

Published in final edited form as:

Biol Psychol. 2011 March ; 86(3): 230–238. doi:10.1016/j.biopsycho.2010.12.003.

Autonomic nervous system reactivity to positive and negative mood induction: The role of acute psychological responses and frontal electrocortical activity

Willem J. Kop, Ph.D.^{1,2}, Stephen J. Synowski, Ph.D.¹, Miranda E. Newell, M.S.², Louis A. Schmidt, Ph.D.³, Shari R. Waldstein, Ph.D.^{4,1}, and Nathan A. Fox, Ph.D.⁵

¹Department of Medicine, University of Maryland, 22 S. Greene Str. Baltimore, MD 21201, USA

²Department of Medical and Clinical Psychology, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Road, Bethesda, MD 20814, USA

³Department of Psychology Neuroscience and Behaviour, McMaster University, 1280 Main Street West, Hamilton, Ont. L8S 4K1, Canada

⁴Department of Psychology, University of Maryland, Baltimore County, 1000 Hilltop Circle, Baltimore, MD 21250, USA

⁵Department of Human Development, University of Maryland, College Park, MD 20742, USA

Abstract

The differential effects of positive versus negative emotions on autonomic nervous system activity are insufficiently understood. This study examined the role of acute mood responses and central nervous system activity on heart rate variability (HRV) using 5-min event recall tasks (happiness and anger recall) and a 5-min Stroop Color Word Test (SCWT) in 20 healthy individuals (mean age 25±4 years, 55% female). HRV was measured in high frequency (HF) and low frequency (LF) domains, and frontal brain activity using electroencephalography (EEG) in the alpha frequency band in F3 and F4. Happiness Recall resulted in increased LF-HRV ($p=0.005$) but not HF-HRV ($p=0.71$). Anger Recall did not change HRV (p -values >0.10). The SCWT produced decreases in HF-HRV ($p=0.001$) as well as LF-HRV ($p=0.001$). The magnitude of feeling “happy” during Happiness Recall was positively correlated with Δ HF-HRV ($p=0.050$), whereas an incongruent mood state (“frustrated”) was associated with smaller Δ HF-HRV ($p=0.070$). Associations between frontal EEG activation and HRV responses were mostly non-significant, except for increased right frontal activation during Happiness Recall which was associated with a decrease in LF/HF ratio ($p=0.009$). It is concluded that positive and negative mood induction result in differential HRV responses, which is related to both task valence and the intensity of task-induced emotions.

Keywords

Autonomic nervous system; heart rate variability; emotion; electroencephalography; brain; negative affect; positive affect

© 2010 Elsevier B.V. All rights reserved.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

INTRODUCTION

Intense emotional arousal can trigger the onset of acute coronary syndromes such as myocardial infarction and sudden cardiac death (Samuels, 2007; Mittleman et al., 1995; Krantz, Kop, Santiago, & Gottdiener, 1996). These adverse cardiac events following emotional perturbations may result from sympathetic nervous system activation and its cardiovascular correlates (reactivity), thereby potentially triggering coronary events via myocardial ischemia and a lowered threshold for ventricular arrhythmias (Krantz et al., 1996; Lane et al., 2005). Evidence further suggests that mental stress-induced myocardial ischemia during ambulatory assessments is precipitated by reduced parasympathetic activity (Kop et al., 2001). The autonomic nervous system correlates of emotions purportedly differ with the valence (i.e., positive or negative states) of emotions (Schwartz, Weinberger, & Singer, 1981; Ekman, Levenson, & Friesen, 1983; Sinha, Lovallo, & Parsons, 1992; Bradley, Codispoti, Cuthbert, & Lang, 2001; Kreibig, 2010), which may have implications for the pathophysiological mechanisms of emotion-triggered cardiac events.

Variables that importantly influence emotional responses involve valence and arousal (Russell, 1980; Diener, Larsen, Levine, & Emmons, 1985). Research on psychological risk factors for cardiovascular and other adverse health outcomes has primarily focused on states with negative emotional valence combined with high levels on the arousal dimension such as anger, distress, anxiety and depression (Suls & Bunde, 2005; Rozanski, Blumenthal, & Kaplan, 1999; Kop, 1999). An increasing body of literature indicates that psychological traits and states characterized by positive emotions are associated with reduced risk of clinical cardiovascular disease (Pressman & Cohen, 2005). However, some evidence also suggests that acute positive emotions (happiness, excitement) can act as triggers of acute coronary syndromes (Samuels, 2007; Phillips, Jarvinen, Abramson, & Phillips, 2004; Engel, 1971). It is possible that emotion-specific differential autonomic nervous correlates play a role in these varying cardiovascular consequences of negative and positive emotions. Markers of autonomic nervous system activity are useful in determining the arousal component of emotional responses (Lang, Greenwald, Bradley, & Hamm, 1993), whereas assessments of brain activity are preferred to differentiate the valence of emotional responses (Marci, Glick, Loh, & Dougherty, 2007; see also Posner et al., 2009). Brain circuits respond differently to aversive (negative) and appetitive (positive) environmental cues as well as memories (Lang & Bradley, 2009). To examine mood states with high levels on the emotional arousal dimension (Russell, 1980), we measured autonomic nervous system correlates in response to happiness and anger, and the role of emotional valence was evaluated by task-induced mood states and electrocortical activity responses.

The health benefits related to positive emotions may operate via reduced cardiovascular activation associated with decreased sympathetic nervous system activation and increased parasympathetic activation. These autonomic nervous system correlates of positive emotions may be explained in part by the substitution of the typical narrow “thought-action response” with a broader response that enables an array of potential thoughts and actions (Fredrickson, 1998). Several theories have been proposed to integrate emotional states with central and autonomic nervous system activity (e.g., Porges, 2007; Wager et al., 2009; Lang & Bradley, 2009; Bradley et al., 2001; Lane & Schwartz, 1987; Fredrickson & Losada, 2005). For purposes of this study, we define emotion in terms of its functional aspects (Frijda, 1986; Thayer & Lane, 2007), characterized by an individual’s response to an environmental event that facilitates the rapid mobilization for action of multiple systems (including cognitive, behavioral, and autonomic nervous system responses) (Hagemann, Waldstein, & Thayer, 2003). Expression of emotions is hypothesized to have developed during evolution from motivational brain circuits to optimize survival chances (Porges, 2007; Lang & Bradley, 2009, see Grossman & Taylor, 2007 for comments on this hypothesis). Emotions modulate

sensory systems and mobilize the organism for action (Bradley, 2009). Evidence suggests that positive emotions are not the reverse of (or absence of) negative emotions, but it remains unclear whether circumplex, vector-based, or non-linear models are needed to best characterize positive vs. negative emotional states (Rubin & Talarico, 2009; Terracciano, McCrae, Hagemann, & Costa, Jr., 2003). The adaptive role of positive emotions is less clear when compared to negative emotions (Fredrickson, 1998), but may involve indirect effects related to modulating reactions to negative challenges as well as personal and societal growth (Fredrickson, 1998; Fredrickson & Losada, 2005) in addition to potentially protective central and autonomic nervous system processes.

Evidence suggests that positive emotions (e.g., joy or happiness) elicit differential heart rate (HR), blood pressure, and peripheral vascular resistance responses as compared to negative emotions including anger, fear, and sadness (Schwartz et al., 1981; Ekman et al., 1983; Sinha et al., 1992; Bradley et al., 2001). Positive emotions also result in altered autonomic nervous system activity, characterized by increased parasympathetic nervous system activity, whereas negative emotions (e.g., anger) result in parasympathetic withdrawal and sympathetic activity (McCraty, Atkinson, Tiller, Rein, & Watkins, 1995). The specific nature of the positive emotion and individual response characteristics are important factors in the autonomic nervous system responses to emotional states (Kreibig, 2010). Positive emotions may also facilitate recovery of cardiovascular measures following negative emotional challenges (Fredrickson & Levenson, 1998). Most positive emotions do not display pronounced autonomic nervous system responses relative to negative emotions (Fredrickson, 1998; Pressman & Cohen, 2005), and emotional responses to recall of past experiences are less differentiated for positive versus negative events (Ellsworth & Smith, 1988; Stephens, Christie, & Friedman, 2010). However, other studies have not found differential cardiovascular responses associated with positive versus negative emotions (Warner & Strowman, 1995). Despite these mixed findings regarding the physiological response patterns associated with the valence of emotions, it can be hypothesized that increased parasympathetic activity is consistent with the hypothesized nonspecific action, referred to as “free activation” (Frijda, 1986) tendencies in response to positive emotions (Fredrickson, 1998), whereas sympathetic nervous system activation can occur with the more narrow-focused thought-action response.

To measure changes in autonomic nervous system activity, several methods have been developed of which heart rate variability (HRV) has been extensively studied (Malliani, Pagani, Lombardi, & Cerutti, 1991; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Pieper & Hammill, 1995; Lombardi, Malliani, Pagani, & Cerutti, 1996). In addition to the non-invasive methodology, an additional advantage of HRV analysis is the possibility of repeated measurements within the same participant, which is relevant to examining acute autonomic nervous system responses in reaction to a series of laboratory tasks with different emotional valence. Spectral analyses of the beat-to-beat R-R intervals based on Fourier analyses can be used to obtain HRV measures in the frequency domain, which are preferred for short-term changes in HRV. Decreased HRV has been used as an index of a shift in autonomic nervous system activity towards increased sympathetic activity and reduced parasympathetic activity, i.e., sympathetic dominance over vagal activity (Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), although methodological challenges remain of some concern (Grossman & Taylor, 2007). High frequency (HF) HRV is considered to be a relatively unbiased index of parasympathetic cardiac input (Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Low frequency (LF) HRV has been used as a measure of sympathetic activity, but this index also contains influences of vagal cardiac control

(Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Eckberg, 1997; Grossman & Taylor, 2007). The HF-HRV component was therefore used as the primary index of parasympathetic activity.

Central nervous system processes may partially mediate associations between emotion and autonomic nervous system reactivity (Hagemann et al., 2003; Porges, 2007; Wager et al., 2009). Electroencephalography (EEG) and imaging techniques such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) have been used to investigate central nervous system responses during positive and negative emotions. Experiences of positive emotion and approach behaviors are commonly associated with left frontal brain activity, whereas the experience and expression of negative emotions and withdrawal/avoidance behaviors are associated with activity of the right frontal lobe (Davidson, 1992; Jones & Fox, 1992; Hagemann, Naumann, Becker, Maier, & Bartussek, 1998; Harmon-Jones, Gable, & Peterson, 2010; Carver & Harmon-Jones, 2009). Activity of the frontal brain structures is involved in eliciting sympathetic outflow, cardiovascular reactivity, myocardial conduction instability, and cardiac arrhythmias (Skinner & Reed, 1981; Lane & Schwartz, 1987). Wittling et al. (1998) showed that the right hemisphere primarily controls sympathetic activity and the left hemisphere mainly controls parasympathetic activity (Wittling, Block, Genzel, & Schweiger, 1998; Wittling & Pfluger, 1990). In addition to emotional valence, motivational and arousal aspects of emotion are important asymmetrical frontal cortical activity (Harmon-Jones et al., 2010). Some research has shown that damage to the right hemisphere is more likely to result in autonomic dysregulation as compared with left hemisphere damage (Hachinski, Oppenheimer, Wilson, Guiraudon, & Cechetto, 1992; Andersson & Finset, 1998). These findings lead to the hypothesis that left frontal cortical activation in response to positive emotional states will result in parasympathetic activation, whereas right frontal activation in response to negative emotional states will lead to increased sympathetic activation.

Some evidence indeed suggests that EEG measures of brain activity are associated with HRV-based indices of autonomic nervous system activity during resting conditions (Foster & Harrison, 2004). We previously showed that asymmetric frontal EEG responses to emotional arousal may elicit different patterns of heart rate and blood pressure reactivity in healthy adults, and that anger-induced right frontal lateralization was associated with higher blood pressure responses, whereas HR responses were related to both left and right frontal activity (Waldstein et al., 2000). Using PET, Marci et al. (2007) found correspondence between anger-induced sympathetic arousal (measured by skin conductance and heart rate) and parasympathetic withdrawal (reduced HF-HRV), with cerebral blood flow responses (left orbitofrontal, left insula, left cerebellum, left mid-temporal gyrus, and right superior temporal gyrus), whereas no such correspondence was found for happiness and sadness (Marci et al., 2007). Thus, differential (right-sided) cerebral lateralization is involved in the relative sympathetic predominance in response to negative vs. positive emotion-induced autonomic nervous system reactivity. Using positive (happiness) and negative (anger) emotional recall tasks and the SCWT, we will examine the role of mood induction and EEG responses in HRV-based autonomic nervous system reactivity.

To investigate the effects of selected emotions on autonomic nervous system activity, this study used laboratory-based recall tasks designed to induce positive emotion (happiness) and negative emotion (anger). The Stroop Color Word Test (SCWT) was used as a third challenge task to examine associations between emotions with autonomic nervous system and EEG responses that were not dependent on personal recall. We tested the hypothesis that positive emotions induced by happiness recall will lead to an increase in HRV-based indices of parasympathetic activity, whereas negative emotions induced by anger recall and SCWT

are expected to result in HRV-based indices of parasympathetic withdrawal. We also investigated whether acute perceived emotions are related to these autonomic indices, and to what extent emotion-induced left frontal cortical responses are associated with HRV-based indices of parasympathetic nervous system activity.

MATERIAL AND METHODS

Participants

Right-handed young adults were included in this investigation (mean age 25 ± 4 years, 55% female; 20% African American). Exclusion criteria were a self-reported history of hypertension, diabetes mellitus, cardiovascular disease, neurological or psychiatric disorder; history of head injury with loss of consciousness (>10 min); use of medications affecting cardiovascular or cerebrovascular function (including oral contraceptives); and obesity ($>25\%$ overweight by Metropolitan Life Insurance Tables) (1983). Results examining blood pressure and heart rate reactivity based on this sample have been described previously (Waldstein et al., 2000). Participants with valid data for heart rate variability analyses were included in the present analyses ($N=20/30$). Reasons for HRV-based exclusions were as follows: Data were not collected on Holter monitoring tapes for participants enrolled in the first phase of the study ($N=7$), ECG abnormalities interfering with HRV analyses ($> 5\%$ ectopic beats) ($N=1$), ambiguous markings of Holter tapes to precisely link ECG with EEG registrations ($N=1$), and equipment failure ($N=1$). Individuals with valid HRV data tended to be older (25 ± 4 yrs. vs. 22 ± 3 yrs, $t(18)=2.20$, $p=0.04$) than those without valid HRV data, but no differences were found in sex, race, education, or resting heart rate. All participants provided written informed consent in accordance with the Institutional Review Board guidelines, and were paid \$20.00 for their participation.

Procedure

Participants were seated in a comfortable chair, completed a brief demographic questionnaire, and were instrumented for ECG and EEG monitoring. A 10-minute rest (baseline) period was followed by completion of tasks that were designed to elicit positive and negative emotions as described previously (Waldstein et al., 2000). Emotion-specific recall tasks involved positive event recall, negative event recall, and the Stroop Color Word Test (SCWT). As part of the overall study protocol, video clips were presented as well; these clips were not used for the present analyses because they were of insufficient duration (< 90 sec) to validly analyze short-term HRV changes. Each task was followed by a 10 min recovery period. The order of the positive (happiness-inducing tasks) and negative (anger/frustration-inducing tasks) emotional tasks was counterbalanced in blocks, and the protocol ended with the SCWT for all conditions to minimize carry over of the SCWT.

ECGs were collected continuously throughout the protocol using a Holter monitor. Self-reported ratings of affect and task engagement were obtained at the end of each baseline and task period.

EEG data were obtained during min 7 – 9 of the initial baseline period (to allow for habituation to the setting); continuously during the visualization (imagery) portion of the happiness and anger recall tasks; and during min 8–9 of the post-recall recovery periods. EEG data were not collected during the verbalization portion of the recall tasks because of the well known effects of facial movement, including speaking, on frontal activity (Pivik et al., 1993).

Laboratory Tasks

Provocation tasks were chosen to elicit positive affect (happiness) or negative affect (anger). Personally relevant recall tasks were used because such tasks induce stronger physiological arousal than tasks that are not personally relevant (Velasco & Bond, 1998). The positive and negative recall tasks involved a 3 minute speech and a subsequent 2 minute imaginary phase during which EEG measures were obtained. We also administered the SCWT to include a negative emotion provocation task that is not personally relevant.

Happiness Recall Task—Participants were asked to discuss an incident (preferably occurring within the past 6 months) that made them feel happy, glad, or cheerful. The participant was asked to recreate the incident from beginning to end relaying what was said and done and describing associated thoughts and feelings. The task was modeled after the previously validated Anger Recall Task (see below). During the imagery portion of the task, the participant was asked to continue to think about this situation, focusing on visualizing different aspects of the situation (e.g. location, persons involved) and concentrating on the associated feelings. They were also cued with signs periodically during the task to assist in focusing their attention (e.g. ‘how did you feel?’).

Anger Recall Task—Participants were asked to discuss an incident (preferably occurring within the past 6 months) that made them feel angry, frustrated, or irritated as described previously (Ironson et al., 1992; Kop et al., 2008). The participant was asked to recreate the incident from beginning to end parallel to the instructions for the Happiness Recall Task (3-minute speech followed by a 2-minute imaginary phase).

Stroop Color Word Test (SCWT)—The SCWT was used to examine the effects of a challenging perturbation task that is known to elicit autonomic and central nervous system responses, but that does not require recall of a personally relevant event (Becker et al., 1996). Participants were asked to press a mouse button (with their dominant, i.e. right hand) corresponding to the display color (blue, red, or green) of the presented word as fast as they could when color words (“red”, “blue”, or “green”) appeared on the computer screen as described previously (Becker et al., 1996). For example, if the target word “blue” was presented in red display color, then the three possible answers were the words “blue” displayed in red letters, the word “green” displayed in blue letters, and the word “red” displayed in green letters; the correct response would then be “red” written in green letters. The pace at which words were displayed was automatically adjusted to a level of difficulty designed to result in 60% correct responses. Participants were told to get as many correct answers as possible in the allotted time and were continuously encouraged by a research assistant to improve performance.

Electrocardiographic Measures

Electrocardiographic (ECG) recordings were obtained using a portable Marquette Series 8500 7-lead ECG monitor (GE-Marquette Medical Systems Information Technologies, Milwaukee, WI) connected to disposable 3M electrodes (3M, London, Ontario) and recorded on cassette tapes. Data were uploaded onto a Dell computer with Microsoft Windows for reduction and analysis using the MARS PC 6.01 software (GE-Marquette). Artifacts and ectopic beats (ventricular and supraventricular beats) were detected using the MARS software as well as visually, and subsequently removed prior to analyses.

Heart Rate Variability (HRV) Analyses

Heart rate variability was used to assess autonomic nervous system activity. Heart rate was determined by the number of beats per minute. HRV was computed from the continuous

ECG recordings by analyzing the variability in the intervals between sinus rhythm heart beats (R-R intervals) (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Frequency domain analyses were used, separating R-R intervals into frequency bands and then determining the power of each frequency band during each phase of the study protocol (Stein, Bosner, Kleiger, & Conger, 1994). Fast Fourier Transform analyses was used to transform R-R intervals into high (HF-HRV; 0.15–0.40 Hz) and low (LF-HRV; 0.04–0.15 Hz) frequency bands, and HRV values are expressed in $\ln(\text{ms}^2)$. HF-HRV was the predominant HRV index under investigation because HF-HRV is a specific marker of parasympathetic activity, whereas LF-HRV may represent both parasympathetic and sympathetic nervous system activity (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Pomeranz et al., 1985). Additionally, we used the LF/HF ratio a measure of sympathovagal balance. A natural logarithmic transform of the power of each frequency band was used to normalize the data and the resultant power is described in $\ln(\text{ms}^2)$.

The window of analysis was set at 1-minute segments. HRV indices were calculated for each minute during the 5-min tasks. The two minute periods before and after each target minute segment were used for HR trending. The average HRV data over the 5-min task periods were used for analyses and compared to the last 5-min segment of the preceding rest periods. The Δ HRV response was calculated based by subtracting task from resting levels.

Acute Emotional Responses

Participants rated items reflecting their level of affective arousal (frustrated, angry, depressed, and happy) and task engagement (interested, in control) on a Likert-type scale of 1 (not at all) to 10 (very much). Change scores (task value minus immediately preceding baseline value) were computed to index task-induced mood responses.

Electroencephalographic (EEG) Recordings

Brain electrical activity was recorded by EEG using a Lycra stretch cap (Electro-Cap, Inc.). The cap electrodes were positioned according to the International 10/20 Electrode Placement System (Jasper, 1958). After abrading each electrode surface, the electrode sites were then filled with a small amount of electrolyte gel, which served as a conductor. Electrode impedances below 10 kV per site and within 500 V between homologous sites were considered acceptable. The EEG was recorded at eight scalp locations, representing the left and right hemispheres and anterior and posterior regions of the brain: left and right mid-frontal (F3, F4), parietal (P3, P4), central (C3, C4) and occipital (O1, O2) regions. All electrodes were referenced to the central vertex (Cz). Electrooculographic (EOG) activity was recorded using two Beckman miniature electrodes, which were placed on the external canthus and the supra-orbital area of the right eye. The EOG signal was used to facilitate subsequent EEG artifact editing. A separate ground electrode was attached to the base of the subject's neck on the dorsal side.

The nine channels were amplified by individual Grass AC Bioamplifiers (Model 7p511). The filter settings for the nine channels were set at 1 Hz (high pass) and 100 Hz (low pass). A calibration signal (10 Hz/0.47 V rms sine wave) was sent through each amplifier prior to data collection for each participant (output 50 μ V, gain of 10,000). The data from all nine channels were digitized on-line at a sampling rate of 512 Hz on a Gateway 486/33C PC in order to prevent aliasing of the EEG data (Pivik et al., 1993). EEG data were stored on optical laser disk prior to analysis. The EEG data were re-referenced to an average reference configuration and visually scored for artifact due to eye blinks and movement. All artifact-free EEG data were analyzed using a discrete Fourier transform, with a Hanning window of

1-s width and 50% overlap. Power (μV^2) was computed in the 8–13 Hz (alpha) frequency band for each electrode site and natural logarithmically transformed. Lower levels in the alpha frequency band indicate activation of the target brain region. A lateralization index was computed in order to examine frontal asymmetry during each of the tasks, calculated as: $(\ln \text{ power [right hemisphere]}) - (\ln \text{ power [left hemisphere]})$. Positive values on this metric represent greater relative left hemisphere (Pivik et al., 1993).

Statistical Analyses

Data are presented as mean \pm standard deviation (s.d.) or percentages when appropriate. HRV data are presented as mean \pm standard error of the mean. Repeated measures analyses of variance were used to examine responses across tasks using two within-subject factors (2 [baseline vs. task] \times 3 [type of task: Happiness Recall, Anger Recall, and SCWT]). Greenhouse-Geisser corrections were used to adjust for non-sphericity of the variance-covariance matrices, which revealed the same results as uncorrected F-values, and uncorrected F values were therefore reported. Stress-induced responses (HRV indices, mood and EEG measures) were further examined using paired t-tests for each of the tasks separately, and partial eta-squared (η^2) values were calculated to quantify the effect sizes of the mood, HRV and EEG responses. To examine whether the tasks resulted in a significant change in the lateralization index, one-sample t-tests were used with 0 as reference value. Non-parametric correlation coefficients (Spearman's rho) were used to examine whether acute mood responses and EEG measures were associated with HRV-indices. We examined HRV levels during the tasks as well as responses Δ from baseline as dependent measures. Data were analyzed with SPSS (version 17) and a p-value of <0.05 was used to indicate statistical significance.

RESULTS

Participant Characteristics

Table 1 displays the participant characteristics. Demographic measures were associated with selected HF indices: age was associated with lower LF-HRV ($\rho = -0.51$, $p = 0.023$). European Americans had higher LF-HRV levels compared to other participants (7.41 ± 0.21 vs. $6.47 \pm 0.29 \ln(\text{ms}^2)$, $t = 2.72$, $p = 0.014$). A higher level of education was associated with lower LF-HRV ($\rho = -0.71$, $p = 0.001$) and lower HF-HRV ($\rho = -0.55$, $p = 0.016$).

The tasks produced the expected emotional responses (Table 2). To examine the differential effects (from pre- to post) of the three tasks (Happiness Recall, Anger Recall, SCWT) on the 6 mood responses (happy, angry, frustrated, depressed, interested, in control), a $2 \times 3 \times 6$ repeated measures analysis of variance was conducted. Main effects were found for the pre-post task response ($F(1,19) = 49.40$, $p < 0.001$, $\eta^2 = 0.72$), type of task ($F(2,38) = 2.94$, $p = 0.065$, $\eta^2 = 0.13$), type of emotion ($F(5,95) = 25.70$, $p < 0.001$, $\eta^2 = 0.58$) and significant interactions: pre-post response \times task type ($F(2,38) = 8.78$, $p = 0.001$, $\eta^2 = 0.32$), pre-post \times emotion type ($F(5,95) = 11.45$, $p < 0.001$, $\eta^2 = 0.37$), task type \times emotion type ($F(10,190) = 19.37$, $p < 0.001$, $\eta^2 = 0.51$), and the pre-post \times task \times emotion interaction ($F(10,190) = 21.18$, $p < 0.001$, $\eta^2 = 0.53$). Specifically, the Happiness Recall task produced significant increases in the following mood measures: “happy,” “interested” and “in control” and a reduction in “frustrated.” The Anger Recall task produced significant increases in all negative emotions and a reduction in happiness and no effect on feeling “in control.” The SCWT resulted in increased negative emotions, reduced happiness, increased interest, and had no effects on feeling depressed or in control, indicating that the SCWT elicited a negative mood response (Table 2).

Positive and Negative Mood Induction and Autonomic Nervous System Activity

To determine the effects (from baseline to task) of the three perturbation tasks on HRV indices, repeated measures analysis of variance were conducted, using a 2×3 within subjects model for each of the 3 HRV indices. For HF-HRV, significant main effects were found for response from baseline ($F(1,18) = 5.82, p = 0.027, \eta^2 = 0.24$), type of task ($F(2,36) = 4.14, p = 0.024, \eta^2 = 0.19$) and a significant interaction between task-responses and task type ($F(2,36) = 12.12, p < 0.001, \eta^2 = 0.24$). Results for LF-HRV were also significant (pre-post main effect $F(1,18) = 7.78, p = 0.012, \eta^2 = 0.30$, task-type main effect $F(2,36) = 7.71, p = 0.002, \eta^2 = 0.30$, and interaction term $F(2,36) = 10.16, p < 0.001, \eta^2 = 0.36$). For the LF/HF ratio, the main effect for task type was significant ($F(2,36) = 3.99, p = 0.027, \eta^2 = 0.18$), whereas no significant effects were observed for pre- vs. post ($F(1,18) = 1.25, p = 0.28, \eta^2 = 0.07$) or the interaction term ($F(2,36) = 0.53, p = 0.58, \eta^2 = 0.03$). No evidence was found for a baseline drift in HRV indices, and resting HRV measures prior to the three provocation tasks were not significantly different.

As shown in Figure 1, positive mood induction by the Happiness Recall task resulted in increased LF-HRV (from 7.03 ± 0.20 to $7.32 \pm 0.21 \ln(\text{ms}^2)$, $F(1,19) = 10.02, p = 0.005, \eta^2 = 0.35$). This response was paralleled by an increase in the LF/HF ratio (from 1.18 ± 0.03 to 1.22 ± 0.02 , $F(1,19) = 3.42, p = 0.080, \eta^2 = 0.15$) and an increase in heart rate (from 71.7 ± 3.2 to 77.3 ± 3.3 bpm, $F(1,19) = 9.55, p = 0.006, \eta^2 = 0.33$). The effect of Happiness Recall on HF-HRV was not significant ($F(1,19) = 0.15, p = 0.71, \eta^2 = 0.01$).

Negative mood induction by Anger Recall had no significant effects on HRV indices (Figure 1) (LF-HRV $F(1,19) = 0.10, p = 0.76, \eta^2 = 0.005$, HF-HRV $F(1,19) = 0.33, p = 0.57, \eta^2 = 0.017$). The LF/HF response was negligible (from 1.20 ± 0.03 to 1.20 ± 0.02 , $F(1,19) = 0.01, p = 0.92, \eta^2 = 0.001$), whereas heart rate increased during Anger Recall (from 71.9 ± 3.5 bpm to 77.6 ± 3.3 bpm ($F(1,19) = 44.82, p < 0.001, \eta^2 = 0.70$)).

The SCWT task produced a decrease from baseline in HF-HRV (from 6.21 ± 0.20 to $5.52 \pm 0.21 \ln(\text{ms}^2)$, $F(1,18) = 4.57, p = 0.001, \eta^2 = 0.44$), consistent with parasympathetic withdrawal. This response was accompanied by decreases in LF-HRV (from 7.12 ± 0.24 to $6.36 \pm 0.23 \ln(\text{ms}^2)$, $F(1,18) = 15.59, p = 0.001, \eta^2 = 0.46$) (Figure 1). The LF/HF response to the SCWT was non-significant (from 1.15 ± 0.03 to 1.17 ± 0.02 , $F(1,18) = 0.17, p = 0.67, \eta^2 = 0.01$). The SCWT-induced Δ HR response (from 68.6 ± 2.8 to 81.7 ± 3.7 bpm, $F(1,18) = 38.91, p < 0.001, \eta^2 = 0.67$) was associated with SCWT-induced Δ LF-HRV ($\rho = 0.53, p = 0.02$).

Acute Emotional Reactions as Related to Task-Induced HRV Responses

Results are presented for *levels* of emotions during each of the tasks and *responses* from the preceding baseline values. As shown in Table 3, emotional state *levels* during the Happiness Recall task revealed a positive correlation between task-induced feeling “happy” and HF-HRV increases from baseline ($\rho = 0.44, p = 0.050$). Participants who reported being frustrated or depressed during the Happiness Recall task had lower LF-HRV ($\rho = -0.60, p = 0.005$ and $\rho = -0.43, p = 0.056$, respectively). When examining emotional *responses* from pre-task baseline levels, it was found that participants who became more “in control” tended to display a higher increase in HF-HRV ($\rho = 0.40, p = 0.083$), whereas individuals who became “frustrated” during the Happiness Recall task displayed a drop in HF-HRV ($\rho = -0.56, p = 0.011$) and consequently an increase in the LF/HF ratio ($\rho = 0.45, p = 0.045$).

The Anger Recall task *levels* of frustration tended to be associated with higher LF-HRV values ($\rho = 0.40, p = 0.080$) and participants who were more interested and in control during Anger Recall had smaller LF/HF HRV changes. Investigation of the mood *responses* to Anger Recall revealed that participants who became more frustrated tended to display a

stronger increase in LF-HRV ($\rho = 0.39$, $p = 0.091$), whereas other no associations were found for other HRV indices.

The SCWT revealed that participants with higher *levels* of being frustrated during the task displayed higher increase in the LF/HF ratio ($\rho = 0.39$, $p = 0.095$), but no associations between mood *responses* and HRV indices were observed.

Brain Activity and HRV Responses to Mood Induction

The tasks induced the anticipated frontal cortical responses. Happiness Recall resulted in left frontal EEG activity (from 2.08 ± 1.03 to $1.82 \pm 0.94 \ln.\mu V^2$, $F(1,18) = 6.18$, $p = 0.023$, $\eta^2 = 0.26$) as well as a tendency for right frontal activity (from 1.96 ± 1.06 to $1.79 \pm 1.03 \ln.\mu V^2$, $F(1,18) = 3.12$, $p = 0.094$, $\eta^2 = 0.15$). The Anger Recall task produced a trend towards the anticipated right frontal activity (from 1.92 ± 0.78 to $1.74 \pm 0.78 \ln.\mu V^2$, $F(1,19) = 2.93$, $p = 0.098$) and no significant left frontal response (from 1.90 ± 0.73 to $1.74 \pm 0.78 \ln.\mu V^2$, $F(1,19) = 2.33$, $p = 0.14$, $\eta^2 = 0.11$). Happiness Recall and Anger Recall did not result in significant changes in the lateralization index (-0.026 ± 0.228 ($p = 0.61$, $\eta^2 = 0.02$), and -0.001 ($p = 0.99$, $\eta^2 = 0.01$), respectively). The SCWT did not result in significant right frontal ($F(1,19) = 0.32$, $p = 0.58$, $\eta^2 = 0.02$) or left frontal ($F(1,19) = 0.01$, $p = 0.93$, $\eta^2 < 0.01$) activation, or a change in the lateralization index ($p = 0.62$, $\eta^2 = 0.01$). When examining the EEG measures jointly in a multivariate repeated measures model (using a 2 [baseline vs. task] \times 3 [Happiness Recall, Anger Recall, SCWT] \times 2 [left vs. right activation]), no significant main or interaction effects were found (p -values > 0.10).

The associations between continuous frontal activation measures and HRV responses were in most cases non-significant and effect sizes were relatively small (median $I \rho I = 0.14$, range -0.40 to 0.58). The only trends for significance were that individuals with high *levels* of frontal activity during the SCWT (i.e., as indicated by lower $\ln.\mu V^2$ values) tended to display higher LF-HRV increases (left frontal $\rho = -0.40$, $p = 0.088$, right frontal $\rho = 0.39$, $p = 0.099$). Evaluation of the *responses* in frontal activity indicated that increased right frontal activation during Happiness Recall was associated with a decrease in LF/HF ratio ($\rho = 0.58$, $p = 0.009$), and the correlation between left frontal activity during Happiness Recall was in the expected direction but not statistically significant ($\rho = 0.36$, $p = 0.12$). The task-induced lateralization indices were not related to HRV responses (Table 4).

DISCUSSION

This study shows that positive (happiness recall) and negative (anger recall and SCWT) mood induction tasks result in differential autonomic nervous system responses as measured by HRV. No direct evidence was found for a generalized increase in parasympathetic nervous system activity (i.e., increased HF-HRV) by the positive mood induction task or parasympathetic withdrawal in response to negative mood induction tasks. However, participants who experienced the anticipated feelings of happiness in response to the positive mood induction task displayed an increase in HF-HRV suggesting increased parasympathetic activity. These data may indicate that physiological responses to positive and negative emotional perturbation tasks can be better quantified if individual responses to tasks are assessed in addition to the hypothesized emotional valence of the task. Additional research with larger and more diverse samples is needed to systematically address this issue and to replicate the observed associations.

Individual differences in autonomic responses may have important implications for the investigation of laboratory-induced physiological responses to mood induction tasks. The elicitation of a “congruent” positive emotional state (i.e., feeling “happy”) during the Happiness Recall task was associated with an increase in HF-HRV from baseline ($\rho =$

0.44, $p = 0.050$). In contrast, “incongruent” mood states of feeling “frustrated” during this task were related to reduced HF-HRV ($\rho = 0.41$, $p = 0.070$; Table 3). Experiences of frustration (scores ≥ 2) during the Happiness Recall task occurred in 25% of participants; 2 (10%) displayed an increase in frustration, 11 (55%) were stable, and 7 became less frustrated). Evidence from the Anger Recall and SCWT pointed to a similar direction, such that individuals with a congruent negative mood response (i.e., frustration) had increases in Δ LF-HRV, whereas participants with increased levels of being “interested” and “in control” during Anger Recall displayed a relative reduction in the Δ LF/HF HRV ratio. It is therefore possible that a combination of task valence and congruent emotional responses are required to elicit parasympathetic activity.

Evidence from the EEG responses revealed a similar pattern, such that participants with paradoxical right frontal lobe activity during Happiness Recall (i.e., negative change scores) displayed a shift towards parasympathetic activation and sympathetic autonomic nervous system withdrawal as indexed by a smaller task-induced increase in the LF/HF ratio ($\rho = 0.58$; Table 4). Further research is needed to examine to what extent these incongruent mood and electrocortical responses reflect stable psychological traits such as hostility and other contextual psychological variables (e.g. trait anger, depression, and anxiety). A recent meta-analysis indicates that autonomic nervous system responses to acute challenge tasks are not consistently related to these chronic contextual psychological measures (Chida & Hamer, 2008). Studies characterizing acute emotional responses combined with brain imaging are needed to address this issue by experimentally manipulating emotional congruencies with task characteristics.

Positive mood induction by the Happiness Recall task resulted in an unanticipated increase in LF-HRV. One interpretation would be that this LF response reflects an increase in sympathetic activity. Such an interpretation would be consistent with the clinical observation that acute states of positive emotions can trigger adverse cardiac events (Samuels, 2007; Phillips et al., 2004; Engel, 1971). Alternatively, some evidence suggests that the LF HRV component partially reflects vagal activation (Berntson et al., 1997; Eckberg, 1997; Grossman & Taylor, 2007), which would be consistent with the intended parasympathetic response of the Happiness Recall task as well as the parallel changes in the HF and LF HRV measures during Anger Recall and the SCWT consistent with vagal withdrawal. No significant changes were noted in the LF/HF ratio during any of the tasks, further indicating that differentiating LF and HF components may not be optimal to evaluate HRV-based indices of parasympathetic versus sympathetic autonomic nervous system reactivity to mood induction tasks in healthy young adults.

One of the strengths of this study is the potential to differentiate between acute perceived emotions and cortical arousal as predictors of autonomic activity (Lane, 2008). Only limited support was found for the hypothesis that EEG-based measures of left frontal cortical activity are associated with HRV indices of a reduced sympathovagal balance during the SCWT (ρ left frontal activity with Δ LF-HRV = -0.40 , $p = 0.088$; Table 4). Prior research suggests that distinct patterns of cardiac activation may result from asymmetric stimulation of the stellate ganglion as a consequence of differences in the topographical distribution of the right and left stellate cardiac nerves (Lane & Schwartz, 1987). However, we found that paradoxical right frontal activity was related to an increase in the Δ LF/HF ratio during the SCWT ($\rho = 0.39$, $p = 0.099$) and no associations were found between frontal lateralization and HRV indices (Table 4). Marci et al. (2007) reported increased brain activity (i.e., increased cerebral blood flow based on PET) in response to anger, but not happiness or sadness provocation, whereas all tasks induced either sympathetic arousal (measured by skin conductance and heart rate), parasympathetic withdrawal (reduced HF-HRV), or both (Marci et al., 2007). However, correlations between emotional-induced brain activity and

HRV measures were not presented by Marci and colleagues. Optimal investigation of emotion-related associations between central and autonomic nervous system activity may require investigation of additional brain areas other than the frontal lobe, particularly the limbic system and related structures. Using PET and multiple positive and negative mood-inducing tasks, the medial visceromotor network has been postulated as the final common pathway by which emotional and cognitive functions result in autonomic nervous system activation (Lane et al., 2009). Negative emotions (e.g., social evaluation) are associated with elevated dorsal pregenual cingulate activity as well as decreased right ventromedial/medial orbital activity, both of which are coupled with HR increases (Wager et al., 2009). In addition, several models have been postulated about cerebral balance, such as the interdependence of anterior and posterior brain structures, or the interhemispheric balance model postulated by Tucker and colleagues (Tucker & Williamson, 1984; Tucker, 1984). However, the cerebral activation in response to emotional provocation tasks is quite variable and not as consistent as previously hypothesized (Wager, Phan, Liberzon, & Taylor, 2003; Murphy, Nimmo-Smith, & Lawrence, 2003). It is possible that EEG measures lack sufficient sensitivity to document association between various regions of cortical activity and autonomic nervous system responses and that PET or fMRI-based imaging techniques are needed to further examine these pathways at cortical and sub-cortical levels.

The present study utilized the computerized SCWT to induce a negative mood state that did not rely on a personally relevant event or involve anger as the primary target emotion. The SCWT was associated with the most pronounced HRV responses, but no changes in the LF/HF ratio were found. Both left and right frontal activity during the SCWT tended to be related to smaller Δ LF-HRV responses (Table 4), suggesting that SCWT-induced frontal activation *per se* may result in a shift towards parasympathetic activation. Standardized computerized tasks that elicit sustained and wider range of positive and negative emotions would be of interest to further investigate relationships between brain and autonomic nervous system responses.

The findings of this study need to be interpreted in the light of several limitations. The sample size was relatively small resulting in low statistical power. This limitation is partially outweighed by the repeated measures design and multimodal emotional and physiological assessments, as well as the relatively large effect size of some of the HRV responses ($\eta^2 > 0.30$). The small sample size precluded multivariate adjustment for potentially confounding factors such as age, sex and race. Parasympathetic withdrawal is the primary mechanism involved in the heart rate response range with moderate mental arousal as observed in the present study. However, the correlation between HR and HRV-indices was not consistent, which may in part be explained by error variance in the HRV measures. Future research is therefore needed to better quantify sympathetic activation such as impedance cardiography-derived shortening of the pre-ejection period. We also did not evaluate the full spectrum of positive and negative mood induction tasks (e.g., relaxation, fear or sadness) and the present results are therefore not generalizable to other positive and negative emotions than those assessed in this study. Some evidence suggests that the duration between life events and testing may affect EEG parameters, and although all events occurred between within 6 months of testing, no systematic control for the time delay between event and laboratory testing was obtained. This study also demonstrated significant heart rate responses to all tasks, whereas EEG-based cerebral responses were less consistent. More sensitive imaging techniques may be needed to establish associations between short-term cortical and subcortical responses with autonomic nervous system activity.

CONCLUSIONS

The present findings suggest that increases in parasympathetic nervous system activity may develop in individuals who respond to conditions that typically elicit a positive mood (e.g., a happy memory or a positive life event) that are accompanied by a congruent response of feeling happy. These emotionally congruent states may have cardio-protective effects. In contrast, if such “happy occasions” (or recollections thereof) are associated with incongruent negative emotions, then transient increases in the sympathovagal balance, consistent with parasympathetic withdrawal may occur. This finding may explain why positive life events can act as triggers of acute coronary syndromes (Samuels, 2007; Phillips et al., 2004; Engel, 1971). In addition, repeated cardiovascular responses in reaction to emotional arousal may provide a link between dispositional psychological traits and cardiovascular disease (Rozanski et al., 1999; Kop, 1999). The present findings suggest that individual differences exist in the response to standardized recall tasks. These differential responses may in part result from stable traits as well as transient environmental challenges associated with elevated distress. Future studies in larger samples and in individuals at high risk of adverse cardiovascular outcomes may shed further light on the inter-relations between positive and negative emotions, central nervous system processes and autonomic nervous system reactivity.

Acknowledgments

This project was supported, in part, by NIH Grants HD17899 (to N.A. Fox). Preparation of this manuscript was supported, in part, by NIH Grants HL079376 (to W.J. Kop). The opinions and assertions expressed herein are those of the authors and are not to be construed as reflecting the views of the Uniformed Services University of the Health Sciences or the United States Department of Defense.

Reference List

- 1983 metropolitan height and weight tables. *Stat.Bull.Metrop.Life Found.* 1983; 64:3–9. [PubMed: 6623350]
- Andersson S, Finset A. Heart rate and skin conductance reactivity to brief psychological stress in brain-injured patients. *Journal of Psychosomatic Research.* 1998; 44:645–656. [PubMed: 9678746]
- Becker LC, Pepine CJ, Bonsall R, Cohen JD, Goldberg AD, Coghlan C, et al. Left ventricular, peripheral vascular, and neurohormonal responses to mental stress in normal middle-aged men and women: Reference group for the psychophysiological investigations of myocardial ischemia (PIMI) study. *Circulation.* 1996; 94:2768–2777. [PubMed: 8941101]
- Berntson GG, Bigger JT, Eckberg DL, Grossman P, Kaufman PG, Malik M, et al. Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology.* 1997; 34:623–648. [PubMed: 9401419]
- Bradley MM. Natural selective attention: orienting and emotion. *Psychophysiology.* 2009; 46:1–11. [PubMed: 18778317]
- Bradley MM, Codispoti M, Cuthbert BN, Lang PJ. Emotion and motivation I: defensive and appetitive reactions in picture processing. *Emotion.* 2001; 1:276–298. [PubMed: 12934687]
- Carver CS, Harmon-Jones E. Anger is an approach-related affect: evidence and implications. *Psychological Bulletin.* 2009; 135:183–204. [PubMed: 19254075]
- Chida Y, Hamer M. Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: a quantitative review of 30 years of investigations. *Psychological Bulletin.* 2008; 134:829–885. [PubMed: 18954159]
- Davidson RJ. Anterior cerebral asymmetry and the nature of emotion. *Brain Cogn.* 1992; 20:125–151. [PubMed: 1389117]
- Diener E, Larsen RJ, Levine S, Emmons RA. Intensity and frequency: dimensions underlying positive and negative affect. *Journal of Personality and Social Psychology.* 1985; 48:1253–1265. [PubMed: 3998989]

- Eckberg DL. Sympathovagal balance: a critical appraisal. *Circulation*. 1997 Nov 4; 96(9):3224–3232. [PubMed: 9386196]
- Ekman P, Levenson RW, Friesen WV. Autonomic nervous system activity distinguishes among emotions. *Science*. 1983; 221:1208–1210. [PubMed: 6612338]
- Ellsworth PC, Smith CA. Shades of joy: Patterns of appraisal differentiating pleasant emotions. *Cognition and Emotion*. 1988; 2:301–331.
- Engel GL. Sudden and rapid death during psychological stress. Folklore or folk wisdom? *Annals of Internal Medicine*. 1971; 74:771–782. [PubMed: 5559442]
- Foster PS, Harrison DW. The covariation of cortical electrical activity and cardiovascular responding. *Int.J.Psychophysiol*. 2004; 52:239–255. [PubMed: 15094247]
- Fredrickson BL. What good are positive emotions? *Review of General Psychology*. 1998; 2:300–319.
- Fredrickson BL, Levenson RW. Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cognition and Emotion*. 1998; 12:191–220.
- Fredrickson BL, Losada MF. Positive affect and the complex dynamics of human flourishing. *Am.Psychol*. 2005; 60:678–686. [PubMed: 16221001]
- Frijda, NH. *The emotions*. Cambridge, UK: Cambridge University Press; 1986.
- Grossman P, Taylor EW. Toward understanding respiratory sinus arrhythmia: relations to cardiac vagal tone, evolution and biobehavioral functions. *Biological Psychology*. 2007; 74:263–285. [PubMed: 17081672]
- Hachinski VC, Oppenheimer SM, Wilson JX, Guiraudon C, Cechetto DF. Asymmetry of sympathetic consequences of experimental stroke. *Arch.Neurol*. 1992; 49:697–702. [PubMed: 1497495]
- Hagemann D, Naumann E, Becker G, Maier S, Bartussek D. Frontal brain asymmetry and affective style: a conceptual replication. *Psychophysiology*. 1998; 35:372–388. [PubMed: 9643052]
- Hagemann D, Waldstein SR, Thayer JF. Central and autonomic nervous system integration in emotion. *Brain Cogn*. 2003; 52:79–87. [PubMed: 12812807]
- Harmon-Jones E, Gable PA, Peterson CK. The role of asymmetric frontal cortical activity in emotion-related phenomena: A review and update. *Biological Psychology*. 2010; 84:451–462.
- Ironson G, Taylor CB, Boltwood M, Bartzokis T, Dennis C, Chesney M, et al. Effects of anger on left ventricular ejection fraction in coronary artery disease. *Am.J.Cardiol*. 1992; 70:281–285. [PubMed: 1632389]
- Jasper HH. The ten-twenty electrode system of the International Federation. *Electroencephalography and Clinical Neurophysiology*. 1958; 10:371–375.
- Jones NA, Fox NA. Electroencephalogram asymmetry during emotionally evocative films and its relation to positive and negative affectivity. *Brain Cogn*. 1992; 20:280–299. [PubMed: 1449758]
- Kop WJ. Chronic and acute psychological risk factors for clinical manifestations of coronary artery disease. *Psychosom Med*. 1999; 61:476–487. [PubMed: 10443756]
- Kop WJ, Verdino RJ, Gottdiener JS, O'Leary ST, Bairey Merz CN, Krantz DS. Changes in heart rate and heart rate variability before ambulatory ischemic events. *J Am Coll Cardiol*. 2001; 38:742–749. [PubMed: 11527627]
- Kop WJ, Weissman NJ, Zhu J, Bonsall RW, Doyle M, Stretch MR, et al. Effects of acute mental stress and exercise on inflammatory markers in patients with coronary artery disease and healthy controls. *Am.J.Cardiol*. 2008; 101:767–773. [PubMed: 18328837]
- Krantz DS, Kop WJ, Santiago HT, Gottdiener JS. Mental stress as a trigger of myocardial ischemia and infarction. *Cardiology Clinics*. 1996; 14:271–287. [PubMed: 8724559]
- Kreibig SD. Autonomic nervous system activity in emotion: A review. *Biological Psychology*. 2010; 84:394–442.
- Lane RD. Neural substrates of implicit and explicit emotional processes: a unifying framework for psychosomatic medicine. *Psychosom.Med*. 2008; 70:214–231. [PubMed: 18256335]
- Lane RD, Laukes C, Marcus FI, Chesney MA, Sechrest L, Gear K, et al. Psychological stress preceding idiopathic ventricular fibrillation. *Psychosom.Med*. 2005; 67:359–365. [PubMed: 15911897]
- Lane RD, McRae K, Reiman EM, Chen K, Ahern GL, Thayer JF. Neural correlates of heart rate variability during emotion. *Neuroimage*. 2009; 44:213–222. [PubMed: 18778779]

- Lane RD, Schwartz GE. Induction of lateralized sympathetic input to the heart by the CNS during emotional arousal: a possible neurophysiologic trigger of sudden cardiac death. *Psychosom.Med.* 1987; 49:274–284. [PubMed: 3602297]
- Lang PJ, Bradley MM. Emotion and the motivational brain. *Biological Psychology.* 2009
- Lang PJ, Greenwald MK, Bradley MM, Hamm AO. Looking at pictures: affective, facial, visceral, and behavioral reactions. *Psychophysiology.* 1993; 30:261–273. [PubMed: 8497555]
- Lombardi F, Malliani A, Pagani M, Cerutti S. Heart rate variability and its sympatho-vagal modulation. *Cardiovascular Research.* 1996; 32:208–216. [PubMed: 8796106]
- Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. *Circulation.* 1991; 84:482–492. [PubMed: 1860193]
- Marci CD, Glick DM, Loh R, Dougherty DD. Autonomic and prefrontal cortex responses to autobiographical recall of emotions. *Cogn Affect.Behav.Neurosci.* 2007; 7:243–250. [PubMed: 17993210]
- McCraty R, Atkinson M, Tiller WA, Rein G, Watkins AD. The effects of emotions on short-term power spectrum analysis of heart rate variability. *Am.J.Cardiol.* 1995; 76:1089–1093. [PubMed: 7484873]
- Mittleman MA, Maclure M, Sherwood JB, Mulry RP, Tofler GH, Jacobs SC, et al. Triggering of acute myocardial infarction onset by episodes of anger. Determinants of Myocardial Infarction Onset Study Investigators. *Circulation.* 1995; 92:1720–1725. [PubMed: 7671353]
- Murphy FC, Nimmo-Smith I, Lawrence AD. Functional neuroanatomy of emotions: a meta-analysis. *Cogn Affect.Behav.Neurosci.* 2003; 3:207–233. [PubMed: 14672157]
- Phillips DP, Jarvinen JR, Abramson IS, Phillips RR. Cardiac mortality is higher around Christmas and New Year's than at any other time: the holidays as a risk factor for death. *Circulation.* 2004; 110:3781–3788. [PubMed: 15596560]
- Pieper SJ, Hammill SC. Heart rate variability: technique and investigational applications in cardiovascular medicine. *Mayo Clinic Proceedings.* 1995; 70:955–964. [PubMed: 7564548]
- Pivik RT, Broughton RJ, Coppola R, Davidson RJ, Fox N, Nuwer MR. Guidelines for the recording and quantitative analysis of electroencephalographic activity in research contexts. *Psychophysiology.* 1993; 30:547–558. [PubMed: 8248447]
- Pomeranz B, Macaulay RJ, Caudill MA, Kutz I, Adam D, Gordon D, et al. Assessment of autonomic function in humans by heart rate spectral analysis. *Am.J.Physiol.* 1985; 248:H151–H153. [PubMed: 3970172]
- Porges SW. The polyvagal perspective. *Biological Psychology.* 2007; 74:116–143. [PubMed: 17049418]
- Posner J, Russell JA, Gerber A, Gorman D, Colibazzi T, Yu S, et al. The neurophysiological bases of emotion: An fMRI study of the affective circumplex using emotion-denoting words. *Hum.Brain Mapp.* 2009; 30:883–895. [PubMed: 18344175]
- Pressman SD, Cohen S. Does positive affect influence health? *Psychological Bulletin.* 2005; 131:925–971. [PubMed: 16351329]
- Rozanski A, Blumenthal JA, Kaplan J. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation.* 1999; 99:2192–2217. [PubMed: 10217662]
- Rubin DC, Talarico JM. A comparison of dimensional models of emotion: evidence from emotions, prototypical events, autobiographical memories, and words. *Memory.* 2009; 17:802–808. [PubMed: 19691001]
- Russell JA. A circumplex model of affect. *Journal of Personality and Social Psychology.* 1980; 39:1161–1178.
- Samuels MA. The brain-heart connection. *Circulation.* 2007; 116:77–84. [PubMed: 17606855]
- Schwartz GE, Weinberger DA, Singer JA. Cardiovascular differentiation of happiness, sadness, anger, and fear following imagery and exercise. *Psychosom.Med.* 1981; 43:343–364. [PubMed: 7280162]
- Sinha R, Lovallo WR, Parsons OA. Cardiovascular differentiation of emotions. *Psychosom.Med.* 1992; 54:422–435. [PubMed: 1502284]

- Skinner JE, Reed JC. Blockade of frontocortical-brain stem pathway prevents ventricular fibrillation of ischemic heart. *Am.J.Physiol.* 1981; 240:H156–H163. [PubMed: 7193421]
- Stein PK, Bosner MS, Kleiger RE, Conger BM. Heart rate variability: a measure of cardiac autonomic tone. *Am.Heart J.* 1994; 127:1376–1381. [PubMed: 8172068]
- Stephens CL, Christie IC, Friedman BH. Autonomic specificity of basic emotions: Evidence from pattern classification and cluster analysis. *Biological Psychology.* 2010; 84:463–473.
- Suls J, Bunde J. Anger, anxiety, and depression as risk factors for cardiovascular disease: the problems and implications of overlapping affective dispositions. *Psychological Bulletin.* 2005; 131:260–300. [PubMed: 15740422]
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation, and clinical use. *Circulation.* 1996; 93:1043–1065. [PubMed: 8598068]
- Terracciano A, McCrae RR, Hagemann D, Costa PT Jr. Individual difference variables, affective differentiation, and the structures of affect. *Journal of Personality.* 2003; 71:669–703. [PubMed: 12932207]
- Thayer JF, Lane RD. The role of vagal function in the risk for cardiovascular disease and mortality. *Biological Psychology.* 2007; 74:224–242. [PubMed: 17182165]
- Tucker DM. Lateral brain function in normal and disordered emotion: interpreting electroencephalographic evidence. *Biological Psychology.* 1984; 19:219–235. [PubMed: 6525383]
- Tucker DM, Williamson PA. Asymmetric neural control systems in human self-regulation. *Psychological Review.* 1984; 91:185–215. [PubMed: 6152836]
- Velasco C, Bond A. Personal relevance is an important dimension for visceral reactivity in emotional imagery. *Cognition and Emotion.* 1998; 12:231–242.
- Wager TD, Phan KL, Liberzon I, Taylor SF. Valence, gender, and lateralization of functional brain anatomy in emotion: a meta-analysis of findings from neuroimaging. *Neuroimage.* 2003; 19:513–531. [PubMed: 12880784]
- Wager TD, Waugh CE, Lindquist M, Noll DC, Fredrickson BL, Taylor SF. Brain mediators of cardiovascular responses to social threat: part I: Reciprocal dorsal and ventral sub-regions of the medial prefrontal cortex and heart-rate reactivity. *Neuroimage.* 2009; 47:821–835. [PubMed: 19465137]
- Waldstein SR, Kop WJ, Schmidt LA, Haufler AJ, Krantz DS, Fox NA. Frontal electrocortical and cardiovascular reactivity during happiness and anger. *Biological Psychology.* 2000; 55:3–23. [PubMed: 11099805]
- Warner RM, Strowman SR. Cardiovascular reactivity and positive/negative affect during conversations. *Journal of Behavioral Medicine.* 1995; 18:141–159. [PubMed: 7563043]
- Wittling W, Block A, Genzel S, Schweiger E. Hemisphere asymmetry in parasympathetic control of the heart. *Neuropsychologia.* 1998; 36:461–468. [PubMed: 9699952]
- Wittling W, Pfluger M. Neuroendocrine hemisphere asymmetries: salivary cortisol secretion during lateralized viewing of emotion-related and neutral films. *Brain Cogn.* 1990; 14:243–265. [PubMed: 2285516]

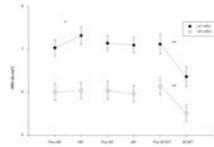


Figure 1.

Effects of positive and negative mood induction on HRV. Responses from baseline to Happiness Recall (HR), Anger Recall (AR) and Stroop Color Word Test (SCWT) were examined for low frequency (LF: closed circles) and high frequency (HF: open circles) heart rate variability (HRV).

* = $p < 0.05$, ** = $p < 0.01$ from preceding baseline.

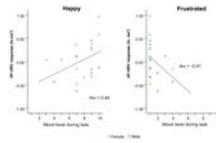


Figure 2. Association between mood responses to positive mood induction by the Happiness Recall task. The horizontal axes present mood experiences during the task (scaled from 1–10) and the vertical axes high frequency (HF) heart rate variability (HRV) in $\ln.ms^2$. Data are presented for female (green circles) and male participants (blue circles) for descriptive purposes.

Table 1

Participant characteristics

	Mean \pm s.d. or N (%)
Age (years)	25 \pm 4
Sex (Female)	11 (55%)
Marital status (Married)	3 (15%)
Race (European American)	12 (60%)
(African American)	4 (20%)
(Asian American)	2 (10%)
(Latino American)	1 (5%)
("Mixed" background)	1 (5%)
Education (years)	16.6 \pm 2.3
Height (m)	67.2 \pm 3.7
Weight (kg)	147.9 \pm 28.2
Systolic BP (mmHg)	115.4 \pm 12.0
Diastolic BP (mmHg)	62.8 \pm 11.8
Heart Rate (bpm)	70.7 \pm 15.2

Table 2
Self-reported affect and engagement in response to Happiness Recall, Anger Recall and Stroop Color Word Task

	Happiness Recall		Anger Recall		Stroop	
	Baseline	Task	Baseline	Task	Baseline	Task
Happy	5.6 ± 2.5	7.8 ± 2.0 **	5.2 ± 2.5	3.1 ± 2.0 **	5.5 ± 2.5	4.4 ± 2.4 *
Angry	1.4 ± 0.7	1.2 ± 0.5	1.2 ± 0.4	6.7 ± 2.8 **	1.3 ± 0.6	3.4 ± 3.0 **
Frustrated	2.0 ± 1.4	1.5 ± 1.0 *	1.7 ± 0.8	5.4 ± 2.7 **	1.6 ± 0.9	5.9 ± 2.7 **
Depressed	1.4 ± 0.8	1.2 ± 0.6	1.3 ± 0.6	3.3 ± 2.6 **	1.3 ± 0.6	1.9 ± 1.9
Interested	4.7 ± 3.0	6.3 ± 2.3 **	4.5 ± 2.7	5.4 ± 2.8 *	4.2 ± 2.7	7.7 ± 1.7 **
In Control	4.5 ± 3.0	5.8 ± 2.7 *	4.6 ± 2.9	4.8 ± 2.8	4.9 ± 3.0	5.1 ± 2.9

* = $p < 0.05$,

** = $p < 0.01$ from baseline

Table 3
 Association between acute mood responses with HRV responses to Happiness Recall, Anger Recall and Stroop Color Word Test

	Happiness Recall				Anger Recall				SCWT			
	LF	HF	LF/HF ratio	LF/HF ratio	LF	HF	LF/HF ratio	LF/HF ratio	LF	HF	LF/HF ratio	LF/HF ratio
Emotion levels during task												
Happy	0.261	0.440*	-0.376	-0.217	0.077	0.077	-0.264	-0.214	0.189	0.189	-0.300	-0.300
Angry	-0.430 ^t	-0.185	-0.071	0.198	0.207	0.207	-0.057	0.003	-0.104	-0.104	0.263 ^t	0.263 ^t
Frustrated	-0.600**	-0.414 ^t	0.003	0.400 ^t	0.170	0.170	0.227	-0.038	-0.267	-0.267	0.394 ^t	0.394 ^t
Depressed	-0.434 ^t	-0.202	-0.058	0.267	0.136	0.136	0.066	-0.049	-0.066	-0.066	0.285	0.285
Interested	0.350	0.277	-0.118	-0.148	0.289	0.289	-0.400 ^t	-0.047	0.039	0.039	-0.145	-0.145
In Control	0.318	0.180	0.085	-0.350	0.149	0.149	-0.479*	-0.022	0.154	0.154	-0.111	-0.111
Emotional responses												
(Δ from baseline)												
Happy	0.122	-0.204	0.143	-0.122	-0.079	-0.079	0.015	-0.233	0.109	0.109	-0.302	-0.302
Angry	-0.405 ^t	-0.260	0.087	0.121	0.170	0.170	-0.124	0.072	-0.110	-0.110	0.240	0.240
Frustrated	-0.134	-0.558*	0.454*	0.388 ^t	0.151	0.151	0.198	-0.015	-0.206	-0.206	0.259	0.259
Depressed	-0.231	0.005	-0.028	0.150	0.137	0.137	-0.064	-0.084	0.005	0.005	0.122	0.122
Interested	0.187	-0.149	0.103	0.072	0.246	0.246	-0.097	-0.034	-0.169	-0.169	0.254	0.254
In Control	0.123	0.396 ^t	-0.264	-0.169	0.105	0.105	-0.220	-0.071	0.178	0.178	-0.091	-0.091

Data present non-parametric (Spearman) correlations

** = $p < 0.01$,

* = $p < 0.05$,

^t = trend: $p < 0.10$.

Table 4

Association between frontal EEG measures with HRV responses to Happiness Recall, Anger Recall and Stroop Color Word Test

	Happiness Recall			Anger Recall			SCWT		
	LF	HF	LF/HF ratio	LF	HF	LF/HF ratio	LF	HF	LF/HF ratio
EEG levels during									
Emotion-inducing tasks ^a									
Left frontal	0.081	0.224	-0.061	-0.014	0.192	-0.149	-0.402 <i>t</i>	-0.302	0.140
Right frontal	0.027	0.248	-0.121	0.041	0.311	-0.153	-0.389 <i>t</i>	-0.228	0.027
Lateralization	-0.194	0.041	-0.062	-0.102	0.340	-0.226	0.093	0.237	-0.147
EEG Response									
(Δ from baseline) ^b									
Left frontal	0.216	-0.037	0.364	-0.114	0.217	-0.214	-0.009	-0.151	0.037
Right frontal	0.232	-0.256	0.582**	-0.105	0.072	-0.126	0.068	-0.223	0.138
Lateralization	-0.074	-0.189	0.103	0.080	-0.096	0.096	0.193	-0.107	.0173

Data present non-parametric (Spearman) correlations

** = $p < 0.01$,

* = $p < 0.05$,

t = trend; $p < 0.10$.

^a Positive correlations between EEG-based levels during tasks with Δ HRV indices indicate that frontal activity is associated with an decrease in the HRV index because brain activity is reflected by lower EEG values. Negative correlations indicate that increased frontal activity is associated with increased Δ HRV indices.

^b Positive correlations between changes in EEG data with Δ HRV indices indicate that increases in frontal activity are associated with a decrease in the HRV index, and negative correlations indicate that frontal activation is associated with increases in Δ HRV indices.