Eur Arch Psychiatry Clin Neurosci (2007) 257:325–329 DOI 10.1007/s00406-007-0724-4

ORIGINAL PAPER

Kosuke Narita · Tetsuhito Murata · Tetsuya Takahashi · Toshihiko Hamada · Hirotaka Kosaka
Haruyoshi Yoshida · Yuji Wada

The association between anger-related personality trait and cardiac autonomic response abnormalities in elderly subjects

Received: 17 July 2006 / Accepted: 22 January 2007 / Published online: 1 April 2007

Abstract Cardiac autonomic response abnormality associated with trait anger has been recognized to elevate blood pressure in daily life, leading to atherosclerotic progression and cardiovascular disease. To clarify the relationship between anger-related personality traits and cardiac autonomic response in healthy elderly subjects, 54 volunteers consisting of 30 male (mean age 62.2 ± 5.4) and 24 female (mean age 58.4 ± 4.6) subjects underwent testing of heart rate variability (HRV) with head-up tilt. For the evaluation of trait anger, we used a questionnaire corresponding to the trait anger score taken from the State and Trait Anger Expression Inventory. Furthermore, we measured carotid intima-medial thickness (IMT) to evaluate atherosclerotic progression in subjects with anger trait. In female subjects, higher trait anger was positively associated with elevated carotid IMT and the suppression of HRV vagal attenuation from the supine to head-up position, and negatively associated with the HRV sympathetic activity in the head-up position and also with the HRV sympathetic response from the supine to head-up position. In male subjects, trait anger was not significantly associated with carotid IMT or any HRV component with or without head-up tilt testing. We conclude that a simple noninvasive measure, short-term HRV with head-up tilt testing, could be a useful method to investigate the association between cardiac autonomic imbalance and increased risk of atherosclerosis associated with trait anger in healthy elderly subjects.

Key words anger personality trait · heart rate variability · autonomic response · postmenopausal female subjects · intima-medial thickness

K. Narita, MD, PhD · T. Murata, MD, PhD (✉)
T. Takahashi, MD, PhD · H. Kosaka, MD, PhD · Y. Wada, MD, PhD
Department of Neuropsychiatry
University of Fukui
Fukui, Japan
Tel.: +81-776/61-8363
Fax: +81-776/61-8136
E-Mail: tmurata@fmsrsa.fukui-med.ac.jp

T. Hamada, PhD · H. Yoshida, MD, PhD
Department of Clinical and Laboratory Science
University of Fukui
Fukui, Japan

Introduction

Short-term recording (usually 2–15 min) of supine heart rate variability (HRV), a noninvasive and convenient measure of cardiac autonomic function, has been used as an indicator of cardiovascular health. Reduced HRV, reflecting abnormalities of cardiac sympatho-vagal modulation, is a predictor of all-cause mortality, arrhythmic events, and sudden death after acute myocardial infarction [1]. However, normal cardiac autonomic functioning is also reflected by the shift from parasympathetic to sympathetic modulation in response to environmental or physiologic stimuli. The HRV response to gravitational stress imposed by postural change is a sensitive measure of the shift in cardiac autonomic balance from parasympathetic predominance at rest to sympathetic predominance, to accommodate the increased cardiac demands of standing [4]. As a laboratory technique to elicit sympathetic activation, postural change has been recognized as a simple noninvasive procedure that is readily quantifiable and correlates well with changes of reproducible behavioral and hemodynamic condition [13].

Anger-related personality traits have been shown to be associated with atherosclerosis and cardiovascular disease (CVD) [2, 7]. Negative affect composed of anger elevates blood pressure (BP) temporarily, which increases atherosclerotic changes and cardiovascular mortality and morbidity [3, 9]. Transient BP elevation associated with anger is suggested to result from predominant sympathetic nerve activation [12].

However, a recent study using the method of short-term recording of supine HRV showed no significant association between anger-related personality traits and HRV components [27]. Most investigations that have shown significant associations between anger-related personality traits and HRV components have used the methods of the long-term 24-h recording of HRV or the HRV response to mental stress [10, 20]. Few studies have examined the relationship between anger-related personality traits and the short-term HRV response to postural change.

The objective of our preliminary study was to examine the association between trait anger and the cardiovascular response of HRV with head-up tilt testing in elderly subjects. In addition, we measured carotid intima-medial thickness (IMT) to evaluate atherosclerotic progression in subjects with anger trait [22]. Measurement of carotid IMT employs B-mode ultrasonography, which can detect morphological changes of the carotid artery, consisting of both an intimal atherosclerotic process and medial hypertrophy [14].

Methods

Subjects

Fifty-four healthy elderly subjects (30 males, mean age 62.2 ± 5.4; 24 females, mean age 58.4 ± 4.6) were recruited from general inhabitants in Fukui prefecture, Japan, using a brochure that described the following criteria for exclusion: history of major atherosclerotic risk factors (such as hypertension, hypercholesterolemia, diabetes mellitus), history of CVD (coronary artery disease, congestive heart failure, or hemodynamically significant valvular disease), history of neurological or psychiatric illness, chronic alcoholism, smoking, obesity with a body mass index (BMI) above 26, and continuous administration of drugs. Only postmenopausal females were included as female subjects. All subjects gave their written informed consent. The protocol of this study was approved by the ethics committee of our university.

Experimental procedure

All subjects underwent BP measurement in the clinic three times (at about 10:00 a.m.) after a 10-min rest in the supine position, and the mean value was calculated as systolic or diastolic BP. The subjects then underwent venous blood sampling from an antecubital vein in the right arm before noon without having eaten breakfast to measure the serum concentration of total cholesterol (TC). After lunch, at 1:00 p.m., for the evaluation of anger-related personality profiles, subjects completed a questionnaire of 10 items (each item consisting of 4-point scale) corresponding to the trait anger score taken from the State and Trait Anger Expression Inventory (STAXI) constructed and validated by Spielberger [23], and a lower score reflected a better psychological status. At 2:30 p.m., the subjects underwent a 30-min ECG for HRV (15 min in the supine position and 15 min in the head-up position). The subjects subsequently underwent B-mode ultrasonographic measurement of carotid artery IMT.

Power spectral analysis of HRV with head-up tilt testing protocol

In a quiet and comfortable environment, the subjects were instructed to lie in the supine position on a bed. After allowing 10 min for stabilization, the ECG was recorded for 15 min as a baseline condition (supine). Afterward, the subjects underwent head-up tilt testing, a passive orthostatic maneuver achieved with a motorized tilt upright (90°). The subjects then underwent ECG (head-up) during a 15-min upright session. During ECG in both positions, the subjects maintained their breathing at the fixed rate of 0.25 Hz in tempo with the sound of a metronome, because the respiratory frequency influences the HRV [21].

Power spectral analysis of the recording of HRV for 256 consecutive seconds was performed sequentially with a fast Fourier transform using a Hamming window (Fukuda-Denshi Co., Tokyo, Japan). The following frequency components of HRV were calculated: the area under the low-frequency (LF component, 0.04–0.15 Hz), high-frequency (HF component, 0.16–0.45 Hz) portions of the spectrum. These components were obtained in absolute values of power (ms^2). HF corresponds to vagally mediated modulation of HRV, and LF corresponds to baroreflex control of the heart rate and reflects mixed sympathetic and parasympathetic modulation of HRV [18]. The LF/HF ratio, as an estimate of sympathovagal balance, was also calculated from the absolute power of both frequency components [25]. ΔLF, ΔHF and ΔLF/HF were also calculated as the difference between the values in the head-up position and the supine position. Because the distribution of the measurements of HRV was skewed, the log transformation of each measure, which produces normal distributions, was applied.

Carotid B-mode ultrasonography

Carotid B-mode ultrasonography was performed using a LOGIQ500 MD MR3 (General Electric Medical Systems), and carotid atherosclerosis was examined using an 8.8-MHz wideband transducer. The carotid IMT was measured as the distance between the lumen-intima interface and the media-adventitia interface at the far wall on each side in the B-mode image according to the method of Handa et al. [6].

Statistical analysis

Statistical analysis was carried out using SPSS software for Windows version 9 (SPSS Japan Inc., Tokyo, Japan). The t-test was used to assess the significance of differences between gender groups. Simple correlation analyses and an analysis of variance of a general linear model were performed to evaluate the relationships among trait anger score, HRV components, carotid IMT, age, BMI, systolic/diastolic BP and serum TC concentration in all subjects, male subjects or female subjects. Furthermore, one-way analysis of variance (ANOVA) was performed to examine the relationships between the high- and low-trait anger subgroups among female subjects with high and low trait anger score.

Results

The demographic characteristics (i.e. age and education), biological parameters (i.e. BMI, systolic/diastolic BP and TC), trait anger score and HRV components (data not shown) showed no significant difference between the males and females. The diastolic BP of male subjects (80.3 ± 8.1 mmHg) was higher than that of female subjects (75.1 ± 10.4 mmHg) ($p < 0.05$). Simple correlation analysis failed to show a significant association between trait anger score and any biological index (age, BMI, systolic/diastolic BP or the serum concentration of TC) in all subjects or in male or female subjects (data not shown).

Table 1 shows the associations between trait anger score and HRV components. There was no significant

Table 1 The association between trait anger score and HRV components

	STAXI trait anger score		
	All subjects	Male (n = 30)	Female (n = 24)
HRV (ln ms^2)			
LF_{supine}	−0.082	0.334	0.192
HF_{supine}	0.088	0.089	0.177
LF/HF_{supine}	−0.176	0.213	−0.027
$LF_{head\text{-}up}$	0.011	0.207	−0.107
$HF_{head\text{-}up}$	0.054	0.154	0.490*
$LF/HF_{head\text{-}up}$	−0.043	−0.046	−0.576**
ΔLF	0.101	−0.137	−0.393
ΔHF	−0.049	0.107	0.449*
ΔLF/HF	0.067	−0.224	−0.534**
IMT (mm)	−0.068	−0.256	0.597**

* $p < 0.05$, ** $p < 0.01$ (Pearson's product–moment correlation coefficients (= r))

Table 2 Comparison between low and high STAXI trait anger group in elderly female subjects

	STAXI trait anger score		
	Low group <15	High group ≧15	p-value
Age (year)	57.2 ± 5.1	59.9 ± 5.3	NS
BMI (kg/m^2)	22.7 ± 1.9	22.8 ± 2.5	NS
Systolic BP (mmHg)	124.7 ± 15.5	122.2 ± 9.0	NS
Diastolic BP (mmHg)	79.2 ± 10.9	71.5 ± 8.3	NS
Total cholesterol (mg/dl)	230.3 ± 29.7	219.3 ± 34.4	NS
HRV (ln ms^2)			
HF_{supine}	2.18 ± 0.40	2.25 ± 0.75	NS
LF/HF_{supine}	0.95 ± 0.14	1.01 ± 0.18	NS
$HF_{head\text{-}up}$	1.57 ± 0.35	2.05 ± 0.65	<0.05
$LF/HF_{head\text{-}up}$	1.39 ± 0.33	1.08 ± 0.25	<0.05
ΔHF	−0.60 ± 0.36	−0.21 ± 0.31	<0.01
ΔLF/HF	0.44 ± 0.35	0.07 ± 0.21	<0.01
IMT (mm)	0.84 ± 0.05	0.91 ± 0.09	<0.05

Values are mean ± SD

association between trait anger score and any HRV component in all subjects or in male subjects. In female subjects, trait anger score was positively correlated with $HF_{head\text{-}up}$ ($r = 0.490$, $p < 0.05$) and ΔHF ($r = 0.449$, $p < 0.05$), and negatively correlated with $LF/HF_{head\text{-}up}$ ($r = -0.576$, $p < 0.01$) and ΔLF/HF ($r = -0.534$, $p < 0.01$). In addition, trait anger score in female subjects was positively correlated with carotid IMT ($r = 0.597$, $p < 0.01$). Using analysis of variance in a general linear model, trait anger in female subjects was significantly associated with $HF_{head\text{-}up}$ ($F = 6.964$, $p = 0.015$), $LF/HF_{head\text{-}up}$ ($F = 10.902$, $p = 0.003$), ΔHF ($F = 5.558$, $p = 0.028$) and ΔLF/HF ($F = 8.790$, $p = 0.007$). Also, anger trait in females was significantly associated with carotid IMT ($F = 12.162$, $p = 0.002$).

For trait anger score, female subjects were classified into two groups according to whether the score was higher or lower than the median (high-trait anger and low-trait anger groups), and HRV components (HF and LF/HF) were compared between the two groups (Table 2). Using one-way ANOVA, the high-trait anger group showed higher $HF_{head\text{-}up}$ and lower ΔHF than the low-trait anger group ($F = 4.827$, $p < 0.05$ and $F = 8.283$, $p < 0.01$, respectively). Also, the high-trait anger group showed lower $LF/HF_{head\text{-}up}$ and lower ΔLF/HF than the low-trait anger group ($F = 7.224$, $p < 0.05$ and $F = 9.975$, $p < 0.01$, respectively). Furthermore, the high-trait anger group showed higher carotid IMT than the low-trait anger group ($F = 4.350$, $p < 0.05$).

Discussion

Our results did not show significant associations between anger trait score and HRV components in all subjects or male subjects. On the other hand, trait anger score was positively associated with ΔHF (or $HF_{head\text{-}up}$) and negatively associated with ΔLF/HF (or $LF/HF_{head\text{-}up}$) in female subjects (Tables 1 and 2).

Since a postural change causes sympathetic activation and vagal withdrawal, the HF power and normalized unit LF (i.e. LF/HF ratio) response to postural change must decrease or increase, respectively [15]. Additionally, HF power is a major contributor to vagally mediated modulation of HRV [18]. Thus, ΔHF could be considered to reflect the amplitude of vagal attenuation due to postural change. Since the increase in LF/HF during head-up tilt is inhibited by β-blockers [15, 18], ΔLF/HF is considered to reflect a sympathetic nerve response to the change from supine to head-up. Attenuated ΔHF and ΔLF/HF have also been identified in subjects with high anxiety symptoms [17] and in some categories of hypertensive and diabetic subjects [5, 16], who have the common feature of predominant sympathetic activity at rest and have been reported to show lower HF power and higher LF/HF ratio in supine HRV than controls. Thus, enhanced baseline sympathetic activity could be thought to cause a smaller amplitude of sympathetic activation due to postural change. Considering our results together with these reports, the decreased ΔHF and ΔLF/HF might indicate that subjects with high trait anger in the female group have a sympathetic predominance at baseline. However, our results failed to observe a significant association between trait anger score and HRV components at baseline, namely LF_{supine}, HF_{supine} and LF/HF_{supine}. Also, a recent report using short-term (256 s) measurement of supine HRV in a population-based study demonstrated no significant association between anger-related personality scores and supine HRV components [27]. To explain this, the following possibilities could be considered. First, HRV with head-up tilt testing might emphasize individual differences of supine HRV because of its sensitive measurement of the shift in cardiac autonomic balance from supine to head-up [4]. Another possibility is that subjects with high trait anger might tend to

have a sympatho-vagal response abnormality, which could decrease the amplitude of sympathetic activation and vagal attenuation due to postural change, resulting in attenuation of ΔHF and ΔLF/HF.

Our results showed a significant positive association between anger trait score and carotid IMT in female subjects (Tables 1 and 2). Also, some previous investigations demonstrated that high anger trait is positively associated with elevated carotid IMT [2, 11, 19]. These findings agree with other previous reports that subjects with high anger trait have increased risk of atherosclerosis [2, 7], because carotid IMT has been reported to be correlated with coronary atherosclerosis and to predict stroke and myocardial infarction [22].

As pathophysiological mechanisms linking trait anger and carotid IMT, several possible hypotheses could be proposed. Raikkonen et al. [19] suggested that trait anger in postmenopausal women might elevate carotid IMT through the development of metabolic syndrome. Considering some previous reports together with our results regarding the relationship between trait anger and HRV components with head-up tilt, it could be speculated that cardiac autonomic response abnormalities associated with trait anger evoke elevated BP and homodynamic irregularity in daily life, leading to atherosclerosis [12].

Some gender differences were found between trait anger and physiological indices in this study. Trait anger was positively associated with carotid IMT only in female subjects, but not male subjects. Several previous studies showed that elevated carotid IMT was correlated with trait anger in postmenopausal women [19], Japanese males and females [11], and untreated hypertensive men [2], but these studies enrolled subjects with traditional CVD risk factors, such as smoking, hypertension or hypercholesterolemia. Also, significant associations in this study were observed between trait anger and HRV components only in female subjects, but not males. There have been few reports investigating gender differences in the relationship between anger-related personality traits and short-term HRV. A previous report using a method of 24-h recording of HRV demonstrated that anger-out score was associated with both higher LF and HF power in males (18–50 years old) but not females [20], and another report showed that an inability to relieve anger by talking to others (anger-discuss) was associated with standard deviation of normal-to-normal intervals (SDNN) and LF power in female subjects [8]. The reasons why some gender differences in the relationship between anger trait and HRV components were found in this study are unclear, but these previous reports regarding associations between anger trait and HRV components or carotid IMT varied in the subject sampling method, psychological assessment, and experimental tool (i.e. 24-h HRV recording method), which may have contributed to the inconsistencies among the results. As another possible reason, the contribution of gender difference in insulin resistance to anger-related personality trait might be proposed. A recent report demonstrated that anger-related personality trait is associated with insulin resistance in healthy females, but not males [24]. Since insulin resistance interacts with predominant sympathetic activity and is recognized as a denominator of metabolic syndrome, which is strongly related to atherosclerotic progression [26], it might be speculated that a greater association between insulin resistance and anger-related personality trait in females than in males leads to these gender differences in HRV components or carotid IMT. Regrettably, we lack definite information to further discuss this point, because estimation of insulin resistance was not done in this study.

This study has major limitations, which are the small sample size and cross-sectional study design. Moreover, the reasons why some gender differences were found between anger trait and HRV components or carotid IMT in this study are unclear. To clarify the causal relationship and also gender differences between anger-related personality traits and HRV response abnormalities or carotid IMT, a prospective investigation with a larger sample size and using short-term HRV with head-up tilt testing in both males and females should be conducted in the future.

In conclusion, our results suggest that a simple noninvasive measure, short-term HRV with head-up tilt testing, might be a useful method to investigate the association between cardiac autonomic imbalance and increased risks of atherosclerosis associated with trait anger in healthy elderly subjects.

References

1. Bigger JT, Fleiss JL, Rolnitzky LM, Steinman RC (1993) The ability of several short-term measures of RR variability to predict mortality after myocardial infarction. Circulation 88:927–934
2. Bleil ME, McCaffery JM, Muldoon MF, Sutton-Tyrrell K, Manuck SB (2004) Anger-related personality traits and carotid artery atherosclerosis in untreated hypertensive men. Psychosom Med 66:633–639
3. Brown DE, James GD, Nordloh L (1998) Comparison of factors affecting daily variation of blood pressure in Filipino-American and Caucasian nurses in Hawaii. Am J Phys Anthropol 106:373–383
4. Carnethon MR, Liao D, Evans GW, Cascio WE, Chambless LE, Heiss G (2002) Correlates of the shift in heart rate variability with an active postural change in a healthy population sample: the atherosclerosis risk in communities study. Am Heart J 143:808–813
5. Guzzetti S, Piccauga E, Casati R, Cerutti S, Lombardi F, Pagani M, Malliani A (1988) Sympathetic predominance in essential hypertension: a study employing spectral analysis of heart rate variability. J Hypertens 6:711–717
6. Handa N, Matsumoto M, Maeda H, Hougaku H, Ogawa S, Fukunaga R, Yoneda S, Kimura K, Kamada T (1990) Ultrasonic evaluation of early carotid atherosclerosis. Stroke 21:1567–1572

7. Harris KF, Matthews KA, Sutton-tyrrell K, Kuller LH (2003) Association between psychological traits and endothelial function in postmenopausal women. Psychosom Med 65:402–409
8. Horsten M, Ericson M, Perski A, Wamala SP, Schenck-Gustafsson K, Orth-Gomer K (1999) Psychosocial factors and heart rate variability in healthy women. Psychosom Med 61:49–57
9. Kikuya M, Hozawa A, Ohokubo T, Tsuji I, Michimata M, Matsubara M, Ota M, Nagai K, Araki T, Satoh H, Ito S, Hisamichi S, Imai Y (2000) Prognostic significance of blood pressure and heart rate variabilities: the Ohasama study. Hypertension 36:901–906
10. Laude D, Girard A, Consoli S, Mounier-Vehier C, Elghozi JL (1997) Anger expression and cardiovascular reactivity to mental stress: a spectral analysis approach. Clin Exp Hypertens 19:901–911
11. Matsumoto Y, Uyama O, Shimizu S, Michishita H, Mori R, Owada T, Sugita M (1993) Do anger and aggression affect carotid atherosclerosis? Stroke 24:983–986
12. McCraty R, Atkinson M, Tiller WA, Rein G, Watkins AD (1995) The effects of emotions on short-term power spectrum analysis of heart rate variability. Am J Cardiol 76:1089–1093
13. Montano N, Ruscone TG, Porta A, Lombardi F, Pagani M, Malliani A (1994) Power spectrum analysis of heart rate variability to assess the changes in sympathovagal balance during graded orthostatic tilt. Circulation 90:1826–1831
14. O'Leary DH, Polak JF, Kronmal RA, Savage PJ, Borhani NO, Kittner SJ, Tracy R, Gardin JM, Price TR, Furberg CD (1996) Thickening of the carotid wall. A marker for atherosclerosis in the elderly? Cardiovascular Health Study Collaborative Research Group Stroke 27:224–231
15. Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, Pizzinelli P, Sandrone G, Malfatto G, Dell'Orto S, Piccaluga E (1986) Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. Circ Res 59:178–193
16. Pagani M, Malfatto G, Pierini S, Casati R, Masu AM, Poli M, Guzzetti S, Lombardi F, Cerutti S, Malliani A (1988) Spectral analysis of heart rate variability in the assessment of autonomic diabetic neuropathy. J Auton Nerv Syst 23:143–153
17. Piccirillo G, Elvira S, Bucca C, Viola E, Cacciafesta M, Marigliano V (1997) Abnormal passive head-up tilt test in subjects with symptoms of anxiety power spectral analysis study of heart rate and blood pressure. Int J Cardiol 60:121–131
18. Pomeranz B, Macaulay RJ, Caudill MA, Kutz I, Adam D, Gordon D, Kilborn KM, Barger AC, Shannon DC, Cohen RJ (1985) Assessment of autonomic function in humans by heart rate spectral analysis. Am J Physiol 248:H151–H153
19. Raikkonen K, Matthews KA, Sutton-Tyrrell K, Kuller LH (2004) Trait anger and the metabolic syndrome predict progression of carotid atherosclerosis in healthy middle-aged women. Psychosom Med 66:903–908
20. Ramaekers D, Ector H, Demyttenaere K, Rubens A, Van de Werf F (1998) Association between cardiac autonomic function and coping style in healthy subjects. Pacing Clin Electrophysiol 21:1546–1552
21. Sakakibara M, Takeuchi S, Hayano J (1994) Effect of relaxation training on cardiac parasympathetic tone. Psychophysiology 31:223–228
22. Simon A, Gariepy J, Chironi G, Megnien JL, Levenson J (2002) Intima-media thickness: a new tool for diagnosis and treatment of cardiovascular risk. J Hypertens 20:159–169
23. Spielberger CD (1988) State-trait anger expression inventory, STAXI, Professional Manual. Odessa, FL, Psychological Assessment Resources
24. Suarez EC (2006) Sex differences in the relation of depressive symptoms, hostility, and anger expression to indices of glucose metabolism in nondiabetic adults. Health Psychol 25:484–492
25. Task Force of the European Society of Cardiology, the North American Society of Pacing and Electrophysiology (1996) Heart rate variability: standards of measurement, physiological interpretation and clinical use. Circulation 93:1043–1065
26. Tataranni PA (2005) Metabolic syndrome: is there a pathophysiological common denominator? World Rev Nutr Diet 94:75–83
27. Virtanen R, Jula A, Salminen JK, Voipio-Pulkki LM, Helenius H, Kuusela T, Airaksinen J (2003) Anxiety and hostility are associated with reduced baroreflex sensitivity and increased beat-to-beat blood pressure variability. Psychosom Med 65:751–756