Challenging frontal lobe capacity using lateralized vestibular stress: A functional cerebral systems approach to a clinical risk for falls

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ABSTRACT

A conceptual model was originally proposed that linked the vestibular modality with executive domains by means of a functional cerebral systems framework. The claim was that frontal regions exert regulatory control over posterior systems for sensation and autonomic functions in a dense, interconnected network with right hemisphere specialization. As a preponderance of evidence demonstrates that a design fluency task is often associated with right frontal functioning, it was hypothesized that proficiency on a design fluency task would yield differences in QEEG and skin conductance after vestibular activation. Fifty-eight total (29 high- and 29 low-fluent performers on the Ruff Figural Fluency Test were subjected to 20 whole-body passive rotations about the neuroaxis at a constant rate of approximately 120 degrees per second. EEG and skin conductance levels were recorded prior to and post-rotation. Analyses were conducted on delta (1-4 Hz.) and beta (13-21 Hz.) frequencies. Overall, delta activity increased from baseline to post-rotation with higher levels at frontal sites, however no group differences were found across conditions. Regarding beta activation, high design fluency was associated with increased beta activation at the right temporal site (T6). In contrast to expectations, beta activity diminished from baseline to post-stress over both groups. Skin conductance levels increased from baseline to post-stress. Methodological considerations are discussed regarding gender issues and procedures of the experiment. The results indicate that vestibular disorientation yields systematic delta changes in the frontal regions, but that future refinements to the vestibular stressor may elicit QEEG and skin conductance differences in fluency groups.

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Introduction

The association of emotion and vestibular dysfunction is not without historical precedence. Although Aristotle did not ascribe to the vestibular system the high stature of inclusion amongst the five classical senses enumerated in *De Anima*, he was aware of vestibular phenomena and made records of dizziness experiences (Wade, 1994). For Aristotle, certain senses provided more problematic differentiation of origin than others. While vision, taste, hearing, and smell were identified with the eyes, tongue, ears, and the nose, respectively, the sense of touch, including sensations of friction, temperature, and pressure, was not as easily pinpointed to one specific organ (Wade, 2003). Likewise, the vestibular system was problematic to localize and consequently can be considered the last of the basic sensory modalities to be discovered. With the confirmation of vestibular end organs in the temporal bone by Prosper Ménière in 1861 (Baloh, 2001), neurological explorations focused on the cranial nerve pathways to the brainstem. The role of the cerebral hemispheres in the experience of dizziness and motion sickness was contested, overlooked, minimized, or even largely ignored. Subsequently, the long-term effect of Aristotle's omission was to preclude the influence of emotion in vestibular system processes.

The literature has implicated emotional influences in sensory modalities including vision (Wittling & Roschmann, 1993), audition (Schmitt, Hartje, & Willmes, 1997; Everhart, Demaree, & Harrison, 2008), somasthesis (Borod, Vingiano, & Cytryn, 1988; Herridge, Harrison, & Demaree, 1997), olfaction (Vermetten & Bremner, 2003), and gustation (Yamamoto, 2006) as well as voluntary motor networks including premotor (Foster & Harrison, 2004; Mollet, Walters, Harrison, & Holland, 2005; Walters & Harrison, 2006; Williamson & Harrison, 2003) and gross motor functions (Demaree, Higgins, Williamson, & Harrison, 2002; Harrison & Pauley, 1990).

Recently, the emotional circuits involved in hostility have been implicated in the modulation of pain (Mollet & Harrison, 2007). It follows then that, dizziness, motion sickness, and vection (an illusory sensation of directional self-motion) may also provoke substantial emotional reactions.

The cerebral relationships underlying emotion and vestibular processes are still a source of conjecture for two broad reasons. First, the limbic and prefrontal contributions to emotion are well established in the psychological literature (for a review see Mesulam, 2000; Williamson & Harrison, 2003). However, the field has not reached a definitive understanding of their role in vestibular processes. Second, as hemispheric laterality has been reliably demonstrated for emotion, the cerebral laterality literature in the vestibular modality has only come to light in the last 15 years. In the previous paper (Carmona, Holland, & Harrison, 2009) it was argued that regulation of dizziness and the experience of negative emotion serves as a concurrent dual task frontal capacity stressor, that increased or exacerbated the vestibular sensory area activation, in much the same way that Kallman and Isaac (1976) predicted that frontal capacity limitation would amplify visual or auditory stimulation. Specifically, the functional cerebral systems approach (Luria, 1973) and the functional cerebral space model (Kinsbourne, 1980) were invoked in order to provide a precedent model to apply to the integration of emotion and vestibular correlates. Parallels for both emotion and the vestibular modality were outlined for associative (frontal/posterior) and lateralized (right/left) activation. From the convergence of the emotion literature and the vestibular literature, a frontal capacity limitation model was postulated that integrated emotion with a dysregulated vestibular system.

But the current model of cerebral regulation depends on the assumption that frontal lobe capacity demands could have disordinate impact on posterior regions. Since negative affective correlates could serve as frontal lobe stressors, it would follow that the research integrating the

frontal lobes would theoretically extend to cases whereby emotional provocation might impact prognosis for vestibular impairments. The current study proposes to test the hypothesis of laterality and frontal lobe capacity by dissociating those individuals who exhibit relatively reduced right frontal capacity from those who exhibit relatively reduced left frontal capacity.

The paper is outlined in the following format: First the paper will review the background literature linking the right hemisphere with predominance for reception of negative emotion and the vestibular modality. Next, the paper will put forth a review of the frontal circuits overlapping the cerebral areas for vestibular processing and emotion. Next, the functional cerebral systems approach will be explicated and an explanation for the appropriateness of the Luria/Kinsbourne model will be provided. To further justify this approach we will indicate the shortcomings of two leading vestibular models which have not appreciated the regulatory capacity role of the frontal lobes in sensory experience and autonomic arousal. Finally, the review will lead to the rationale for the current investigation. The intent of the current experiment is to provide a theoretical foundation for the regulatory role of the frontal lobes over right hemisphere vestibular processing. We propose to use a non-verbal fluency measure in the service of this goal. Ultimately if evidence can be provided that challenges to frontal functional capacity compromises regulation of vestibular sensory regions, then this would provide the theoretical basis for the assertion that findings for executive involvement in the vestibular literature are based on the processing limitations of the frontal lobes to process both stress demands and vestibular correlates.

Evidence for Laterality in Emotion: The Right Hemisphere Predominance for Negative Emotions

In models for hemispheric specialization of emotion, it has become increasingly apparent that the hemisphere that is specialized for vestibular perceptual aspects is also specialized for

perception and experience of negative emotion broadly including anger, fear, and disgust (for recent comprehensive reviews of emotion theories see also Cox & Harrison, 2008; Demaree, Everhart, Youngstrom, & Harrison, 2005; Mollet & Harrison, 2007). Most prominently, the right hemisphere model asserts that expressive and receptive features of emotion are predominantly relegated to the right hemisphere (Heilman & Gilmore, 1998; Heilman, Scholes, & Watson, 1975). Other models have purported a left-hemisphere specialization for positive emotions (Davidson 1998; Davidson & Fox, 1982; Tucker, 1981).

In support of right hemisphere specialization are experiments demonstrating right hemisphere dominance in the visual modality for perception of negative emotional faces (Mandel, Tandon, & Asthana, 1991; Adolphs, Damasio, Tranel, & Damasio, 1996; Herridge, Harrison, Mollet, & Shenal, 2004; Wittling & Roschmann, 1993), and eye gaze during emotional provocation (Borod et al., 1988; Tucker, Roth, Arneson, & Buckingham, 1977); in the auditory modality with respect to emotional prosodic speech (Borod, Andelman, Obler, Tweedy, & Welkowitz, 1992; Borod et al., 1998, 2000; Emerson, Harrison, & Everhart, 1999; Schmitt et al., 1997); and through the somatosensory modality for negative emotional facial gestures (Herridge et al., 1997). It should be reiterated that these sensory modalities correspond to those that contribute to the vestibular network.

Heilman has postulated that emotional experiences are predicated on three dimensions: valence, motivation (approach/withdrawal), and arousal (Heilman & Gilmore, 1998). The importance of considering all dimensions in the study of emotion is underscored by the example of Wager, Phan, Liberzon, & Taylor (2003), who conducted a fairly recent meta-analysis of over 65 neuroimaging studies of emotion and brain asymmetry, in which they considered the dimensions of valence and motivation but neglected to include studies that controlled for arousal.

As a result, the meta-analysis failed to find support for the right hemisphere model. Yet when the level of arousal is controlled, evidence for right hemispheric dominance of negative emotion is supported (Canli, Zhao, Glover, & Gabrieli, 1998). The pertinence of arousal in the design of emotion paradigms is not a new concern (see Pizzagalli, Shackman, & Davidson, 2003 for cautions). Ultimately the issue of arousal is noted because the vestibular system mechanisms in the cortical sites are also intimately shaped by arousal as well (Furman, O'Leary, & Wolfe, 1981).

Evidence for Laterality in the Vestibulo-cortical Network

Technological advances in imaging have provided support for right hemisphere superiority for the vestibular modality. Specifically, several studies using a variety of methodologies have noted greater right hemisphere activation in the vestibular cortical projection areas (Bottini et al.1994, 2001; Dieterich et al., 1998, 2003; Fasold et al., 2002; Friberg, Olsen, Roland, Paulson, & Lassen, 1985; Janzen et al., in press; Kahane, Hoffmann, Minotti, & Berthoz, 2003; Lobel et al., 1998; Schlindwein et al., 2008). Moreover, diffusion tensor tractography has noted asymmetrically denser right-hemisphere white matter tracts connecting the posterior temporal lobe with the intraparietal lobe, which would anatomically support greater capacity for multimodal integration in these areas (Barrick, Lawes, Mackay, & Clark, 2007).

Kahane et al. (2003) directly stimulated the vestibular cortical areas while patients underwent epileptic foci localization. They found that patients endorsed counterclockwise (leftward) sensations of vection four times as much as clockwise vection, and that these were found most often with right hemisphere stimulation. This is consistent with studies of caloric irrigation (Dieterich et al., 2003) and otolith stimulation (Schwindlein et al., 2008) endorsing a sensation of leftward tilt commensurate with right hemisphere activations. Furthermore, evidence

indicates that right hemisphere integrity is associated with leftward gaze direction (Borod et al., 1988; Meador et al., 1989).

Neglect, the Right Hemisphere, and Vestibular Contributions

Support for right hemisphere dominance of vestibular function also derives from the hemineglect literature. Hemineglect refers to a disorder whereby the patient seemingly ignores the side of space contralateral to the impaired hemisphere. Neglect has been noted to occur in sensory modalities including vision (Heilman, Watson, & Valenstein, 2003), somatosensory (Smania & Agliotti, 1995), and audition (De Renzi, Gentilini, & Barbieri, 1989). The preponderance of hemineglect patients are characterized by temporal and parietal lesions in the right hemisphere (Critchley, 1966; Gainotti, Messerli, & Tissot, 1972; Heilman, Watson, & Valenstein, 2003; Leibovitch et al., 1998; Meador et al., 1988; Mort et al., 2003), especially in areas corresponding to vestibular functioning (Karnath, Himmelbach, & Kuker, 2003). This overlap has led some researchers to surmise that spatial neglect reflects a failure of vestibular processing of spatial representation at the cortical level (Brandt, 1999; Karnath & Dieterich, 2006; Philbeck, Behrmann, & Loomis, 2001).

In line with this theory, rehabilitative vestibular therapies that incorporate compensation for right hemisphere neglect syndromes (left neglect of extrapersonal space) have met with preliminary, but not indisputable success. Vestibular therapies have demonstrated temporary remission of hemineglect symptoms (mostly in right hemisphere impaired patients) through incorporating whole-body clockwise rotation (Philbeck et al., 2001) or left auditory canal caloric stimulation (Geminiani & Bottini, 1992; Bottini et al., 2005). Moreover, in a derivative of the classic study by Bisiach & Luzzati (1978) which validated representational hemineglect by having hemineglect subjects describe well-known landmarks in a Milan plaza from memory,

Rode and Perenin (1994) used a similar paradigm and found that irrigation of the left auditory canal (stimulation of the right hemisphere) improved memory recall of landmarks on the left side of a map of France.

Essentially then, the evidence suggests that the multiple inputs that converge in the vestibular cortical areas and the pronounced dominance of these inputs in the right hemisphere argue for overlapping neural networks underlying neglect. This asymmetry in vestibular functioning is consistent with the right hemisphere's predominance in models of global attention (Goldberg, Podell, & Lovell, 1994; Heilman, Watson, & Valenstein, 2003; Mesulam, 2000). In fact, Heilman and Van Den Abell (1980) provided evidence that while the left hemisphere is specialized for surveying the contralateral side of space, the right hemisphere is predominant for allocating attentional resources to both sides of extrapersonal space. These models would suggest that the vestibular contributions at the cerebral level may possibly serve as an adaptive sensory precursor to stages of attentional allocation favoring specialization of the right hemisphere.

Functional Cerebral Systems Theory

The functional cerebral systems model provides an explanatory account for the interconnectivity of the disparate cerebral, brainstem, and cerebellar systems. Luria's conception of a functional system entails "a complex dynamic 'constellation' of connections, situated at different levels of the nervous system, that, in the performance of the adaptive task, may be changed with the task itself remaining unchanged." (Luria, 1966, p.22). Luria proposed an organization of cerebral systems in which multiple units of the brain are connected through a hierarchy of analyzer modules for cortical tone and arousal, sensation and perception, and executive (regulatory) and inhibitory functions. His appreciation of distinct but cooperative functional areas of the brain and his organization of brain function into three distinct units is

instrumental in providing a model to understand cerebral systems interaction (Tupper, 1999). Fundamental to this model is the understanding that impairments can arise at different levels of a biological system and yet still appear similar in the functional outcome.

Luria's model provides for the organization of collective and interdependent cerebral systems, but insufficiently delineates the conditions under which emotion and vestibular processing interact. His model of shared organization of cerebral systems does not address the cognitive capacity limitations of dual task processing in vestibular functions. This aspect is important to emphasize since orientation in space and balance depend on concurrent streams of input from the visual, proprioceptive, and vestibular inputs. In effect, Luria's model outlines a hiearchy of relationships, but not the mechanisms by which the dynamic interplay exists among the cerebro-vestibular systems to suggest asymmetrical specialization for the vestibular modality.

In the introduction to Marcel Kinsbourne's *Asymmetrical Function of the Brain* (1978), Kinsbourne indirectly provides an example of emerging asymmetrical vestibular function, when he notes that, under normal circumstances, it is biologically adaptive for decreased laterality in cerebral processing during whole-body turning about the neuroaxis. Essentially, locomotion and balance are dependent on the ability to attend to both sides of space and on sensorimotor coordination across both hemibodies. Kinsbourne recognized that in healthy individuals vestibular encoding is processed at parallel levels within both hemispheres. However when the systems are compromised, asymmetrical functioning appears in behavior such as, for example, falling secondary to dizziness. In circumstances during which the demands of a task are not overly challenging, performance may, in fact, be enhanced. Excessive demands, however, utilize increasing cerebral resources such as attention allocation, autonomic control, and behavioral

comportment resulting in decreased cerebral capacity. During imperfect compensation for challenges, this imbalance may reveal specialized predominance of one hemisphere over another with heightened laterality effects.

Kinsbourne extended Luria's functional systems approach to propose a more specific model of cerebral activation under challenge conditions. According to his functional cerebral space model (Kinsbourne, 1980), cerebral networks devoted to multiple tasks can result in either facilitation or impairment of concurrent performance depending on the degree of task relatedness and how "close" in physical space those networks are to each other. If the tasks are highly related and are processed in close proximity within the brain, then performance of the task is expedited by the fact that a common network is shared. However, if the tasks are dissimilar and yet share common cerebral networks, then this would predict a poor outcome of performance. Essentially, excessive demands utilize increasing cerebral resources such as attention allocation, autonomic control, and behavioral comportment resulting in decreased cerebral capacity.

Dual processing tasks exemplify Kinsbourne's theory of shared cerebral space yielding interference in behavioral outcomes. In dual processing tasks, the processing of one task may lead to interference of a second consecutive or concurrent task (see Pashler, 1994, for a review of various dual task models). Proficient completion of dual tasks is dependent on the extent to which neural networks overlap. For example, in a dual processing task Chan and Newell (2008) found that performance of different primary task processes (object recognition versus spatial localization) was impacted by the degree of similarity to the distractor task, rather than by which sensory modality the task utilized. Essentially performance of these tasks entailed separate cerebral pathways for an object recognition task and a spatial perception task. When a distractor task that was slightly different also activated these same pathways, the resultant conflict yielded

interference in the primary task. Whether the distractor tasks were performed with either the visual or haptic stimuli was irrelevant since the modalities themselves have different primary sensory areas. Hence there is no conflict stemming from the dissimilarity in the sensory modalities.

Kinsbourne's model originally delineated the circumstances under which the hemispheres divided specialization for dissimilar tasks. Although, dual task performance can be more proficient when the tasks draw on resources within the same hemisphere (e.g. Hiscock & Kinsbourne, 1977; Yazgan, Wexsler, Kinsbourne, Peterson, & Leckman, 1995), this is not always the case (Boles & Law, 1998). Applying the model to the hemispheric specialization of emotion, Root, Wong, and Kinsbourne (1998) used a dual task approach of facial affect recognition and choice reaction time for each hand. They surmised that if emotional faces were presented concurrently to both cerebral hemispheres, responses would be faster at the left hand if the face displayed negative emotions, while response times would be faster at the right hand for positive emotions. The results of their facial affect recognition task showed that as expected, performance was most efficient when the hemisphere of emotional processing and the response hand were congruent; the right hand was faster for positive emotions, whereas the left hand was faster for negative emotions.

Current vestibular models fail to sufficiently incorporate prefrontal contributions in the sensory experiences of dizziness or disorientation. Most of these models extend to the sensory regions only. For example, Brandt and associates have postulated a sensory conflict model whereby they specified the circumstances under which certain sensory areas inhibit other sensory areas (Brandt & Dieterich, 1999; Brandt et al., 2002). For example, vestibular activation by caloric irrigation or Galvanic stimulation bilaterally activates the vestibular cortex, while

concurrently suppressing the visual association cortex (Brandt et al., 2002; Wenzel et al., 1996). Likewise, when the stimulus is primarily visual in nature the opposite pattern occurs, with relative suppression of the vestibular sensory areas (Brandt et al., 1998; Dieterich, Bucher, Seelos, & Brandt, 1998). In addition, this mutually inhibitory relationship has been demonstrated in comparisons of vestibular and somatosensory stimulation (Bense et al., 2001). Yet no attention has been devoted to the frontal lobe's role in selectively regulating the modalities.

Another previous model designed from studies of anxiety resulting from vestibular complaints comes close. According to Jacob, Furman, & Perel (1996), patients with vestibular disorders rely primarily on visual (Dieterich, Bauermann, Best, Stoeter, & Schlindwein, 2008) and secondarily on proprioceptive (Bles, de Jong, & de Wit, 1984) cues in order to negotiate the environment. However when this information is inadequate, deceptive, or confusing, the patient is integrating erroneous sensory information. The patients learn to become wary of false sensory integration, which develops into a constellation of fears about falling or the propensity to fall. The authors refer to this disorder as "space and motion discomfort" and have proposed inclusion into the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2000) as a separate entity. Support for the assertion comes from an experimental vestibular paradigm combining whole body rotation and mental arithmetic (Yardley et al., 1992). High state anxious individuals exhibited a significantly increased slow phase nystagmus component of the VOR (Yardley et al. 1992), indicative of risk for dizziness. Essentially, patients with emotional dysregulation combined with a failure to regulate the kinesthetic and visual inputs in vection display diminished performance on a frontal cognitive task relative to controls (further studies will be discussed in the section on anxiety and vestibular integration).

Like Brandt's sensory conflict model, the space and motion discomfort model restricts itself to sensory areas and does not incorporate a role for frontal regulatory mechanisms which were postulated by vestibular researchers in the animal literature (Akbarian et al., 1994; Nishiike et al., 2000). Although the model fails to specify the prefrontal mechanisms underlying these processes, it appears consistent with Kinsbourne's pre-existing cognitive load model, which is dependent on frontal capacity integration of multiple sensory inputs.

Hanes and McCollum (2006) raise a concern that the interpretation of the dual concurrent task literature may be complicated by unevenly taxing demands. That is, for example, a vestibular task such as maintaining postural balance on a tilting platform may inherently draw more cognitive resources compared with a concurrent mental arithmetic task, not due to cerebral competition, but simply because of the urgency of prioritizing safety. While this may be a legitimate caveat, this concern does not detract from the evidence suggesting the vestibular patient will likely exhibit relatively greater difficulty with dual processing compared with the normal patient. Vestibular demands impact cognitive processing in normal and brain damaged individuals, and indices of cognitive demands, even when the vestibular handicaps are minimized by keeping the participants stationary or seated, still indicate a differential impact on normal and impaired people.

In contrast, a pertinent limitation with the shared space model is that it was designed from studies using readily observable behavioral dual task paradigms such as performing a concurrent motor task and a speech task, or finger tapping and a speech task. Consequently, it fails to appreciate that autonomic regulation of emotion also serves as a cerebral demand task limiting prefrontal resources for other cognitive activities. Experiments within this laboratory have fruitfully extended the functional cerebral systems notion of dual processing demands to include

autonomic regulation through studies examining the impact of cognitive frontal tasks on cerebral activation and cardiovascular functioning in both hostile populations and anxiety-prone populations.

For example, Williamson & Harrison (2003) used concurrent fluency tasks sensitive to activation of either the left or right frontal lobe (also see Foster & Harrison, 2004) with directional and disordinate impact on parasympathetic and sympathetic activation in high hostile men. High hostile men demonstrated increased systolic blood pressure in response to a design fluency task challenging the capacity of the right frontal system (Foster & Harrison, 2004), whereas a verbal fluency task challenging left frontal capacity (Benton & de Hamsher, 1976) resulted in decreased systolic pressure. Moreover, high hostiles evidenced more perseverative errors in the design fluency task, a common clinical finding with diminished right frontal capability. Finally, Everhart and Harrison (2002) found that fluency tasks also negatively impacted heart rate and verbal generativity for anxious-depressed subjects.

If it is accepted that frontal lobe capacity to regulate cognitive processes is limited by the dual task of processing internal psychophysiological states, and therefore that cognition and autonomic functions compete for frontal lobe resources, then it is not altogether implausible that vestibular functions might also utilize crucial cerebral space that competes with executive processing, as well has behavioral restraint. This competition would predict a deleterious impact on the ability to preserve behavioral composure when stressed by concurrent vestibular challenge.

Hostility and the vestibular modality: Evidence for functional cerebral systems

Hostility may affect the vestibular modalityif there is a disturbed factor in the functional cerebral systems network. Specifically, in line with the Kinsbourne's cerebral space model,

anomalous vestibular sensations may arise if the overlapping substrates involved in a particular phase of anger processing clash with those involved in vestibular processing. Though the precise structures involved in both have not clearly been identified (but see Balaban & Thayer, 2001), it is sufficient to note that in rare cases where cortical areas associated with anger reactivity, autonomic lability, and vestibular sensation are impacted (namely right parietal and temporal regions) there may be a conflict that elicits vestibular symptoms. With regards to hostility, this laboratory has found ample evidence to support frontal regulation influence over the anterior temporal region (proximal to the amygdala and anterior insula) for autonomic reactivity (Demaree et al., 1996; Foster & Harrison, 2002, 2004; Williamson & Harrison, 2003).

Luria (1973) first noted cases of spatial delusions in the temporoparietal cortical areas, whereby patients believed that they were simultaneously present in two separate towns. Later, Everhart, Demaree, Harrison, & Williamson (2001) described the case of a man who sustained a closed head injury secondary to a motor vehicle accident. The patient reported feeling as if he were "forced into a box" and hurtled through extrapersonal space. The patient reported extreme hostility and homicidal cognitions associated with loss of spatial control during these delusions. EEG was recorded during an anger induction phase in which the patient was encouraged to imagine a spatial episode. Results indicated marked right hemisphere beta activation at temporoparietal electrode sites during the session.

The case study by Everhart et al. (2001) illustrates the possibility that vestibular interactions with anger or hostility may converge on a theme of control. Although evidence to support this hypothesis is sparse at this time, the assertion has been supported in the case study by Nighoghossian, Trouillas, Vighetto, & Phillipon (1992), who describe a patient with a right internal capsule infarct whose spatial delusion consisted of sensations of traveling through

European cities on various days. He insisted that he was leaving the house, despite the protests of his family.

Recently, research in this laboratory, using rotary vestibular stimulation, has found a relationship between vestibular function, hostility, and arousal that was only predicted on theoretical grounds using the functional cerebral systems approach. Twenty whole body rotations about the vertical neuroaxis elicited differences in autonomic arousal as a function of hostility level in a healthy population (Carmona Holland, Stratton, & Harrison, 2008). In contrast, a mild orthostatic tilt test, comprised of simply elevating the body to the upright position did not (Sloane et al. 2001). The conflicting findings suggest that strongly lateralized and/or unpleasant emotional vestibular paradigms, such as those inducing dizziness for example, may be a crucial factor in considering the relationship between vestibular and emotional networks.

In support of this assertion our laboratory pursued a case study using quantitative EEG recorded scalp activity in a woman with an anoxic encephalopathy who complained of extreme anger episodes and concomitant frequent panic attacks (Carmona, Holland, Foster, Harrison, & Harrison, 2008). EEG was recorded while the woman was instructed to mentally rehearse imagery of a recently stressful attack. The stress imagery yielded prominent bilateral delta activity at the frontal lobes and a surge in right hemisphere beta, with clinical correlates of sympathetic activation (profuse sweating), leftward vection, left facial synergy, and fear. A single-subject experiment was undertaken to follow up the case study's findings on frontal/temporal interactions. In order to maximally tax frontal capacity in the vestibular modality, a female volunteer was subjected to whole-body passive rotation to the point of dizziness and nausea under recording of Quantitative EEG pre- and post-rotation. As the model

predicted, there was heightened delta band activity across the frontal electrode sites and pronounced beta, over the right temporal and parietal sites. Delta band activity has been associated with adverse states such as mental lethargy (Fernandez et al., 1995), as well as with various pathological disorders such as Major Depressive Disorder (Nyström, Matousek, & Hällström, 1986), Schizophrenia (Fehr et al., 2003), and Alzheimer's Disease (Valladares-neto et al., 1995). Beta band activity is typically associated with increases in arousal to emotionally charged stimuli (Foster & Harrison, 2002; Ray & Coles, 1985; Schellberg, Besthorn, Pfleger, & Gasser, 1993). Interestingly, in our single-subject experiment the delta at the frontal sites was slightly higher at the right frontal sites, consistent with our right-hemisphere hypothesis, though this did not reach the level of statistical significance.

A Proposed Integration Model for Competing Vestibular and Emotion Systems

The findings from the hostility experiments and case studies have led to the formulation of a theory for the relationship amongst cerebral systems. It is proposed that under vestibular challenges frontal resources are burdened to capacity, with diminished ability to allocate resources for attenuating activations in the posterior regions. The prognosis for maintenance of composure under stress will likely be poorer for those prone to risk for decompensation to stress from intense negative experiences. This stress may include concerns of dizziness, disorientation, motion sickness, anxiety or even anger over lack of control for some individuals. These negative experiences compound the task of maintaining vestibular processes including maintenance of balance and coordination, Vestibulo-ocular reflexes (VOR), arterial blood pressure during posture and locomotion, cardiovascular control, vasodynamics of blood circulation during gravity challenges, and coordination of gastrointenstinal responses. In effect, if cognitive capacity limitations of the prefrontal region are exceeded, this would predict a poor outcome for

maintaining composure of sensorimotor coordination underlying balance as well as the critical autonomic features for restraining arousal. The experience of salient negative emotional associations within the right hemisphere result in a challenge that further depletes the resources of the prefrontal regions for regulation over key limbic areas in vestibular processes.

The more posterior the impact of the dysfunction in the right hemisphere, the more disorientation and possibly dizziness, without negative affective correlates becomes the dominant clinical feature. For example, posterior temporal lobe and posterior insula impairments appear to be associated more with dizziness and disorientation, than with unpleasant concomitants such as anxiety or nausea (Brandt, Bötzel, Yousry, Dieterich, & Schulze, 1995; Bogousslavsky, Caruzzo, Meuli, & Maeda, 1997; Cereda, Ghika, Maeda, & Bogousslavsky, 2002; Papathanasiou et al., 2006). The temporal lobes appear to be a crucial transitional and interface zone linking the spatial processing properties of the vestibular cortex (which formally extends into the superior temporal lobe) and the autonomic properties of the prefrontal and limbic emotional centers. Anatomical support for this assertion can be found in the research linking right asymmetry for temporal and parietal interconnectivity (Barrick et al., 2007; Spena, Gatignol, Capelle, & Duffau, 2006).

Summary of Main Points

This paper has indicated historical linkages with the vestibular system and emotional functions. In merging disparate perspectives to inform a neuropsychological understanding the following main points are summarized:

First, the research has established that the shared networks are asymmetrically distributed within the hemispheres. The preponderance of evidence from optokinetic, caloric irrigation, and direct stimulation studies in the vestibular literature suggests that the right hemisphere appears

specialized for vestibular awareness, including perception of the horizontal displacement and illusory motion. Furthermore, the onset for this lateralization may be as early as during prenatal development. The hemineglect literature provides behavioral implications for right hemisphere superiority in the multisensory vestibular areas.

Second, there is evidence that the vestibular brainstem apparatus contributes substantially to distributed multisensory areas within the cerebral hemispheres. The vestibular nuclei are the first point of input from the vestibulocochlear cranial nerve and project to the cerebral hemispheres. The vestibular cortical areas have linkages to the motor and premotor cortex areas for balance and voluntary movement coordination. The cortical areas also have linkages both directly and indirectly with limbic areas within the prefrontal regions. The prefrontal regions are implicated in a number of concurrent regulatory roles including attention to internal affective state, affective modulation of motor components, attenuation of sensory overload, determining the appropriate sensory input to attend, autonomic inhibition during stressful vestibular challenges, and affective appraisal of dizziness and disorientation.

Extension of the model to the present study

In the current paper, we propose to test the hypothesis that frontal lobe capacity determines the posterior sensory activation during vestibular challenge using a whole-body rotary paradigm. Given the vast literature implicating the right hemisphere in vestibular and emotional regulation, we would expect that those who exhibit reduced right frontal capacity to regulate posterior sensory and autonomic structures would be more likely to exhibit greater reactivity in these areas to a vestibular stressor. We operationalize those with reduced frontal capacity by considering relative performance on a nonverbal design fluency.

Research with nonverbal fluency tasks tend to show a significant reliance on greater right hemisphere functional integrity (Foster, Williamson, & Harrison, 2005; Jones-Gotman & Milner, 1977; Ruff, Allen, Farrow, Niemann, & Wylie, 1994). Both education and age also affect design fluency (Kraybill & Suchy, 2008; Salthouse, Atkinson, & Berish, 2003). Moreover, research has implicated design fluency inefficiency for hostility (commonly associated with right hemisphere dysfunction) and deleterious impact on cardiovascular regulation (Williamson & Harrison, 2003) and blood-glucose levels (Holland & Harrison, 2008; Walters & Harrison, 2006). Foster et al. (2005) utilized performance on a design fluency task as a grouping factor by dividing the sample population into high design fluent and low design fluent groups and investigating QEEG differences at right frontal sites. They found that the low design fluent group exhibited greater delta magnitude at the right frontal sites (F2, F4, and F8).

Demakis and Harrison (1997) found a small, but significant association between letter fluency and design fluency. Hence, it is probable that those who are proficient at the design fluency task might also be proficient at the verbal fluency task. In fact, using a factor analysis design Salthouse (2005) found that both executive tasks load highly on processing speed ability. Given that dizziness may have bifrontal effects (but greater right hemisphere sensory area effects) it then follows that examining the effects of frontal integrity must take into account the within-person's performance on *both* verbal and nonverbal performance. The current experiment proposes to examine performance on a non-fluency measure (the Ruff Figural Fluency Test) using a two phase experimental design to examine the effects of dizziness-inducing whole-body rotation in a population of relatively low non-verbally proficient and high verbally proficient individuals. In the first phase we will acquire our fluency groups. In the second phase we will

record QEEG and skin conductance level both before and following whole-body rotation and compare performance on a nonverbal fluency task following rotation.

Hypotheses

1. The current experiment intends to replicate and extend the results of Foster et al. (2005). It is expected that both groups to show pronounced delta activity in the frontal lobes following whole-body rotation, but moreso at right frontal sites commensurate with our hypothesis. Furthermore it is expected that the right frontal delta activity would be greater for those with low design fluency.

2. Additionally, QEEG activation will be compared for both groups following whole body rotation according to the procedures of Carmona et al. (2008). It is expected that those with low design fluency will exhibit greater beta activation over posterior sites, consistent with the previous case study as well as the abundant line of hostility research showing this pattern under stress. In line with the theory of frontal regulatory capacity over posterior sites under stress challenge, it would be expected that if indeed vestibular stimulation disproportionately impacts right hemisphere sensory areas, then right frontal lobe capacity to regulate these areas would be diminished.

3. It is predicted that those who perform poorly on a design task will exhibit greater perseverative designs on the design fluency task as compared with those who perform well.

4. It is hypothesized that following rotation, administration of the design fluency task will yield proportionately smaller improvements on the second administration of the design fluency task compared with the first administration given that vestibular stimulation should impact the right hemisphere.

5. It is predicted that there will be no significant GSR differences between the two groups at baseline.

6. It is predicted that GSR differences will increase following rotation. This increase will be greater for those assigned to the low design fluency group as compared with those in the high fluency group given that increases in GSR has been associated with vestibular induced motion discomfort.

Methods

Participants

The research was approved by the Psychology Department Human Participants Committee and by the Institutional Review Board of Virginia Polytechnic Institute and State University. Participants were women, age 18-25, recruited from the undergraduate psychology pool at Virginia Tech. They were granted extra credit and a chance for a bonus raffle for two $20 amazon gift certificates for their participation. A total of 293 participants attended the laboratory screening. Descriptive statistics of the screening phase are listed in Table 1. From the laboratory screening, 82 participants volunteered to return for the experimental phase in which EEG and GSR were recorded.

From this dataset, the analyses were conducted on 58 participants who met group criteria (29 per group). The final group screening resulted in the categorization of 29 participants who performed poorly on a design fluency task (Low RFFT group) and 29 participants who excelled on the design fluency task (High RFFT group). Those in the Low RFFT group had to score at least a half standard deviation below the mean of the entire sample, whereas those assigned to the High RFFT had to demonstrate performance above a half standard deviation above the mean of the entire sample.

The selection of women was based on the literature showing that women exhibit significantly more dizziness and lightheadedness complaints compared with men (Bailey, Sloane, Mitchell & Preisser; Baloh & Halmagyi, 1996; Kwong & Pimlott, 2005); are at greater risk for orthostatic hypotension and dizziness (Wu, Yang, Lu, Wu, & Chang, 2008); and show higher risks of falling due to vertigo (Jönsson, Sixt, Landahl, & Rosenhall, 2004).

Self-Report Measures

Participants were administered the Medical History Questionnaire (see Appendix A), the Coren, Porac, and Duncan Laterality Inventory, the State Trait Anxiety Inventory-Trait, the Cook-Medley Hostility Questionnaire, and the Beck Depression Inventory-II. Those completing the second phase of the experiment were administered the State Trait Anxiety Inventory- State and the Nausea Profile.

The Medical History Questionnaire screened for the purposes of excluding the following pathologies: disturbances of vestibular functioning, including dizziness, vertigo, ataxic gait, nystagmus, ear infections, inner ear problems, tinnitus, hearing aid use, headaches, Ménières Disease, pathology of the middle/inner ear, and other significant neurological disorders. Those without significant medical or neurological problems that could compromise the findings were eligible to participate in the study.

Participants' hemibody preference or "handedness" was assessed using the Coren, Porac, and Duncan Laterality Inventory (Coren, Porac, & Duncan 1979; CPD). This inventory examined hemibody preference through thirteen questions targeting participant's functional preference for use of the left or the right hand, foot, eye, and ear. Test-retest reliability yielded a 98% concordance between lateral preference and behavioral indicators over the course of one year (Coren, Porac, & Duncan, 1978). Right hemibody preference was assigned positive values

while left hemibody preference was assigned negative values. The scores ranged from +13 (full right hemibody preference) to -13 (full left hemibody preference). A score of zero indicated perfect ambidexterity. Participants were included if they meet the score requirement of +5 or above on this instrument.

In addition, emotion questionnaires were administered for exploratory purposes. Anxiety was assessed via scores on the State Trait Anxiety Inventory (STAI) (Spielberger, 1983). The STAI is a well-known self-report measure consisting of two 20-item forms designed to assess transient, temporary State anxiety (S-Anxiety, Form Y-1) and another form designed to assess "relatively stable individual differences in anxiety proneness" (Trait anxiety, T-Anxiety, Form Y-2) (Spielberger, 1983) . The STAI has been used in over 3,000 studies and has been translated into more than 40 languages (Spielberger, 1989). The scale has been shown to have excellent test-retest reliability and internal consistency (Barnes, Harp, & Jung, 2002; Quek, Low, Razack, Loh, & Chua, 2004).

Hostility was assessed by scores on the Cook-Medley Hostility Scale (CMHS; Cook & Medley, 1954). The CMHS is a well-known 50- item self-report measure derived as a subscale of the MMPI (Hathaway & McKinley, 1943). The CMHS is the most widely used measure of hostility (Contrada & Jussim, 1992). The scale has been shown to have excellent test-retest reliability and internal consistency (Christensen, Wiebe, & Lawton, 1997; Smith & Frohm, 1985).

Apparatus

Laboratory. All testing was conducted in a sound-attenuated room located within the Behavioral Neuroscience Laboratory at Virginia Tech. Following administration of the Informed Consent Forms and a review of medical history the participant was led into the lab room and

seated in a black leather cushioned rotary-capable Engage™ Seating chair (Krueger International, Inc.) mounted on a 24 x 48 in. wooden platform with the base elevated 7.5 in. from the ground floor (see Carmona et al., 2008). The participant's total elevation from the ground floor was approximately 28 inches. A 556CN dual timer oscillator circuit (Radioshack, Inc.) was configured with a 2 in. diameter speaker and attached unobtrusively to the back of the chair using Velcro attached to a plastic housing unit to protect the circuit. The circuit served as a digital timer that emitted a 50 db click every 3 seconds to guide the assistant in regulating rotary velocity (see Carmona et al., 2008). The rotary chair was enclosed by several white sheets mounted from the ceiling to create a 6 ft. circular enclosure in order to limit visual stimulation. Onset and cessation of rotation was fixed in the same position across participants. The participant was seated upright and facing forward.

The administration of all manipulations was controlled and double-blinded. Participants were unaware of group designation. In addition, the preparation and manipulation were administered by assistants who had no foreknowledge of the participant's group designation. All inventories were scored by the assistants. Furthermore, all participant EEG preparation, rotary stress, and fluency test procedures were performed entirely by assistants. The primary investigator served in a supervisory capacity. In addition, all instructions were standardized and recorded onto CD for playback via CD player speaker [Koss model 4X/Plus] during the entire duration of the experiment in order to further minimize experimenter bias. As in Carmona et al. (2008) undergraduate assistants were trained in the application and timed pacing of rotary stress using various weights and practice volunteers for one month prior to initial engagement in the experiment. Following seating of the participant in the chamber and supervision of electrode placement, both the experimenter and the recording apparatus were entirely out of view for the

duration of the experiment to control for experimenter bias effects. The participant was isolated within the curtain chamber for the duration of the experiment with the exception of the entry of the assistant for rotation.

Design Fluency Measure. Participants completed the Ruff Figural Fluency Test (RFFT) (Ruff, Light, & Evans, 1987; Ruff, 1988), a measure of design fluency. Participants were given a repeating matrix of dots within a series of grids from which participants are instructed to connect two or more dots using straight lines in order to create a unique design. There are five trials of different dot matrices with increasing levels of distraction and effort (Ruff et al., 1987). The RFFT is scored by counting the total number of unique designs generated minus the number of perseverative errors per trial. A perseverative error is defined as a repetition of a previously generated design on that trial. The RFFT has been demonstrated to be an effective measure for right hemisphere function in patients with lesions in the right frontal region of the brain (Baldo, et al., 2001; Ruff, et al., 1994) as well as in healthy individuals (Foster, et al., 2005). Moreover the RFFT has shown acceptable concurrent validity with the Design Fluency Test (r = .38), another test reputable test of nonverbal generativity (Demakis & Harrison, 1997). In previous experiments we have administered the RFFT to assess normal, hostile, and anxious populations (Demakis & Harrison, 1997; Everhart & Harrison, 2002; Foster, et al., 2005; Williamson & Harrison, 2003).

Letter Fluency Measure. Participants completed the Controlled Oral Word Association Test (COWAT) (Benton & Hamsher, 1976). The test is comprised of 5 trials of letters presented to the participant (see Appendix B). Participants must orally generate as many words as possible within a 1 min. timeframe. They are not permitted to endorse proper nouns, numbers, or the same word with various suffixes. A perseverative error is defined as a repetition of a previously

generated word on that trial. Typically the letters administered are F, A, and S. Alternate forms also utilize letter sets CFL and PRW, which are comparable in difficulty (Ruff, Light, Parker, & Levin, 1996). Furthermore, evidence indicates that there is no significant difference between total verbal output for FAS and CFL letter combinations (Lacy, et al., 1996; Troyer, 2000). Therefore in order to align the number of trials of the verbal fluency task with the nonverbal fluency task, we have included the letters "C" and "L" in addition to the standard FAS test. Additionally, in the current experiment the participants will be required to write their responses as opposed to respond verbally, however this should have a negligent effect on their performance as written responses have been demonstrated to correlate highly with oral performance (r = .81) (Cohen & Stanczak, 2000).

Physiological apparatus

QEEG. In order to test the hypothesis of right hemisphere fluctuation from pre- to post-rotation, quantitative electroencephalography (EEG) activity will be collected and analyzed using the EEG Analysis System software developed by the James Long System (Canoga Lake, NY.). EEG will be recorded and digitized at a sampling rate of 512 Hz, with a high-pass filter set at 0.1 Hz, and a low pass filter set at 100 Hz. Participants were fitted with a custom-made, appropriately-sized cap enabling 22-channel recording with tin electrodes for EEG and tin drop electrodes for left canthus recording of EOG (Electrocap International). The impedance for each electrode was below 5 kohms. The 19 electrode sites from which data were recorded were arranged in accordance with the International 10/20 System. A common-vertex reference (CZ) was recorded and averaged-ear references were computationally derived consistent with prior experiments in this laboratory (e.g. Everhart, et al., 2008). Frequency bandwidths were classified as: delta = 1-4 Hz and beta = 13-21 Hz. The removal of eye and muscle artifact from

recorded EEG data was done manually before analyses. In order to record eye movements, horizontal EOG was recorded from the outer canthus of each eye and supraorbitally above and infraorbitally from the left eye (vertical eye movements). EEG was examined and analyzed using software from the James Long Company.

Galvanic Skin Response (GSR). Skin conductance was collected and analyzed using the Physiology Analysis System software developed by the James Long System (Canoga Lake, NY.). Participants were instructed to wash their hands with non-abrasive Ivory liquid soap (Procter & Gamble Co.) and warm water upon entry to lab for the second portion of the experiment. Isotonic 1cm. diameter silver-silver chloride electrodes (JLC, Canoga Lake, NY.) were placed on the volar surface of the medial phalanges of the left hand with salt-free electrode gel (Spectra 360, Parker Laboratories, Inc.). Electrodermal activity was recorded via transducers connected by cable to a 32-channel bioamplifier, which was then routed to an A/D interface converter. Skin conductance was examined and analyzed using software from the James Long Company.

Procedures

See Appendix C, D, and E for diagrams of the experimental protocol with complete administrative instructions. In Phase I, participants were scheduled via the SONA Experiment Management System for testing on the RFFT measure with the goal of assembling two experimental groups based on their proficiency on the RFFT (Low RFFT and High RFFT groups). Participants completed an Informed Consent Form (see Appendix F). Next they were administered the Medical History Questionnaire, Laterality Inventory, STAI-T (Form Y-2), BDI-II, and CMHS. Once they completed these questionnaires, they were either administered the RFFT or the COWAT first. The order of administration was counterbalanced. Pending

determination of eligibility for group entry, participants were scheduled for participation in phase II of the experiment.

To participate in Phase II of the experiment participants were required to indicate consent to participate via a second Informed Consent Form (see Appendix F). Shortly after arriving at the laboratory the participant was seated at a desk. They completed the STAI-S (Form Y-1). After completion, the participant was taken by the experimenter assistant to the enclosure, seated in the chair and prepared for EEG recording.

Baseline. Following placement of the electrodes and preparation of the recording apparatus, the experimenter assistant departed the enclosure. The experimenter entered the enclosure to verify proper placement of electrodes, reconfirm impedance levels, and proper positioning of the chair in at the starting point. The participants were instructed to sit quietly, to relax, to keep their eyes closed, and to remain as still as possible throughout the remainder of the procedures. To put the participant at ease and to mitigate the risks of extreme dizziness and nausea associated with vestibular stimulation, the experimenter verbally informed the participants of their freedom to terminate the experiment at any time and that the maximum duration of the rotation exposure was about 1 minute. The importance of remaining as still as possible and keeping their eyes closed was emphasized in an effort to minimize movement artifact during recording. The participant was permitted an opportunity to ask any questions regarding this phase of the investigation. After two minutes of relaxation baseline, EEG recordings commenced. A total of 120 one-second epochs constituted the QEEG baseline measurement. Two minutes of electrodermal recording were also taken simultaneously.

Rotation. The rotation paradigm was identical to Carmona et al. (2008). Each participant underwent angular rotation in the clockwise direction at a rate of approximately 120 º/s as paced

by the timer circuit. Onset and cessation of rotation occurred at the same starting position. After 20 rotations, the participant was abruptly stopped and the experimenter initiated 2 minutes of EEG recordings and GSR recordings. Following this period, a 2-minute rest period commenced.

Post-stress. Following EEG recordings the participants completed the RFFT. Participants were required to complete the STAI-S again. Before leaving the lab, the participant was debriefed and provided the opportunity to have any questions answered about the experiment. At any time following participation, participants were able to have access to the study's website, which included notice of Internal Review Board study approval.

Results

An alpha level of .05 was used for all statistical tests.Since the order of fluency test administration was counterbalanced, a 2-way Analysis of Variance (ANOVA) was conducted to test for the interaction effects of Order (RFFT administered first, COWAT administered first) by Group (Low RFFT vs. High RFFT) on raw fluency test scores. The purpose of the analysis was to ensure that the groups did not differ based on whether they received a verbal fluency task or nonverbal fluency task first. There was no significant Group x Order interaction effects for either the COWAT ($F(1, 54) = .05$, $p = .89$) nor the RFFT ($F(1, 54) = 2.24$, $p = .14$), confirming that the completion of either the RFFT or COWAT first did not significantly impact overall fluency performance.

Questionnaires/Group Descriptive Statistics

For the High and Low RFFT groups, the scores on the CMHS, STAI, BDI-II, laterality questionnaire and Nausea Profile are listed in Table 2. A one-way between groups ANOVA was performed on the laterality questionnaire to ensure statistical equivalence in laterality.

Accordingly, there was no significant difference between group scores on the laterality questionnaire, $F(1, 56) = .008$, $p = .93$.

For descriptive purposes only, one-way ANOVAs were performed on the scores of the Laterality Questionnaire, STAI-T, CMHS, BDI-II, and NP. The results do not indicate group differences on any of these self-report measures (see Table 3). The STAI-S was administered at baseline and immediately following rotation. The effects of Group (Low RFFT vs. High RFFT) and the repeated measure of Condition (Baseline and Post-stress) on STAI-S scores were tested by a balanced mixed two- factor Analysis of Variance (ANOVA) design (see Table 4). There were no significant differences noted on this questionnaire.

Initial RFFT Task

To demonstrate that both experimental groups differed on the overall total number of designs produced, a one-way analysis of variance (ANOVA) was performed using the total number of designs produced as the dependent variable. Both groups differed on the total number of designs produced, $F(1, 56) = 543.3$, $p < .01$. As expected, the High RFFT group ($M = 115.8$, $SD = 12.7$) produced over twice the mean number of designs as compared with the Low RFFT group ($M = 55.4$, $SD = 5.6$).

A 2-way mixed design analysis of variance (ANOVA) was performed using number of designs produced as the dependent variable (see Table 5). Data were analyzed with the between groups factors of Group (Low RFFT vs. High RFFT) and with the repeated measure of Trial (Trials 1-5). All post-hoc pairwise comparisons were made using Tukey's Honestly Significance Difference Test to control for Type I error (Winer, 1971).

There was a main effect for Group ($F(1, 56) = 543.3$, $p < .01$). The results confirmed a significant difference in design productivity per trial for those assigned to the High RFFT group

(M = 23.15, SD = 3.6) as compared with those in the Low RFFT group (M = 11.1, SD = 2.1). In addition, the analyses yielded a main effect for Trial ($F(4, 224) = 8.6$, $p < .01$). As displayed in Figure 1, post-hoc analyses revealed that there were no differences in number of designs produced between Trials 2-5. Overall, Trial 1 produced the least number of designs.

To compare the two groups on the overall number of RFFT errors produced, a one-way analysis of variance (ANOVA) was performed using the total number of errors produced as the dependent variable. Both groups differed on the total number of RFFT errors produced, $F(1, 56) = 11.5$, $p = .001$. Most likely due to the higher productivity, the High RFFT group (M = 1.6, SD = 2.1) produced a greater total number of errors as compared with the Low RFFT group (M = 4.7, SD = 4.4).

EEG Results

An alpha level of .05 was used for all statistical tests. Separate mixed design analyses of variance (ANOVAs) were performed using delta and beta bandwidth as the dependent variables. The effects of Group (Low RFFT vs. High RFFT), Condition (Baseline and Post-stress) and Site (F8 and T6) on mean delta bandwidth (1-4 Hz) magnitude (μV) and mean beta bandwidth (13-21 Hz) magnitude (μV) were tested by a balanced mixed three-factor Analysis of Variance (ANOVA) design (see Table 6 and 7). All post-hoc pairwise comparisons were made using Tukey's Honestly Significance Difference Test to control for Type I error (Winer, 1971).

For delta activity, a significant main effect was found for Condition, $F(1, 56) = 4.27$, $p = .04$. For both groups, delta activity increased from baseline (M = 6.7 μV, SD = 2.1) to post-rotation (M = 7.0 μV, SD = 2.7). A significant main effect was also found for Site, $F(1, 56) = 52.1$, $p < .01$. Overall, the frontal site, F8, yielded M = 8.1 μV (SD = 2.6), whereas T6 yielded M = 5.6 μV (SD = 1.4). Moreover, an interaction effect of Condition x Site was found; $F(1, 56) =$

9.2, p = .004 (See Figure 2). Post-hoc analyses reveal that at T6 delta magnitude levels were significantly lower than their respective levels at F8. Delta activity was greater at F8 following rotation, as compared to baseline.

For Beta, a significant main effect was found for Group, $F(1, 56) = 8.1$, p = .006. Overall, the Low RFFT group, yielded $M = 2.9$ μV ($SD = .8$), whereas the High RFFT group yielded $M = 3.4$ μV ($SD = 1.2$). A second main effect was found for Site, $F(1, 56) = 118.4$, $p < .001$. Overall, the frontal site, F8, yielded $M = 2.50$ μV ($SD = .5$), whereas T6 yielded $M = 3.78$ μV ($SD = 1.0$). Moreover, an interaction effect of Group x Site was found; $F(1, 56) = 4.84$, p = .032 (See Figure 3). The High RFFT group yielded greater beta activation than the Low RFFT group at T6. There was no difference between the two groups on beta activation at F8, which was generally lower than at the posterior site, T6. Additionally, an interaction effect of Site x Condition was found; $F(1, 56) = 10.3$, p = .002 (See Figure 4). Post-hoc analyses reveal that there was no difference between the beta activity levels at F8. Post-hoc analyses reveal that T6 beta magnitude levels were significantly higher than their levels at F8, with slightly higher beta activity at Baseline than at Post-rotation levels.

Analyses were expanded to examine the mean theta bandwidth (5-7 Hz) magnitude (μV) (see Table 8). A significant main effect was found for Condition, $F(1, 56) = 9.36$, p = .003. Overall, theta decreased from baseline ($M = 2.99$ μV, $SD = 1.1$) to post-rotation ($M = 2.86$ μV, $SD = 1.1$). In addition, a second main effect was found for Site, $F(1, 56) = 19.9$, $p < .01$. Overall, the frontal site, F8, yielded $M = 2.7$ μV ($SD = .74$), whereas T6 yielded $M = 3.2$ μV, ($SD = 1.3$).

Moreover, an interaction effect of Group x Site was found; $F(1, 56) = 5.6$, p = .02 (See Figure 4). Post-hoc analyses reveal that theta at T6 for the High RFFT group was altogether greater than for theta at T6 for all other levels (see Figure 5). A second interaction was also noted

for Site x Condition; $F(1, 56) = 9.23$, p = .004 (See Figure 6). Essentially theta activity declined slightly at T6 following rotation, whereas theta activity remained stable at F8 over the conditions.

Skin Conductance Level Results

An alpha level of .05 was used for all statistical tests. Separate mixed design analyses of variance (ANOVAs) were performed using skin conductance level (μS) as the dependent variable (see Table 9). Data were analyzed with the between groups factors of Group (Low RFFT vs. High RFFT) and with the repeated measure of Condition (Baseline and Post-stress). All post-hoc pairwise comparisons were made using Tukey's Honestly Significance Difference Test to control for Type I error (Winer, 1971). One subject from the Low RFFT group (subject 11) and one subject from the High RFFT group (subject 17) were excluded from the analyses due to equipment malfunction whereby the electrode required replacement.

A significant main effect was found for Condition, $F(1, 54) = 46.60$, $p < .0001$. As expected, participants' skin conductance increased significantly from baseline to post-rotation. Prior to rotation (Baseline) the mean skin conductance level was 5.19 μS ($SD = 3.26$), whereas following rotation (Post-stress) the mean skin conductance level was 6.14 μS ($SD = 3.67$). No other significant main or interaction effects were noted. Hypotheses predicting a Group x Condition interaction were not confirmed.

In order to reduce skew and kurtosis common to SCL data, mixed design analyses of variance (ANOVAs) were performed using the log of SCL as the dependent variable as recommended by Venables and Christie (1973). Next, Data were again analyzed with the between groups factors of Group (Low RFFT vs. High RFFT) and with the repeated measure of Condition (Baseline and Post-stress). The ANOVA results are displayed in Table 10.

Again, a significant main effect was found for Condition, $F(1, 54) = 43.66$, $p < .0001$. With transformed scores, participants' skin conductance increased significantly from baseline to post-rotation. Prior to rotation (Baseline) the mean skin conductance level was 1.49 ($SD = .55$), whereas following rotation (Post-stress) the mean skin conductance level was 1.65 ($SD = .60$). A trend was noted for the interaction of Group x Condition was found; $F(1, 54) = 2.94$, $p = 0.09$. Essentially, at baseline both groups were equivalent, but following rotation the High RFFT group had greater Log SCL than the Low RFFT group (see Figure 7). No other significant main or interaction effects were noted. Hypotheses predicting a Group x Condition interaction were not confirmed.

Second RFFT Administration

One subject in the Low RFFT group did not complete the second administration of the RFFT. Therefore the calculations were performed using the General Linear Model procedure. A 2-way mixed design analysis of variance (ANOVA) was performed using number of designs produced as the dependent variable (see Table 11). Data were analyzed with the between groups factors of Group (Low RFFT vs. High RFFT) and with the repeated measure of Trial (Trials 1-5). All post-hoc pairwise comparisons were made using Tukey's Honestly Significance Difference Test to control for Type I error (Winer, 1971).

As before, the High RFFT group ($M = 27.2$, $SD = 3.4$) outperformed the Low RFFT group ($M = 14.8$, $SD = 4.1$) on each trial. In addition, the analyses yielded a main effect for Trial ($F(4, 216) = 8.8$, $p < .01$). As displayed in Figure 8, post-hoc analyses revealed that there were no differences in number of designs produced between Trials 1-3. Trials 2, 4, and 5 also did not differ. On the second administration a Group x Trial interaction emerged ($F(4, 216) = 5.9$, $p < .01$. As displayed in Figure 9, the High RFFT group produced a greater number of designs on

Trials 1-3 as opposed to Trial 5-4, whereas there were no differences in productivity across Trials 1-5 for the Low RFFT group.

To compare the two groups on the overall number of RFFT errors produced on the second administration, a one-way analysis of variance (ANOVA) was performed using the total number of errors produced as the dependent variable. Both groups again differed on the total number of RFFT errors produced, $F(1, 55) = 41.6$, $p < 01$. Most likely due to the higher productivity, the High RFFT group ($M = 6.1$, $SD = 4.1$) produced a greater total number of errors as compared with the Low RFFT group ($M = .9$, $SD = 1.2$).

In order to compare the performance of the initial RFFT administration with the second administration, the percentage of change score was calculated to examine the change from the first to the second administration. Next, a one-way analysis of variance (ANOVA) was performed using the change score as the dependent variable. Both groups differed on the amount of change exhibited from the first to the second administration ($F(1, 55) = 5.5$, $p = 02$). The Low RFFT group exhibited a greater mean proportionate increase in design productivity ($M = 23\%$, $SD = 17\%$) as compared to the High RFFT group ($M = 15\%$, $SD = 7\%$).

COWAT Task

To compare the two groups on the overall total number of words produced, a one-way analysis of variance (ANOVA) was performed using the total number of words produced as the dependent variable. Both groups differed on the total number of words produced, $F(1, 56) = 6.5$, $p = .01$. As expected, the High RFFT group ($M = 70.7$, $SD = 15.1$) produced a greater total number of words as compared with the Low RFFT group ($M = 61.4$, $SD = 12.3$). The outcome was expected given the positive correlation between non-verbal and verbal fluency (Demakis & Harrison, 1997).

A 2-way mixed design analysis of variance (ANOVA) was performed using number of words produced as the dependent variable (see Table 12). Data were analyzed with the between groups factors of Group (Low RFFT vs. High RFFT) and with the repeated measure of Trial (Trials 1-5). All post-hoc pairwise comparisons were made using Tukey's Honestly Significance Difference Test to control for Type I error (Winer, 1971).

There was a main effect for Group ($F(1, 56) = 6.9$, $p = .01$). As expected, per each trial, those who performed well on the RFFT also performed well on the COWAT ($M = 12.3$, $SD = 3.7$) as compared with those in the Low RFFT group ($M = 14.2$, $SD = 3.3$). In addition, the analyses yielded a main effect for Trial ($F(4, 224) = 18.9$, $p < .01$). As displayed in Figure 10, post-hoc analyses revealed that Trial 3 (words beginning with letter "s") produced the most words, whereas Trial 2 (words beginning with letter "c") produced the least words. There was no significant difference in production for Trials 1, 4, and 5.

To compare the two groups on the overall number of COWAT errors produced, a one-way analysis of variance (ANOVA) was performed using the total number of errors produced as the dependent variable. The groups did not differ on the total number of errors produced, $F(1, 56) = 2.3$, $p = .13$ (High RFFT: $M = 1.7$, $SD = 2.0$; High RFFT: $M = 2.6$, $SD = 2.6$).

Discussion

The first hypothesis concerned changes in Delta at the right frontal site from baseline to post-rotation was only partially supported. As expected, delta activation increased following rotation. In addition, the results indicate that delta activation change was greatest at the frontal site (F8) as compared with the posterior site. This outcome was predicted by Carmona et al. (2009). Delta activity was expected to increase at the frontal lobe in order to reflect cerebral activation changes from baseline. Specifically, localized increases in delta wave activity during

awake periods in individuals have often been an index of brain dysfunction or abnormality (Fernandez et al., 1995; Foster et al., 2005).

There was relatively little change in delta activity in the posterior sites. It was also expected that those in the Low RFFT group would exhibit a greater increase in delta activation at F8 as compared with the High RFFT group since non-verbal fluency scores are generally associated with right frontal activation. In a previous study, Foster et al. (2005) had administered the RFFT to 15 high and 15 low RFFT fluency men, finding that high delta activity at F8 was associated with poor RFFT performance. The current study did not replicate these findings as the Mixed Factor ANOVA did not yield a main effect of Group or interaction effect of Group x Condition x Site. The reasons for the lack of interaction findings in this bandwidth is discussed below.

The second hypothesis concerned beta activation at the posterior sites. It was expected that those in the Low RFFT group would exhibit greater beta activation in posterior sites, consistent with the finding by Carmona et al. (in preparation) as well as the abundant line of hostility research showing this pattern under stress (Cox & Harrison, 2008; Herridge, Harrison, Mollet, & Shenal, 2004; Mollet & Harrison, 2006, 2007; Rhodes, Harrison, & Demaree, 2002; Williamson & Harrison, 2003). In line with our theory of frontal regulatory capacity over posterior sites it was predicted that vestibular stress would disproportionately impact posterior regions under the regulatory control of the right frontal lobe. Heightened stress would yield activation to the point of diminished capacity secondary to destabilizing vestibular stress.

The results were again partially supportive of these assertions. Overall, posterior site beta activity (site T6) was greater for both groups as compared with frontal beta activity. However, greater beta activity was found in the High RFFT group at T6 as opposed to the Low RFFT

group (see Figure 3). In addition, a Site x Condition interaction suggests that beta activity, while higher at the posterior sites, decreased slightly following rotation at T6. While it was expected that vestibular stimulation would yield increased beta activity, consistent with the increase in beta activity found in emotional processing studies (e.g. Foster & Harrison, 2002; Schellberg, et al., 1993; Ray & Coles, 1985) it is possible that vestibular-based exertion was yielding a cerebral calming effect. Previous literature has discussed the beneficial sedative effects of vestibular stimulation (MacLean & Baumeister, 1982; Sandler & Voogt, 2001).

It is possible that the vestibular stimulation did not reach the threshold necessary to produce a distressing effect. The State-Trait Anxiety Inventory S-scale provides support for this potential explanation as the subjective ratings of anxiety did not significantly differ between the baseline and post-stress conditions and there were no differences in the subjective ratings between groups as well. The vestibular literature has frequently pointed to a relationship between anxiety and vestibular dysfunction. However, whether the nature of the relationship is associative, causal, interrelated, or simply false positive findings are still a matter of debate (Jacob, Furman, & Perel, 1996). Evidence supports either view as vestibular disorders such as vestibular neuritis, can lead to increased anxiety complaints (Eagger, Luxon, Davies, Coello, & Ron, 1992; Pollak, Klein, Rafael, Vera, & Rabey, 2003; Yardley, Masson, Verschuur, Haacke, & Luxon, 1992). Likewise it has been demonstrated that anxiety can create vestibular dysfunctional symptoms when the aural apparati are intact (Staab, 2006; Sklare Stein, Pikus, & Uhde, 1990). In the current experiment, it was expected that whole-body rotation would induce increased sensory stimulation and demands on the limbic, which in turn would result in diminished inhibitory capacity of the frontal lobes during vestibular challenges. The lack of endorsement of significant changes from baseline to post-rotation, combined with the lack of

beta activation in the posterior regions suggest that the whole-body rotation did not provide the optimal stimulation necessary to test this this hypothesis.Moreover, the Nausea Profile also corroborates these conclusions as it suggested that participants experienced little gastrointestinal discomfort following rotation. Specifically, examination of Tables 2 and 3 indicates that both groups experienced very little nausea sensation.

One of the possibilities for the non-significant findings in the vestibular paradigm concerns the issue of gender and cerebral laterality. For example, research in the emotional domain suggests that sex differences in emotional processing and laterality exist between men and women (Crews & Harrison, 1994; Emerson & Harrison, 1990; Harrison, Gorelczenko, & Cook, 1990). The preponderance of the research argues that women appear to be less lateralized than men during emotional provocation, tending to utilize resources from the left hemisphere in conjuction with right hemisphere activation (Everhart, Shucard, Quatrin, & Shucard, 2001; Mollet & Harrison, 2007). The current experiment did not examine sex-related differences in response to a vestibular stressor. While the vestibular modality tends to rely on the right hemisphere for vestibular specialization (see Carmona et al., 2009), activation is also noted to be in the left hemisphere as well, as maintaining balance requires coordination of both hemispheres for spatial awareness. In the previous experiment, the skin conductance of high hostile men were compared with low hostile men after whole-body rotation, and high hostile men demonstrated increased arousal to the vestibular stressor compared with low hostile men. However, the current experiment examined the fluency construct in women. There were no differences in hostility among the women (see Table 3). Therefore, future investigations may benefit from examination of the vestibular paradigm in a sample of men.

Previous studies have found EEG differences in the theta bandwidth following exposure to a vestibular stimulus. For example, Chelen (1993) demonstrated that passive whole body rotation yielded pronounced theta activation across the frontal lobes, whereas Park et al. (2008) found decreases in theta activity at Fz following an indoor motion driving simulator task. More specifically, Buzsáki (2005) related theta activity in maze-trained rats to be attributable to parahippocampal modulation of accounting for spatial map networking and travel distances. Therefore, given the precedence of theta activity in the literature, exploratory analyses were conducted to examine the theta bandwidth.

Overall, theta activity decreased at T6 over the conditions and was fairly stable at frontal sites over both conditions. However, the Group x Site interaction found that overall, the High RFFT group manifested with greater theta activity at T6 (see Figure 5). The difference may be due to the High RFFT group's increased abilities to generate complex spatial designs for the RFFT. Hence individual's in this group may have demonstrated an enhancement in performance of design generation linked to temporal activation, when the frontal lobe activation is stable. Interestingly, the High RFFT group also produced a greater number of perseverative designs on both the first and second administration of the RFFT, suggesting failure to recall a previously executed design. However, the increase in design errors was associated with the overall increased design productivity.

In addition to recorded EEG, skin conductance was selected as a dependent measure of sympathetic arousal based on physiological and theoretical considerations. First, selection was based on the proposition that electrodermal activity emanates from eccrine glands along the palmar and plantar surfaces which are innervated primarily by sympathetic cholinergic pathways (Dawson, Schell, & Filion, 2000). Therefore, the measure is theoretically considered to be more

reflective of purely sympathetic contributions due to the pathways arising strictly and directly from the thoracolumbar segment of the spinal chain rather than the cervical-lumbar segment, which is comprised by primarily parasympathetic pathways (Dawson, Schell, & Filion, 2000; Hugdahl, 1995). Moreover, selection was based on the results of our previous findings (Carmona et al., 2008), which reported differences in high and low hostile men after whole-body passive rotation.

Regarding the current experiment, it was originally hypothesized that no significant skin conductance level differences would be found at baseline. Skin conductance scores were analyzed by in raw data format and through a log transformation. Statistically, the results were similar. For both, the groups were essentially equivalent at baseline. The hypothesis that skin conductance overall would increase following rotation was also confirmed. The results mirror those of Carmona et al. (2008), which found that skin conductance increased following whole-body passive rotation.

Originally it was hypothesized that an increase in skin conductance would be greater in the Low RFFT group following whole body passive rotation. It was expected that this interaction would mirror the interaction effect of Carmona et al. (2008), whereby high hostile men were more prone to heightened skin conductance after brief, whole-body rotation about the neuroaxis beyond that developed by low hostile men. The results were interpreted to provide indirect support for frontocerebral modulation of sympathetic arousal after vestibular stimulation. Similarly, in the current experiment it was hypothesized that the Low RFFT group would show increased sympathetic arousal (via skin conductance), due to purportedly reduced frontal capacity. However, the hypothesis was not confirmed as no group differences were found. The

results are not unexpected given that no differences between the groups were found for frontal delta activity in the EEG recordings.

However, in the log transformed skin conductance, a non-significant trend of significance was found between the High and Low RFFT groups across the conditions. As displayed in Figure 10, the groups were nearly equivalent at baseline, however the High RFFT group manifested greater skin conductance activation after rotation as compared with the Low RFFT group. Nonetheless, the difference was not significant and no other significant results were found for skin conductance.

In this experiment, we have focused on sympathetic nervous system indices of arousal, but it is worth noting that the diffuseness of the vestibular system includes the interplay of both the sympathetic and parasympathetic autonomic systems (Kaufmann & Battacharya, 2002). For example, vestibular disorder phenomena frequently includes features such as flushing, sweating, and somatic thermal fluctuation which indicate sympathetic contributions to the disordered state, with nausea, orthostatic dysregulation, and fainting, suggesting parasympathetic contributions. Hence, since parasympathetic activity was not assessed, it is possible that the non-significant findings for group differences were due to involvement of parasympathetic activation. Future studies may consider examining the low frequency /high frequency vagal ratio of participants after a vestibular stressor.

The RFFT was administered a second time after the initial screening process. Naturally, as the groups were selected for composition based on their RFFT scores, it was expected that those differences in RFFT performance would persist on the second administration. Consequently, differences in the proportion of improvement from the initial screening were calculated for the Low and High RFFT groups and the proportion score was analyzed. The

results indicate that while the High RFFT group continued to show a dominance of design fluency, the mean increase from the screening to the administration following rotation (14%) was proportionately smaller than the Low RFFT group (22%). The results suggest a ceiling effect in terms of performance on the RFFT for the High RFFT group. However, the results can also be interpreted to suggest that the initial performance of the Low RFFT group may have underestimated their innate fluency capabilities. This interpretation would likely have implications for the construction of the fluency groups altogether, which may explain why group differences amongst the physiological dependent variables were absent across conditions.

Altogether, the functional cerebral systems model found support for overall reduced frontal activation following rotation, based on an increase in delta activity and an increase in skin conductance following rotation. However, limited support was found for a laterality component, based on the beta bandwidth findings.

There were a number of methodological considerations in the current experiment. First, the intent of the experiment was to provide a theoretical basis for continuation of investigation into the role of cerebral involvement in vestibular processes in an elderly population. However, the findings of the current experiment may not be generalizable given that the current sample involved a sample of healthy, young, college-age women. Second, the self-report of the anxiety inventory indicate that the participants did not experience a significant increase in distress. One future consideration may be to incorporate a manipulation check to examine the extent of the inducement of dizziness and subjective displeasure after whole-body rotation for possible adjustment of number of rotations or rotation rate. Third, it may also be possible that participants had varying degrees of familiarity with the RFFT. A future manipulation check should inquire as to whether participants have completed the RFFT or a similar non-verbal design fluency task.

For procedural modifications, future studies may wish to incorporate a standard set of head movements to be completed during the whole body passive rotation as seen in vestibular experiments examining nausea (Chelen et al., 1993; Himi et al., 2004). Head movements during whole-body passive rotation would maximize the sensory conflict that would occur between the visual, proprioceptive, and vestibular inputs. It is likely the case that this mismatch would result in unpleasant sensations of disorientation and/or nausea, resulting in a challenge to frontal lobe resources, which may result in a differential cerebral demand on those who are proficient and are not proficient at a design fluency task. Moreover, further analyses may also consider examination of other sites besides F8 and T6. While the results do not support the use of a design fluency task for predicting falling in the elderly, due to vestibular distress, the results indicate that vestibular disorientation yields systematic changes in the frontal and posterior regions which could have a deleterious impact on the elderly's capacity to maintain balance in the face of insurmountable cerebral challenges.

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Figure 1. Total initial design fluency across Trials (1-5).

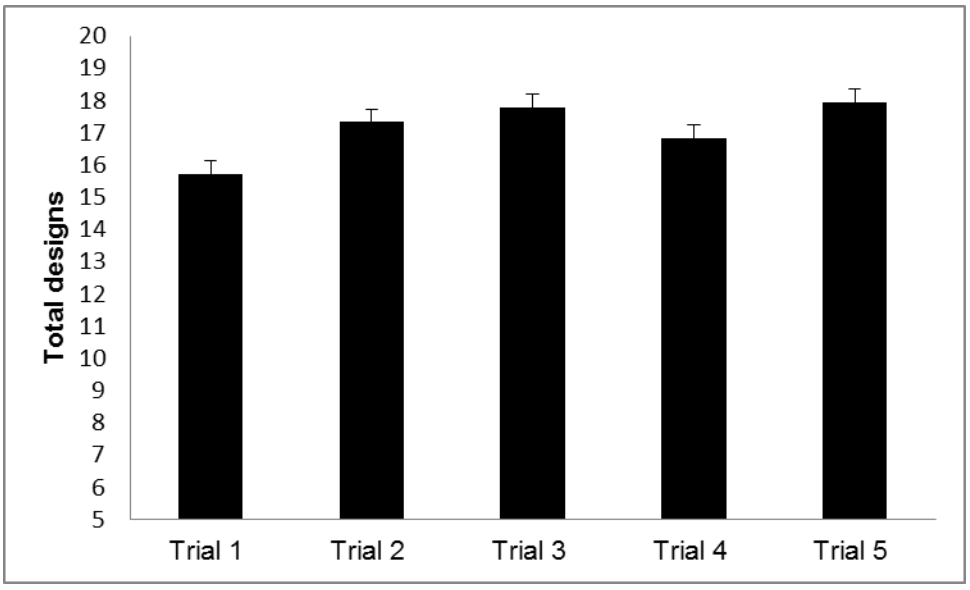


Figure 2. Mean delta magnitude (μV) as a function of Condition and Site.

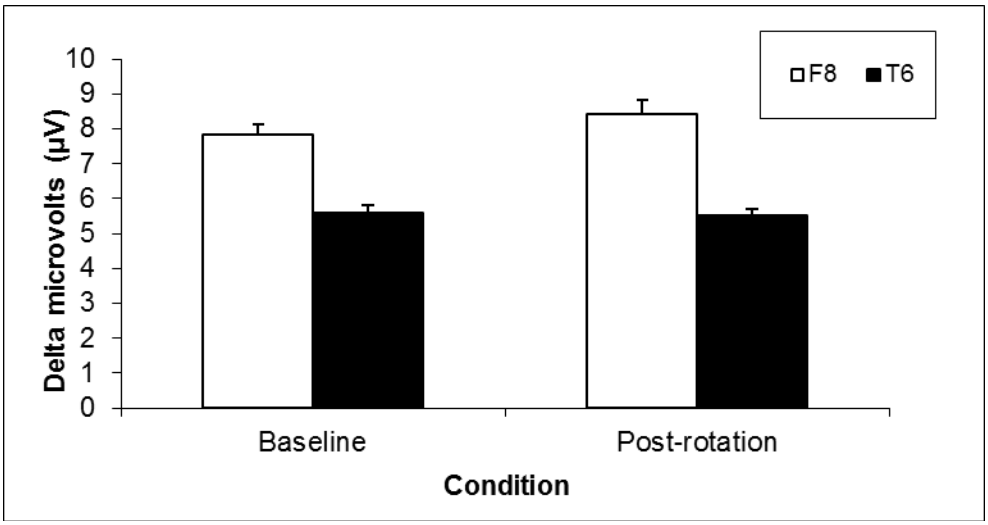


Figure 3. Mean beta magnitude (μV) as a function of Group and Site.

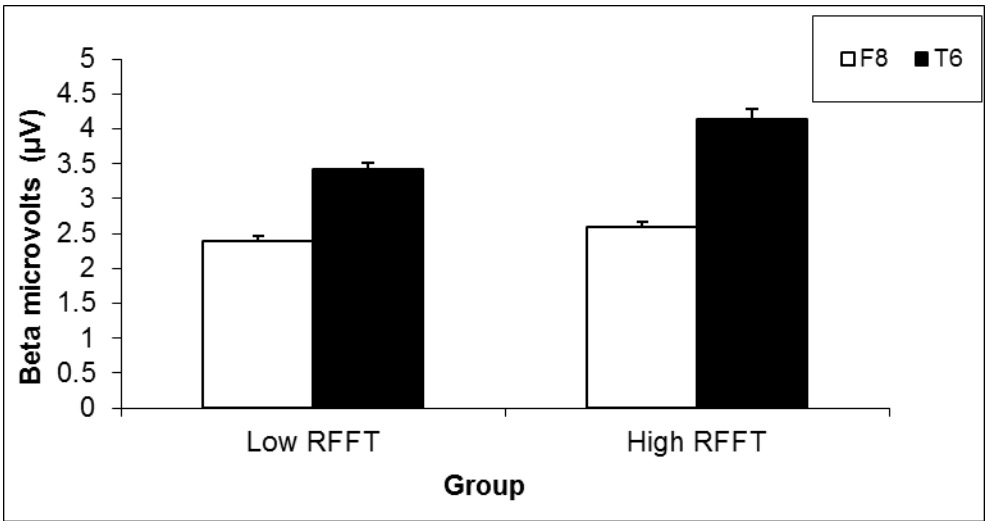


Figure 4. Mean beta magnitude (μV) as a function of Condition and Site.

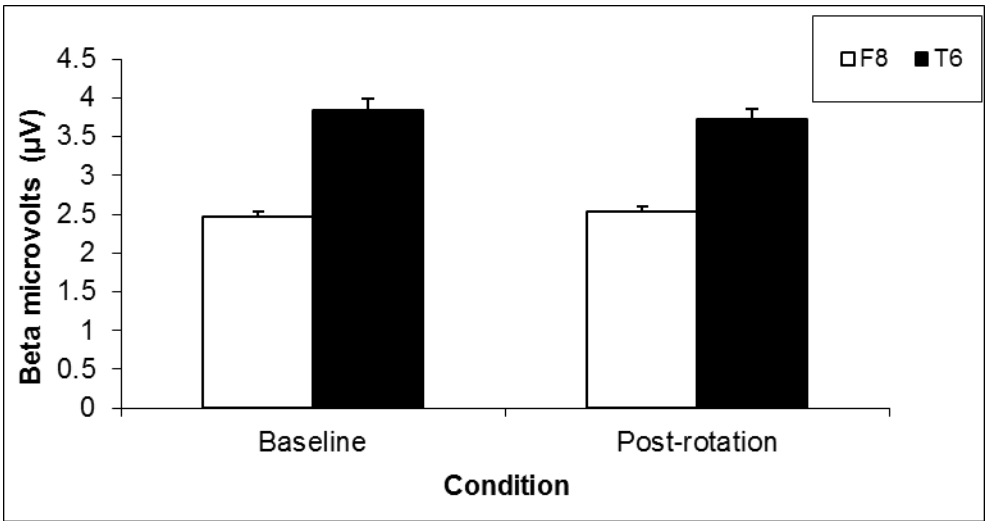


Figure 5. Mean theta magnitude (μV) as a function of Group and Site.

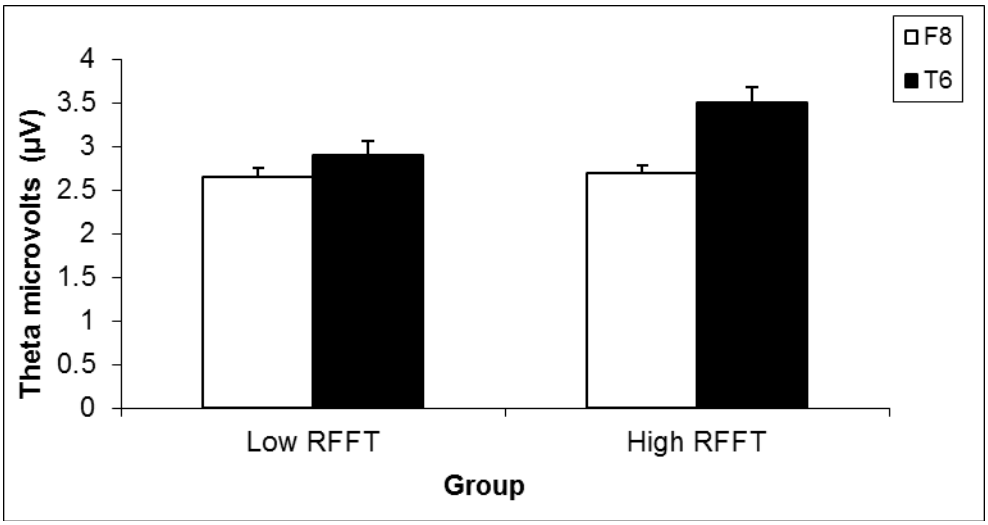


Figure 6. Mean theta magnitude (μV) as a function of Site and Condition.

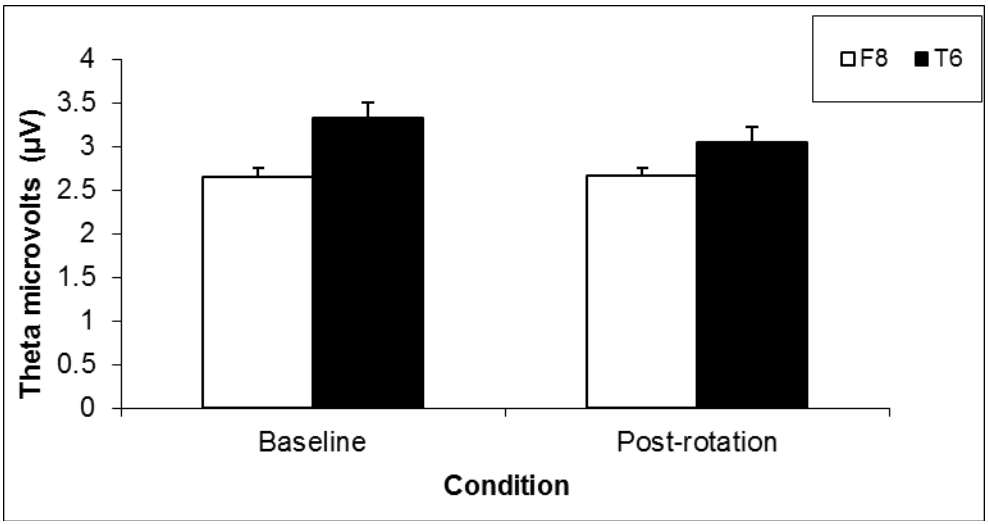


Figure 7. Trend of significance for Log of skin conductance as a function of Group and Site.

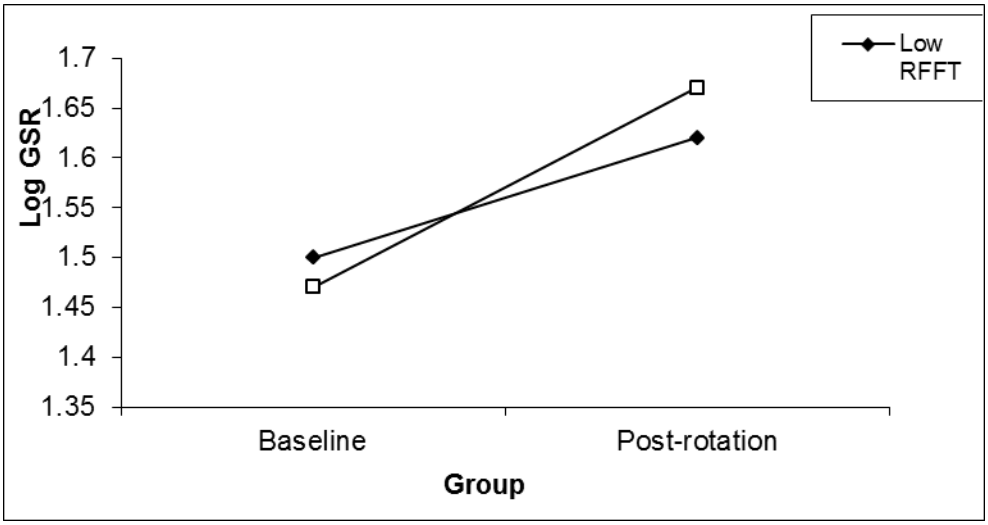


Figure 8. Total design fluency across Trials (1-5) on the second administration.

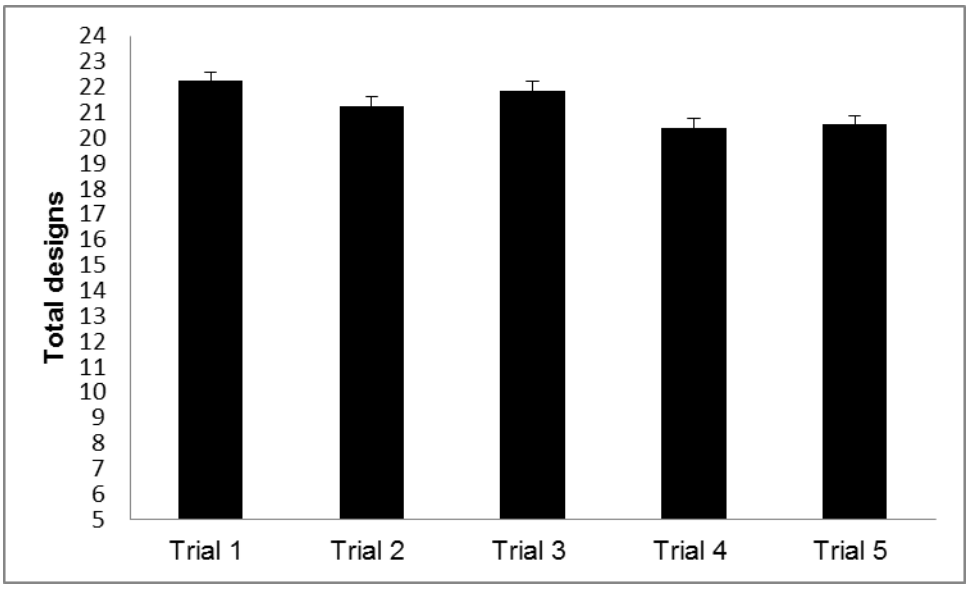


Figure 9. Number of designs as a function of Group and Trial.

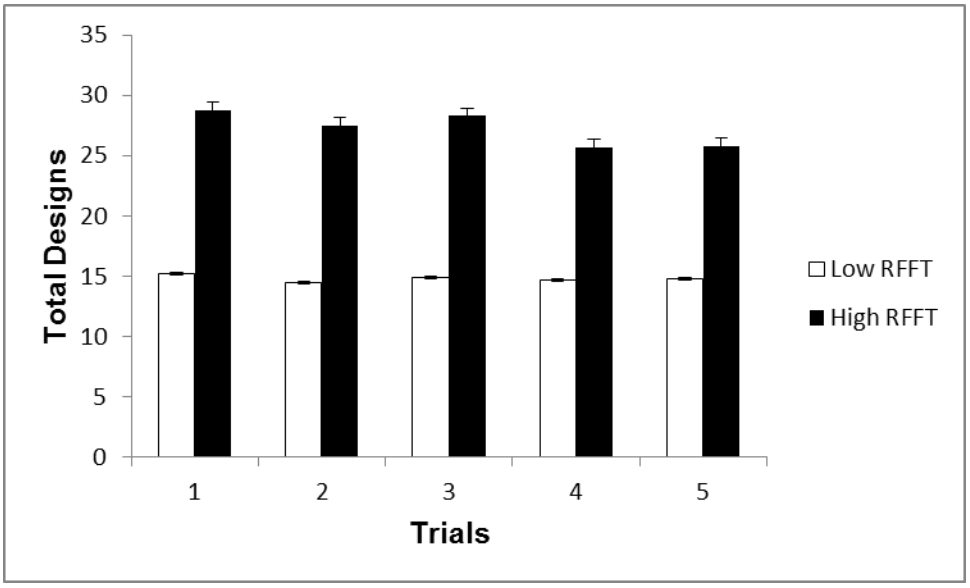


Figure 10. Total letter fluency performance across Trials (1-5) of the COWAT.

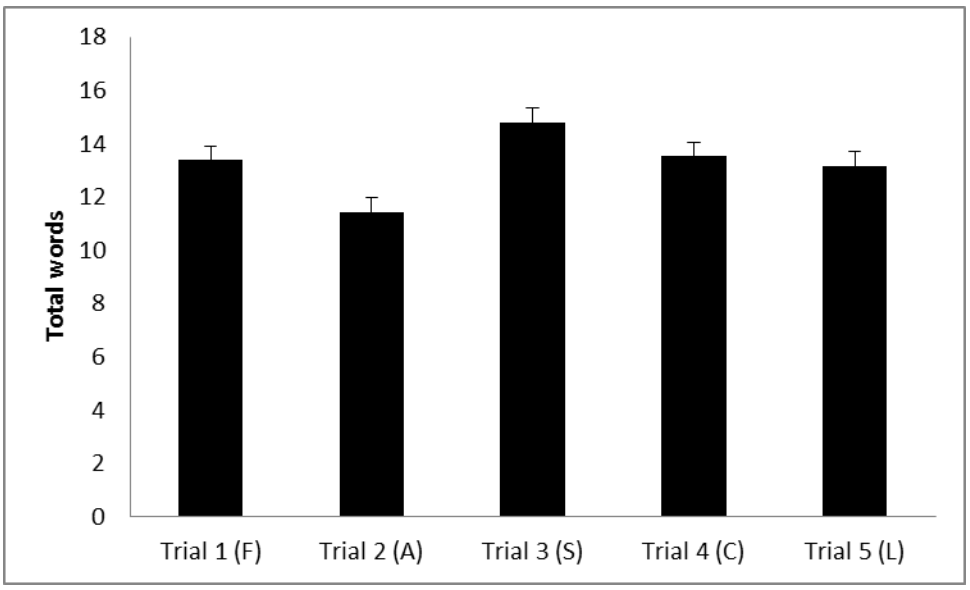


Table 1: Summary of means and standard deviations for self-report measures of Phase 1.

	CMHS[1]	**STAI-T**[2]	**BDI-II**[3]	**LQ**[4]
	16.4 (7.1) N = 289	38.7 (10.5) N = 292	9.1 (8.7) N = 289	8.5 (5.0) N = 290

			COWAT[5] N = 293			
Trial 1	*Trial 2*	*Trial 3*	*Trial 4*	*Trial 5*	*Total*	*Errors*
13 (3.2)	11.5 (3.1)	15.2 (7.0)	13.4 (3.0)	12.9 (3.2)	65.6 (12.6)	1.9 (2.2)
			RFFT[6] N = 293			
Trial 1	*Trial 2*	*Trial 3*	*Trial 4*	*Trial 5*	*Total*	*Errors*
15.4 (5.3)	16.9 (5.1)	17.5 (5.3)	17 (5.0)	18.3 (5.3)	84.8 (24.1)	3.1 (3.4)

1. Cook-Medley Hostility Scale.
2. Spielberger State-Trait Anxiety Inventory (Trait).
3. Beck Depression Inventory-II.
4. Laterality Questionnaire.
5. Ruff Figural Fluency Test
6. Controlled Oral Word Association Test

Table 2: Summary of means and standard deviations for self-report measures of the RFFT groups.

Group	**CMHS**[1]	**STAI-T**[2]	**STAI-S1**[3]	**STAI-S2**[4]	**BDI-II**[5]	**LQ**[6]	**NP**[7]
Low RFFT	15.9 (5.5) N = 29	40.2 (9.5) N = 29	33.6 (8.7) N = 27	34.1 (8.8) N = 27	9.7 (6.1) N = 28	10.1 (2.5) N = 29	18.6 (15.6) N = 28
High RFFT	16.3 (7.6) N = 29	38.3 (10.1) N = 29	29.4 (8.7) N = 28	31.4 (8.5) N = 28	7.6 (6.8) N = 29	10.1 (3.3) N = 29	20.4 (16.3) N = 27

1. Cook-Medley Hostility Scale.
2. Spielberger State-Trait Anxiety Inventory (Trait).
3. Spielberger State-Trait Anxiety Inventory (State) administered at baseline.
4. Spielberger State-Trait Anxiety Inventory (State) administered post-rotation.
5. Beck Depression Inventory-II.
6. Laterality Questionnaire.
7. Nausea Profile.

Table 3: Summary of Analysis of Variance (ANOVA) sources for self-report measures.

Source	**df**	**SS**	**F value**	**Pr > F**
Laterality Questionnaire				
Group	1	.07	.008	.92
Cook-Medley Hostility Questionnaire				
Group	1	2.9	.07	.80
Nausea Profile				
Group	1	41.3	.16	.69
State-Trait Anxiety Inventory (Trait)				
Group	1	50.3	.52	.47
Beck Depression Inventory-II				
Group	1	62.4	1.5	.23

Note: * P<.05; **P<.01

Table 4: Summary of State-Trait Anxiety Inventory (State) Analysis of Variance (ANOVA) sources.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	321.9	2.6	.11
Condition	1	.41.2	1.54	.23
Group x Condition	1	13.7	.51	.48

Note: * P<.05; **P<.01

Table 5: Summary of initial RFFT Analysis of Variance (ANOVA) sources for the High and Low RFFT groups.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	10548	543.3	**<.01****
Trial	4	189.3	8.6	**<.01****
Group x Trial	4	13.3	.60	.66

Note: * P<.05; **P<.01

Table 6: Summary of delta (μV) Analysis of Variance (ANOVA) sources.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	2.2	.26	.61
Condition	1	3.9	4.3	**.04***
Group x Condition	1	.04	.04	.84
Site	1	385	52.1	**<.01****
Group x Site	1	17.8	2.4	.13
Condition x Site	1	7.1	9.2	.004
Group x Condition x Site	1	.17	.22	.64

Note: * P<.05; **P<.01

Table 7: Summary of beta (μV) Analysis of Variance (ANOVA) sources.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	12.1	8.1	.006
Condition	1	.058	.48	.49
Group x Condition	1	.13	1.05	.31
Site	1	94.8	118.4	**<.01****
Group x Site	1	3.9	4.8	**.03***
Condition x Site	1	.47	10.3	**.002***
Group x Condition x Site	1	.07	1.5	.22

Note: * P<.05; **P<.01

Table 8: Summary of theta (μV) Analysis of Variance (ANOVA) sources.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	5.27	1.48	.23
Condition	1	1.01	9.4	**.003****
Group x Condition	1	.07	.65	.42
Site	1	16.41	19.91	**<.01****
Group x Site	1	4.6	5.6	**.02***
Condition x Site	1	1.01	24.1	**<.01****
Group x Condition x Site	1	.01	.24	.63

Note: * P<.05; **P<.01

Table 9: Summary of skin conductance Analysis of Variance (ANOVA) sources.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	7.86	.33	.57
Condition	1	25.5	46.6	**<.01****
Group x Condition	1	.42	.76	.39

Note: * P<.05; **P<.01

Table 10: Summary of the logarithm of skin conductance Analysis of Variance (ANOVA) sources.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	.0008	.001	.97
Condition	1	.67	43.7	**<.01****
Group x Condition	1	.04	2.9	.09

Note: * P<.05; **P<.01

Table 11: Summary of the second administration of the RFFT Analysis of Variance (ANOVA) sources for the High and Low RFFT groups.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	10771	208.2	**<.01****
Trial	4	139.5	8.8	**<.01****
Group x Trial	4	93.0	5.9	**<.01****

Note: * P<.05; **P<.01

Table 12: Summary of COWAT Analysis of Variance (ANOVA) sources for the High and Low RFFT groups.

Source	**df**	**SS**	**F value**	**Pr > F**
Group	1	266.5	6.9	**.01***
Trial	4	342.4	18.9	**<.01****
Group x Trial	4	13.4	.74	.57

Note: * P<.05; **P<.01

Appendices

Appendix A

Medical History Questionnaire

1	Do you have any history of congenital or developmental problems?	Yes	No
2	Do you have any history of learning disabilities or special education?	Yes	No
3	Do you have any history of hypoglycemia (low blood glucose)?	Yes	No
4	Do you have any history of hyperglycemia (diabetes)?	Yes	No
5	Are you experiencing blood glucose problems at present?	Yes	No
6	Do you have any history of hypertension? (high blood pressure)	Yes	No
7	Do you have any history of hypotension? (low blood pressure)	Yes	No
8	Do you have any history of hyperthyroidism?	Yes	No
9	Do you have any history of hypothyroidism?	Yes	No
10	Have you ever suffered a head injury resulting in a hospital stay longer than 24 hours?	Yes	No
11	Have you ever been knocked out or rendered unconscious (more than 5 minutes)?	Yes	No
12	Have you ever suffered "black-out" or fainting spells?	Yes	No
13	Do you have a history of other neurological disorders (e.g. stroke or brain tumor)?	Yes	No
14	Have you ever received psychiatric/psychological care or counseling?	Yes	No
15	Have you ever been hospitalized in a psychiatric facility/hospital?	Yes	No
16	Have you ever been diagnosed with a psychiatric/psychological disorder?	Yes	No
17	Have you ever been administered any (neuro)psychological tests or measures?	Yes	No
18	Do you have a history of substance abuse or alcohol abuse?	Yes	No
19	Do you have any history of heart disease?	Yes	No
20	Do you have any history of pancreatic disease?	Yes	No
21	Are you currently taking any prescription blood-thinning medications?	Yes	No
22	Do you have a history of high blood pressure?	Yes	No
23	Do you have any uncorrected visual or hearing impairments?	Yes	No
24	Are you able to read, write, and speak English effectively?	Yes	No
25	Do you consume three or more alcoholic more than two nights a week?	Yes	No
26	Have you ever experienced a medical or psychiatric condition that could potentially affect cognitive functioning, such as stroke, electroconvulsive treatment, epilepsy, brain surgery, encephalitis, meningitis, multiple sclerosis, Parkinson's Disease, Huntington's Chorea, Alzheimer's dementia, Schizophrenia, or Bipolar Disorder?	Yes	No
27	Have you ever used smoked or used tobacco products?	Yes	No
28	Do you use any unprescribed or "illegal/street" drugs?	Yes	No
29	Are you taking any of the following medications: antidepressant, antianxiety,	Yes	No

	antipsychotic?		
30	Are you taking any allergy or cold medication?	Yes	No
31	Do you frequently experience migraine headaches?	Yes	No
32	Do you have a history of chronic earache that lasted more than a month?	Yes	No
33	Do you often experience pressure in the inner ear?	Yes	No
34	Do you frequently hear a persistent ringing, buzzing, or hissing sound?	Yes	No
35	Have you ever been diagnosed with any of the following vestibular disorders: Orthostatic dysregulation, Meniere's Disease, Cogan's syndrome, Labyrinthine Infarct, Neurolabyrinthitis?	Yes	No
36	Do you have a history of panic attacks or agoraphobia?	Yes	No
37	Do you frequently experience sensations of nausea?	Yes	No
38	Do you frequently experience dizziness?	Yes	No
39	Do you currently participate in gymnastics, ballet, aircraft control?	Yes	No

If you answered "yes" to any of the above please explain fully:

_____

_____

_____

_____

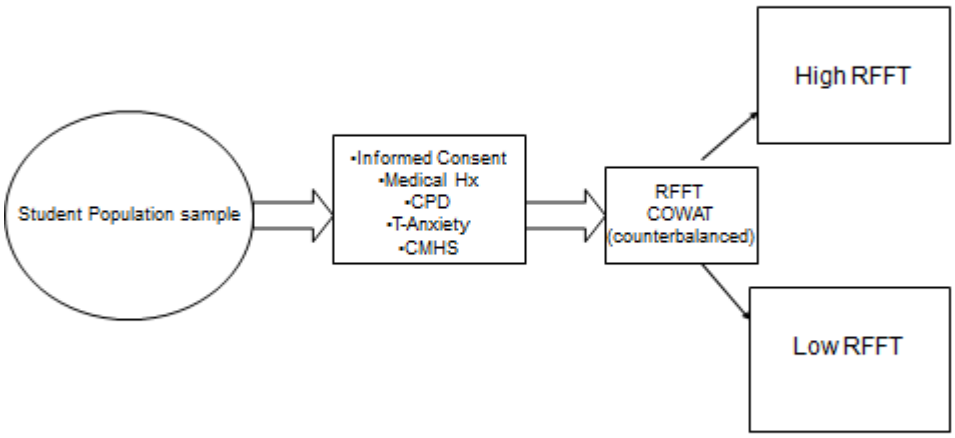
Appendix B

Controlled Oral Word Association Test Sheet

Subject #: _____
Date: _____

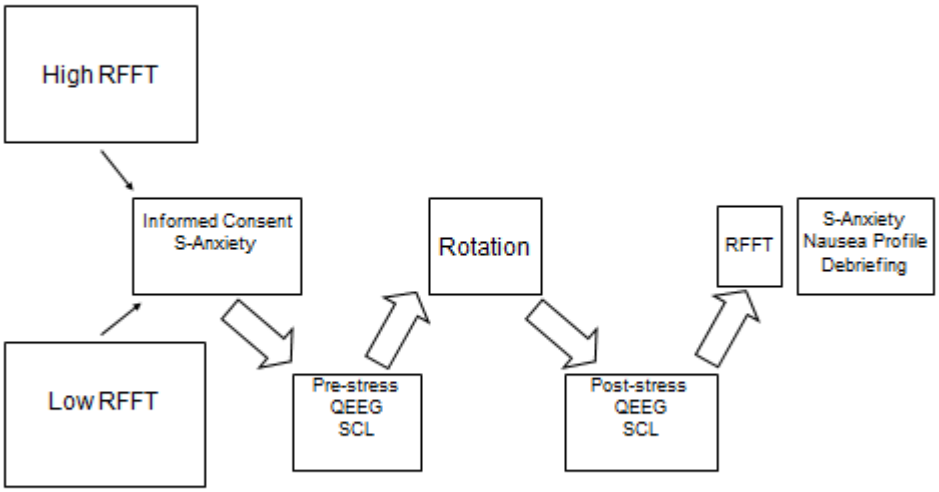
Appendix C

Diagram of Phase I



Appendix D

Diagram of Phase II



Appendix E

Vestibular Experiment Script

Please fill out this informed consent form. If you have any questions please let me know.

[Administer informed consent form (IC)]

In addition to your signature, please print your name legibly so we can be sure to assign credit correctly.

After they finish the IC:

Do you have any questions about this form? Now please turn off your cell phone.

After they complete the IC take up the form and place in the participant's folder.
*The IC will eventually be separated but not before we ensure extra credit.

Do not give any other forms before you receive the IC and ensure there are no questions.

[Administer the Med Hx, Laterality, STAI (T), BDI-II, and CMHS]

Please complete these self-report questionnaires. The instructions are on the forms but please let me know if you have questions.

After they complete the forms take them up and place them in the folders.

Now please listen while the CD player plays the instructions.

[Insert either disk 1 or disk 2. Press play]

CD Player

For Part I (Disk 1)
[1]
Thank you for participating in the study. In this portion of the study you will complete two cognitive tests. If you can hear me, please raise your left hand. [pause]. *If you would like the volume raised please tell the Proctor at this point.* [pause].

[Now give either the RFFT trial or the COWAT]

[2]
COWAT Instructions:

In this next exercise I am going to say a letter of the alphabet to you, and I want you to write as many words as you can think of that begin with that letter. But none of the words can be proper names of people or places. For instance, if I gave you the letter B, you could write "brook ,bottle, black" and so forth, but you could not say "Betty" since that is a person's name, nor could you say "Boston" since that is the proper name of a place. Also, do not give me the same word with different endings, such as "big, bigger, biggest." Finally do not write any numbers. For example if I gave you the letter "T" do not write "31, 32, 33." Do you have any questions?
[Pause]

[3]
Now look at the page in front of you. Once you are given the letter you will write as many unique words as you can think of in the first column. If you finish the first column, go on to the second column and continue until you are told to stop.

[4]
The first letter we will use is F. Go ahead and write as many words as you can think of that begin with the letter "F." BEGIN.

[1 min. Proctor, make sure no one is cheating.]

STOP. Finish the last word you were working on and put your pen down. Now turn the page.
[Pause CD until everyone has the next page.]

[5]
The next letter we will use is the letter "A." Go ahead and write as many words as you can think of that begin with the letter "A." BEGIN.

STOP. Finish the last word you were working on and put your pen down. Now turn the page.
[Pause CD]

[6]
The next letter we will use is the letter "S." Go ahead and write as many words as you can think of that begin with the letter "S." BEGIN.

STOP. Finish the last word you were working on and put your pen down. Now turn the page.
[Pause CD]

[7]
The next letter we will use is the letter "C." Go ahead and write as many words as you can think of that begin with the letter "C." BEGIN.

STOP. Finish the last word you were working on and put your pen down. Now turn the page.
[Pause CD]

[8]

The next letter we will use is the letter "L." Go ahead and write as many words as you can think of that begin with the letter "L." BEGIN.

STOP. Finish the last word you were working on and put your pen down. [Pause CD]

[Proctor, take up the COWAT forms INDIVIDUALLY, and put them in the subject's folder. Be sure not to mix them up! Administer the first trial of the RFFT. Hand them a marker]

[9]

In front of you are three squares, each containing five dots. Note that the arrangement of the five dots is always the same. You will be asked to connect two or more dots to create a unique design by always using straight lines. The purpose of this test is for you to make as many unique designs as quickly as possible. Remember that each design must be different in some way from the others. Now practice with the 3 squares in front of you. [Pause CD]

[Proctor, look at the practice trial and ensure the participant is performing the task correctly. Provide feedback. Reiterate the rule "*Remember to connect two or more dots to make unique designs as quickly as possible*."]

[10]

Now turn the page. On this page, please draw as many different patterns as quickly as possible. Start in the upper left square and work from left to right. Just connect two or more dots with a straight line. Work as quickly as possible and make every pattern different. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [wait 3 sec.]
[Pause CD]

[After the trial, collect trial 1 and place in the correct folder. Be sure not to mix them up!
Next, give Trial 2]

[11]

Trial 2:
The instructions are the same for this trial. Please draw as many different patterns as quickly as possible. Just connect two or more dots with a straight line. This page is for practice. Begin.
[Pause CD]

[12]
Now turn the page. Start in the upper left hand corner. Just connect two or more dots to make unique designs. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [wait 3 sec.]
[Pause CD]

[13]

Trial 3:
For this trial, please use the marker in front of you. The instructions are the same for this trial. This page is for practice. Begin. [Pause CD]

[14]
Now turn the page. Start in the upper left hand corner. Ready--BEGIN.

STOP. Finish the last design you were working on and put your marker down. [wait 3 sec.]
[Pause CD]

[15]

Trial 4:
Now you can put the marker aside and use the pen instead. For this trial, the instructions are the same for this trial. This page is for practice. Begin.
[Pause CD]

[16]
Now turn the page. Start in the upper left hand corner. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [wait 3 sec.]
[Pause CD]

[17]

Trial 5:
The instructions are the same for this trial. This page is for practice. Begin. [Pause CD]

[18]

Now turn the page. Start in the upper left hand corner. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [wait 3 sec.]

[Proctor, take up the RFFT forms, and put them in the subject's folder. Be sure not to mix them up! Take up markers]

Now please sit quietly and wait a few minutes while I see the primary supervisor.

Vestibular Experiment Script

Please fill out this informed consent form. If you have any questions please let me know.

[Administer informed consent form (IC)]

In addition to your signature, please print your name legibly so we can be sure to assign credit correctly.

After they finish the IC:

Do you have any questions about this form? Now please turn off your cell phone.

After they complete the IC take up the form and place in the participant's folder.
*The IC will eventually be separated but not before we ensure extra credit.

Do not give any other forms before you receive the IC and ensure there are no questions.

[Administer the Med Hx, Laterality, STAI (T), BDI-II, and CMHS]

Please complete these self-report questionnaires. The instructions are on the forms but please let me know if you have questions.

After they complete the forms take them up and place them in the folders.

Now please listen while the CD player plays the instructions.

[Insert either disk 1 or disk 2. Press play]

CD Player

For Part I (disk 2)

[1]
Thank you for participating in the study. In this portion of the study you will complete two cognitive tests. If you can hear me, please raise your left hand. [pause]. *If you would like the volume raised please tell the Proctor at this point.* [pause].

[Now give either the RFFT trial or the COWAT]

[2]

In front of you are three squares, each containing five dots. Note that the arrangement of the five dots is always the same. You will be asked to connect two or more dots to create a unique design by always using straight lines. The purpose of this test is for you to make as many unique designs

as quickly as possible. Remember that each design must be different in some way from the others. Now practice with the 3 squares in front of you. [Pause CD]

[Proctor, look at the practice trial and ensure the participant is performing the task correctly. Provide feedback. Reiterate the rule "*Remember to connect two or more dots to make unique designs as quickly as possible*."]

[3]

Now turn the page. On this page, please draw as many different patterns as quickly as possible. Start in the upper left square and work from left to right. Just connect two or more dots with a straight line. Work as quickly as possible and make every pattern different. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [Pause CD]

[After the trial, collect trial 1 and place in the correct folder. Be sure not to mix them up! Next, give Trial 2]

[4]

Trial 2:
The instructions are the same for this trial. Please draw as many different patterns as quickly as possible. Just connect two or more dots with a straight line. This page is for practice. Begin. [Pause CD]

[5]
Now turn the page. Start in the upper left hand corner. Just connect two or more dots to make unique designs. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [Pause CD]

[6]

Trial 3:
For this trial, please use the marker in front of you. The instructions are the same for this trial. This page is for practice. Begin. [Pause CD]

[7]
Now turn the page. Start in the upper left hand corner. Just connect two or more dots to make unique designs. Ready--BEGIN.

STOP. Finish the last design you were working on and put your marker down.[Pause CD]

[8]

Trial 4:

Now you can put the marker aside and use the pen instead. For this trial, the instructions are the same. This page is for practice. Begin. [Pause CD]

[9]
Now turn the page. Start in the upper left hand corner. Just connect two or more dots to make unique designs. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [Pause CD]

[10]

Trial 5:
The instructions are the same for this trial. This page is for practice. Begin. [Pause CD]

[11]

Now turn the page. Start in the upper left hand corner. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [Pause CD]

[Proctor, take up the RFFT forms, and put them in the subject's folder. Be sure not to mix them up! Take up markers]

[12]
COWAT Instructions:

In this next exercise I am going to say a letter of the alphabet to you, and I want you to write as many words as you can think of that begin with that letter. But none of the words can be proper names of people or places. For instance, if I gave you the letter B, you could write "brook ,bottle, black" and so forth, but you could not say "Betty" since that is a person's name, nor could you say "Boston" since that is the proper name of a place. Also, do not give me the same word with different endings, such as "big, bigger, biggest." Finally do not write any numbers. For example if I gave you the letter "T" do not write "31, 32, 33." Do you have any questions? [Pause]

[13]
Now look at the page in front of you. Once you are given the letter you will write as many unique words as you can think of in the first column. If you finish the first column, go on to the second column and continue until you are told to stop.

[14]
The first letter we will use is F. Go ahead and write as many words as you can think of that begin with the letter "F." BEGIN.

[1 min. Proctor, make sure no one is cheating.]

STOP. Finish the last word you were working on and put your pen down. Now turn the page. [Pause CD until everyone has the next page.]

[15]
The next letter we will use is the letter "A." Go ahead and write as many words as you can think of that begin with the letter "A." BEGIN.

STOP. Finish the last word you were working on and put your pen down. Now turn the page. [Pause CD]

[16]
The next letter we will use is the letter "S." Go ahead and write as many words as you can think of that begin with the letter "S." BEGIN.

STOP. Finish the last word you were working on and put your pen down. [wait 3 sec.] *Now turn the page.* [Pause CD]

[17]
The next letter we will use is the letter "C." Go ahead and write as many words as you can think of that begin with the letter "C." BEGIN.

STOP. Finish the last word you were working on and put your pen down. [wait 3 sec.] *Now turn the page.* [Pause CD]

[18]
The next letter we will use is the letter "L." Go ahead and write as many words as you can think of that begin with the letter "L." BEGIN.

STOP. Finish the last word you were working on and put your pen down.

[Proctor, take up the COWAT forms INDIVIDUALLY, and put them in the subject's folder. Be sure not to mix them up! Administer the first trial of the RFFT. Hand them a marker]

Now please sit quietly and wait a few minutes while I see the primary supervisor.

Vestibular Experiment Script

Part II

Preparation

Please fill out this informed consent form. If you have any questions, please let me know.

[Administer informed consent form]

In addition to you signature, please print your name legibly so we can be sure to assign credit correctly

After she finishes the informed consent form:

Do you have any questions?

[After she completes the IC, take up the form and place with the participant's folder; the IC will eventually be separated but not until we ensure extra credit for this part of the experiment]

[Ask the participant if she needs to use the bathroom or get something to drink before startingWhen she returns, place "experiment" sign on door and place "out of order" sign on bathroom door]

Prep for cap fitting:

[Wipe target areas with rubbing alcohol and Nu-Prep]

Get the appropriate sized belt for the participant; have her place the belt right underneath the bust with metal circles facing forward.

Measure the head circumference and longitudinal length using the nasion and the inion. 10% of the distance between the inion and nasion on longitudinal measure- Mark placement for FP1 and FP2. Retrieve proper sized cap for participant.

Place disposable sponge discs on electrodes FP1and FP2. Also place collars on eye electrodes and skin conductance electrodes. Fill skin conductance electrodes with salt gel solution.

Apply electro-gel to each electrode (cap, eyes, and ears); make sure the participant is not in any discomfort with the application of the gel.

Hook up cap and SC electrodes to bioamp. Calibrate and check impedance levels

Experiment

After checking impedances:

Please relax and get accustomed to your surroundings. Step out of the chamber. Turn off impedance amp and turn all switches to "ref" except for 18, 21, and 22. Channel 18 should

always be closed. Channels 21 and 22 should be switched to “open”. Switch bioamp to “Calibrate”

[**Light on**]

Please close your eyes and remain as still as possible. Switch bioamp back to “Normal”. Give “thumbs up” signal. [Baseline measure for 2 min]

[**Light off**] Unhook electrodes.

Please keep your eyes open, looking straight ahead, with your feet tucked in. After the rotation is finished, please close your eyes and remain as still as possible.

Proceed with rotation. After 20 rotations, stop. Remind the participant to close their eyes. Hook up electrodes and give signal. Step out of chamber. Give signal

[**Light on**] EEG recording for 2 mins or so

[**Light off**] administer the RFFT to the participant. Start CD player.

CD Player

[1]

The purpose of this test is for you to make as many unique designs as quickly as possible. Remember that each design must be different in some way from the others. Now practice with the 3 squares in front of you. [Pause CD]

[Proctor, look at the practice trial and ensure the participant is performing the task correctly. Provide feedback. Reiterate the rule “*Remember to connect two or more dots to make unique designs as quickly as possible*.”]

[2]

Now turn the page. On this page, please draw as many different patterns as quickly as possible. Start in the upper left square and work from left to right. Just connect two or more dots with a straight line. Work as quickly as possible and make every pattern different. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [Pause CD]

[3]

Now turn the next two blank pages until you get to the practice sheet. [Pause CD]

[4]

Trial 2:

The instructions are the same for this trial. Please draw as many different patterns as quickly as possible. Just connect two or more dots with a straight line. This page is for practice. Begin. [Pause CD]

[5]
Now turn the page. Start in the upper left hand corner. Just connect two or more dots to make unique designs. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [Pause CD]

[6]

Now turn the next two blank pages until you get to the practice sheet. [Pause CD]

[7]

Trial 3:
For this trial, please use the marker in front of you. The instructions are the same for this trial. This page is for practice. Begin. [Pause CD]

[8]

Now turn the page. Start in the upper left hand corner. Just connect two or more dots to make unique designs. Ready--BEGIN.

STOP. Finish the last design you were working on and put your marker down.
[Pause CD]

[9]

Now turn the next two blank pages until you get to the practice sheet. [Pause CD]

[10]

Trial 4:
Now you can put the marker aside and use the pen instead. For this trial, the instructions are the same. This page is for practice. Begin. [Pause CD]

[11]
Now turn the page. Start in the upper left hand corner. Just connect two or more dots to make unique designs. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down.

[Pause CD]

[12]
Now turn the next two blank pages until you get to the practice sheet. [Pause CD]

[13]

Trial 5:
The instructions are the same for this trial. This page is for practice. Begin. [Pause CD]

[14]

Now turn the page. Start in the upper left hand corner. Ready--BEGIN.

STOP. Finish the last design you were working on and put your pen down. [wait 3 sec.]
[Pause CD]

[Proctor, take up the RFFT forms, and put them in the subject's folder. Be sure not to mix them up! Take up markers]

Appendix F

Informed Consent I

Title of Experiment: Cerebral activation to whole-body rotation.
Principle Investigator: David W. Harrison
Co-Investigator: Joseph E. Carmona

I. Purpose of this research

You are invited to participate in a study about cognition and health. This questionnaire will assess medical, interpersonal and personality characteristics.

II. Procedures

To accomplish the goals of this study, you will be asked to complete a questionnaire about emotion and health. Based on the answers provided in the questionnaire you may or may not be contacted for participation in the second portion of the study. Next you will be given two cognitive tests. Each take about 10 minutes to perform.

III. Risks

There will be minimal discomfort associated with the completion of the questionnaires or tests.

IV. Benefits

Your participation in this research will help clinical psychologists better understand correlates of cognition and health. No promise of benefits has been made to encourage you to participate.

V. Anonymity and Confidentiality

The results of this study will be confidential. The information you provide will not include information that can identify you (e.g., name, etc.). Instead, a subject number will be used on all forms you complete. Only the subject number will be used to identify you during data analysis and during the write up of the study results. The only instance in which confidentiality may be broken is to avert risk of harm to self or others. In the event that this occurs a referral will be made to the appropriate agencies (law enforcement).

VI. Compensation

You may receive one extra credit point for the psychology class you enrolled in. You are also eligible to be entered in a raffle to win a $20 gift certificate from Amazon.com. For alternative methods of receiving extra credit, talk to your professor. If, as a result of this

procedure, you should seek counseling, treatment will be made available at the Psychological Services Center and the University Counseling Center.

VII. Freedom to Withdraw

You are free to withdraw from this study at any time without penalty. If you choose to withdraw, you will still receive the extra credit and will not be penalized by any reduction in points. Talk to your professor if alternative forms of extra credit are desired.

X. Subject Permission

I have read and understand the informed consent and conditions of this project. I have had 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.

_____

Participant Signature Date

_____

Date of Birth Age

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

_____

Joseph E. Carmona
jcarmona@vt.edu
540-231-6914

_____

David W. Harrison, PhD.
dwh@vt.edu
540-231-4422

_____

Dr. David M. Moore
IRB Chair
moored@vt.edu
540-231-4991

Informed Consent II

Title of Experiment: Cerebral activation to whole-body rotation.
Principle Investigator: David W. Harrison
Co-Investigator: Joseph E. Carmona

I. Purpose of this research

You are invited to participate in a study about brain activity, cognition, and dizziness. This study will involve measurements of brain activation and cognitive tests after rotation in a specially designed chair.

II. Procedures

To accomplish the goals of this study, we will prepare your forehead, left cheek, and ear lobes with an alcohol and specialized EEG cleaning solution. You will then be fitted with a cap that collects electroencephalographic (EEG) information from the scalp. The EEG cap will include use of a water-soluble gel on the scalp. You will be asked to remove all jewelry. You will also be fitted with skin electrodes and gel on the surface of your left-hand fingers in order to collect skin conductance. Once fitted with the cap and finger electrodes, you will then be asked to complete some surveys that take approximately 5 minutes in length. After completion of the cognitive task, you may be asked to sit still while being rotated in a chair for about 1 minute. After rotation, you will be asked to take some more surveys (5 minutes).

This project will take approximately 75-90 minutes.

III. Risks

You may experience some discomfort associated with the rotating chair. You may develop motion sickness, including the feelings of nausea, headache, dizziness, and vomiting during or following procedures. You are free to request termination of rotation at any time simply by telling the investigator that you feel too uncomfortable to continue. You will not be penalized in any way if you request early termination of rotation. You may terminate experiment at any point without penalty.

IV. Benefits

Your participation in this research will help provide for a better understanding physiological correlates of cognition, balance, and stress. No promise of benefits has been made to encourage you to participate. You may receive a synopsis or summary of this research when it is completed. Please give a self addressed stamped envelope to the experimenter if you wish for a synopsis.

Immediately following completion of the experiment you will have the opportunity to discuss the nature and purpose of the research. Any questions you may have regarding the study will be answered at this time or anytime thereafter should questions arise later.

V. Anonymity and Confidentiality

The results of this study will be confidential. The information you provide will not include information that can identify you (e.g., name, etc.). Instead, a subject number will be used on all forms you complete. Only the subject number will be used to identify you during data analysis and during the write up of the study results. The only instance in which confidentiality may be broken is to avert risk of harm to self or others. In the event that this occurs a referral will be made to the appropriate agencies (law enforcement). Treatment will be made available at the Psychological Services Center and the University Counseling Center. Should you desire counseling the university will not be responsible for payment.

VI. Compensation

You may receive two extra credit points for participation in the project. You are also eligible to be entered in another raffle to win a $20 gift certificate from Amazon.com. You will need to speak with your instructor to verify that the instructor accepts bonus credits. For alternative methods of receiving extra credit, talk to your professor. No other compensation is offered in connection with this project.

VII. Freedom to Withdraw

You are free to withdraw from this study at any time without penalty. If you choose to withdraw, you will still receive the extra credit and will not be penalized by any reduction in points.

X. Subject permission

I have read and understand the informed consent and conditions of this project. I have had 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.

_____

Participant Signature Date

_____

Date of Birth Age

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

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