Graded Cerebral Activation to Noise:

Behavioral and Cardiovascular Effects

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Abstract

Research has indicated that the frontal and temporal lobes are involved in the mediation of heart rate and blood pressure. However, whereas these regions of the brain have been identified in the mediation of heart rate and blood pressure, the specific cerebral processes involved in determining the direction and magnitude of change in heart rate and blood pressure has not been adequately addressed. The present paper proposes that changes in the magnitude of cerebral activation between the left and right frontal and temporal lobes is partly that which determines the direction and magnitude of changes in heart rate and blood pressure. The present investigation sought to test part of this proposition, namely, that increasing magnitude of cerebral activity within the right anterior temporal region generates increasing levels of sympathetic control of heart rate and blood pressure and that the right lateral frontal region acts to inhibit sympathetic activity. A total of 45 right handed men, with no history of significant head injury, were exposed to 55 dB, 75 dB, and 90 dB white noise presentations. Right frontal lobe functioning was assessed by performance on the Ruff Figural Fluency Test (RFFT), with the participants scoring in the lower one-third classified as Low Fluency. Those scoring in the upper one-third were classified as High Fluency. Quantitative electroencephalography, measured at 19 electrodes sites arranged according to the International 10/20 System, as well as heart rate and blood pressure responses to white noise presentation were measured. Although the results failed to support any of the hypotheses concerning the effects of varying intensity of white noise on

cardiovascular activity, partial support was found for the hypotheses that varying intensity of white noise would generate differential changes in high beta magnitude between the Low and High Fluency groups. The results are discussed in terms of support for the model being tested. Alternative explanations of the findings are also provided that demonstrate correspondence between the QEEG and cardiovascular data. Finally, limitations of the model and the methods of the present investigation are discussed and suggestions for improvement are provided.

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This dissertation is in memory of my father, Gary David Foster, whose untimely passing has been difficult for me and my entire family.

Table of Contents

Table Captions

Table Captions (continued)

Figure Captions

Figure Captions (continued)

Figure Captions (continued)

Graded Cerebral Activation to Noise:

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A relationship between the brain and cardiovascular functioning, particularly heart rate and blood pressure, has been hypothesized to exist for some time. Nearly 160 years ago, James Braid stated that every part of the body supplied by nerves has a distinct corresponding point in the brain (1843/1976). Shortly thereafter, Hughlings Jackson (1875) similarly stated that the movements of not only the body, but also the movements of the arteries and viscera were represented in the cerebrum (as cited by Hoff, Kell, & Carroll, 1963). The time since these early statements were made has been filled with a veritable multitude of research investigations empirically testing this hypothesized relationship. The result has been a wealth of accumulated knowledge supporting not only the relationship between the brain and changes in heart rate and blood pressure, but also the cerebral sites associated with such a relationship. Indeed, reviews of the relevant literature spanning the late 19th and early 20th centuries have consistently demonstrated that stimulation of or lesions within the frontal and temporal lobes, motor and premotor cortices, and insular cortices produced changes in heart rate and blood pressure (Delgado, 1960; Hoff et al., 1963). More recent reviews of the literature have indicated that the medial prefrontal and insular regions, in particular, are involved in the regulation of heart rate and blood pressure (Cechetto & Saper, 1990; Neafsey, 1990; Verberne & Owens, 1998).

However, whereas the neuroanatomical sites associated with heart rate and blood pressure have been identified, the specific cerebral processes involved in determining the direction and magnitude of change in heart rate and blood pressure has not been adequately

addressed. Indeed, Wittling (1995) has stated that only preliminary information exists concerning the effects that the cerebral lateralization of physiological functions exerts on the strength and course of physiological functioning. The present paper will propose that the relative magnitude of cerebral activation between the left and right frontal and temporal regions is part of that which determines the direction and magnitude of change in heart rate and blood pressure. This proposition will be developed and supported by reviewing research findings demonstrating the involvement of the frontal and temporal lobes, including the insular regions, in the regulation of heart rate and blood pressure. Subsequently, research demonstrating the hemispheric lateralization of the parasympathetic and sympathetic nervous systems will be discussed. This discussion will then be followed by an integration of research concerning the cerebral and cardiovascular effects of increasing task difficulty, subjective intensity of pain, and increasing intensity of white noise presentation. A rationale for empirically testing the hypothesized relationship between changes in heart rate and blood pressure and the relative magnitude of cerebral activation across the right frontal and temporal regions will then be presented. Finally, specific methodology for investigating this relationship will be proposed.

Cerebral Sites Involved in the Regulation of Heart Rate and Blood Pressure

Research findings from diverse areas have implicated the involvement of specific cerebral areas in the regulation of heart rate and blood pressure. More specifically, as will be discussed in the following paragraphs, the cerebral basis of heart rate and blood pressure regulation has been investigated by assessing the cardiovascular effects of both lesions within and stimulation of the brain. Further evidence is provided by an integration of research involving the cardiovascular and cerebral effects of emotional arousal. The results of this

research have consistently indicated that the frontal and temporal lobes, including the insular regions, are involved in the mediation of heart rate and blood pressure.

Cardiovascular and Cerebral Effects of Emotional Arousal

Research has consistently indicated that the induction of positive emotions, such as happiness or mirth, results in significant changes in heart rate (Averill, 1969; Foster & Webster, 2001; Foster, Webster, & Williamson, 2002-2003; Levenson, Ekman, & Friesen, 1990; Schwartz, Weinberger, & Singer, 1981; Sinha, Lovallo, & Parsons, 1992; Vrana, 1993) and systolic blood pressure (Schwartz et al., 1981; Sinha et al., 1992). Similarly, negative emotions are associated with alterations in cardiovascular functioning. More specifically, anger and hostility have generated significant increases in heart rate (Demaree & Harrison, 1997a; Demaree, Harrison, & Rhodes, 2000; Ekman, Levenson, & Friesen, 1983; Foster, 2001a; Foster & Webster, 2001; Foster, Smith, & Webster, 1998-1999; Levenson et al., 1990; Schwartz et al., 1981; Sinha et al., 1992; Vrana, 1993) and systolic blood pressure (Demaree & Harrison, 1997a; Demaree et al., 2000; Foster, 2001a; Schwartz et al., 1981; Sinha et al., 1992). Additionally, the experimental induction of sadness has resulted in increases in heart rate (Averill, 1969; Ekman et al., 1983; Schwartz et al., 1981; Sinha et al., 1992) and systolic blood pressure (Averill, 1969; Sinha et al., 1992).

Alterations in cerebral activity across various regions of the brain are associated with the experimental induction of both positive and negative emotions as well. Positively and negatively valenced emotions are associated with alterations in cerebral activity within the temporal lobes as well as the lateral, medial, and orbital frontal areas (Robinson, 1995). More specifically, the experimental induction of positive emotions has resulted in lowered cerebral activation within the right prefrontal and bilateral temporal-parietal areas (George et al., 1995). Investigations

have found increased cerebral activation within the right temporal (Cole & Ray, 1985; Schellberg, Besthorn, Pfleger, & Gasser, 1993), left temporal (Davidson, 1993; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Sidorova, Kostyunina, & Kulikov, 1992; Tomarken, Davidson, Wheeler, & Doss, 1992), as well as the bilateral anterior, middle, and posterior temporal regions (Lane, Reiman, Ahern, Schwartz, & Davidson, 1997). Furthermore, positive emotions have generated increased cerebral activity within the frontal lobes (Sidorova & Kostyunina, 1993) and the medial prefrontal region (Lane, Reiman, Bradley, et al., 1997; Lane, Reiman, Ahern, et al., 1997).

Increased cerebral activity within the frontal lobes has been generated by the induction of negative emotions, such as anger, fear, and sorrow (Sidorova & Kostyunina, 1993). Additionally, hostility is associated with decreased right frontal lobe activity (Everhart & Harrison, 1995) and increased activity across the right hemisphere (Herridge, Harrison, & Demaree, 1997) as well as the right temporal lobe (Demaree & Harrison, 1997; Everhart & Harrison, 1995). Induction of disgust has generated increased cerebral activity within the right frontal and temporal lobes (Davidson, 1993; Davidson et al., 1990). Furthermore, fear results in decreased right frontal (Johanson et al., 1998) and increased left temporal activation (Sidorova et al., 1992). Increased cerebral activation within the right temporal lobe has resulted from the induction of negative emotion through the threat of electric shock as well (Konovalov & Serikov, 1990). Induction of sadness has resulted in increased activation within the bilateral insular (George et al., 1995; Partiot, Grafman, Sadato, Wachs, & Hallett, 1995; Reiman et al., 1997) as well as the bilateral anterior, middle, and posterior temporal regions (Lane, Reiman, & Ahern et al., 1997).

Significant relationships between cerebral activity in the frontal and temporal lobes and emotional memory intensity and age have also been found. Specifically, significant correlations were found between changes in cerebral activity and the intensity of mirthful memories at the left frontal and right temporal and parietal areas (Foster, Williamson, & Harrison, 2003) and the intensity of angry memories at the bilateral frontal, temporal, and parietal regions (Foster & Harrison, 2002a). Foster and Harrison (in press), using a sample of women, found a significant relationship between varying ages of angry memories and cerebral activity at the right frontal, left temporal, and bilateral parietal regions. Foster, Thompson, and Harrison (2004a) replicated this study using a sample of men and found significant correlations between ages of angry memories and changes in cerebral activity at the bilateral frontal and left parietal regions.

Taken together, the results of the investigations concerning the cerebral effects of emotions indicate that altered cerebral activity within the prefrontal and temporal lobes is common to both positively and negatively valenced emotions. Thus, given the aforementioned research findings of increases in heart rate and blood pressure in response to both positive and negative emotions, it may be inferred that the prefrontal and temporal lobes are concurrently involved in the regulation of these cardiovascular measures. However, based on this integration of research findings involving the cardiovascular and cerebral effects of emotional arousal, only inferences may be made in drawing conclusions concerning the involvement of the frontal and temporal lobes in cardiovascular functioning. More direct evidence for the involvement of these regions of the brain is provided by the results of investigations concerning the cardiovascular effects of lesions and stimulation of the brain.

Lesion Studies

Lesions arising from multiple sources, including tumors and strokes, are known to produce significant alterations in cardiovascular functioning. Indeed, fatal cardiac arrhythmias and sudden cardiac death may result from the alterations in cardiac regulation that accompany lesions within the central nervous system (Talman, 1985). Cerebrovascular insults and traumatic brain injuries to the right hemisphere also to result in significantly reduced heart rate responses to stress (Andersson & Finset, 1998). Strokes within the right hemisphere have resulted in supraventricular tachycardia (Lane, Wallace, Petrosky, Schwartz, & Gradman, 1992), cardiac arrhythmias (Barron, Rogovski, & Hemli, 1994), and reduced heart rate variability (Naver, Blomstrand, & Wallin, 1996). Although the aforementioned investigations have implicated the involvement of the right hemisphere in cardiovascular functioning, other studies have identified specific regions of the cerebral hemispheres. For instance, left insular lesions have resulted in increased sympathetic tone and decreased parasympathetic tone (Oppenheimer, Kedem, & Martin, 1996). Additionally, strokes localized within the region of the insula, particularly the right insula, are associated with reduced heart rate variability and increased incidence of sudden cardiac death (Tokgozoglu et al., 1999).

Lesion studies have implicated the frontal lobes in the regulation of cardiovascular functioning as well. More specifically, Rush, Everett, Adams, and Kuscke (1977) reported the case of a patient who presented with recurrent episodes of paroxysmal atrial tachycardia and who was found to have a right frontal lobe tumor. Additionally, the episodes of tachycardia subsequently ceased upon removal of the tumor. The role of the right frontal lobe in regulating cardiovascular functioning is further supported by research concerning the effects of hostility on heart rate and blood pressure. High levels of hostility are linked to decreased activation of the

right frontal lobe (Demaree & Harrison, 1996; Everhart & Harrison, 1995), which may result from cerebrovascular accidents. Additionally, high hostile individuals evidence greater increases in heart rate and blood pressure in response to stressors, as compared to low hostile individuals (Demaree & Harrison, 1997a; Demaree et al., 2000).

Stimulation Studies

Researchers have known for some time that stimulation of the frontal lobes generates significant changes in cardiovascular functioning (Hoff & Green, 1936). More recently, research has indicated that gross stimulation of the frontal lobes through electroconvulsive therapy (ECT) results in significant increases in heart rate and blood pressure (Keilson, Hauser, & Magrill, 1989; Wells & Davies, 1987). The effects of ECT on heart rate and blood pressure have been demonstrated for both bilateral and right unilateral ECT. More specifically, bilateral ECT has generated significant increases in systolic blood pressure (O'Donnell & Webb, 1986; Prudic et al., 1987), diastolic blood pressure (Prudic et al., 1987), and heart rate (Lane, Zeitlin, Abrams, & Swartz, 1989; Prudic et al., 1987). Similarly, right unilateral ECT generates significant increases in systolic and diastolic blood pressure (Prudic et al., 1987), heart rate (Lane et al., 1989; Prudic et al., 1987), as well as rate-pressure product, or the product of heart rate and systolic blood pressure (Webb et al., 1990). The cardiovascular effects of bilateral and right unilateral ECT appear to be roughly equivalent, with both generating similar increases in heart rate as well as systolic and diastolic blood pressure (Lane et al., 1989; Prudic et al., 1987). However, the cardiovascular effects of left versus right unilateral ECT have been found to differ. Whereas left and right unilateral ECT produce significant increases in heart rate and comparable peak heart rate values, right unilateral ECT generates greater overall increases in heart rate (Swartz, Abrams, Lane, DuBois, & Srinivasaraghavan, 1994). Thus, generally speaking, gross

stimulation of the frontal lobes through bilateral as well as left and right unilateral ECT has generated significant increases in heart rate as well as systolic and diastolic blood pressure.

Stimulation of more localized regions of the frontal lobes through high rate transcranial magnetic stimulation has resulted in significant changes in heart rate and blood pressure. For instance, Foerster, Schmitz, Nouri, and Claus (1997) applied high rate transcranial magnetic stimulation to the C3, C4, FZ, and PZ electrode sites, arranged according to the International 10/20 System. The results indicated that high rate transcranial magnetic stimulation applied at all four electrode sites generated significant increases in heart rate. A biphasic blood pressure response was found, with an initial increase in systolic and diastolic blood pressure followed by a significant reduction in systolic and diastolic blood pressure. Similarly, Claus, Foerster, Schmitz, Bochannek, and Nouri (1999) found increased heart rate following application of high rate transcranial magnetic stimulation at the C3, C4, FZ, and PZ electrode sites. No significant differences in heart rate and blood pressure were noted among any of the sites stimulated (Claus et al., 1999; Foerster et al., 1997). However, as with ECT, the cardiovascular effects of high rate transcranial magnetic stimulation may be due to gross stimulation of cerebral tissue. Indeed, Foerster et al. (1997) stated that their results may have been due to an nonspecific arousal reaction, rather than to direct stimulation of autonomic cerebral areas.

Other investigations have more directly stimulated the cerebral areas thought to be involved in cardiovascular functioning. Research has indicated that stimulation of the medial prefrontal area elicits bradycardia and depressor responses (Cechetto & Saper, 1990; Verberne, Lam, Owens, & Sartor, 1997; van der Molen, 2000; Verberne & Owens, 1998). Zbrozyna and Westwood (1993) found that stimulation of the medial prefrontal area inhibits the tachycardia that occurs during the Defense-Aggression Reaction in rats. Likewise, al Maskati and Zbrozyna

(1989) found that electrical and chemical stimulation of the medial prefrontal area inhibited the cardiovascular and motor response of the defense reaction in rats. Additionally, electrical stimulation of the medial prefrontal area has inhibited conditioned increases in blood pressure in rats (Zbrozyna & Westwood, 1991). Other investigators have found that electrical stimulation of the ventral portion of the medial prefrontal area elicits depressor responses as well (Hardy & Holmes, 1988).

Reviews of the literature have indicated that stimulation of not only the medial prefrontal (Cechetto & Saper, 1990; Verberne & Owens, 1998) and frontal regions (Delgado, 1960; Hoff et al., 1963), but also stimulation of the temporal (Delgado, 1960; Hoff et al., 1963) and insular regions (Cechetto & Saper, 1990; Delgado, 1960; Neafsey, 1990; Oppenheimer, 1992; Verberne & Owens, 1998) results in changes in cardiovascular functioning. The right insula, in particular, is thought to be associated with sympathetic cardiovascular responding (Oppenheimer, 1993). Evidence supporting the role of the right insula in sympathetic responding is provided by Oppenheimer, Gelb, Girvin, and Hachinski (1992). They found that stimulation of the right insula in humans produced more frequent tachycardia and pressor responses than stimulation of the left insula. Conversely, stimulation of the left insula generated more frequent bradycardia and depressor responses. Investigations involving stimulation of the rat insular region have yielded similar findings. For instance, electrical stimulation of the rat insular region has generated significant changes in cardiovascular functioning as well as increased sympathetic nerve activity measured at the renal nerve (Cechetto & Chen, 1990). Changes in cardiovascular functioning resulting from electrical stimulation of the rat insular region were obtained by Yasui, Breder, Saper, and Cechetto (1991). Finally, Oppenheimer, Saleh, and Cechetto (1992) found that tachycardia resulted from stimulation of the rat insular region.

Specific Role of the Medial Prefrontal and Insular Cortices in Cardiovascular Regulation

The aforementioned investigations indicate that the medial prefrontal and insular regions are involved in the regulation of heart rate and blood pressure. However, each of these regions of the brain may have a different role in the regulation of these cardiovascular measures. As indicated earlier, stimulation of the right insular region results in increases in both heart rate and blood pressure (Oppenheimer et al., 1992). Thus, the insular region appears to have an excitatory effect on cardiovascular functioning (Verberne et al., 1997).

Conversely, the aforementioned research findings suggest an inhibitory role for the medial prefrontal region. Indeed, the frontal lobes, and the medial prefrontal area in particular, are known to be inhibitory in nature (Lane, Reiman, Ahern, & Thayer, 2000; Neafsey, 1990; Tucker & Derryberry, 1992; Verberne et al., 1997). The inhibitory nature of the medial prefrontal region in regulating cardiovascular functioning arises from the extensive connections that this region of the brain has with other areas of the cerebrum. The frontal lobes are known to have extensive connections with all other areas of the brain through numerous corticocortical and commissural pathways (Afifi & Bergman, 1998; Luria, 1980; Walsh & Darby, 1999). The medial prefrontal region, in particular, is known to have intimate interconnections with the insular regions (Benarroch, 1993). Furthermore, the medial prefrontal and insular regions both have reciprocal interconnections with the dorsomedial nucleus of the thalamus (Afifi & Bergman, 1998; Cummings, 1995; Niedermeyer, 1998; Nolte, 1999; Verberne & Owens, 1998; Walsh & Darby, 1999), which may then serve as a gate in passing messages from one area of the cortex to another (Sherman & Guillery, 2001). Thus, given the inhibitory nature of the medial prefrontal region and the intimate reciprocal connections with the insular regions, it may be inferred that the medial prefrontal region is inhibitory over heart rate and blood pressure.

Additionally, some researchers have suggested that the medial prefrontal region is inhibitory over cardiovascular functioning through its connections with the rostral ventrolateral medulla (Verberne & Owens, 1998). However, since the connections between the medial prefrontal and rostral ventrolateral medulla are sparse, these connections may not play a major role in inhibiting cardiovascular functioning (Verberne et al., 1997).

The neuroanatomical basis for the inhibitory role of the medial prefrontal region is consistent with the aforementioned research findings concerning the cardiovascular effects of stimulating this area of the brain. As stated earlier, the results of numerous investigations have indicated that stimulation of the medial prefrontal region results in either bradycardia and depressor responses (Hardy & Holmes, 1988) or the inhibition of conditioned increases in heart rate and blood pressure (al Maskati & Zbrozyna, 1989; Zbrozyna & Westwood, 1991; Zbrozyna & Westwood, 1993). Although the aforementioned research used rats as subjects, the role of the medial prefrontal region in inhibiting heart rate and blood pressure may be inferred from studies using human subjects. Specifically, clinical evidence is provided by research concerning the cardiovascular effects of tumors localized to the frontal lobes. As stated earlier, Rush et al. (1977) reported the case of a patient who exhibited recurrent episodes of atrial tachycardia and who was diagnosed with a right frontal lobe tumor. The recurrent episodes of atrial tachycardia would be expected in this case since the lesion within the right frontal lobe would preclude inhibition of the right insula. Essentially, the lesion disinhibited the right insular area, which is excitatory in nature, thereby permitting tachycardia to ensue. Further, it is worth noting that once the tumor was removed and the right frontal lobe returned to a more functional state, the episodes of atrial tachycardia ceased.

The inhibitory role of the frontal lobes in regulating cardiovascular functioning is further illustrated by the neuropsychophysiological model of hostility championed by Harrison and his colleagues. This model proposes that the right frontal lobe is responsible for inhibiting or regulating sympathetic cardiovascular functioning and hostility (Demaree & Harrison, 1997b). Consistent with this proposition, high hostile individuals, as compared to low hostile individuals, were found to possess decreased beta activity in the right frontal lobe (Demaree & Harrison, 1996; Everhart & Harrison, 1995) and increased right temporal activity (Demaree, 1995; Demaree & Harrison, 1997a; Everhart & Harrison, 1995). Based on these findings of decreased right frontal and increased right temporal activity, high hostile individuals should possess a relative disinhibition of sympathetic cardiovascular activity. Indeed, research has indicated that high hostile individuals not only have greater resting levels of heart rate (Demaree et al., 2000) but also have significantly greater increases in heart rate (Demaree, 1995; Demaree & Harrison, 1997a; Demaree et al., 2000) and systolic blood pressure (Demaree et al., 2000) to stress. Thus, the decreased right frontal lobe activity among high hostile individuals results in an inability to inhibit right temporal lobe activity, and hence increases in heart rate and blood pressure, particularly when exposed to stressors. The finding that decreased right frontal lobe activity among high hostile individuals leads to the disinhibition of right posterior systems, including the temporal lobe, and hence sympathetic cardiovascular activity is a result of the hemispheric lateralization of the sympathetic and parasympathetic nervous systems.

Hemispheric Lateralization of Sympathetic and Parasympathetic Nervous Systems

Herridge, Harrison, and Demaree (1997) found differences between low and high hostile men in habituation of skin conductance responses to affective facial configurations generated by the participants of the investigation. However, differences in skin conductance emerged only at

the left hand, thereby providing evidence for an asymmetrical cerebral representation of the sympathetic nervous system. Further evidence supporting the hemispheric lateralization of the sympathetic and parasympathetic nervous systems is provided by the results of an investigation that assessed the cardiovascular effects of localized unilateral electrical stimulation of the brain (Oppenheimer et al., 1992). More specifically, the investigation by Oppenheimer et al. (1992) found that whereas bradycardia and depressor responses were generated from stimulation of the left insula, stimulation of the right insula generated tachycardia and pressor responses. Numerous other investigations have been conducted assessing the lateralization of the sympathetic and parasympathetic nervous systems. The results of investigations using either lateralized visual stimuli or hemispheric inactivation have generally supported the results of the Oppenheimer et al. (1992) investigation.

Cardiovascular Effects of Lateralized Visual Stimuli

A series of investigations by Hugdahl, Franzon, Andersson, and Walldebo (1983) assessed the changes in heart rate resulting from the presentation of either slides with neutral content or emotionally arousing content using the visual half-field technique. The results indicated that whereas presentation of either the neutral or the emotional slides to the left hemisphere resulted in a significant reduction in heart rate, presentation to the right hemisphere resulted in a significant increase in heart rate. The findings of Hugdahl et al. (1983) were replicated and extended by Wittling and his colleagues, who found significant changes in systolic and diastolic blood pressure responding as well. For instance, Wittling (1990) found that presentation of a positive emotional film to the right hemisphere resulted in a significantly greater increase in systolic and diastolic blood pressure than presentation of the same film to the left hemisphere. However, the conclusions that may be drawn from the results of these

investigations regarding the lateralization of the sympathetic and parasympathetic nervous systems are limited since heart rate and blood pressure may not be ideal indicators of sympathetic and parasympathetic activity. More conclusive support for the hemispheric lateralization of the sympathetic and parasympathetic nervous systems is provided by the results of investigations measuring the effects of lateralized film presentations on ventricular myocardial contractility and heart rate variability, as these may be more ideal measures of sympathetic and parasympathetic activity, respectively. Specifically, in comparing the effects of presenting a negative film to the right versus the left hemisphere, Wittling, Block, Schweiger, and Genzel (1998) found that presentation to the right hemisphere resulted in significantly greater ventricular myocardial contractility. Additionally, using power spectral analysis to assess differences in heart rate variability between lateralized presentations of a negative film, Wittling, Block, Genzel, and Schweiger (1998) found that significantly greater high frequency power values resulted from presentation to the left hemisphere as compared to the right hemisphere.

Cardiovascular Effects of Hemispheric Inactivation

Power spectral analysis has been used in conjunction with intracarotid sodium amobarbital injections to assess and further support the hemispheric lateralization of the sympathetic and parasympathetic nervous systems. More specifically, research using power spectral analysis to assess the differences in heart rate variability resulting from intracarotid sodium amobarbital injections has indicated that inactivation of the left cerebral hemisphere results in a significant increase in the low frequency/high frequency ratio, indicating a shift toward increased sympathetic activity (Yoon, Morillo, Cechetto, & Hachinski, 1997). Conversely, inactivation of the right hemisphere has resulted in a decrease in the low frequency/high frequency ratio, suggesting a shift toward increased parasympathetic activity,

although this decrease failed to achieve statistical significance (Yoon et al., 1997). Further, the investigation by Yoon et al. (1997) found increases and decreases in heart rate following left and right hemisphere inactivation, respectively, although these differences in heart rate failed to achieve statistical significance. However, Hilz et al. (2001) found that inactivation of the right hemisphere generated a significant decrease in blood pressure and a significant increase in high frequency power of heart rate. Further, inactivation of the left hemisphere generated increases in heart rate and blood pressure and low frequency power of heart rate (Hilz et al., 2001). Other investigators have also found significant increases in heart rate following left hemisphere inactivation and significant decreases in heart rate following right hemisphere inactivation (Lane, Novelly, Cornell, Zeitlin, & Schwartz, 1988; Zamrini et al., 1990). Thus, taken together, the aforementioned research findings indicate that the sympathetic and parasympathetic control of heart rate and blood pressure is lateralized to the right and left cerebral hemispheres, respectively.

Relationship Between Magnitude of Cerebral Activation and Cardiovascular Functioning

Pool and Ransohoff (1949) stated that the variability in the magnitude of autonomic effects obtained from stimulation of the cingulate gyri, including changes in blood pressure, respiration rate, and heart rate, may be a reflection of the strength of the stimulus used. Further, Lacey and Lacey (1970) stated that increases in afferent cardiovascular feedback to the central nervous system will result in an increased rate of termination of autonomic activity. These two statements share a common presumption, namely, that a relationship exists between the magnitude of cerebral activation and the magnitude or degree of change in cardiovascular functioning. Indeed, support for the existence of such a relationship may be provided by the

results of research involving the cardiovascular and cerebral effects of increased task difficulty, experimentally induced pain, and white noise stimulation.

Cardiovascular and Cerebral Effects of Increasing Task Difficulty

Research has consistently indicated that increasing levels of task difficulty generate increasing changes in cardiovascular responding. For instance, Sherwood, Davis, Dolan, and Light (1992) conducted an investigation in which the participants were permitted to choose among performing either an easy or a difficult mental arithmetic task while measures of cardiovascular reactivity were assessed. Unbeknownst to the participants, all participants actually received the same arithmetic problems, regardless of choice. The findings indicated that choosing the difficult task generated significantly greater increases in myocardial contractility, cardiac output, and systolic blood pressure than choosing the easy arithmetic task. The results of a subsequent investigation found that both systolic blood pressure and heart rate increased as the choice of difficulty level for mental arithmetic tasks increased (Sherwood, Royal, & Light, 1993). Other researchers have assessed cardiovascular reactivity to mental arithmetic problems that actually do differ in level of difficulty. Consistent with previous findings, the results of these investigations found that the performance of difficult mental arithmetic tasks generates significantly greater increases in heart rate than the performance of easy arithmetic tasks (Carroll, Turner, & Hellawell, 1986; Carroll, Turner, & Prasad, 1986). Further, Harrison and Kelly (1989) found that increases in blood pressure and heart rate, secondary to heightened white noise levels, improved arithmetic performance.

The results of numerous other investigations using a variety of different tasks have supported a relationship between increasing task difficulty and increasing changes in heart rate and blood pressure. For instance, an investigation manipulating the level of difficulty of a

computer simulated flight program found that heart rate variability increased significantly more during the difficult sections of the flight than during the easy sections (Veltman & Gaillard, 1993). Additionally, research has found that significantly increased heart rate as well as systolic and diastolic blood pressure result from a more difficult simulated flight task, as compared to an easy flight task (Veltman & Gaillard, 1996). An investigation by Harrell and Clark (1985) measured cardiovascular responses to varying levels of difficulty on a rotary pursuit task in which the participants were instructed to use a stylus to maintain contact with a target disk while it rotated on a turntable. The results indicated that heart rate increased with increasing levels of difficulty on the pursuit task. Similar findings of a relationship between increased task difficulty and increased cardiovascular responding have been found in investigations manipulating the level of difficulty on a working memory task (Backs & Seljos, 1994) as well as a digit recognition task (Wright, Williams, & Dill, 1992). Both of these investigations found that increasing task difficulty resulted in increases in heart rate. Additionally, the investigation by Wright et al. (1992) found that systolic blood pressure was sensitive to the level of difficulty. Finally, an investigation by Fournier, Wilson, and Swain (1999) examined the cardiovascular effects of increasing levels of task difficulty on the Multi-Attribute Task Battery. This task battery consisted of a light detection task, gauge monitoring, visual tracking, and an auditory communication task. The results indicated that as the level of difficulty increased changes in heart rate increased.

The investigation by Fournier et al. (1999) examined the cerebral effects of increasing levels of task difficulty on the Multi-Attribute Task Battery. The results indicated that increasing levels of task difficulty resulted in increasing levels of cerebral activity, as measured by changes in cerebral electrical activity within the alpha (10 to 12 Hz) bandwidth range. Similarly, greater

levels of task difficulty on a visual probability matching task and an auditory memory task have generated greater reductions in alpha (7.5 to 13.5 Hz) spectral power within the parietal and occipital regions and greater increases in theta (3.5 to 7.5 Hz) spectral power in the left frontal regions (Gundel & Wilson, 1992). Furthermore, increasing levels of task difficulty using a visual discrimination task (Boiten, Sergeant, & Geuze, 1992) and a selective attention task (Van Winsum, Sergeant, & Geuze, 1984) have resulted in increases in the amount and duration of alpha event related desynchronization within the parietal and occipital lobes. Increasing the level of difficulty in a working memory task has been associated with increasing spectral power within the theta (4 to 7.5 Hz) bandwidth at the anterofrontal midline region as well as decreasing amplitude of alpha (7.5 to 14 Hz) at the parietocentral and occipitoparietal regions (Gevins, Smith, McEvoy, & Yu, 1997). Similar findings have been obtained in investigations using fMRI to assess the magnitude of cerebral activation. For instance, increasing working memory load generated increasing signal changes in fMRI within the dorsolateral prefrontal region (Manoach et al., 1997). Finally, increasing levels of difficulty for an attention to speed discrimination task have resulted in increasing cerebral activation as measured by fMRI within the right frontal and insular regions (Sunaert, Van Hecke, Marchal, & Orban, 2000).

Cardiovascular and Cerebral Effects of Experimentally Induced Pain

The experimental induction of pain has been shown to produce significant increases in heart rate and blood pressure (Hilgard, 1969; Moltner, Holzl, & Strian, 1990; Peckerman et al., 1991; Wolf & Hardy, 1941). An investigation by Wolf and Hardy (1941) measured systolic and diastolic blood pressure responses to pain induced through the cold pressor technique whereby the participants were instructed to immerse their left hand into a cold water bath. The results indicated that at the instant pain was experienced there was a concomitant sharp increase in

systolic and diastolic blood pressure. The increase in systolic and diastolic blood pressure reached maximum levels at the same time that the participants reported the pain had reached its maximum. Furthermore, the blood pressure responses to pain decreased with decrements in the level of pain. A positive correlation was found to exist between the intensity of the pain experienced and the increases in systolic and diastolic blood pressure. Other investigations have found significant positive correlations between the intensity of perceived pain resulting from the cold pressor technique and changes in systolic blood pressure (Hilgard, 1969) and mean arterial pressure (Peckerman et al., 1991). The investigation by Peckerman et al. (1991) compared high versus low reactors in the intensity of perceived pain, with high and low reactors being classified on the basis of peak increases in mean arterial pressure. The results indicated that those classified as high reactors reported significantly higher ratings of perceived pain than those classified as low reactors. Additionally, changes in heart rate were found to be sensitive to ratings of perceived pain. Specifically, research has indicated that increases in the perceived intensity of pain induced through phasic heat stimulation is associated with increasing changes in heart rate (Moltner et al., 1990).

Research has indicated that subjective pain experiences are positively correlated with the amplitude of the positive component of cerebral evoked responses within the central and parietal regions of the brain (Carmon, Dotan, & Sarne, 1978). A significant positive correlation has been found between the subjective intensity of pain and cerebral activation as measured by fMRI. Specifically, relative to mild levels of subjective pain, intense pain has been associated with greater fMRI signal intensity within the anterior cingulate cortex (Davis, Taylor, Crawley, Wood, & Mikulis, 1997). Other investigations have found a positive relationship between pain intensity and regional cerebral blood flow within the insular and medial prefrontal regions,

among other areas of the cerebrum (Coghill, Sang, Maisog, & Iadarola, 1999; Derbyshire et al., 1997).

Cardiovascular and Cerebral Effects of White Noise Stimulation

Presentation of white noise has often been used as a procedure for generating changes in cardiovascular functioning. For instance, Knott and Bulmer (1985) examined differences in heart rate responses resulting from presentation of 100 dB white noise between alcoholics and normal controls. The results indicated that the group of normal controls exhibited a significant increase in heart rate, which was greater than that for the group of alcoholics. Harrison and Kelly (1989) used white noise presented at 80 dB to investigate age differences in the effects of sensory stimulation on cardiovascular functioning. White noise was found to generate a significant increase in systolic blood pressure for both the younger (median age 19 years) and the older (median age 77 years) groups. Further, the increase in systolic blood pressure was found to persist throughout the testing for the older group but not for the younger group. Additionally, in comparing the cardiovascular effects of equally intense white noise and tone stimuli, Graham and Slaby (1973) found that broad band white noise (50 to 10,000 Hz) presented at 85 dB for a duration of 5 seconds generated a significantly greater increase in heart rate than a 1000 Hz sine-wave tone presented at the same intensity and duration. Further, although the cardiovascular responses to both types of stimuli habituated after 5 successive presentations, white noise continued to generate a greater increase in heart rate than the tone even on the final presentation.

The investigation by Graham and Slaby (1973) used white noise presentation to examine the cardiovascular effects of orienting and defense responses. Indeed, among investigations seeking to examine the autonomic consequences of orienting and defense responses, white noise has often been used as a method for generating changes in cardiovascular functioning (Raskin,

Hattle, Harris, & DeYoung, 1967; Raskin, Kotses, & Bever, 1969a; Raskin, Kotses, & Bever, 1969b; Turpin, Schaefer, & Boucsein, 1999; Uno & Grings, 1965). The typical methodological protocol employed in such investigations involves measuring cardiovascular responses to repeated presentations of white noise at varying levels of intensity. Thus, these investigations may not only serve to demonstrate the cardiovascular effects of white noise presentation but also the cardiovascular effects of stimulus intensity. For instance, Uno and Grings (1965) investigated the effects of presenting white noise of 60, 70, 80, 90, and 100 dB on heart rate, finger blood volume, and pulse volume. The results indicated that whereas heart rate was significantly affected by stimulus intensity, there was no consistent pattern of either acceleration or deceleration of heart rate. However, a near linear relationship was found to exist between the intensity of white noise and changes in finger blood pulse volume.

A series of investigations conducted by David Raskin and his colleagues examined the effects of varying levels of intensity of white noise on heart rate to investigate the cardiovascular indicators of orienting and defense responses (Raskin et al., 1967; Raskin et al., 1969a; Raskin et al., 1969b). The findings indicated not only that white noise induced changes in heart rate but also that a relationship existed between changes in heart rate and the intensity of white noise. For instance, using white noise intensities of 40, 60, 80, 100, and 120 dB, heart rate was reported to increase with increases in stimulus intensity (Raskin et al., 1967). A subsequent investigation using the same intensity levels of white noise found that 120 dB of white noise generated greater increases in heart rate than all other levels of intensity of white noise (Raskin et al., 1969a). Finally, following repeated presentations of white noise consisting of 80 and 120 dB, the 120 dB condition was found to generate a significantly greater increase in heart rate than the 80 dB condition for the initial presentations of white noise. The results of other investigations have

found a significant difference in the magnitude of change in heart rate resulting from 60 and 100 dB of white noise (Turpin et al., 1999) as well as 80, 90, and 100 dB of white noise (O'Gorman & Jamieson, 1977), with higher intensities generating greater increases in heart rate. Similar findings have also been found to exist between changes in systolic and diastolic blood pressure and intensity of white noise (Martinik & Opltova, 1986).

Presentation of white noise has also produced changes in cerebral functioning, as measured by electroencephalography. For instance, Schellenberg et al. (1989) examined differences in EEG power resulting from 85 dB white noise stimulation among schizophrenic patients and normal controls. The results indicated that the normal controls exhibited significant increases in beta 2 (21 to 40 Hz) power at the right parietal and frontal sites, using bipolar electrode placements consisting of F3/F4, F3/P3, and F4/P4 arrangements. Similarly, Giannitrapani (1970) found that presentation of white noise generated increased amplitude of beta (i.e. greater than 21 Hz) at the prefrontal, lateral frontal, frontal, and temporal electrode sites. Further, the amplitude of beta was higher at the right temporal site relative to the left temporal site. Other investigators have also found increases in beta power within the temporal lobes resulting from white noise presentation. Nicholls, Schier, Stough, and Box (1999) investigated cerebral asymmetry in temporal processing by having participants judge whether they detected a gap in a 300 millisecond burst of 70 dB white noise while EEG was being recorded. The results indicated a bilateral increase in beta (13 to 25 Hz) power at the temporal region, with higher beta power over the left temporal region as compared to the right temporal region. Presentation of 80 dB white noise has also generated increased beta (13 to 30 Hz) spectral power within the occipital region, although the increase failed to achieve statistical significance (San Martini, Venturini, Zapponi, & Loizzo, 1979).

A relationship between changes in EEG activity and intensity of white noise stimulation has been reported in the literature. Roth, Dorato, and Kopell (1984) investigated intensity and task effects on event related potentials using 65, 80, 95, and 110 dB white noise bursts. The participants were exposed to 50 millisecond bursts of each intensity level of white noise while performing a visual tracking task, sitting passively, or by reacting to the white noise burst by pressing a button. The results indicated that the amplitude of the P50 component of the event related potential recorded at the CZ electrode site increased with increasing stimulus intensity across all three experimental conditions.

Additional Supportive Evidence

Several recent investigations have sought to determine whether correlations exist between changes in cerebral activity and cardiovascular functioning. For instance, the results of an investigation by Waldstein et al. (2000) indicated that during recollection of angry memories, significant negative correlations were found between changes in alpha (8 to 13 Hz) power and heart rate reactivity at the left and right medial frontal lobes. Further support for the existence of a relationship between cerebral activity and cardiovascular functioning is provided by the results of an investigation by Kubota and colleagues, who found a significant negative correlation between changes in cardiac sympathetic index and theta (6 to 7 Hz) power at the FZ electrode site (Kubota et al., 2001). Other investigators found that, during a mental arithmetic task, significant coherence emerged between changes in gamma power and not only changes in heart rate at the bilateral frontal and right parietal but also changes in systolic blood pressure at the left frontal and right parietal regions (Umeno et al., 2003).

A recent investigation by Foster and Harrison (2004) used quantitative electroencephalography to assess more directly the relationship between magnitude of cerebral

activity and magnitude of changes in heart rate and blood pressure. More specifically, quantitative electroencephalographic recordings were taken from a group of right handed women while heart rate and blood pressure responses to recollection of angry memories were concomitantly recorded. The results indicated that the magnitude (μV) of alpha activity (8.0 to 13.0 Hz) was significantly negatively correlated with changes in heart rate and systolic and diastolic blood pressure within the right anterior temporal lobe and the right posterior region. Thus, as the magnitude of cerebral activity increased so too did changes in heart rate and blood pressure.

The findings of the Foster and Harrison (2004) investigation were replicated in a subsequent investigation using a group of right handed men (Foster & Harrison, 2002b). As with the previous study, quantitative electroencephalography as well as heart rate and systolic and diastolic blood pressure recordings were obtained in response to recollection of angry memories. The results indicated that changes in the magnitude (μV) of high beta (21.0 to 32.0 Hz) were positively correlated with changes in heart rate and systolic blood pressure at the right posterior temporal lobe. Changes in heart rate were positively correlated with changes in high beta magnitude at the left posterior temporal lobe, right prefrontal region, and left medial frontal lobe, as well as other sites across the cerebral cortex. Finally, changes in diastolic blood pressure were positively correlated with changes in high beta magnitude at the left prefrontal region. Further, a subsequent investigation found that changes in high beta magnitude were significantly correlated with changes in heart rate at the bilateral temporal, parietal, and frontal lobes, with changes in systolic blood pressure at the left frontal and temporal lobes, and with changes in diastolic blood pressure at the left frontal and right temporal lobes (Foster, Thompson, & Harrison, 2004b).

Integration of Findings

Based on an integration of the aforementioned research findings involving the cardiovascular and cerebral effects of increasing levels of task difficulty, subjective intensity of induced pain, and increasing intensity of white noise presentation, it may be inferred that increasing magnitude of cerebral activity generates increasing levels of change in heart rate and blood pressure. Additionally, this relationship between increasing magnitude of cerebral activity and increasing levels of change in heart rate and blood pressure would exist at those cerebral sites associated with cardiovascular functioning, namely, the frontal and temporal lobes, including the insular regions. However, given that the frontal area is largely inhibitory and that the temporal lobes are largely excitatory, the specific cardiovascular effects of increasing magnitude of cerebral activity will be dependent upon the relative level of cerebral activation between these regions. As mentioned previously, research has indicated that the sympathetic and parasympathetic nervous systems are lateralized to the right and the left hemispheres, respectively. Thus, the specific cardiovascular effects of increasing magnitude of cerebral activity within the frontal and temporal lobes will be dependent upon which hemisphere is associated with the higher level of cerebral activation. Altogether, the relative magnitude of cerebral activity between the left and right frontal and temporal lobes regions will determine the overall changes in the direction and in the magnitude of heart rate and blood pressure.

More specifically, since the right hemisphere has been associated with sympathetic control of cardiovascular functioning, it may be inferred that increasing magnitude of cerebral activity within the right temporal lobe would generate increasing levels of sympathetic control of cardiovascular functioning. Hence, increasing magnitude of cerebral activity within the right temporal lobe would result in increases in heart rate and blood pressure. Conversely, increasing

magnitude of cerebral activity within the left temporal lobe would generate increasing levels of parasympathetic control of cardiovascular functioning, and hence decreases in heart rate and blood pressure. However, since different regions of the brain act in concert (Kinsbourne, 1982), both hemispheres of the brain may be activated in the control of cardiovascular functioning. Indeed, as Wittling (1997) has stated, there exists a division of responsibility between the two cerebral hemispheres in the cerebral control of cardiac activity such that the two hemispheres act in concert to promote cardiovascular functioning. The concurrent activation of both cerebral hemispheres in the mediation of heart rate and blood pressure is consistent with the view of the two hemispheres existing in a reciprocally balanced relationship with each hemisphere opposing and complementing the other (Tucker, 1981). Hence, increases in heart rate and blood pressure may result from relatively greater magnitude of cerebral activation within the right temporal lobe, as compared to the left temporal lobe. Additionally, decreases in heart rate and blood pressure may result from relatively greater magnitude of cerebral activity within the left temporal lobe, as compared to the right temporal lobe. Thus, the temporal lobe with the higher magnitude of cerebral activation will determine the direction of change in heart rate and blood pressure. Further, the magnitude or degree of change in heart rate and blood pressure will be a function of the relative differences in the magnitude of cerebral activation among the two temporal lobes. Thus, the greater the difference in magnitude of cerebral activity between the two temporal lobes the greater the change in heart rate and blood pressure.

Support for the application of the positions that a division of responsibility exists between the two hemispheres (Wittling, 1997) and that the two hemispheres exist in a reciprocally balanced relationship (Tucker, 1981) to the cerebral mediation of cardiovascular functioning is provided by the results of an investigation by Foster and Harrison (2002b). Specifically, Foster

and Harrison (2002c) sought to test the hypothesis that increasing levels of right hemisphere activation, relative to left hemisphere activation, would be associated with increasingly greater baseline levels of heart rate and blood pressure. Conversely, it was also hypothesized that as magnitude of cerebral asymmetry shifts to heightened levels of left hemisphere activation lower baseline levels of heart rate and blood pressure would result. Given the inhibitory influence of the frontal lobes, the direction of the correlations between the frontal and temporal lobes was also expected to differ. To test these hypotheses a group of 29 men were asked to relax while baseline quantitative electroencephalographic measures were recorded at 19 electrode sites arranged according to the International 10/20 System, as well as measures of heart rate and blood pressure. Cerebral asymmetry scores were then calculated by subtracting the magnitude at each right hemisphere electrode from that at each homologous left hemisphere electrode site. The results indicated that cerebral asymmetry in low and high beta magnitude at the medial frontal lobes was negatively correlated with baseline diastolic blood pressure. Cerebral asymmetry in low and high beta magnitude at the occipital lobes was positively correlated with baseline systolic blood pressure. Finally, resting heart rate was negatively correlated with low beta asymmetry at the medial frontal lobes and positively correlated with low beta asymmetry at the posterior temporal and parietal lobes.

The present paper proposes that to explain the cerebral mediation of heart rate and blood pressure a functional cerebral systems perspective must be applied. As Luria (1980) has stated, any function of the brain is derived from within a "functional system" that is directed toward the performance of some particular task. Further, any system consists of a group of interconnected acts that operate in concert to promote a corresponding effect. A function is comprised of a group of highly differentiated and interchangeable elements that act as a complex and dynamic

system performing some adaptive task. Further, each discrete region or subsystem of the brain must interact to successfully complete any behavior or function. Thus, a functional cerebral systems perspective of the cerebral mediation of cardiovascular activities necessitates the inclusion of other regions of the brain that are involved in the mediation of heart rate and blood pressure.

The frontal lobes, particularly the medial prefrontal region, are inhibitory over the more posterior regions of the brain, including the temporal lobes. Thus, increasing magnitude of cerebral activity within the frontal regions will result in increasing levels of inhibition of the respective temporal lobes. Hence, depending upon which frontal region is associated with the greatest magnitude of cerebral activation there will exist a greater level of inhibition of either sympathetic or parasympathetic activity. For instance, increasing magnitude of cerebral activation within the right frontal region would result in increasing levels of inhibition of the right temporal lobe, and thus sympathetic control of heart rate and blood pressure. Conversely, decreasing magnitude of cerebral activity within the right frontal region will result in decreasing levels of inhibition of the right temporal lobe, thereby disinhibiting sympathetic control of heart rate and blood pressure. The same relationship between magnitude of cerebral activity within the frontal and temporal lobes applies to the left hemisphere, and thus parasympathetic activity. Thus, the direction of change in heart rate and blood pressure will be partly determined by the differences in magnitude of cerebral activity between the left and the right frontal regions, which will ultimately determine the differences in magnitude of cerebral activity between the left and the right temporal lobes. For instance, higher magnitude of cerebral activity within the left frontal region relative to the right frontal region will result in heightened inhibition of the left temporal lobe (parasympathetic activity) relative to the right temporal lobe (sympathetic

activity). This pattern of cerebral activity would then result in general increases in heart rate and blood pressure due to the greater level of inhibition of parasympathetic activity. Furthermore, the magnitude or degree of change in heart rate and blood pressure will be partly determined by the differences in magnitude of cerebral activity between the left and right frontal regions. Essentially, the greater the difference in cerebral activity between the frontal regions the greater the difference between the temporal lobes and hence the greater the change in heart rate and blood pressure.

Rationale

The purpose of the present investigation was to empirically test part of the proposed model of the cerebral mediation of heart rate and blood pressure (Foster, 2001b). Specifically, the present investigation sought to test the proposition that increasing magnitude of cerebral activity within the right temporal lobe generates increasing levels of sympathetic activation of heart rate and blood pressure. Additionally, the contribution of the right frontal lobe in inhibiting sympathetic activation of heart rate and blood pressure was examined. Given these intentions, a method for stimulating the right temporal lobe to varying degrees of intensity was proposed. Furthermore, a method for assessing the contribution of the right frontal lobe was proposed.

Figural Fluency: Assessment of Right Frontal Lobe Functioning

Fluency, as a neuropsychological construct, refers to the ability to employ one or more strategies that maximize response production while minimizing or avoiding response repetition (Ruff, Allen, Farrow, Niemann, & Wylie, 1994) under timed and limited search conditions (Demakis & Harrison, 1997). Tasks that involve measuring fluency have often been used as indicators of frontal lobe functioning since they are sensitive to frontal lobe impairment (Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001; Tucha, Smely, & Lange, 1999). Research has

indicated that right frontal lobe impairment, in particular, is associated with decreased performance on measures of figural fluency (Jones-Gotman & Milner, 1977; Ruff et al., 1994). More specifically, Jones-Gotman and Milner (1977) developed a task sensitive to anterior lesions of the non-dominant hemisphere. To meet this objective they developed a task, referred to as the Design Fluency task, comprised of two parts or conditions that each require the individual to draw as many unique designs as possible. The free condition component of the task required participants to draw as many unique designs as possible within four minutes, with the restriction that the designs could not represent actual objects or patterns that could be named. Simply drawing scribbles was also restricted. The fixed condition was the same as the free condition except that the participants were limited to drawings that contained only four lines. Jones-Gotman and Milner (1977) found that patients with right frontal and right fronto-central lesions were more impaired in their performance on both conditions than patients with either right temporal, right parietal, left parietal, or left frontal lesions.

Ruff and his colleagues also found that decreased performance on the Design Fluency task was associated with right frontal lobe impairment (Ruff et al., 1994). However, Ruff et al. (1994) pointed out several limitations to the Design Fluency task, including the fact that no normative data are available for this task and that the responses are not easily quantified or scored. Hence, Ruff and his colleagues developed a test of figural fluency with the intention of providing psychometric properties appropriate for clinical adaptation (Ruff, Light, & Evans, 1987). The result of this endeavor, known as the Ruff Figural Fluency Test (RFFT), has been found to be both reliable (Ruff et al., 1987) and sensitive to lesions resulting from head injuries with significantly impaired performance on the RFFT being associated with those with head injuries relative to controls (Ruff, Evans, & Marshall, 1986). An investigation by Ruff et al.

(1994) compared the performance of those with left and right posterior and anterior lesions on the RFFT. The results indicated that the patients with right frontal lobe pathology generated significantly fewer designs than those with left frontal, left posterior, or right posterior lesions. Foster, Beck, and Harrison (2003) found that men with heightened right frontal delta magnitude performed more poorly on the RFFT than men with lower right frontal delta magnitude. Additionally, Demakis and Harrison (1997) found that performance on the RFFT was positively correlated with performance on both conditions of the Design Fluency task. Other research has found that the right frontal lobe is critically involved in figural fluency, and specifically performance on the RFFT (Demakis, 1995). Thus, for purposes of the present investigation, relative impairments in right frontal lobe functioning would be evidenced by decreased performance on the RFFT.

White Noise: Graded Activation of the Right Temporal Lobe

Presentation of white noise has generated increased activation, as measured by EEG, within the occipital (San Martini et al., 1979), parietal (Schellenberg et al., 1989), and temporal (Giannitrapani, 1970; Nicholls et al., 1999) cortices. Additionally, research has indicated that a relationship exists between increasing intensity of white noise and changes in EEG activity in that the amplitude of the P50 component of the event related potential increased with increasing stimulus intensity of white noise (Roth et al., 1984). The results of other research investigations have not only found increased activation within the temporal cortices using a variety of auditory stimuli and measures of cerebral activation but have also supported the existence of a relationship between intensity of auditory stimulation and magnitude of cerebral activation.

Research has indicated that presentation of various auditory stimuli generates increased cerebral activity within the auditory cortex (Colder & Tanenbaum, 1999; Downar, Crawley,

Mikulis, & Davis, 2000; Frey, Kostopoulos, & Petrides, 2000; Jancke, Shah, Posse, Grosse-Ryuken, & Muller-Gartner, 1998) and in particular the superior temporal gyri (Hall et al., 2001; Millen, Haughton, & Yetkin, 1995; Mohr, King, Freeman, Briggs, & Leonard, 1999) and insular regions (Linden et al., 1999). The types of stimuli utilized in these investigations have included verbal stimuli (Colder & Tanenbaum, 1999; Jancke et al., 1998; Millen et al., 1995; Mohr et al., 1999), tones (Hall et al., 2001; Jancke et al., 1998; Linden et al., 1999; Millen et al., 1995), the sound of running water and croaking frogs (Downar et al., 2000), as well as the sound of violent car crashes (Frey et al., 2000).

Many of these studies have used functional magnetic resonance imagining (fMRI) to investigate the relationship between stimulus intensity and magnitude of cerebral activation. For instance, increasing the intensity of verbal stimuli from 65 to 110 dB has been found to generate increasing magnitude of cerebral activation within the bilateral superior temporal gyri or auditory cortices, with greater activation in the left than in the right auditory cortex (Mohr et al., 1999). Colder & Tanenbaum (1999) found that the intensity of fMRI measured at the auditory cortex increased with increasing stimulus intensity of verbal stimuli. Similarly, Hall et al. (2001) reported that the magnitude of cerebral activation within the temporal lobes increased in a linear fashion as the stimulus intensity of a 300 Hz tone increased from 65 to 85 dB.

Presentation of tones of varying intensity in conjunction with EEG has been used to investigate the relationship between increasing stimulus intensity and changes in cerebral activation. For instance, Adler and Adler (1989) used 1 kHz tones presented at a range of intensities from 30 dB to 90 dB to investigate this relationship. The results indicated that the amplitudes of the N100 and P175 components of the auditory evoked potential recorded at the FZ and CZ electrode sites increased with increasing stimulus intensity. Similarly, Reite,

Zimmerman, Edrich, and Zimmerman (1982), using 1 kHz tones presented at 40, 60, 80, and 100 dB, found that the mean auditory evoked potential increased in amplitude with increasing stimulus intensity at an electrode placed ¼ of the distance up from T4 to C4.

Although many of the aforementioned investigations used auditory stimuli other than white noise, the results of these investigations nonetheless support the findings of Roth et al. (1984) who used white noise in demonstrating the existence of a positive relationship between increasing stimulus intensity and changes in cerebral activation. Indeed, that auditory stimuli in general, and white noise in particular, have demonstrated such a relationship is not surprising given that audition is represented in the superior temporal gyri (Afifi & Bergman, 1998; Walsh & Darby, 1999). However, it should be mentioned that, even though a positive relationship has been found between increasing stimulus intensity and changes in cerebral activity, the ultimate nature of the relationship is likely curvilinear.

Integration of Findings

Based on an integration of these research findings, it is proposed that binaural presentation of graded levels of white noise should result in varying intensities of cerebral activation within the temporal lobes. Given that stimulation of the temporal lobes has been found to generate significant changes in heart rate and blood pressure, white noise presentation should result in such increases in cardiovascular functioning. Indeed, as mentioned previously, research has supported this proposition. Further, given the inhibitory role of the frontal lobes over the more posterior regions of the brain (Crawford et al., 1998; Crawford, Knebel, & Vendemia, 1998; Demaree & Harrison, 1996; Demaree et al., 2000; Lane et al., 2000; Neafsey, 1990; Tucker & Derryberry, 1992; Verberne et al., 1997), including the temporal lobes, the changes in cardiovascular functioning that result from graded white noise presentation should be

inhibited or disinhibited to the extent that the frontal lobes are functional or dysfunctional. Hence, given that the RFFT is sensitive to right frontal lobe functioning, performance on this measure should be reflected in the changes in heart rate and blood pressure that result from graded white noise presentation.

The changes in cerebral activity resulting from graded white noise presentation should be reflected in quantitative electroencephalography. As mentioned previously, the beta bandwidth has been used in investigations of the neurophysiological changes accompanying white noise presentation (Giannitrapani, 1970; Nicholls et al., 1999; San Martini et al., 1979; Schellenberg et al., 1989). Research has indicated, though, that functional differences exist between the low versus high beta bandwidths (Crawford, Clarke, & Kitner-Triolo, 1996). Given that white noise presentation has been found specifically to generate changes in the high beta bandwidth (Giannitrapani, 1970; Schellenberg et al., 1989), high beta (21 to 32 Hz) magnitude (μV) was measured in the present investigation. However, the low beta (13 to 21 Hz) magnitude was also included for the purposes of conducting exploratory analyses and gaining a greater understanding of the cerebral effects of white noise presentation.

Previous research has found significant changes in beta power (Nicholls et al., 1999) and amplitude (Giannitrapani, 1970) at the right anterior temporal lobe resulting from white noise presentation. Further, significant correlations between changes in high beta magnitude and cardiovascular activity have been found at the right anterior temporal lobe (Foster et al., 2004b). Research has also indicated orbitofrontal involvement in the processing of unpleasant auditory information (Frey et al., 2000). The orbitofrontal regions has also been implicated in the regulation of cardiovascular activity (Delgado, 1960; Neafsey, 1990). Using a 19 electrode site arrangement, the right lateral frontal lobe electrode site (F8) is the closest approximation to the

right orbitofrontal region. Hence, the present investigation measured changes in low and high beta magnitude at the right lateral frontal (F8) and the right anterior temporal (T4) lobes. Support for measuring low and high beta magnitude at these locations is also provided by the results from research conducted by Harrison and colleagues. Specifically, changes in cerebral activity at the right lateral frontal (F8) and anterior temporal lobe (T4) have been used to investigate right frontal lobe inhibition/disinhibition of the right posterior cerebral systems involved in the regulation of cardiovascular activity (Demaree & Harrison, 1996; Demaree et al., 2000).

Variables

Independent Variables

Group. The independent variable of Group was based on performance on the Ruff Figural Fluency Test and was comprised of two levels, Low Fluency and High Fluency.

Condition. The variable of Condition consisted of four levels, including the baseline or no white noise as well as the 55 dB, 75 dB, and 90 dB presentations of white noise (A scale reference of .002 Dynes/cm^2).

Electrode Site. The variable of Electrode Site consisted of two levels and was comprised of the right lateral frontal (F8) and right anterior temporal (T4) electrode sites, arranged according to the International 10/20 System.

Trial. The variable Trial consisted of two levels, including the pre-white noise and post-white noise administrations of the Ruff Figural Fluency Test. It should be mentioned that this variable only applied to analysis of performance on the Ruff Figural Fluency Test before and after white noise presentation and not to the analysis of changes in heart rate and blood pressure.

Dependent Variables

Design Fluency. Scores on the Ruff Figural Fluency Test, i.e. the total number of unique designs produced, were used as a measure of design fluency.

Cardiovascular. The cardiovascular variables included heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP).

Quantitative Electroencephalography. Quantitative electroencephalography (QEEG) was used to measure low beta (13 to 21 Hz) and high beta (21 to 32 Hz) magnitude (μV) at the aforementioned electrode sites.

Hypotheses

Design Fluency

Hypothesis 1: A Group x Condition x Trial interaction on RFFT scores was expected. Specifically, relative to the High Fluency group, the Low Fluency group was exhibited greater increases in performance on the RFFT from the first or pre-white noise administration to the second or post-white noise administration. The greatest increase in performance on the RFFT was predicted to occur following the 90 dB condition and the least increase in RFFT scores following the 55 dB condition. Further, performance on the RFFT was predicted to be greater for the High Fluency group relative to the Low Fluency group at both administrations of the RFFT.

Cardiovascular

Hypothesis 2: A Group x Condition interaction on heart rate and blood pressure was expected. Specifically, relative to the High Fluency group, the Low Fluency group was expected to exhibit greater increases in heart rate and blood pressure from the baseline or no white noise condition to the 90 dB, 75 dB, and the 55 dB white noise conditions, with the 90 dB condition

generating the greatest increase in heart rate and blood pressure, followed by the 75 dB condition, and the 55 dB condition generating the least increase. Differences in the amount of increase in heart rate and blood pressure between the High and Low Fluency groups were also expected, with the High and Low Fluency groups differing the most in the 90 dB condition, followed by the 75 dB condition, and differing the least in the 55 dB condition in terms of the amount of increase in heart rate and blood pressure.

Hypothesis 3: A Condition main effect for heart rate and blood pressure was expected such that the increases in heart rate and blood pressure would be greatest from the no white noise condition to the 90 dB white noise condition, followed by the 75 dB condition, with the 55 dB condition generating the least increase in heart rate and blood pressure.

Quantitative Electroencephalography

Hypothesis 4: A Group x Condition x Electrode Site interaction was expected for low and high beta magnitude. Specifically, relative to the High Fluency group, the Low Fluency group was expected to exhibit greater reductions in low and high beta magnitude from the no white condition to the 90 dB, 75 dB, and the 55 dB white noise conditions, with the 90 dB condition generating the greatest reduction in low and high beta magnitude, followed by the 75 dB condition, and the 55 dB condition generating the least reduction. Differences in the amount of reduction of low and high beta magnitude between the High and Low Fluency groups were also expected, with the High and Low Fluency groups differing the most in the 90 dB condition, followed by the 75 dB condition, and differing the least in the 55 dB condition in terms of the amount of reduction of low and high beta magnitude. Further, the reductions in low and high beta magnitude were expected to be greater at the right anterior temporal lobe site as compared to the right lateral frontal lobe site.

Hypothesis 5: A Group x Condition interaction for low and high beta magnitude was expected such that, relative to the High Fluency group, the Low Fluency group was expected to exhibit greater overall reductions in low and high beta magnitude from the no white noise condition to the three different white noise conditions, with the 90 dB condition generating the greatest reduction, followed by the 75 dB condition, and the 55 dB condition generating the least reduction. As before, differences in the amount of reduction of low and high beta magnitude between the High and Low Fluency groups were also expected, with the High and Low Fluency groups differing the most in the 90 dB condition, followed by the 75 dB condition, and differing the least in the 55 dB condition in terms of the amount of reductions in low and high beta magnitude.

Hypothesis 6: A Condition x Electrode Site interaction for low and high beta magnitude was expected such that, relative to the right lateral frontal lobe site, the right anterior temporal lobe site was expected to exhibit greater reductions in low and high beta magnitude from the no white noise condition to the three white noise conditions, with the 90 dB condition generating the greatest reduction, followed by the 75 dB condition, and the 55 dB condition generating the least reduction.

Hypothesis 7: A Condition main effect for low and high beta magnitude was expected such that the reduction in low and high beta magnitude were expected to be greatest from the no white noise condition to the 90 dB white noise condition, followed next by the 75 dB condition, with the 55 dB condition generating the least reduction in low and high beta magnitude.

Additionally, although no specific hypotheses were generated, quantitative electroencephalographic data were collected from not only the F8 and T4 electrode sites but all other left and right frontal (F1, F2, F3, F4, F7) and temporal (T3, T5, T6) lobe electrode sites as

well. The QEEG data collected from these additional electrode sites were included in additional analyses that served to elucidate further the changes in cerebral functioning following presentation of white noise. More specific reasons for these additional analyses will be discussed at the time the results from such analyses are presented.

Methods

Participants

A total of 45 right-handed men, with an age range of 18 to 29 years (M = 20.02, SD = 2.04), volunteered to participate in exchange for extra credit in their undergraduate psychology course, earning one point of extra credit for each session in which they participated. Handedness was assessed using the Coren, Porac, and Duncan (CPD) Laterality Questionnaire (Coren, Porac, & Duncan, 1979; see Appendix A). The participants were selected based on their scores from the CPD and their responses on a questionnaire assessing history of head injury and medical illnesses or problems (HIMHI; see Appendix B). These questionnaires were administered to all participants in an initial screening session, during which the RFFT was also administered. To be considered for inclusion the participants had to score a minimum of +5 on the CPD and identify both biological parents as being right-handed. The scores on the CPD for the entire sample of participants ranged from a minimum of +5 to a maximum of +13 (M = 9.36, SD = 2.75). Additional inclusion criteria included no reported history of significant head injury, no currently experienced psychological dysfunction or complaints, and no current prescription psychotropic or pain medication usage, as indicated by their responses on the HIMHI. Men were used exclusively due to the differences in lateralized brain functions that exist between men and women, particularly with respect to the cerebral mediation of cardiovascular functioning (Ahern et al., 2001; Wittling, 1995). It should be mentioned that of the 45 participants, the data for one

participant had to be discarded due to equipment malfunction. Hence, the data for that participant were not used for any statistical analysis.

Apparatus

Coren, Porac, and Duncan Laterality Questionnaire. The CPD (Coren, Porac, & Duncan, 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 are from –13 to +13.

Head Injury/Medical History Inventory. The HIMHI is a self-report questionnaire consisting of 15 forced choice questions to which the participants respond either "yes" or "no". The HIMHI assesses history of head injury and neurological dysfunction through questions concerning whether the respondent has experienced severe head trauma, stroke, seizures, paralysis, or other neurological problems or surgeries. Additional questions address psychological or psychiatric problems, medical conditions or illnesses, visual or hearing impairments, alcohol and illicit drug use, prescription medications, as well as problems or pain related to movement of limbs.

Ruff Figural Fluency Test. The Ruff Figural Fluency Test (RFFT) was administered to assess design fluency. The RFFT (Ruff et al., 1987) is a measure of nonverbal fluency consisting of five individual parts, with each part consisting of a different stimulus pattern. More specifically, each of the five parts contains a 5 x 7 array of 35 unique stimulus items, with each stimulus item being comprised of a 5-dot matrix. Additionally, three sample stimulus items for the participant to complete to gain practice preceded each part of the test. The participants were instructed to draw as many unique designs as possible by connecting two or more of the dots within each of the matrices within a time limit of one-minute. The total number of unique

designs produced and the number of perseverative errors (repetitions of the same design within a single test sheet or part) were calculated. The accuracy of scoring was ensured through systematically comparing each design produced against the remaining productions. Test-retest reliability for the RFFT is approximately .76 (Ruff et al., 1987).

White Noise. White noise was presented binaurally using a Lafayette Instruments Model 15800 White Noise Generator (Lafayette Instrument Co., Lafayette, IN). The decibel (dB) level of white noise was set and checked using a Model dB-307 Metrologger Noise Dosimeter (Metrosonics, Inc., Rochester, NY).

Cardiovascular. Heart rate (HR) measured in beats per minute, systolic blood pressure (SBP), and diastolic blood pressure (DBP), both measured in mmHg, 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 3mmHg/sec. The manufacturer provides accuracy specifications of ±3 mmHg 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 & 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, Gorelczenko, & Kelly, 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 2.5cm above the antecubital space. The cuff was removed and repositioned over the brachial artery in the event of an error reading.

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).

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.

Procedure

The initial phase of the experiment consisted of a screening session in which all participants were first greeted and then asked to sign an Informed Consent Form (see Appendix C). The participants were then provided with a brief description of the experiment, as well as the experimental protocol for the initial screening session and informed that some of them may be asked to return for a second phase of the experiment. Following the description of the experiment the CPD, HIMHI, and the RFFT were administered. The 45 participants that met the aforementioned inclusion criteria were then used to construct two groups of 15 participants each.

Group assignment was based on their performance on the RFFT, specifically, the total number of unique designs produced. Participants scoring in the lower one-third of the total sample were assigned to the Low Fluency group and those scoring in the upper one-third of the total sample were assigned to the High Fluency group. All statistical analyses were then conducted using the Low and High Fluency groups. Following the initial screening session, the 45 participants that met the inclusion criteria were invited to return for a second phase of the experiment, during which cardiovascular and quantitative electroencephalographic responses to three different intensity levels of white noise were measured.

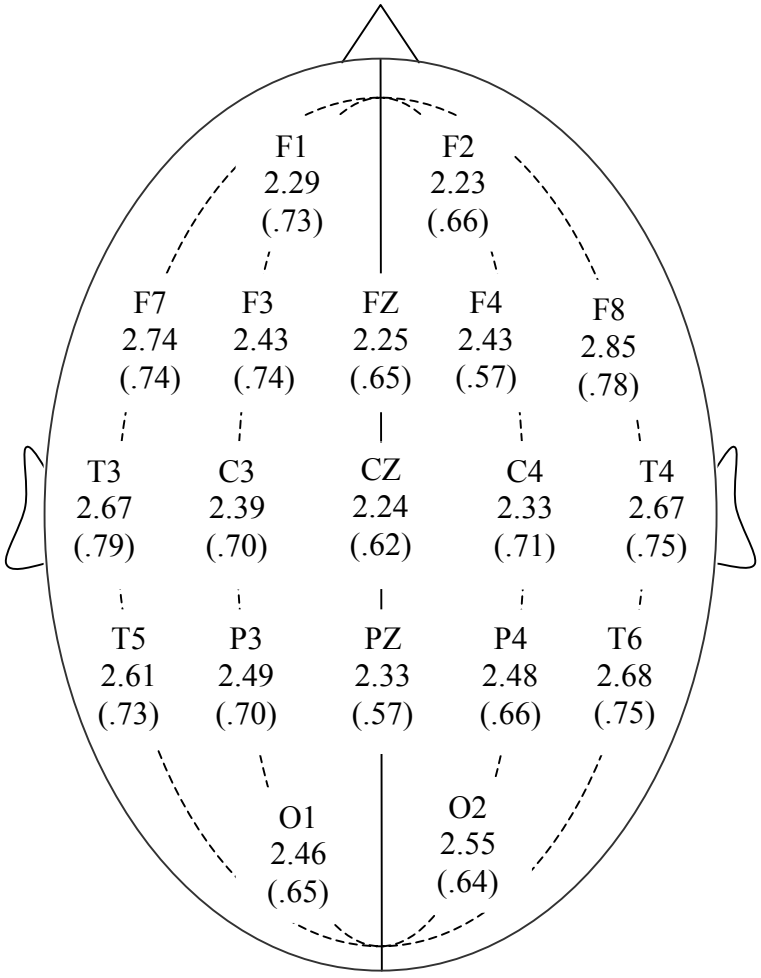
Those participating in the second phase of the experiment were invited to the neuropsychological laboratory and provided with an opportunity to ask questions regarding the experiment. Subsequent to answering any questions, the participants were fitted for the lycra electrode cap, which was attached to the participant's scalp according to the appropriate anatomical landmarks. To ensure a securely positioned electrode cap, therefore minimizing artifacts related to electrode movement, the cap was attached with elastic straps connected to a body harness placed around the participant's chest. Each electrode site involved in measuring QEEG was prepped with NuPrep prior to injecting electrode gel to minimize resistance between the electrode and the scalp, thereby reducing artifact related to high impedance levels. The electrodes for measuring EOG activity over the left and right eyes were then attached. More specifically, a bipolar arrangement of electrodes was used for each eye, with one electrode placed about 2cm above the supraorbital margin and the other electrode placed over the cheekbone. The electrode sites used to measure EOG activity were prepped with an alcohol pad to minimize resistance between the electrode and the surface of the skin. Once the electrode cap and the electrodes used to measure EOG activity were attached, the impedance levels for all

electrodes used to measure QEEG were checked. Impedance levels for all electrodes were below 5kΩ, and in most instances below 3kΩ. The average interelectrode impedance level between any two electrodes did not exceed 700Ω. Consult Figure 1 for the mean impedance levels and standard deviations for each electrode site.

Once the impedance levels were checked, the participants were seated in the middle of a Controlled Acoustical Environments (Industrial Acoustics Company, Inc., Bronx, NY) sound attenuated chamber, with interior dimensions of 48" x 40" x 78". The blood pressure cuff of the digital blood pressure monitor was then attached to the participant's left arm. The participants were then instructed to sit quietly, to relax, to keep their eyes closed, and to remain as still as possible throughout the remainder of the procedures. They were then informed that in a few minutes they would hear white noise presented through a speaker located just above and behind their head. Additionally, they were informed that three different intensity levels of white noise would be presented. The importance of remaining as still as possible and keeping their eyes closed was then reviewed in an effort to minimize such actions and the artifacts associated with them. They were then permitted another opportunity to ask any questions regarding this phase of the investigation.

Baseline measurements of heart rate, systolic and diastolic blood pressure, and QEEG activity were obtained following the aforementioned instructions. The baseline measurements for cardiovascular functioning and QEEG activity were noncontemporaneous, occurring at different times in the beginning of the second phase of the investigation. Further, all baseline measurements were obtained in the absence of white noise, with the sound attenuated chamber having a 45 dB level (A scale reference .002 Dynes/cm²). The establishment of baseline cardiovascular functioning occurred in two stages. Initial baseline measurements of heart rate

Figure 1. Average impedance levels for each electrode site.



Note. Average impedance level for each site is presented under the site identifier with standard deviation in parentheses. The average impedances for the reference and ground sites were 2.36 (.77) and 2.43 (.73), respectively.

and blood pressure were obtained 3 minutes following the initiation of relaxation. 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. The QEEG baseline measurement was obtained during the minute prior to the first cardiovascular baseline measurement. A total of 45 one-second epochs constituted the QEEG baseline measurement. 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. Electro-oculographic measurements were obtained during the recording of QEEG activity.

Following completion of the baseline cardiovascular and QEEG measurements, the participants were given an additional 2 minutes to relax before being exposed to the experimental manipulation, i.e. white noise presentation. All participants were exposed to white noise of three different intensity levels, 55 dB, 75 dB, and 90 dB (A scale reference .002 Dynes/cm^2). The order of presentation of white noise was randomized to prevent contamination from potential carry-over effects. Given that the decibel level of white noise was manually manipulated, measurements of the accuracy in setting the decibel levels were obtained to ensure that the participants were exposed to approximately the same level of intensity of white noise. These measurements were not obtained in the presence of the participants. Measurements of accuracy were obtained by randomly setting each of the three different decibel levels of white noise over 10 trials. The results of these analyses indicated that the decibel level for all three white noise intensities did not differ by ± .8 decibels, with no single intensity setting exceeding .8 dB above or .3 dB below the desired intensity level. White noise was presented through a speaker mounted

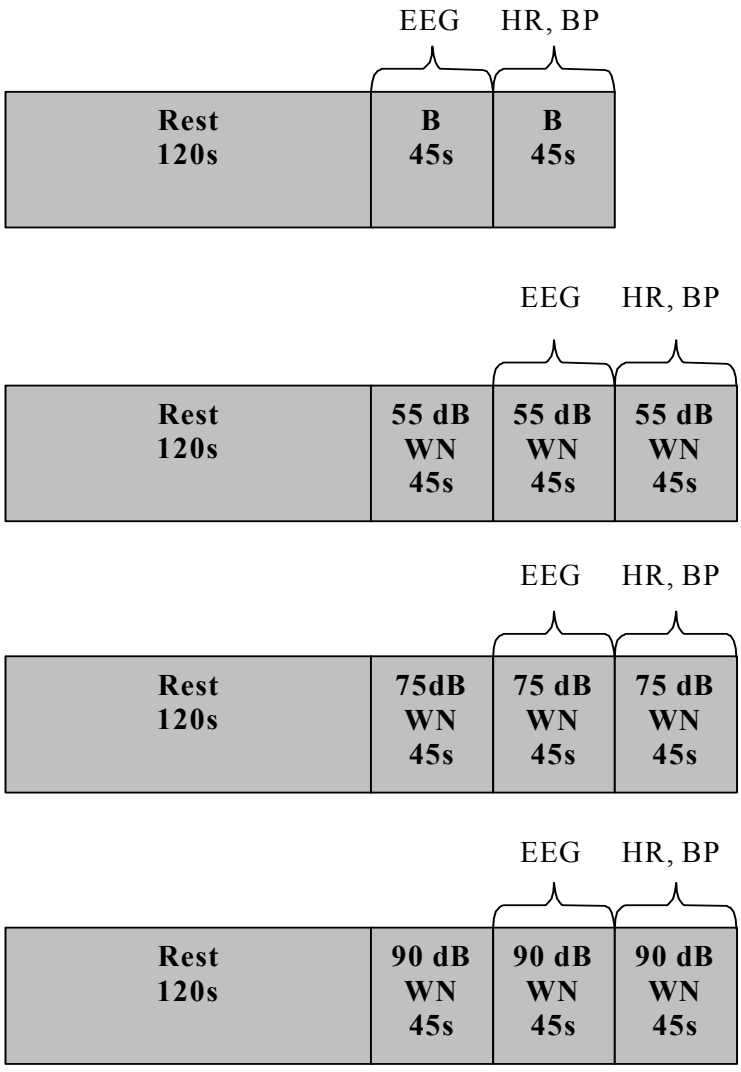
on a shelf located approximately 12" above and 6" behind the participant's head. Consistent positioning of the participant's head at the desired location, relative to the speaker, ensured minimization of field effects. The participants were initially exposed to each presentation of white noise for 45 seconds, after which QEEG activity was recorded. As with the baseline condition, a total of 45 one-second QEEG epochs were obtained for each of the three white noise presentations. Further, EOG activity was recorded during the measurement of QEEG activity. Following the measurement of QEEG, heart rate and blood pressure responses to each presentation of white noise were obtained. Quantitative electroencephalographic and cardiovascular measurements were obtained while the participants were exposed to the white noise. Hence, altogether, the participants were exposed to each intensity level of white noise for approximately 135 seconds. Approximately two minutes separated each presentation of white noise. Figure 2 presents a schematic diagram of the procedures. Subsequent to the final presentation of white noise and the recording of QEEG and cardiovascular measures, the participants exited the sound attenuated chamber and all electrodes as well as the electrode cap were removed. Immediately following the removal of the electrode cap and electrodes, which required approximately two to three minutes, the participants were seated at a table and asked to complete the RFFT for a second time. The participants were then thanked for their participation and dismissed.

Results

Data Reduction

Quantitative Electroencephalography. Prior to conducting statistical analyses, each one-second QEEG epoch resulting from the baseline and the experimental conditions was individually reviewed for the purpose of removing the one-second epochs suspected of

Figure 2. Schematic diagram of methods used.



containing artifacts resulting from muscle movements or other contaminants. Specifically, artifacting of the epochs involved deleting any one-second epoch noted to contain QEEG activity whose magnitude exceeded ±50μV 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.

Difference scores were also calculated for some analyses by subtracting the magnitude (μV) value resulting from the baseline or no white noise condition from the magnitude (μV) value resulting from each of the three white noise conditions. These difference scores were calculated for each of the electrodes used in the analyses.

Analyses

Group Descriptive Statistics

As mentioned previously, the data for one participant was discarded and not used for the statistical analyses. Since this participant would have been assigned to the Low Fluency group given his performance on the RFFT, the data for this participant was replaced by substituting this participant with the participant who scored the next lowest on the RFFT. The ages of the participants in the Low Fluency group ranged from 18 to 29 years (M = 19.93, SD = 2.71), with the High Fluency group ranging in age from 18 to 24 years (M = 20.20, SD = 1.78). Additionally, scores on the CPD ranged from 5 to 13 for the Low Fluency group (M = 9.07, SD = 2.94) and from 5 to 13 for the High Fluency group (M = 8.73, SD = 2.79). A one-way between groups ANOVA performed on the CPD scores, to ensure that the groups did not differ in degree of laterality or right-handedness, indicated that the Low and High Fluency groups were statistically equivalent in their scores on the CPD, $F(1, 27) = .102$, $p = .75$.

Design Fluency

Group Differences. Scores on the RFFT for the Low Fluency group ranged from 47 to 86 (M = 69.93, SD = 10.57), with the High Fluency group RFFT scores ranging from 103 to 133 (M = 115.40, SD = 10.35). A one-way between groups ANOVA performed on the RFFT scores, to ensure that the Low and High Fluency groups differed significantly in terms of their performance on the RFFT, indicated that the RFFT scores for the Low Fluency group were significantly less than that for the High Fluency group, $F(1,27) = 141.66$, $p = .0001$.

Pre and Post White Noise Administrations. To analyze the effects of white noise on performance on the RFFT, 6 different groups were created based on RFFT performance and the last white noise intensity condition to which they were exposed. Hence, the six groups were comprised of the following; Low Fluency-55 dB, Low Fluency-75 dB, Low Fluency-90 dB, High Fluency-55 dB, High Fluency-75 dB, and High Fluency-90 dB. The results of a 2 (Group) x 3 (Condition) x 2 (Trial) mixed factorial ANOVA, with the between subjects factors of Group and Condition and the repeated factor of Trial, indicated significant main effects for Group, Condition, and Trial, $F(1, 24) = 108.37$, $p = .0001$ and $F(2, 24) = 6.87$, $p = .004$, and $F(1,24) = 154.04$, $p = .0001$, respectively. Thus, the overall performance of the Low Fluency group (M = 80.97, SD = 12.24) was significantly lower than that of the High Fluency group (M = 127.24, SD = 13.20). Additionally, overall performance on the RFFT was found to vary as a function of Condition, with participants exposed to the 55 dB condition as the last white noise condition scoring the lowest (M = 100.29, SD = 17.41), participants exposed to the 90 dB condition scoring in the middle (M = 106.57, SD = 17.87), and participants exposed to the 75 dB condition as the final white noise condition scoring the highest (M = 108.75, SD = 16.57). Multiple comparisons using Tukey's HSD test indicated that all possible comparisons were

statistically significant with the exception of the comparison between the 55 dB and 90 dB conditions (see Table 1). Scores on the RFFT were found to significantly increase from the first administration (M = 92.67, SD = 10.46) to the second or post white noise administration (M = 115.53, SD = 14.97), irrespective of group assignment. All interaction effects were nonsignificant. Consult Table 2 for the ANOVA source table for RFFT performance.

Cardiovascular

Heart Rate. The results of a 2 (Group) x 4 (Condition) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factor of Condition, indicated that all main and interaction effects were nonsignificant. Table 3 presents the ANOVA source table for heart rate.

Systolic Blood Pressure. The results of a 2 (Group) x 4 (Condition) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factor of Condition, indicated a significant main effect for Condition, $F(3, 84) = 3.00$, $p = .04$ (see Figure 3). However, multiple comparisons using Tukey's HSD indicated that no two conditions were significantly different in terms of heart rate. Due to this discrepancy, a subsequent, more refined, repeated measures AVOVA was conducted on systolic blood pressure, using Condition as a repeated factor. The results indicated a nonsignificant effect of Condition on systolic blood pressure, $F(3, 116) = .36$, $p = .78$. No other main or interaction effects were found to be statistically significant. Table 4 presents the ANOVA source table for systolic blood pressure.

Diastolic Blood Pressure. The results of a 2 (Group) x 4 (Condition) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factor of Condition, indicated that all main and interaction effects were nonsignificant. Table 5 presents the ANOVA source table for diastolic blood pressure.

Table 1

Means and standard deviations for performance on the RFFT as a function of Condition.

Condition	Mean	Standard Deviation
55 dB White Noise	100.29 a	17.41
75 dB White Noise	108.75	16.57
90 dB White Noise	106.57 a	17.87

Note. Means with the same letter are not significantly different. Multiple comparisons based on Tukey's HSD test.

Table 2

ANOVA source table for RFFT scores.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 24	32109.07	32109.07	108.37	.0001
Condition	2, 24	4072.62	2036.31	6.87	.0044
Trial	1, 24	7843.27	7843.27	154.04	.0001
Group x Condition	2, 24	0.00	0.00	0.00	1.00
Group x Trial	1, 24	9.60	9.60	0.19	.67
Condition x Trial	2, 24	189.39	94.69	1.86	.18
Group x Condition x Trial	2, 24	77.74	38.87	0.76	.48

Table 3

ANOVA source table for heart rate.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	242.25	242.25	0.39	.54
Condition	3, 84	222.76	74.25	1.95	.13
Group x Condition	3, 84	4.42	1.47	0.04	.99

Figure 3. Systolic blood pressure (mmHg) as a function of Condition.

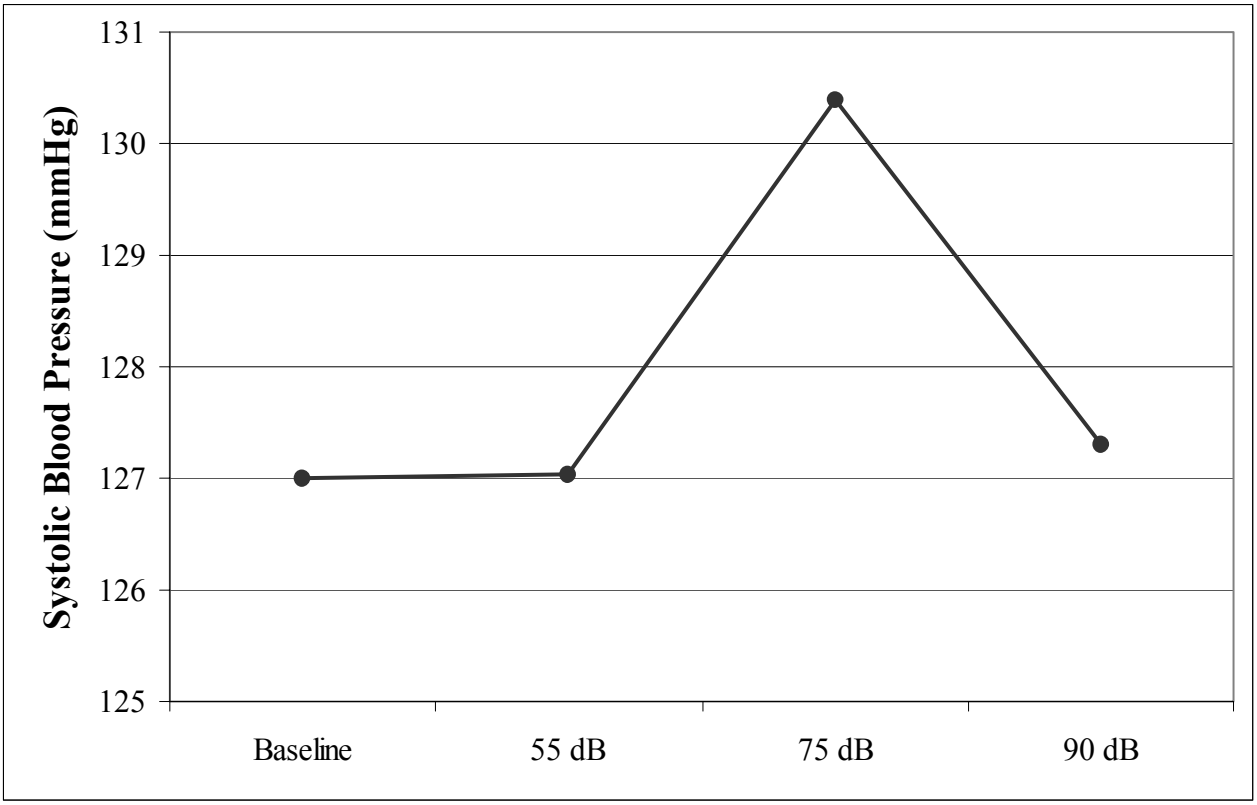


Table 4

ANOVA source table for systolic blood pressure.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	653.33	653.33	0.77	.39
Condition	3, 84	245.00	81.67	3.00	.04
Group x Condition	3, 84	63.00	21.00	0.77	.51

Table 5

ANOVA source table for diastolic blood pressure.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	583.00	583.00	0.51	.48
Condition	3, 84	629.27	209.76	1.40	.25
Group x Condition	3, 84	619.94	206.65	1.38	.25

Quantitative Electroencephalography

Low Beta. The results of a 2 (Group) x 4 (Condition) x 2 (Electrode Site) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factors of Condition and Electrode Site, indicated a significant Group x Electrode Site interaction, $F(1, 28) = 4.48$, $p = .04$ (see Table 6). Multiple comparisons using Tukey's HSD indicated that low beta magnitude at the right anterior temporal lobe electrode site for the High Fluency group ($M = 6.30$, $SD = 2.57$) was significantly lower than for the Low Fluency group ($M = 7.06$, $SD = 2.62$). Additionally, the main effect of Electrode Site was statistically significant, $F(1, 28) = 5.11$, $p = .03$ (see Table 7). Multiple comparisons using Tukey's HSD indicated that the right anterior temporal lobe electrode site ($M = 6.68$, $SD = 2.60$) evidenced heightened low beta magnitude as compared to the right lateral frontal lobe electrode site ($M = 6.08$, $SD = 2.05$). No other main or interaction effects were noted to be statistically significant (see Table 8 for the ANOVA source table for low beta magnitude at the right lateral frontal and anterior temporal lobes). However, the Condition x Electrode Site interaction was noted to closely approach the level of statistical significance, $F(3, 84) = 2.64$, $p = .06$.

To understand more completely the effects of white noise presentation on frontal and temporal lobe activity a more broad analysis of the data was performed using all electrodes sites across the frontal and temporal lobes. The results of a 2 (Group) x 4 (Condition) x 10 (Electrode Site) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factors of Condition and Electrode Site, indicated a significant Condition x Electrode Site interaction, $F(27, 756) = 2.18$, $p = .0005$. Multiple comparisons using Tukey's HSD indicated that the 55 dB condition resulted in a significant reduction in low beta magnitude at the right

Table 6

Means and standard deviations for low beta magnitude as a function of Group and Electrode Site.

Group	Electrode Site	Mean	Standard Deviation
Low Fluency	F8	5.90	1.51
Low Fluency	T4	7.06 *	2.62
High Fluency	F8	6.26	2.58
High Fluency	T4	6.30 *	2.57

Note. Multiple comparisons based on Tukey's HSD test. * indicates a significant difference at the .05 level of significance. Values are reported in μV.

Table 7

Means and standard deviations for low beta magnitude as a function of Electrode Site.

Electrode Site	Mean	Standard Deviation
F8	6.08	2.05
T4	6.68	2.60

Note. Multiple comparisons based on Tukey's HSD test. * indicates a significant difference at the .05 level of significance. Values are reported in μV.

Table 8

ANOVA source table for low beta magnitude at the right lateral frontal and anterior temporal lobes.

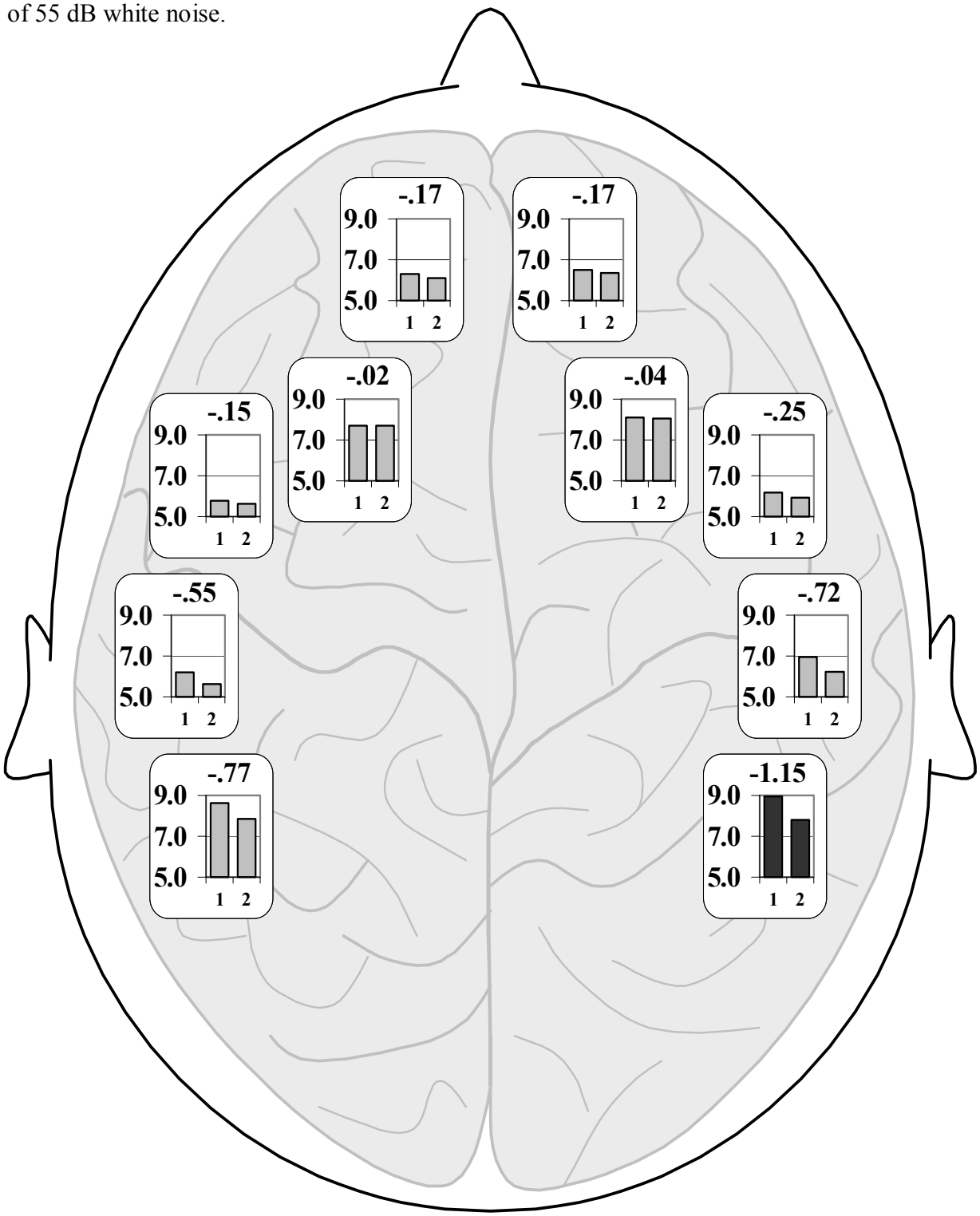
Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	2.28	2.28	0.06	.81
Condition	3, 84	8.16	2.72	1.99	.12
Electrode Site	1, 28	21.36	21.36	5.11	.03
Group x Condition	3, 84	0.97	0.32	0.24	.87
Group x Electrode Site	1, 28	18.70	18.70	4.48	.03
Condition x Electrode Site	3, 84	2.83	0.94	2.64	.06
Group x Condition x Electrode Site	3, 84	0.94	0.31	0.88	.46

posterior temporal lobe (T6, Baseline $M = 8.96$, $SD = 3.14$; 55 dB White Noise $M = 7.81$, $SD = 2.56$; see Figure 4). Significant changes in low beta magnitude were not noted at any other electrode site or for any of the other conditions (see Figures 5 and 6). Additionally, the Electrode Site main effect was found to be statistically significant, $F(9, 252) = 23.89$, $p = .0001$ (see Table 9). No other main or interaction effects were noted to be statistically significant (see Table 10 for the ANOVA source table for low beta magnitude at all electrodes sites within the bilateral frontal and temporal lobe).

Additional analyses were performed on the difference scores obtained by subtracting the baseline low beta μV from the low beta μV resulting from presentation of each intensity level of white noise. The purpose for these additional analyses was threefold. First, these analyses aided in determining whether significant differences existed between homologous comparisons of electrode sites for any given condition in terms of changes in low beta μV from the baseline to the 55 dB, 75 dB, and 90 dB white noise conditions. Further, these analyses were necessary to determine whether significant differences existed between the frontal and temporal lobe electrode sites in terms of changes in low beta μV. Finally, questions regarding whether significant differences existed in terms of changes in low beta μV for any given electrode across the three conditions were answered.

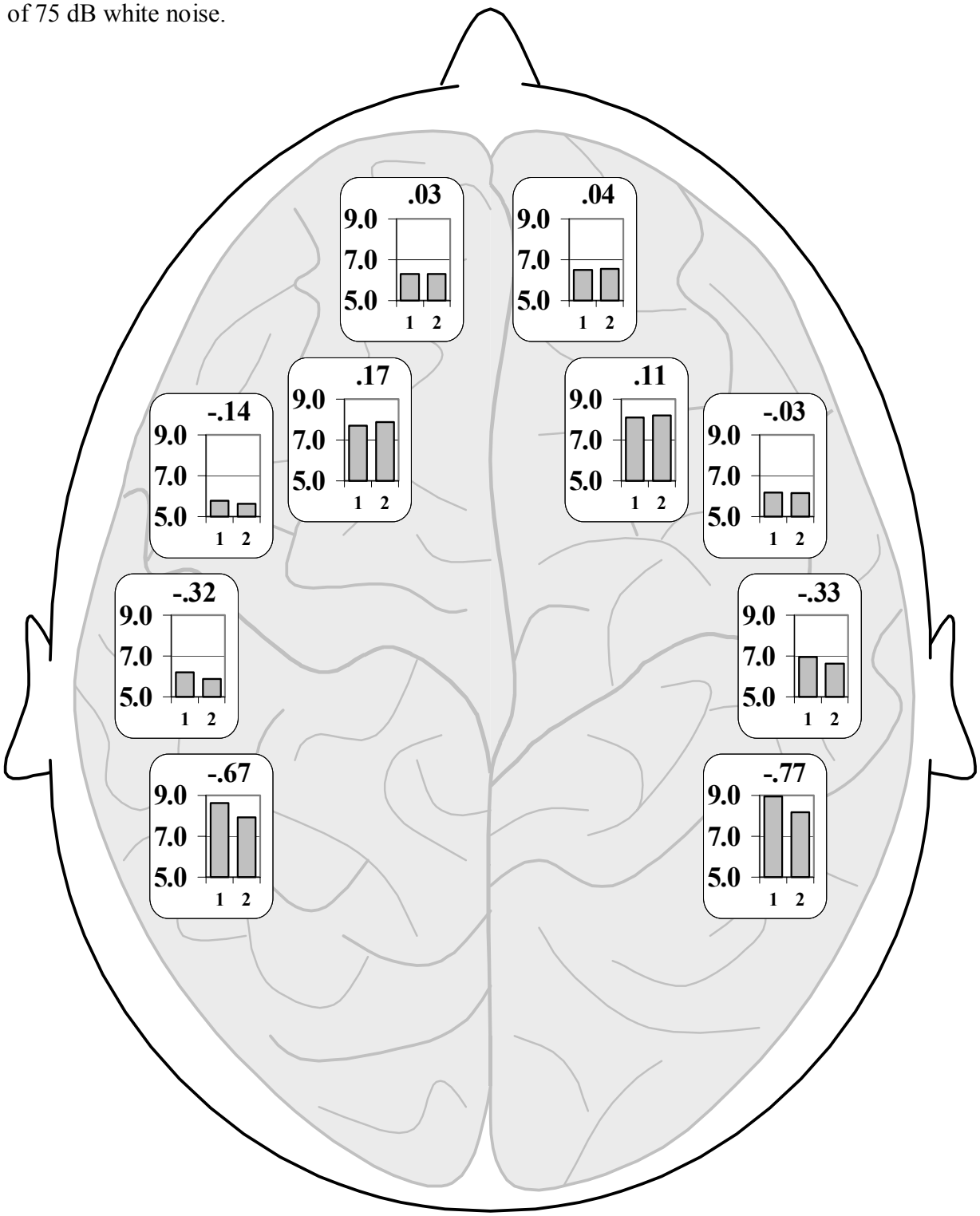
The results of a 2 (Group) x 3 (Condition) x 10 (Electrode Site) mixed factorial ANOVA on the low beta difference scores, with the between subjects factor of Group and the repeated factors of Condition and Electrode Site, indicated a significant Condition x Electrode Site interaction, $F(18, 504) = 1.83$, $p = .02$ (see Table 11). Multiple comparisons using Tukey's HSD indicated that no two homologous electrode sites differed significantly in terms of changes in

Figure 4. Low beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 55 dB white noise.



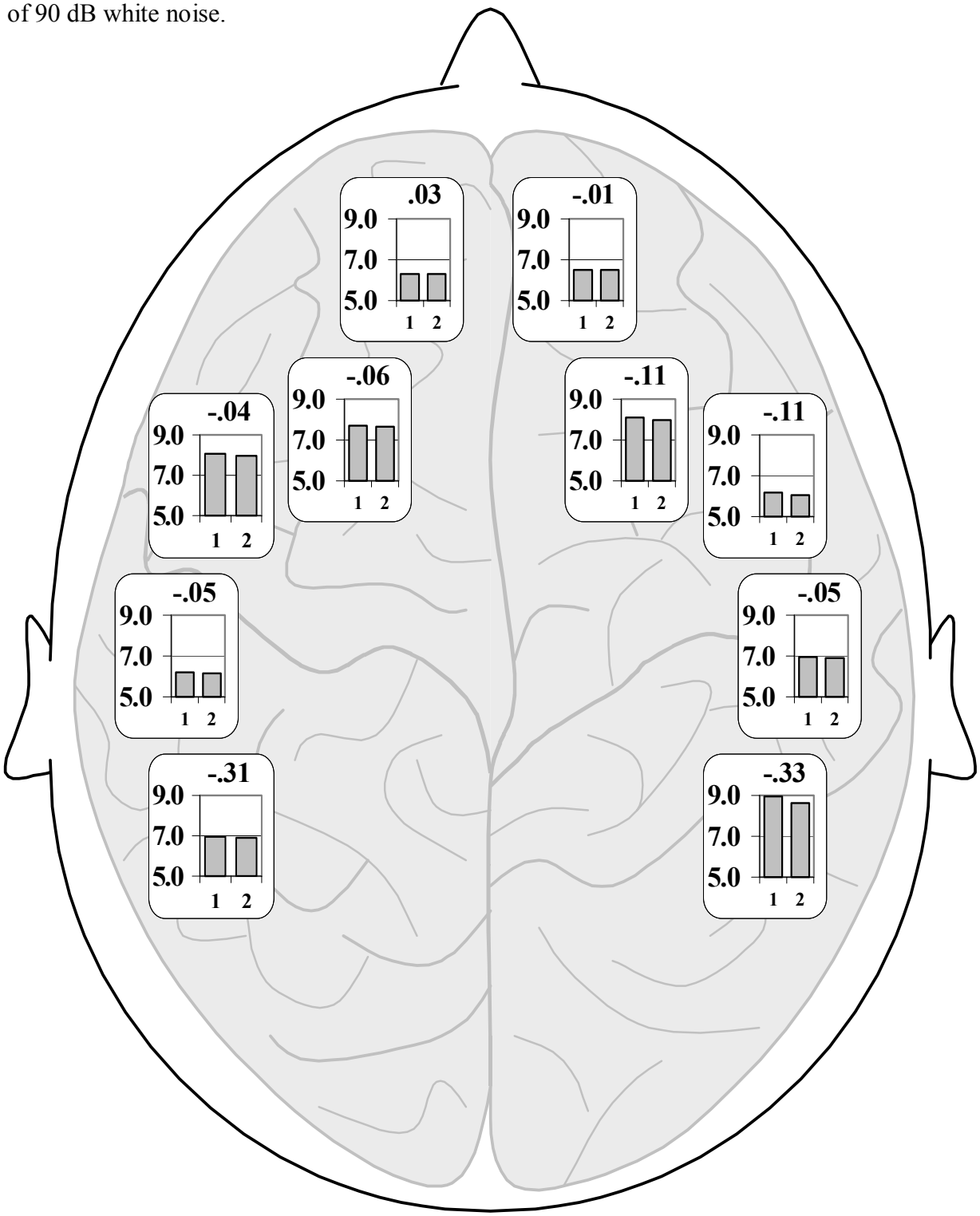
Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 55 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 55 dB white noise condition in magnitude (μV).

Figure 5. Low beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 75 dB white noise.



Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 75 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 75 dB white noise condition in magnitude (μV).

Figure 6. Low beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 90 dB white noise.



Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 90 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 90 dB white noise condition in magnitude (μV).

Table 9

Means and standard deviations for low beta magnitude as a function of Electrode Site.

Electrode Site	Mean	Standard Deviation
F1	6.25 b, c	1.83
F2	6.48 b, c	2.00
F3	7.73 a	2.48
F4	8.07 a	2.65
F7	5.69 c	1.68
F8	6.08 b, c	2.05
T3	5.97 b, c	2.16
T4	6.68 b	2.60
T5	8.18 a	2.19
T6	8.39 a	2.80

Note. Values are reported in μV. Means with the same letter are not significantly different. Multiple comparisons based on Tukey's HSD test.

Table 10

ANOVA source table for low beta magnitude at bilateral frontal and temporal lobes.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	23.97	23.97	0.17	.68
Condition	3, 84	25.86	8.62	1.61	.19
Electrode Site	9, 252	1144.76	127.20	23.89	.0001
Group x Condition	3, 84	1.30	0.43	0.08	.97
Group x Electrode Site	9, 252	64.71	7.19	1.35	.21
Condition x Electrode Site	27, 756	29.12	1.08	2.18	.0005
Group x Condition x Electrode Site	27, 756	12.08	0.45	0.91	.61

Table 11

Means and standard deviations of low beta magnitude difference scores as a function of Condition and Electrode Site.

Condition	Electrode Site	Mean	Standard Deviation
55 dB White Noise	F1	-0.17 b, c, d, e	1.03
	F2	-0.17 b, c, d, e	1.03
	F3	-0.02 c, d, e	1.45
	F4	-0.04 c, d, e	1.37
	F7	-0.14 b, c, d, e	1.04
	F8	-0.25 b, c, d, e	1.18
	T3	-0.55 a, b, c, d, e	1.79
	T4	-0.72 a, b, c	1.57
	T5	-0.77 a, b	1.93
	T6	-1.15 a	2.52
75 dB White Noise	F1	0.03 d, e	1.03
	F2	0.04 e	1.17
	F3	0.17 e	1.36
	F4	0.11 e	1.63
	F7	-0.14 b, c, d, e	1.04
	F8	-0.03 c, d, e	1.32
	T3	-0.32 b, c, d, e	1.30
	T4	-0.33 b, c, d, e	1.62
	T5	-0.69 a, b, c, d	1.75
	T6	-0.77 a, b	2.39

Table 11

Continued

Condition	Electrode Site	Mean	Standard Deviation
90 dB White Noise	F1	0.03 d, e	0.79
	F2	-0.01 c, d, e	0.89
	F3	-0.06 b, c, d, e	1.18
	F4	-0.11 b, c, d, e	1.12
	F7	-0.04 c, d, e	1.21
	F8	-0.11 b, c, d, e	1.08
	T3	-0.05 b, c, d, e	2.01
	T4	-0.05 b, c, d, e	1.63
	T5	-0.31 b, c, d, e	1.91
	T6	-0.33 b, c, d, e	2.19

Note. Values are reported in μV. Means with the same letter are not significantly different. Multiple comparisons based on Tukey's HSD test.

low beta magnitude from the baseline condition to any of the three white noise conditions. Further analyses did reveal that the right posterior temporal lobe was associated with greater changes in low beta magnitude than each of the right frontal electrode sites for the 55 dB condition (T6, *M* = -1.15, *SD* = 2.52; F2, *M* = -.17, *SD* = 1.03; F4, *M* = -.04, *SD* = 1.37; F8, *M* = -.25, *SD* = 1.18; see Figure 7) as well as the 75 dB condition (T6, *M* = -.77, *SD* = 2.39; F2, *M* = .04, *SD* = 1.17; F4, *M* = .11, *SD* = 1.63; F8, *M* = -.03, *SD* = 1.32; see Figure 8). Additionally, the left posterior temporal lobe was associated with greater changes in low beta magnitude than the left lateral frontal lobe for the 55 dB condition (T5, *M* = -.77, *SD* = 1.93; F3, *M* = -.02, *SD* = 1.45; see Figure 9) as well as the 75 dB condition (T5, *M* = -.69, *SD* = 1.75; F3, *M* = .17, *SD* = 1.36; see Figure 10). Finally, multiple comparisons using Tukey's HSD indicated that the 55 dB condition generated significantly greater reductions in low beta magnitude than the 75 dB condition at the right posterior temporal lobe (55 dB, *M* = -1.15, *SD* = 2.52; 75 dB, *M* = -.77, *SD* = 2.39; see Figure 11).

A significant main effect for Electrode Site was also found, $F(9, 252) = 2.72$, $p = .005$. Multiple comparisons using Tukey's HSD indicated that the low beta magnitude at the right posterior temporal lobe was significantly lower than at the right and left lateral frontal lobes (see Table 12). No other main or interaction effects were noted to be statistically significant (see Table 13 for the ANOVA source table for low beta magnitude difference scores).

Additional analyses were performed to determine whether significant differences existed between homologous comparisons of electrode sites at the baseline and at post white noise. The results of a 2 (Group) x 4 (Condition) x 2 (Hemisphere) x 10 (Electrode Site) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factors of Condition,

Figure 7. Changes in low beta magnitude (μV) at the right frontal and posterior temporal lobes as a function of 55 dB white noise.

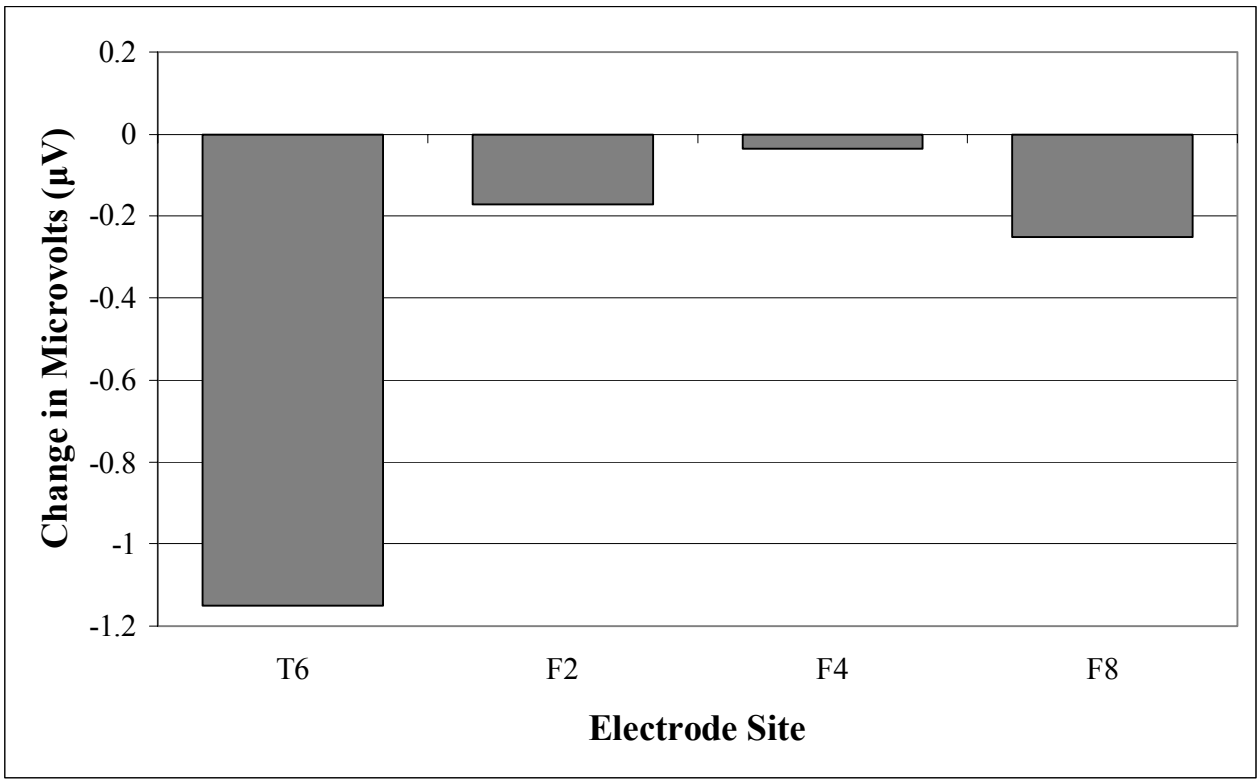


Figure 8. Changes in low beta magnitude (μV) at the right frontal and posterior temporal lobes as a function of 75 dB white noise.

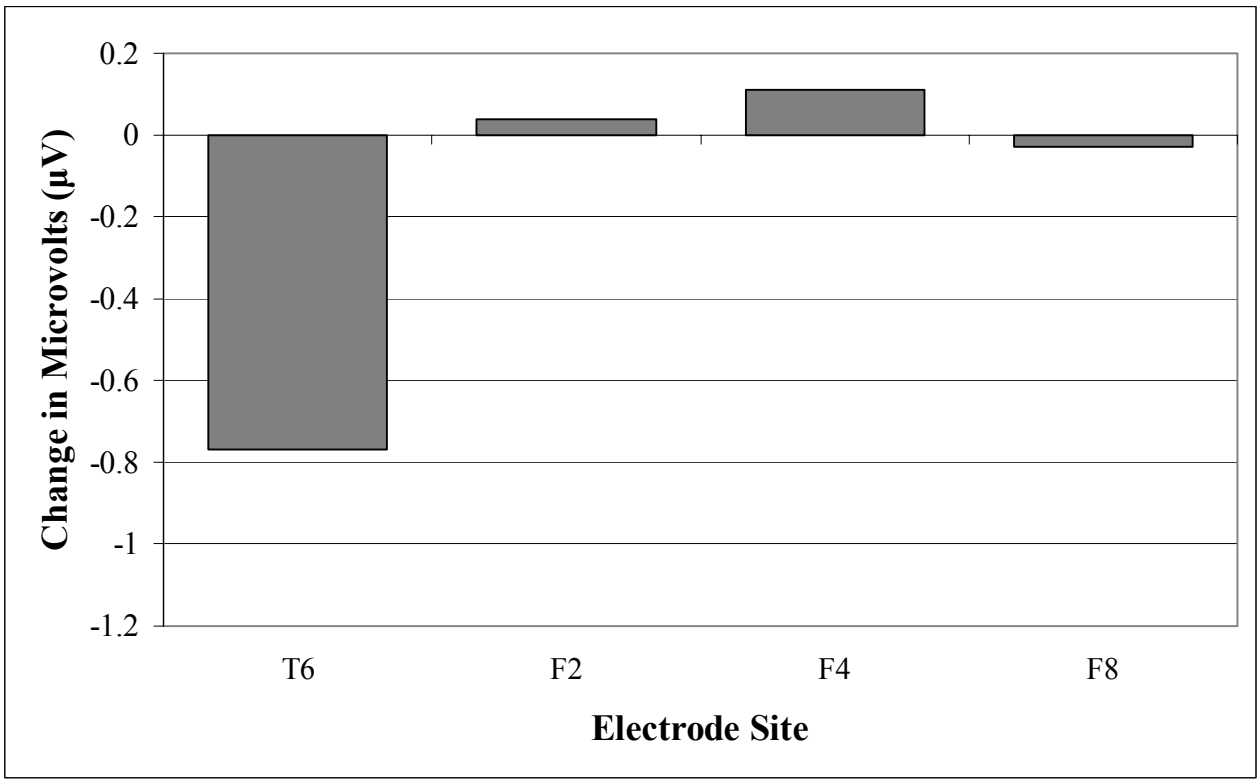


Figure 9. Changes in low beta magnitude (μV) at the left lateral frontal and posterior temporal lobes as a function of 55 dB white noise.

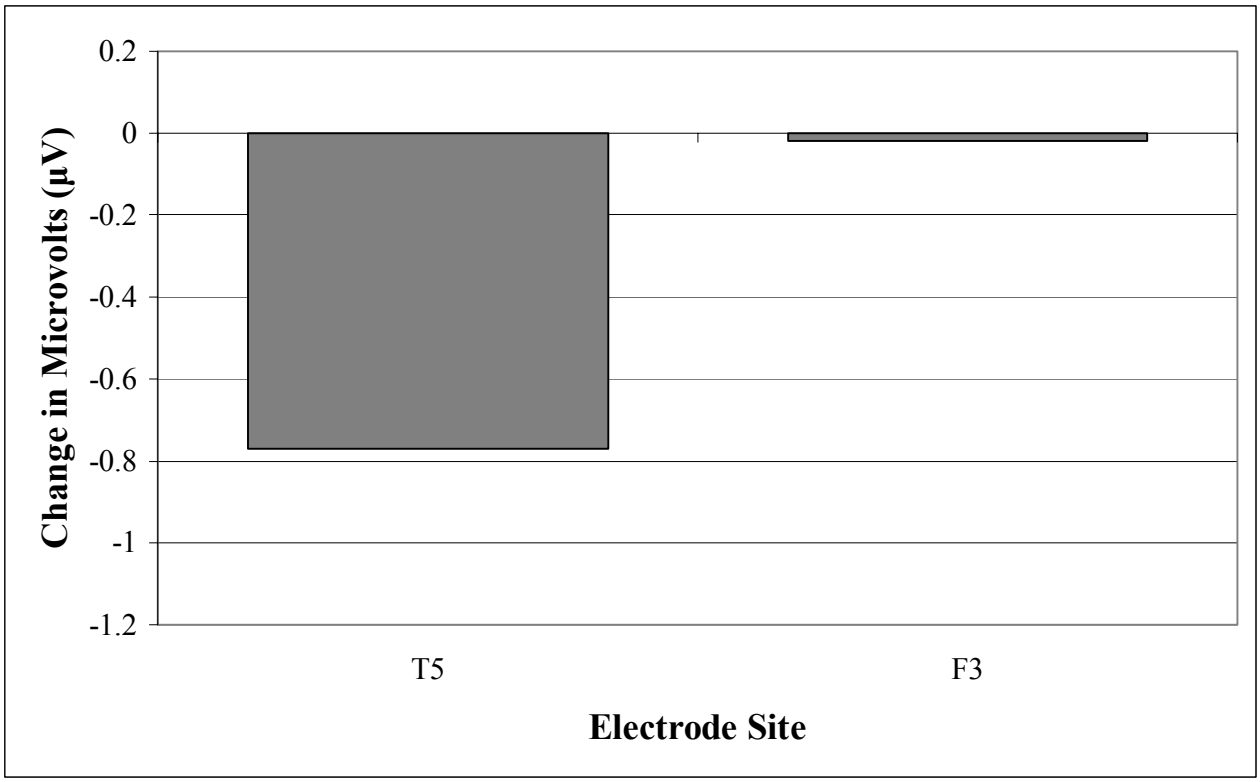


Figure 10. Changes in low beta magnitude (μV) at the left lateral frontal and posterior temporal lobes as a function of 75 dB white noise.

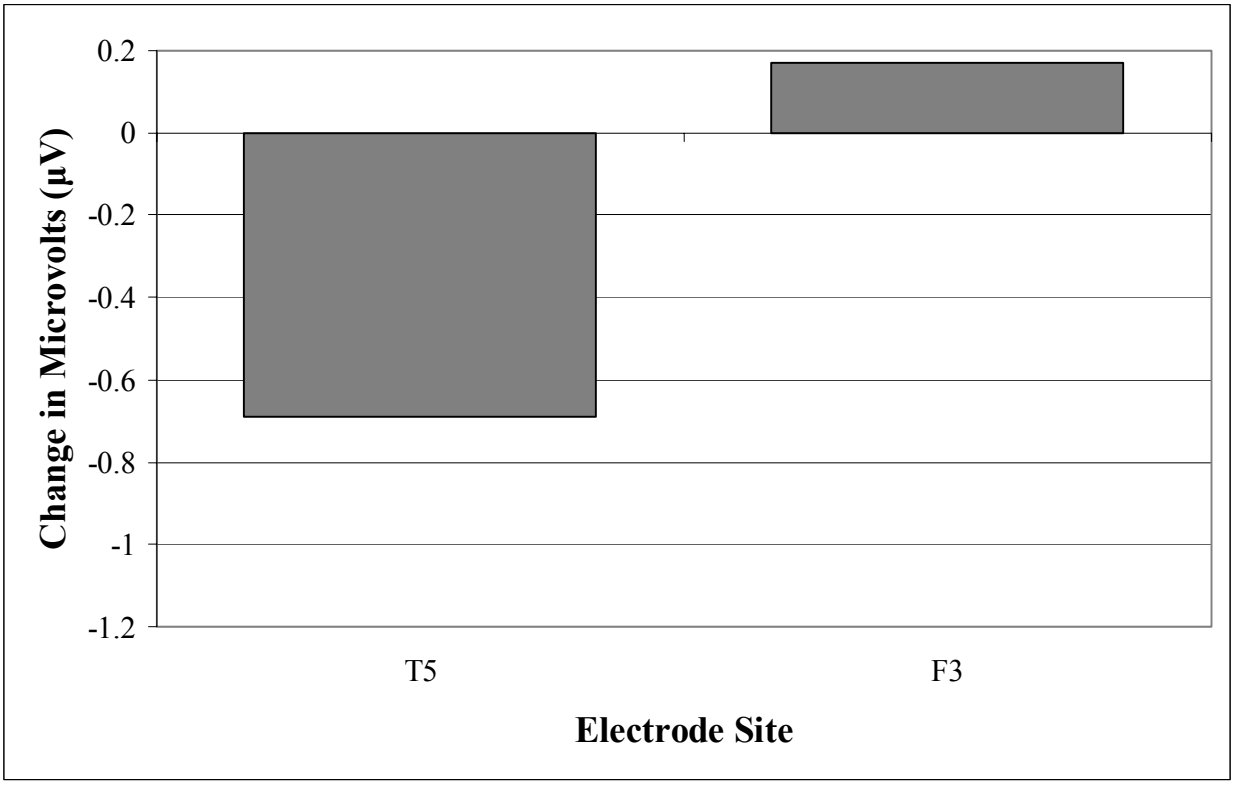


Figure 11. Changes in low beta magnitude (μV) at the right posterior temporal lobe as a function of varying white noise intensity.

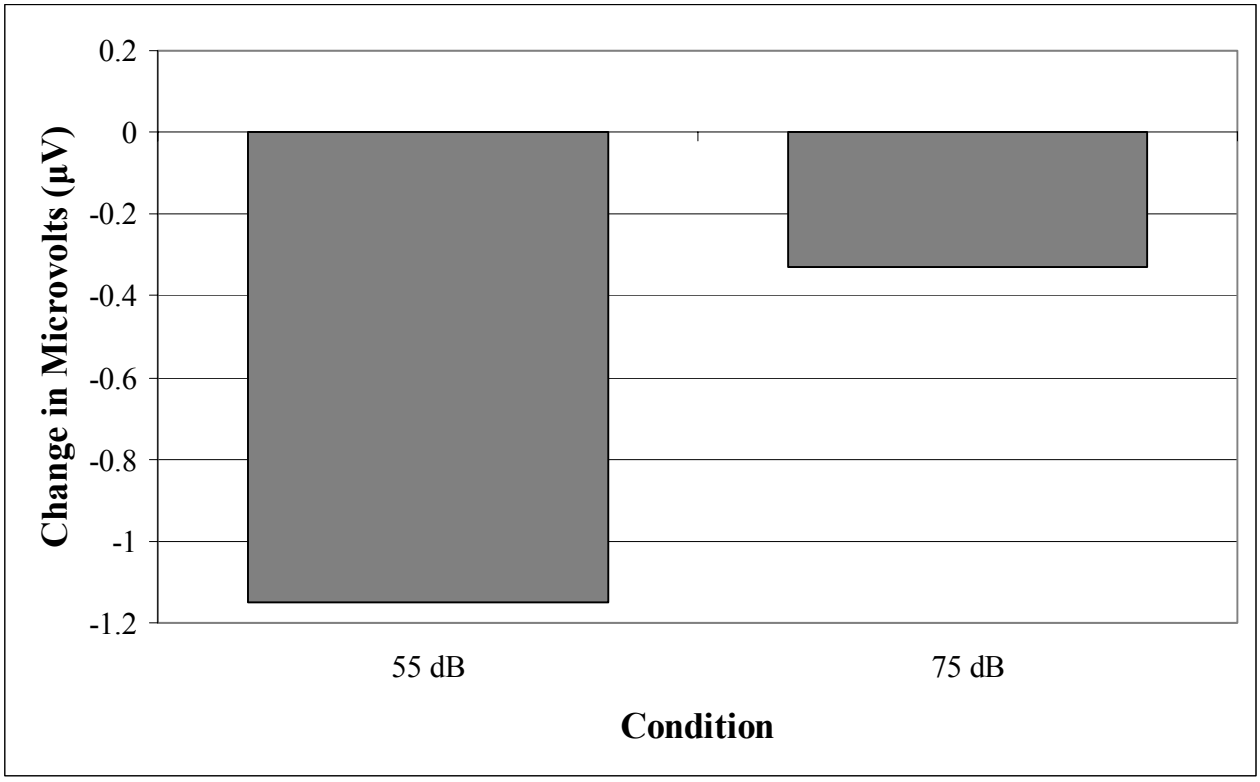


Table 12

Means and standard deviations of low beta magnitude difference scores as a function of Electrode Site.

Electrode Site	Mean	Standard Deviation
F1	-0.04 a, b	0.95
F2	-0.05 a, b	1.04
F3	0.03 a	1.32
F4	-0.01 a	1.37
F7	-0.11 a, b	1.09
F8	-0.13 a, b	1.10
T3	-0.31 a, b	1.71
T4	-0.37 a, b	1.58
T5	-0.59 a, b	1.86
T6	-0.75 b	2.32

Note. Values are reported in μV. Means with the same letter are not significantly different. Multiple comparisons based on Tukey's HSD test.

Table 13

ANOVA source table for low beta magnitude difference scores.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	0.15	0.15	0.00	.95
Condition	2, 56	13.78	6.89	1.83	.17
Electrode Site	9, 252	57.17	6.35	2.72	.005
Group x Condition	2, 56	1.27	0.63	0.17	.85
Group x Electrode Site	9, 252	15.83	1.76	0.75	.66
Condition x Electrode Site	18, 504	14.85	0.82	1.83	.02
Group x Condition x Electrode Site	18, 504	8.11	0.45	1.00	.46

Hemisphere, and Electrode Site indicated a significant main effect for Hemisphere, $F(1, 28) = 4.75$, $p = .04$. Multiple comparisons using Tukey's HSD indicated heightened low beta magnitude over the right hemisphere ($M = 7.14$, $SD = 2.62$) as compared to the left hemisphere ($M = 6.76$, $SD = 2.31$). No other main or interaction effects involving the variable Hemisphere were statistically significant (see Table 14 for the ANOVA source table for low beta magnitude involving the Hemisphere variable).

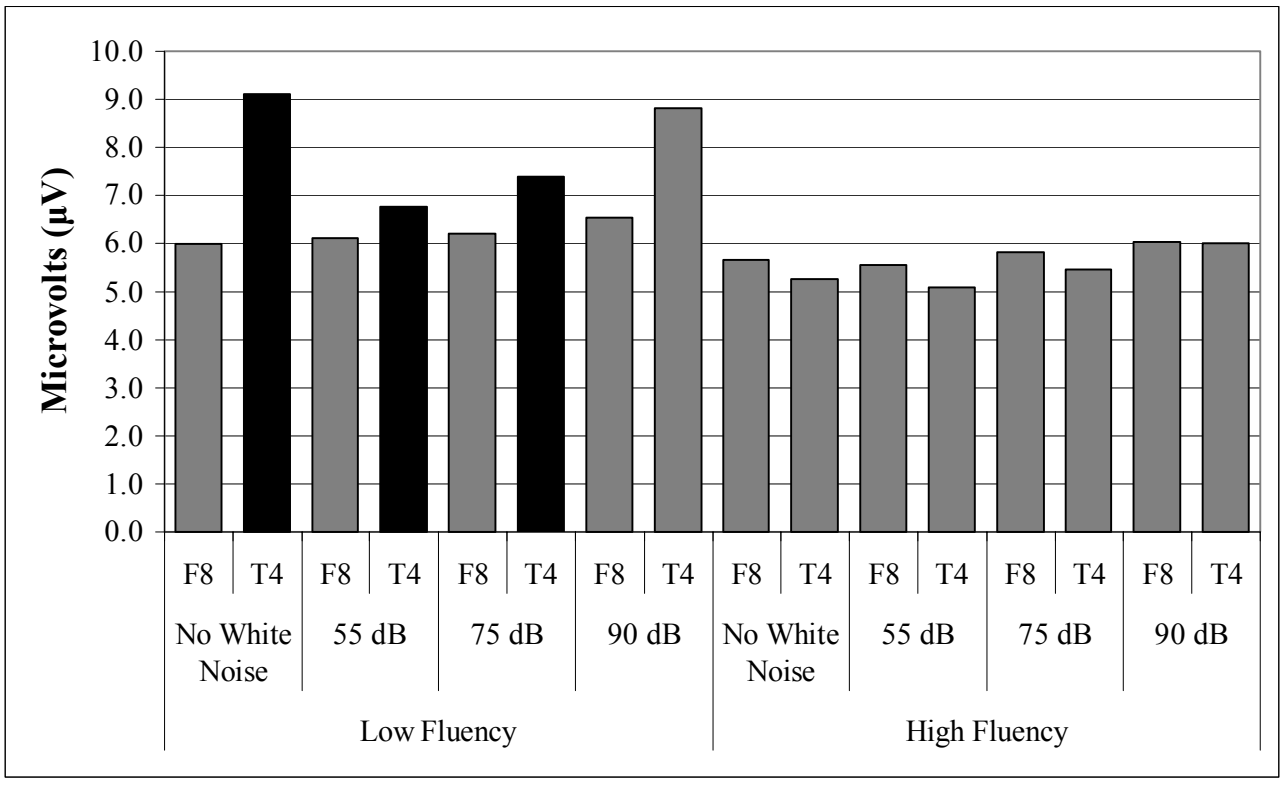
High Beta. The results of a 2 (Group) x 4 (Condition) x 2 (Electrode Site) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factors of Condition and Electrode Site, indicated a significant Group x Condition x Electrode Site interaction, $F(3, 84) = 3.13$, $p = .03$ (see Figure 12). Multiple comparisons using Tukey's HSD indicated that both the 55 dB (Baseline, $M = 9.11$, $SD = 5.23$; 55 dB White Noise, $M = 6.76$, $SD = 3.07$) and the 75 dB (Baseline, $M = 9.11$, $SD = 5.23$, 75 dB White Noise, $M = 7.39$, $SD = 3.94$) conditions resulted in a significant reduction of high beta magnitude at the right anterior temporal lobe electrode site for the Low Fluency group. No significant reduction in high beta magnitude was noted at the right lateral frontal lobe electrode site for the Low Fluency group. Additionally, no significant changes in high beta magnitude were noted at either electrode site for the High Fluency group. A significant Condition x Electrode Site interaction was also noted, $F(3, 84) = 3.96$, $p = .01$ (see Figure 13). Multiple comparisons using Tukey's HSD indicated that the 55 dB condition resulted in a significant reduction in high beta magnitude at the anterior temporal lobe (Baseline, $M = 7.18$, $SD = 4.36$; 55 dB White Noise, $M = 5.92$, $SD = 2.70$). No significant changes in high beta magnitude were noted for any other electrode site or any other condition. Finally, the main effect for Condition was statistically significant, $F(3, 84) = 3.71$, $p = .01$ (see Figure 14). However, multiple comparisons using Tukey's HSD indicated that no

Table 14

ANOVA source table for low beta magnitude involving the Hemisphere variable.

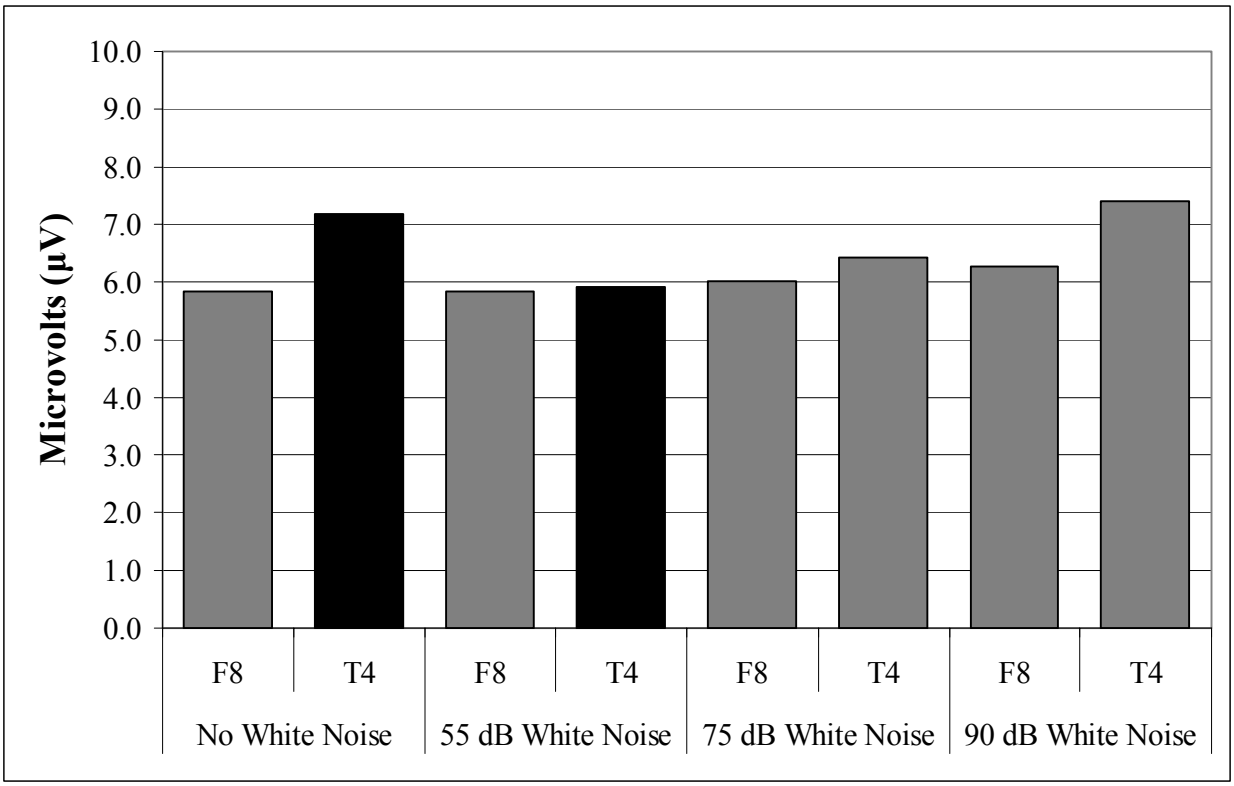
Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	23.97	23.97	0.17	.68
Condition	3, 84	25.86	8.62	1.61	.19
Hemisphere	1, 28	42.49	42.49	4.75	.04
Electrode Site	4, 112	1092.53	273.13	35.39	.0001
Group x Condition	3, 84	1.30	0.43	0.08	.97
Group x Hemisphere	1, 28	9.08	9.08	1.02	.32
Group x Electrode Site	4, 112	52.14	13.03	1.69	.16
Condition x Hemisphere	3, 84	0.88	0.29	0.41	.75
Condition x Electrode Site	12, 336	26.96	2.25	3.24	.0002
Hemisphere x Electrode Site	4, 112	9.74	2.43	1.20	.31
Group x Condition x Hemisphere	3, 84	1.79	0.60	0.83	.48
Group x Condition x Electrode Site	12, 336	8.27	0.69	1.00	.45
Group x Hemisphere x Electrode Site	4, 112	3.49	0.87	0.43	.79
Condition x Hemisphere x Electrode Site	12, 336	1.28	0.11	0.44	.94
Group x Condition x Hemisphere x Electrode Site	12, 336	2.02	0.17	0.70	.75

Figure 12. Right lateral frontal and anterior temporal lobe high beta magnitude (μV) as a function of Group, Condition, and Electrode Site.



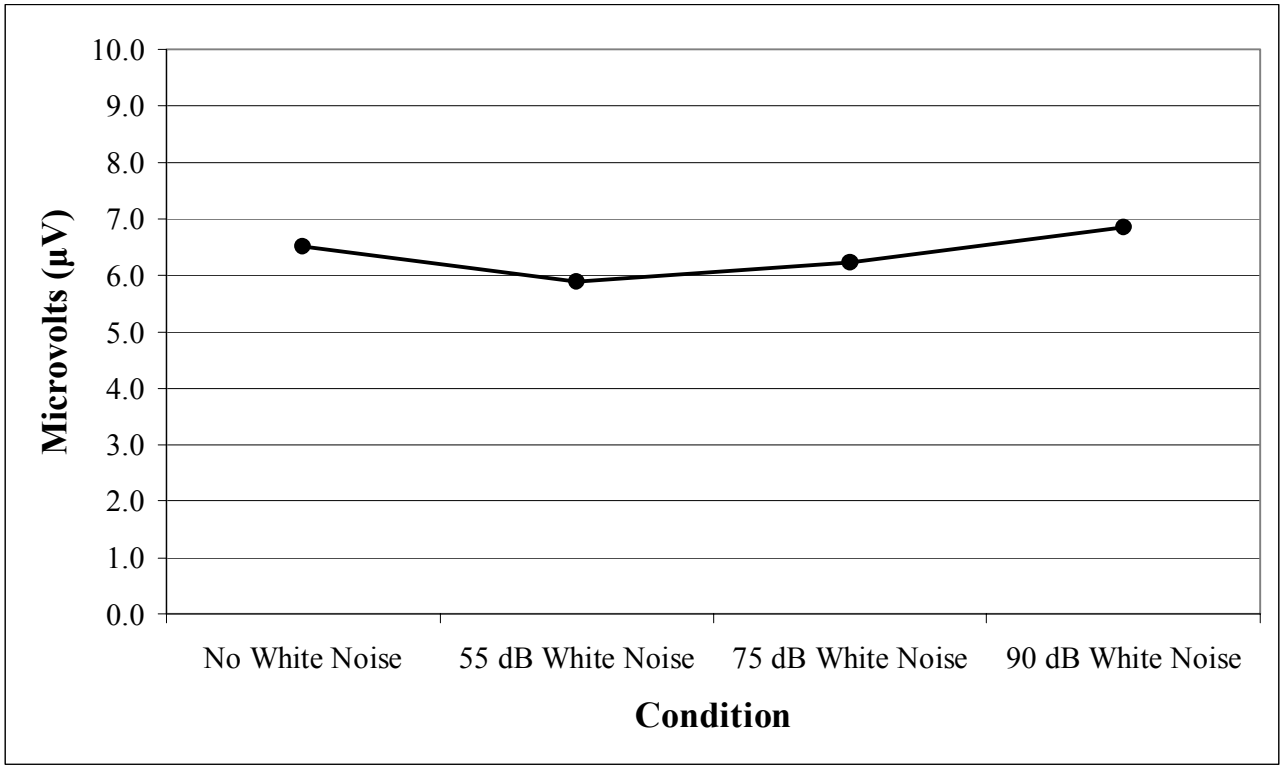
Note. Graphs in black represent statistically significant differences, i.e. $p < .05$.

Figure 13. Right lateral frontal and anterior temporal lobe high beta magnitude (μV) as a function of Condition and Electrode Site.



Note. Graphs in black represent statistically significant differences, i.e. $p < .05$.

Figure 14. Right lateral frontal and anterior temporal lobe high beta magnitude (μV) as a function of Condition.



condition resulted in a significant reduction of high beta magnitude from the baseline. All other main and interaction effects were nonsignificant (see Table 15 for the ANOVA source table for high beta magnitude at the right lateral frontal and anterior temporal lobes).

As with the data for low beta magnitude, a more broad analysis of the data was performed to understand more completely the effects of white noise presentation on bilateral frontal and temporal lobe activity. The results of a 2 (Group) x 4 (Condition) x 10 (Electrode Site) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factors of Condition and Electrode Site, indicated a significant Group x Condition x Electrode Site interaction, $F(27, 756) = 1.60$, $p = .03$. Multiple comparisons using Tukey's HSD indicated significant reductions in high beta magnitude at the anterior temporal lobe for both the 55 dB (Baseline, $M = 9.11$, $SD = 5.23$; 55 dB White Noise, $M = 6.76$, $SD = 3.07$) and the 75 dB (Baseline, $M = 9.11$, $SD = 5.23$; 75 dB White Noise, $M = 7.39$, $SD = 3.94$) conditions, but only for the Low Fluency group (see Figures 15 and 16, respectively). Significant changes in high beta magnitude were not noted at any electrode site for the 90 dB condition (see Figure 17). Additionally, no significant reductions in high beta magnitude were noted for any electrode site or condition for the High Fluency group.

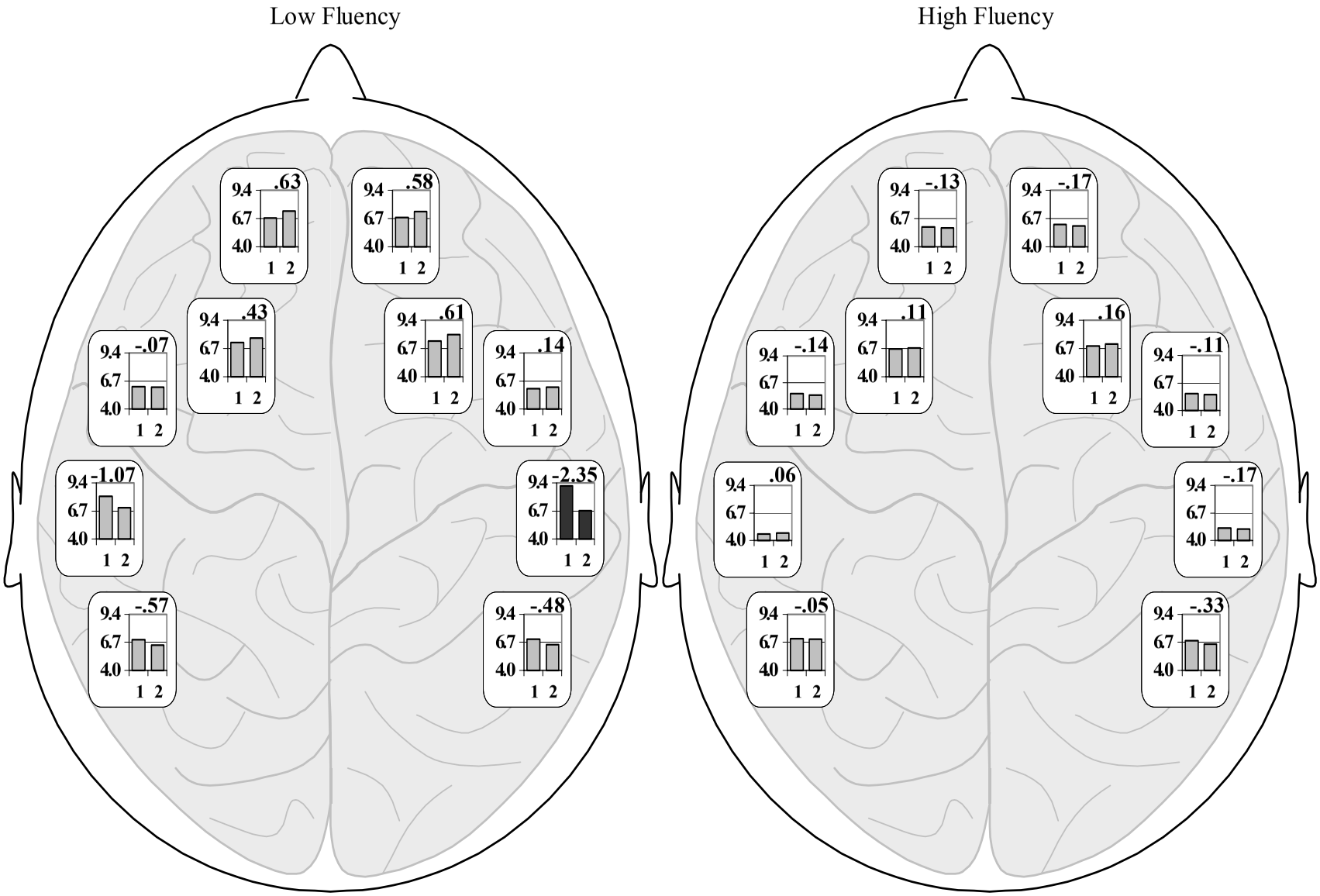
A significant Condition x Electrode Site interaction was found, $F(27, 756) = 2.01$, $p = .002$. However, multiple comparisons using Tukey's HSD indicated that no white noise condition resulted in a significant reduction of high beta magnitude at any electrode site (see Figures 18 through 20). The Group x Electrode Site interaction was also statistically significant, $F(9, 252) = 2.84$, $p = .003$ (see Table 16). Multiple comparisons using Tukey's HSD indicated heightened high beta magnitude for the Low Fluency group, as compared to the High Fluency group, at the left anterior temporal lobe (Low Fluency, $M = 7.77$, $SD = 3.97$; High Fluency,

Table 15

ANOVA source table for high beta magnitude at the right lateral frontal and anterior temporal lobes.

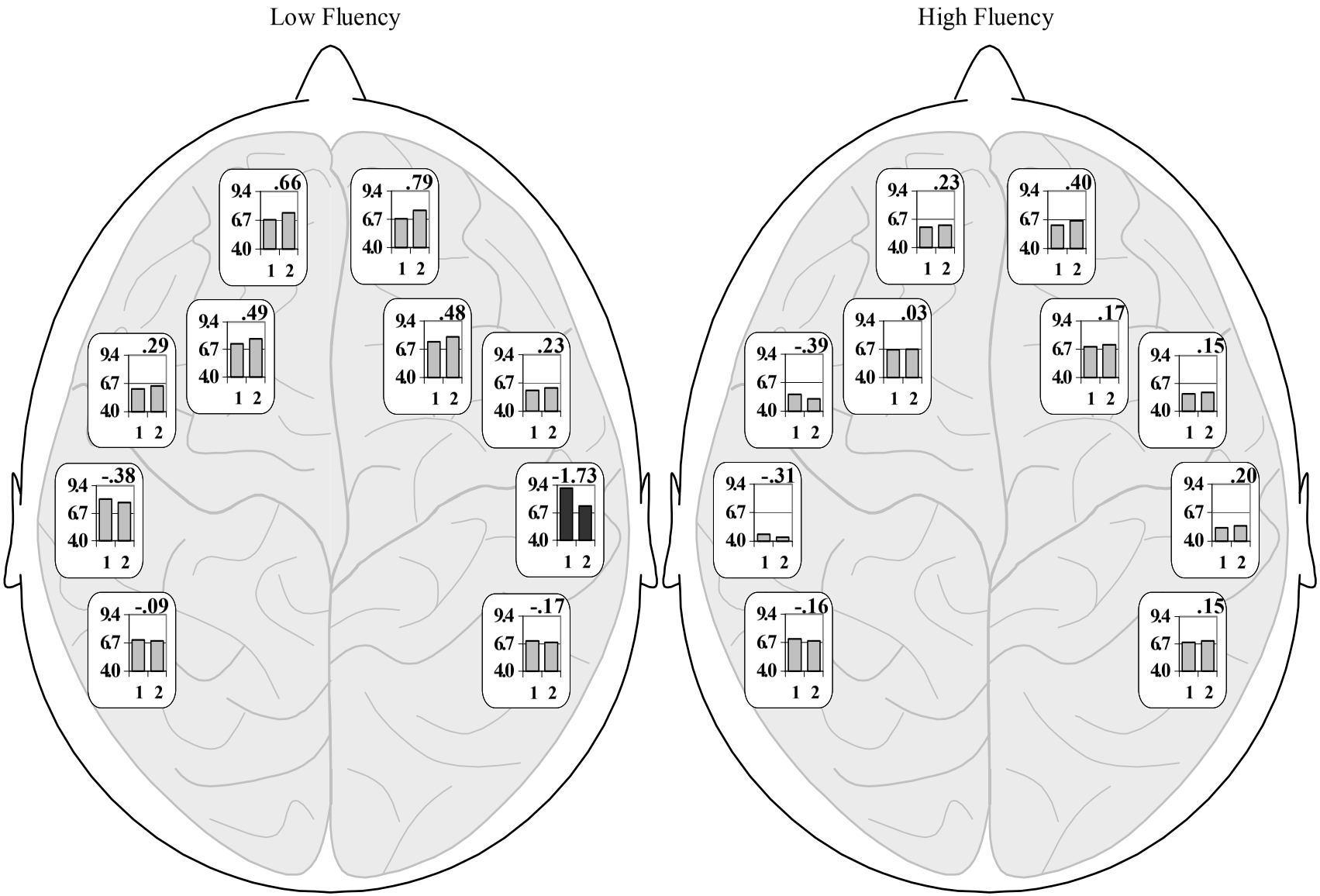
Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	136.35	136.35	3.56	.07
Condition	3, 84	30.02	10.01	3.71	.01
Electrode Site	1, 28	33.38	33.38	1.97	.17
Group x Condition	3, 84	9.42	3.14	1.16	.33
Group x Electrode Site	1, 28	67.95	67.95	4.02	.06
Condition x Electrode Site	3, 84	16.27	5.42	3.96	.01
Group x Condition x Electrode Site	3, 84	12.90	4.30	3.13	.03

Figure 15. High beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 55 dB white noise and design fluency.



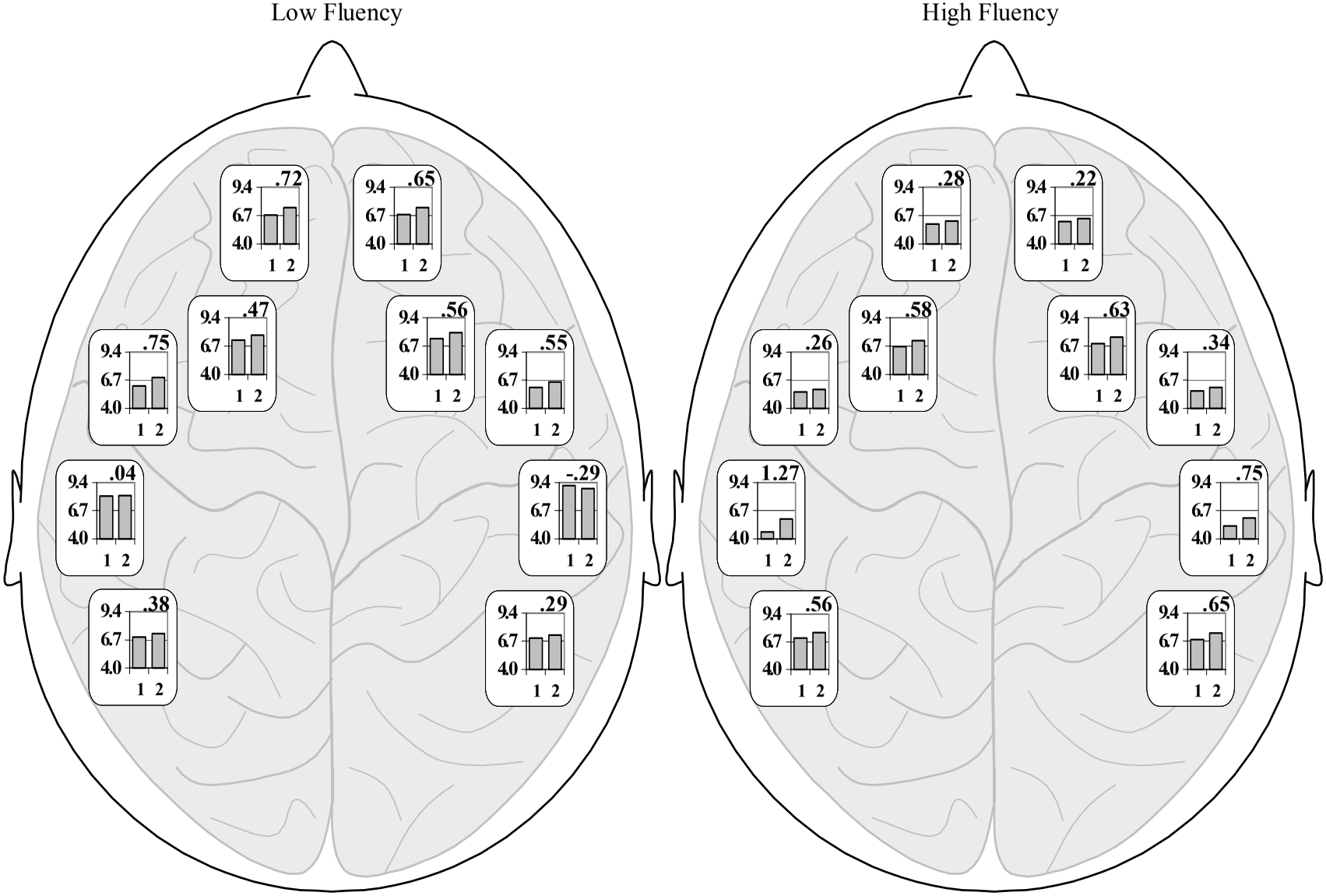
Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 55 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 55 dB white noise condition.

Figure 16. High beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 75 dB white noise and design fluency.



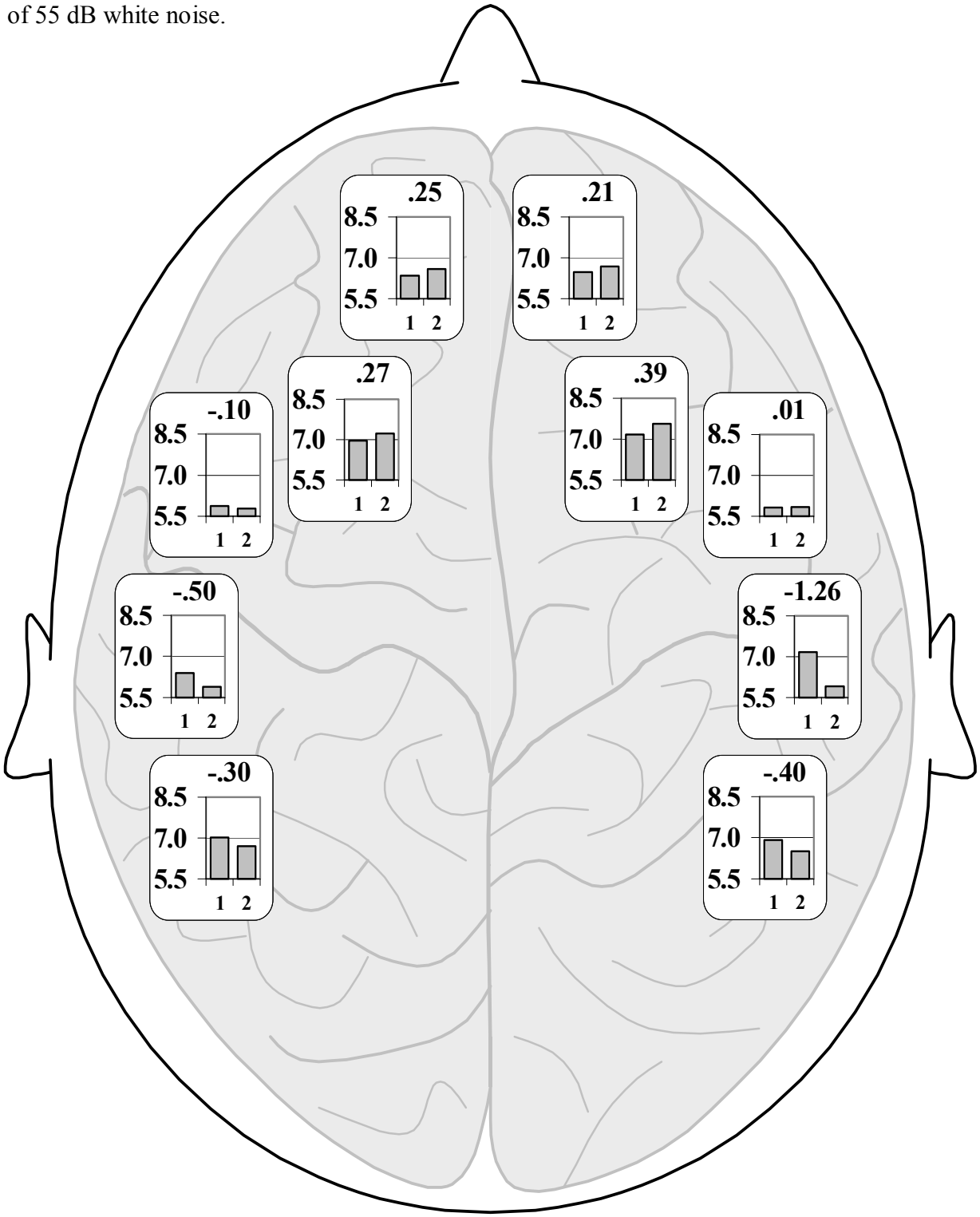
Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 75 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 75 dB white noise condition.

Figure 17. High beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 90 dB white noise and design fluency.



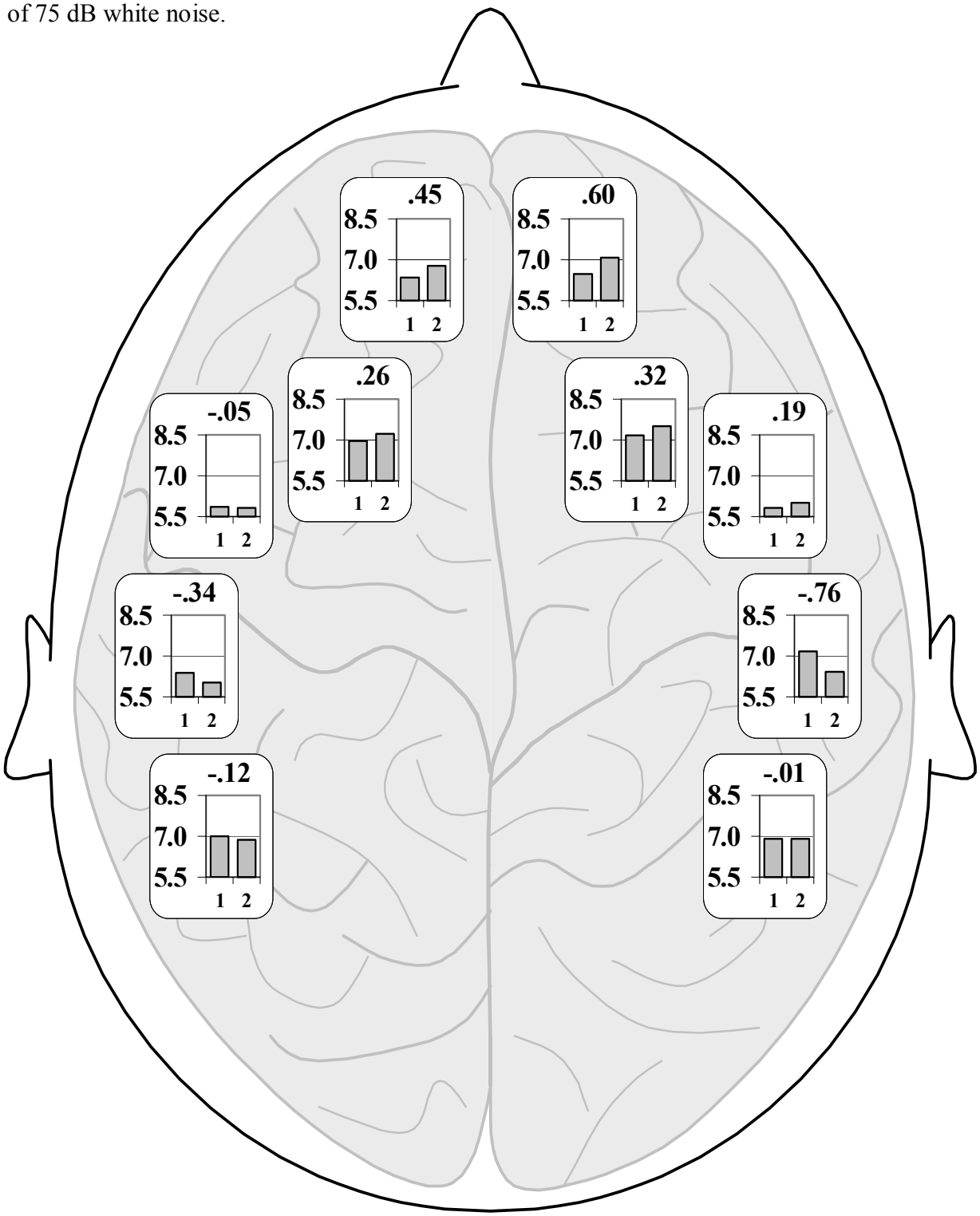
Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 90 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 90 dB white noise condition.

Figure 18. High beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 55 dB white noise.



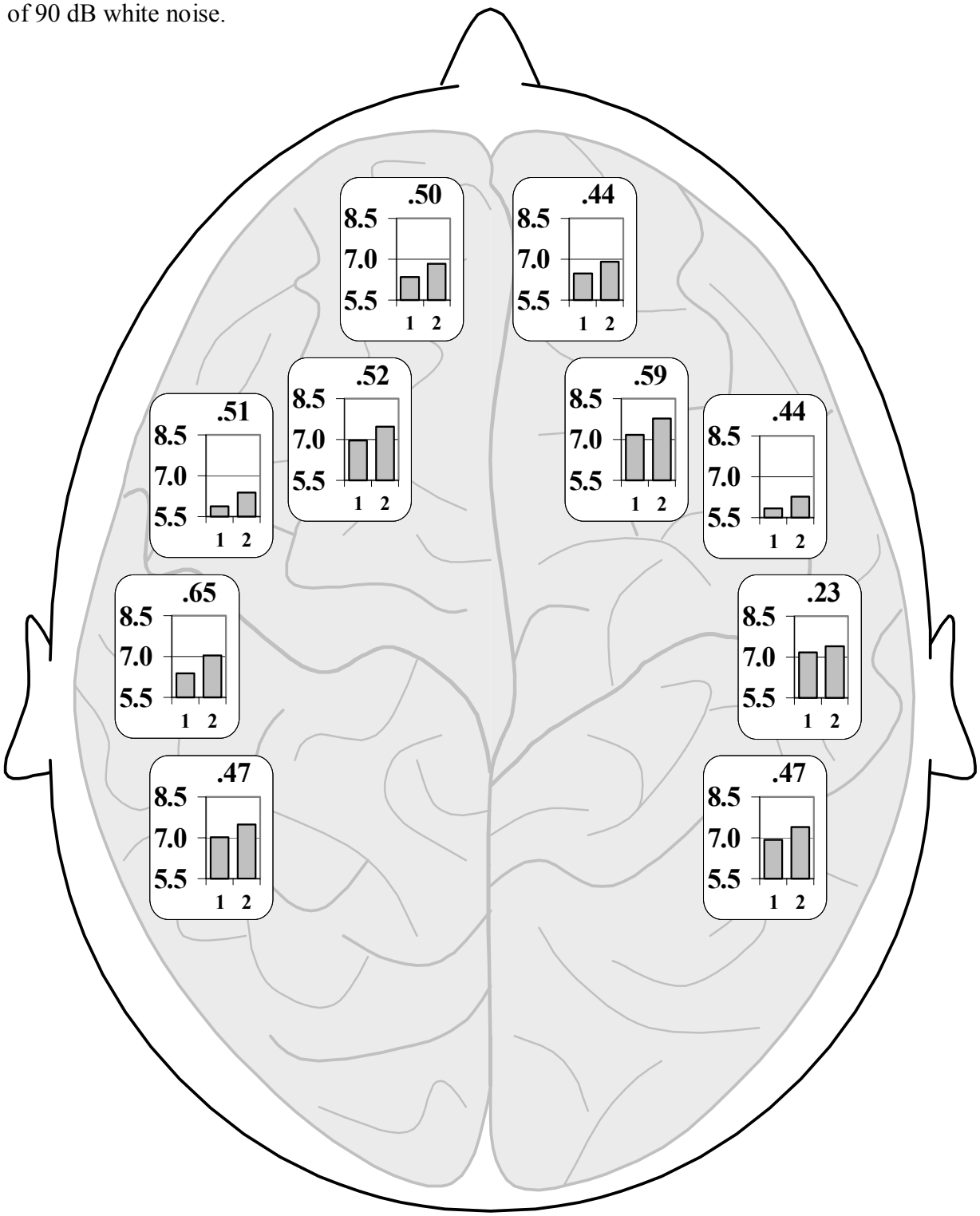
Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 55 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 55 dB white noise condition in magnitude (μV).

Figure 19. High beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 75 dB white noise.



Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 75 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 75 dB white noise condition in magnitude (μV).

Figure 20. High beta magnitude (μV) at the bilateral frontal and temporal lobes as a function of 90 dB white noise.



Note. Graphs in black represent statistically significant differences, i.e. $p < .05$, between the baseline or no white noise condition (1) and the 90 dB white noise condition (2). The numbers at the top of each graph represent the difference between the baseline and the 90 dB white noise condition in magnitude (μV).

Table 16

Means and standard deviations for high beta magnitude as a function of Group and Electrode Site.

Group	Electrode Site	Mean	Standard Deviation
Low Fluency	F1	7.26	2.46
	F2	7.28	2.49
	F3	7.61	2.29
	F4	7.82	2.55
	F7	6.42	1.88
	F8	6.21	1.84
	T3	7.77 *	3.97
	T4	8.02 *	4.58
	T5	6.89	1.29
	T6	6.89	1.79

Table 16

continued

Group	Electrode Site	Mean	Standard Deviation
High Fluency	F1	6.02	1.61
	F2	6.28	1.91
	F3	6.83	2.04
	F4	7.18	2.24
	F7	5.50	1.71
	F8	5.77	2.07
	T3	4.91 *	2.20
	T4	5.45 *	2.12
	T5	7.14	2.13
	T6	6.97	2.13

Note. Values are reported in μV. * represents statistically significant differences between the means of these homologous comparisons. Multiple comparisons based on Tukey's HSD test.

M = 4.91, *SD* = 2.20) as well as the right anterior temporal lobe (Low Fluency, *M* = 8.02, *SD* = 4.58; High Fluency, *M* = 5.45, *SD* = 2.12).

The main effect for Condition was also found to be statistically significant, $F(3, 84) = 4.92$, $p = .003$ (see Figure 21). Multiple comparisons using Tukey's HSD indicated that the 90 dB White Noise condition resulted in a significant increase in high beta magnitude from the baseline (Baseline, *M* = 6.61, *SD* = 2.50; 90 dB White Noise, *M* = 7.10, *SD* = 2.63). Finally, the main effect for Electrode Site was statistically significant, $F(9, 252) = 2.79$, $p = .004$ (see Table 17). No other main or interaction effects were noted to be statistically significant (see Table 18 for the ANOVA source table for high beta magnitude at all electrode sites within the bilateral frontal and temporal lobes).

Further analyses were performed, as with the low beta magnitude data, on the difference scores obtained by subtracting the high beta μV resulting from the baseline from the high beta μV resulting from presentation of each intensity level of white noise. The purposes for these analyses were the same as for the data for low beta magnitude and, hence, will not be repeated here. The results of a 2 (Group) x 3 (Condition) x 10 (Electrode Site) mixed factorial ANOVA on the high beta difference scores, with the between subjects factor of Group and the repeated factors of Condition and Electrode Site, indicated a significant Condition x Electrode Site interaction, $F(18, 504) = 1.84$, $p = .01$ (see Table 19). Multiple comparisons using Tukey's HSD indicated that no two homologous electrode sites differed significantly in terms of changes in high beta magnitude from the baseline condition to any of the three white noise conditions. Further analyses did reveal, however, that the right anterior temporal lobe was associated with greater changes in high beta magnitude than all of the right frontal electrode sites for the 55 dB

Figure 21. High beta magnitude (μV) as a function of Condition.

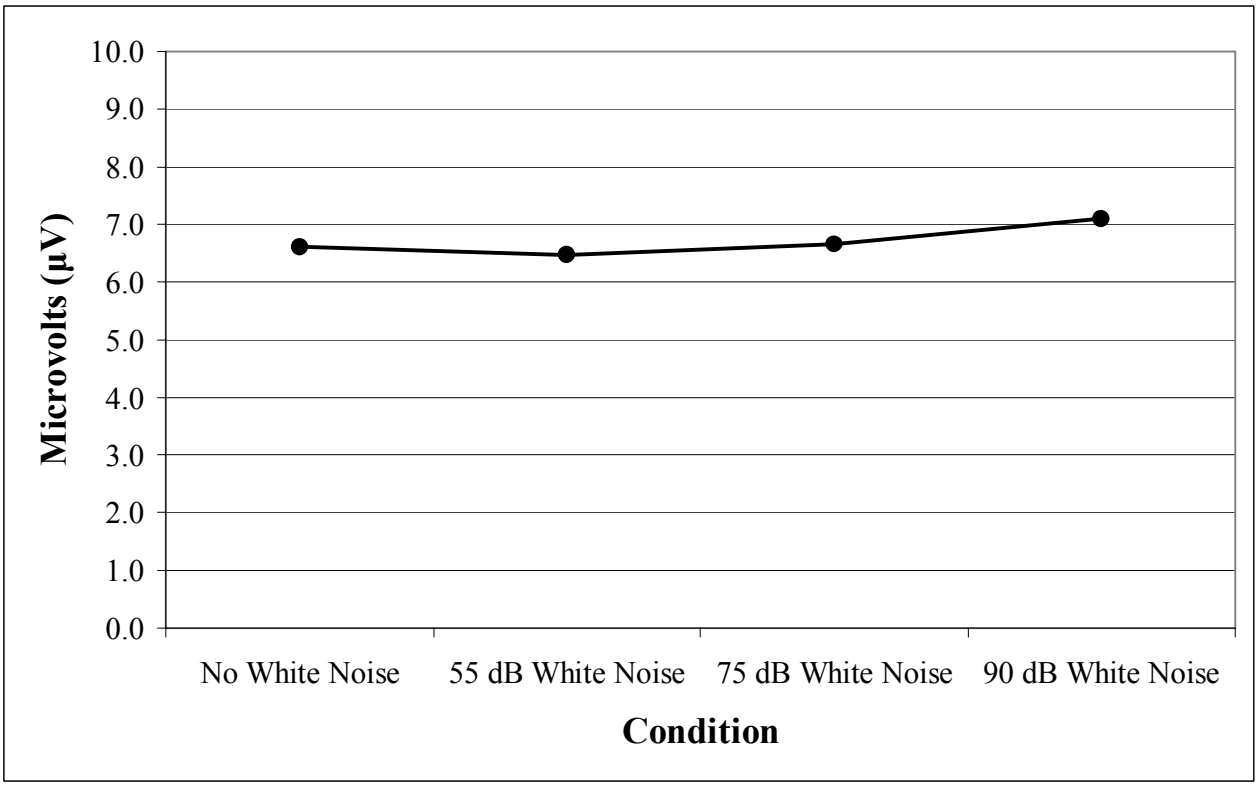


Table 17

Means and standard deviations for high beta magnitude as a function of Electrode Site.

Electrode Site	Mean	Standard Deviation
F1	6.64 a, b	2.04
F2	6.78 a, b	2.20
F3	7.22 a, b	2.17
F4	7.50 a	2.40
F7	5.96 b	1.80
F8	5.99 b	1.96
T3	6.34 a, b	3.09
T4	6.73 a, b	3.35
T5	7.02 a, b	1.71
T6	6.93 a, b	1.96

Note. Values are reported in μV. Means with the same letter are not significantly different. Multiple comparisons based on Tukey's HSD test.

Table 18

ANOVA source table for high beta magnitude at bilateral frontal and temporal lobes.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	307.55	307.55	3.19	.09
Condition	3, 84	65.48	21.83	4.92	.003
Electrode Site	9, 252	271.61	30.18	2.79	.004
Group x Condition	3, 84	1.57	0.52	0.12	.95
Group x Electrode Site	9, 252	276.23	30.69	2.84	.003
Condition x Electrode Site	27, 756	53.32	1.97	2.01	.002
Group x Condition x Electrode Site	27, 756	42.43	1.57	1.60	.03

Table 19

Means and standard deviations of high beta magnitude difference scores as a function of Condition and Electrode Site.

Condition	Electrode Site	Mean	Standard Deviation
55 dB White Noise	F1	0.25 c, d, e, f	1.19
	F2	0.21 b, c, d, e, f	0.85
	F3	0.27 c, d, e, f	0.75
	F4	0.39 c, d, e, f	1.01
	F7	-0.10 b, c, d, e, f	1.31
	F8	0.01 b, c, d, e, f	0.91
	T3	-0.50 a, b, c	2.65
	T4	-1.26 a	2.84
	T5	-0.31 a, b, c, d, e, f	1.64
	T6	-0.40 a, b, c, d	1.35
75 dB White Noise	F1	0.45 c, d, e, f	1.08
	F2	0.60 e, f	1.58
	F3	0.26 c, d, e, f	1.05
	F4	0.32 c, d, e, f	1.10
	F7	-0.05 b, c, d, e, f	1.49
	F8	0.19 b, c, d, e, f	0.90
	T3	-0.34 a, b, c, d, e	2.11
	T4	-0.76 a, b	2.56
	T5	-0.12 b, c, d, e, f	1.67
	T6	-0.01 b, c, d, e, f	1.33

Table 19

Continued

Condition	Electrode Site	Mean	Standard Deviation
90 dB White Noise	F1	0.50 d, e, f	1.10
	F2	0.44 c, d, e, f	1.08
	F3	0.52 d, e, f	0.97
	F4	0.59 e, f	1.32
	F7	0.51 d, e, f	1.86
	F8	0.44 c, d, e, f	1.25
	T3	0.65 f	3.44
	T4	0.23 b, c, d, e, f	3.25
	T5	0.47 c, d, e, f	1.75
	T6	0.47 c, d, e, f	1.74

Note. Values are reported in μV. Means with the same letter are not significantly different. Multiple comparisons based on Tukey's HSD test.

condition (T4, *M* = -1.26, *SD* = 2.84; F2, *M* = .21, *SD* = .85; F4, *M* = .39, *SD* = 1.01; F8, *M* = .01, *SD* = .91; see Figure 22) as well as greater changes in high beta magnitude than the right lateral frontal and prefrontal electrode site for the 75 dB condition (T4, *M* = -.76, *SD* = 2.56; F2, *M* = .60, *SD* = 1.58; F4, *M* = .32, *SD* = 1.10; see Figure 23). The degree of changes in high beta magnitude was also found to differ significantly at the right anterior temporal lobe between the 55 dB condition (*M* = -1.26, *SD* = 2.84) and the 90 dB condition (*M* = .23, *SD* = 3.25; see Figure 24). Additionally, the changes in high beta magnitude at the left anterior temporal lobe resulting from the 90 dB condition (*M* = .65, *SD* = 3.44) was found to differ significantly from both the 55 dB condition (*M* = -.50, *SD* = 2.65) and the 75 dB condition (*M* = -.34, *SD* = 2.11) at this electrode site (see Figure 25).

The Group x Electrode Site interaction was also found to be statistically significant, $F(9, 252) = 2.61$, $p = .007$ (see Table 20). Multiple comparisons using Tukey's HSD indicated a significant difference in the degree of changes in high beta magnitude at the right anterior temporal lobe between the Low Fluency group (*M* = -1.46, *SD* = 3.74) and the High Fluency group (*M* = .26, *SD* = 1.38). Additionally, the degree of changes in high beta magnitude at the right anterior temporal lobe electrode site (T4; *M* = 1.46, *SD* = 3.74) was significantly different than that at the right prefrontal (F2; *M* = .68, *SD* = 1.47), dorsal frontal (F8; *M* = .55, *SD* = 1.42), and the lateral frontal lobe (F4; *M* = .31, *SD* = 1.26) electrode sites, but only for the Low Fluency group. The degree of changes in high beta magnitude at the left anterior temporal lobe electrode site (T3; *M* = -.46, *SD* = 3.32) was also noted to be significantly different than at the left prefrontal electrode site (F1; *M* = .67, *SD* = 1.43), though once again only for the Low Fluency group. The degree of changes in high beta magnitude was not noted to differ significantly between any two comparisons of electrode sites for the High Fluency group.

Figure 22. Changes in high beta magnitude (μV) at the right frontal and anterior temporal lobes as a function of 55 dB white noise.

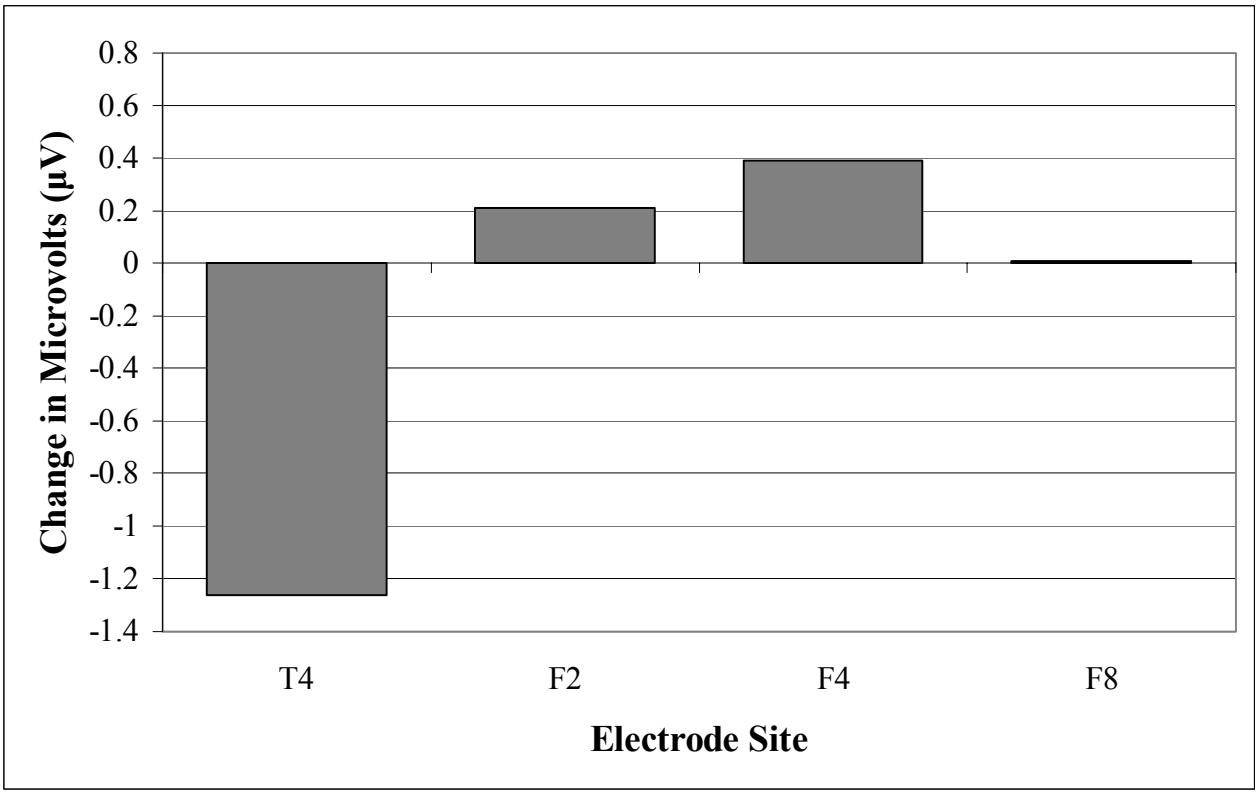


Figure 23. Changes in high beta magnitude (μV) at the right frontal and anterior temporal lobes as a function of 75 dB white noise.

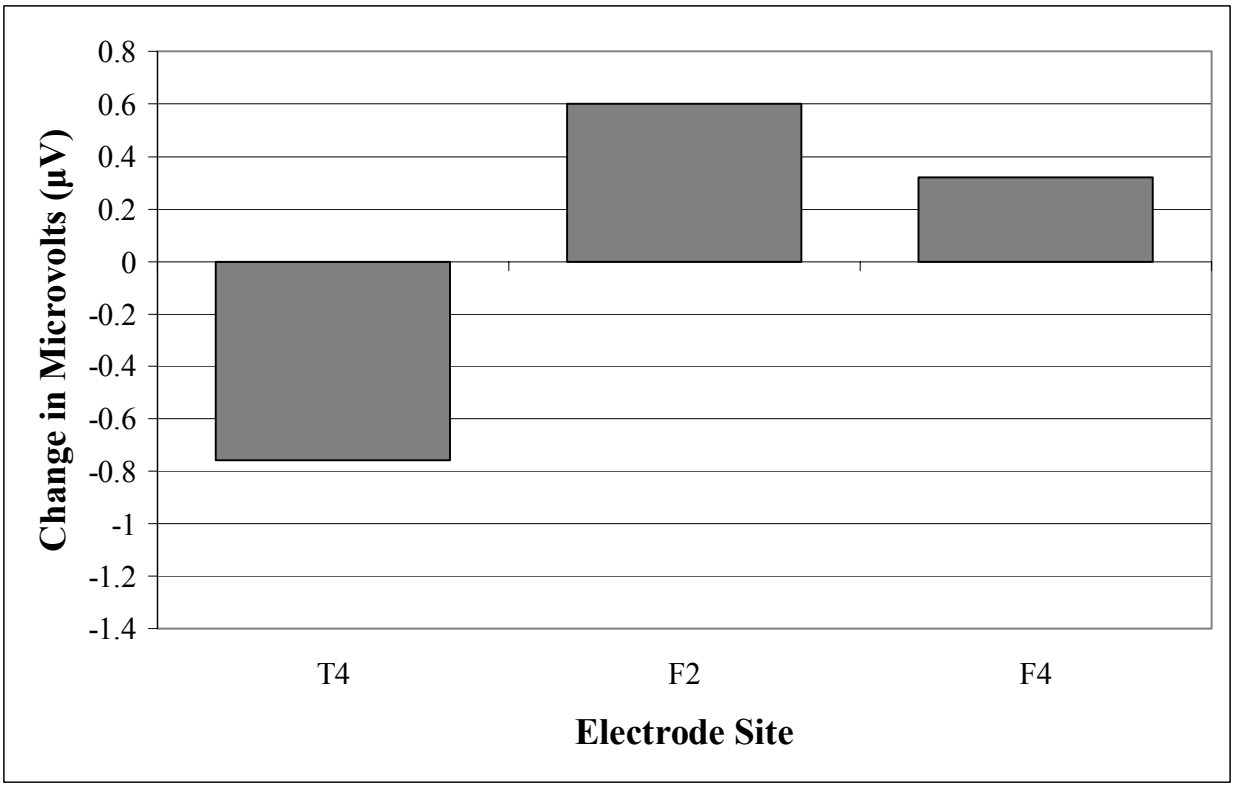


Figure 24. Changes in high beta magnitude (μV) at the right anterior temporal lobe as a function of varying white noise intensity.

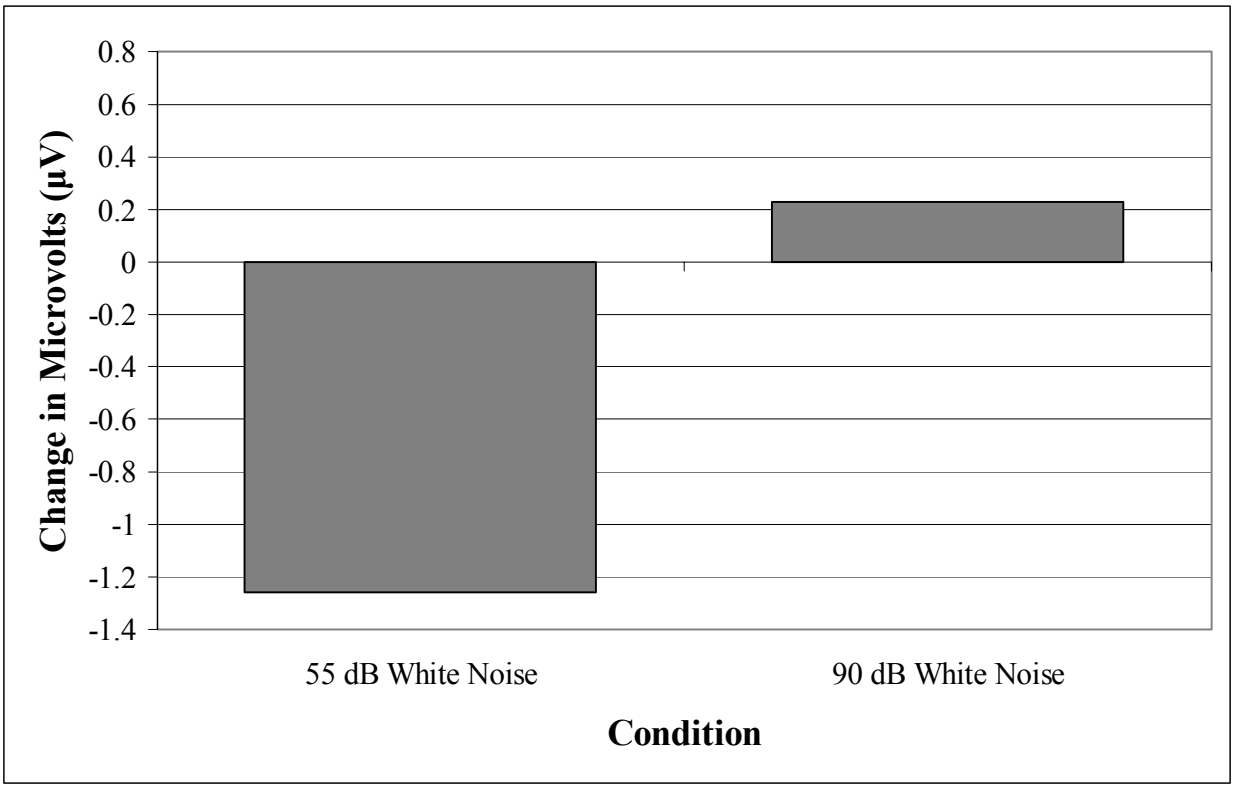


Figure 25. Changes in high beta magnitude (μV) at the left anterior temporal lobe as a function of varying white noise intensity.

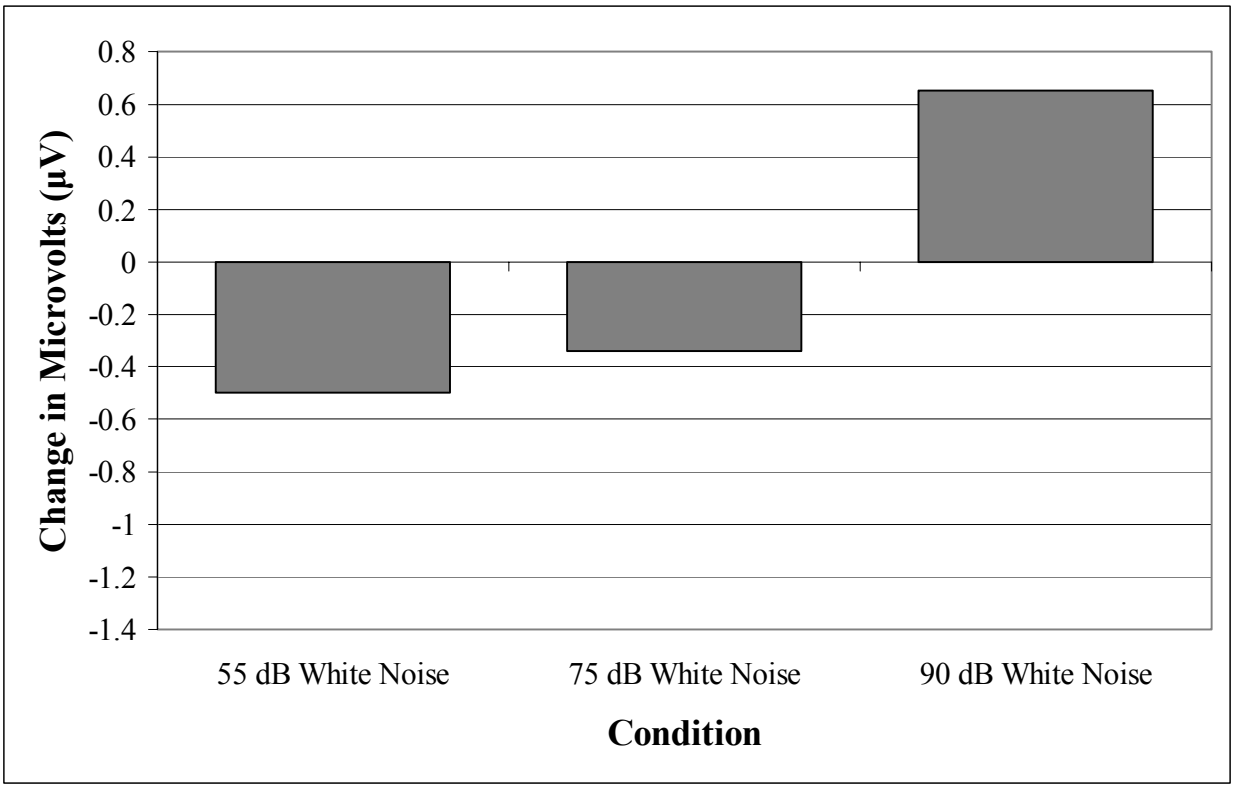


Table 20

Means and standard deviations for high beta magnitude difference scores as a function of Group and Electrode Site.

Group	Electrode Site	Mean	Standard Deviation
Low Fluency	F1	0.67 c	1.43
	F2	0.68 c	1.47
	F3	0.46 b, c	0.95
	F4	0.55 c	1.42
	F7	0.32 b, c	1.21
	F8	0.31 b, c	1.26
	T3	-0.46 a, b	3.32
	T4	-1.46 a	3.74
	T5	-0.09 b, c	1.48
	T6	-0.12 b, c	1.48

Table 20

Continued

Group	Electrode Site	Mean	Standard Deviation
High Fluency	F1	0.13 b, c	0.57
	F2	0.15 b, c	0.81
	F3	0.24 b, c	0.90
	F4	0.32 b, c	0.77
	F7	-0.09 b, c	1.87
	F8	0.12 b, c	0.76
	T3	0.34 b, c	2.13
	T4	0.26 b, c	1.38
	T5	0.12 b, c	1.91
	T6	0.16 b, c	1.55

Note. Values are reported in μV.

The main effect for Electrode Site was statistically significant, $F(9, 252) = 2.07$, $p = .03$ (see Table 21). Multiple comparisons using Tukey's HSD indicated that the changes in high beta magnitude at the right anterior temporal lobe (T4, $M = -.60$, $SD = 2.56$) were significantly different from the right prefrontal (F2, $M = .41$, $SD = 1.14$), the right lateral frontal (F4, $M = .43$, $SD = 1.10$), and the left prefrontal (F1, $M = .40$, $SD = 1.00$) electrode sites. Finally, the main effect for Condition was statistically significant, $F(2, 56) = 8.71$, $p = .0005$ (see Figure 26). Multiple comparisons using Tukey's HSD indicated that the changes in high beta magnitude resulting from the 90 dB condition ($M = .48$, $SD = 1.92$) were significantly different than both the 55 dB condition ($M = -.14$, $SD = 1.61$) and the 75 dB condition ($M = .05$, $SD = 1.55$). All other main and interaction effects were nonsignificant (see Table 22 for the ANOVA source table for high beta magnitude difference scores).

Additional analyses were performed to determine whether significant differences existed between homologous comparisons of electrode sites at the baseline and at post white noise. The results of a 2 (Group) x 4 (Condition) x 2 (Hemisphere) x 10 (Electrode Site) mixed factorial ANOVA, with the between subjects factor of Group and the repeated factors of Condition, Hemisphere, and Electrode Site indicated that no main or interaction effect was statistically significant (see Table 23 for the ANOVA source table for high beta magnitude involving the Hemisphere variable).

Discussion

The hypothesis of a main effect for Trial on RFFT scores was supported by the results of the present investigation. Specifically, as hypothesized, scores on the RFFT were found to increase significantly from the baseline or pre-white noise administration to the post-white noise administration of the RFFT. However, the results failed to support any of the hypothesized

Table 21

Means and standard deviations for high beta magnitude difference scores as a function of Electrode Site.

Electrode Site	Mean	Standard Deviation
F1	0.40 a	1.00
F2	0.41 a	1.14
F3	0.35 a, b	0.93
F4	0.43 a	1.10
F7	0.12 a, b	1.54
F8	0.22 a, b	1.01
T3	-0.06 a, b	2.73
T4	-0.60 b	2.56
T5	0.01 a, b	1.70
T6	0.02 a, b	1.52

Note. Values are reported in μV. Means with the same letter are not significantly different. Multiple comparisons based on Tukey's HSD test.

Figure 26. Changes in high beta magnitude (μV) as a function of Condition.

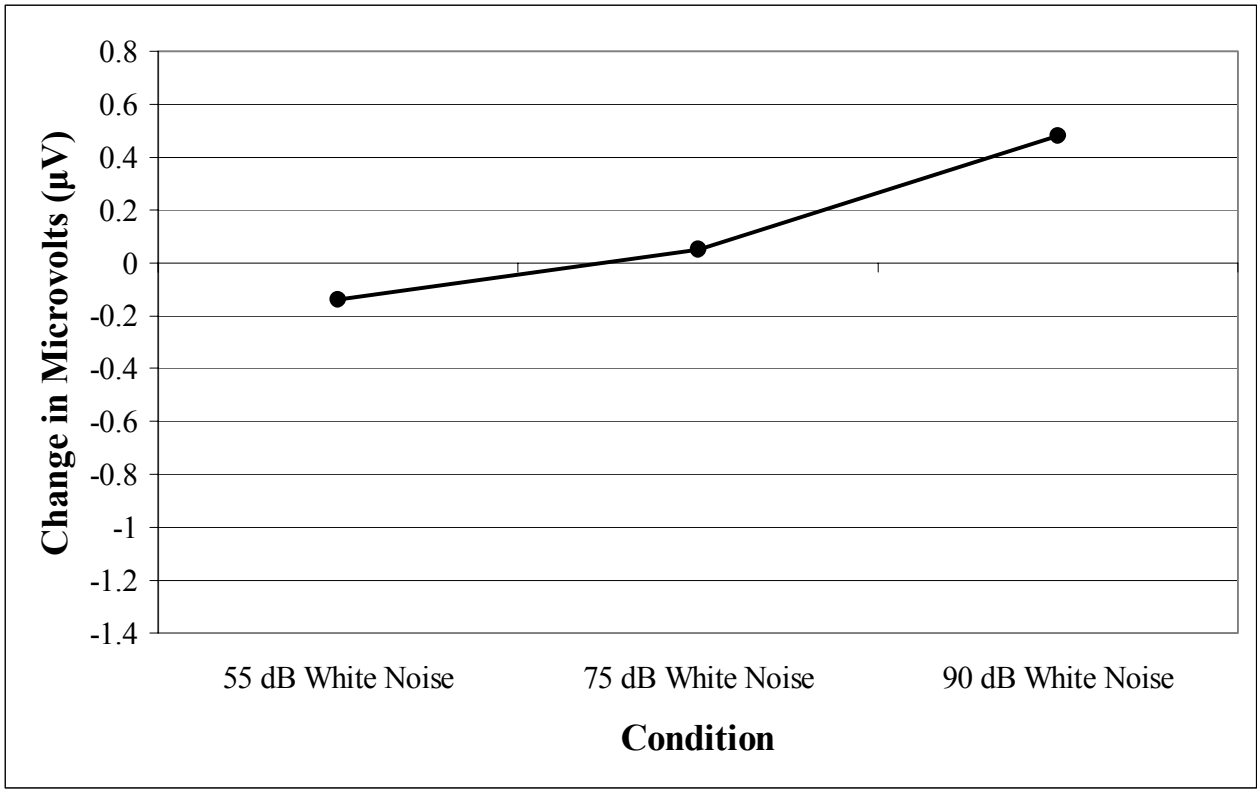


Table 22

ANOVA source table for high beta magnitude difference scores.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	1.79	1.79	0.07	.79
Condition	2, 56	61.67	30.83	8.71	.0005
Electrode Site	9, 252	80.69	8.97	2.07	.03
Group x Condition	2, 56	1.12	0.56	0.16	.85
Group x Electrode Site	9, 252	101.77	11.31	2.61	.007
Condition x Electrode Site	18, 504	33.15	1.84	1.97	.01
Group x Condition x Electrode Site	18, 504	16.99	0.94	1.01	.45

Table 23

ANOVA source table for high beta magnitude involving the Hemisphere variable.

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 28	307.55	307.55	3.19	.09
Condition	3, 84	65.48	21.83	4.92	.003
Hemisphere	1, 28	6.95	6.95	0.80	.38
Electrode Site	4, 112	255.90	63.97	3.51	.01
Group x Condition	3, 84	1.57	0.52	0.12	.95
Group x Hemisphere	1, 28	2.83	2.83	0.33	.57
Group x Electrode Site	4, 112	270.03	67.51	3.70	.007
Condition x Hemisphere	3, 84	1.36	0.45	0.38	.77
Condition x Electrode Site	12, 336	47.32	3.94	2.60	.003
Hemisphere x Electrode Site	4, 112	8.77	2.19	0.56	.69
Group x Condition x Hemisphere	3, 84	4.48	1.49	1.27	.29
Group x Condition x Electrode Site	12, 336	29.60	2.47	1.63	.08
Group x Hemisphere x Electrode Site	4, 112	3.37	0.84	0.22	.93
Condition x Hemisphere x Electrode Site	12, 336	4.64	0.39	0.96	.49
Group x Condition x Hemisphere x Electrode Site	12, 336	8.36	0.70	1.73	.06

interactions for RFFT scores. Hence, given the lack of any significant interaction effects, the data indicate that scores on the RFFT increased significantly from the pre to the post white noise presentation, irrespective of group or condition (white noise intensity level). A potential explanation for this finding is that white noise presentation, regardless of intensity level, facilitated performance on the RFFT such that performance improved after exposure to white noise. This finding is consistent with research findings of improved performance on cognitive tasks both during (Baker & Holding, 1993; Bhattacharya, Tripathi, & Kashyap, 1989; Carlson, Rama, Artchakov, & Linnankoski, 1997) and following (Harrison & Kelly, 1989) exposure to white noise. However, the likelihood that the result is due to practice effects cannot be excluded and constitutes a more plausible explanation.

Unlike the data concerning pre- and post-white noise performance on the RFFT, the cardiovascular data were not supportive of any of the hypotheses forwarded. Although the main effect for Condition on systolic blood pressure was found to be statistically significant, multiple comparisons failed to indicate significant differences between any two comparisons of the conditions. Hence, a subsequent repeated measures ANOVA was performed using only the Condition variable. The results indicated that no significant differences existed between the baseline or no white noise condition and any of the three white noise conditions. Similarly, presentation of white noise, irrespective of intensity, was not found to generate any significant changes in heart rate or diastolic blood pressure for either the Low or High Fluency groups.

The fact that significant increases in heart rate and systolic blood pressure were not found for the three white noise intensity levels is perplexing given that previous research has found that heart rate (O'Gorman & Jamieson, 1977; Raskin et al., 1967; Turpin et al., 1999) and systolic blood pressure (Martinik & Opltova, 1986) increase with increasing intensity of white noise

(Harrison & Kelly, 1989). The lack of significant findings for diastolic blood pressure may have resulted from excessive measurement error given the modest correlation (r = .52) found between the oscillometric and ausculatory methods of measuring diastolic blood pressure for the blood pressure monitor used in the present investigation (Harrison & Kelly, 1987). A further explanation for this discrepancy of findings may be the fact that the duration of white noise presentation differed between the present investigation and those investigations finding significant effects for heart rate and blood pressure. For instance, Graham and Slaby (1973) presented white noise for only a 5 second duration. Further, as was mentioned earlier, many of the investigations assessing the cardiovascular effects of white noise have done so within the context of examining orienting and defense responses (Raskin et al., 1967; Raskin et al., 1969a; Raskin et al., 1969b; Turpin et al., 1965; Uno & Grings, 1965). Hence, for those investigations, white noise was presented for only a brief amount of time, in terms of milliseconds.

The present investigation, in contrast, presented white noise for a total duration of approximately 135 seconds. This extended period of white noise presentation was both by design and by necessity. Specifically, an initial 45-second period of white noise was used as an adaptation period wherein no cardiovascular or quantitative electroencephalographic measures were obtained. However, to ensure that a sufficient number of QEEG epochs would be obtained white noise had to be presented for a sufficient amount of time, in the present case for 45 seconds. Further, white noise continued through the measurement of heart rate and blood pressure, which required an additional 45 seconds or so. Citing research demonstrating that noise fails to induce sustained increases in autonomic activity and that adaptation to noise occurs within minutes, Jennings (1986) expressed doubt as to whether noise is capable of inducing sustained arousal, such as changes in autonomic activity. Thus, the possibility exists that the

initial cardiovascular effects of white noise were either not detected or dissipated since white noise had been presented for 90 seconds before measures of heart rate and blood pressure were obtained.

The possibility of habituation occurring to the extended exposure of white noise presentation may explain the dissipation of cardiovascular effects. Research has indicated that repeated exposure to white noise results in habituation of both heart rate (Turpin & Siddle, 1978) and blood pressure (Linden, Frankish, & McEachern, 1985). Turpin, Schaefer, and Boucsein (1999) found that the initial increases in heart rate resulting from exposure to 100 dB white noise returned to baseline levels within approximately 8 seconds. Given these findings, the possibility certainly exists that the initial effects of white noise presentation on cardiovascular functioning in the current investigation dissipated as a result of habituation, resulting in no significant changes in heart rate or blood pressure being detected.

However, to investigate fully the reasons why changes in cardiovascular functioning did not differ as a function of performance on the RFFT and/or white noise intensity the QEEG data must be considered. Similar findings with the QEEG data may aid in explaining the results from the cardiovascular data, especially if those regions of the brain involved in regulating heart rate and blood pressure were inconsistently affected by varying intensity of white noise presentation. For instance, the QEEG data may indicate that white noise presentation did not result in significant changes in cerebral activity at the right frontal and/or temporal lobes.

As with the cardiovascular data, the QEEG data were generally not supportive of the hypotheses forwarded. Although the data for low beta magnitude failed to support any of the hypotheses forwarded, the data for high beta magnitude indicated a significant interaction between Group, Condition, and Electrode Site. Consistent with the hypothesis forwarded,

presentations of 55 dB and 75 dB white noise resulted in significant reductions in high beta magnitude at the right anterior temporal lobe for the Low Fluency group, as compared to the High Fluency group. Analysis of the difference scores indicated that significant group effects also emerged with respect to the degree of changes in high beta magnitude resulting from white noise presentation. Specifically, white noise presentation resulted in a significant difference between the Low and High Fluency groups in terms of the degree of changes in high beta magnitude at the right anterior temporal lobe. Further, white noise presentation also resulted in a significant difference between the right anterior temporal and right lateral frontal sites in terms of the degree of change in high beta magnitude, but only for the Low Fluency group. However, though differences emerged between the Low and High Fluency groups in terms of significant changes in high beta magnitude as a result of white noise presentation, the differences were not always significant or in the hypothesized location or direction. For instance, white noise presentation of any intensity level failed to generate significant changes in high beta magnitude at the right lateral frontal lobe for either the Low or the High Fluency group. Further, none of the three white noise presentations generated significant changes in high beta magnitude at the right anterior temporal lobe for the High Fluency group. The effect of varying intensity of white noise on high beta magnitude also did not conform to the hypothesis given that presentation of 90 dB white noise failed to generate significant changes in high beta magnitude. Additionally, although significant group effects emerged with respect to the degree of changes in high beta magnitude at the right anterior temporal lobe and between the right anterior temporal lobe and the right lateral frontal lobe, the differences did not vary as a function of white noise intensity.

The results also indicated a significant interaction between Condition and Electrode Site, as hypothesized. Consistent with the hypothesis forwarded, presentation of white noise resulted

in a significant reduction in high beta magnitude at the right anterior temporal lobe. Analysis of the difference scores also indicated that, as hypothesized, presentations of both 55 dB and 75 dB white noise generated greater changes in high beta magnitude at the right anterior temporal lobe as compared to the right lateral frontal lobe, which actually evidenced an increase in high beta magnitude. Further analysis of the difference scores indicated a significant difference in the degree of changes in high beta magnitude between presentations of 55 dB and 90 dB white noise. However, the changes in high beta magnitude were not always in the hypothesized location or direction. For instance, contrary to the hypothesis, no significant reduction in high beta magnitude was noted at the right lateral frontal lobe for any of the three white noise presentations. Additionally, presentation of 90 dB white noise did not generate a significant difference in high beta magnitude between the right anterior temporal and right lateral frontal lobes. The effect of varying intensity of white noise on high beta magnitude also did not conform to the hypothesis, given that the only significant difference in high beta magnitude found to exist between the three white noise intensity levels was between the 55 dB and 90 dB white noise presentations. Indeed, the effects of varying white noise intensity were actually in the opposite direction of that hypothesized. Specifically, whereas presentation of 55 dB white noise generated a significant reduction in high beta magnitude at the right anterior temporal lobe, presentations of 75 dB and 90 dB white noise resulted in a nonsignificant reduction and increase in high beta magnitude, respectively. These results are potentially reminiscent of traditional arousal theories that hypothesize curvilinear changes resulting from heightened arousal. Such a finding may implicate the contributions of cerebral laterality with regard to right hemisphere systems contribution to sympathetic tone.

Finally, as hypothesized, the main effect for Condition was significant. Consistent with the hypothesis, analysis of the difference scores indicated that presentation of 90 dB white noise resulted in a significant difference in the degree of changes in high beta magnitude than both 55 dB and 75 dB white noise presentations. However, once again, the direction of the changes was not in the hypothesized direction given that presentations of both 90 dB and 75 dB white noise generated increases in high beta magnitude and presentation of 55 dB white noise resulted in a decrease in high beta magnitude. Indeed, although the main effect for Condition was significant, multiple comparisons indicated that none of the three white noise intensity levels generated significant changes in high beta magnitude from the baseline or no white noise condition.

As with the cardiovascular data, the quantitative electroencephalographic data generally do not correspond well with previous findings. More specifically, previous research has found a relationship between intensity of white noise and increasing amplitude of the P50 component of the event related potential at the CZ electrode site (Roth et al., 1984). Although the present investigation used quantitative electroencephalography as opposed to event related potentials, a similar relationship may be expected to exist. However, no significant incremental increases were found in the present investigation. This discrepancy may be partially explained by the fact the present investigation measured QEEG at the frontal and temporal regions, unlike the Roth et al. (1984) investigation which used a central vertex location. Inconsistencies also emerge with respect to the overall effects of white noise on changes in low and high beta magnitude. For instance, whereas research has found increased low beta power at the left and right temporal lobes (Nicholls et al., 1999), the present investigation found no significant changes in low beta magnitude at the right anterior temporal lobe. Previous research has also found significant increases in high beta amplitude at the temporal lobes (Giannitrapani, 1970). However, the

present investigation found significant reductions in high beta magnitude at the right temporal lobe. Furthermore, whereas presentation of white noise has been found to generate significant increases in high beta power at the frontal lobes (Schellenberg et al., 1989), the present investigation found no significant changes in high beta magnitude at the right lateral frontal lobe.

The lack of consistent findings between previous research and the present investigation may once again be a result of the use of a longer period of white noise presentation in the present investigation. Many of the previous investigations used white noise presentation of a very brief duration, in terms of milliseconds (Nicholls et al., 1999; Roth et al., 1984). Additionally, the decibel levels were not consistent across investigations, with the present investigation using levels of 55 dB, 75 dB, and 90 dB and none of the previous investigations using these same levels of intensity. Thus, generally the methods used across investigations have not corresponded well. Hence, it is not surprising that the results have not been consistent either.

The discrepancy between the present findings and previous research as well as the general lack of support for the hypotheses forwarded may also be due to habituation. Research has found that the amplitude of the P300 component of the event related potential habituates to repeated presentations of white noise (Hirano, Russell, Ornitz, & Liu, 1996). Thus, as with the cardiovascular data, the possibility exists that, as a result of habituation to the white noise, the initial effects of white noise presentation on cerebral activity largely dissipated by the time measures of cerebral activity were obtained.

Although the findings of the present investigation were not consistent with previous research and generally not supportive of the hypotheses, the degree of correspondence between the cardiovascular and the QEEG data is of more critical importance given the purpose of this investigation. The essential question is whether the QEEG data both support and help to explain

the cardiovascular data given the model being tested. Support for the model being tested would be provided if the QEEG data indicate that presentation of white noise resulted in patterns of cerebral activity across the right anterior temporal and right lateral frontal lobes that would generate no significant changes in heart rate and blood pressure. As an example, given that, according to the model being tested, the right frontal lobe is associated with inhibition of and the right temporal with excitation of the sympathetic nervous system, equivalent levels of cerebral activity within these regions of the brain would result in no significant changes in heart rate or blood pressure. Additionally, equivalent levels of cerebral activity at these regions of the brain across both the Low and High Fluency groups and the three white noise intensity levels would result in not only no significant changes in heart rate or blood pressure but also no significant differences or interactions between these groups or conditions.

However, as mentioned previously, the data indicated that presentation of 55 dB and 75 dB white noise resulted in significant reductions in high beta magnitude at the right anterior temporal lobe, but only for the Low Fluency group. Further, across the Low and High Fluency groups, 55 dB and 75 dB white noise presentations generated reduction in high beta magnitude at the right anterior temporal lobe, relative to the increased high beta magnitude at the right lateral frontal lobe. Hence, presentation of 55 dB and 75 dB white noise resulted in relative heightened sympathetic influences on heart rate and blood pressure for the Low Fluency group. Given this overall pattern of cerebral activity, significant increases in heart rate and blood pressure would be expected for the Low Fluency group, with either no changes or slight increases in heart rate and blood pressure for the High Fluency group. This expectation, though, is in direct contrast to the results, which indicated no significant changes in heart rate and blood pressure for either the Low or the High Fluency groups. Thus, at first glance the QEEG data do not lend any support to

the cardiovascular data. Further, given the lack of correspondence the cardiovascular and QEEG data, the results do not provide support for the model being tested.

A fundamental problem, though, with the interpretation of the findings as discussed thus far is that the influence of the left temporal and frontal lobes is not taken into consideration. This problem becomes particularly salient when considering that research has indicated that a relationship exists between the magnitude of cerebral asymmetry at the frontal lobes and baseline measures of cardiovascular functioning (Foster & Harrison, 2002b). The aforementioned interpretation of the data has essentially ignored the influence of the left hemisphere, and hence the parasympathetic nervous system, on heart rate and blood pressure. Clearly, given the critical and just as influential role of the left hemisphere in regulating heart rate and blood pressure, as indicated by research demonstrating left hemisphere involvement in the regulation of cardiovascular activity (Lane et al., 1988; Yoon et al., 1997; Zamrini et al., 1990), any analyses of the data should include the left temporal and frontal lobes. Further, given that white noise was presented bilaterally, changes in high beta magnitude would also have occurred at the left temporal and frontal lobes. Hence, with the methods used in this investigation, both sympathetic and parasympathetic influences on heart rate and blood pressure likely existed.

An additional, albeit related, problem with the aforementioned interpretation of the findings is that it is not entirely consistent with a functional cerebral systems perspective of the cerebral regulation of heart rate and blood pressure. A functional cerebral systems perspective of the cerebral regulation of cardiovascular functioning, by definition, necessitates inclusion of all areas of the cerebrum involved in regulating heart rate and blood pressure. A functional cerebral systems perspective is consistent with research findings indicating that numerous areas across the left and right frontal and temporal lobes are associated with changes in cardiovascular

functioning. For instance, as was previously discussed, lesions localized within the left (Oppenheimer et al., 1996) and right (Tokgozoglu et al., 1999) insula result in significant changes in cardiovascular functioning. The right (Demaree & Harrison, 1997a; Rush et al., 1977) and left (Foster & Harrison, 2002a) frontal lobes have also been associated with changes in heart rate and blood pressure. Several reviews of the literature have indicated that the frontal lobes, prefrontal cortex, temporal lobes, and insular cortices are all associated with changes in cardiovascular functioning (Cechetto & Saper, 1990; Delgado, 1960; Hoff et al., 1963; Neafsey, 1990; Verberne & Owens, 1998).

The functional cerebral systems model of cardiovascular functioning tested in the present investigation proposes that all of the aforementioned areas of the cerebrum are involved in the regulation of heart rate and blood pressure. Specifically, the model proposes that the left and right frontal lobes are differentially involved in inhibiting parasympathetic and sympathetic influences on heart rate and blood pressure, respectively. Additionally, the left and right temporal lobes are excitatory in nature, with regard to parasympathetic and sympathetic influences on heart rate and blood pressure, respectively. The model also proposes that incremental increases or decreases in the magnitude of cerebral activity will result in corresponding incremental changes in heart rate and blood pressure, depending on the location of such changes in cerebral activity. Differences in the extent of spreading activation across homologous regions of the cerebrum, in addition to differences in magnitude of activation, will also mediate changes in cardiovascular functioning. Finally, the relative differential activation of the left and right frontal and temporal lobes will ultimately determine whether heart rate and blood pressure increase or decrease as well as the magnitude of such changes.

Given the problems inherent in focusing the analyses solely on the right lateral frontal and anterior temporal lobes, a more complete and accurate interpretation of the findings would include all electrodes sites within the left and right temporal and frontal lobes. Hence, the data were reanalyzed with the expectation that correspondence would be found between the cardiovascular and QEEG data once all electrodes sites were included in the data. Thus, these unplanned post-hoc analyses focused on the location and magnitude of significant changes in low and high beta magnitude within the bilateral frontal and temporal lobes. Additional analyses were performed on the differences in the degree of changes in low and high beta magnitude between homologous comparisons of electrode sites as well. Post-hoc analyses were also performed on the differences in low and high beta magnitude between homologous comparisons of electrode sites at the baseline and during presentation of white noise presentation. The intention of this last set of analyses was to obtain evidence regarding whether a shift occurred from parasympathetic predominance to sympathetic predominance or visa versa.

Contrary to the results of the original analyses, post-hoc analyses revealed significant effects of white noise presentation on low beta magnitude. As may be seen in Figures 4 through 6, whereas presentation of 75 dB and 90 dB white noise generated no significant changes in low beta magnitude, presentation of 55 dB white noise resulted in a significant reduction in low beta magnitude at the right posterior temporal lobe. However, no differences between the Low and High Fluency groups were found. Analysis of the difference scores indicated that differences in the magnitude of change between the right frontal and right posterior temporal lobe were also found. Specifically, significant differences were found in the magnitude of change between the right posterior temporal and all three right frontal sites for presentations of both 55 dB and 75 dB white noise. Similar differences were also found between the left posterior temporal and frontal

lobes in that significant differences were found in the magnitude of change between the left posterior temporal and left lateral frontal lobes for presentation of both 55 dB and 75 dB white noise. Significant differences in the degree of changes in low beta magnitude was also noted between presentations of 55 dB and 75 dB white noise at the right posterior temporal lobe. Once again, though, no differences emerged between the Low and High Fluency groups in terms of differences in the degree of change in low beta magnitude. Further, it is important to note, for reasons to be discussed shortly, that analysis of the difference scores also indicated that no significant differences existed in the magnitude of change in low beta between any two homologous comparisons of electrode sites. Additionally, whereas reduced low beta magnitude was found over the left hemisphere relative to the right hemisphere, low beta magnitude at the baseline and during white noise presentation did not vary as a function of either group, white noise intensity, or location.

The results of analyses for high beta magnitude were generally the same as before. Specifically, as may be seen in Figures 15 through 17, presentation of 55 dB and 75 dB white noise generated significant reductions in high beta magnitude at the right anterior temporal lobe, but only for the Low Fluency group and at no other electrode site. As before, presentation of 90 dB white noise failed to generate significant changes in high beta magnitude at any electrode site for either the Low or High Fluency group. Thus, no significant changes in high beta magnitude were found at any electrode site across the left frontal and temporal lobes. However, analysis of the difference scores indicated a significant difference in the magnitude of change in high beta resulting from 90 dB white noise and that resulting from both 55 dB and 75 dB white noise at the left anterior temporal lobe. Further, as with the data for low beta magnitude, analysis of the difference scores indicated that no significant differences in magnitude of change in high beta

existed between any two homologous comparisons of electrode sites. Further, the results of analyses conducted to determine whether differences existed between homologous comparisons of electrode sites at the baseline and during white noise indicated that no differences in high beta magnitude existed at either the baseline or during white noise for any group, white noise intensity, or location.

Collectively, this reanalysis of the data indicated that presentation of 55 dB and 75 dB white noise generated significant reductions in high beta magnitude at the right anterior temporal lobe, and hence increased sympathetic influence on heart rate and blood pressure. Additionally, the differences in the magnitude of changes in high beta magnitude resulting from 55 dB and 75 dB white noise presentation significantly differed between not only the right anterior temporal lobe and the right frontal lobe but also between the left posterior temporal and left frontal lobes. Thus, when initially considering this pattern of cerebral activity, significant increases in heart rate and blood pressure would be expected for the Low Fluency group. This expectation, though, is once again in direct contrast with the results, which found no significant changes in either heart rate or blood pressure. Hence, yet again, the results do not support correspondence existing between the cardiovascular and the QEEG data, thereby providing no support for the model being tested.

However, it is of critical importance to remember that no significant differences were noted between homologous comparisons of electrode sites in terms of either the degree of changes in low or high beta magnitude or the magnitude of low or high beta at baseline and during white noise presentation. Thus, whereas significant reductions in high beta magnitude were found at the right anterior temporal lobe for both 55 dB and 75 dB white noise presentation, these changes in high beta magnitude did not differ significantly from those observed at the left

temporal lobe. As a result, equivalent influences from the left and right temporal lobes, and hence the parasympathetic and sympathetic nervous systems, existed on cardiovascular activity. Given this critical finding, the expectation would be that no significant changes in heart rate and blood pressure would result from presentation of white noise of any intensity or for any group. Further, this expectation is entirely consistent with the results of the present investigation. Hence, the data provide both correspondence between the cardiovascular and QEEG data as well as partial support for the model being tested.

Although the aforementioned reinterpretation of the data provides initial partial support for the model by demonstrating correspondence between the QEEG and cardiovascular data, at least four problems exist regarding the reinterpretation. First, the reinterpretation of the data is not represented in the hypotheses that were being tested in this investigation. Changes in low and high beta magnitude within the left frontal and temporal lobes were not included in the hypotheses. Indeed, the hypotheses were restricted to the right lateral frontal and right anterior temporal lobes, hence other areas within the frontal and temporal lobes were also excluded. As a result, the findings as reviewed in the reinterpretation were not part of statistical analyses as originally planned. Given the lack of correspondence between the hypotheses as originally conceived and the reinterpretation of the data, the reinterpretation should be circumspectively evaluated.

However, a potentially more serious problem is that the reinterpretation of the data, and the model being tested in general, is predicated on the hemispheric lateralization of the parasympathetic and sympathetic nervous systems to the left and right hemispheres, respectively. Hence, research findings contrary to this supposition represent potential problems with not only the reinterpretation of the data but also the model being tested. Not all research findings are

consistent with the view of hemispheric lateralization of the parasympathetic and sympathetic nervous systems. Wittling (1990) presented data suggesting that whereas presentation of an emotional film to the right hemisphere generated increases in blood pressure, presentation of the same film to the left hemisphere either produced no significant changes in blood pressure or resulted in a reduction of blood pressure. However, this pattern of results was not found for all participants. Indeed, increases in systolic blood pressure following right hemisphere presentation and decreases or not changes in systolic blood pressure following left hemisphere presentation were found for less than half of the participants. Further, close to one-quarter of those in the left hemisphere presentation condition were found to exhibit increases in systolic blood pressure.

Other researchers have also presented data that suggests hemispheric lateralization of the parasympathetic and sympathetic nervous systems, but with nonsignificant results (Oppenheimer et al., 1996; Yoon et al., 1997). Further, Naver et al. (1996) presented data that were contradictory to the notion of hemispheric lateralization of the parasympathetic and sympathetic nervous systems. Specifically, Naver et al. (1996) found that strokes localized to the right hemisphere were associated with reduced heart rate variability, as compared to strokes localized to the left hemisphere. Ahern et al. (2001) found that intracarotid sodium amobarbital injections into either hemisphere resulted in increased heart rate. Thus, although the preponderance of findings supports the lateralization of the parasympathetic and sympathetic nervous systems to the left and right hemispheres, respectively (Andersson & Finset, 1998; Barron et al., 1994; Hugdahl et al., 1983; Lane et al., 1988; Oppenheimer et al., 1992; Wittling, 1995; Wittling, 1997; Wittling, Block, Genzel et al., 1998; Wittling, Block, Schweiger et al., 1998; Zamrini et al., 1990), the findings to the contrary represent obstacles that must be considered.

An additional problem is that changes in low and high beta magnitude at areas other than the frontal and temporal lobes were not measured. Research has indicated that other areas of the cerebrum are also associated with changes in cardiovascular activity. For instance, Foster and Harrison (2002a) found that changes in heart rate were significantly correlated with changes in high beta magnitude at the left and right parietal lobes and the parietal vertex. Similarly, Foster and Harrison (2004) found that changes in diastolic blood pressure were significantly correlated with changes in alpha and low beta magnitude at the bilateral parietal lobes and the parietal vertex. This investigation also found significant correlations between changes in systolic blood pressure and changes in low beta magnitude at the bilateral parietal lobes. Another investigation by Foster, Thompson, and Harrison (2004b) also found that changes in heart rate were significantly correlated with changes in high beta magnitude at the bilateral parietal lobes. Further, an investigation by Foster and Harrison (2002b) found that baseline systolic blood pressure was significantly correlated with resting magnitude of cerebral asymmetry, as measured by high beta magnitude asymmetry scores obtained by subtracting the high beta magnitude of right hemisphere electrodes from that of the homologous left hemisphere electrodes, at the occipital lobes. This investigation also found that baseline heart rate was significantly correlated with low beta magnitude asymmetry at the parietal lobes. Hence, these findings support the notion that other areas of the cerebral cortex may be involved in regulating heart rate and blood pressure. Given that the present investigation did not obtain measurements of cerebral activity at either the parietal or occipital lobes, the possibility exists that undetected influences on heart rate and blood pressure also existed from these additional regions of the cerebrum.

A final problem with the reinterpretation of the data, and the findings in general, concerns the fact that significant differences in heart rate and blood pressure responses to white noise

stimulation were not found between the Low and High Fluency groups, as defined by performance on the RFFT. Research has found that performance on the RFFT is sensitive to head injuries (Ruff et al., 1986) as well as frontal lobe lesions (Baldo et al., 2001), particularly lesions or dysfunction localized to the right frontal lobe (Ruff et al., 1994). Further, a recent investigation by Foster, Beck, and Harrison (2003) indicated that individuals who performed more poorly on the RFFT were characterized by heightened delta activity at the right frontal lobe, relative to those who perform well on the RFFT. Hence, given that performance on the RFFT is sensitive to right frontal lobe dysfunction, the Low Fluency group would be expected to possess relative right frontal dysfunction and hence right temporal dysregulation or an inability to inhibit activity within the right temporal lobe. As a result, heart rate and blood pressure responses to white noise presentation would be expected to be greater among the Low Fluency group as compared to the High Fluency group. However, as mentioned previously, no significant group effects were found.

The lack of significant group effects may have been the result of several factors, such as the possibility that individuals perform well on the RFFT but yet possess right frontal dysfunction. However, once again, a more plausible explanation concerns the fact that the influence of the left hemisphere was not assessed. Specifically, although the Low Fluency group performed poorly on the RFFT, suggesting right frontal lobe dysfunction, the question arises as to whether this was *relative* dysfunction. The left frontal lobe may have been equally or perhaps even more dysfunctional that the right frontal lobe. Hence, these individuals would have been classified as Low Fluency in the present investigation, but yet possess relative left frontal dysfunction. This would have resulted in relative disinhibition or dysregulation of the left temporal lobe, and hence parasympathetic activity, as opposed to the right temporal lobe, and

hence sympathetic activity. As a result, the data may have been washed out since the Low Fluency group may have been comprised of not only individuals whose heart rate and blood pressure increased, as hypothesized, but also individuals whose heart rate and blood pressure decreased.

Notwithstanding the aforementioned limitations and the fact that the findings do not support some of the hypotheses forwarded, the findings are generally consistent with the model being tested in that the QEEG and cardiovascular data were found to correspond. Support for the hypotheses concerning the effects of white noise intensity on changes in cardiovascular functioning as well as low and high beta magnitude may not have been as critical as consistency between the cardiovascular and QEEG data. That being said, though, it is quite difficult, perhaps even erroneous, to attempt to provide support for a model through null findings. Many aspects of the investigation could have been altered or improved to provide not only a more direct and better test of the model but also stronger support. For instance, the present investigation used binaural presentation of white noise, but yet concentrated the analyses on the right frontal and temporal lobes. The results may have provided more direct support for the model had white noise been presented to each ear individually and cerebral activity measured at electrode sites not only at the frontal and temporal lobes but also at the parietal and occipital lobes. Additionally, the methods would have been improved with the assessment of relative left and right frontal lobe dysfunction. These changes to the methods would provide not only a more direct test of the model but would also be more consistent with a functional cerebral systems perspective of the cerebral regulation of cardiovascular functioning.

The impact of using binaural presentation of white noise on the findings of the present investigation becomes particularly salient when considering that this is the major difference

between the methods used in this investigation and those used successfully in our lab to investigate the neuropsychophysiological effects of hostility. Numerous investigations have been conducted in our lab demonstrating the effects of right frontal lobe disinhibition of posterior cerebral systems on cardiovascular activity (Demaree, 1995; Demaree & Harrison, 1997a; Demaree et al., 2000; Williamson & Harrison, 2003). Whereas the present investigation used performance on the RFFT, our lab has generally used the Cook Medley Hostility Scale (CMHS, Cook & Medley, 1954) to assess right frontal lobe functioning. However, both the RFFT (Ruff et al., 1986) and the CMHS (Demaree & Harrison, 1996; Everhart & Harrison, 1995) are known to be sensitive to right frontal lobe functioning. The primary difference involves the stressor or stimulus used to generate changes in cerebral activity. Whereas the methods used in our lab result in relatively heightened right hemisphere activity through placing the left hand of the participants into an ice-water bath, the present investigation used binaural presentation of white noise, which likely resulted in bilateral changes in cerebral activity. This methodological difference may explain the disparity in findings between the present investigation and the numerous investigations of our lab that have used the cold-presser task. Specifically, contrary to the present findings, our lab has consistently found that, as compared to low hostile individuals, high hostile individuals evidence significantly greater heart rate (Demaree, 1995; Demaree & Harrison, 1997a; Demaree et al., 2000) and systolic blood pressure (Demaree et al., 2000) in response to the cold-presser task applied to the left hand. Thus, the results of the present investigation may have provided more direct support for the model being tested had a more lateralizing stressor been used, such as the cold-presser task. Further, to assess that aspect of the model addressing the effects of varying magnitude of cerebral activity, the temperature of the ice-water bath may be manipulated.

Given the aforementioned limitations, improvements or even significant alterations to the present methods may have provided a more direct test of the model being tested. Discussing and providing explanations for findings that do not support the hypotheses tested is quite difficult, certainly much more difficult than discussing findings that support the hypotheses tested. As mentioned earlier, although the post-hoc analyses conducted in the present investigation indicate correspondence between the cardiovascular and QEEG data, such analyses are potentially erroneous and should be circumspectively evaluated. The lack of support for the hypotheses in the present investigation may have resulted from problems with the validity of the model being tested, from methodological limitations, or a combination of the two. However, it is felt that the aforementioned methodological limitations are likely the reason for the present findings and lack of support for the hypotheses, especially given the support for the model being tested based on the literature review and integration. It is hoped that, based on the problems identified in the discussion, the present methodological limitations may be improved or corrected in future investigations of the model and that through revised and improved methodology the model may receive more direct testing and support.

Perhaps the greatest implication of the present investigation, particularly given the reinterpretation of the data, is that it demonstrates the importance of considering and investigating the cerebral mediation of cardiovascular functioning from a functional cerebral systems perspective. As originally conceived, the hypotheses and analyses only concerned the right frontal and temporal lobes. However, as was evident, considering only the right hemisphere in interpreting the data neglected the influence of the left hemisphere. Consistency between the cardiovascular and QEEG data emerged only when the left and right frontal and temporal lobes were included in the analyses and interpretation. This underlies the importance

of analyzing the contributions of each area of the brain in mediating cardiovascular functioning and the fact that analyzing only a single area or anything less than the entire brain is incomplete and will not likely yield a full understanding of the relationship between cerebral and cardiovascular functioning.

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Appendix A

Coren, Porac, and Duncan (CPD) Laterality Questionnaire

Circle the appropriate number after each item:	Right	Left	Both
With which hand would you throw a ball to hit a target?	1	-1	0
With which hand do you draw?	1	-1	0
With which hand do you use an eraser on paper?	1	-1	0
With which hand do you remove the top card when dealing?	1	-1	0
With which foot do you kick a ball?	1	-1	0
If you wanted to pick up a pebble with your toes, which foot would you use?	1	-1	0
If you had to step up onto a chair, which foot would you place on the chair first?	1	-1	0
Which eye would you use to peep through a keyhole?	1	-1	0
If you had to look into a dark bottle to see how full it was, which eye would you use?	1	-1	0
Which eye would you use to sight down a rifle?	1	-1	0
If you wanted to listen to a conversation going on behind a closed door, which ear would you place against the door?	1	-1	0
If you wanted to listen to someone's heartbeat, which ear would you place against their chest?	1	-1	0
Into which ear would you place the earphone of a transistor radio?	1	-1	0

# of Right + # of Left = Total Score

If mother left or right hand dominant? _____ _____ + _____ = _____

Is father left or right hand dominant? _____

Appendix B

Head Injury/Medical History Inventory (HIMHI)

Name: _____ Birth date: _____

Have you ever experienced or been diagnosed with any of the following, or are you experiencing of the following at present? Please circle the appropriate response and explain any "Yes" responses below.

1.	Severe head trauma or injury?	Yes	No
2.	Stroke or aneurysm?	Yes	No
3.	Learning disabilities (problems with reading, writing, or comprehension)?	Yes	No
4.	Epilepsy or seizures?	Yes	No
5.	Paralysis?	Yes	No
6.	Neurological surgery?	Yes	No
7.	Other neurological or nervous system problems?	Yes	No
8.	Alcohol or drug problems?	Yes	No
9.	Are you using alcohol or drugs (other than for prescribed purposes) at present?	Yes	No
10.	Past psychological/psychiatric problems?	Yes	No
11.	Are you currently taking any prescription medications?	Yes	No
12.	Are you currently suffering from any medical conditions or illnesses?	Yes	No
13.	Do you have any uncorrected vision problems?	Yes	No
14.	Do you have an uncorrected hearing impairment?	Yes	No
15.	Do you have any problems or pain with movement (e.g., severe hand, arm, or shoulder pain with movement)?	Yes	No

Please explain any "Yes" responses above:

Appendix C

Graded Cerebral Activation to Noise:
Behavioral and Cardiovascular Effects

Justification

Research has indicated that the frontal and temporal lobes are involved in the mediation of heart rate and blood pressure. For instance, stimulation of the frontal lobes through transcranial magnetic stimulation has been found to result in significant changes in heart rate and blood pressure. Further, stimulation of the medial prefrontal area has elicited bradycardia and depressor responses. The finding that stimulation of the medial prefrontal area elicits bradycardia and depressor responses suggests that the frontal lobes are inhibitory relative to the cerebral mediation of heart rate and blood pressure. Conversely, the temporal regions are known to be excitatory in nature. More specifically, stimulation of the temporal lobes, and in particular the insular cortices, has been found to generate significant increases in heart rate and blood pressure. Thus, the frontal and temporal lobes possess different roles in the regulation of these cardiovascular measures. However, the specific cardiovascular effects of stimulating these regions of the brain depend on which cerebral hemisphere is stimulated due to the hemispheric lateralization of the sympathetic and parasympathetic nervous systems.

Research has indicated that the sympathetic and parasympathetic nervous systems are lateralized to the right and left cerebral hemispheres, respectively. For instance, whereas stimulation of the right insular cortex has resulted in tachycardia and pressor responses, stimulation of the left insular cortex has resulted in bradycardia and depressor responses. Furthermore, research has indicated that presentation of emotional films to the right hemisphere has resulted in significant increases in heart rate and blood pressure while presentation to the left hemisphere has resulted in significant decreases in heart rate and blood pressure. Support for the lateralization of the sympathetic and parasympathetic nervous systems is also provided by the results of investigations assessing the cardiovascular effects of unilateral hemispheric inactivation through intracarotid sodium amobarbital injections. Specifically, increases and decreases in heart rate and blood pressure have resulted from left and right hemisphere inactivation, respectively.

Integrating the aforementioned research findings suggests that stimulation of the right frontal lobe will result in inhibition of the sympathetic nervous system whereas stimulation of the left frontal lobe will result in inhibition of the parasympathetic nervous system. Additionally, while stimulation of the right insular cortex or temporal lobe will result in sympathetic excitation, stimulation of the left insular cortex or temporal lobe will result in parasympathetic excitation. However, the specific effects of stimulating each of these regions of the brain are dependent on the magnitude of cerebral activation resulting from the stimulation.

Research has consistently indicated that increasing levels of task difficulty generate increasing changes in cardiovascular responding. Further, increasing levels of task difficulty have also been found to generate increasing levels of cerebral activation. Thus, by integrating these findings it may be inferred that increasing levels of cerebral activation are associated with increasing changes in heart rate and blood pressure. Additional support for a relationship between changes in cerebral activation and changes in heart rate and blood pressure is provided by research that has correlated these measures. Specifically, positive correlations were found between changes in beta magnitude of EEG measured at the frontal and temporal regions of the brain and changes in heart rate and blood pressure among a group of male participants. Further,

significant negative correlations have been found between changes in alpha magnitude of EEG measured at the frontal and temporal regions of the brain and changes in heart rate and blood pressure among a group of female participants.

Based on an integration of all the aforementioned research findings a new model for the cerebral mediation of heart rate and blood pressure may be proposed. Specifically, given that the right hemisphere is associated with the sympathetic nervous system, it may be inferred that increasing levels of cerebral activation within the right temporal lobe will result in increasing levels of sympathetic control of heart rate and blood pressure. Conversely, increasing levels of cerebral activation within the left temporal lobe will result in increasing levels of parasympathetic control of heart rate and blood pressure. However, as mentioned previously, the frontal lobes are also involved in the mediation of these cardiovascular measures. Thus, whereas increasing levels of cerebral activation within the right frontal lobe will result in increasing levels of inhibition of the sympathetic nervous system, increasing levels of cerebral activation within the left frontal lobe will yield increasing levels of inhibition of the parasympathetic nervous system. Thus, overall changes in heart rate and blood pressure will be determined by the relative levels of cerebral activation across the left and right frontal and temporal lobes.

The purpose of the present investigation is to empirically test part of this proposed model of the cerebral mediation of heart rate and blood pressure. Specifically, the present investigation seeks to test the proposition that increasing magnitude of cerebral activity within the right temporal lobe, as induced through presentation of white noise, generates increasing levels of sympathetic activation of heart rate and blood pressure. Additionally, the contribution of the right frontal lobe, as assessed by the Ruff Figural Fluency Test, in inhibiting sympathetic activation of heart rate and blood pressure will be examined. More specifically, it is hypothesized that greater impairment in right frontal lobe functioning will result in relative disinhibition of the right temporal lobe and hence heart rate and blood pressure. It is anticipated that the results of this research will contribute to a greater understanding of how specific changes in the direction and magnitude of heart rate and blood pressure are reflected by concomitant changes in cerebral activation. This research is also vital as it seeks to integrate the fields of neuropsychology and psychophysiology in explaining the cerebral mediation of heart rate and blood pressure. This research will also use human subjects as this is the group to which the results will be generalized and this is also the group which is of primary interest to the investigators.

Procedures

Participants will consist of approximately 60 right handed men with an age range of approximately 18 to 26 years. Right handed men will be utilized as participants as research has indicated that important differences exist in the lateralization of functions, and specifically the lateralization of the sympathetic and parasympathetic nervous systems, between right and left handed individuals as well as between men and women. Hence, including both right and left handed men and women would render the data uninterpretable. The subject pool will consist of undergraduate students who are enrolled in an introductory to psychology or other undergraduate courses. Recruitment opportunities will be made available through allowing the participants to review a short description of the experiment that will be included in the consent form, which will be located on the fifth floor of Derring Hall. This short description will be attached to a sign-up sheet which will provide information concerning how to contact the principle investigator and which will also inform the participants to report at this location should they sign-up for the experiment.

The participants will be asked initially to read and sign the consent form and will then be given the opportunity to ask questions regarding the experiment. They will then be given a brief description of the experiment. All participants will then be given the Ruff Figural Fluency Test, the Design Learning Test, the Rey Auditory Verbal Learning Test, Trail Making Parts A and B, Trail Making Parts C and D, the Coren, Porac, and Duncan (CPD) Laterality Questionnaire (see Appendix A), the Head Injury/Medical History Questionnaire (HIMHQ; see Appendix B), and the Cook Medley Hostility Scale (CMHS; see Appendix C). The criterion for inclusion is right handedness, as defined by a minimum score of 7 on the CPD and both biological parents identified as being right handed. Additionally, only those who present with no significant neuropsychological impairments, as evidenced by responses on the HIMHQ, will be considered for inclusion.

Subsequent to completion of the aforementioned tests and questionnaires, the participants will be fitted for a lycra electrode cap containing 19 electrodes arranged according to the International 10/20 system. Electrodes for measuring EMG will then be placed over the participant's eyes. The participants will then be connected to the NeuroSearch-24, which will be used to record their electroencephalogram. Subsequently, the participants will be connected to a Norelco Model HC3501 Digital Blood Pressure Monitor, which will be used to measure diastolic and systolic blood pressure as well as heart rate. The participants will then be given five to eight minutes to relax with their eyes closed so that baseline recordings of heart rate, blood pressure, and electroencephalography may be obtained. Each participant will be exposed to three levels of intensity of white noise (55dB, 70dB, and 85dB) presented binaurally following completion of the baseline measurements of heart rate, blood pressure, and electroencephalography. The white noise will be presented for a duration of approximately 45 seconds, during which time EEG activity will be recorded. Immediately following presentation of the white noise cardiovascular responses will also be recorded. The participants will be asked to complete the Ruff Figural Fluency Test for a second time following presentation of the last white noise stimulus and disconnection from the physiological recording equipment. Subsequently, they will be afforded the opportunity to ask questions regarding the experiment, thanked for their participation, and dismissed. It is anticipated that the duration of the entire experiment should last no longer than approximately two hours and thirty minutes.

Risks from Participating in the Study

Risks are minimal since precautions are being followed. Participants will be informed that they may discontinue the experiment at any time. Concerning the use of EEG recordings, we follow procedures that are approved by the FDA and recommended by the equipment distributors. However, the participants may feel some discomfort during this experiment due to the placement and inflation of blood pressure equipment on their arm. Also, participation in EEG data collection may produce discomfort. Specifically, the placement of ear electrodes may pinch their ears and be potentially painful. Wearing the electro-cap may produce pressure around the head that is uncomfortable, or even painful. Other risks include slight discomfort from the abrasive cleaning and trivial risk of infection. To ensure the participant's safety from infection, all electrodes, electrode caps, and other equipment contacting the participant's body will be thoroughly sanitized before each experimental session. The experimenters will also wear rubber gloves while applying electrode caps and individual electrodes. These gloves will be thrown away immediately afterwards. Electrode gel injectors and blunt needles for attaching to the injector come in enclosed packages and are sanitized by the distributor. They will also be

discarded after each subject. All containers and materials will be cleaned and/or disposed of following procedures approved for BioHazard disposition. Containers for the injectors and needles will be clearly labeled "BioHazard" and will be separate from other laboratory waste.

Cotton swabs are used to apply the cleanser to the skin (other than scalp) and disposed of immediately afterwards. Electrodes are lightly scrubbed with Ivory soap to remove any gel and then sterilized, according to standardized procedures with a solution purchased from the manufacturer. Given these procedures, the likelihood of transmission of human diseases is quite minimal if not nonexistent. Using standardized procedures, the participant's skin is cleansed to reduce impedance from natural oils at each electrode site. These cleansings are accomplished with the use of NuPrep, a commonly used antiseptic cleanser accepted in EEG research laboratories in the US and Europe. Such procedures are recommended by the Electrocap company and follow US government hospital regulations. Since participants who have skin allergies have been shown to rarely experience a skin rash from NuPrep, subjects are asked if they have ever had an skin allergies. If so, only rubbing alcohol will be used to clean the skin. The researchers will monitor any recommended changes and update procedures when necessary.

Further safeguards that will be used to minimize any discomfort include the continuous opportunity to terminate the experiment without penalty to themselves (as in loosing extra credit points) should they ever feel uncomfortable. A thorough debriefing discussing any issues that may be of concern to the participants will also be provided at the end of the experiment. At that time the participants will be given ample opportunity to ask any additional questions about the research that they feel were inadequately addressed by the debriefing. Should the investigator find that the mental or physical health of the participant is in jeopardy the will be immediately referred to the Psychological Services Center, the Student Health Center, and/or Respond.

Expected Benefits

Participation will provide the participants with an increased understanding of the relationship between neuropsychological functioning and physiological responding by providing them with a description of the research and expected findings after the completion of their participation. Participants will also benefit from an increased understanding of how psychological research is conducted. Finally, the participants will benefit from participation through earning extra credit in their undergraduate psychology course.

Confidentiality

Confidentiality will be maintained by allowing only those directly involved with the experiment access to the data collected. The principle investigator will also hold all data collected and will store this data in a locked filing cabinet. Under no circumstances will anyone other than those directly involved in the experiment be allowed access to this information. By doing so the identity of those participating will be held in strict confidence. Neither video nor audio recordings of the participants will be employed.

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY
Informed Consent for Participants
Of Investigative Projects

Graded Cerebral Activation to Noise:
Behavioral and Cardiovascular Effects
Paul S. Foster and David W. Harrison

I. The Purpose of This Research

The purpose o this research investigation is to study the relationship between neuropsychological functioning and psychophysiological responding. Specifically, this research concerns the psychophysiological sequelae of graded stimulation of the auditory cortex and the mediation of these responses by the frontal lobes. Electroencephalographic (EEG) recordings will be taken as a measure of auditory cortex stimulation and frontal lobe activation. It is anticipated that your participation will also aide in developing a greater understanding of how the cerebrum mediating cardiovascular functioning. The neuropsychophysiological measures will include changes in heart rate, blood pressure, and EEG.

II. Procedures

All participants will be initially asked to read and sign this informed consent form. You will then participate in an initial screening session at which time you will be asked complete the following tests: Ruff Figural Fluency Test; Design Learning Test; Rey Auditory Verbal Learning Test; Trail Making Parts A and B; Trail Making Parts C and D; Coren, Porac, and Duncan Laterality Questionnaire; Head Injury/Medical History Questionnaire; and the Cook Medley Hostility Scale. It is anticipated that approximately 60 participants will be included in this initial screening. Your phone number and e-mail address will also be collected during the initial screening session so that you may be contacted to arrange a date for participating in the second part of the experiment. If contacted for the second part of the experiment you may be connected to a NeuroSearch-24, which will be used to record your electroencephalogram (EEG) and the electromyographic (EMG) of your left and right eyes. You may also be connected to a Norelco Digital Blood Pressure Monitor, which will be used to measure systolic and diastolic blood pressure as well as heart rate. Subsequent to connection to this neuropsychophysiological equipment you will be seated in a comfortable chair and asked to close your eyes and relax for five to eight minutes so that a baseline physiological measurement may be recorded. You will then be exposed to three levels of intensity of white noise presented to both ears. The white noise will be presented for 45 seconds, during which time EEG and EMG activity will be recorded. Immediately following exposure to each level of intensity of white noise blood pressure and heart rate recordings will also be obtained. You may also be asked to complete the Ruff Figural Fluency Test for a second time following exposure to all three trials of white noise presentation. You will then be afforded the opportunity to ask any questions regarding the experiment.

You will be required to participate in two separate sessions, the initial screening session and the experimental session. Participation in the initial screening session should take no longer than approximately one hour and participation in the experimental session should take no longer than approximately one hour and thirty minutes. The room where the initial screening session is to be completed is listed on the sign-up sheet and the slip for your records. The experimental session will be held in the physiological laboratory on the fifth floor of Derring Hall.

III. Risks

You may feel some discomfort during this experiment due to the placement and inflation of the blood pressure cuff on your arm. Also, participation in EEG data collection may produce discomfort. Specifically, the placement of ear electrodes may pinch your ears and be potentially painful. Wearing the electro-cap may produce pressure around your head this is uncomfortable. Other risks include slight discomfort from the abrasive cleaning and trivial risk of infection. To ensure your safety from infection, all electrodes, electrode caps, and other equipment contacting the body will be thoroughly sanitized before each experimental session. The experimenters will also wear rubber gloves while applying electrode caps and individual electrodes. These gloves will be thrown away immediately afterwards. Electrode get injectors and blunt needles for attaching to the injector come in enclosed packages and are sanitized by the distributor. They will also be discarded after use. All containers and materials will be cleaned and/or disposed of following procedures approved for BioHazard disposition. Container(s) for the injectors and needles will be clearly labeled "BioHazard" and will be separate from other laboratory waste.

Cotton swabs are used to apply the cleanser to the skin (other than scalp) and disposed of immediately afterwards. Electrodes are lightly scrubbed with Ivory soap to remove any gel and then sterilized, according to standardized procedures with a solution purchased from the manufacturer. Given these procedures, the likelihood of transmission of human diseases in quite minimal if not nonexistant. Using standardized procedures, your skin is cleansed to reduce impedance from natural oils at each electrode site. These cleansings are accomplished with the use of NuPrep, a commonly used antiseptic cleanser accepted in EEG research laboratories in the US and Europe. Such procedures are recommended by the Electrocap company and follow US government hospital regulations. Since those ho have skin allergies have been shown to rarely experience a skin rash from NuPrep, you will be asked if you have ever had any skin allergies. If so, only rubbing alcohol will be used to clean the skin. The researchers will monitor any recommended changes and update procedures when necessary.

Safeguards that will be used to minimize your discomfort include the continuous opportunity to terminate the experiments without penalty to yourself (as in loosing extra credit points) should you ever feel uncomfortable. A thorough debriefing discussion any issues that may be of concern to you will also be provided at the end of the experiment. At that time you will be given ample opportunity to ask any additional questions about the research that you feel were inadequately addressed by the debriefing.

IV. Benefits of this Project

It is anticipated that the results of this research will contribute to a greater depth of understanding of the relationship between neuropsychological functioning and cardiovascular responding. This increased understanding has potentially important clinical implications regarding the cardiovascular effects of cerebrovascular disorders. Benefits for the participants include an increased awareness of how professional research is conducted. Also, through participating in this experiment and signing this consent from you agree to the fact that no promise or guarantee of benefits have induced you to participate. Finally, you are more than welcome to contact the principle investigator with any questions about the experiment or to request a summary of the results of this experiment.

V. Extent of Confidentiality

Your identity will be held strictly confidential both during and following the experiment. All data collected during the experiment will be held by the principle investigator in a secured location. Furthermore, the results of your participation will be confidential and at no time will

the researchers release the specific results from your participation in this experiment to anyone other than individuals working on this project without your prior consent. However, confidentiality may be broken if you are perceived to be a threat to yourself or others, in which case the investigators will contact the appropriate authorities. You will not be audio or video taped during any portion of this experiment.

VI. Compensation

You will be compensated for your participation in this experiment by earning one extra credit point for the initial screening session and two extra credit points for the experimental session. The extra credit points will be added to the sum of your grades in your psychology course. You may want to verify how these points will be used with your course instructor. Also, alternative means for earning extra credit may be undertaken without participating in this experiment. Should you be interested in these alternatives you should contact your course instructor who will promptly provide you with instructions on how to do so.

VII. Freedom to Withdraw

Please note that your participation is entirely voluntary, that you can refuse to answer any question, you can withdraw your consent and participation at any time without penalty or reduction in extra credit points earned, and you can have the results of your participation returned, removed from the experimental records, or destroyed.

VIII. Approval of Research

This research has been approved, as required, by the Institutional Review Board for Research Involving Human Subjects at Virginia Polytechnic Institute and State University and by the Department of Psychology.

IX. Participant's Responsibilities

I voluntarily agree to participate in this study. I have the following responsibilities:

- To answer all question truthfully and respond to the best of my ability.
- To remain still during the recording of all physiological measures.
- To follow the directions of the investigator.
- To inform the experimenter of any history of head trauma, epilepsy, or any cardiac problems.

X. Participant's Permission

I have read and understand the Informed Consent and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent for participation in this project. If I participate, I may withdraw at any time without penalty. I agree to abide by the rules of this project.

_____ _____

Signature Date

Should I have any questions about this research or its conduct, I may contact:

Paul S. Foster 231-6581 Principle Investigator	D. W. Harrison, Ph.D. 231-4422 Chair, HSC
David Moore, Ph.D. 231-5281	Department of Psychology 231-6581 Chair, IRB Research Division

Biographical Sketch

Mr. Foster graduated from Kennesaw State College with a Bachelor of Science in Psychology in 1993. He graduated Summa Cum Laude from Kennesaw State College in 1994 with a Bachelor of Science in Public and Social Services. Mr. Foster earned his Master of Science in Clinical Psychology from Georgia Southern University in 1996. He is currently a fourth year Doctoral student in the Department of Psychology at Virginia Tech. As a graduate student Mr. Foster has conducted numerous investigations concerning the psychophysiological sequelae and differentiation of various emotions and emotion induction methods. He has also conducted numerous research projects concerning the relationships between neuropsychological functioning and cardiovascular responding. He has 3 publications in this field in which he is first author and 4 other relevant articles which are currently being considered for publication. He also has 8 relevant presentations at regional conference meetings. He also won the Southeastern Psychological Association's Special Topics Research Competition for graduate students in 1998 and was the third place winner in the Social Science and Humanities Category of the 17th Annual Research Symposium of Virginia Tech in 2001. Finally, Mr. Foster was awarded two grants from the Georgia Southern University Graduate Student Professional Development Fund.

Dr. Harrison graduated with Highest Distinction in 1978 from the University of New Mexico with a Bachelor of Science in Psychology/Biology. He earned a Master of Science in Biopsychology from the University of Georgia in 1980. A Doctor of Philosophy in Bio/Neuropsychology was then earned from the University of Georgia in 1983. Dr. Harrison has been employed as an Associate Professor of Psychology since 1991 and as the Director of the Laboratory of Neuropsychology since 1985. Previously, Dr. Harrison was also employed as an Assistant Professor of Psychology at Virginia Tech from 1985 to 1990. Dr. Harrison's research experiences include having 55 publications as well as another 14 articles that have been submitted for publication. Dr. Harrison also has 11 different papers and presentations at international conference meetings, 33 presentations at national meetings, and another 61 papers and presentations at regional meetings. In addition, Dr. Harrison has chaired 15 Dissertations and 10 Theses. All of these research experiences have involved issues pertaining to neuropsychology. Dr. Harrison has also been awarded 12 different grants over the last decade. Finally, Dr. Harrison is currently a Board-Certified Forensic Examiner, Diplomate of the American Board of Vocational Neuropsychology, a Licensed Psychologist in the state of Virginia, a Licensed Rehabilitation Provider, and a Certified Neurotherapist.

Virginia Tech
VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

Institutional Review Board

Dr. David M. Moore
IRB (Human Subjects) Chair
Assistant Vice Provost for Research Compliance
CVM Phase II – Duckpond Dr., Blacksburg, VA 24061-0442
Office: 540/231-4991; FAX 540/231-6033
e-mail: moored@vt.edu

MEMORANDUM

TO: David Harrison
Paul Foster
Psychology 0436

FROM: David M. Moore

DATE: Januray 31, 2002

SUBJECT: **Expedited Approval – "**Graded Cerebral Activation to Noise: Behavioral and Cardiovascular Effects" – IRB # 02-031

This memo is regarding the above-mentioned protocol. The proposed research is eligible for expedited review according to the specifications authorized by 45 CFR 46.110 and 21 CFR 56.110. As Chair of the Virginia Tech Institutional Review Board, I have granted approval to the study for a period of 12 months, effective January 30, 2002.

Approval of your research by the IRB provides the appropriate review as required by federal and state laws regarding human subject research. It is your responsibility to report to the IRB any adverse reactions that can be attributed to this study.

To continue the project past the 12 month approval period, a continuing review application must be submitted (30) days prior to the anniversary of the original approval date and a summary of the project to date must be provided. My office will send you a reminder of this (60) days prior to the anniversary date.

cc: File

Appendix D

Name: _____ Subject Number: _____

Age: _____ Order: _____

Ruff Total Number of Unique Designs: _____
Ruff Total Number of Perseverations: _____
Ruff Error Ratio: _____

Baseline 1 SBP: _____
Baseline 1 DBP: _____
Baseline 1 MAP: _____
Baseline 1 HR: _____

Baseline 2 SBP: _____
Baseline 2 DBP: _____
Baseline 2 MAP: _____
Baseline 2 HR: _____

Mean Baseline SBP: _____
Mean Baseline DBP: _____
Mean Baseline MAP: _____
Mean Baseline HR: _____

55 dB SBP: _____
55 dB DBP: _____
55 dB MAP: _____
55 dB HR: _____

75 dB SBP: _____
75 dB DBP: _____
75 dB MAP: _____
75 dB HR: _____

90 dB SBP: _____
90 dB DBP: _____
90 dB MAP: _____
90 dB HR: _____

Baseline

High Beta:	Low Beta:	High Alpha:	Low Alpha:	Theta:	High Delta:	ohms:
FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____
FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____
F3: _____	F3: _____	F3: _____	F3: _____	F3: _____	F3: _____	F3: _____
F4: _____	F4: _____	F4: _____	F4: _____	F4: _____	F4: _____	F4: _____
F7: _____	F7: _____	F7: _____	F7: _____	F7: _____	F7: _____	F7: _____
F8: _____	F8: _____	F8: _____	F8: _____	F8: _____	F8: _____	F8: _____
T3: _____	T3: _____	T3: _____	T3: _____	T3: _____	T3: _____	T3: _____
T4: _____	T4: _____	T4: _____	T4: _____	T4: _____	T4: _____	T4: _____
T5: _____	T5: _____	T5: _____	T5: _____	T5: _____	T5: _____	T5: _____
T6: _____	T6: _____	T6: _____	T6: _____	T6: _____	T6: _____	T6: _____
C3: _____	C3: _____	C3: _____	C3: _____	C3: _____	C3: _____	C3: _____
C4: _____	C4: _____	C4: _____	C4: _____	C4: _____	C4: _____	C4: _____
P3: _____	P3: _____	P3: _____	P3: _____	P3: _____	P3: _____	P3: _____
P4: _____	P4: _____	P4: _____	P4: _____	P4: _____	P4: _____	P4: _____
O1: _____	O1: _____	O1: _____	O1: _____	O1: _____	O1: _____	O1: _____
O2: _____	O2: _____	O2: _____	O2: _____	O2: _____	O2: _____	O2: _____
FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____
CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____
PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____
						Ref: _____
						Gnd: _____

55dB

High Beta:	Low Beta:	High Alpha:	Low Alpha:	Theta:	High Delta:
FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____
FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____
F3: _____	F3: _____	F3: _____	F3: _____	F3: _____	F3: _____
F4: _____	F4: _____	F4: _____	F4: _____	F4: _____	F4: _____
F7: _____	F7: _____	F7: _____	F7: _____	F7: _____	F7: _____
F8: _____	F8: _____	F8: _____	F8: _____	F8: _____	F8: _____
T3: _____	T3: _____	T3: _____	T3: _____	T3: _____	T3: _____
T4: _____	T4: _____	T4: _____	T4: _____	T4: _____	T4: _____
T5: _____	T5: _____	T5: _____	T5: _____	T5: _____	T5: _____
T6: _____	T6: _____	T6: _____	T6: _____	T6: _____	T6: _____
C3: _____	C3: _____	C3: _____	C3: _____	C3: _____	C3: _____
C4: _____	C4: _____	C4: _____	C4: _____	C4: _____	C4: _____
P3: _____	P3: _____	P3: _____	P3: _____	P3: _____	P3: _____
P4: _____	P4: _____	P4: _____	P4: _____	P4: _____	P4: _____
O1: _____	O1: _____	O1: _____	O1: _____	O1: _____	O1: _____
O2: _____	O2: _____	O2: _____	O2: _____	O2: _____	O2: _____
FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____
CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____
PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____

75dB

High Beta:	Low Beta:	High Alpha:	Low Alpha:	Theta:	High Delta:
FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____
FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____
F3: _____	F3: _____	F3: _____	F3: _____	F3: _____	F3: _____
F4: _____	F4: _____	F4: _____	F4: _____	F4: _____	F4: _____
F7: _____	F7: _____	F7: _____	F7: _____	F7: _____	F7: _____
F8: _____	F8: _____	F8: _____	F8: _____	F8: _____	F8: _____
T3: _____	T3: _____	T3: _____	T3: _____	T3: _____	T3: _____
T4: _____	T4: _____	T4: _____	T4: _____	T4: _____	T4: _____
T5: _____	T5: _____	T5: _____	T5: _____	T5: _____	T5: _____
T6: _____	T6: _____	T6: _____	T6: _____	T6: _____	T6: _____
C3: _____	C3: _____	C3: _____	C3: _____	C3: _____	C3: _____
C4: _____	C4: _____	C4: _____	C4: _____	C4: _____	C4: _____
P3: _____	P3: _____	P3: _____	P3: _____	P3: _____	P3: _____
P4: _____	P4: _____	P4: _____	P4: _____	P4: _____	P4: _____
O1: _____	O1: _____	O1: _____	O1: _____	O1: _____	O1: _____
O2: _____	O2: _____	O2: _____	O2: _____	O2: _____	O2: _____
FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____
CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____
PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____

90dB

High Beta:	Low Beta:	High Alpha:	Low Alpha:	Theta:	High Delta:
FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____	FP1: _____
FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____	FP2: _____
F3: _____	F3: _____	F3: _____	F3: _____	F3: _____	F3: _____
F4: _____	F4: _____	F4: _____	F4: _____	F4: _____	F4: _____
F7: _____	F7: _____	F7: _____	F7: _____	F7: _____	F7: _____
F8: _____	F8: _____	F8: _____	F8: _____	F8: _____	F8: _____
T3: _____	T3: _____	T3: _____	T3: _____	T3: _____	T3: _____
T4: _____	T4: _____	T4: _____	T4: _____	T4: _____	T4: _____
T5: _____	T5: _____	T5: _____	T5: _____	T5: _____	T5: _____
T6: _____	T6: _____	T6: _____	T6: _____	T6: _____	T6: _____
C3: _____	C3: _____	C3: _____	C3: _____	C3: _____	C3: _____
C4: _____	C4: _____	C4: _____	C4: _____	C4: _____	C4: _____
P3: _____	P3: _____	P3: _____	P3: _____	P3: _____	P3: _____
P4: _____	P4: _____	P4: _____	P4: _____	P4: _____	P4: _____
O1: _____	O1: _____	O1: _____	O1: _____	O1: _____	O1: _____
O2: _____	O2: _____	O2: _____	O2: _____	O2: _____	O2: _____
FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____	FZ: _____
CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____	CZ: _____
PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____	PZ: _____

Appendix E

Instruction for the Ruff Figural Fluency Test

Begin with the sample items for Part 1. Say the following:

In front of you are three squares, each containing five dots. Note that the arrangement of the five dots is always the same. I want you to connect two or more dots by always using straight lines. The purpose of the test is for you to make as many patterns (or figures) as possible, but each pattern has to be different in some way from all the others.

Following completion of the sample, turn the page and then say:

On this page, please draw as many different patterns (or figures) as possible. Start in the upper left square and work from left to right (examiner points out correct order). Just connect at least two dots with a straight line. Remember, work as quickly as possible and make every pattern different. Get ready –go.

Paul S. Foster
1320-1 Ephesus Church Road
Chapel Hill, NC 27517
(919) 933-6628
pfoster@vt.edu

EDUCATION

Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Doctor of Philosophy, Clinical Psychology
Presently enrolled; Current GPA 3.69
Title of Dissertation: Graded Cerebral Activation to Noise: Behavioral and Cardiovascular Effects.
Major Advisor: David W. Harrison, Ph.D.

Georgia Southern University
Statesboro, Georgia
Master of Science, Clinical Psychology
June 8, 1996; GPA 3.71
Title of Thesis: The Effects of Enacted Fantasy Aggression on Psychophysiological Arousal Resulting From Frustration.
Major Advisor: Edward W. L. Smith, Ph.D.

Kennesaw State College
Marietta, Georgia
Bachelor of Science, Public and Social Services
Summa Cum Laude
June 18, 1994; GPA 4.00

Kennesaw State College
Marietta, Georgia
Bachelor of Science, Psychology
June 19, 1993; GPA 3.20

INTERNSHIPS

University of North Carolina at Chapel Hill School of Medicine
Chapel Hill, North Carolina
09/02/03 to present
As the Adult Neuropsychology intern I have participated in the following rotations:

Adult Clinical Neuropsychology. Performed comprehensive neuropsychological evaluations utilizing a flexible battery approach within the Department of Physical Medicine and Rehabilitation. Supervision conducted by Karla Thompson, Ph.D.

Behavioral Medicine. Performed psychological assessments to aide in determining heart and lung transplant candidacy. Supervision conducted by Eileen Burker, Ph.D.

The Devereux Foundation
Kennesaw, Georgia
01/94 to 03/94
Completed an internship as a work adjustment instructor at a residential treatment facility for adolescents diagnosed with a variety of emotional, behavioral, psychiatric, and developmental disabilities. Duties consisted of vocational adjustment instruction, social skills training, work milieu skills training, and independent living skills training.

EXTERNSHIP

Lewis-Gale Department of Psychological Medicine
Salem, Virginia
05/28/99 to 02/02/00
Conducted clinical outcome research with the purposes of tracking therapeutic progress and determining the efficacy of the services provided. Duties consisted of compiling, organizing, and analyzing data from a database of approximately 2900 patients. A final report of the findings was written and recommendations provided.

PRACTICA

Virginia Tech Psychological Services Center
Blacksburg, Virginia
08/24/98 to 04/30/03
Fall 1999 to Spring 2000: Provided therapy services for adults, adolescents, and couples with a range of presenting psychological difficulties and referral questions. Psychological assessments were also conducted for purposes of assessing learning disabilities and providing recommendations for special accommodations as well as strategies for coping with learning disabilities and maximizing learning experiences. Therapy and assessment activities were supervised by Lee D. Cooper, Ph.D., Licensed Psychologist.

Fall 1998 to Spring 1999; Fall 2000 to present: Provided neuropsychological assessments consisting of standardized testing and syndrome analysis for individuals with a variety of referral questions. Neuropsychological assessments also often consisted of quantitative electroencephalography to provide for convergent validation of suspected cerebral dysfunction. Rehabilitation of individuals with cerebral dysfunction was also performed. Neuropsychological assessment and rehabilitation activities were supervised by David W. Harrison, Ph.D., Licensed Psychologist.

Ogeechee Area Mental Health
Swainsboro, Georgia
01/12/96 to 05/29/96
Provided counseling and therapy services to prevent and reduce the disabling effects of mental illness, mental retardation and the abuse of alcohol and drugs. Counseling and therapy services were supervised by Pamela O. Carr, Licensed Professional Counselor, at Ogeechee Area Mental Health and by James L. Pugh, Ph.D., Licensed Psychologist, at Georgia Southern University.

The Regents Center for Learning Disorders
Statesboro, Georgia
09/27/95 to 12/04/95
Performed comprehensive standardized psychological assessments for students experiencing academic difficulties stemming from suspected learning disabilities. Assessment activities and weekly team meetings at The Regents Center for Learning Disorders were supervised by Merry Gallagher, Ed.D., Licensed Psychologist. Practicum class meetings at Georgia Southern University were supervised by James L. Pugh, Ph.D., Licensed Psychologist.

CLINICAL WORK EXPERIENCE

Respond at the Lewis-Gale Medical Center
Salem, Virginia
07/06/99 to present
Employed as an Assessment Specialist conducting interviews and assessments for individuals requesting or requiring behavioral health interventions or psychiatric care. Assessments were conducted for purposes of referring patients to the appropriate level of intervention, including outpatient counseling or psychiatric services, intensive outpatient programs, partial hospitalization, or inpatient hospitalization. As an Assessment Specialist responsibilities also included facilitating access of benefits through the precertification process and completing the necessary paperwork for those patients requiring partial or inpatient hospitalization. All assessments were staffed with a psychiatrist.

Neuropsychological and Counseling Services
Salem, Virginia
08/30/02 to 12/27/02
Employed as a neuropsychological technician with duties consisting of conducting clinical interviews and administering and scoring neuropsychological tests. An additional responsibility was the measurement of quantitative electroencephalography, including attachment of electrodes, data acquisition, data artifacting, interpretation of data, and generation of reports.

Respond at Montgomery Regional Hospital
Blacksburg, Virginia
10/12/01 to 01/12/02
Served as the Interim Coordinator with duties consisting of writing monthly admissions/assessment reports and staff time sheet reports. Staff meetings were also coordinated for purposes of communicating and discussing changes in hospital and EMTALA regulations and procedures. Staff on-call and office hours were also coordinated.

Southern Psychological Services
Statesboro, Georgia
06/10/96 to 07/30/98
Administered comprehensive psychological evaluations covering a variety of referral questions including Medicaid evaluations and the assessment of learning disorders and Attention Deficit Disorder. Psychological evaluations were also conducted to aide in determining eligibility for receiving disability benefits through the Social Security Administration. All psychological evaluations were supervised by Steve Chester, Ph.D., Licensed Psychologist.

The Devereux Foundation
Kennesaw, Georgia
07/09/93 to 09/17/94
Employed as a Mental Health Technician at a residential treatment facility for children and adolescents with a variety of emotional, behavioral, developmental, and psychiatric disabilities. Duties consisted of monitoring the patient living unit and patient activities during the course of the shift, supervising patients assigned to my care, and supervising program components and activities. Supervised patients assigned to me according to the program structure operating at the time.

Power Over Panic, Inc.
Atlanta, Georgia
08/93 to 08/94
Volunteered as the Co-Chairman of the Northside group of Power Over Panic, a nonprofit organization devoted to helping those suffering from fears, phobias, panic, and anxiety. Responsibilities included leading group discussion, disseminating information, and supporting members when they requested help coping with their anxiety or panic. Groups met monthly and were supervised by Steven Garber, Ph.D., Licensed Psychologist.

TEACHING EXPERIENCE

Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Graduate Instructor
07/05/99 to 04/30/03
Employed as an instructor of several laboratory and full courses with a range of 15 to 75 students. Duties consisted of creating and grading examinations, assigning and grading research projects and papers, and providing assistance to students. The following courses were taught: Senior Seminar in Clinical Neuropsychology, Principles of Psychological Research, Abnormal Psychology, Psychology of Learning, Cognitive Psychology Lab, Social Psychology Lab, Developmental Psychology Lab.

Graduate Teaching Assistant
08/24/98 to 05/05/99
Employed as a teaching assistant for four Introduction to Psychology recitation courses from the Fall Semester of 1998 to the Spring Semester of 1999, with approximately 35 students per course. Duties consisted of teaching laboratory section associated with an introduction to psychology class, creating and administering quizzes and essays, grading quizzes and essays, and providing assistance to students.

PUBLICATIONS

Foster, P. S. (in press). Use of the Calmset 3 Biofeedback/Relaxation System in the assessment and treatment of chronic nocturnal bruxism. *Applied Psychophysiology and Biofeedback.*

Foster, P. S., & Harrison, D. W. (2004). The covariation of cortical electrical activity and cardiovascular responding. *International Journal of Psychophysiology, 52,* 239-255.

Foster, P. S., & Harrison, D. W. (in press). Cerebral correlates of varying ages of emotional memories. *Cognitive and Behavioral Neurology.*

Foster, P. S., Webster, D. G., & Williamson, J. B. (2002-2003). The psychophysiological differentiation of actual, imagined, and recollected mirth. *Imagination, Cognition and Personality, 22,* 163-180.

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Tirnauer, L., Smith, E. W. L., & Foster, P. S. (1996). The American Academy of Psychotherapists research committee survey of members. *Voices, 32,* 87-94.

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Williamson, J. B., Foster, P. S., & Harrison, D. W. (2003). Functional cerebral asymmetry as a function of hostility. *Psychophysiology, 40(Suppl.),* S92.

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Foster, P. S., & Harrison, D. W. (2001). The neurophysiology of pessimism. *Archives of Clinical Neuropsychology, 16,* 724.

Williamson, J. B., Shenal, B. V., Rhodes, R., Foster, P. S., & Harrison, D. W. (2000). Quantitative EEG assessment of an adolescent with expressive aprosodia. *Archives of Clinical Neuropsychology, 15,* 787.

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SUBMITTED FOR PUBLICATION

Foster, P. S., Williamson, J. B., & Harrison, D. W. (submitted). The Ruff Figural Fluency Test: Heightened right frontal lobe delta activity as a function of performance. *Archives of Clinical Neuropsychology.*

Foster, P. S., & Harrison, D. W. (submitted). The Design Learning Test: A visuospatial analog of the Rey Auditory Verbal Learning Test. *Applied Neuropsychology.*

MANUSCRIPTS IN PREPARATION

Foster, P. S., & Harrison, D. W. (in preparation). The functional cerebral activation model: Cerebral mediation of cardiovascular functioning.

Foster, P. S., & Harrison, D. W. (in preparation). Magnitude of cerebral asymmetry at rest: Covariation with baseline cardiovascular activity.

Foster, P. S., & Harrison, D. W. (in preparation). Recollection of angry memories: Effects of intensity and age on cerebral activity.

PRESENTATIONS

Foster, P. S., Thompson, K., & Harrison, D. W. (2004, February). *Varying ages of angry memories: Electroencephalographic correlates.* Poster session to be presented at the annual meeting of the International Neuropsychological Society, Baltimore, MD.

Foster, P. S., Thompson, K., & Harrison, D. W. (2004, February). *Recollection of mirthful memories: Covariation of electrophysiological and cardiovascular responding.* Poster session to be presented at the annual meeting of the International Neuropsychological Society, Baltimore, MD.

Foster, P. S., Beck, A. L., & Harrison, D. W. (2003, October). *The Ruff Figural Fluency Test: Heightened right frontal lobe delta activity as a function of performance.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Dallas, TX.

Foster, P. S., Beck, A. L., & Harrison, D. W. (2003, October). *The Figure Arrangement Test: Differential performance between low and high hostile men.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Dallas, TX.

Foster, P. S., Beck, A. L., Mollet, G. A., & Harrison, D. W. (2003, October). *Homologous hemispheric comparisons using quantitative electroencephalography: Is symmetry to be expected?* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Dallas, TX.

Williamson, J. B., Foster, P., & Harrison, D. W. (2003, October). *Functional cerebral asymmetry as a function of hostility.* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Chicago, IL.

Foster, P. S., Williamson, J., & Harrison, D. W. (2003, October). *That was so funny you can even see it on my brain!* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Chicago, IL.

Foster, P. S., Williamson, J., & Harrison, D. W. (2003, October). *The effects of varying intensity of white noise on cerebral activation.* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Chicago, IL.

Beck, A. L., Mollet, G. A., Foster, P. S., Walters, R. P., & Harrison, D. W. (2003, March). *Thalamic syndrome: Lateralized multimodal hallucinations.* Poster session presented at the 19th Annual Graduate Research Symposium of Virginia Tech, Blacksburg, VA.

Foster, P. S., & Harrison, D. W. (2003, March). *That was so funny you can even see it on my brain!* Poster session presented at the 19th Annual Graduate Research Symposium of Virginia Tech, Blacksburg, VA.

Harrison, D. W., Walters, R. P., Williamson, J. B., & Foster, P. S. (2002, October). *Lateralized visual hallucinations: Analysis of affective valence.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Miami, FL.

Foster, P. S., & Harrison, D. W. (2002, October). *Quantitative electroencephalographic assessment of an individual with comorbid depression and panic attacks.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Miami, FL.

Foster, P. S., & Harrison, D. W. (2002, October). *Blazing trails with the right hemisphere: A homologous version of Trail Making Parts A and B.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Miami, FL.

Foster, P. S., Williamson, J. B., & Harrison, D. W. (2002, October). *The Design Learning Test: Assessment of learning using the right hemisphere.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Miami, FL.

Williamson, J. B., Harrison, D. W., & Foster, P. S. (2002, October). *Emotion and arousal: A functional cerebral systems analysis.* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Washington, DC.

Foster, P. S., & Harrison, D. W. (2002, October). *Now I'm really angry, just look at my brain!* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Washington, DC.

Foster, P. S., & Harrison, D. W. (2002, October). *Resting magnitude of cerebral asymmetry: Baseline cardiovascular correlates.* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Washington, DC.

Foster, P. S., & Harrison, D. W. (2002, October). *Changes in magnitude of cerebral activation: Covariation with cardiovascular responding.* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Washington, DC.

Foster, P. S., Beck, A., & Harrison, D. W. (2002, October). *Intensity and age effects of angry memories on cardiovascular responding.* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Washington, DC.

Foster, P. S. & Harrison, D. W. (2002, April). *Now I'm really angry, just look at my brain!* Poster session presented at the 18th Annual Graduate Research Symposium of Virginia Tech, Blacksburg, VA.

Foster, P. S., & Harrison, D. W. (2001, November). *The neurophysiology of pessimism.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, San Francisco, CA.

Foster, P. S., & Harrison, D. W. (2001, November). *Quantitative electroencephalographic outcome assessment of expressive dysphasia and dysprosodia.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, San Francisco, CA.

Christie, I. C., Williamson, J. B., Foster, P. S., & Park, A. (2001, October). *Autonomic responses to laterally presented rhythm and melody.* Poster session presented at the annual meeting of the Society for Psychophysiological Research, Montreal, Canada.

Foster, P. S. (2001, April). *Emotional memories: The relationship between age of memory and the corresponding psychophysiological responses.* Paper presented at the Spring Convention of the Virginia Psychological Association Convention, Roanoke, VA.

Foster, P. S. (2001, April). *The relationship between the subjective intensity of emotional memories and cardiovascular responding.* Poster session presented at the annual meeting of the Eastern Psychological Association, Washington, D.C.

Foster, P. S., & Harrison, D. W. (2001, March). *The relationship between magnitude of cortical activation and cardiovascular responding.* Poster session presented at the 17th Annual Graduate Research Symposium of Virginia Tech, Blacksburg, VA.

Higgins, D., Williamson, J. B., Beck, A., Foster, P. S., & Harrison, D. W. (2001, March). *Frontal lobe deterioration: Sex differences in aging effects.* Poster session presented at the annual meeting of the Southeastern Psychological Association, Atlanta, GA.

Foster, P. S., & Harrison, D. W. (2001, March). *The relationship between magnitude of cortical activation and cardiovascular responding.* Poster session presented at the annual meeting of the Southeastern Psychological Association, Atlanta, GA.

Williamson, J. B., Shenal, B. V., Rhodes, R. D., Foster, P. S., & Harrison, D. W. (2000, November). *Quantitative EEG assessment of an adolescent with expressive aprosodia.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Orlando, FL.

Foster, P. S., Williamson, J. B., & Harrison, D. W. (2000, November). *Quantitative Electroencephalogram of an individual diagnosed with nonfluent dysphasia.* Poster session presented at the annual meeting of the National Academy of Neuropsychology, Orlando, FL.

Williamson, J. B., Foster, P., & Harrison, D. (2000, March). *Differential effects of hostility level on nonverbal and verbal fluency.* Poster session presented at the annual meeting of the Southeastern Psychological Association, New Orleans, LA.

Foster, P. S., Webster, D. G., & Williamson, J. B. (1999, March). *The psychophysiological differentiation of actual, imagined, and recollected mirth.* Poster session presented at the annual meeting of the Southeastern Psychological Association, Savannah, GA.

Foster, P. S., Smith, E. W. L., & Webster, D. G. (1998, March). *The psychophysiological differentiation of actual, imagined, and recollected anger.* Paper presented at the annual meeting of the Southeastern Psychological Association, Mobile, AL.

Foster, P. S., Webster, D. G., & Smith, E. W. L. (1997, April). *The psychophysiological differentiation of emotional memories.* Poster session presented at the annual meeting of the Southeastern Psychological Association, Atlanta, GA.

AWARDS

Research project entitled *Now I'm Really Angry, Just Look at My Brain!* was the first place winner in the Social Sciences and Humanities Category of the 18th Annual Research Symposium of Virginia Tech in 2002.

Research project entitled *The Relationship Between Magnitude of Cortical Activation and Cardiovascular Responding* was the third place winner in the Social Sciences and Humanities Category of the 17th Annual Research Symposium of Virginia Tech in 2001.

Research project entitled *The Psychophysiological Differentiation of Actual, Imagined, and Recollected Anger* was a winner of the 1998 Southeastern Psychological Association's Special Topics Research Competition for graduate students.

GRANTS AWARDED

Research project entitled *The Psychophysiological Differentiation of Emotional Memories* was supported in part by a grant from the Georgia Southern University Graduate Student Professional Development Fund.

Research project entitled *The Psychophysiological Differentiation of Actual, Imagined, and Recollected Mirth* was supported in part by a grant from the Georgia Southern University Graduate Student Professional Development Fund.

HONORS

President's List: 08/24/92; 12/12/92; 03/19/93; 06/11/93; 12/14/93; 03/23/94; 06/18/94
Dean's List: 03/16/92; 06/08/92

EDITORIAL ACTIVITIES

Guest editor of the *Journal of Gender, Culture, and Health*.
Assisted in preparation of:
Chandler, H. K., & Finney, J. W. (1999). *Exploring psychology: Reader and Workbook.* New York: Primis Custom Publishing.

PROFESSIONAL MEMBERSHIPS

Graduate Student Affiliate of the American Psychological Association
Member of the Volunteer State Archaeological Society
Student Member of the National Academy of Neuropsychology
Student Member of the Society for Psychophysiological Research *Revised 12/20/03*