($\dot{V}O_{2\,peak}$) ($t = 3.8$, $P < 0.001$), and lower MAP ($t = 1.7$, $P = 0.05$) had longer RR (adj. $R^2 = 0.27$), while participants who were younger ($t = 2.1$, $P = 0.021$), had lower HR ($t = 2.6$, $P = 0.006$) and higher MAP ($t = 1.7$, $P = 0.05$) had a larger SDRR (adj. $R^2 = 0.23$). In contrast, men ($t = 2.1$, $P = 0.02$), older participants ($t = 2.4$, $P = 0.01$), and participants having higher perceived stress ($t =$

Table 3
Pearson bivariate correlation coefficients among the time and frequency domains of heart rate variability ($N = 92$).

	RR	SDRR	LF	HF	TF	HF/TF	LF/HF	LnSDRR	LnLF	LnHF	LnTF	LnHF/TF
SDRR	0.42**											
LF	0.31**	0.82**										
HF	0.41**	0.79**	0.74**									
TF	0.35**	0.92**	0.88**	0.85**								
HF/TF	0.13	0.03	−0.04	0.45	0.01							
LF/HF	−0.19	−0.11	0.10	−0.36**	−0.12	−0.66**						
LnSDRR	0.42**	0.95**	0.72**	0.69**	0.79**	0.06	−0.08					
LnLF	0.33**	0.80**	0.80**	0.57**	0.67**	−0.02	0.20	0.87**				
LnHF	0.45**	0.77**	0.59**	0.81**	0.66**	0.57**	−0.47**	0.82**	0.71**			
LnTF	0.42**	0.94**	0.74**	0.71**	0.81**	0.07	−0.08	0.99**	0.88**	0.84**		
LnHF/TF	0.21*	0.04	0.00	0.44**	0.03	0.94**	−0.75**	0.06	0.02	0.60**	0.07	
LnLF/HF	−0.21*	−0.09	0.16	−0.41**	−0.09	−0.81**	0.90**	−0.07	0.25*	−0.51**	−0.08	−0.81**

**$P < 0.01$,
*$P < 0.05$ (two-tailed); Ln (natural log).

Table 4
Descriptive statistics (mean ± S.D.) for Cohen's Perceived Stress Scale (CPSS), cardiorespiratory fitness ($\dot{V}O_{2peak}$), respiration, and measures of heart rate variability compared between low (≤ 5) and high (> 5) scorers on the short form of the Taylor Manifest Anxiety Scale (TMAS)[a]

	Low ($n = 65$)	High ($n = 27$)	P-value (two-tailed)
TMAS	3.66 ± 1.1	8.22 ± 2.27	0.001
CPSS	14.2 ± 5.5	21.5 ± 6.5	0.001
$\dot{V}O_{2peak}$ (ml kg min^{-1})	44.1 ± 11.8	45.6 ± 11.8	0.583
Peak R–R power (Hz)	0.25 ± 0.04	0.28 ± 0.04	0.017
Respiration rate (breaths min$^{-1)}$	17.7 ± 5.1	20.4 ± 5.4	0.042
Ventilation (L min^{-1})	9 ± 2.6	9.7 ± 3.7	0.315
RR interval (ms)	1066 ± 173	1102 ± 173	0.354
SDRR (ms)	66.7 ± 32	64.2 ± 25.3	0.716
LF (ms^2)	606 ± 699	535 ± 425	0.623
LF/HF	1.32 ± 1.16	1.58 ± 1.45	0.364
HF (ms^2)	693 ± 851	640 ± 750	0.778
HF/TF	0.27 ± 0.15	0.28 ± 0.17	0.842
TF (ms^2)	2649 ± 3162	2139 ± 1503	0.426
LnSDRR	4.1 ± 0.46	4.1 ± 0.43	0.877
LnLF	5.9 ± 1.06	5.9 ± 0.97	0.910
LnLF/HF	−0.04 ± 0.79	0.05 ± 0.96	0.647
LnHF	5.9 ± 1.2	5.9 ± 1.1	0.817
LnHF/TF	−1.49 ± 0.62	−1.49 ± 0.68	0.965
LnTF	7.43 ± 0.95	7.37 ± 0.88	0.796

[a]*Abbreviations:* LF, low frequency; HF, high frequency; LF/HF, index of cardiac parasympathetic activity; LF/TF, low frequency/total power — normalized HF — index of cardiac parasympathetic activity; Ln, natural log.

1.9, $P = 0.038$) had lower HF/TF (adj. $R^2 = 0.17$). Men ($t = 2.4$, $P = 0.09$) and older participants ($t = 2.0$, $P = 0.023$) had higher LF/TF (adj. $R^2 = 0.15$).

4. Discussion

These results provide limited support for an attenuated parasympathetic modulation of HRV among healthy, physically fit, and mentally well adults who reported elevated perceptions of emotional stress during the past week. This finding did not extend to trait anxiety as measured by the TMAS (Fuller, 1992). Though another study (King et al., 1993) found that exercise training reduced trait anxiety and perceived stress, as measured by the instruments we employed, our results indicate that the relationship between the high frequency component of HRV and perceived stress is independent of trait anxiety and cardiorespiratory fitness among physically active adults. A blunted vagal modulation of heart rate would provide a plausible mechanism by which persistent emotional stress might affect cardiovascular health negatively, as vagal tone has been associated with the threshold for ventricular arrhythmia during emotional stress (Verrier and Lown, 1981).

Our finding that trait anxiety was unrelated to all HRV indices is not consistent with a prior report on 90 normotensive adults aged 25–45 years (Watkins et al., 1998). In that study, trait anxiety was inversely related to respiratory sinus arrhythmia (RSA) ($r = -0.26$) and baroreflex-mediated modulation of heart period ($r = -0.30$). The non-normalized HF component of HRV (0.13–0.5 Hz) was used to estimate RSA, while the LF component (0.07–0.1299 Hz) normalized to systolic blood pressure variability was used to estimate baroreflex-mediated control of HR. The exercise habits and fitness levels of the participants were not reported. In that well done study,

Table 5
Descriptive statistics (mean ± S.D.) for the Taylor Manifest Anxiety Scale (TMAS), cardiorespiratory fitness ($\dot{V}O_{2peak}$), respiration, and measures of heart rate variability compared between low (< 20) and high (≥ 20) scorers on Cohen's Perceived Stress Scale (CPSS)[a]

	Low stress ($n = 64$)	High stress ($n = 28$)	*P*-value (two-tailed)
CPSS	12.9 ± 4.0	24.2 ± 4.7	0.001
TMAS	4.1 ± 1.6	7.2 ± 3.2	0.001
$\dot{V}O_{2peak}$(ml kg^{-1} min^{-1})	43.4 ± 11.2	48.2 ± 12.7	0.076
Peak R–R power (Hz)	0.25 ± 0.04	0.27 ± 0.05	0.077
Respiration rate (breaths min$^{-1)}$	18 ± 5.4	20 ± 5.1	0.109
Ventilation (L min^{-1})	9.18 ± 3	9.23 ± 3	0.950
RR interval (ms)	1063 ± 169	1096 ± 183	0.415
SDRR (ms)	63.6 ± 30.4	71 ± 31.7	0.310
LF (ms^2)	584 ± 705	553 ± 419	0.836
LF/HF	1.32 ± 1.28	1.48 ± 1.12	0.584
HF (ms^2)	707 ± 911	591 ± 599	0.552
HF/TF	0.28 ± 0.15	0.23 ± 0.15	0.125
TF (ms^2)	2409 ± 3097	2720 ± 2142	0.642
LnSDRR	4.1 ± 0.44	4.20 ± 0.50	0.364
LnLF	5.85 ± 1.06	5.96 ± 1.0	0.646
LnLF/HF	−0.08 ± 0.84	0.10 ± 0.81	0.359
LnHF	5.9 ± 1.15	5.85 ± 1.19	0.808
LnHF/TF	−1.4 ± 0.56	−1.7 ± 0.70	0.047
LnTF	7.30 ± 0.93	7.50 ± 0.97	0.324

[a]*Abbreviations:* LF, low frequency; HF, high frequency; LF/HF, index of cardiac parasympathetic activity; LF/TF, low frequency/total power — normalized HF — index of cardiac parasympathetic activity; Ln, natural log.

it is not clear whether the small correlation between trait anxiety and HF might have been extinguished had the higher heart rate observed in the high anxious participants been controlled. Though blood pressure was analyzed as a covariate, no mention was made about a similar control for HR. Another explanation for the different results is their use of the State-Trait Anxiety Inventory (STAI). We used the TMAS in order to permit a direct comparison with the study by Fuller (1992). Though both scales measure trait anxiety, they are only moderately correlated. Nonetheless, in a separate study of physically active adults aged 25–40 years conducted in our laboratory, we found no relationship between trait anxiety measured by the STAI and the HF and LF components of HRV measured in a semi-recumbent position at rest or during active and passive laboratory stressor tasks (Jackson et al., in press). More study is needed to determine which components of the anxiety construct might be most influential on HRV, as both self-rated tension and the LF component of HRV have been found to be independent risk factors for the development of hypertension among middle-aged men in the Framingham Heart Study cohort (Markovitz et al., 1993; Singh et al., 1998).

Short-term HRV is regulated by cardiac sympathetic and parasympathetic nervous system activity. Responses of HRV (Pagani et al., 1991; McCraty et al., 1995; Sloan et al., 1997; Rossy and Thayer, 1998) during emotional tasks have been used as indexes of autonomic regulation as related to aging or cardiovascular disease. Previous research on fitness or exercise habits and HRV that pertained to emotional stress has been limited to laboratory studies that used acute stressor tasks (Saito and Nakamura, 1995; Boutcher et al., 1998; Wood et al., 1998).

Our observations are consistent with an earlier cross-sectional study of sedentary adults that reported no correlation between $\dot{V}O_{2peak}$ and spec-

tral components of HRV, despite a small relationship between $\dot{V}O_{2\,peak}$ and SDRR (Colflesh et al., 1997). In contrast, Goldsmith et al. (1997)) reported a high correlation between $\dot{V}O_{2\,peak}$ and non-normalized HF power among 37 men and women aged 22–44 years who had varying fitness levels. Age was unrelated to HRV in that sample. Earlier reports of enhanced vagal tone among aerobically fit participants used respiratory sinus arrhythmia to estimate cardiac-vagal tone (Kenney, 1985; Barney et al., 1988; Dixon et al., 1992; Boutcher et al., 1998). However, the scope of those methods and a lack of agreement among exercise studies that have examined the autonomic regulation of resting heart rate (HR) place limits on a conclusion that the bradycardia typically observed in higher fit participants reflects an enhanced vagal tone (Berntson et al., 1993; Buckworth et al., 1994; Graham et al., 1996).

More recently, Boutcher et al. (1998) conducted a study comparing HRV in trained male runners, untrained men who had low resting heart rates, and untrained men who had normal resting heart rates. Men who were exercise trained or were sedentary but had low resting heart rates each had higher power in HF (0.12–0.40 Hz) and LF (0.07–0.11 Hz) spectra of HRV measured at baseline. They also had a greater rate of decrease in each of those spectra during the first minute of a 2.5-min Stroop color-word conflict test taken in a seated posture (Boutcher et al., 1998). The implications of those results for autonomic control of heart rate are not clear. HF was not normalized to total power and was not adjusted for possible influences of breathing rate, while LF was not normalized to total blood pressure. Also, a minimum of approximately 5 min is recommended for stable sampling of the short-term spectral components of HRV (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

We recognize the influence of respiration rate on the cardiac cycle (Brown et al., 1993), but it is usually not feasible to fully control respiration in clinical settings like the one we have described. Sympathetic influence on the respiratory component of HRV, in the absence of adrenergic blockade, limits a clear interpretation of the HF spectrum of HRV as an index of cardiac vagal tone in many circumstances (Grossman and Kollai, 1993; Jennings and McKnight, 1994; Dunlap and Pfeifer, 1999). We attempted to control for influence by the sympathetic nervous system on the HF and LF components of HRV by measuring heart period in the supine position. Moreover, we found that respiration rate (either estimated by the RSA frequency at peak spectral power during R–R assessment or measured by spirometry soon after), though higher among the anxious participants, was not related to perceived stress or to the indices of HRV.

The mechanism of the resting bradycardia typically observed in aerobically trained individuals has not been established by scientific consensus (Seals and Chase, 1989; Berntson et al., 1993; Buckworth et al., 1994; Graham et al., 1996). Results of several cross-sectional (Lewis et al., 1980; Katona et al., 1982) and training studies (Maciel et al., 1985) of men that examined the heart period after beta-adrenergic-, vagal-, and double-blockade suggest that the exercise training-induced bradycardia is primarily mediated by non-autonomic factors resulting in a lower intrinsic rate among fit participants (Berntson et al., 1993). In contrast, cross-sectional (Smith et al., 1989) and exercise training (Ekblom et al., 1973) studies of men using similar blockade procedures have demonstrated that, in addition to non-autonomic factors, exercise-induced bradycardia results from increased vagal tone and a small reduction in sympathetic activity, resulting in an altered sympatho-vagal autonomic balance. Using double autonomic blockade, Shi et al. (1995) reported that the bradycardia observed occurred concomitantly with a 27% increase in $\dot{V}O_{2\,peak}$ among young men after 8 months of endurance exercise training and was a result of increased vagal tone with no change in intrinsic rate.

Though our participants were more fit than is typical for sedentary people their age, we do not believe that their level or range of fitness constrained the test of the effects of fitness on HRV, or the generalizability of these results to other normotensive middle-aged adults who are physically active. Both men and women in our sample

had a $\dot{V}O_{2peak}$ that was within the normal range for people who remain physically active and maintain low percent body fat from age 25 until middle-age (Jackson et al., 1995, 1996).

5. Summary

A small inverse relationship between perceived emotional stress during the past week and the normalized HF component of HRV was observed. This indicates a blunted cardiac-vagal modulation of HRV among men and women who perceived more stress. That relationship was independent of age, gender, trait anxiety, and cardiorespiratory fitness. We conclude that the cardiac-vagal spectral component of HRV may be sensitive to the recent experience of persistent emotional stress, regardless of people's dispositions toward experiencing anxiety and their levels of physical fitness.

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