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Cerebral moderation of cardiovascular functioning: A functional cerebral systems perspective

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ABSTRACT

Objective: Interhemispheric and intrahemispheric balance models may be integrated to gain an increased understanding of how cerebral systems are involved in the regulation of heart rate and blood pressure. We sought to examine the relationship between left and right frontal and posterior activity and resting heart rate and blood pressure. Based on this integration, we predicted that lateral (left minus right hemisphere) and longitudinal (frontal minus posterior regions) asymmetry in cerebral activity would be related to resting measures of heart rate and blood pressure.
Methods: Resting heart rate, blood pressure, and EEG (low and high beta) were obtained in a sample of 42 men. Physiological measures were obtained during an eyes closed resting period.
Results: Our results provided partial support, finding significant correlations between resting heart rate and not only frontal lobe lateral asymmetry but also frontal–parietal asymmetry.
Conclusions: These results provide support for the relative differential associations of the left and right frontal and parietal lobes and cardiovascular activity.
Significance: Previous research has not examined cerebral control of cardiovascular functioning from a functional cerebral systems perspective. The results are discussed as they relate to research on aggression and hostility.

1. Introduction

The brain functions in a concerted, coordinated, and integrated manner, with no single region being exclusively associated with any given function. Petr Anokhin wrote extensively about the functional systems organization of brain functioning as it relates to goal-directed behavior (see Egiazaryan and Sudakov, 2007; Sudakov, 1998). Luria (1980) also wrote about the concept of a functional cerebral system. Extensive networks of intrahemispheric and interhemispheric fibers connect each region of the brain. Through these extensive connections activation in one region may spread to other regions of the brain, permitting the brain to act as a functional system. However, the precise influence of one area on another varies along both longitudinal and lateral anatomical axes.

Regarding the longitudinal axis, the frontal lobes are known to be inhibitory in nature, providing an inhibitory influence particularly over the posterior regions of the brain. Derek Denny-Brown (1950, 1956, see also Denny-Brown and Chambers, 1958) proposed a mutually inhibitory model of fronto-posterior functioning such that the frontal lobes are inhibitory over the posterior regions and the posterior regions, in turn, are inhibitory over the frontal lobes. Further, lesions within either area will disrupt this balance and result in a transcortical functional release of the functions associated with the intact region. Hence, lesions of the frontal lobes will release the approach-related behaviors (exploration) of the parietal lobes, and parietal lobe lesions will release the withdrawal-related behaviors (avoidance) of the frontal lobes.

A balance model has also been proposed to characterize the relationship between the left and right cerebral hemispheres. Specifically, Tucker (1981) proposed that the two hemispheres exist in a reciprocally balanced relationship, with each hemisphere opposing and complementing the other. Thus, increased activation in hemispheric region will result in decreased activation in the homologous region. This relationship may be observed through functional imaging of the effects of motor movement. Allison and colleagues found that hand movements generate increased activation in the sensorimotor area contralateral to the hand that is moved and deactivation in the ipsilateral area (Allison et al., 2000).

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An integration of these models indicates the dynamic system and interplay that exists within the brain and this integration suggests that activation or deactivation in one area of the brain may have specific, cascading interhemispheric and intrahemispheric effects on distal regions. As an example, relative right frontal lobe activation may lead to increased inhibition, i.e. decreased activation, of the right posterior region, and hence a shift of interhemispheric balance favoring the left posterior region. The increased left posterior activity may also result from the imbalance that is created across the frontal lobes. The relative right frontal lobe activation will generate deactivation at the left frontal lobe and hence disinhibition of the left posterior region (see Fig. 1A). However, relative left frontal lobe activation may lead to increased inhibition of the left posterior region, and the subsequent imbalance of activity will result in increased right posterior activity. As before, the increased right posterior activity may also result from the imbalance of activity across the frontal lobes, with decreased right frontal activity and hence disinhibition of right posterior functioning (see Fig. 1B).

This model of the dynamic interplay between the left and right frontal and posterior regions of the brain may be used to increase our understanding of how the brain may modulate cardiovascular functioning. Extensive research has been conducted examining the influence of the frontal, temporal, and parietal regions on heart rate and blood pressure. Research has consistently found a relationship between changes in heart rate and blood pressure and measures of cerebral activity at these locations, such as electroencephalography (EEG) (Foster and Harrison, 2004; Kubota et al., 2001; Umeno et al., 2003; Waldstein et al., 2000) and positron emission tomography (Gianaros et al., 2004; Lane et al., 2000). However, given that the modulation of heart rate and blood pressure is represented within a differentiated functional cerebral system, the nature of the relationship between cerebral activity and cardiovascular functioning will vary along both lateral and longitudinal neuroanatomical axes.

The two cerebral hemispheres seem to possess contrasting roles in regulating changes in heart rate and blood pressure. Wittling (1997) postulated that the left hemisphere is associated with parasympathetic activity and the right hemisphere is associated with sympathetic activity. The results of a seminal investigation by Wittling (1990) found that presentation of a positively valenced emotional film to the right hemisphere generated significantly greater increases in systolic and diastolic blood pressure than presentation of the same film to the left hemisphere. Subsequent investigations by Wittling and colleagues have provided further support for parasympathetic lateralization to the left hemisphere (Wittling et al., 1998a) and sympathetic lateralization to the right hemisphere (Wittling et al., 1998b). Further, stimulation of the left insula is associated with bradycardia and depressor responses, and stimulation of the right insula is associated with tachycardia and pressor responses (Oppenheimer et al., 1992). Inactivation of the left hemisphere following intracarotid sodium amobarbital (ISA) injections generates sympathetic predominance (Yoon et al., 1997). Zamrini et al. (1990) reported increased heart rate following left ISA injection and decreased heart rate following right ISA injection. Finally, Hilz et al. (2001) found that right hemisphere ISA injection generated significant decreases in blood pressure and a significant increase in the high frequency component of power spectral analysis of heart rate and blood pressure. Conversely, left hemisphere ISA injection was accompanied by increased heart rate and blood pressure.

Along the longitudinal axis, the temporal and posterior regions possess an excitatory role in regulating heart rate and blood pressure (Cechetto and Saper, 1990; Neafsey, 1990; Oppenheimer,

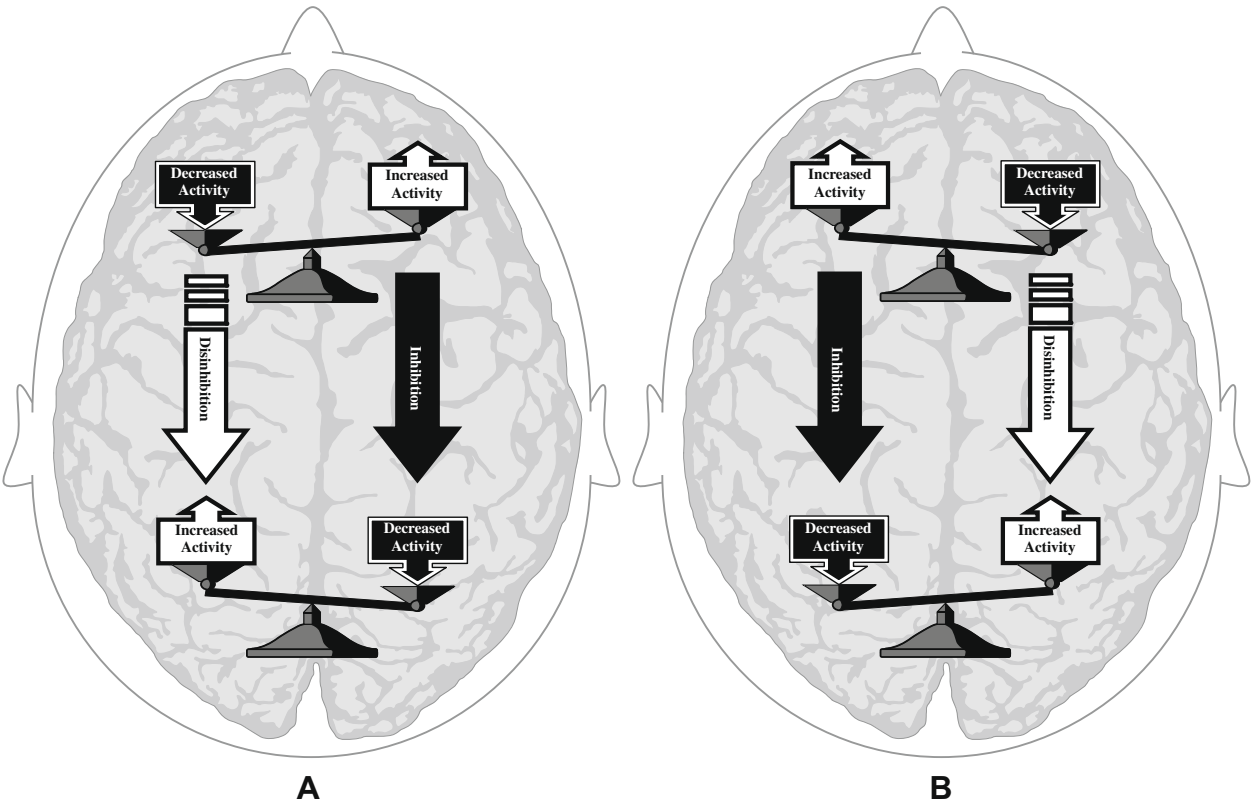

Fig. 1. (A) The cascading effects of right frontal activation on the left frontal and posterior regions of the brain. (B) The cascading effects of left frontal activation on the right frontal and posterior regions of the brain.

1992). As mentioned previously, stimulation of the left insular cortex produces increased parasympathetic tone and hence bradyphrenia and depressor responses, but stimulation of the right insular cortex produces increased sympathetic tone and hence tachycardia and pressor responses (Oppenheimer et al., 1992). Further, consistent with the inhibitory role of the frontal lobes, stimulation of the medial prefrontal regions generates bradycardia and depressor responses (Hardy and Holmes, 1988) and inhibition of conditioned increases in heart rate and blood pressure (al Maskati and Zbrozyna, 1989; Zbrozyna and Westwood, 1991, 1993). Further, high hostile individuals are associated with decreased activity at the right frontal lobe (Demaree and Harrison, 1996; Everhart and Harrison, 1995). When exposed to stressful situations, such as left hand cold pressor, the right frontal lobe of high hostile individuals is unable to inhibit activation of the right posterior regions associated with sympathetic activity. Thus, when exposed to left hand cold pressor, high hostile individuals experience heightened heart rate and blood pressure (Demaree and Harrison, 1997; Demaree et al., 2000).

An increased understanding of the cerebral modulation of heart rate and blood pressure may be derived by integrating all of the aforementioned research. Whereas the left frontal lobe may have a role in inhibiting the left posterior regions associated with parasympathetic excitation, the right frontal lobe may inhibit the right posterior regions associated with sympathetic excitation. Further, given an integration of the aforementioned balance models, one may suppose that a cascading effect of changes in cerebral activity may exist. Specifically, relative right frontal activation will generate increased inhibition of the right posterior region as well as decreased left frontal lobe activation, resulting in increased left posterior (parasympathetic) activity. Conversely, relative left frontal lobe activation will cause increased inhibition of the left posterior region as well as decreased right frontal lobe activity, resulting in increased right posterior (sympathetic) activity.

We recently reported the results of an investigation that sought to test this model by conducting a series of correlations between resting measures of heart rate and blood pressure and lateral asymmetry of cerebral activity (Foster and Harrison, 2006). Lateral asymmetry of cerebral activity was calculated by using difference scores obtained subtracting the EEG amplitude of each right hemisphere electrode site from the amplitude of each homologous left hemisphere electrode site. Given the model, it was hypothesized that significant negative correlations would be found between resting measures of heart rate and blood pressure and alpha (8–13 Hz) amplitude (magnitude or μV) asymmetry across the frontal lobe electrode sites. Further, we also hypothesized that significant positive correlations would be found between resting measures of heart rate and blood pressure and alpha amplitude asymmetry across the temporal and posterior electrode sites. The findings generally supported the hypotheses, with significant negative correlations between heart rate and frontal alpha amplitude asymmetry and significant positive correlations between systolic and diastolic blood pressure and temporal and parietal alpha amplitude asymmetry.

Our previous investigation, though, only tested part of the model due to the fact that only correlations between indices of lateral asymmetry of cerebral activity and heart rate and blood pressure were conducted. Hence, the influence of only the lateral neuroanatomical axis was directly evaluated. This investigation sought to test the full model by conducting a series of correlations using indices of both lateral asymmetry of cerebral activity and longitudinal asymmetry. Similar to our previous investigation, lateral cerebral activity asymmetry was defined as a difference score obtained by subtracting the low beta (13–21 Hz) and high beta (21–32 Hz) magnitude (μV) of each right hemisphere electrode from the low and high beta magnitude of each homologous left hemisphere electrode. Longitudinal cerebral activity asymmetry was defined as a difference score obtained by subtracting the low and high beta magnitude of each posterior (temporal, parietal, and occipital) electrode site from the low and high beta magnitude of each frontal lobe electrode site of the same hemisphere. Hence, positive and negative lateral cerebral activity scores represent relatively greater right and left hemisphere activity, respectively. Further, positive and negative longitudinal cerebral activity scores represent relatively greater posterior and frontal activity, respectively. Thus, regarding the lateral neuroanatomical axis, we predicted that significant negative correlations would be found between resting measures of heart rate and blood pressure and asymmetry of low and high beta magnitude across the frontal lobe electrode sites. Further, we predicted that significant positive correlations would be found between resting measures of heart rate and blood pressure and asymmetry of low and high beta magnitude across the posterior electrode sites. Regarding the longitudinal neuroanatomical axis, we predicted that significant negative correlations would be found between resting measures of heart rate and blood pressure and asymmetry of low and high beta magnitude across the left hemisphere electrode sites. Finally, we predicted that significant positive correlations would be found between resting measures of heart rate and blood pressure and asymmetry of low and high beta magnitude across the right hemisphere electrode sites.

2. Methods

2.1. Participants

A total of 42 males, with an age range of 18–29 years (M = 20.02, SD = 2.10), agreed to participate in exchange for extra credit in their undergraduate introduction to psychology course. All participants were right handed, as assessed by their scores on the Coren, Porac, and Duncan Laterality Questionnaire (CPD). Specifically, to be included the participants had to score a +5 on the CPD and indicate that both parents were right handed. The participants had no history of significant head injury, neurological illness or psychological disturbance, and were not presently taking any medications. Participants were treated in accordance with the ethical principles of the American Psychological Association.

2.2. Apparatus

2.2.1. Coren, Porac, and Duncan Laterality Questionnaire

The CPD (Coren et al., 1979) is a self-report questionnaire consisting of 13 questions assessing lateral preference for the hand, foot, eye, and ear. Responses are scored as +1 for "right", −1 for "left", and 0 for "both". Thus, the range of scores possible on the CPD is from −13 to +13.

2.2.2. Cardiovascular

Heart rate (HR) measured in beats per minute, systolic blood pressure (SBP) and diastolic blood pressure (DBP), both measured in mm Hg, were measured using a Norelco Model HC3501 Digital Blood Pressure Monitor (North American Philips Corporation, Stamford, CT). Systolic and diastolic blood pressure were assessed using the oscillometric measurement method with automatic inflation of the cuff. Exhaust was performed automatically at a rate of 3 mm Hg/s. The manufacturer provides accuracy specifications of ±3 mm Hg for blood pressure and ±5% for pulse measurements. Correlation coefficients of .84, .52, and .80 between the oscillometric and ausculatory methods of measuring systolic blood pressure, diastolic blood pressure, and heart rate, respectively, have been reported in the literature (Harrison and Kelly, 1987). The procedure for measuring heart rate, systolic blood pressure, and diastolic

blood pressure adhered to the basic requirements of the Association for the Advancement of Medial Instrumentation an the American Heart Association (see Harrison et al., 1988). The left arm of the participants was partially extended, supported, and positioned at approximately the fourth intercostal space with the palm facing upward. The location of the brachial artery was performed by palpitation. The cuff was then snuggly positioned on the left upper arm over the brachial artery, or approximately 2.5 cm above the antecubital space. The cuff was removed and repositioned over the brachial artery in the event of an error reading.

2.2.3. Quantitative electroencephalography

Quantitative electroencephalography (QEEG) was measured using a NeuroSearch-24 (Lexicor Medical Technology, Inc., Boulder, CO). Monopolar QEEG recordings, with linked ear references, were obtained using a lycra electrode cap (Electro-Cap International, Inc., Eaton, OH) containing 19 pure tin electrodes filled with ECI electrode gel. Electrodes were arranged according to the International 10/20 System. Silver–silver chloride electrodes filled with conductive paste were used for the ear reference electrodes. A Model 1089 mk II Checktrode Electrode Tester (Lexicor Medical Technology, Inc., Boulder, CO) was used to check the impedance levels. Measurement and analysis of electroencephalography adhered to the standards set forth by Pivik et al. (1993).

2.2.4. Electro-oculography

Auxiliary channels of the NeuroSearch-24 and silver–silver chloride electrodes filled with ECI electrode gel were used to measure electro-oculography (EOG) activity over the participant's left and right eyes.

2.3. Procedure

This project was approved by the university Institutional Review Board. The participants initially signed an informed consent form, and were provided with a brief description of the study. The lycra electrode cap was then fitted and attached to the participant's scalp using the appropriate anatomical landmarks. To ensure a securely positioned electrode cap, therefore minimizing artifacts from electrode movement, the cap was attached with elastic straps connected to a body harness placed around the participant's chest. The impedance levels for all electrodes were then checked, with all impedances falling below 5 kΩ and in most instances below 3 kΩ. The average interelectrode impedance level between any two electrodes did not exceed 700 Ω. The electrodes for measuring EOG activity over the left and right eyes were then attached.

The participants were then seated in a chair located in a sound attenuated chamber. Once seated the blood pressure cuff of the Digital Blood Pressure Monitor was attached to the participant's left arm. The participants were then instructed to sit quietly and relax, keeping their eyes closed and to remain as still as possible throughout the remainder of the study. Approximately two minutes following these instructions the QEEG baseline measurement was obtained, consisting of forty-five 1-s epochs. Quantitative electroencephalography was measured using a sampling rate of 256 Hz, with frequencies less than or equal to 2 Hz eliminated by a high pass filter. The NeuroSearch-24 software digitizes the EEG signal and provides amplitude and power for specified bandwidths, based on spectral analysis. Low (13–21 Hz) and high (21–32 Hz) beta magnitude (amplitude) was obtained and used in statistical analyses. We chose to use low and high beta magnitude in this investigation since our sample was comprised solely of men, in contrast to our previous investigation which used a sample of women and alpha magnitude (Foster and Harrison, 2006). Research has indicated that significant differences exist is resting asymmetry of alpha amplitude between men and women (Glass, 1968; Ray et al., 1976; Wada et al., 1994). Indeed, we have found that alpha is significantly correlated with changes in heart rate and blood pressure in women (Foster and Harrison, 2004), but that low and high beta are significantly correlated with changes in heart rate and blood pressure in men (Foster and Harrison, 2002b). Electro-oculographic measurements were obtained during the recording of QEEG activity.

Baseline measurements of heart rate, systolic and diastolic blood pressure were then obtained. The establishment of baseline cardiovascular functioning occurred in two stages. Initial baseline measurements of heart rate and blood pressure were obtained following the measurement of QEEG activity. Immediately following the initial baseline measurement of cardiovascular functioning, a second baseline measurement of heart rate and blood pressure was obtained. The measurements obtained from these two recordings were then averaged, with this average constituting the baseline from which the statistical analyses were conducted.

3. Results

3.1. Data reduction

Prior to conducting statistical analyses, each 1-s QEEG epoch resulting from the baseline condition was individually reviewed for the purpose of removing the 1-s epochs suspected of containing artifacts resulting from muscle movements or other contaminants. Specifically, artifacting of the epochs involved deleting any 1-s epoch noted to contain QEEG activity whose magnitude exceeded ±50 μV as well as those containing artifacts related to eye movements, as identified by EOG activity. Finally, epochs suspected of contamination by electrode movements and EKG artifacts were deleted. This process resulted in an average of about 24% of the epochs being deleted for any given subject. These deleted epochs were not used for calculating EEG amplitude. Hence, all analyses (i.e. lateral and longitudinal correlations) were based on the same number of accepted, artifact-free epochs.

For the purpose of conducting correlational analyses between resting measures of cardiovascular functioning and QEEG, difference scores were calculated based on the low and high beta magnitude of each electrode site. More specifically, to examine the lateral neuroanatomical axis, the low beta magnitude from each right hemisphere electrode site was subtracted from the low beta magnitude from each homologous left hemisphere electrode site (i.e. left hemisphere minus right hemisphere). Further, to examine the longitudinal neuroanatomical axis, the low beta magnitude from each posterior electrode site was subtracted from the low beta magnitude of each frontal lobe electrode site of the ipsilateral hemisphere. This same procedure was then used to calculate difference scores for the high beta bandwidth. Correlational analyses were then conducted between these low and high beta differences scores and the averaged baseline measurements of heart rate and blood pressure.

3.2. Analyses

Given the number of correlations involved, alpha was set at $p < .01$. Regarding the lateral neuroanatomical axis, significant negative correlations were found between baseline heart rate and low beta magnitude asymmetry at the frontal poles and dorsolateral frontal region. A significant negative correlation was also found between baseline heart rate and high beta magnitude asymmetry at the dorsolateral frontal region (see Table 1). Baseline heart rate was not significantly correlated with low or high beta magnitude asymmetry at the posterior regions. Further, resting measures of

Table 1
Lateral asymmetry correlations between low and high beta and all three cardiovascular measures

	HR	SBP	DBP		HR	SBP	DBP
Low beta				*High beta*			
F1–F2	−**.426** (**.002**)	.273 (.040)	−.196 (.107)	F1–F2	−.122 (.220)	.033 (.418)	−.099 (.266)
F3–F4	−**.471** (**.001**)	.297 (.028)	−.246 (.058)	F3–F4	−**.360** (**.010**)	.119 (.226)	−.181 (.125)
F7–F8	−.242 (.061)	−.004 (.490)	−.328 (.017)	F7–F8	−.245 (.059)	−.014 (.466)	−.293 (.030)
T3–T4	−.252 (.054)	.228 (.074)	−.270 (.042)	T3–T4	−.224 (.077)	.296 (.028)	−.082 (.303)
T5–T6	−.198 (.104)	.213 (.088)	−.089 (.287)	T5–T6	−.346 (.012)	.177 (.131)	−.181 (.126)
C3–C4	−.104 (.256)	.119 (.226)	−.197 (.106)	C3–C4	−.055 (.364)	−.003 (.492)	−.008 (.479)
P3–P4	.139 (.191)	.198 (.104)	−.303 (.026)	P3–P4	−.209 (.092)	.225 (.076)	−.277 (.038)
O1–O2	−.238 (.064)	.281 (.036)	−.013 (.467)	O1–O2	−.281 (.036)	.225 (.076)	−.139 (.190)

Note. Correlations in bold typeface are statistically significant ($p < .01$). HR represents baseline heart rate, SBP represents baseline systolic blood pressure, and DBP represents baseline diastolic blood pressure.

systolic and diastolic blood pressure were not significantly correlated with low or high beta asymmetry at either the frontal lobes or the posterior regions. Regarding the longitudinal neuroanatomical axis, significant positive correlations were found between heart rate and low beta magnitude asymmetry between all three right frontal lobe electrode sites and the right parietal lobe. Additionally, a significant negative correlation was found between resting systolic blood pressure and low beta magnitude asymmetry between the left frontal and left parietal electrode sites (see Table 2). No significant correlations were found using high beta magnitude asymmetry (see Table 3).

Additionally, as a type of manipulation check, we conducted a series of correlations between the frontal lobe low and high beta magnitude asymmetries and the posterior low and high beta magnitude asymmetries. Given the model driving our hypotheses, we would expect negative correlations to exist between the frontal and posterior magnitude asymmetries. As before, alpha was set at $p < .01$ to control for the number of correlations. The results indicated that significant positive correlations existed between the frontal lobe low beta asymmetry and both temporal lobe and parietal lobe low beta asymmetry. No correlations using high beta asymmetries were significant (see Table 4).

4. Discussion

We sought to investigate the relationship between indices of lateral and longitudinal asymmetry of cerebral activity and resting heart rate and blood pressure. Our findings provided partial support for our hypotheses. Specifically, as predicted we found negative correlations between resting heart rate and low beta magnitude asymmetry at the frontal poles and dorsolateral frontal regions as well as high beta magnitude at the dorsolateral frontal region. Regarding asymmetry along the longitudinal axis, we found a significant negative correlation between resting systolic blood pressure and low beta magnitude asymmetry for the left frontal and left parietal regions. Additionally, significant positive correlations were found, as predicted, between resting heart rate and low beta magnitude asymmetry for the right frontal and right parietal regions. Hence, all of the significant correlations in our study were in accordance with our a-priori predictions.

Our findings indicate that patterns of asymmetry across the left and right frontal and then across the right frontal and right parietal regions are involved in resting heart rate. Specifically, asymmetry in activity favoring the left frontal lobe is associated with higher resting heart rate and as this asymmetry shifts to relatively higher right frontal lobe activity resting heart rate decreases. The relationship between resting heart rate and right frontal lobe activity then continues along the longitudinal neuroanatomical axis such that asymmetry favoring right frontal lobe activity was associated with lower resting heart rate and as activity decreased at the right frontal lobe and increased at the right parietal region resting heart rate increased. Thus, the findings provide support for a division of responsibility between the left and right frontal and posterior regions in regulating cardiovascular functioning. Our findings are also consistent with the view that the left hemisphere is involved in parasympathetic influences on cardiovascular functioning and the right hemisphere is involved in sympathetic influences. However, whereas our results provide support for the lateralization of the parasympathetic and sympathetic nervous systems to the left

Table 2
Longitudinal correlations axis correlations between low beta and all three cardiovascular measures

	HR	SBP	DBP		HR	SBP	DBP
Longitudinal axis–left hemisphere				*Longitudinal axis–right hemisphere*			
F1–T3	.306 (.024)	−.200 (.103)	−.013 (.467)	F2–T4	.125 (.25)	−.013 (.467)	−.029 (.428)
F1–T5	.184 (.121)	−.157 (.160)	.066 (.338)	F2–T6	.079 (.310)	−.005 (.488)	.019 (.453)
F1–C3	.097 (.271)	−.160 (.156)	.211 (.090)	F2–C4	.160 (.155)	−.168 (.144)	.134 (.199)
F1–P3	.260 (.048)	−.336 (.015)	.166 (.147)	F2–P4	**.366** (**.009**)	−.243 (.061)	.026 (.436)
F1–O1	.235 (.067)	−.326 (.018)	−.053 (.368)	F2–O2	.165 (.148)	−.213 (.088)	−.039 (.404)
F3–T3	.274 (.039)	−.052 (.372)	.083 (.300)	F4–T4	.178 (.129)	.078 (.311)	−.105 (.254)
F3–T5	.163 (.151)	−.002 (.495)	−.070 (.329)	F4–T6	.152 (.169)	.102 (.260)	−.073 (.323)
F3–C3	.093 (.279)	.103 (.259)	.001 (.498)	F4–C4	.349 (.012)	.008 (.480)	−.020 (.449)
F3–P3	.272 (.041)	−.227 (.074)	.054 (.367)	F4–P4	**.422** (**.003**)	−.166 (.146)	−.042 (.396)
F3–O1	.217 (.083)	−.206 (.096)	−.154 (.166)	F4–O2	.209 (.092)	−.116 (.231)	−.106 (.252)
F7–T3	.318 (.020)	−.242 (.061)	.125 (.215)	F8–T4	.189 (.116)	.017 (.458)	.015 (.462)
F7–T5	.182 (.124)	−.195 (.108)	−.045 (.389)	F8–T6	.116 (.233)	.024 (.440)	.060 (.353)
F7–C3	.079 (.309)	−.206 (.095)	.048 (.382)	F8–C4	.221 (.080)	−.109 (.246)	.195 (.108)
F7–P3	.252 (.053)	−**.364** (**.009**)	.075 (.318)	F8–P4	**.389** (**.005**)	−.214 (.087)	.058 (.358)
F7–O1	.231 (.071)	−.354 (.011)	−.138 (.192)	F8–O2	.193 (.111)	−.177 (.131)	−.001 (.498)

Note. Correlations in bold typeface are statistically significant ($p < .01$). HR represents baseline heart rate, SBP represents baseline systolic blood pressure, and DBP represents baseline diastolic blood pressure.

Table 3
Longitudinal correlations axis correlations between high beta and all three cardiovascular measures

	HR	SBP	DBP		HR	SBP	DBP
Longitudinal axis–left hemisphere				*Longitudinal axis–right hemisphere*			
F1–T3	.250 (.055)	−.118 (.229)	.130 (.206)	F2–T4	.099 (.265)	.095 (.275)	.083 (.300)
F1–T5	.180 (.127)	−.046 (.386)	.129 (.208)	F2–T6	−.026 (.434)	.093 (.279)	.051 (.375)
F1–C3	−.053 (.369)	.031 (.422)	.060 (.352)	F2–C4	−.037 (.408)	.017 (.456)	.104 (.255)
F1–P3	.148 (.174)	−.183 (.123)	.092 (.282)	F2–P4	.123 (.219)	−.125 (.215)	.026 (.435)
F1–O1	.219 (.082)	−.291 (.031)	.071 (.327)	F2–O2	.017 (.457)	−.164 (.150)	−.035 (.412)
F3–T3	.275 (.039)	−.083 (.301)	.118 (.229)	F4–T4	.199 (.104)	.110 (.244)	.102 (.260)
F3–T5	.257 (.050)	−.001 (.497)	.133 (.201)	F4–T6	.196 (.106)	.136 (.194)	.096 (.272)
F3–C3	.015 (.463)	.157 (.160)	.065 (.342)	F4–C4	.263 (.046)	.062 (.248)	.210 (.091)
F3–P3	.255 (.051)	−.173 (.137)	.100 (.264)	F4–P4	.354 (.011)	−.134 (.198)	.064 (.343)
F3–O1	.267 (.044)	−.287 (.033)	.068 (.334)	F4–O2	.138 (.191)	−.157 (.160)	−.018 (.455)
F7–T3	.279 (.037)	−.171 (.139)	.038 (.404)	F8–T4	.221 (.080)	.065 (.341)	.100 (.265)
F7–T5	.239 (.064)	−.130 (.206)	.004 (.491)	F8–T6	.206 (.095)	.015 (.462)	.070 (.329)
F7–C3	.000 (.500)	−.088 (.289)	−.148 (.174)	F8–C4	.259 (.049)	−.103 (.258)	.164 (.150)
F7–P3	.213 (.088)	−.297 (.028)	−.055 (.365)	F8–P4	.339 (.014)	−.227 (.074)	.038 (.405)
F7–O1	.250 (.055)	−.348 (.012)	−.007 (.481)	F8–O2	.137 (.193)	−.214 (.087)	−.031 (.423)

Note. Correlations in bold typeface are statistically significant ($p < .01$). HR represents baseline heart rate, SBP represents baseline systolic blood pressure, and DBP represents baseline diastolic blood pressure.

and right hemisphere, respectively, the results are also limited by the fact that our measures of cardiovascular functioning, and our primary finding, included heart rate. Cardiac functioning is simultaneously influenced by both the parasympathetic and sympathetic nervous systems at any given time, due to dual innervation of the sinoatrial node by both the vagus nerve and sympathetic fibers (Andreassi, 2000; Hugdahl, 1995). Hence, more conclusive statements would be possible by using more precise measures of parasympathetic and sympathetic functioning. For instance, impedance cardiography may be used as a measure of sympathetic activity (Andreassi, 2000) and heart rate variability as a measure of parasympathetic functioning (Friedman et al., 2002; Stein and Kleiger, 1999).

The central role played by the right frontal lobe in our study, as well as the relationship between heart rate and asymmetry along the longitudinal axis in the right hemisphere, is also consistent with the literature supporting a primary role of the right hemisphere in cardiovascular regulation. Some have suggested that the right hemisphere has a more dominant role in the cerebral regulation of cardiovascular functioning (Ahern et al., 2001; Lane and Jennings, 1995). Indeed, research has indicated that supraventricular tachycardia (Lane et al., 1992) and reduced heart rate variability (Colivicchi et al., 2004) result from right hemisphere strokes. Further, patients with right hemispheric strokes show an increased susceptibility to cardio-autonomic dysfunction (Strittmatter et al., 2003) and strokes lateralized to the right insula have been associated with sympathetic activation (Meyer et al., 2004), systolic hypertension (Cereda et al., 2002), and reduced heart rate variability (Tokgozoglu et al., 1999).

Anomalies in resting heart rate have been also extensively studied in the field of antisocial and aggressive behavior. Low resting heart rate has been identified as among the most replicated biological correlates of antisocial and aggressive behavior in child and young adults (Raine, 2003). Among the mechanisms discussed by Raine et al. (2002) to explain the relationship between low heart rate and aggressive and antisocial behavior is the right hemispheric dysfunction often seen in this population. Aggression following severe closed head injury is associated with lower cerebral blood flow in the right hemisphere, as measured by single photon emission computerized tomography (Oder et al., 1992). Further, violent behaviors in individuals with antisocial personality disorder are associated with relatively greater cortical thinning at the right medial inferior frontal and lateral sensory motor cortices (Narayan et al., 2007). Wang et al. (2003) reported that individuals with antisocial personality erred to the right when performing the line bisection task, indicating left hemisphere activation and right hemisphere deactivation. Antisocial behaviors are associated with relative right hemisphere dysfunction even in individuals as young as 3 years of age, as evidence by their performance on tests of spatial and verbal functioning (Raine et al., 2002). Our findings are consistent with this literature in that decreased activity at the right parietal region, relative to the right frontal lobe, was associated with lower resting heart rate.

Our findings are also consistent with the literature on the effects of hostility on cardiovascular regulation. Individuals with heightened hostility possess decreased right frontal lobe activity (Demaree and Harrison, 1996; Everhart and Harrison, 1995). Consequently, high hostile individuals also exhibit disinhibition of the right posterior cerebral systems involved in regulating sympathetic influences on cardiovascular activity, as evidenced by heightened heart rate and blood pressure responses to stress (Demaree and Harrison, 1997; Demaree et al., 2000). Thus, when exposed to situational stressors such as a cold pressor task involving the left hand, the right frontal lobe is unable to inhibit activation of the cerebral systems in the right posterior regions responsible for regulating sympathetic activity. Our results are consistent with these findings in that asymmetry across the right hemisphere characterized by lower activity at the right frontal lobe and higher activity at the right posterior region was associated with heightened heart rate.

Our results also indicated that asymmetry across the left frontal and left parietal regions was related to resting systolic blood pressure. Indeed, the overall findings indicated that resting heart rate was significantly related to longitudinal asymmetry in the right hemisphere and resting systolic blood pressure only in the left hemisphere. Additionally, resting systolic blood pressure was not

Table 4
Correlations among lateral frontal and posterior low and high beta magnitudes

	T3–T4	T5–T6	C3–C4	P3–P4	O1–O2
Low beta					
F1–F2	**.498 (.0004)**	.304 (.025)	**.438 (.002)**	.213 (.088)	−.081 (.304)
F3–F4	**.598 (.00001)**	.337 (.015)	**.575 (.00003)**	.169 (.142)	.022 (.444)
F7–F8	**.447 (.002)**	.101 (.263)	**.533 (.0001)**	.221 (.08)	−.011 (.473)
High beta					
F1–F2	.238 (.065)	.172 (.138)	−.129 (.208)	.104 (.256)	−.238 (.065)
F3–F4	.286 (.033)	.269 (.043)	−.024 (.440)	.302 (.026)	.029 (.428)
F7–F8	.155 (.164)	.204 (.097)	.239 (.063)	.303 (.025)	.000 (.498)

Note. Correlations reported in bold typeface are statistically significant ($p < .01$).

significantly related to lateral asymmetry at any two homologous sites. The reason for this dissociation between systolic blood pressure and heart rate is unknown. Our previous investigation also indicated a dissociation between cerebral asymmetry and resting heart rate and blood pressure. Specifically, whereas resting heart rate was associated with lateral asymmetry across the frontal and parietal lobes, resting systolic and diastolic blood pressure were related to lateral asymmetry across the temporal and parietal lobes (Foster and Harrison, 2006). Taken together, these findings seem to suggest that different functional cerebral systems may exist that are involved in regulating heart rate and blood pressure. This would seem to be consistent with the concept of directional fractionation, i.e. that the overall pattern of psychophysiological responding to stressors may be fractionated in different directions such as with increases in heart rate and decreases in skin conductance (see Hugdahl, 1995; Andreassi, 2000).

Although our findings support the model being investigated and indicate that patterns of cerebral activity across the left and right frontal and posterior regions are involved in the regulation of heart rate, this study is limited by the fact that only baseline measurements of cardiovascular functioning and cerebral activity were obtained. As mentioned previously, the brain is dynamic and future investigations will need to be conducted to determine whether the lateral and longitudinal cerebral asymmetries identified in this investigation are also involved in situations that involve changes in cardiovascular functioning. For instance, future research should explore whether these asymmetries exist following recollection of emotional memories, which is known to produce changes in both cerebral (Foster and Harrison, 2002a; Lane et al., 1997; Markowitsch et al., 2003; Reiman et al., 1997) and cardiovascular functioning (Foster and Webster, 2001; Labouvie-Vief et al., 2003; Marci et al., 2007). An investigation by Gianaros and colleagues sought to examine whether heightened cardiovascular activity was associated with greater activity in the orbitofrontal cortex, cingulated cortex, or in the insular cortex (Gianaros et al., 2005). Their findings indicated that individuals with high cardiovascular reactivity to a behavioral stressor exhibited greater posterior cingulated cortex activation. However, this investigation did not assess the potential relationship between changes in cardiovascular activity and changes or shifts in lateral and/or longitudinal cerebral activation asymmetry.

Although we found significant correlations in the predicted locations and directions, it should be mentioned that the amount of variability explained (i.e. coefficient of determination) is relatively low. Specifically, regarding the lateral asymmetry correlations, the coefficient of determination for the heart rate and low beta F1–F2 correlation was .182, with coefficients of .222 and .130 for the heart rate and low beta and high beta F3–F4 correlations, respectively. Regarding the longitudinal correlations, the coefficients indicated that less than 20% of the variance was explained (.133 for systolic blood pressure and F7–P3; .134 for heart rate and F2–P4, .178 for heart rate and F4–P4; and .151 for heart rate and F8–P4). Thus, much of the variability between the lateral and longitudinal asymmetries and baseline heart rate and blood pressure was not accounted for by these findings. The unexplained variance may be due to the influence of other regions of the brain, such as the subcortical structures that are involved in regulating heart rate and blood pressure. Although we analyzed the lateral and longitudinal asymmetries, another equally important dimension lies along the neuraxis in the brainstem. For instance, the Central Autonomic Network (CAN, Benarroch, 1993) is proposed as a central component of an internal regulation system that is responsible for modulating visceromotor responses. The CAN consists of a number of structures, including the medial prefrontal and insular cortices, amygdale, hypothalamus, periaqueductal gray matter, parabrachial complex, nucleus of the solitary tract, and ventrolateral medulla. Additionally, the pattern and strength of the lateral and longitudinal correlations found in the present investigation may change or increase given an activation paradigm. Certainly, the results of the present investigation should be replicated using a paradigm that involves generating changes in cardiovascular functioning.

The use of an activation-type paradigm would also be useful to further investigate the model on which our hypotheses were based. Given our model, we would expect negative correlations to exist between the frontal lobe and posterior region asymmetries. However, we found positive correlations, meaning that the asymmetries in baseline activity were in the same direction across the left and right frontal and posterior regions. Clearly, this is in contrast with the model that drove our hypotheses. The finding of positive correlations was not expected given that our primary findings were consistent with our hypotheses. The reason that frontal and posterior region asymmetries were positively correlated may be related to the fact that this study investigated resting measures of cerebral and cardiovascular activity. Previous research has supported both components of the model. Hand movements generate activation in the sensorimotor cortex contralateral to the hand that is moved and deactivation in the ipsilateral sensorimotor cortex (Allison et al., 2000). Research has also generally supported Denny-Brown's proposal that a mutually inhibitory relationship exists between the frontal and posterior regions (Vilensky and Gilman, 1997). Perhaps these relationships become manifest in research paradigms that involve more localized cerebral activation. A lateralized stressor such as the cold pressor paradigm used by Harrison and colleagues (Demaree and Harrison, 1997; Demaree et al., 2000) may generate local changes in cerebral activity that conform more closely to the model. Future research will need to be conducted to determine whether this is, in fact, the case.

Our findings are also based on a sample comprised entirely of men. Significant differences in alpha amplitude between men and women have been reported (Wada et al., 1994), as well as differences in total spectral amplitude at specific sites (Briere et al., 2003). Others have noted significant gender differences in various cardiovascular functions, including heart rate (Nagy et al., 2000) and heart rate variability (Stein et al., 1997). Due to these reported differences, we limited our sample to only men in this investigation. Indeed, our previous investigation (Foster and Harrison, 2006) used a sample of women, and the results were not consistent in some regards with this study. Both investigations found negative correlations between resting heart rate and frontal lobe asymmetry. However, whereas our previous investigation also found significant positive correlations between resting systolic and diastolic blood pressure and asymmetry at the temporal lobes, the present investigation did not find any significant relationships between lateral asymmetry and blood pressure at any site. Hence, further studies should attempt to examine whether differences exist between men and women in the functional cerebral system regulation of cardiovascular activity.

The potential confounding effects of artifact removal should also be mentioned. The process of removing epochs suspected of containing artifacts involved reviewing each epoch to determine whether electrode movement, EOG, or EKG affected the recording of EEG. The vast majority of removed epochs seemed to be primarily affected by EOG. However, we cannot rule out that EKG contributed to artifacts. Hence, the possibility exists that this process introduced a bias such that epochs associated with higher heart rate, or even lower heart rate, were differentially removed.

Finally, as mentioned previously, our findings are consistent with a division of responsibility between the left and right frontal and parietal regions in regulating cardiovascular functioning. However, our study was limited not only by the use of baseline or rest-

ing measures of cardiovascular and brain activity but also by the correlational design. Thus, although our findings provide support for a functional cerebral system in regulating cardiovascular activity, more conclusive statements cannot be made with the available data since an activation-type paradigm involving changes in cardiovascular activity was not used. The data from the present investigation preclude statements regarding cause–effect relationships. Future research should investigate whether differential changes in activation across the left and right frontal lobes and posterior regions are associated with increases or decreases in cardiovascular activity, preferably using an experimental design.

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