Available online at www.sciencedirect.com

SCIENCE DIRECT®

International Journal of Psychophysiology 53 (2004) 183–195

INTERNATIONAL JOURNAL OF PSYCHOPHYSIOLOGY

www.elsevier.com/locate/ijpsycho

Alexithymia predicts attenuated autonomic reactivity, but prolonged recovery to anger recall in young women

Serina A. Neumann[a,*], John J. Sollers III[b,1], Julian F. Thayer[b,2], Shari R. Waldstein[b,c,d,e,3]

[a] *Cardiovascular Behavioral Medicine Research Training Program, Department of Psychiatry, University of Pittsburgh, 4015 O'Hara Street, 506 OEH, Pittsburgh, PA 15260, USA*

[b] *Laboratory of Personality and Cognition, National Institute on Aging Gerontology Research Center, Room 2-C-08 5600 Nathan Shock Drive, Baltimore, MD 21224-6825, USA*

[c] *Department of Psychology, University of Maryland, Baltimore County, 1000 Hilltop Circle, Baltimore, MD 21250, USA*

[d] *Geriatric Research Education and Clinical Center, University of Maryland School of Medicine, 1000 Hilltop Circle, Baltimore, MD 21250, USA*

[e] *Baltimore Veterans Affairs Medical Center, and Gerontology Research Center, National Institute on Aging, 1000 Hilltop Circle, Baltimore, MD 21250, USA*

Received 2 December 2003; received in revised form 29 January 2004; accepted 25 March 2004

Abstract

Alexithymia has been prospectively associated with all-cause mortality and with cardiovascular morbidity. Here, stress-induced autonomic reactivity and recovery were examined as potential pathways linking alexithymia to cardiovascular disease. The relation of alexithymia to blood pressure, heart rate, and other cardiovascular parameters derived from impedance cardiography ($N=80$) and heart rate variability ($N=40$) was evaluated during rest, an anger recall task and recovery in women (ages 18–30). During anger recall, alexithymia was associated with significantly attenuated heart rate and stroke index reactivity, greater low frequency power, and with marginally dampened blood pressure and high frequency power reactivity. Overall, this response pattern suggests blunted sympathetic activation and diminished vagal withdrawal. Alexithymia was also related to slower diastolic blood pressure and quicker preejection period recovery implying abbreviated sympathetic arousal and possibly greater vagal modulation. These results impart some evidence for the hypoarousal model of alexithymia during reactivity, but the hyperarousal model during recovery. Autonomic dysregulation during and following acute emotional stress is suggested as a possible physiological pathway connecting alexithymia to cardiovascular disease.
© 2004 Elsevier B.V. All rights reserved.

Keywords: Alexithymia; Autonomic reactivity; Recovery; Impedance cardiography; Heart rate variability

* Corresponding author. Tel.: +1-412-624-8855; fax: +1-412-624-9108.

E-mail addresses: neumannsa@msx.upmc.edu (S.A. Neumann), sollersj@grc.nia.nih.gov (J.J. Sollers), thayer@lpc.grc.nia.nih.gov (J.F. Thayer), waldstei@umbc.edu (S.R. Waldstein).

[1] Tel.: +1-410-558-8285; fax: +1-410-558-8690.

[2] Tel.: +1-410-558-8612; fax: +1-410-558-8690.

[3] Tel.: +1-410-455-2374; fax: +1-410-455-1055.

0167-8760/$ - see front matter © 2004 Elsevier B.V. All rights reserved.
doi:10.1016/j.ijpsycho.2004.03.008

1. Introduction

Alexithymia has traditionally been described as having difficulties with identifying, describing, regulating, and expressing one's emotions (Sifneos, 1973; Taylor et al., 1985). This cognitive–affective construct has also been related to being preoccupied with external events, having problems with distinguishing among thoughts, feelings, and bodily sensations, and having a poverty of imagination, daydreams and fantasies (Taylor et al., 1985). Alexithymia has been prospectively associated with all-cause mortality (Kauhanen et al., 1996) and has shown cross-sectional relations to hypertension (Jula et al., 1999) myocardial infarction (Kojima et al., 2001), coronary artery spasm (Numata et al., 1998) and coronary heart disease (Kauhanen et al., 1996).

It is thought that alexithymia may influence cardiovascular and other organic diseases through several physiological, behavioral, cognitive, or social pathways. For instance, Lumley et al. (1996) reviewed several studies showing that alexithymia was associated with negative affect, unhealthy behaviors, nonadherence to medical regimens, and social isolation (also see Linden et al., 1996; Waldstein et al., 2002; Fukunishi et al., 1999; Friedlander et al., 1997; Nemiah et al., 1997; Newton and Contrada, 1994; Wehmer et al., 1995; Berenbaum and Irvin, 1996; Haviland et al., 1988; Kauhanen et al., 1991; Lumley and Norman, 1996; Valkamo et al., 2001). However, more research is needed with respect to the exploration of potential physiological pathways connecting alexithymia to cardiovascular disease.

In this regard, it has been hypothesized that autonomic nervous system (ANS) dysregulation may play a mechanistic role underlying the relation of alexithymia and cardiovascular health (Lumley et al., 1996; Linden et al., 1996; Waldstein et al., 2002; Fukunishi et al., 1999; Friedlander et al., 1997). Dysregulation of the autonomic nervous system (i.e., reduced basal parasympathetic and/or exaggerated activation of sympathetic modulation of heart rate and contractility) has been hypothesized to play a role in cardiovascular disease pathogenesis (e.g., Sloan et al., 1999; Kop, 1999, 2003; Rozanski et al., 1999). However, two opposing models have emerged in the prior literature with respect to alexithymia and ANS regulation—the hypoarousal versus the hyperarousal model of alexithymia. The hypoarousal model of alexithymia posits that dampened sympathetic nervous system activation and limited affective reactivity is associated with alexithymia during emotional provocation. In contrast, the hyperarousal model poses that alexithymia is related to higher tonic levels of sympathetic activity and/or exaggerated sympathetic reactivity (and possibly parasympathetic withdrawal) to emotional stressors. If alexithymia is indeed associated with hyperarousal, then it is possible that this personality factor may be linked to cardiovascular disease pathogenesis via enhanced cardiovascular reactivity (Lumley et al., 1996; Linden et al., 1996; Waldstein et al., 2002; Fukunishi et al., 1999; Friedlander et al., 1997; Nemiah et al., 1997; Newton and Contrada, 1994; Wehmer et al., 1995; Berenbaum and Irvin, 1996; Rozanski et al., 1999; Davis et al., 2000; Everson et al., 1995; Prkachin et al., 2001; Neumann and Waldstein, 2001; Sinha et al., 1992). However, as reviewed below, evidence for hyperarousal versus hypoarousal has been mixed in psychophysiological studies of alexithymia.

Several studies have investigated the association between alexithymia and tonic and acute ANS responses to laboratory-based stressors (Linden et al., 1996; Waldstein et al., 2002; Fukunishi et al., 1999; Friedlander et al., 1997; Nemiah et al., 1997; Newton and Contrada, 1994; Wehmer et al., 1995) using reliable and well-validated measures of alexithymia such as the Toronto Alexithymia Scale (Taylor et al., 1985). In support of the hypoarousal model, three studies (i.e., Linden et al., 1996; Newton and Contrada, 1994; Wehmer et al., 1995) found that alexithymic men and women displayed attenuated heart rate responses to emotion-provoking tasks. Additionally, Friedlander et al. (1997) found attenuated heart rate reactivity for women, but not for men. Another study (Nemiah et al., 1997) found smaller increases in oxygen consumption during mental arithmetic and emotional imagery among alexithymic men and women. In contrast, Waldstein et al. (2002) found support for the hyperarousal model. These authors found that, in a sample of older men, alexithymia was related to exaggerated blood pressure reactivity to anger-provoking tasks. Thus, five of six studies supported the hypoarousal model of alexithymia, whereas one found evidence for the hyperarousal model of alexithymia during stress.

Tonic levels of physiological function have also been examined with respect to alexithymia, and provide support for the hyperarousal model. Three of

seven studies (Fukunishi et al., 1999; Friedlander et al., 1997; Newton and Contrada, 1994) suggest that alexithymia is associated with higher baseline levels of sympathetic activity (i.e., sympathovagal balance as measured by heart rate variability) (Fukunishi et al., 1999), skin conductance (Friedlander et al., 1997), and heart rate (Newton and Contrada, 1994)) in groups of relatively young men and women.

In light of the inconsistent findings regarding alexithymia and autonomic function, the present study further examined the relation of alexithymia to resting and reactive autonomic activity. Measures of autonomic poststress recovery were also examined given that prolonged poststress recovery may be another important linkage between alexithymia and cardiovascular disease (Friedlander et al., 1997). Consistent with the prior literature, and since alexithymics are acknowledged to have various problems with emotion recognition and expression, an emotion (specifically anger) provoking task was used here to elicit autonomic activation.

It has also been hypothesized that alexithymics experience problems with accurately characterizing their affective responses to emotional stressors which can result in discordant self-ratings of affect in comparison to their physiological and behavioral responses (Lumley et al., 1996; Newton and Contrada, 1994; Berenbaum and Irvin, 1996). For instance, Newton and Contrada (1994) found that compared to nonalexithymics, alexithymic women reported greater negative affect, but evinced lower heart rate responses to a speech task. Similarly, Berenbaum and Irvin (1996) found that although alexithymic men and women displayed greater nonverbal anger responses during an anger provocation task, they reported their experiences as more pleasant than their counterparts. In contrast, Friedlander et al. (1997) found that alexithymic women concordantly reported greater self-reported displeasure and arousal concomitant with greater electrodermal activity than their nonalexithymic counterparts. Moreover, Waldstein et al. (2002) noted no significant differences between alexithymics' and non-alexithymics' state affect ratings despite the enhanced cardiovascular reactivity found among alexithymic men. Due to these inconsistencies, it remains unclear whether negative affect is directly related to ANS function in alexithymics. Consequently, self-reported state anger was examined here in relation to resting, reactive, and recovery measures of ANS function.

The primary purpose of the present study was to investigate the relations among alexithymia, resting autonomic function, and autonomic reactivity (i.e., blood pressure, heart rate, impedance derived measures and HRV) induced by a personally relevant anger recall task, and poststress recovery in healthy, young women. This study contributes to the prior research by utilizing both impedance cardiography and heart rate variability measures when investigating cardiovascular reactivity and recovery in relation to alexithymia. As a secondary aim, state anger was examined in association with alexithymia and concurrent physiological function. In light of the evidence presented thus far, it was hypothesized that, upon evaluating the relation between alexithymia and autonomic measures during the baseline, task, and recovery periods in separate multiple regression analyses: (1) Alexithymia would be associated with higher tonic cardiovascular function and sympathetic activity (and possibly lower vagal tone). (2) Alexithymia would be related to smaller changes in cardiovascular and autonomic reactivity during the anger recall task and thus quicker recovery (return to baseline). (3) Alexithymia would be associated with greater self-reported state anger (discordant with physiological findings) at rest and during the task and recovery periods.

2. Methods

2.1. Participants

The sample and relevant data of the present investigation were derived from a previous study examining the effects of hostility and distraction on cardiovascular and autonomic recovery (Neumann et al., in press). Eighty female university students (18–30 years) were recruited from introductory psychology courses and from advertisements at the University of Maryland, Baltimore County (UMBC). The sample included 54% Caucasian, 34% African-American, and 12% Asian-American participants approximating the ethnic stratums at UMBC. All participants were nonsmokers (by self-report); were nonobese (body mass index (BMI) < 30 kg/m^2 (The National Heart Lung et al., 1998)); were normotensive (resting systolic blood pressure (SBP) < 140 mm Hg and diastolic blood pressure (DBP) < 90 mm Hg) (Joint National Com-

mittee on Detection, Evaluation, and Treatment of High Blood Pressure, 1997); and reported no history of hypertension, cardiovascular or pulmonary disease, psychiatric disorder, or use of medications that would alter cardiovascular function. The characteristics of the sample are provided in Table 1. Participants were requested to withhold use of caffeine for 12 h and alcohol for 24 h before their session. Informed consent was acquired according to the UMBC's Institutional Review Board guidelines. Each participant completed a 2-h laboratory session and was remunerated with a choice of two course credits toward their psychology class or with US$10.00 for completing the study.

2.2. Psychosocial measures

The 26-item Toronto Alexithymia Scale (TAS-26) was used to assess alexithymia (Taylor et al., 1985). The items are scaled on a 5-point likert scale from 1—strongly disagree to 5—strongly agree with possible scores ranging from 26 to 36. The TAS-26 and the 20-item TAS (TAS-20) are the most widely accepted, used, and psychometrically sound instruments utilized to measure alexithymia in current research. The TAS was developed in response to problems with earlier instruments having insufficient reliability and validity (Taylor et al., 1985; Bagby et al., 1988, 1994). The TAS-26 has a test–retest reliability equal to 0.82 and the construct validity of this scale is considered adequate. A score of 74 or greater on the TAS-26 suggests clinically significant levels of alexithymia.

State anger was measured using the S-Anger subscale of the State-Trait Anger Expression Inventory (STAXI) (Spielberger, 1988). The 10 items of the subscale were rated on a 4-point likert scale and were summed (scores range from 10 to 40) with higher scores indicating greater state anger. The internal consistency of the subscale is around 0.84 and validity has been supported by several studies (Spielberger, 1988).

Table 1
Sample characteristics for low and high Alexithymia groups

Characteristic	Alexithymia			
	Low ($n=37$)		High ($n=42$)	
	M	S.D.	M	S.D.
Demographics				
Age (years)	19.0	(1.6)	18.9	(1.4)
BMI (kg/m^2)	22.0*	(3.5)	24.0*	(2.7)
Education (years)	12.8	(1.4)	12.5	(1.0)
Caffeine intake (8 oz. drinks/day)	1.1	(0.9)	1.2	(0.9)
Alcohol intake (drinks/week)	0.3*	(0.7)	1.2*	(2.0)
Positive family history of hypertension	38%		41%	
Psychosocial measures				
Baseline state anger	10.2	(0.6)	10.4	(0.9)
Task state anger	18.4	(4.9)	19.2	(7.0)
Recovery state anger	13.2	(3.6)	12.8	(4.6)

*$p<0.01$.

2.3. Cardiovascular measures

SBP, DBP and mean arterial pressure (MAP) were assessed oscillometrically using a Critikon Dinamap Vital Signs Monitor (cuff on nondominant arm) [Model # 8100; Critikon, Tampa, FL]. Heart rate (HR) was derived from the electrocardiogram (ECG) measured from two electrodes attached bilaterally to the chest. Heart sounds were obtained by a Hewlett-Packard Contact Transducer (Model # 21050A) positioned at the second intercostal space on the left sternal border. The ECG and heart sound signals were filtered and amplified by Grass biological amplifiers. Impedance derived signals (dZ/dt) were used to estimate cardiac preejection period (PEP), stroke volume (SV), left ventricular ejection time (LVET), cardiac output (CO), and total peripheral resistance (TPR) [IFM Minnesota Impedance Cardiograph, Model # 304B]. A tetrapolar band-electrode configuration was used (two bands placed around the neck and two around the chest) (Sherwood et al., 1990). ECG, heart sounds, dZ/dt (first derivative of the change in thoracic impedance), and Zo (basal thoracic impedance) signals were acquired continuously employing computerized analog to digital conversion at a rate of 1000 samples per second (Debski et al., 1991).

Using VU-AMS (version 4.4, TD-FPP, Vrïe Universiteit, Amsterdam), heart rate variability (HRV) data were measured (Klaver et al., 1994). R wave-to-R wave intervals were derived continuously on-line from a 3-lead ECG. The recording methodology, reliability and validity of the VU-AMS has been described previously in more detail (see De Geus et al., 1995).

3. Procedures

Each participant was seated in a sound-attenuated, temperature-controlled room. They completed a 15-min baseline period, a 3-min anger recall task, and a 10-min recovery period. Participants were asked to rest quietly and to try not to move around, talk, or sleep during the rest period. For the task, the experimenter provided instructions for the personally relevant anger recall task (Valkamo et al., 2001; Neumann and Waldstein, 2001; Neumann et al., in press; Ironson et al., 1992; Waldstein et al., 2000). Each participant was asked to speak aloud in the presence of the female experimenter for 3 min about a recent event occurring within the last year and that continued to make her very angry, frustrated, irritated, or upset. After an angry event was selected (an event that would generate moderately high intensity anger), the participant prepared silently for 30 s for her upcoming recall. After the 30-s preparation, she was prompted to speak for the entire 3 min. Each participant was reminded to "keep speaking" if pauses of 3 s occurred and was asked questions to intensify and incite moderately high levels of anger. The types of events that were discussed included significant interpersonal problems/arguments with their partner, close friends, roommates, and family members, as well as situations involving discrimination. As part of a previous experiment (Neumann et al., in press), the participants were randomly assigned to undergo either a distraction technique (i.e., reading a neutral article about the possibility of life in outer space (Weinberger et al., 1979)) or a standard recovery period (i.e., identical instructions as for the initial baseline period with no reading or implemented distractions) during the recovery period. This recovery manipulation variable will not be discussed in the present report since it did not interact with alexithymia scores, but it was nonetheless entered into the final regression analyses to account for concomitant covariation. The S-anger subscale was administered prior to the task in order to measure baseline levels of affect. Following the recovery period, the participant completed the S-anger subscale for the task and recovery periods separately, and then the TAS was completed. Blood pressure was taken every 90 s during the baseline and every 60 s during the task and recovery periods ($N = 80$), which is in accordance with psychometric criteria (Debski et al., 1991; Klaver et al., 1994; Ironson et al., 1992). ECG and impedance cardiography were assessed continuously throughout the rest and task periods ($N = 80$). HRV measures were collected on the latter 40 participants.[4]

3.1. Data reduction

Blood pressure data (i.e., SBP and DBP) were averaged for the baseline period and the anger recall task. For the baseline measures, the last three blood pressure readings were averaged (Kamarck et al., 1992; 1993). For the recall task, the mean of the three obtained blood pressure readings was calculated. ECG and impedance waveforms were ensemble-averaged and scored in 30-s intervals for the baseline, task, and recovery periods utilizing computer software programmed at the University of Pittsburgh (Debski et al., 1991). Stroke volume was calculated using a fixed value of 135 Ω cm for blood resistivity (SV = LVET*dZ/dT*12/zo2*rho) (Kubicek et al., 1966). Cardiac output was computed as (HR*SV)/1000. Stroke volume index (SI) and cardiac index (CI) was calculated by dividing SV and CO by body surface area [weight $(\mathrm{kg})^{425} \times \mathrm{height}\ (\mathrm{cm})^{725} \times 0.007184$] in order to adjust for participant differences in body mass. Total peripheral resistance was calculated using the equation MAP/CO × 80. PEP and LVET were coded in ms using the intervals of the Q-wave of the digitized ECG to the B-point of the dZ/dt waveform and the B-point to X-wave of the dZ/dT waveform (i.e., coincident with the closure of the aortic valve—the second heart sound). The impedance cardiography measures were then averaged for the baseline, task, and recovery periods (Kamarck et al., 1993).

Time and frequency domain analyses were performed for the HRV measures utilizing a custom package. Time domain analysis provided root mean

[4] Using GPOWER software (Erdfelder et al., 1996), an a priori power analysis was performed for multiple regression analyses with one main predictor regressed on the HRV reactivity measures. The power analysis revealed that for power = 0.80, larger effect sizes ($f^2 = 0.33$) (effect size was calculated using HF power reactivity estimates during a speech task; Berntson et al., 1994) and alpha = 0.05 26 participants were needed to provide sufficient power for this experiment (Erdfelder et al., 1996). The equipment to measure HRV was available only for the latter 40 of the 80 participants.

of successive differences in R–R intervals (r-MSSD) and heart rate (HR). To obtain both low-frequency (LF: 0.04–0.15 Hz) and high-frequency (HF: 0.15–0.4 Hz) power components, spectral analyses using an autoregressive algorithm were performed according to the Task Force guidelines (1996). HF power has been shown to primarily reflect fluctuations in heart rate through the respiratory cycle that are moderated by parasympathetic (vagal) efferent input to the SA node (Friedman and Thayer, 1998). In contrast, LF power is proportional to cardiac sympathetic activity, but is also influenced by parasympathetic tone (Friedman and Thayer, 1998; Pagani et al., 1992). To interpret LF power, LF/HF ratios were calculated (Malliani et al., 1990). Higher LF/HF ratios represent relative increased sympathetic activity and/or decreased parasympathetic activity. To normalize the distribution of scores, spectral estimates of power were logarithmically transformed (ln) for the baseline (15-min epoch), task (3-min epoch), and recovery (10-min epoch) periods and will be noted with an "$_L$" (Malliani et al., 1994). Regarded as reliable estimates of autonomic balance (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology et al., 1996; Malliani et al., 1994), normalized values for the spectral estimates of power were also created (i.e., LF and HF power/(total power − DC components)) for descriptive purposes and will be indicated with an "$_N$" in Tables 2 and 3.

Arithmetic change scores (task mean value − base-baseline mean value) were calculated to assess the reactivity of SBP, DBP, HR, PEP, SI, CI, TPR, r-MSSD, LF_L power, and HF_L power responses during the recall task (Kamarck et al., 1992). These change scores were then used in the regression analyses for the task period. For recovery, the area under the recovery curve minus the baseline (i.e., excursions) was computed for each participant and each cardiovascular measure (i.e., SBP, DBP, HR, PEP, CI, SI, TPR) (Protter and Morrey, 1970). The following equation was used to compute the recovery excursions: Excursion=[0.5∗fixed time interval ((cardiovascular measure at time 1)+(2∗cardiovascular measure at time 2)+(2∗cardiovascular measure at time 3)+...+(cardiovascular measure at last time point)) − (baseline cardiovascular measure∗the fixed time interval)]; where fixed time interval = 60-s averages for blood pressure and 30-s averages for HR and impedance measures, and each time point (e.g., time 1) represents a blood pressure value taken every 60 s, or for HR and impedance measures, corresponds to 30-s averages until the end of the 10-min recovery period. Spectral estimates of power estimating the HRV components during recovery are the standard for assessing HRV (Task Force of the European Society of Cardiology and the North American Society of Pacing and

Table 2
Baseline and task cardiovascular and HRV means (and standard deviations) for low and high Alexithymia groups

	Alexithymia			
	Low ($n=37$)		High ($n=42$)	
	M	S.D.	M	S.D.
Baseline measures				
SBP (mm Hg)	106	(6.3)	106	(7.8)
DBP (mm Hg)	56	(5.9)	58	(6.1)
HR (bpm)	76	(11.2)	77	(10.2)
PEP (s)	102	(12.2)	107	(12.2)
CI (l/min/m^2)	4.3	(0.9)	4.1	(1.0)
SI (ml/beat/m^2)	60	(15.0)	56	(16.7)
TPR (dyn/cm^5/s)	912	(214)	920	(250)
HRV				
r-MSSD (ms)	42.9	(23.6)	46.8	(30.9)
LF_L (Hz)	2.74	(0.32)	2.60	(0.41)
HF_L (Hz)	2.80	(0.51)	2.81	(0.62)
LF/HF_L (Hz)	1.14	(0.85)	1.01	(1.29)
LF_N (Hz)	0.44	(0.16)	0.40	(0.20)
HF_N (Hz)	0.49	(0.16)	0.56	(0.21)
Task measures				
SBP (mm Hg)	123	(9.0)	120	(10.4)**
DBP (mm Hg)	73	(9.1)	72	(8.0)**
HR (bpm)	92	(14.4)	89	(9.8)*
PEP (s)	99	(12.7)	106	(13.2)
CI (l/min/m^2)	4.2	(1.1)	4.0	(1.0)
SI (ml/beat/m^2)	45	(13.2)	45	(13.3)*
TPR (dyn/cm^5/s)	1189	(374)	1192	(392)
HRV				
r-MSSD (ms)	33.0	(19.5)	40.9	(21.4)
LF_L (Hz)	2.89	(0.38)	3.02	(0.45)*
HF_L (Hz)	2.35	(0.59)	2.59	(0.61)**
LF/HF_L (Hz)	1.31	(0.33)	1.19	(0.16)
LF_N (Hz)	0.68	(0.20)	0.64	(0.16)
HF_N (Hz)	0.25	(0.18)	0.25	(0.11)

$N=80$ for SBP, DBP, HR and impedance measures; $N=40$ for HRV measures.
*p's < 0.01.
**p's < 0.10.

Electrophysiology et al., 1996) and were not computed as excursions.

High and low alexithymia groups were created based on the median of the TAS scores [MDN = 64: Low TAS < 64 ($n = 37$); High TAS ≥ 64 ($n = 42$)]. These groups were created solely for descriptive purposes, whereas continuous alexithymia scores were used in the primary regression analyses.

4. Results

4.1. Preliminary analyses

TAS scores ranged from 35 to 86 ($M = 61$, S.D. = 12) with 11 (13%) participants scoring in the clinical range. The distribution of high and low alexithymia participants were uniformly distributed between the distraction conditions (Chi-square = 0.014, n.s.). Pearson r correlations or analysis of variance (ANOVA) were performed to examine the relation of alexithymia to sample characteristics (i.e., age, education, BMI, self-reported average alcohol and caffeine consumption, parental history of hypertension, and ethnicity) and resting autonomic function (see Table 1). Pearson r correlational analyses yielded significant positive correlations of alexithymia with alcohol intake (r (79) = 0.26, $p < 0.02$) and BMI (r (79) = 0.23, $p < 0.04$). Due to these significant findings, Pearson r correlations were conducted to evaluate the relation between BMI and alcohol intake to autonomic function during the resting, task, and recovery periods. BMI was positively correlated with average baseline SBP (r (80) = 0.43, $p < 0.0001$) and PEP reactivity (r (76) = 0.23, $p < 0.04$), and was negatively related to HR reactivity (r (78) = − 0.22, $p < 0.05$) as well as LF (r (37) = − 0.31, $p < 0.052$) and HF (r (38) = − 0.24, $p < 0.057$) during recovery. Alcohol intake was marginally (and negatively) associated with baseline HR (r (79) = − 0.21, $p < 0.07$) and positively related to baseline SI (r (79) = 0.25, $p < 0.03$). No other significant results were noted with respect to these analyses. As a result, BMI was entered as a covariate for the

Table 3
Cardiovascular and HRV recovery means (and standard deviations)

Alexithymia group	Cardiovascular recovery and HRV measures	Recovery			
		1 min	2 min	5 min	10 min
Low	SBP (mm Hg)	113 (8.1)	110 (7.5)	108 (7.7)	107 (3.1)
High	SBP (mm Hg)	111 (10.8)	109 (8.1)	107 (7.2)	107 (7.4)
Low	DBP (mm Hg)*	58 (7.0)	54 (7.9)	53 (7.6)	51 (8.1)
High	DBP (mm Hg)*	60 (7.9)	58 (6.9)	56 (7.4)	55 (8.1)
Low	HR (bpm)	83 (18.9)	80 (14.5)	78 (12.4)	80 (13.0)
High	HR (bpm)	79 (10.0)	78 (9.5)	78 (9.0)	80 (12.5)
Low	PEP (s)*	97 (14.0)	99 (12.6)	100 (13.2)	101 (13.3)
High	PEP (s)*	103 (14.3)	106 (14.1)	107 (11.9)	108 (12.2)
Low	CI ($l/min/m^2$)	4.0 (1.1)	4.2 (1.0)	4.3 (1.1)	4.2 (1.0)
High	CI (l/min/m2)	4.0 (1.1)	4.1 (1.0)	4.1 (1.0)	4.1 (1.0)
Low	SI ($ml/beat/m^2$)	52 (16.5)	58 (17.0)	59 (17.1)	57 (13.5)
High	SI ($ml/beat/m^2$)	51 (14.5)	54 (15.3)	53 (15.0)	53 (14.9)
Low	TPR ($dyn/cm^5/s$)	1032 (336)	971 (267)	943 (270)	929 (244)
High	TPR ($dyn/cm^5/s$)	1037 (317)	976 (314)	964 (297)	919 (252)
Low	r-MSSD (ms)	–	–	–	39.1 (25.7)[a]
High	r-MSSD (ms)	–	–	–	45.5 (24.4)[a]
Low	LF_L (Hz)	–	–	–	2.72 (0.39)[a]
High	LF_L (Hz)	–	–	–	2.72 (0.38)[a]
Low	HF_L (Hz)	–	–	–	2.67 (0.53)[a]
High	HF_L (Hz)	–	–	–	2.79 (0.51)[a]
Low	LF/HF_L (Hz)	–	–	–	1.69 (1.67)[a]
High	LF/HF_L (Hz)	–	–	–	1.45 (2.16)[a]
Low	LF_N (Hz)	–	–	–	0.48 (0.20)[a]
High	LF_N (Hz)	–	–	–	0.42 (0.19)[a]
Low	HF_N (Hz)	–	–	–	0.45 (0.19)[a]
High	HF_N (Hz)	–	–	–	0.50 (0.20)[a]

Notes to Table 3:
$N = 80$ for SBP, DBP, HR and impedance measures; $N = 40$ for HRV measures.
[a] Average of entire 10-min recovery period.
* $p < 0.007$.

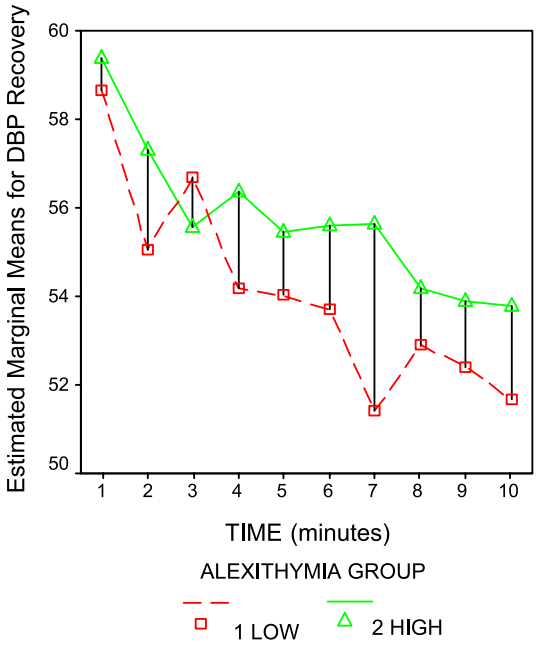

Fig. 1. Alexithymia groups on DBP poststress recovery.

multiple regression analyses on SBP, HR, PEP, and HRV responses during the baseline, task and recovery periods. Alcohol intake was entered as a covariate for multiple regression analyses concerning HR and SI measures.

4.2. Alexithymia and autonomic reactivity during anger recall[5]

Multiple regression analyses were performed on SBP, DBP, HR, PEP, SI, CI, TPR, LF_L power, and HF_L power arithmetic change scores with alexithymia as the predictor (controlling for BMI for SBP, HR, PEP, and HRV reactivity). As expected, alexithymia predicted significantly attenuated HR ($\beta = -0.40$, $p < 0.001$, $r^2 = 0.14$) and SI ($\beta = 0.33$, $p < 0.002$, $r^2 = 0.10$) reactivity (see Table 2). Marginally significant relations were noted between alexithymia and attenuated blood pressure reactivity [SBP ($\beta = -0.20$, $p < 0.09$, $r^2 = 0.04$) and DBP ($\beta = -0.21$, $p < 0.06$, $r^2 = 0.04$)]. Additionally, alexithymia predicted significantly greater LF_L power ($\beta = 0.45$, $p < 0.009$, $r^2 = 0.18$) and marginally smaller changes in HF_L power ($\beta = 0.29$, $p < 0.08$, $r^2 = 0.08$) during the recall task (see Table 2).

4.3. Alexithymia and autonomic recovery

Multiple regression analyses examined alexithymia as a predictor of autonomic recovery from anger recall (i.e., SBP, DBP, HR, PEP, SI, CI, and TPR excursions and LF_L power and HF_L power). Respective autonomic reactivity means and distraction condition were entered prior to alexithymia in these analyses to account for concomitant covariation. Unexpectedly, the results showed that alexithymia was significantly associated with hampered DBP recovery ($\beta = 0.31$, $p < 0.007$, $r^2 = 0.10$) combined with shorter PEP ($\beta = 0.32$, $p < 0.003$, $r^2 = 0.10$) recovery (see Table 3 and Figs. 1 and 2).

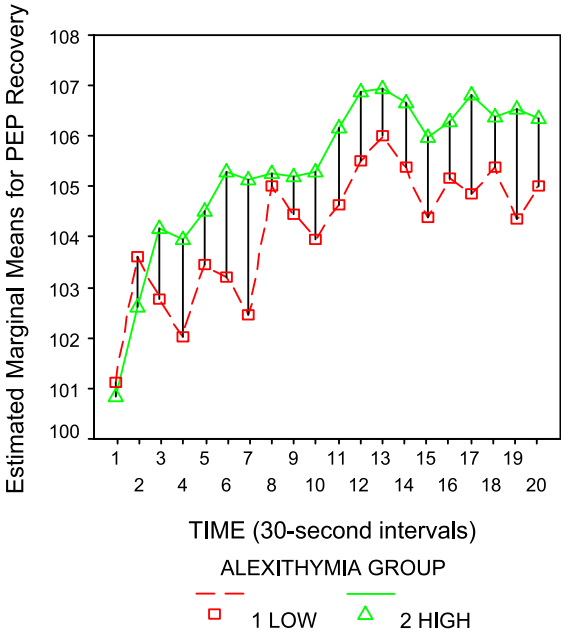

Fig. 2. Alexithymia groups on PEP poststress recovery.

[5] In order to interpret the results of the present study, it is important to note the overall effects of the anger recall task on autonomic function which were described more extensively in a prior report (Neumann et al., in press) and briefly described here. Specifically, the anger recall task elicited significant increases for blood pressure, HR, TPR, LF power, and LF/HF ratio and significant decreases for r-MSSD, CI, SI, and HF power. In other words, the anger recall task elicited a vascular resistance response pattern implying relative increases in alpha-adrenergic sympathetic activity as well as a reduction in parasympathetic activity and a shift in sympathetic autonomic activity.

4.4. Alexithymia and state anger

Pearson *r* correlations were computed to examine the relation between alexithymia and self-reported state anger during the baseline, anger recall and recovery periods. Contrary to our hypotheses, no significant associations were found for alexithymia and state anger (see Table 1).

5. Discussion

The present study examined the relation of alexithymia to autonomic reactivity during a personally relevant anger recall task and poststress recovery using impedance cardiography and heart rate variability measures. Results of the investigation revealed that alexithymia was related to select measures of autonomic dysregulation during and following anger recall. However, alexithymia was not related to higher tonic autonomic function, nor was it associated with state anger at rest or during anger recall.

More specifically, alexithymia was significantly related to attenuated HR and SI reactivity and greater LF power during anger recall. With the exception of Waldstein et al. (2002), our findings are consistent with prior research showing that alexithymia is associated with attenuated physiological reactivity (i.e., Linden et al., 1996; Nemiah et al., 1997; Newton and Contrada, 1994; Wehmer et al., 1995) supporting the hypoarousal model of alexithymia. In contrast to Waldstein et al. (2002), the five studies supporting the hypoarousal model (i.e., the present study, Linden et al., 1996; Nemiah et al., 1997; Newton and Contrada, 1994; Wehmer et al., 1995) consisted of considerably younger participants which may, at least in part, account for the differential reactivity patterns. Interestingly, prior studies examining psychopathology and specifically anxiety disorders have also found attenuated cardiovascular reactivity to stressors (e.g., Friedman and Thayer, 1998). Our results also extend prior research findings by measuring heart rate variability thus allowing for the assessment of differential autonomic influences. The significantly greater LF_L power reactivity found for alexithymics were interpreted by evaluating LF/HF ratios (see Table 2) showing that those with higher alexithymia scores had lower LF/HF ratios than those with lower alexithymia scores (1.19 vs. 1.31) (Malliani et al., 1990). Thus, the blunted cardiovascular reactivity found here appeared to arise from attenuated sympathetic activation and diminished vagal withdrawal.

Alexithymia was also significantly related to slower DBP recovery. In contrast, faster PEP recovery was associated with alexithymia suggesting abbreviated sympathetic arousal as compared to those with lower alexithymia scores. Importantly, these associations were independent of task reactivity levels. However, because effects of the sympathetic nervous system on the heart have been shown to be dependent on the surrounding vagal activity (for review see Uijtdehaage and Thayer, 2000; Levy, 1984; Tulppo et al., 1998), it is key that changes in cardiac reactivity associated with presumed sympathetic activity be interpreted in conjunction with concurrent vagal activity. In this regard, inspection of the normalized and logarithmically transformed values of HF power for task and recovery periods (see Tables 2 and 3) suggests the possibility of increased levels of vagal control of the heart during recovery for those with higher alexithymia scores. While prior research has found delayed physiological poststress recovery related to anxiety disorders and other forms of psychopathology (Friedman and Thayer, 1998), to the authors' knowledge, this is the first study to document that alexithymia is associated with prolonged cardiovascular recovery from anger.

The self-reported state anger ratings were unrelated to alexithymia in this study, which was also found by Waldstein et al. (2002). On the one hand, these findings may indicate that alexithymics were as emotionally engaged in the anger recall task as their counterparts. On the other hand, it is possible that this absence of relation may be due to a response bias. All the participants were directly informed that anger was an expectation during the task. Consequently, this information may have biased the alexithymics' affect ratings. The tendency for alexithymics to have difficulties with identifying and recognizing their emotions may likely be more of a problem when in an ambiguous emotional context. Berenbaum and Irvin (1996) suggest that nonverbal/behavioral ratings of emotional expression may be a more valid measure of state anger than self-reports. Hence, the validity of our self-reported state anger ratings from alexithymics may be considered somewhat questionable due to a possible response expectancy bias. Nonverbal anger

expression measures may be a beneficial measure to include in future research.

Consistent with several prior studies (e.g., Linden et al., 1996; Waldstein et al., 2002; Newton and Contrada, 1994; Wehmer et al., 1995), our results did not yield any significant relations between alexithymia and higher baseline levels of heart rate. In fact, no significant relations were noted between alexithymia and baseline levels of any of the other cardiovascular and autonomic function measures. In contrast, one prior study reported greater tonic sympathetic activity and diminished vagal tone (i.e., greater LF/HF ratios) in a sample of young, Japanese men ($N = 26$) with high TAS scores as compared to their counterparts (Fukunishi et al., 1999). The inconsistencies found between the present findings and the results of this prior study (Fukunishi et al., 1999) may stem from sample differences in gender and/or culture, and/or not experimentally or statistically controlling for body mass.

We also found that alexithymia was associated with greater alcohol intake and body mass index, as has been found in previous work (e.g., Helmers and Mente, 1999). Such maladaptive behaviors and risk factors may serve as mechanisms by which alexithymia is related to cardiovascular disease or general mortality. However, in the present report, the relations found among alexithymia and the cardiovascular measures are independent of self-reported alcohol consumption and body mass index. Additional studies are needed to further understand potential interrelations among alexithymia, other relevant psychosocial and behavioral factors, and cardiovascular morbidity and mortality.

It is important to note several strengths and limitations of the present investigation. With respect to strengths, the racial composition of our female sample is rather unique including 54% Caucasians, 34% African-Americans, and 12% Asian-Americans. Additionally, the present study improved upon prior alexithymia reactivity research by providing both hemodynamic and differential autonomic influences in regulating blood pressure and heart rate and by evaluating poststress recovery. Furthermore, excursion computations provide more reliable recovery estimates than other statistical techniques (e.g., repeated measures ANOVA or time to recover) since they are based on all of the relevant data points and rate of change.

There are also several limitations. First, our findings cannot be extrapolated to men. Although there has been a general paucity of research examining the role of psychosocial factors in relation to autonomic function in women, this method limits the generalizability of our findings. Some studies have noted physiological differences between alexithymic men and women (e.g., Friedlander et al., 1997; Nemiah et al., 1997; Lumley and Sielky, 2000). Second, we sampled one type of stressor specifically targeting the main emotion of anger. Therefore, we are limited in our ability to determine whether our findings are stressor specific. Nonetheless, anger has indeed been associated with increased cardiovascular disease risk (e.g., Friedlander et al., 1997; Nemiah et al., 1997; Newton and Contrada, 1994; Wehmer et al., 1995) and thus pertinent to the study of alexithymia and cardiovascular disease risk. Third, several other psychosocial factors (such as depression, social isolation, and anger-inhibition) and behavioral factors (e.g., sleep disturbance and substance abuse) and noncompliance with medical regimens have been related to alexithymia (see Lumley et al., 1996; Linden et al., 1996) and cardiovascular morbidity (Jula et al., 1999; Kojima et al., 2001; Numata et al., 1998) and all-cause mortality (Kauhanen et al., 1996). All of these factors were not accounted for in the present study and therefore, we cannot ascertain whether the significant relations observed here result from problems with affect regulation or from poor lifestyle habits for example. Fourth, only select measures of cardiovascular reactivity and recovery were found to be significantly related to alexithymia which may be a result of spurious findings, small effect sizes, or it may be explained by an unmeasured variable. However, when accounting for the number of multiple regression analyses performed (by adjusting alpha < 0.01), the relations of alexithymia to select cardiovascular reactivity and recovery remain significant (p's ranging from 0.001 to 0.009). The absence of significant relations among alexithymia and all of the cardiovascular measures may also be due, at least in part, to not having more participants with clinically significant levels of alexithymia ($n = 11$). Samples including more extreme levels of low and high alexithymia may provide a more robust distinction between these groups on cardiovascular reactivity to and recovery from angry events. Moreover, differences in task

performance between individuals with relatively lower versus higher levels of alexithymia may possibly explain the differential cardiovascular reactivity we found. Although we do not have objective measures of speech amount or a count of "keep speaking" prompts, the experimenter was trained to maintain emotional disclosure levels and speech rate as consistent as possible across subjects.

6. Conclusions

In sum, our results suggest that greater levels of alexithymia are related to attenuated autonomic reactivity to angry events, but prolonged recovery following anger in young women. Our findings impart some evidence for the hypoarousal model of alexithymia during anger provoking events, but the hyperarousal model during recovery. Overall, these results suggest that autonomic nervous system dysregulation during and following emotion provocation may, at least in part, play a role in the connection between alexithymia and cardiovascular disease. These findings warrant further replication and explication in future investigations.

Acknowledgements

This research was supported, in part, by the National Institutes of Health Grant AG15112 and HL07560-19.

References

Bagby, R.M., Taylor, G.J., Atkinson, L., 1988. Alexithymia: a comparative study of three self-report measures. J. Psychosom. Res. 32, 107–116.

Bagby, R.M., Parker, J.D.A., Taylor, G.J., 1994. The twenty-item Toronto Alexithymia Scale: I. Item selection and cross validation of the factor structure. J. Psychosom. Res. 38, 23–32.

Berenbaum, H., Irvin, S., 1996. Alexithymia, anger, and interpersonal behavior. Psychother. Psychosom. 65, 203–208.

Berntson, G.G., Cacioppo, J.T., Binkley, P.F., Uchino, B.N., Quigley, K.S., Fieldstone, A., 1994. Autonomic cardiac control: III. Psychological stress and cardiac response in autonomic space as revealed by pharmacological blockades. Psychophysiology 31, 599–608.

Davis, M.G., Matthews, K.A., McGrath, C.E., 2000. Hostile attitudes predict elevated vascular resistance during interpersonal stress in men and women. Psychosom. Med. 62, 17–25.

de Geus, E.J.C., Willemsen, G.H.M., Klaver, C.H.A.M., van Doornen, L.J.P., 1995. Ambulatory measurement of respiratory sinus arrhythmia and respiration rate. Biol. Psychol. 41, 205–227.

Debski, T.T., Kamarck, T.W., Jennings, J.R., Young, L.W., Eddy, M.J., Zhang, Y., 1991. A computerized test battery for the assessment of cardiovascular reactivity. Int. J. Bio-Med. Comput. 27, 277–289.

Erdfelder, E., Faul, F., Buchner, A., 1996. GPOWER: a general power analysis program. Behav. Res. Methods Instrum. Comput. 28, 1–11.

Everson, S.A., McKey, B.S., Lovallo, W.R., 1995. Effect of trait hostility on cardiovascular responses to harassment in young men. Int. J. Behav. Med. 2, 172–191.

Friedlander, L., Lumley, M.A., Farchione, T., Doyal, G., 1997. Testing the alexithymia hypothesis: physiological and subjective responses during relaxation and stress. J. of Nerv. Ment. Dis. 185, 233–239.

Friedman, B.H., Thayer, J.F., 1998. Autonomic balance revisited: panic anxiety and heart rate variability. J. Psychosom. Res. 44, 133–151.

Fukunishi, I., Sei, H., Morita, Y., Rahe, R.H., 1999. Sympathetic activity in alexithymics with mother's low care. J. Psychosom. Res. 46, 579–589.

Haviland, M.G., Shaw, D.G., MacMurray, J.P., Cummings, M.A., 1988. Validation of the Toronto Alexithymia Scale with substance abusers. Psychother. Psychosom. 50, 164–170.

Helmers, K.F., Mente, A., 1999. Alexithymia and health behaviors in healthy male volunteers. J. Psychosom. Res. 47, 635–645.

Ironson, G., Taylor, C.B., Boltwood, M., Bartzokis, T., Dennis, C., Chesney, M., Spitzer, S., Segall, G.M., 1992. Effects of anger on left ventricular ejection fraction in coronary artery disease. Am. J. Cardiol. 70, 281–285.

Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, 1997. The sixth report of the Joint National Committee on detection, evaluation, and treatment of high blood pressure (JNC V). Arch. Int. Med. 157, 2413–2446.

Jula, A., Salminen, J.K., Saarijarvi, S., 1999. Alexithymia: a facet of essential hypertension. Hypertension 33, 1057–1061.

Kamarck, T.W., Jennings, J.R., Debski, T.T., Glickman-Weiss, E., Johnson, P.S., Eddy, M.J., Manuck, S.B., 1992. Reliable measures of behaviorally-evoked cardiovascular reactivity from a PC-based battery: results from student and community samples. Psychophysiology 29, 17–28.

Kamarck, T.W., Jennings, J.R., Manuck, S.B., 1993. Psychometric applications in the assessment of cardiovascular reactivity. Homeostatis 34, 5–6.

Kauhanen, J., Julkunen, J., Salonen, J.T., 1991. Alexithymia and perceived symptoms: criterion validity of the Toronto Alexithymia Scale. Psychother. Psychosom. 56, 247–252.

Kauhanen, J., Kaplan, G.A., Cohen, R.A., Julkunen, J., Salonen, J.T., 1996. Alexithymia and risk of death in middle-aged men. J. Psychosom. Res. 41, 541–549.

Klaver, C.H.A.M., de Geus, E.J.C., de Vries, J., 1994. Ambulatory monitoring system. In: Maarse, F.J. (Ed.), Computers in Psychology. Applications, Methods and Instrumentations, vol. 5. Swets and Zeitlinger, Lisse, pp. 254–268.

Kojima, M., Frasure-Smith, N., Lesperance, F., 2001. Alexithymia following myocardial infarction: psychometric properties and correlates of the Toronto Alexithymia Scale. J. Psychosom. Res. 51, 487–495.

Kop, W.J., 1999. Chronic and acute psychological risk factors for the clinical manifestations of coronary artery disease. Psychosom. Med. 61, 476–487.

Kop, W.J., 2003. The integration of cardiovascular behavioral medicine and psychoneuroimmunology: new developments based on converging research fields. Brain Behav. Immun. 17, 233–237.

Kubicek, W.G., Karnegis, J.N., Patterson, P.P., Witsoe, D.A., Mattson, R.H., 1966. Development and evaluation of an impedance cardiograph system. Aerosp. Med. 37, 1208–1212.

Levy, M.N., 1984. Cardiac sympathetic–parasympathetic interactions. Fed. Proc. 43, 2598–2602.

Linden, W., Lenz, J.W., Stossel, C., 1996. Alexithymia, defensiveness and cardiovascular reactivity to stress. J. Psychosom. Res. 41, 575–583.

Lumley, M.A., Norman, S., 1996. Alexithymia and healthcare utilization. Psychosom. Med. 58, 197–202.

Lumley, M.A., Sielky, K., 2000. Alexithymia, gender, and hemispheric functioning. Compr. Psych. 41, 352–359.

Lumley, M.A., Stettner, L., Wehmer, F., 1996. How are alexithymia and physical illness linked? A review and critique of pathways. J. Psychosom. Res. 41, 505–518.

Malliani, A., Lombardi, F., Pagani, M., Cerutti, S., 1990. Clinical exploration of the autonomic nervous system by means of electrocardiography. Ann. N.Y. Acad. Sci. 601, 234–246.

Malliani, A., Pagani, M., Lombardi, F., 1994. Physiology and clinical implications of variability of cardiovascular parameters with focus on heart rate and blood pressure. Am. J. Cardiol. 73, 3C–9C.

Nemiah, J.C., Sifneos, P.E., Apfel-Savitz, R., 1997. A comparison of oxygen consumption of normal and alexithymic subjects in response to affect-provoking thoughts. Psychother. Psychosom. 28, 167–171.

Neumann, S.A., Waldstein, S.R., 2001. Similar patterns of cardiovascular response during emotional activation as a function of affective valence and arousal and gender. J. Psychosom. Res. 50, 245–253.

Neumann, S.A., Waldstein, S.R., Sollers III, J.J., Thayer, J.F., Sorkin, J.D., in press. Hostility and distraction have differential influences on cardiovascular recovery responses to anger recall in women. Health Psychol.

Newton, L.T., Contrada, R.J., 1994. Alexithymia and repression: contrasting emotion-specific coping styles. Psychosom. Med. 56, 457–462.

Numata, Y., Ogata, Y., Oike, Y., Matsumura, T., Shimada, K., 1998. A psychobehavioral factor, alexithymia, is related to coronary spasm. Jpn. Circ. J. 62, 409–413.

Pagani, M., Rimoldi, O., Malliani, A., 1992. Low-frequency components of cardiovascular variabilities as markers of sympathetic modulation. Trends Pharmacol. Sci. 13, 50–54.

Prkachin, K.M., Mills, D.E., Zwaal, C., Husted, J., 2001. Comparison of hemodynamic responses to social and nonsocial stress: evaluation of an anger interview. Psychophysiology 38, 879–885.

Protter, M.H., Morrey, C.B., Jr., 1970. College calculus with analytic geometry. Addison-Wesley Series in Mathematics. Addison-Wesley, Reading, MA, pp. 255–266 and 544–547.

Rozanski, A., Blumenthal, J.A., Kaplan, J., 1999. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. Circulation 99, 2192–2217.

Sherwood, A., Allen, M.T., Fahrenberg, J., Kelsey, R.M., Lovallo, W.R., van Doornen, L.J.P., 1990. Methodological guidelines for impedance cardiography. Psychophysiology 27, 1–23.

Sifneos, P.E., 1973. The prevalence of 'alexithymic' characteristics in psychsomatic patients. Psychother. Psychosom. 22, 255–262.

Sinha, R., Lovallo, W.R., Parsons, O.A., 1992. Cardiovascular differentiation of emotions. Psychosom. Med. 54, 422–435.

Sloan, R.P., Shapiro, P.A., Bagiella, E.B., Myers, M.M., Gorman, J.M., 1999. Cardiac autonomic control buffers blood pressure variability responses to challenge: a psychophysiologic model of coronary artery disease. Psychosomat. Med. 61, 58–68.

Spielberger, C.D., 1988. Manual for the State-Trait Anger Expression Inventory (STAXI). Psychological Assessment Resourses, Odessa, FL.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Circulation 93, 1043–1065.

Taylor, G.J., Ryan, D., Bagby, R.M., 1985. Toward the development of a new self-report alexithymia scale. Psychother. Psychosom. 44, 191–199.

The National Heart Lung, and Blood Institute Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults, 1998. Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. J. Am. Diet Assoc. 98, 1178–1191.

Tulppo, M.P., Maekikallio, T.H., Seppaenen, T., Airaksinen, H.V., Huikuri, H.V., 1998. Heart rate dynamics during accentuated sympathovagal interaction. Am. J. Physiol. 274, H810–H816.

Uijtdehaage, S.H., Thayer, J.F., 2000. Accentuated Antagonism in the control of human heart rate. Clinic. Auton. Res. 10, 107–110.

Valkamo, M., Hintikka, J., Honkalampi, K., Niskanen, L., Koivumaa-Honkanen, H., Viinamaki, H., 2001. Alexithymia in patients with coronary heart disease. J. Psychosom. Res. 50, 125–130.

Waldstein, S.R., Kop, W.J., Schmidt, L.A., Haufler, A.J., Krantz, D.S., Fox, N.A., 2000. Frontal electrocortical and cardiovascular reactivity during happiness and anger. Biol. Psychol. 5, 3–23.

Waldstein, S.R., Kauhanen, J., Neumann, S.A., Katzel, L.I., 2002. Alexithymia and cardiovascular risk in older adults: psychoso-

cial, psychophysiological, and biomedical correlates. Psychol. Health 17, 597–610.

Wehmer, F., Brejnak, C., Lumley, M.A., Stettner, L., 1995. Alexithymia and physiological reactivity to emotion-provoking visual scenes. J. of Nerv. Ment. Dis. 183, 351–357.

Weinberger, D.A., Schwartz, G.E., Davidson, R.J., 1979. Low-anxious, high-anxious, and repressive coping styles: psychometric patterns and behavioral and physiological responses to stress. J. Abnorm. Psychology 88, 369–380.