Attention to Change:

A Neurological Investigation

A Thesis

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DEDICATIONS

To my husband Greg,

and children Ethan, Thea, and Sinjin,

for providing me perspective,

and to my mother,

Linda Z. Lease, Ph.D.,

who always said,

"The 'P' in Ph.D. is for perseverance."

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ABSTRACT
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Neglect, a dramatic manifestation of attention, is a primary source of stroke-related long-term disability (Katz, Hartman-Maeir, Ting, & Soroker, 1999). Subtle attention deficits (extinction) may recover more slowly than overt symptoms (neglect). (Heir, Mondlock, & Caplan, 1983) While often not captured by traditional bedside tests, subtle deficits have real-world implications. To guide rehabilitation efforts, it is clinically relevant to examine attention in a 6-month post-lesion population.

The visual attention tasks in this study were a simple detection task and a change detection task. A novel component of this study was vigilance, sustaining attention over extended periods of time. The frontal lobes are associated with vigilance (e.g. Manly & Robertson, 1997), and the frontal-parietal cortex mediates spatial attention (e.g. Beck, Rees, Frith, & Lavie, 2001). This study examined the role of the frontal-parietal network in detecting change at spatial locations over time.

Nine frontal-lesioned, seven parietal-lesioned, and twenty-nine non-lesioned individuals were administered the Detection and Monitoring Tasks. The Detection Task required participants to report whether the letter 'T' appeared, and if so, in which hemispace. The Monitoring Task involved bilateral stimuli presentation of two blinking red 'T's. The participants were asked to report whether either letter changed to green, and if so, in which hemispace. The temporal component of both tasks was for the stimuli to appear (Detect Task) or change (Monitor Task) over the time intervals of 200, 800, 1400, or 2000 msec.

Detection Task: No participants showed an accuracy performance difference over time or hemispace. Contralesional accuracy performance was worse than Ipsilesional performance for Parietals, but not Frontals. Contralesional RT performance, relative to Ipsilesional, was worse for all patients. Parietals performed significantly worse (accuracy and RT) than Controls.

Monitoring Task: No participants showed a performance difference over time. Surprisingly, all participants showed a hemispheric difference. Accuracy performance for right hemispace was significantly worse than left hemispace. This left-hemispace advantage may explain why patients did not demonstrate worse Contralesional accuracy performance, relative to Ipsilesional, despite slower Contralesional RT performance. Parietals performed worse (accuracy and RT) than Controls. A trend suggested better accuracy performance for Frontals relative to Parietals.

1. INTRODUCTION

Brain lesion, often the result of stroke, can lead to long-term difficulty performing daily living tasks. One year post-injury, stroke survivors demonstrate dissatisfaction correlating with activity limitation and restricted participation. (Hartman-Maeir, Soroker, Ring, Avni, & Katz, 2007) Neglect, a dramatic manifestation of attention, is a primary source of stroke-related long-term disability (Katz, Hartman-Maeir, Ting, & Soroker, 1999). While overt attentional deficits following brain lesion may subside, subtle attentional deficits may persist. For example, in right hemisphere stroke patients, Hier, Mondlock, and Caplan (1983) note rapid recovery for left neglect but slower recovery for extinction. Subtle attentional deficits may not be captured with traditional bedside tests, but they still have implications for real-world activities such as driving. For the purpose of guiding rehabilitative efforts, it is clinically relevant to examine attentional deficits in a 6-month post lesion population.

This dissertation reports a brain-behavior study of attention. The empirical literature supporting the idea that frontal-parietal brain areas mediate attentional behavior (e.g. Beck, Rees, Frith, & Lavie, 2001) was reviewed, with neglect examined as a specific attentional behavior. The attentional tasks utilized in this study were a simple visual detection task and a monitoring/ detecting change task. A novel component of this study was time. Vigilance performance, sustained attention, was observed by evaluating performance at extended time points on both the Detection and Monitoring tasks. The purpose of this study was to examine the role of the frontal-parietal network in detecting change at spatial locations over time.

1.1 Unilateral Neglect

Unilateral spatial neglect is a striking disorder of cognition associated with diminished awareness for space opposite to the damaged cerebral hemisphere. This diminished awareness has also been referred to as spatial neglect, hemineglect, imperception, hemi-inattention, and visual-spatial agnosia. (Diller & Riley, 1993)

Neglect patients fail to observe and react to new meaningful information presented in hemispace contralateral to their brain lesion. (Heilman, Bowers, Valenstein, & Watson, 1987) Hemianopia, a visual field cut or blindness for half of vision in each eye, may occur when one side of primary visual cortex (or all the fibers leading to that side) is damaged. (Lezak, 1995) Despite the absence of hemianopia, when a neglect patient's attention is attracted by a stimulus presented in the visual field on the same side as their lesion, he or she may fail to perceive stimuli in the visual field opposite their lesion. (Denny-Brown, Meyer, & Horenstein, 1952; Patterson & Zangwill, 1944) Behaviors demonstrated by these patients include ignoring food on the side of a plate that is opposite the lesion, neglecting to dress the side of the body that is opposite the lesion, and failing to groom the side of the face that is opposite the lesion. (Filley, 1995; Chatterjee & Mennemeier, 1998; Beschin & Robertson, 1997) This failure to respond to objects or situations in contralesional space cannot be explained by a sensorimotor deficit (Heilman, 1979; Heilman, KM, Valenstein, E., & Watson, RT, 2000) such as a conjugate gaze deviation towards right visual field, a deficit found in neglect as well as homonymous hemianopia (Gainotti, 1993). Despite the resemblance between hemineglect and hemianopia, there is a distinct difference. Patients with hemianopia learn to behaviorally compensate for their lack of vision by turning their head, while

patients with visual neglect do not appreciate the part of the world they are missing and may demonstrate a lack of concern and awareness of their deficit. (Filley, 1995) This lack of awareness is particularly dramatic when neglect patients do not recognize or deny ownership of their own contralesional limb. (Babinski, 1914, as cited in Filley, 1995)

Neglect syndrome occurs much more often and is more severe for lesions to the right hemisphere than the left (e.g., Mesulam, 1981; Gainotti, Messerli, & Tissot, 1972). The syndrome commonly results from right parietal lesions (Vallar & Perani, 1987), resulting in neglect for the left side of space. Attentional theories of neglect, discussed later, address this hemispheric asymmetry. In addition to visual deficits, neglect symptoms may manifest themselves in other sensory modalities, such as tactile or auditory deficiencies. (Watson & Heilman, 1979; Bisiach, Capitani, & Porta, 1985; Sandifer, 1946) Given that parietal lobes are crucial in visual-motor integration, it is not surprising that a left parietal lesion can lead to constructional impairment. For example left parietal lesion individuals require significantly more time to complete a visual construction task than control or right parietal lesion individuals (Mack & Levine, 1982). However, typically the incidence and severity of defect is greater for right hemisphere lesions. In the same study, in terms of frequency, right lesioned patients were inferior to left lesioned patients and controls. (Mack & Levine, 1982)

Assessment of Neglect 1.1.1

There are numerous tasks used to clinically detect neglect and assess its severity. The simplest test, line bisection, requires no specific test material. The patient is asked to mark the exact middle of several horizontal lines of differing lengths and the discrepancy distance is measured. (Filley, 1995) Patients with left-sided neglect usually put their

mark to the right of the actual middle. (Schenkenberg, Bradford, & Ajax, 1980) Another simple task is the cancellation task. Patients are asked to 'cancel' certain targets from an array, such as in the letter cancellation task where the subject is to cross off randomly placed letters on a sheet of paper. Typically, left-sided targets are neglected (Albert, 1973) and often patients ignore targets close to the body (Chatterjee, Thompson, & Ricci, 1999). Reading tasks and drawing tasks, such as copying a flower or drawing a clock from memory, are also used. In drawings, neglect patients usually omit details on the side of the drawing opposite their lesion. (Lezak, 1995)

Neglect patients can also be assessed for extinction to double simultaneous stimulation. Extinction occurs when a stimulus, perceived in isolation, is no longer perceived when a competing stimulus is presented simultaneously. (Bender & Furlow, 1945; Denny-Brown, Meyer, & Horenstein, 1952) This unilateral inattention or suppression to one side of the body may be present during visual, tactile, and auditory evaluation (Filley, 1995; Heilman, Pandya, & Geshwind, 1970) and even during assessment of weights simultaneously placed in a patient's hands (Chatterjee & Thompson, 1998). The deficit in extinction seems to be attentional, since patients can detect single contralesional events (e.g. Bender, 1952). Additionally, if a patient is asked to ignore the ipsilesional event during simultaneous presentation, the probability of detecting the contralesional event increases. (Karnath, 1988)

Extinction is not necessarily a mild form of neglect (Driver et al., 1997) and moreover, can be viewed as a separate, dissociated phenomenon. (Halligan & Robertson, 1992) Clear evidence of this notion is the demonstration of neglect in the absence of extinction. Goodrich and Ward (1997) describe a patient with left neglect following a

right parietal infarct that shows 'anti-extinction'. Patient V.H.'s performance was better for detection of contralesional targets when presented simultaneously than when the contralesional target appeared alone. (Goodrich & Ward, 1997)

Theories of Neglect 1.1.2

Attentional Theories. Attentional theories view neglect as a disorder of spatial attention. Many attentional theories attempt to explain why left neglect is more common and more severe than right neglect. Kinsbourne hypothesizes that each hemisphere generates a vector of spatial attention toward contralateral space, and that the contralateral hemisphere inhibits these vectors. (Kinsbourne, 1970; 1987) Additionally, the vector of the left hemisphere is powerfully directed in comparison to the right hemisphere's weaker vector. Thus, after right-brain damage, the vector of the left hemisphere is unhampered resulting in a powerful orientation to the right. In contrast, after left-brain damage, the orientation to the left is not as powerful because the right hemisphere's natural vector is only weakly directed. Hence, left-sided neglect is more common and more severe than right-sided neglect.

In contrast to Kinsbourne's theory, Heilman and Mesulam posit that the right hemisphere is dominant for arousal and spatial attention. (Heilman & Van Den Abell, 1980; Mesulam, 1981; Mesulam, 1990) Essentially, the right hemisphere mediates both visual fields and the left mediates the right field. Evidence supporting right hemisphere dominance includes greater EEG slowing for right-brain lesion patients in comparison to left-brain damage patients and diminished GSR for right-brain lesion patients in comparison to left-brain lesion and normal control patients (Heilman, Schwartz, & Watson, 1978). Heilman and colleagues hypothesize that the right hemisphere can direct

attention into both hemispaces, while the left hemisphere only directs attention into contralateral space. (PET studies support the idea that the right hemisphere can direct attention into both hemispaces. (Corbetta, Meizen, Shulman, & Peterson, 1993)) After a left-brain lesion, the right can compensate for the damage by directing attention into both hemispaces resulting in less severe neglect. However, after right-brain damage, the left hemisphere is unable to compensate by directing attention into left hemispace, resulting in much more severe left-sided neglect.

Posner and coworkers explained spatially directed attention as composed of the elementary operations 'shift', 'engage', and 'disengage'. (Posner & Dehaene, 1994; Posner, Walker, Friedrich, & Rafal, 1984) The disengage operation is altered in patients with neglect due to middle cerebral artery (MCA) infarct (Morrow & Ratcliff, 1987) as well as in patients with extinction and/or more focal parietal lobe damage (Posner, Walker, Friedrich, & Rafal, 1984). Patients with right superior parietal damage have a 'disengage deficit' where they have selective impairment for disengaging attention from right-sided stimuli, prior to shifting and engaging left-sided stimuli. (Posner, Walker, Friedrich, & Rafal, 1984) This disengage deficit may contribute to some neglect symptoms, especially visual extinction. (Chatterjee & Coslett, 2003)

The aforementioned theories explain neglect as a deficit in selection for perception. It has also been posited that processing may involve selecting spatial locations for actions. (Watson, Valenstein, & Heilman, 1978) 'Intentional' neglect is the notion that patients are disinclined to initiate movements or move towards or into contralateral hemispace. (Heilman & Valenstein, 1979; Watson, Valenstein, & Heilman, 1978; Heilman, Bowers, Coslett, Whelan, & Watson, 1985) Analogously, Posner and

colleagues proposed a posterior and an anterior attentional network, where the anterior attentional network is involved in selecting locations for actions. A further suggestion is that action preparation may be critical to perception. (Rizzolatti, Matelli, & Pavesi, 1983) Although attention and intention are usually inextricably linked, as attention is typically directed to objects on which one intends to act, several researchers have devised clever ways of separating the two by using pulleys, mirrors, and cameras. (e.g., Coslett, Bowers, Fitzpatrick, Haws, & Heilman, 1990; Bisiach, Geminiani, Berti, & Rusconi, 1990; Tegner & Levander, 1991) Generally, these studies tried to separate where patients were looking from where their limbs were acting.

Representational Theories. Bisiach and colleagues have proposed that the behavioral deficits of neglect patients may be due to the inability to form adequate contralateral mental representations of space. (Bisiach, Capitani, Luzzatti, & Perani, 1981; Bisiach, 1993) A classic study by Bisiach & Luzzatti (1978) involved patients imagining the Piazza del Duomo in Milan, Italy and reporting what they saw from two perspectives: looking across the square towards the cathedral, and looking from the cathedral across the square. Patients reported only the right half of the imagined scene, resulting in their report of different structures for each condition. In addition to difficulty recalling contralateral representations from memory, neglect patients may also be deficient in forming new contralateral representations. (Bisiach, Luzzatti, & Perani, 1979)

Representational theories of neglect are often contrasted to attentional and intentional theories, however this distinction may not be necessary. Researchers often view attention directed to stimuli, but Farah and colleagues emphasize that attention is

allocated to internal representations of stimuli. (Farah, Wallace & Vecera, 1993) Simple visual elements such as color, movement, and form are preattentively processed at different locations within the visual cortex. (Van Essen, Felleman, DeYoe, Ollavaria, & Knierman, 1990) Internal representations are formed by binding these simple visual elementary features into percepts. We become aware of external stimuli by mentally reconstructing them. (Chatterjee, 2002)

1.1.3 Cortical Lesions

Neglect is a more common and more severe result of right hemisphere damage. (Mesulam, 1981; Heilman, Watson, & Valenstein, 1985; Weintraub & Mesulam, 1987) The typical lesion encompasses the right inferior parietal lobe, Brodmann Areas 39 and 40. (Heilman, Watson, & Valenstein, 1994) Neglect has also been observed following dorsolateral prefrontal (DLPFC) (Heilman & Valenstein, 1972; Husain & Kennard, 1996) and cingulate lesions (Watson, Heilman, Cauthern, & King, 1973), as well as basal ganglia (Hier, Davis, Richardson, & Mohr, 1977) and thalamic lesions (Watson, Valenstein, & Heilman, 1981). Severe neglect is more probable when the posterior-superior longitudinal fasciculus and the inferior-frontal fasciculus are damaged in addition to cortical areas. (Leibovitch et al., 1998)

Due to the observation that different cortical and subcortical lesions produce neglect, Heilman and coworkers posit a distributed network that mediates spatially directed attention. (Heilman, 1979; Watson et al., 1981) Mesulam (Mesulam, 1981; 1990) proposed a similar model, where distinct regions within a large-scale network execute distinct aspects of one's interaction within their spatial environment. He suggested lesion to the posterior parietal area results in perceptual aspects of neglect, and

lesions to the dorsolateral prefrontal area produce deficient contralesional exploratory behavior. While it is frequently cited that parietal lesions produce perceptual manifestations of neglect and frontal lesions produce response or motor aspects of neglect, this association is not consistently reported. (Chatterjee, 2002)

1.2 Neuroimaging Studies of Spatial Attention and Representation

Positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) are non-invasive methods for imaging the human brain at work. Both methods allow the exploration of neurophysiological changes during specific cognitive tasks. As previously mentioned, some theorists hypothesize that the right hemisphere directs attention to right and left space, with the left hemisphere directing attention only contralesionally. (Heilman, Watson, & Valenstein, 1985; Heilman & Van Den Abell, 1980; Mesulam, 1981) Thus, one would predict greater right than left hemisphere activation for attention shifts in either direction, but preferential left hemisphere activation for attention directed to the right. PET results support these predictions. Greater right hemisphere activation is observed when attention is shifted to both right and left hemispace, and greater left hemisphere activation is observed when attention is shifted to right hemispace. (Corbetta, Meizen, Shulman, & Peterson, 1993; Gitelman et al., 1999; Kim et al, 1999)

In a review of brain imaging and attentional control in the human brain, Nobre (2001a) notes that many studies have investigated visual spatial orienting. Brain areas revealed most consistently include the posterior parietal cortex and the intraparietal sulcus (banks of Brodmann Areas 7 and 19), as well as frontal regions including the frontal eye field (Brodmann Area 6). Similarly, animal lesion and single cell

neurophysiological studies support the idea that parietal and frontal association cortices mediate spatial attention and awareness. (e.g. Burcham, Corwin, Stoll & Reep, 1997; Mountcastle, Lynch, Georgeopolous, Sakata, & Acuna, 1975)

Corbetta et al. (1993) found that when attention was shifted to peripheral locations, the superior parietal and superior frontal cortex were more active, relative to when attention was maintained at the center of gaze. While activation was bilateral, activation was greatest in the hemisphere contralateral to the attended target. Other areas that were active, albeit not consistently, include the right inferior parietal cortex (Boardman Area 40), superior temporal sulcus (Boardman Area 22) and the anterior cingulate. Other studies utilizing PET as well as fMRI also found shifting attention resulted in an increase in blood flow around the intraparietal sulcus. (Nobre, Sebestyen, Gitelman, & Mesulam, Frackowiak, & Frith, 1997; Corbetta, 1998) Corbetta and coworkers (1998) also found activity in the post-central and pre-central sulcus during shifts of attention and similar activity increases in right intra-parietal sulcus and precentral cortex during sustained attention directed to a peripheral location, rather than simply shifting to a peripheral location.

The DLPFC is another region engaged when visual attention is shifted to different locations. Rosen and colleagues (1999) found endogenous strategy-driven and exogenous stimulus-related orienting activated bilateral parietal as well as dorsal premotor regions, including the frontal eye fields. (Rosen et al., 1999) In addition, right DLPFC was activated selectively in the endogenous condition. Since DLPFC lesions produce attentional disorders (Heilman & Valenstein, 1972; Husain & Kennard, 1996), it is plausible these areas are linked to posterior parietal areas involved in directed spatial

attention. (Chatterjee, 2002) Neural activation in the parietal and frontal cortices in neuroimaging and electrophysiological studies clearly show spatial attentional processes during covert orienting. (Corbetta, 1998) Furthermore, the frontoparietal network enhances visual processing by interacting with the ventral visual system during object analysis. (Corbetta, 1998)

1.3 Dorsal and Ventral Visual Pathways

There is evidence of multiple cortical areas highly specialized for processing specific visual features (e.g. Zeki & Shipp, 1988) and there are distinct anatomical pathways for visual processing (Ungerleider and Mishkin, 1982). Identifying *what* involves a subsystem conveying information about form and another conveying information about color. Both terminate in the inferior temporal cortex, in a complex of areas important for the recognition of form. Object features are represented in this ventral visual pathway. Identifying *where* involves another system that terminates in the posterior parietal cortex. Spatial features are represented in this dorsal visual pathway. If visual information is processed in parallel pathways, how is this information integrated into a cohesive perception? One research question has been to determine the mechanism by which the brain associates these streams of information. The name given to this unspecified mechanism is the binding mechanism. (Kandel, Schwartz, & Jessell, 1995)

It has been hypothesized that attention to spatial locations is necessary to accurately bind object features. (Treisman & Gelade, 1980) Divided or reduced attention (Arguin, Cavanagh, Joanette, 1994) as well as inaccurate/degraded spatial information (Friedman-Hill, Robertson, & Treisman, 1995) disrupts feature binding. Patient R.M., with bilateral parieto-occipital lesions, demonstrated the selective inability

to combine colors and shapes, despite intact visual fields and normal uncorrected visual acuity. (Friedman-Hill, Robertson, & Treisman, 1995) Patient R.M. was able to recognize letters and shapes, but had difficulty in correctly binding the elementary features of color and size when two or more shapes were presented. When R.M. was presented with two colored letters (e.g. a red X and a blue O) and asked to report the name and color of the first letter he saw, he reported illusory conjunctions (e.g. a red O or a blue X) at a rate of 13% even when display times lasted 10 seconds. When presented with geometrical shapes of various sizes and asked to report which shape was taller, he made significantly more errors for stimuli presented simultaneously compared to those presented sequentially, even when total display time was twice as long in the simultaneous condition. The better performance on the sequential presentation suggests he had spatial deficits in binding errors, rather than a generalized problem in feature integration for forming perceptual objects. When tested, this patient was unable to judge relative or absolute visual locations. The authors interpreted the data to suggest explicit spatial information associated with the dorsal pathway is necessary for feature binding.

1.4 Monitoring for Change

The ability to detect changes in the environment can be consequential for survival. Attending to changes in one's sensory environment is necessary for evaluating and modifying behavior in the presence of threats or opportunities. Abrupt changes in the environment often draw our attention and preferentially enter our awareness. (Downar, Crawley, Mikulis, & Davis, 2000) When the ability to attend to stimuli is diminished, as in neglect syndrome, awareness of the stimuli is diminished as well. (Halligan & Marshall, 1998a)

Detection of change in the sensory environment was investigated with fMRI. (Downar, Crawley, Mikulis, & Davis, 2000) The visual task was to passively observe a red or blue abstract shape that alternated independently. The cortical areas responsive to the visual sensory modality included bilateral visual association cortices of the dorsal and ventral visual-processing streams as well as activation in the right superior parietal lobule, on the superior bank of the intraparietal sulcus. Downar and colleagues (2000) also investigated areas of activation for auditory and tactile modalities. A 'transition' was defined as a change from one stimulus modality to another. During detection of these sensory changes, the greatest overall volume of activation was in the right temporoparietal junction (TPJ). Other areas of high activation included right inferior frontal cortex, left anterior cingulate and supplementary motor areas, and right anterior insula. There was smaller activation in left TPJ and inferior frontal cortex, right posterior insula, and right middle temporal gyrus. The authors noted that the areas activated and their degree of activation agree well with neuroanatomical correlates of sensory neglect. Downar and colleagues (2000) comment that the amount of overlap between known anatomical correlates of neglect and the areas activated in their study highlights the intimate relationship between detection of salient sensory events and selection of salient stimuli for entry into awareness.

EEG studies also support the involvement of the TPJ and inferior frontal gyrus in detection of change in sensory input. P3a ERP is normally elicited by unexpected, task irrelevant stimuli, and P3b ERP is normally elicited by expected, task-relevant stimuli. Knight and colleagues found lesions of the TPJ result in an amplitude reduction of the P3a and the P3b ERP for visual, auditory and tactile stimuli. (Yamaguchi & Knight,

1991b; Swick & Knight, 1988; Knight, Scabini, Woods, & Clayworth, 1989; Yamaguchi & Knight, 1991a) In addition, there was a reduction in P3a for all three modalities for prefrontal lesions, although parietal lesions immediately dorsal to TPJ did not affect P3a or P3b. (Swick & Knight, 1998; Yamaguchi & Knight, 1991a; Knight, 1984)

By utilizing error-related negativity (ERN), an event-related brain potential that reflects anterior cingulate action monitoring, Knight further investigated prefrontal lesion patients. (Gehring & Knight, 2000) Action monitoring was defined as detecting errors and behavioral conflict. The letter-discrimination task required selective attention to the relevant stimulus feature of color. Subjects were presented two letters, one red and one green, and were cued to respond to only one color (e.g. green). The subject indicated whether the cued letter was an "H" or an "S" via a hand squeeze. Half of the trials were congruent trials where the irrelevant flanking letter was identical to the target letter (i.e. green H, red H). The other half of the trials were incongruent letters (i.e. green H, red S) requiring the subject to ignore the distracting letter (analogous to the Stroop task). Patients and control subjects had approximately the same proportion of errors, with mean correct RT significantly slower for the PFC group. Control subjects had greater ERN activity for error trials than for correct trials, while DLPFC lesion subjects demonstrated no difference in ERN activity between correct and error trails. Additionally, frontally damaged patients had fewer error corrections and showed less force inhibition on error trials, suggesting impaired capacity for control, despite intact monitoring capacity. The authors conclude the DLPFC appears to interact with the anterior cingulate cortex in monitoring behavior and in guiding compensatory systems. (Gehring & Knight, 2000)

Activation has also been found for the DLPFC and parietal lobes for a task involving the detection of visual change. (Beck, Rees, Frith, & Lavie, 2001) Subjects were instructed to detect changes in one of two peripherally presented images while simultaneously detecting letters. Subjects were presented face or place images 2° to the left and right of a fixation in a 'flicker method' involving cycling the images on and off every 500 msec. The flicker method was utilized because stimuli presented in this fashion can result in change detection, consciously detecting a visual change, or change blindness, being functionally blind for the exact same stimuli. Participants were asked to indicate via key press whether a change occurred in one of the two images. In the simultaneous letter detection task, subjects were presented two letter strings 2.4° above and below the fixation and were asked to make a button press for the target letter X. This task ensured subjects maintained fixation. Conscious change detection was defined as the comparison of detected and undetected change trials. For the conscious detection of change, functional MRI scans revealed activation in the ventral visual stream and frontoparietal cortex. Regardless of whether the stimulus was a face change or place change, the three areas activated by conscious change detection included: fusiform gyrus, bilateral parietal lobe, and right DLPFC. Areas of activation common for both face and place conscious change detection included the parietal and DLPFC, but no ventral areas. Change blindness was defined as the comparison of trials where no change was detected , despite a genuine change, verses no-change trials. For change blindness, initial analysis revealed activation in the fusiform gyrus (close to the area activated for consciously detected face change), lingual gyrus and inferior frontal gyrus, but no dorsal activation. When this portion of the study was replicated with measures added to control for eye

movement, no activation was found. Thus, conscious dorsal activity in the parietal lobes and DLPFC was associated solely with conscious detection of change. These findings conflict with the traditional view of a 'conscious' ventral pathway and 'unconscious' dorsal pathway. (Milner & Goodale, 1995) Previous research on visual awareness typically emphasizes the ventral visual pathway to the exclusion of the dorsal pathway, but this data suggests visual awareness arises from an interaction of the ventral and dorsal streams.

1.5 Vigilance

Normal attentional function theories posit visual stimuli are competing for limited attentional resources. (e.g. Treisman, 1969; Duncan, 1980) Some stimuli are selected and reach awareness while others are disregarded or fail to reach awareness. (Duncan & Humphreys, 1989) Extinction may be a spatially specific exaggeration of normal attentional limitation, with contralesional stimuli 'losing' in the competition for selection. (Ward, Goodrich, & Driver, 1994) When two items are presented simultaneously, the ipsilateral stimulus 'wins' the competition for selection. However, when the contralesional stimulus appears alone, it can still attract attention to itself because there are no other competitors.

Object (what) and spatial (where) information provide effective frameworks for focusing perception and action. In our dynamic world, time (when) information might also be useful for filtering awareness. (Nobre, 2001b) The temporal moment of a stimulus that appears or changes may afford additional imperative information for the integration and analysis of its elements. (Nobre, 2001b) For example, complex movements require temporal coordination. (Georgopoulos, 2000)

Vigilance is active when selective attention is utilized over an extended period of time. Sustained attention is useful for activities such as hitting a ball with a bat, which requires integrating the timing of the throw with the spatial location of the ball. Pastimes that require sustained attention include daily life activities such as reading, as well as safety-critical aspects of driving. (Bunce, 2001) While the ability to attend over a period is important for recreational activities, it is imperative for road and highway safety. (Bunce, 2001) The frontal cortex appears to be associated with the ability to maintain a vigil. (Manly & Robertson, 1997; Robertson, Manly, Andrade, Baddeley, & Yiend, 1997)

Just as with object and spatial information, task demands can influence vigilance performance due to temporal capacity limitations of selective attention. Events occurring in close temporal proximity compete for limited resources and can interfere with one another. (Pashler, 1994; Raymond, Shapiro, & Arnell, 1992) A vigilance taxonomy, consistent with resource theory (Kahneman, 1973), was proposed by Parasuraman and Davies (1977) where task parameters affect performance by placing attentional demands on a limited-capacity information processing system. Task manipulations, such as changing event rate or the type of the stimulus discrimination to be made, can increase the demands on attentional resources. Behaviorally, these manipulations are observed by lower average performance and declines in performance as a temporal epoch progresses.

2. CURRENT STUDY

Although there is a broad similarity of symptoms, neglect is a heterogeneous disorder with a wide variety of symptoms. (Chatterjee, 1998; Halligan & Marshall, 1998b; Stone, Halligan, Marshall, & Greenwood, 1998) It is this heterogeneity of neglect symptoms that has made it possible to study the dynamics (attention and representation) of spatial systems. (Chatterjee, 1998) There is a preponderance of evidence demonstrating frontal and parietal cortices are neuroanatomical correlates of sensory neglect. Similarly, frontal-parietal networks, particularly in the right hemisphere, mediate spatial attention and representations.

The current study is an investigation of attention, utilizing right and left focal brain damaged patients. After recovery from brain damage, patients continue to demonstrate functional disabilities, some of which may manifest as very subtle deficits. Sustained attention is crucial for activities such as driving and subtle vigilance deficits may prove detrimental in terms of road and highway safety. Deficits in detecting environmental changes may also be detrimental for other safety-critical activities. Beck and colleagues (Beck, Rees, Frith, & Lavie, 2001) demonstrated that conscious detection of change is associated with activation of the parietal lobes and DLPFC. Thus, one would predict that lesions to this network would affect attention to visual changes in the environment, or monitoring ability, in addition to simple detection ability. Preliminary pilot data with patients support this hypothesis. Furthermore, it was predicted that deterioration of accuracy performance would increase over time because of temporal capacity limitations. This decrement was expected to be greater for patients with frontal lesions since the frontal cortex seems to be associated with the ability to maintain a vigil.

The present study proposes to examine the ability of frontal and parietal lesion patients in detecting stimuli and monitoring changes in visual stimuli over a temporal epoch. The monitoring task involves visual attention directed to changes in the elementary stimulus feature color. Research hypotheses are described further below.

2.1 Hypotheses for Detection Task

For the Detection Task, it was predicted that patients with frontal and parietal damage would be less accurate in detecting contralesional stimuli than ipsilesional stimuli, and that the deficit would be further exaggerated for detection later in the temporal epoch on their contralesional side. In a pilot study, four right brain damaged patients were slower or less accurate in detection of an event in left hemispace than on the right. A decline in performance over a temporal epoch was expected because of increased spatial and temporal attentional demand on their limited-capacity processing system. It was predicted that age-matched controls would show no hemispheric differences and that performance over the temporal epoch would not change.

2.2 Hypotheses for Monitoring Task

For the Monitoring Task, it was predicted that patients with frontal and parietal damage would be less accurate in monitoring change in contralesional stimuli than ipsilesional stimuli, and that the deficit would be further exaggerated for monitoring change later in the temporal epoch on their contralesional side. In a pilot study, all four right brain damaged patients were slower or less accurate in monitoring an event change in left hemispace than on the right. A decline in performance over the temporal epoch was expected because of increased spatial and temporal attentional demand on their limited-capacity processing system. It was predicted that age-matched controls would

show no hemispheric differences and that performance over the temporal epoch would not change.

Finally, it was expected that patients with frontal damage would have greater difficulty with the monitoring task over a temporal epoch, in comparison to patients with parietal damage. Frontal regions appear to be predominantly involved in attentional control, monitoring, and vigilance. Pilot data show that the two patients with contralesional monitoring deficits (for a single stimulus onset) had larger lesions involving prefrontal cortices.

3. METHOD

3.1 Subjects

Forty-five individuals participated in this study. Sixteen experimental subjects comprised of nine frontal (five left frontal, four right frontal) and seven parietal (four right parietal, three left parietal) lesioned subjects were recruited from the patient database of the Center for Cognitive Neuroscience (CCN) at the University of Pennsylvania. Twenty-nine neurologically normal age-matched control subjects were recruited from the surrounding Philadelphia area and the surrounding Hazleton, PA area.

The Patient Coordinator locates and screens potential patients that are obtained from monitoring admissions at the Hospital of the University of Pennsylvania, Temple University Hospital, and the Philadelphia Veterans Administration Medical Center. This database is comprised of approximately 170 patients with relatively stable focal lesions resulting from an infarct or tumor resection, as well as patients with other relatively restricted lesions. Each patient is administered a comprehensive neuropsychological battery to assess a variety of cognitive capacities. All database patients received an MRI study to precisely measure and localize their lesioned area if MRI or CT results are not already available.

3.1.1 General Inclusion/ Exclusion Criteria

Experimental subjects included had a frontal or parietal brain lesion, confirmed by a neurologist viewing MRI or CT head scans. Operationally, frontal lesions included, but were not restricted to, damage to Brodmann Areas 44 and 45. Parietal lesions included, but were not restricted to, damage to Brodmann Areas 39 and 40. As this study

investigated a brain-behavior relationship, inclusion criteria were based on brain lesion area to examine subsequent behavior.

All experimental subjects were in the chronic stages, at least six months post-injury. Both the control and experimental groups were given a Handedness Inventory (Briggs & Nebes, 1975). Subjects who demonstrated left-handedness, as determined by a score lower than nine, were excluded. Both groups were administered the Ishihara Pseudoisochromatic Plates to determine whether they had a red/green color perception anomaly. A score of six or more errors lead to exclusion from the study. A Visual Field Screening task was also administered to both groups to determine whether participants had a dense visual cut. Performance was at or above 75% correct for inclusion in this study. Additionally, subjects with a history of drug or alcohol abuse, a history of dementia or other diseases of the central nervous system (e.g. multiple sclerosis or epilepsy), or psychiatric problems were excluded.

3.1.2 Attrition

A total of 11 subjects were excluded from the study for not meeting inclusion criteria or due to incomplete task data. All subjects were compensated monetarily ($15 per hour and parking/transportation/babysitting reimbursement) for their scheduled time regardless of whether they were included in the study. Four subjects were excluded due to extensive drug or alcohol histories that were not reported during previous screening procedures. One individual was excluded due to a 20-year depression history that was not reported during previous screening. Another individual was excluded due to depressive symptomology including decreased sleep in the three days previous to testing and crying during testing reportedly due to the death of her son nine years prior. Two

subjects were excluded due to high Ishihara Plate errors (thirteen and ten errors). Two patients were excluded from the data analysis because they did not complete the full set of trials for the Monitor task. Finally, one control subject was excluded from the study after stating she had experienced a "mild" hemorrhage which had not been previously reported.

3.2 Procedure

The study was reviewed and approved by the Institutional Review Boards of the University of Pennsylvania and Drexel University. The study was reviewed with all subjects by the examiner and all subjects gave informed consent. Subjects were assured confidentiality and were given time to ask any questions before beginning any tasks.

Locations of testing included a testing room at the University of Pennsylvania (Penn), patient's homes, and two testing rooms created in homes in Haddonfield, NJ and in Hazleton, PA. For the Control subjects, 55% were tested at Penn, 24% in Haddonfield, and 21% in Hazleton. For the Frontal subjects, 44% were tested at Penn and 56% were tested in their own homes. For the Parietal subjects, 71% were tested at Penn and 29% were tested in their own homes.

Total testing time was approximately three hours. The duration of testing was deemed too lengthy for some patients, resulting in a few patients being tested over two sessions rather than one. For the Frontal subjects, 33% were tested over two sessions. For the Parietal subjects, 29% were tested over two sessions. All control subjects were tested in one session.

First, the screening tasks were administered in the following order: Hand Preference Questionnaire, Ishihara Plates, and the Visual Field Screening Task. If

subjects met the cut-off requirements of these tasks, they were administered the remaining tests in either of the following pseudo-random orders: Detection Task, BIT, and Monitor Task; or Monitor Task, BIT, and Detection Task. The BIT was administered for descriptive purposes. The Visual Field Screening Task, Detection Task, and Monitoring Task were all created with PsyScope (Cohen, MacWhinney, Flatt, & Provost, 1993). For these three tasks, stimuli were presented on a MacIntosh laptop computer. Subjects were paid $15 per hour and reimbursed for expenses such as parking, babysitting, and meals. When all measures were completed, subjects were provided with the opportunity to ask questions regarding the study.

3.3 Measures

3.3.1 Screening and Descriptive Measures

Hand Preference Questionnaire. This questionnaire is a revision of Annett's (1967) hand preference questionnaire. Briggs and Nebes' (1975) hand preference questionnaire requires the subject to indicate their hand preference for 12 tasks using a five-point scale. Left preferences are scored as negative and right preferences are scored as positive, providing a range of scores from -24 (most left-handed) to +24 (most right-handed). The authors designated scores of +9 and above, right-handed, and scores -9 and below, left-handed, with scores in-between considered ambidextrous. Using this scoring method, the authors found 14% of a group of 1599 college students tested were designated as non-right-handers, a proportion in agreement with the literature. For the current study, a score above 9 was required for inclusion in the study.

Ishihara Pseudoisochromatic Plates. The Ishihara pseudoisochromatic test is the most widely used screening test for red-green color deficiency. (Birch, 1997) The 38-

plate edition is recommended for clinical use. (Birch & McKeever, 1993) The test is most reliable when the transformation and disappearing digit plates (2-17) are used, rather than all 20 screening plates as recommended by the publishers. (Birch, 1997) The subject is asked to tell the examiner the numbers that he or she sees on a given page. They are also told that on some pages they will not see a number. The maximum number of errors for the Transformation and Vanishing plates is 16. Birch (1997) tested 401 people with red-green color deficiency and determined that the combined sensitivity of the Transformation and Vanishing plates is 97.5% on six errors and 99.0% on three errors. The same author tested 471 normal subjects and determined that the combined specificity of the Transformation and Vanishing plates is 95.4 for 6 errors and 94.1 for 3 errors. (Birch & McKeever, 1993) For the current project, subjects were excluded if they made six or more errors.

Visual Field Screening Task. It has been argued that visual field deficits (VFD) exacerbate behavioral manifestations of neglect. However, Halligan, Marshall, and Wade (1990) found severity of neglect did not differ significantly between patients with and without VFD, which lead them to believe poor functional recovery in many VFD patients is due to "the association of sensory loss with the underlying causal factor of neglect." (p. 487) The purpose of the Visual Field Screening task was to screen for dense visual cuts. A warning tone indicated the start of the trial. At 800 msec the letter “T” (0.91° x 1.14° visual angle) appeared on the left or right side of a fixation mark for 600 msec. The stimulus was either a standard red T or a green T, matched for luminance of the red T. Subjects were instructed to keep their eyes on the fixation mark and indicate, via key press with their ipsilesional hand (or right hand for non-patients), the location (left or

right) of the letter "T". Trials terminated after 2500 msec following the onset of the stimuli. Stimuli were presented randomly in two blocks of 20 trials (10 red and 10 green), with a total of 20 trials for each hemispace. If performance was below 75% for either hemispace, the subject was excluded.

Behavioural Inattention Test. Wilson, Cockburn, and Halligan (1987a) created the Behavioural Inattention Test (BIT) to measure unilateral visual neglect. It is comprised of 'conventional' as well as 'behavioral' sub-tests. Only the conventional sub-tests were administered for this project. The six conventional sub-tests included line crossing, star cancellation, figure and shape copying, line bisection, representational drawing, and letter cancellation. For the line-crossing task, the patient is presented with 40 one-inch long lines and asked to cross out all the lines. The patient's score is the total number of lines crossed. Because the four lines in the center are not scored, the maximum score is 36 (18 left, 18 right). The letter cancellation task requires the patient to cancel all the E's and R's from five lines of 34 letters. The maximum score for this task is 40 (20 left, 20 right). For the line bisection task, the patient is instructed to divide three horizontal eight-inch lines at the center. The score is the deviation between the actual center of the lines and the patient's estimated centers. If the patient marks all three lines within 1/2 inch of the center, a total score of nine points was given. Among tests of inattention, line bisection is one of the least sensitive. (Lezak, 1995) The star cancellation task is a jumble of letters, words, and stars. The target stimuli are 56 small stars, two of which are crossed out by the examiner during demonstration. The total score for this task is 54 points (27 left, 27 right). This task correlates well with other inattention tests (Lezak, 1995) and accurately identified an entire group of patients with

inattention. (Halligan, Marshall, and Wade, 1989) The figure and shape copying sub-test involves copying a star, a cube, and a daisy, while the representational drawing task involves drawing a clock face with numbers, a man or a woman, and a butterfly. All of the stimuli for these two sub-tests are bilaterally symmetrical. (Lezak, 1995) Scoring is based on the completeness of the drawings. Copying is more sensitive than drawing for patients with right-sided strokes (Halligan, Cockburn, & Wilson, 1991) and generally, drawings are less sensitive in eliciting inattention than cancellation tasks (e.g. Halligan, Marshall, & Wade, 1989). Inter-rater reliability, parallel form reliability, and test-retest reliability correlations for the entire BIT ranged from .91 to .99. The validity correlation, measured by comparing the behavioural battery to the conventional battery, was .92. Information from the BIT was used for descriptional purposes in this study.

3.3.2 Dependent Variable Measures

The Detection Task of the current study, where subjects were asked to detect the red letter "T" appearing in left or right hemispace, is similar to attention tasks utilized in other visual attention studies. The Monitoring Task of the current study is a change detection variation of the Detection Task, where subjects are asked to detect a color change (red to green) of one of the bilaterally presented flashing red "T's".

Posner and colleagues utilized colored letters in their work on the integration of color and shape information. (Prinzmetal, Presti, & Posner, 1986) Colored letters were also used in another study (noted earlier) examining binding of the elementary features of color and size. Treisman and colleagues presented a patient with two colored letters (e.g. a red X and a blue O) and asked him to report the name and color of the first letter seen. (Friedman-Hill, Robertson, & Treisman, 1995) In a separate study of feature integration

by Briand and Klein (1987), subjects were asked to search for the target letter (R) while being presented distracter letters that could result in illusory conjunctions (PQ) or not (PB). Another similar attention task is Gehring and Knight's (2000) letter-discrimination task (noted earlier), which required selective attention to the relevant stimulus feature of color. Subjects were presented two letters, one red and one green, and were cued to respond to only one color (e.g. green).

In the Monitoring Task of the current study, bilateral "T" letters flashed every 200 msec, similar to the flicker presentation method of visual stimuli by Beck, Rees, Frith, and Lavie (2001). In Beck et al.'s (2001) study (discussed earlier), subjects were instructed to detect changes in one of two peripherally presented images while simultaneously detecting letters. Face or place images appeared to the left and right of a fixation in a 'flicker method' involving cycling the images on and off every 500 msec. Participants indicated via key press whether a change occurred in one of the two images.

Detection Task. This task was used to determine whether patients with frontal and parietal damage had deficits in their ability to detect stimuli onset contralesionally when they were required to be vigilant over a period of time. Specifically, we examined their ability to detect when a target appeared either to the left or right of a fixation mark when stimuli were presented at 200, 800, 1400, or 2000 msec after a warning tone. Prior to the start of this experimental task, stimulus duration was manipulated via a titration version of the task until performance on a central fixation point was between 60-75% correct. The purpose of titrating stimulus duration at a central location was to make task difficulty for patients and controls comparable. Titrating stimulus duration is a procedure utilized by other researchers for presentation of centrally displayed stimuli (Whyte,

Polansky, Fleming, Coslett, & Cavallucci, 1995) as well as single contralesional stimuli (Baylis, Gore, Rodriguez, & Shisler, 2001).

For the titrated version of the Detection task, subjects were instructed to look at a central fixation mark. Following a warning beep and an interstimulus interval of 200, 800, 1400, or 2000, the letter "T" appeared at the center of the screen monitor for 500 msec. The stimuli were a standard red T or a green T, matched for luminance of the red T. Approximately 15% of the trials were catch trials where no stimulus appeared. Subjects were instructed to keep their eyes on a central fixation mark and to indicate, via key press, whether the letter "T" appeared. Trials terminated after 2500 msec following the onset of the stimuli. Stimuli were presented randomly in a single block of 12 trials (ten with stimuli and two catch trials). Stimulus duration was increased or decreased in 100 msec increments for the next set of 12 trials based on the subject's task performance. Once performance was between approximately 60 and 75% correct (seven to nine correct responses), an additional 24 trials were administered. If performance was still between 60 and 75% correct (14-18 correct responses), that became the stimulus duration for the entirety of the Detection Task. It should be noted that many participants had greater than 75% correct at the lowest level of stimulus duration. Thus, further decreases in stimulus duration were not possible.

For the actual experimental Detection task, stimulus duration was previously determined, as described above, but for this description, will be 200 msec. For the Detection task (Figure 1.), the letter "T" (0.91° x 1.14° visual angle) appeared on the left or right side of a fixation mark for 200 msec at 200, 800, 1400, or 2000 msec after a warning tone. The stimuli were a standard red T or a green T, matched for luminance of

the red T. There were also catch trials where no T appeared after the warning tone. Subjects were instructed to keep their eyes on the fixation mark and indicate, via key press with their ipsilesional hand (right hand for control subjects), the location (left, right, or neither) of the letter "T". Trials terminated after 2500 msec following the onset of the stimuli. Approximately 15% of the trials were catch trials where no stimulus appeared. Stimuli were presented randomly in 4 blocks of 56 trials (48 with stimuli and 8 catch trials), with a total of 96 trials for each hemispace, excluding catch trials. If an individual performed at less than chance on the no-stimulus catch trials, their data was to be thrown out. This did not occur. Total number of accurate detection responses for each of the four stimuli onsets (200, 800, 1400, and 2000 msec) was calculated for each hemispace for each subject.

Monitoring Task. This task was used to determine if patients with right frontal or parietal damage were insensitive to changes in stimuli, even when explicitly aware of the presence of these stimuli as demonstrated on their performance in the previous detection task. Specifically, we examined their ability to detect when a target changes color in left and right hemispace when stimuli were presented at 200, 800, 1400, or 2000 msec after a warning tone. Prior to the start of this task, stimulus duration was determined for each subject in a titration version of the task, so that performance on a central fixation point was between 60-75% correct. Again, the purpose of titrating stimulus duration at a central location was to make task difficulty for patients and controls comparable.

For the titrated version of the Monitoring task, subjects were instructed to look at a central red "T" that appeared to blink. The stimuli came on the computer screen for 190 msec epochs, with 10 msec blank/non-stimuli inter-stimulus intervals (ISI's), such

that the red “T” appeared to flash at the center of the screen. (The purpose of the flash was to probe for the ability to monitor change while avoiding potential confounding effects of simply detecting an offset of a stimulus as might have occurred in a pilot version of the experiment.) The central stimulus changed to a green “T” at 200, 800, 1400, or 2000 msec after the start of the stimulus presentation and remained green for 190 msec with another 10 msec blank ISI immediately following. For catch trials, no change occurred during this 200 msec period of time. Immediately following this change (or no change) and ISI, the stimulus returned to the flashing central red “T” presentation. The patient again had 2500 msec to respond following the onset of the stimulus change. Subjects were instructed to keep their eyes on the central fixation mark and indicate whether a change occurred by responding with their ipsilesional hand (right hand for control subjects). Stimuli were presented randomly in a single block of 12 trials (ten with stimuli and two catch trials). Stimulus duration was increased or decreased in 100 msec increments for the next set of 12 trials based on the subject's task performance. Once performance was between approximately 60 and 75% (seven to nine correct responses), an additional 24 trials were administered. When performance remained between 60 and 75% (14-18 correct responses), that duration became the stimulus duration for the entirety of the Monitoring Task. As in the detection task, several participants had greater than 75% correct at the lowest level of stimulus duration. Thus, further decreases in stimulus duration were not possible.

For the actual experimental Monitoring task, stimulus duration was previously determined, as described above, but for this description, will be 190 msec. For the Monitoring task (Figure 2.), following a warning tone, a red letter “T” was presented on

both the left and right side of a fixation mark. The stimuli came on the computer screen for 190 msec epochs, with 10 msec blank/non-stimuli inter-stimulus intervals (ISI's), such that the red "T" appeared to flash on both sides simultaneously. One of the stimuli changed to a green "T" at 200, 800, 1400, or 2000 msec after the start of the bilateral stimulus presentation and remained green for 190 msec with another 10 msec blank ISI immediately following. For catch trials, no change occurred during this 200 msec period of time. Immediately following this change (or no change) and ISI, the stimulus returned to the flashing bilateral red "T" presentation. The patient again had 2500 msec to respond following the onset of the stimulus change. Subjects were instructed to keep their eyes on the central fixation mark and indicate the location of the change (left, right, no change) by responding with their ipsilesional hand (right hand for patients). Approximately 33% of the trials were catch trials where no stimulus appeared. Stimuli were presented randomly in 5 blocks of 56 trials with approximately 100 trials for each hemispace, excluding catch trials. If an individual performed at less than chance on the no-stimulus catch trials, their data was to be thrown out. This did not occur. Total number of accurate monitoring responses for each of the four stimuli onsets (200, 800, 1400, and 2000 msec) was calculated for each hemispace, for each subject.

4. RESULTS

4.1 Demographics

Demographic information for the patient and control groups can be found in Tables 1 and 2. In regards to ethnicity, the control group was 34% non-Caucasian, the frontal group 33% non-Caucasian, and the parietal group 43% non-Caucasian. Regarding gender composition of the groups, the control group was 34 % male (10 male, 19 female), the frontal group 33 % male (3 male, 6 female), and the parietal group 57 % male (4 male, 3 female). It should be noted that of the patient sub-groups, the right frontal group was entirely female and the left parietal group entirely male. To examine possible effects of gender, ANOVAs were utilized. Please refer to section 4.4.4 Gender Differences for ANOVA results.

The dependent measures, detection performance and monitoring performance, were analyzed separately. Analysis was with three groups (control, frontal, parietal), rather than five (control, left frontal, right frontal, left parietal, right parietal) because the research questions did not specifically aim to compare left and right brain damaged individuals. Rather, the inclusion of both left and right brain damaged patients was to increase generalizability of the research findings

A 3-way ANOVA revealed no significant differences between controls and patients for age (F (2, 42) = .749, p = .479), handedness on the hand preference questionnaire (F (2, 42) = 2.699, p = .079), or errors on the Ishihara Plates (F (2, 42) = 1.905, p = .161). However, there was a significant difference with education, F (2, 42) = 11.104, p = .002. Multiple post hoc comparisons using Bonferroni's correction revealed the Control group had significantly more years of education than the Parietal group, with

an average of 15.9 years for the Controls and 12.4 for the Parietals, $t = 3.47$, $p = .019$. (Pearson correlation coefficients revealed no significant relationship at the .05 level between education and left hemispace or right hemispace accuracy, for either the Detect or Monitoring Tasks.) There was also a significant difference for BIT scores, $F(2, 42) = 3.979$, $p = .026$, with Controls (average BIT score: 143) performing better than Parietal patients (average BIT score: 139), $t = 3.71$, $p = .022$. For subject performance on screening and descriptive measures refer to Table 3.

Because some analyses utilized solely patient data, a second ANOVA was run to more directly contrast the two patient groups. For the ANOVA run on the demographic data with control subjects excluded, there were no differences found for age, education, handedness, or errors on the Ishihara Plates. There was a difference, at the .057 level, between Frontal and Parietal patients on the BIT. Frontals performed better than Parietals ($F(1, 14) = 4.448$, $p = .053$), with an average score of 142 for Frontals, and an average score of 139 for Parietals. A post hoc ANOVA on right brain damage verses left brain damage demographics revealed no significant differences between the groups.

4.2 Detection Task

On the Detection Task, to determine whether controls had equivalent performance in both visual fields and whether there was a performance difference over time for the patient groups, but no performance difference over time for the age-matched control group, a 3 (group: frontal, parietal, control) x 2 (hemispace: left vs. right) x 4 (time: 200, 800, 1400, and 2000 msec stimulus onset) repeated measures ANOVA was conducted. Due to the lack of a lesion in the control group, the performance data for this analysis was coded left visual field and right visual field performance, rather than ipsilesional and

contralesional performance. There were no significant differences for the three-way interaction or any of the two-way interactions. The main effects for time and hemispace were also not significant. There was a main effect for group, F (2, 42)= 4.159, p = .022. For ANOVA results, refer to Table 5. Bonferroni corrected pairwise comparisons revealed that the Parietal group had lower accuracy performance than the Control group, t = 8.615, p = .020. See Table 4 and Figure 3 for group accuracy performance on the Detection Task. It is worth mentioning that the Parietal group also performed worse than the Frontal group, but this did not reach significance (p = .09). The larger variance in the two patient groups, relative to the control group, may have decreased the ability to detect a significant difference if there was one. For hemispace performance by subgroup, see Figure 4.

To specifically address whether patients had worse performance in contralesional space, relative to ipsilesional space, a 2 (lesion area: frontal vs. parietal) x 2 (performance: ipsilesional vs. contralesional) x 4 (time: 200, 800, 1400, and 2000 msec stimulus onset) repeated measures ANOVA was conducted. For this analysis, the controls were excluded and the performance data was recoded as ipsilesional and contralesional. The three-way interaction for time x performance x lesion area was not significant, F (3,12) = 3.133, p = .066, but there was a trend suggesting that the factors differentially effect each other. (See Figure 5.) The two-way interactions, for time x lesion area and time x performance (ipsilesional/ contralesional), were not significant. The main effect for time was also not significant. The interaction of performance (ipsilesional/contralesional) and lesion area (Frontal/ Parietal) was significant, F (1, 14)= 4.521, p = .052, indicating that ipsilesional and contralesional performance are

differentially effected, depending on lesion area. (See Figure 6.) The main effect for performance was significant at the .060 level, F (1, 14) = 4.196, p = .060. There was a hint of a group (lesion area) main effect, but it was not significant, (F (1, 14) = 3.571, p = .080). Essentially, ipsilesional performance was better than contralesional performance, but only for the Parietal group. (Frontal: t (8) = -.05, p = .961; Parietal: t (6) = 2.655, p = .038) See Table 6 for ANOVA results.

4.3 Monitoring Task

For the Monitoring task, identical to the analysis for the Detection task, a 3 (group: frontal, parietal, control) x 2 (hemispace: left vs. right) x 4 (time: 200, 800, 1400, and 2000 msec stimulus onset) repeated measures ANOVA was conducted. There were no significant differences for the three-way interaction or any of the two-way interactions. The main effect for time was not significant. The main effects for hemispace, F (1, 42) = = 57.898, $p \leq .000$, and group, F (2, 42) = 4.748, p = .014, were significant. For hemispace, left performance was significantly better than right performance, regardless of group. (Control: t (28) = 5.893, $p \leq .000$; Frontal: t (8) = 3.614, p = .007; Parietal: t (6) = 5.114, p = .002) Regarding the group main effect, Bonferroni corrected multiple comparisons reveal the Parietal group was significantly worse than the Control group, t = 20.489, p = .011. Interestingly, the Frontals' performance appears to be equivalent to that of Controls. See Figures 7 and 8 for performance on the Monitoring Task and refer to Table 5 for ANOVA results.

To address whether patients had worse performance in contralesional space, relative to ipsilesional space, a 2 (lesion area: frontal vs. parietal) x 2 (performance: ipsilesional vs. contralesional) x 4 (time: 200, 800, 1400, and 2000 msec stimulus onset)

repeated measures ANOVA was run. This analysis was also used to determine whether frontal patients had worse performance than parietal patients over a temporal epoch. For this analysis, the controls were again excluded and the performance data was recoded as ipsilesional and contralesional. The three-way interaction was not significant. None of the two-way interactions were significant. Finally, none of the main effects were significant. Although, there was a trend observed at the .068 level for the main effect of group, suggesting Frontals had better performance than Parietals. (F (1, 14) = 3.896, p = .068) See Figure 9 for Frontal and Parietal ipsilesional and contralesional performance and refer to Table 6 for ANOVA results.

4.4 Post Hoc Analyses

4.4.1 Detect Verses Monitor

Because of the differences in accuracy performance observed on the two experimental tasks, the Detection and Monitoring Tasks were contrasted to each other to determine whether the difference was statistically significant. The Detection and Monitor tasks were compared utilizing a 2 (task: Detect vs. Monitor) x 2 (hemispace: left vs. right) ANOVA. The interaction of hemispace and task was significant, F (1,44) = 59.924, $p \leq .000$. The main effects of hemispace, F (1,44) =67.248, $p \leq .000$, and task, F (1,44) = 85.415, $p \leq .000$, were each significant. Essentially, performance is better in left hemispace, but only for the Monitor Task. See Figure 10 for all participants' performance on the Detect and Monitor tasks.

4.4.2 Left Verses Right

To contrast right brain damaged to left brain damaged patients, a 2 (lesion side: left vs. right) x 2 (performance: ipsilesional vs. contralesional) x 4 (time: 200, 800, 1400,

and 2000 msec stimulus onset) repeated measures ANOVA was run for both the Detection and Monitor tasks. No significant differences were found for the Detection task. The only significant difference found for the Monitor task was for the performance x lesion side interaction, $\underline{F}$ (1,14) = 32.692, $\underline{p} \leq .000$. For left brain damaged individuals, ipsilesional performance (performance in left hemispace) was greater than contralesional performance. For right brain damaged individuals, contralesional performance (performance in left hemispace) was greater than ipsilesional performance. Essentially, performance was better for left hemispace, as found in previous analyses. See Figure 8.

4.4.3 Single Case Analysis

In order to determine whether there were any apparent patterns within the data in regards to lesion location and specific task deficits, the patient data was examined separately by single case analysis. Each patient was compared to the control group. (e.g. Mycroft, Mitchell, & Kay, 2002) A 2 (group: individual patient vs. control group) x 4 (time: 200, 800, 1400, and 2000 msec stimulus onset) repeated measures ANOVA was utilized to derive an F value for each patient for each of the two tasks for left and right hemispace. There was no obvious relationship between brain lesion location and experimental task behavior for either the Detect or Monitor tasks. See Single Case Analysis Table 7 for a complete list of $\underline{p}$ values. See Table 8 for patient lesion and history information.

One left frontal patient (#1) had significantly different performance from the control group for stimuli presented in right hemispace in the Detection task, $\underline{F}$ (1, 28) = 4.515, $\underline{p} = .043$, and significantly different performance in left hemispace on the Monitor task, $\underline{F}$ (1, 28) = 21.32, $\underline{p} \leq .000$. Three different patients, a right frontal, a left parietal,

and a right parietal, were different from the control group for Detection stimuli presented in both left and right hemispace. (Right Frontal (#8): Left Hemispace: F (1, 28) = 3.867, p = .059, Right Hemispace: F (1, 28) = 6.816, p = .014; Left Parietal (#10): Left Hemispace: F = 6.599, p = .016, Right Hemispace: F (1, 28) = 22.26, p ≤ .000; Right Parietal (#16): Left Hemispace: F (1, 28) = 11.05, p = .002, Right Hemispace: F (1, 28) = 7.696, p = .010) The aforementioned right parietal patient (#16) was also significantly different (F (1, 28) = 12.18, p = .002) from the age-matched control group for stimuli presented in left hemispace for the Monitor task. Finally, another right parietal patient (#13) was also significantly different (F (1, 28) = 9.652, p = .004) from the control group for stimuli presented in left hemispace. No obvious patterns between task and lesion were observed.

4.4.4 Gender Differences

In order to determine whether there was an effect of gender on accuracy performance, oneway ANOVAs were utilized. On the Detection Task, there were no significant differences for gender for either left (F (1, 43) = 1.137, p = .292) or right hemispace (F (1,43) = .406, p = .528) performance accuracy. On the Monitoring task, there was also no significant effect for gender for right (F (1, 43) = .457, p = .503) hemispace performance. However, there was a significant gender effect for left hemispace performance, F (1, 43) = 7.147, p = .011, on the Monitoring Task. Females had significantly worse accuracy performance scores at stimuli onset time points 8, 14, and 20 seconds, but not at 2 seconds. (Examining groups separately, Control females had significantly worse performance than Control males at 2 and 20 seconds; Frontal females had significantly worse performance than Frontal males at 14 and 20 seconds; Parietal

males and females did not perform differently across the time points for left hemispace on the Monitoring Task.)

A 3 (group: frontal, parietal, control) x 2 (hemispace: left vs. right) x 4 (time: 200, 800, 1400, and 2000 msec stimulus onset) repeated measures ANOVA was repeated for the Monitoring Task, but with gender as a covariate. Main effects for group ($p = .004$) and hemispace ($p = .000$) were again observed. Main effects for time ($p = .016$) and gender ($p = .031$) were also observed, however the interaction for time and gender was also significant ($p = .049$). As noted in the previous paragraph, with all groups combined, males performed significantly better than females at 8, 14, and 20 second stimuli onsets in left hemispace. No group differences were observed for right hemispace Monitoring Task performance across time.

Oneway ANOVAs were also utilized to evaluate whether there was a significant influence of gender on ipsilesional and contralesional performance for patient data. On the Detection Task there were no significant differences of gender for either ipsilesional ($F (1, 14) = .739$, $p = .404$) or contralesional ($F (1, 14) = .058$, $p = .814$) performance accuracy. On the Monitoring task, there was no significant gender difference for contralesional ($F (1, 14) = .111$, $p = .744$) performance accuracy, and a trend was suggested for ipsilesional ($F (1, 14) = 4.30$, $p = .057$) performance, with males performing more accurately than females.

4.4.5 Reaction Time Analyses

To determine whether there were any reaction time differences, the performance accuracy repeated measures ANOVAs were replicated with reaction time performance. Please refer to Tables 9, 10, and 11 for participant mean reaction time performance and

ANOVA results. It should be noted that subjects were not instructed to respond quickly, only accurately. Thus, findings from this post hoc analysis should be viewed with some skepticism.

The 3 (group) x 2 (hemispace performance) x 4 (time) repeated measures ANOVA performed on the Detection Task revealed a main effect for group, F (2, 42) = 4.779, p = .013. Parietal (mean: 783 msec) reaction times were slower than Control (mean: 591 msec) reaction times. A main effect for time was also observed, F (3, 4) = 3.641, p = .021. Bonferroni corrected pairwise comparisons reveal reaction time for 800 msec stimuli onset (mean: 696 msec) was slower than reaction for either the 1400 msec stimuli onset (mean: 676 msec; p = .043) or the 2000 msec stimuli onset (mean: 669 msec, p = .021). There was not a main effect for group. No interactions were significant.

The 3 (group) x 2 (hemispace performance) x 4 (time) repeated measures ANOVA performed on the Monitoring Task revealed a main effect for group, F (2, 42) = 6.909, p = .003. Parietal (mean: 909 msec) reaction times were slower than Control (mean: 691 msec) reaction times. No other significant differences were observed.

The 2 (group) x 2 (ipsilesional/ contralesional performance) x 4 (time) repeated measures ANOVA performed on patient reaction time data of the Detection Task revealed a main effect for ipsilesional/ contralesional performance, F (1, 14) = 9.899, p = .007. Response time for Contralesional stimuli (768 msec) was slower than that of Ipsilesional stimuli (691 msec). No other significant differences were observed.

The 2 (group) x 2 (ipsilesional/ contralesional performance) x 4 (time) repeated measures ANOVA performed on patient reaction time data of the Monitoring Task also

revealed a main effect for ipsilesional/ contralesional performance, F (1, 14) = 10.714, p = .006. Mean reaction time for Contralesional stimuli (888 msec) was slower than that of Ipsilesional stimuli (810 msec). No other significant differences were observed.

5. DISCUSSION

In the Detection Task, controls did not show a hemispheric difference (left visual field verses right visual field) or a performance difference over time, as was predicted. However, the patients did not show an accuracy performance difference over time either. Unexpectedly, reaction time analyses revealed response time for Detection stimuli presented at 8 sec onset was slower than response time at 14 sec or 20 sec onset. Parietal lesion subjects, but not Frontal lesion subjects, had worse accuracy performance for contralesional space. Reaction time analyses reveal both Frontal and Parietal subjects are slower for contralesional space. Parietal lesioned patients performed significantly worse than Controls in terms of accuracy and reaction time performance.

For the Monitoring Task, Controls did not show an accuracy or reaction time performance difference over time, nor did the Frontal or Parietal patients. Surprisingly, a hemispheric difference in accuracy performance was observed for all study subjects. Accuracy performance for right hemispace was significantly worse than performance for left hemispace for controls and patient groups. Frontal and Parietal patients did not have worse accuracy performance in contralesional space, but did have worse (slower) reaction time performance for contralesional space. Given the observed left hemispace advantage in accuracy performance, it is likely that any accuracy differences between ipsilesional and contralesional space would be diminished. Although there were no specific predictions, Parietal lesioned patients again performed significantly worse (reaction time and accuracy) overall than Controls.

It was expected that Frontal lesion subjects would have worse performance than their Parietal counterparts on the Monitoring Task, with worse performance over the

temporal epoch. However, a trend suggesting better accuracy performance for Frontal patients was found. There was no difference over time.

Several post hoc analyses were run to further explore the data. From contrasting the Detection and Monitoring Tasks to each other it was determined that Monitoring accuracy performance is better in left hemispace, as noted earlier. Single case analysis, comparing patients individually to the control group, did not demonstrate clear links between brain lesion location and behavior deficit (accuracy) on the experimental tasks.

While not explicitly stated, it was suggested that decrements found in accuracy performance would be more pronounced for right brain damaged subjects because behavioral manifestations of decrement are more prevalent in right, than left, brain damaged individuals (i.e. left neglect is much more prominent than right neglect), immediately following brain injury. A post hoc ANOVA excluding control data was used to compare right to left brain damaged patients for both the Detection and Monitor tasks. Performance accuracy differences between right and left brain damaged subjects were not found for either of the two tasks. Lesion side differences were also not observed with reaction time data for either of the two tasks. This may be due to experimental testing occurring more than six months post injury. As noted earlier, a significant interaction in the Monitoring task indicated that accuracy performance was better in left hemispace, regardless of side (right or left) of lesion.

5.1 Alternate Considerations

The current study is one of the first to evaluate attention and sustained attention of frontal and parietal patients over a temporal epoch. It was surprising that there were no accuracy differences over time for either the frontal or the parietal group for either of the

two attention tasks. It was also surprising that reaction time performance analysis of the Detection Task found worse (slower) performance for an earlier time point, 8 sec, relative to later time points, 14 and 20 secs. Perhaps the time points for the stimuli onsets should be further evaluated. One consideration is that the 10 sec breaks between each individual trial might not have been long enough. Although a regular break preceded each new trial, a full trial of 2, 8, 14, and 20 sec possible stimuli onsets preceded by a short break might be experienced as longer onsets than intended. For example, a subsequent 2 sec onset might be experienced as a 32 sec (2 sec: new onset + 20 sec: previous trial + 10 sec: break) onset, an 8 sec onset like a 38 sec onset, etc. It is also possible that the expected time differences in accuracy performance for these novel tasks are so subtle, they would only be detected if testing occurred closer to the date of injury. A decrease in performance over time might be evident in patients that were tested less than six months post injury. Additionally, reaction times may be more sensitive than percentage of correct trials to detect performance differences across time. If subjects were instructed to respond quickly, as well as accurately, future studies might evaluate reaction times.

Another surprising finding was the lack of decrement in patient accuracy performance for contralesional space. In the Monitoring task, the observed hemispace effect in accuracy performance may have masked any contralesional differences. However, in the Detection Task, while Parietals did show a performance accuracy decrease in contralesional space, Frontal patients did not show this difference. It is possible that the performance accuracy decrease observed for Parietal subjects is linked to the subtle, but significant, decrease in performance on the paper and pencil attention task, the BIT. When contrasted directly, the Parietal group had significantly lower BIT

scores than the Frontal group. Of note, worse contralesional performance was observed with the post hoc reaction time analysis. Both Frontals and Parietals had slower reaction times for contralesional space.

Parietals had worse accuracy and reaction time performance than the Control group for both the Detection and Monitor tasks. The decrease in performance on the experimental detection and monitoring tasks, in conjunction with the decrease in performance on the BIT, demonstrate that attention is impaired in Parietal lesion patients at six months or more post injury. Additionally, the decrease in contralesional reaction time for both the Frontal and Parietal subjects further reiterates that subtle deficits may persist following injury.

In general, the lesioned subjects performed quite well. Patient accuracy performance, particularly the Frontal group, was quite similar to that of the Control subjects. Additionally, the lesioned subjects of this study appear to have relatively better performance than the pilot subjects mentioned earlier in this text. It is possible that the average lesion size for the patient subjects of this study is smaller than the average lesion size for the pilot subjects. A volumetric analysis might provide some insight regarding extent of lesion. As well, there may be differences in severity of illness.

A trend (at the .068 level) suggests that frontal lesion subjects differ from their parietal counterparts on the Monitoring task, but in the opposite direction than was predicted. Frontal lesion individuals tended to have better overall accuracy performance than parietal patients. (See Figures 7 and 9.) This may be a reflection of the significant difference in BIT scores. When directly contrasted to each other, the parietals had significantly poorer BIT scores. Regardless, observation of the frontal patient

performance in the context of the control subject performance suggests these two groups perform equally well. (See Figure 7 for the Monitoring Task and Figure 3 for similar performance on the Detection task.) This equivalent performance suggests the trend of worse parietal performance, relative to frontal performance, may reflect the parietal difference from the controls, and not the frontals per se.

The most unexpected result from the study was the a main effect for hemispace on the Monitoring Task for all subjects, regardless of lesion or lack of a lesion, in which accuracy performance for left hemispace was significantly better than right hemispace. Inconsistent with expectations, both left brain damaged and right brain damaged subjects had better accuracy performance for left hemispace on this sustained attention task. (See Figure 8.)

It is possible that the Monitoring task was so taxing to the visual system that a strategy was utilized to maximize performance. An individual might selectively attend to one visual field to increase the number of correct responses, rather than trying to attend to both visual fields simultaneously. To counteract this possible strategy, subjects were asked to fixate on the central fixation point and their eyes were monitored during practice trials. However, it is unclear whether they continued to fixate on the fixation point during actual testing since their eye gaze was not monitored during actual testing. Future studies might consider shorter testing durations and the use of an eye-tracking device. If the strategy of attending to one visual field was adopted by subjects, it is still not clear why all participants would selectively attend to left visual field.

Another possible explanation for poorer right visual field performance for all subjects on the Monitoring task is that this is an effect of aging. It is possible that as one

ages, sustained attention tasks increase in difficulty. As seen in the experimental data of this study, the age- matched controls also were significantly worse for right visual field performance. Age-related declines in sustained attention have been substantiated. However, it should be noted that in these studies, differences were found in individuals over the age of 65 (Parasuraman, Nestor, & Greenwood, 1989) and 70 (Filley & Cullum, 1994). For the current study, the average participant age was 61. A future study might compare younger controls to older subjects on the Monitoring task.

Whether the decrease in monitoring is due to an adopted strategy or an effect of aging, it is still unclear why performance was better for left rather than right visual space. For auditory tasks, performance is generally better for the right ear. (e.g. Saetrevik & Hugdahl, 2007) In the dichotic listening literature there is the well-known right ear advantage for verbal stimuli, indicating a left hemisphere processing preference. (e.g. Hiscock, Cole, Benthall, Carlson, & Ricketts, 2000) A left hemisphere temporal-processing advantage is also suggested in the tactile literature. (Nicholls & Lindell, 2000) In regards to the visual system, literature also supports a left-hemisphere advantage. Deason and Marsolek (2005) presented stimuli on a monitor controlled by a Apple Power Macintosh, and had subjects place their heads in a chin rest to ensure a 50 cm eye distance from the monitor. They found subjects had a strong left-hemisphere advantage when observing words written in uppercase and lowercase words, although a weaker effect was found for "AlTeRnAtInG-cAsE" words. Of interest, they reported a reversal of the left-hemisphere advantage, with a numerical trend suggesting a right-hemisphere advantage, for words presented in an unfamiliar word format (visual prototype font). Another group of researchers have also suggested a general right-

hemisphere specialization for attentional processing. Hollander, Corballis, and Hamm (2005) found a left-visual-field advantage while investigating attentional blink (AB) in a neurologically intact population. In regards to the observed asymmetry, they noted that the right-hemispheric specialization for spatial attention might override any of the left-hemispheric advantage for temporal processing. (Hollander, Corballis, & Hamm, 2005) Similar to the Monitoring task of this study, a bilateral presentation of stimuli was used and the result was found in neurologically intact participants.

It is possible that better left performance on the experimental task is related to learned left-to-right reading. Learned visual scanning from left-to-right might lead people to be more attentive to/ aware of, the left side of their visual field for word or letter stimuli. It might lead individuals to automatically visually orient to the left side when initially processing visual input. The target stimulus for the experimental tasks was the letter 'T'. Although these tasks did not require reading, the letter stimulus itself may trigger a learned reading behavior of initially looking left. Essentially, what might occur is information processing modulation by the 'letterness' of the target stimuli, in a top-down fashion, such that learned left-to-right scanning is automated. To investigate this further, one might use a geometric shape for a stimulus object, rather than a letter, in a future study. Also, as previously suggested, one could utilize a visual eye-tracking system to monitor eye gaze.

5.2 Challenges and Limitations

One limitation of the study was the significant differences observed in the demographic data. The BIT scores and education level may explain the experimental performance differences observed for the parietal subjects, relative to the control

subjects. Scores on a behavioral inattention task might inherently be correlated with the data. A participant's intellectual ability, as indicated by education level, might influence perceptual ability, or vice versa. (Although, Pearson correlations did not reveal a significant relationship between education and accuracy performance.) Also, gender differences were observed for left hemispace and ipsilesional accuracy performance on the Monitoring task. Demographic differences are unavoidable due to the limited availability of specific brain lesion patients. With small patient groups, any differences in demographic data can become more pronounced.

Another limitation of the study was the low power observed. A larger sample size of patients might yield different results. To increase the patient yield of suitable database subjects, future studies might consider utilizing frontal-parietal lesioned individuals in addition to the discreet frontal and parietal lesioned subjects. Single case analysis in conjunction with brain mapping analysis could be utilized to evaluate behavioral performance that differed from the control group.

Another criticism of the study may be that none of the patients had neglect. For this brain-behavior study, inclusion was based on lesion location, not neglect behavior. The BIT was utilized in this study for descriptive purposes. All patients in this study scored above the neglect cutoff (130/146) on the BIT. Future behavioral studies might consider inclusion criteria that establish neglect syndrome for lesion subjects.

While beyond the means of the current study, evaluation of possible confounds such as a precise measure of visual acuity, might be informative. While not administered to every participant, a "Visual Acuity: MIS Pocket Vision" measure was utilized in an attempt to ensure a minimal level of visual acuity, at least 20/50 for each eye. A more

extensive measure of acuity might prove beneficial in future studies. Other factors that might influence the results include presence of cataracts, presence of astigmatism, and amount of time between lesion onset and testing. Future studies evaluating sustained attention might consider these variables.

Another limitation of the study is the proscribed testing situation, which may decrease the application of the results to a real world setting. Participants were performing a very specific task in an artificial setting. Thus it is a small stretch to transfer the findings to specific real world tasks such as driving. A future study might be to devise a task that involves detecting stimuli in a simulated driving experience.

Another factor that could influence the results was the use of different testing locations. Many patient subjects were tested in their homes (often in living or dining rooms), rather than at the University of Pennsylvania. Also, many control subjects were tested in a testing room created in the examiner's home or in the home of the mother of the examiner. The location of windows and various other light sources, as well as various sounds (roadwork being performed near a participant's home during home testing), may have inadvertently diverted the subjects' attention during the experimental tasks despite attempts to minimize these effects. However, the different testing environments may also prove to be an asset to the study, since it makes the significant findings more robust.

5.3 Clinical Implications

The findings of this study provide new information suggesting a left-visual-field advantage for the visual system during change detection (monitoring). This is at odds with the general literature, but is supported by the attentional blink study of Hollander,

Corballis, and Hamm (2005) as well as by the right-hemisphere trend observed in Deason and Marsolek's (2005) study involving visual prototype font. If future studies confirm the finding of a right hemisphere processing advantage for detecting changes, this information might be useful for designing improvements in real-world detection tasks. For example, with airport baggage screening, one might ensure that the visual monitor presentation of items is from left to right to increase detection of contraband items. Other potential applications might include improving the design of controls and/or screen monitors of airplane cockpits, car dashboards, or security systems involving the monitoring of several visual displays simultaneously. By adding features that divert eye gaze rightward, right visual field change detection might increase and strain from fatigue or stress might decrease.

If the finding of decreased attention and decreased monitoring ability for parietal patients more than six months post injury were replicated, application of the study results might be to direct rehabilitation efforts. Although a parietal patient may perform quite well on an overt attention task, training and subsequent evaluation on subtle detection and monitoring tasks might prove beneficial prior to returning to certain everyday tasks. Specific rehabilitative efforts might help a parietal lesioned patient detect a car that 'suddenly' appears in right visual field while they are driving or crossing a crosswalk.

In a similar vein, the reaction time decrease observed for detecting and monitoring contralesional stimuli found for both Frontal and Parietal lesioned individuals reiterates the subtle differences that persist more than 6 months post injury. Additional retraining focused on attention in contralesional space may improve participation in activities of daily living as well as general life satisfaction.

LIST OF REFERENCES

Albert, M.L. (1973). A simple test of visual neglect. *Neurology, 23*, 658-664.

Annett, M. (1967). The binomial distribution of right, mixed and left handedness. *Quarterly Journal of Experimental Psychology, Nov: 19(4):* 327-333.

Arguin, M., Cavanagh, P., & Joanette, Y. (1994). Visual feature integration with an attention deficit. *Brain and Cognition, 24*, 44-56.

Babinski, J. (1914). Contribution a l'etude des troubles mentaux dans l'hemiplegie organique cerebrale (anosognosie). *Revue Neurologique; 27*: 845-847.

Baylis, G.C., Gore, C.L., Rodriguez, P.D., & Shisler, R.J. (2001). Visual extinction and awareness: The importance of binding dorsal and ventral pathways. *Visual Cognition, 8*, 359-379.

Beck, D.M., Rees, G., Frith, C.D., & Lavie, N. (2001). Neural correlates of change detection and change blindness. *Nature Neuroscience, 4(6),* 645-650.

Bender, M.B. (1952). *Disorders in perception.* Springfield, IL: Charles C. Thomas.

Bender, M.B. & Furlow, C.T. (1945). Phenomenon of visual extinction and homonymous field and psychological principles involved. *Archives of Neurology and Psychiatry, 53*, 29-33.

Beschin, N. & Robertson, I.H. (1997). Personal versus extrapersonal neglect: a group study of their dissociation using a reliable clinical test. *Cortex, 33*, 379-384.

Birch, J. (1997). Efficiency of the Ishihara test for identifying red-green colour deficiency. *Opthal Physiol Opt*, *17(5)*: 403-408.

Birch, J. & McKeever, L. M. (1993). Survey of the accuracy of new pseudoisochromatic plates. *Opthal Physiol Opt, 13(1)*: 35-40.

Bisiach, E. (1993). Mental representation in unilateral neglect and related disorders: The twentieth Bartlett Memorial lecture. *Quarterly Journal of Experimental Psychology, A, 46(3)*, 435-461.

Bisiach, E., Capitani, E., Luzzatti, & Perani, d. (1981). Brain and conscious representation of outside reality. *Neuropsychologia, 19*, 543-551.

Bisiach, E., Capitani, E. & Porta, E. (1985). Two basic properties of space representation in the brain: Evidence from unilateral neglect. *Journal of Neurology, Neurosurgery and Psychiatry, 48*, 141-144.

Bisiach, E., Geminiani, G., Berti, A., & Rusconi, M.L. (1990). Perceptual and premotor factors of unilateral neglect. *Neurology, 40*, 1278-1281.

Bisiach, E., & Luzzatti, C. (1978). Unilateral neglect of representational space. *Cortex, 14*, 129-133.

Bisiach, E., Luzzatti, C., & Perani, D. (1979). Unilateral neglect, representational schema and consciousness. *Brain, 102*, 609-618.

Briand, K.A., & Klein, R.M. (1987). Is Posner's "beam" the same as Treisman's "glue"?: On the relation between visual orienting and feature integration theory. *Journal of Experimental Psychology, Human Perception and Performance, 13(2),* 228-241.

Briggs, G. G. & Nebes, R. D. (1975). Patterns of hand preference in a student population. *Cortex, Sep: 11(3):* 230-238.

Burcham, K.J., Corwin, J.V., Stoll, M.L., & Reep, R.L. (1997). Disconnection of medial agranular and posterior parietal cortex produces multimodal neglect in rats. *Behavioral Brain Research, 90*, 187-197.

Bunce, D. (2001). Age differences in vigilance as a function of health-related physical fitness and task demands. *Neuropsychologia, 39(8)*, 787-797.

Chatterjee, A. (2002). Neglect. In M. D'Esposito (Ed.), *Neurological Foundations of Cognitive Neuroscience*. The MIT Press: Cambridge, MA.

Chatterjee, A. (1998). Motor minds and mental models in neglect. *Brain and Cognition, 37*, 339-349.

Chatterjee, A. & Coslett, H.B. (2003). Neglect: Cognitive Neuropsychological Aspects. In T. Feinberg & M.J. Farah (Eds.) *Behavioral Neurology and Neuropsychology*. Oxford University Press.

Chatterjee, A. & Mennemeier, M. (1998). Diagnosis and treatment of spatial neglect. In R.B. Lazar (Ed.), *Principles of Neurologic Rehabilitation*. McGraw-Hill: Chicago, Illinois.

Chatterjee, A. & Thompson, K.A. (1998). Weigh(t)ing for awareness. *Brain and Cognition, 37*, 477-490.

Chatterjee, A., Thompson, K.A., & Ricci, R. (1999). Quantitative analysis of cancellation tasks in neglect. *Cortex, 35*, 253-262.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112(1),* 155-159.

Cohen, J.D., MacWhinney, B., Flatt, M., & Provost, J. (1993). PsyScope: A new graphic interactive environment for designing psychology experiments. *Behavioral Research Methods, Instruments, and Computers, 25(2),* 257-271.

Corbetta, M. (1998). Frontoparietal cortical networks for directing attention and the eye to visual locations: identical, independent, or overlapping neural system. *Proceedings of the National Academy of Sciences, 95*, 831-838.

Corbetta, M., Meizen, F.M., Shulman, G.L., & Peterson, S.E. (1993). A PET study of visuospatial attention. *Journal of Neuroscience, 11*, 1202-1226.

Coslett, H.B., Bowers, D., Fitzpatrick, E., Haws, B., & Heilman, K.M. (1990). Directional hypokinesia and hemispatial inattention in neglect. *Brain, 113*, 475-486.

Deason, R. G. & Marsolek, C. J. (2005). A critical boundary to the left-hemisphere advantage in visual-word processing. *Brain and Language, 92(3),* 251-261.

Denny-Brown, D., Meyer, J.S., & Horenstein, S. (1952). The significance of perceptual rivalry resulting from parietal lesion. *Brain, 75*, 433-471.

Diller, L. & Riley, E. (1993). The behavioural management of neglect. In I.H. Robertson & J.C. Marshall (Eds.), *Unilateral Neglect: Clinical and Experimental Studies* (pp. 293-308). East Sussex: Lawrence Erlbaum Associates Ltd.

Downar, J., Crawley, D. P., Mikulis, D. J., & Davis, K. D. (2000). A multimodal cortical network for the detection of changes in the sensory environment. *Nature Neuroscience, 3(3)*, 277-283.

Driver, J., Mattingley, J. B., Rorden, C., & Davis, G. (1997). Extinction as a paradigm measure of attentional bias and restricted capacity following brain injury. In P. Their and H.-O Karnath (Eds.), *Parietal lobe contributions to orientation in 3D space*. Springer-Verlag, Heidelberg.

Duncan, J. (1980). The locus of interference in the perception of simultaneous stimuli. *Psychological Review, 87*, 272-300.

Duncan, J. & Humphreys, G. W. (1989). Visual search and stimulus similarity. *Psychological Review, 96*, 433-458.

Farah, M.J., Wallace, M.A., & Vecera, S.P. (1993). "What" and "where" in visual attention: Evidence from the neglect syndrome. In I.H. Robertson & J.C. Marshall (Eds.), *Unilateral Neglect: Clinical and Experimental Studies*. (pp. 123-137). Hillsdale. NJ: LEA.

Filley, C. (1995). *Neurobehavioral Anatomy*. University Press of Colorado: Niwot, Colorado.

Filley, C. M. & Cullum, C. M. (1994). Attention and vigilance functions in normal aging. *Applied Neuropsychology, 1(1&2)*, 29-32.

Friedman-Hill, S. R., Robertson, L. C., & Treisman, A. (1995). Parietal contributions to visual feature binding: Evidence from a patient with bilateral lesions. *Science, 269*, 853-855

Gainotti, G. (1993). The role of spontaneous eye movements in orienting attention and in unilateral neglect. In I.H. Robertson & J.C. Marshall (Eds*.), Unilateral Neglect: Clinical and Experimental Studies* (pp. 107-122). East Sussex: Lawrence Erlbaum Associates Ltd.

Gainotti, G., Messerli, P., & Tissot, R. (1972). Qualitative analysis of unilateral and spatial neglect in relation to laterality of cerebral lesions. *Journal of Neurology, Neurosurgery, and Psychiatry, 35*, 545-550.

Gehring, W. J. & Knight R. T. (2000). Prefrontal-cingulate interactions in action monitoring. *Nature Neuroscience, 3(5),* 516-520.

Gitelmen, D.R., Nobre, A.C., Parrish, T.B., LaBar, K.S., Kim, Y.H., Meyer, J.R., & Mesulam, M. (1999). A large-scale distributed network for covert spatial attention: Further anatomical delineation based on stringent behavioural and cognitive controls. *Brain, 122*, 1093-1106.

Georgopoulos, A.P. (2000). Neural aspects of cognitive motor control. *Current Opinions in Neurobiology, 10*, 238-241.

Goodrich, S. J., & Ward, R. (1997). Anti-extinction following unilateral parietal damage, *Cognitive Neuropsychology, 14(4),* 595-612.

Halligan, P.W., Cockburn, J., & Wilson, B.A. (1991). The behavioural assessment of visual neglect. *Neuropsychological Rehabilitation, 1*, 5-32.

Halligan, P. W. & Marshall, J. C. (1998a). Neglect of awareness. *Consciousness and Cognition, 7*, 356-380.

Halligan, P.W. & Marshall, J.C. (1998b). Visuo-spatial neglect: the ultimate deconstruction. *Brain and Cognition, 37(3)*, 419-438.

Halligan, P.W., Marshall, J.C., & Wade, D.T. (1989). Visualspatial neglect: underlying factors and test sensitivity. *Lancet, 14(2),* 908-911.

Halligan, P.W., Marshall, J.C., & Wade, D.T. (1990). Do visual field deficits exacerbate visuo-spatial neglect? *Journal of Neurology, Neurosurgery, and Psychiatry, 53(6)*, 487-491.

Halligan, P. W. & Robertson, I. H. (1992). The assessment of unilateral neglect. In J. R. Crawford, D. M. Parker, & W. W. McKinlay (Eds.), *A Handbook of Neuropsychological Assessment* (pp. 151-175). Hillsdale, NJ; Lawrence Erlbaum.

Hartman-Maeir, A., Soroker, N., Ring, H., Avni, N., & Katz, N. (2007). Activities, participation and satisfaction one-year post stroke. *Disability and Rehabilitation, 29(7)*, 559-566.

Heilman, K.M. (1979). Neglect and related disorders. In K.M. Heilman & E. Valenstein (Eds.), *Clinical Neuropsychology* (pp. 268-307). Oxford: Oxford University Press.

Heilman, K., Bowers, D., Coslett, H., Whelen, H., & Watson, R.T. (1985). Directional hypokinesia: prolonged reaction times for leftward movements in patients with right hemisphere lesions and neglect. *Neurology, 35*, 855-859.

Heilman, K.M., Bowers, D., Valenstein, E., & Watson, R.T. (1987). Hemispace and hemispatial neglect. In M. Jeannerod (Ed.), *Neurophysiological and neuropsychological aspects of spatial neglect* (pp. 115-150). Amsterdam: Elsevier.

Heilman, K.M., Pandya, D.N., & Geschwind, N. (1970). Trimodal inattention following parietal lobe ablations. *Transactions of the American Neurological Association, 95*, 259-261.

Heilman, K.M., Schwartz, H.D., & Watson, R.T. (1978). Hypoarousal in patients with the neglect syndrome and emotional indifference. *Neurology, 28*, 229-232.

Heilman, K.M. & Valenstein, E. (1972). Frontal lobe neglect in man. *Neurology, 22*, 660-664.

Heilman, K.M. & Valenstein, E. (1979). Mechanisms underlying hemispatial neglect. *Annals of Neurology, 5*, 166-170.

Heilman, K. & Van Den Abell, T. (1980). Right hemisphere dominance for attention: the mechanisms underlying hemispheric asymmetries of inattention (neglect). *Neurology, 30*, 327-330.

Heilman, K.M., Watson, R.T., & Valenstein, E. (1985). Neglect and related disorders. In K.M. Heilman & E. Valenstein (Eds.), *Clinical Neuropsychology*, (2nd ed., pp. 243-293). New York: Oxford University Press.

Heilman, K.M., Watson, R.T., & Valenstein, E. (1994). Localization of lesions in neglect and related disorders. In A. Kertesz (Ed*.), Localization and Neuroimaging in Neuropsychology* (pp.495-524). New York: Academic Press.

Heilman, K.M., Valenstein, E., & Watson, R.T. (2000). Neglect and related disorders. *Seminars in Neurology, 20(4),* 463-470.

Hier, D., Mondlock, J., Caplan, L. (1983). Recovery of behavioral abnormalities after right hemisphere stoke. *Neurology, 33*, 345-350.

Henley, S., Pettit, P., Todd-Pokropek, L., & Tupper, J. (1985). Who goes home? Predictive factors in stoke recovery. *Journal of Neurology, Neurosurgery, and Psychiatry, 48*, 1-6.

Hier, D.B., Davis, K.R., Richardson, E.P., & Mohr, J.P. (1977). Hypertensive putaminal hemorrhage. *Annals of Neurology, 1*, 152-159.

Hiscock, M., Cole, L. C., Benthall, J. G., Carlson, B. L., & Ricketts, J. M. (2000). Toward solving the inferential problem in laterality research: Effects of increased reliability on the validity of the dichotic listening right-ear advantage. *Journal of the International Neuropsychological Society, 6(5)*, 539-547.

Hodges, J.R. (1998). *Cognitive Assessment for Clinicians*. Oxford: Oxford University Press.

Hollander, A., Corballis, M. C., & Hamm, J. P. (2005). Visual-field asymmetry in dual-stream RSVP. *Neuropsychologia, 43*, 35-40.

Husain, M., & Kennard, C. (1996). Visual neglect associated with frontal lobe infarction. *Journal of Neurology, 243*, 652-657.

Kahneman, D. (1973). *Attention and Effort.* Prentice-Hall, Englewood Cliffs, NJ.

Kandel, E.R., Schwartz, J.H. & Jessell, T.M. (1995*). Essentials of Neural Science and Behavior*. Simon & Schuster: Stamford, Connecticut

Karnath, H.O. (1988). Deficits of attention in acute and recovered visual hemi-neglect. *Neuropsychologia, 26*, 27-43.

Katz, N. Hartman-Maeir, A., Ring, H. & Soroker, N. (1999). Functional disability and rehabilitation outcome in right hemisphere damaged patients with and without unilateral spatial neglect. *Archives of Physical and Medical Rehabilitation, 80(4*), 379-384.

Kim, Y.H., Gitelman, D.R., Nobre, A.C., Parrish, T.B., LaBar, K.S., & Mesulam, M.-M. (1999). The large-scale neural network for spatial attention displays multifunctional overlap but differential asymmetry. *Neuroimage, 9*, 269-277.

Kinsbourne, M. (1970). The cerebral basis of lateral asymmetries in attention. *Acta Psychologica, 33*, 193-201.

Kinsbourne, M. (1987). Mechanisms of unilateral neglect. In M. Jeannerod (Ed.), *Neurophysiological and Neuropsychological Aspects of Spatial Neglect*, (pp. 69-86). New York: North Holland.

Knight, R. T. (1984). Decreased response to novel stimuli after prefrontal lesions in man. *Electroencephalography and Clinical Neurophysiology, 59*, 9-20.

Knight, R.T., Scabini, D., Woods, D. L., & Clayworth, C. C. (1989). Contribution of temporal-parietal junction to the human auditory P3. *Brain Research, 502*, 109-116.

Leibovitch, F.S., Black, S.E., Caldwell, C.B., Ebert, P.L., Ehrlich, L.E., & Szalai, J.P. (1998). Brain-behavior correlations in hemispatial neglect using CT and SPECT. *Neurology, 50*, 901-908.

Lezak, M. (1995). *Neuropsychological Assessment*, 3rd ed., New York: Oxford University Press.

Mack, J.L. & Levine, R.N. (1981). The basis of visual constructional disability in patients with unilateral cerebral lesions. *Cortex, 17(4),* 515-531.

Manly, T. & Robertson, I.H. (1997). Sustained attention and the frontal lobes. In: P.M.A. Rabbitt, (Ed.), *Methodology of Frontal and Executive Function*. Psychology Press, Hove, UK.

Mesulam, M.-M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of Neurology, 10*, 725-727.

Mesulam, M.-M. (1990). Large-scale neurocognitive networks and distributed processing for attention, language and memory. *Annuals of Neurology, 28*, 597-613.

Milner, D. & Goodale, M. (1995). *The Visual Brain in Action*. Oxford University Press; Oxford.

Morrow, L.A. & Ratcliff, G. (1987). Attentional mechanisms in clinical neglect. *Journal of Clinical and Experimental Neuropsychology, 9*, 74-75.

Mountcastle, V.V., Lynch, J.C., Georgeopolous, A., Sakata, H., & Acuna, C. (1975). The influence of attentive fixation upon the excitability of the light sensitive neurons of the posterior parietal cortex. *Journal of Neuroscience, 1*, 1218-1245.

Mycroft, R. H., Mitchell, D. C., & Kay, J. (2002). An evaluation of statistical procedures for comparing an individual's performance with that of a group of controls. *Cognitive Neuropsychology, 19(4)*, 291-299.

Nicholls, M. D. & Lindell, A.K. (2000). A left hemisphere, but not right hemispace, advantage for tactual simultaneity judgments, *Perceptual Psychophysics, 62(4)*, 717-725.

Nobre, A.C. (2001a). The attentive homunculus: now you see it, now you don't. *Neuroscience and Biobehavioral Reviews, 25(6)*, 477-496.

Nobre, A.C. (2001b). Orienting attention to instants in time. *Neuropsychologia, 39(12),*1317-1328.

Nobre, A.C., Sebestyen, G.N., Gitelman, D.R., Mesulam, M.-M., Frackowiak, R.S., & Frith, C.D. (1997). Functional localization of the system for visuospatial attention using positron emission tomography. *Brain, 120*, 515-533.

Parasuraman, R. & Davies, D.R. (1977). A taxonomic analysis of vigilance performance. In R.R. Mackie (Ed.), *Vigilance: Theory, Operational Performance, and Physiological Correlates* (pp. 559-574). Plenum, New York.

Parasuraman, R., Nestor, P. G., & Greenwood, P. (1989). Sustained-attention capacity in young and older adults. *Psychology and Aging, 4*, 339-345.

Pashler, H. (1994). Dual-task interference in simple tasks: data and theory. *Psychology Bulletin, 116*, 220-244.

Posner, M.I., & Dehaene, S. (1994). Attentional networks. *Trends in Neurosciences, 17*, 75-79.

Posner, M., Walker, J., Friedrich, F., & Rafal, R. (1984). Effects of parietal injury on covert orienting of attention. *Journal of Neuroscience, 4*, 1863-1874.

Patterson, A. & Zangwill, O.L. (1944). Disorders of visual space perception associated with lesions of the right cerebral hemisphere. *Brain, 67*, 331-358.

Prinzmetal, W., Presti, D.E., & Posner, M.I. (1986) Does attention affect visual feature integration? *Journal of Experimental Psychology, Human Perception and Performance, 12(3),* 361-369.

Raymond, J.E., Shapiro, K.L. & Arnell, K.M. (1992). Temporary suppression of visual processing in an RSVP task: an attentional blink*? Journal of Experimental Psychology and Human Perception Performance, 18*, 849-860.

Rizzolatti, G., Matelli, M., & Pavesi, G. (1983). Deficits in attention and movement following the removal of postarcuate (area 6) and prearcuate (area 8) cortex in macaque monkeys. *Brain, 106*, 655-673.

Robertson, I.H., Manly, T., Andrade, J., Baddeley, B.T., & Yiend, J. (1997). 'Oops!': performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia, 35*, 747-758.

Rosen, A.C., Rao, S.M., Caffarra, P., Scaglioni, A., Bobholz, J.A., Woodley, S.J., Hammeke, T.A., Cunningham, J.M., Prieto, T.E., & Binder, J.R. (1999). Neural basis of endogenous and exogenous spatial orienting. A functional MRI study. *Journal of Cognitive Neuroscience, 11(2)*, 135-152.

Sandifer, P.H. (1946). Anosognosia and disorders of body scheme. *Brain, 69*, 122-137.

Saetrevik, B. & Hugdahl, K. (2007). Priming inhibits the right ear advantage in dichotic listening: Implications for auditory laterality. *Neuropsychologia, 45*, 282-287.

Schenkenberg, T., Bradford, D.C., & Ajax, E. T. (1980). Line bisection and unilateral visual neglect in patients with neurologic impairments. *Neurology, 30*, 509-517.

Stone, S.P., Halligan, P.W., Marshall, J.C., & Greenwood, R.J. (1998). Unilateral neglect: a common but heterogeneous syndrome. *Neurology, 50(6),* 1902-1905.

Stone, S.P., Patel, P., & Greenwood, R.J. (1993). Selection of acute stroke patients for treatments of visual neglect. Journal of Neurology, *Neurosurgery, and Psychiatry, 56*, 463-466.

Swick, D. & Knight, R. T. (1998). In R. Parasuraman (Ed.) *The Attentive Brain* (pp. 144-162). MIT Press, Cambridge, Massachusetts.

Tegner, R. & Levander, M. (1991). Through the looking glass: A new technique to demonstrate directional hypokinesia in unilateral neglect. *Brain, 114*, 1943-1951.

Treisman, A.M. (1969). Strategies and models of selective attention. *Psychological Review, 76*, 282-299.

Treisman, A. & Gelade, G. (1980). A feature-integration theory of attention. *Cognitive Psychology, 12(1),* 97-136.

Ungerleider, L.G. & Mishkin, M. (1982). Two cortical visual systems. In D.J. Ingle, M.A. Goodale, & R.J.W. Mansfield (Eds.), *Analysis of Visual Behavior*. Cambridge, MA: MIT Press.

Vallar, G. & Perani, D. (1987). The anatomy of spatial neglect in humans. In M. Jeannerod (Ed.), *Neurophysiological and Neuropsychological Aspect of Spatial Neglect*, (p. 235-258). Amsterdam: North-Holland.

Van Essen, D.C., Felleman, D.J., DeYoe, E.A., Ollavaria, J., & Knierman, J. (1990). Modular and hierarchical organization of extrastriate visual cortex in the macaque monkey. *Cold Spring Harbor Symposia on Quantitative Biology, 55*, 679-696.

Ward, R. Goodrich, S.J. & Driver, J. (1994). Grouping reduces visual extinction: Neuropsychological evidence for weight-linkage in visual selection. *Visual Cognition, 1*, 101-129.

Watson, R.T. & Heilman, K.M. (1979). Thalamic neglect. *Neurology, 29*, 290-294.

Watson, R.T., Heilman, K.M., Cauthern, J.C., & King, F.A. (1973). Neglect after cingulectomy. *Neurology, 23*, 1003-1007.

Watson, R.T., Valenstein, E., & Heilman, K.M. (1978). Nonsensory neglect. *Annals of Neurology, 3*, 505-508.

Watson, R. T., Valenstein, E., & Heilman, K.M. (1981). Thalamic neglect. *Archives of Neurology, 38*, 501-506.

Whyte, J., Polansky, M., Fleming, M., Coslett, H.B., & Cavallucci, C. (1995). Sustained arousal and attention after traumatic brain injury. *Neuropsychologia, 33(7)*, 797-813.

Weintraub, S., & Mesulam, M.-M. (1987). Right cerebral dominance in spatial attention: further evidence based on ipsilateral neglect. *Archives of Neurology, 44*, 621-625.

Wilson, B., Cockburn, J., & Halligan, P. (1987). *Behavioural Inattention Test.* Fareham, England: Thames Valley Test Company.

Yamaguchi, S. & Knight, R. T. (1991a). Anterior and posterior association cortex contribution to the somatosensory P300. *Journal of Neuroscience, 11*, 2039-2054.

Yamaguchi, S. & Knight, R. T. (1991b). P300 generation by novel somatosensory stimuli. *Electroencephalography and Clinical Neurophysiology, 78*, 50-55.

Zeki, S. & Shipp, S. (1988). The functional logic of cortical connections. *Nature, 335(6188)*, 311-317.

APPENDIX A: TABLES

Table 1: Participant Age and Education

	Age		Education	
	M	SD	M	SD
Control	61.7	11.4	15.9	2.9
Frontal	56.7	10.2	13.3	2.2
L Frontal	57.2	10.8	13.2	1.8
R Frontal	56.0	11.0	13.5	3.0
Parietal	62.6	13.1	12.4	3.3
L Parietal	69.3	8.5	12.7	2.9
R Parietal	57.4	14.7	12.3	4.0

Table 2: Participant Gender and Ethnicity

Gender	Male		Female	
	n	%	n	%
Control	10	34.5	19	65.5
Frontal	3	33.3	6	66.7
L Frontal	3	60.0	2	40.0
R Frontal	0	0.0	4	100.0
Parietal	4	57.1	3	42.9
L Parietal	3	100.0	0	0.0
R Parietal	1	25.0	3	75.0

Ethnicity	Caucasian		African American		Hispanic	
	n	%	n	%	n	%
Control	19	65.5	9	31.0	1	3.45
Frontal	6	66.7	3	33.3	0	0.0
L Frontal	4	80.0	1	20.0	0	0.0
R Frontal	2	50.0	2	50.0	0	0.0
Parietal	4	57.1	3	42.9	0	0.0
L Parietal	2	66.7	1	33.3	0	0.0
R Parietal	2	50.0	2	50.0	0	0.0

Table 3: Participant Handedness, Ishihara Plate Errors (Ishihara), and Behavioral Inattention Task (BIT) Performance

	Handedness		Ishihara		BIT	
	M	SD	M	SD	M	SD
Control	20.7	2.2	.6	.8	143.0	3.2
Frontal	18.0	4.0	.9	1.1	142.4	2.4
L Frontal	16.8	2.8	.8	1.3	141.6	2.9
R Frontal	19.5	5.3	1.0	.8	143.5	1.3
Parietal	20.4	4.4	1.4	1.8	139.3	3.6
L Parietal	18.3	6.0	1.3	2.3	136.3	2.3
R Parietal	22.0	2.7	1.5	1.7	141.5	2.6

Table 4: Participant Mean Accuracy Performance for Detection and Monitoring Tasks (LH=Left Hemispace, RH=Right Hemispace)

Group	Detect LH		Detect RH		Monitor LH		Monitor RH	
	M	SD	M	SD	M	SD	M	SD
Control	93.8	6.8	93.9	6.3	85.0	10.0	65.3	23.3
Frontal	93.9	7.1	92.7	8.3	79.8	18.7	61.9	23.8
L Frontal	95.0	6.3	94.0	7.8	79.1	24.8	57.3	27.7
R Frontal	92.6	8.8	91.1	9.8	80.8	10.3	67.6	20.3
Parietal	85.9	9.1	84.6	11.3	71.1	15.2	38.2	8.7
L Parietal	86.8	9.5	80.3	15.1	74.3	8.0	34.3	8.1
R Parietal	85.2	10.3	87.8	8.4	68.7	20.1	41.1	8.9

Table 5: Participant Accuracy Performance: 3 (Group: Control, Frontal, Parietal) x 2 (Hemispace (Hemi) Performance: Left, Right) x 4 (Time: 200, 800, 1400, and 2000 msec) Repeated Measures ANOVA for Detection and Monitoring Tasks

Detection Task	F	df	p	Observed Power	Effect Size*
Group	4.159	2, 42	.022	.702	.165
Hemi	1.530	1, 42	.223	.227	.035
Time	.450	3, 40	.718	.132	.033
Group X Hemi	.778	2, 42	.466	.174	.036
Group X Time	.891	6, 80	.505	.333	.063
Time X Hemi	1.363	3, 40	.268	.334	.093
Group X Hemi X Time	1.433	6, 80	.212	.528	.097

Monitoring Task	F	df	p	Observed Power	Effect Size*
Group	4.748	2, 42	.014	.763	.184
Hemi	57.898	1, 42	.000	1.000	.580
Time	1.668	3, 40	.189	.403	.111
Group X Hemi	1.854	2, 42	.169	.365	.081
Group X Time	.807	6, 80	.567	.302	.057
Time X Hemi	.685	3, 40	.567	.182	.049
Group X Hemi X Time	2.031	6, 80	.071	.706	.132

* Partial Eta Squared

Table 6: Patient Accuracy Performance: 2 (Group: Frontal, Parietal) x 2 (Ipsi/ Contra Performance: Ipsilesional, Contralesional) x 4 (Time: 200, 800, 1400, and 2000 msec) Repeated Measures ANOVA for Detection and Monitoring Tasks

Detection Task	F	df	p	Observed Power	Effect Size*
Group	3.571	1, 14	.080	.421	.203
Ipsi/Contra	4.196	1, 14	.060	.479	.231
Time	.286	3, 12	.835	.090	.067
Group X Ipsi/Contra	4.521	1, 14	.052	.508	.244
Group X Time	1.397	3, 12	.291	.280	.259
Time x Ipsi/Contra	1.671	3, 12	.226	.330	.295
Group X Ipsi/Contra X Time	3.133	3, 12	.066	.576	.066
Monitoring Task	**F**	**df**	**p**	**Observed Power**	**Effect Size***
Group	3.896	1, 14	.068	.452	.218
Ipsi/Contra	.235	1, 14	.635	.074	.016
Time	.410	3, 12	.749	.109	.093
Group X Ipsi/Contra	.095	1, 14	.762	.060	.007
Group X Time	1.070	3, 12	.398	.221	.211
Time X Ipsi/Contra	1.399	3, 12	.291	.281	.259
Group X Ipsi/Contra X Time	.221	3, 12	.880	.080	.052

* Partial Eta Squared

Table 7: Single Case Analysis: F test p Values for Accuracy Performance in Left and Right Hemispace

	Detection Task		Monitoring Task	
Patient (Lesion)	Left	Right	Left	Right
#1 (Left Frontal)	.186	.043	.000	.089
#2 (Left Frontal)	.653	.528	.276	.270
#3 (Left Frontal)	.456	.768	.383	.744
#4 (Left Frontal)	.882	.538	.340	.749
#5 (Left Frontal)	.372	.538	.330	.239
#6 (Right Frontal)	.456	.538	.371	.207
#7 (Right Frontal)	.880	.538	.570	.533
#8 (Right Frontal)	.059	.014	.127	.918
#9 (Right Frontal)	.487	.730	.681	.699
#10 (Left Parietal)	.016	.000	.098	.318
#11 (Left Parietal)	.654	.150	.877	.102
#12 (Left Parietal)	.991	.861	.213	.236
#13 (Right Parietal)	.237	.329	.004	.170
#14 (Right Parietal)	.882	.891	.750	.258
#15 (Right Parietal)	.550	.855	.898	.612
#16 (Right Parietal)	.002	.010	.002	.326

Table 8: Patient Lesion and Demographic Information

#	Sex	Age	Edu	Yrs*	History	Lesion Side	Location
1	F	61	12	~1	Stroke	Left	Frontal-Temporal
2	F	40	12	~2	Stroke	Left	Frontal, insular cortex
3	M	64	12	~3	Hemorrhage	Left	Frontal
4	M	67	16	~2.5	Stroke	Left	Frontal
5	M	54	14	~5	Stroke	Left	Frontal
6	F	53	12	~5	Hemorrhage	Right	Frontal
7	F	62	12	~1.5	Stroke	Right	Frontal-temporal
8	F	42	12	~3	Stroke	Right	Frontal-temporal
9	F	67	18	~6	Tumor resection	Right	Frontal
10	M	78	11	~3	Stroke	Left	Parietal
11	M	61	16	~5	Stroke	Left	Parietal-temporal, cerebellum
12	M	69	11	~1	Stroke	Left	Parietal
13	F	72	12	~1	Stroke	Right	Parietal-temporal
14	M	48	9	~1.5	Stroke	Right	Parietal-temporal
15	F	42	10	~1	Stroke	Right	Parietal
16	F	68	18	~3	Stroke	Right	Parietal

* Years between lesion and testing

Table 9: Participant Mean Reaction Time (msec) Performance for Detection and Monitoring Tasks (LH=Left Hemispace, RH=Right Hemispace, IPSI= Ipsilesional, CON=Contralesional)

Group	Detect LH		Detect RH		Monitor LH		Monitor RH	
	M	SE	M	SE	M	SE	M	SE
Control	608	19	575	29	683	16	696	26
Frontal	675	80	678	73	755	61	815	75
Parietal	826	90	741	58	895	91	919	88

Group	Detect IPSI		Detect CON		Monitor IPSI		Monitor CON	
	M	SE	M	SE	M	SE	M	SE
Frontal	646	73	707	79	739	67	832	67
Parietal	737	50	830	94	876	82	938	95

Table 10: Participant Reaction Time Performance: 3 (Group: Control, Frontal, Parietal) x 2 (Hemispace (Hemi) Performance: Left, Right) x 4 (Time: 200, 800, 1400, and 2000 msec) Repeated Measures ANOVA for Detection and Monitoring Tasks

Detection Task	F	df	p	Observed Power	Effect Size*
Group	4.779	2, 42	.013	.765	.185
Hemi	2.807	1, 42	.101	.374	.063
Time	3.641	3, 40	.021	.757	.214
Group X Hemi	.981	2, 42	.384	.209	.045
Group X Time	1.416	6, 80	.219	.522	.096
Time X Hemi	.644	3, 40	.591	.173	.046
Group X Hemi X Time	.735	6, 80	.623	.275	.052

Monitoring Task	F	df	p	Observed Power	Effect Size*
Group	6.909	2, 42	.003	.905	.248
Hemi	2.035	1, 42	.161	.286	.046
Time	1.337	3, 40	.276	.328	.091
Group X Hemi	.470	2, 42	.628	.122	.022
Group X Time	1.003	6, 80	.429	.374	.070
Time X Hemi	.799	3, 40	.502	.207	.057
Group X Hemi X Time	.434	6, 80	.854	.170	.032

* Partial Eta Squared

Table 11: Patient Reaction Time Performance: 2 (Group: Frontal, Parietal) x 2 (Ipsi/ Contra Performance: Ipsilesional, Contralesional) x 4 (Time: 200, 800, 1400, and 2000 msec) Repeated Measures ANOVA for Detection and Monitoring Tasks

Detection Task	F	df	p	Observed Power	Effect Size*
Group	1.022	1, 14	.329	.156	.068
Ipsi/Contra	9.899	1, 14	.007	.833	.414
Time	1.495	3, 12	.266	.298	.272
Group X Ipsi/Contra	.513	1, 14	.486	.103	.035
Group X Time	.595	3, 12	.630	.139	.129
Time x Ipsi/Contra	.998	3, 12	.427	.208	.200
Group X Ipsi/Contra X Time	1.637	3, 12	.233	.324	.290

Monitoring Task	F	df	p	Observed Power	Effect Size*
Group	1.269	1, 14	.279	.183	.083
Ipsi/Contra	10.714	1, 14	.006	.860	.434
Time	.715	3, 12	.561	.159	.152
Group X Ipsi/Contra	.477	1, 14	.501	.099	.033
Group X Time	1.618	3, 12	.237	.321	.288
Time X Ipsi/Contra	2.702	3, 12	.092	.509	.403
Group X Ipsi/Contra X Time	1.888	3, 12	.185	.369	.321

* Partial Eta Squared

APPENDIX B: FIGURES

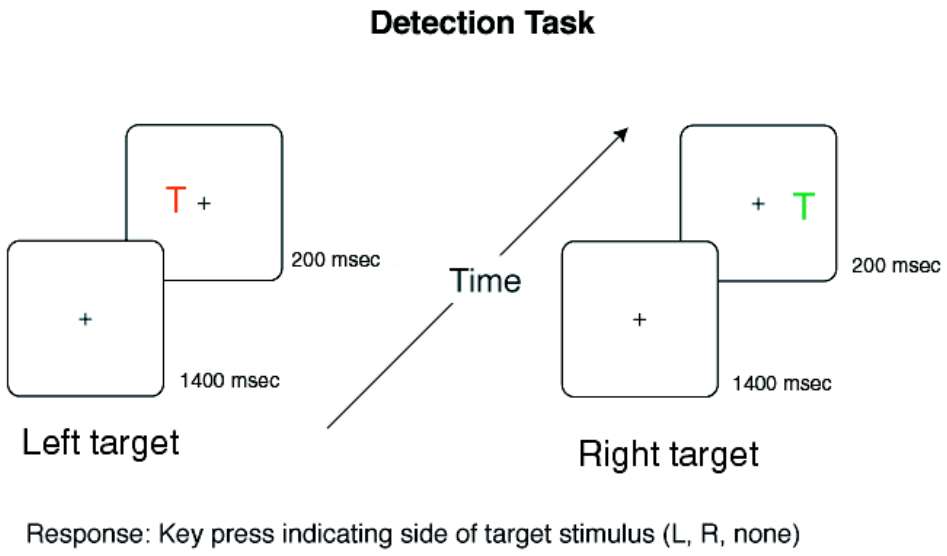

Figure 1. Detection Task

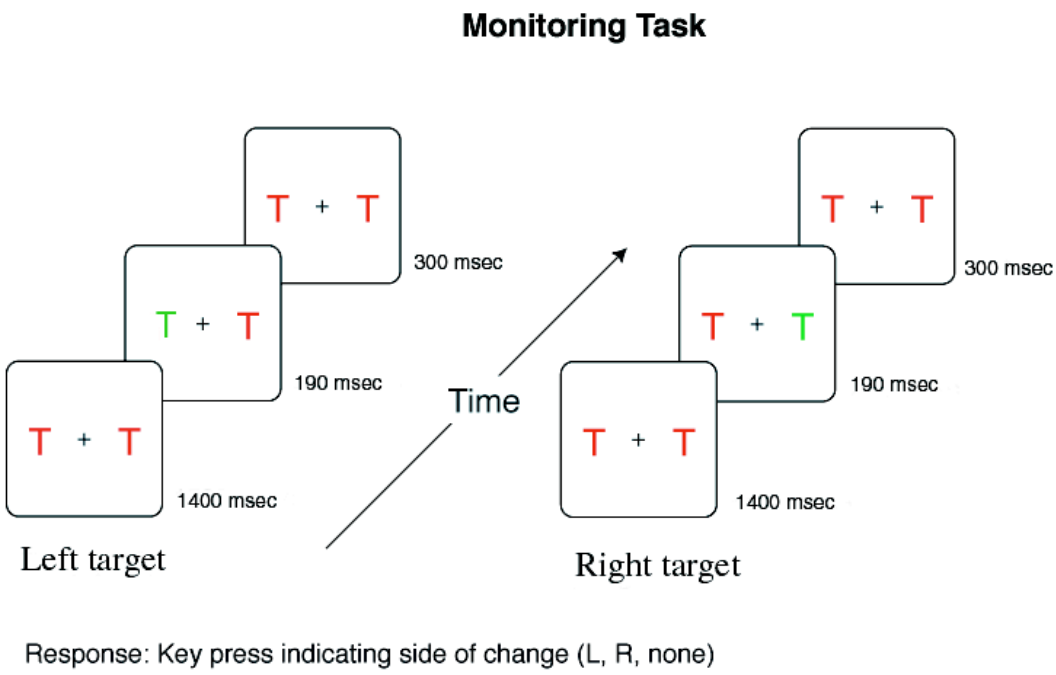

Figure 2. Monitoring Task

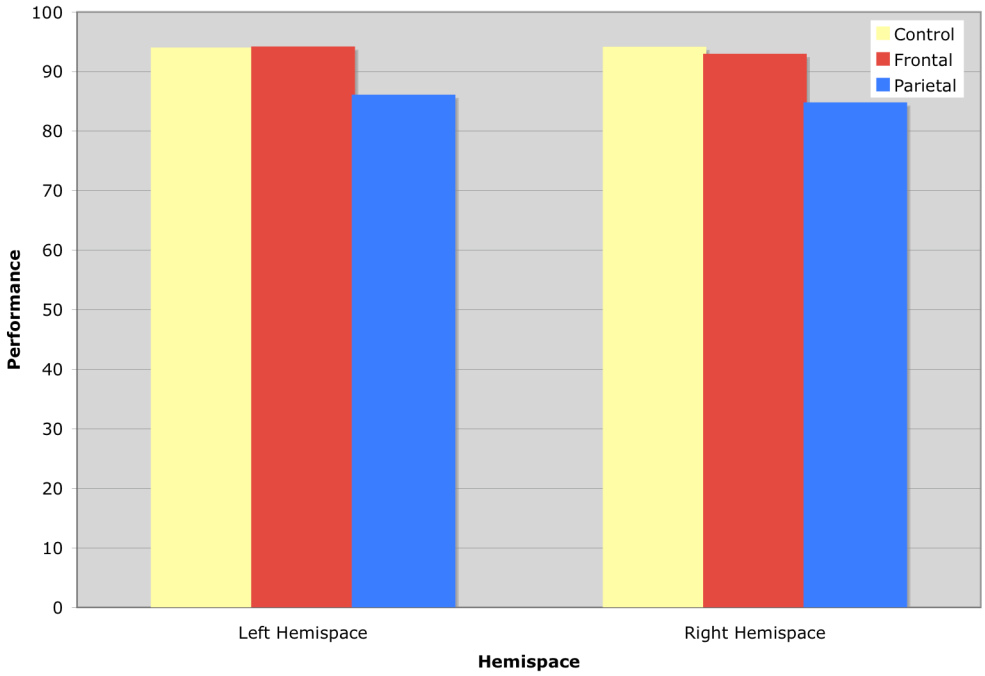

Figure 3. Detection Task: Performance by Hemispace

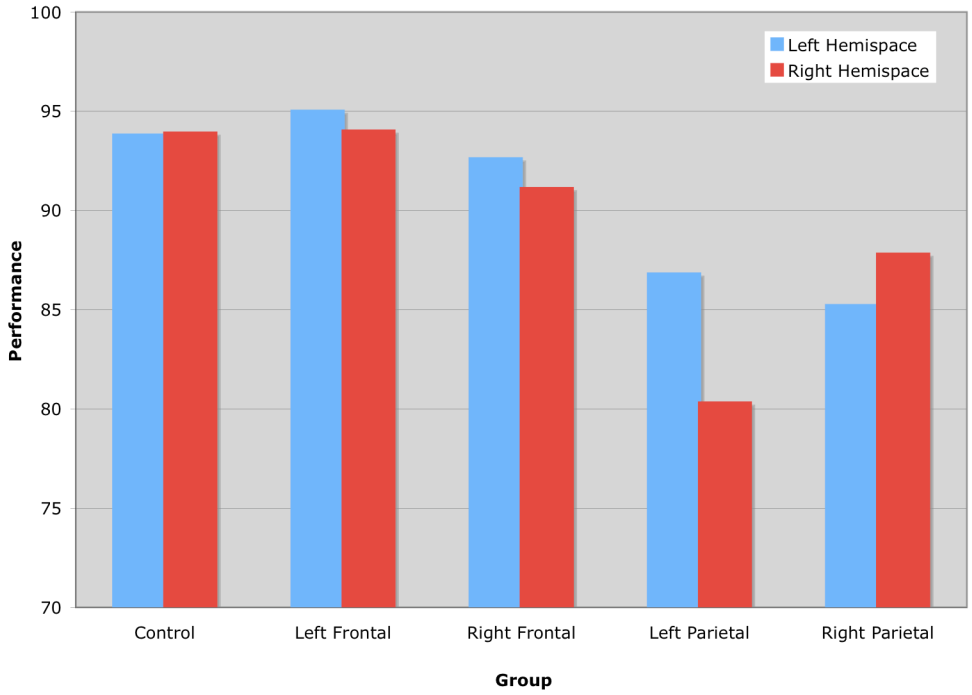

Figure 4. Detection Task: Performance by Hemispace Across Subgroups

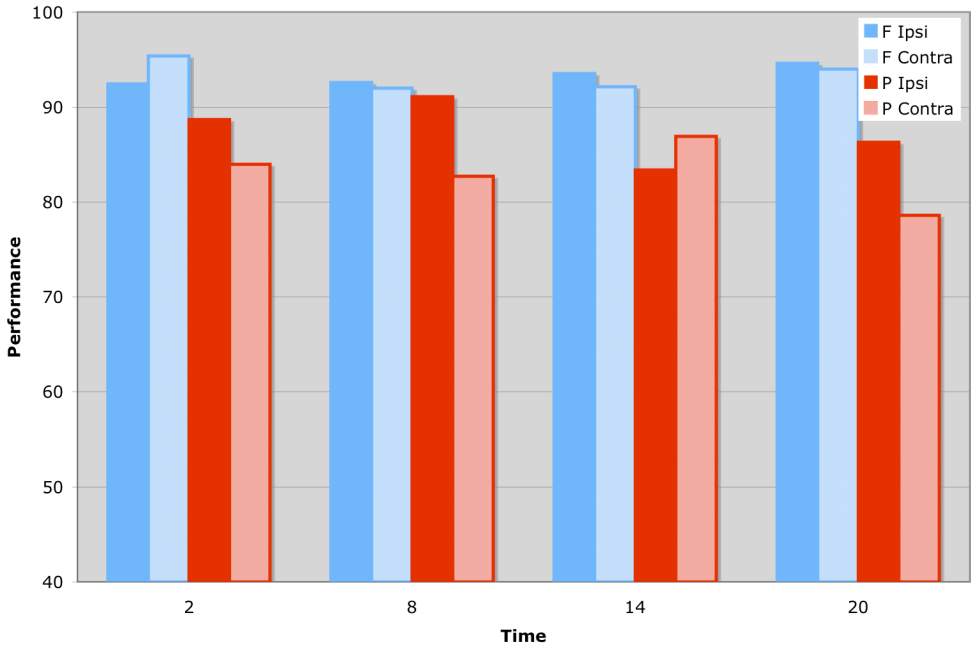

Figure 5. Detection Task: Frontal (F) and Parietal (P) Ipsilesional (Ipsi) and Contralesional (Contra) Performance Across Time

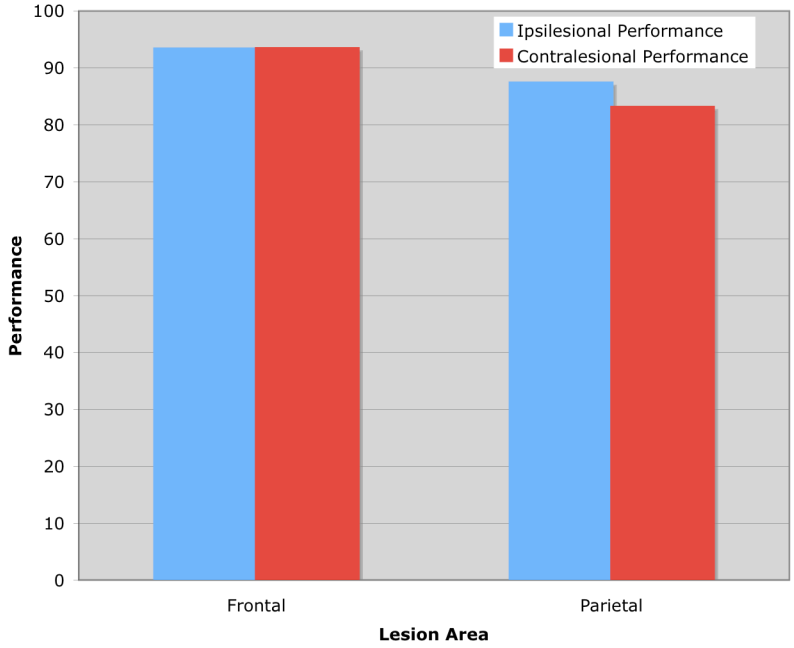

Figure 6. Detection Task: Frontal and Parietal Ipsilesional and Contralesional Performance

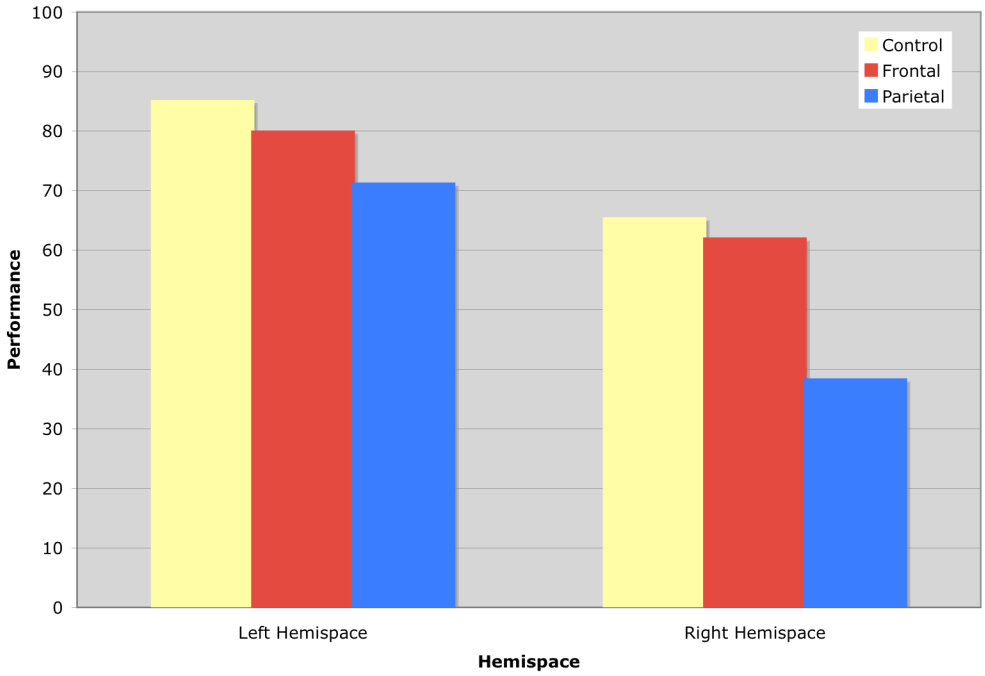

Figure 7. Monitoring Task: Performance by Hemispace

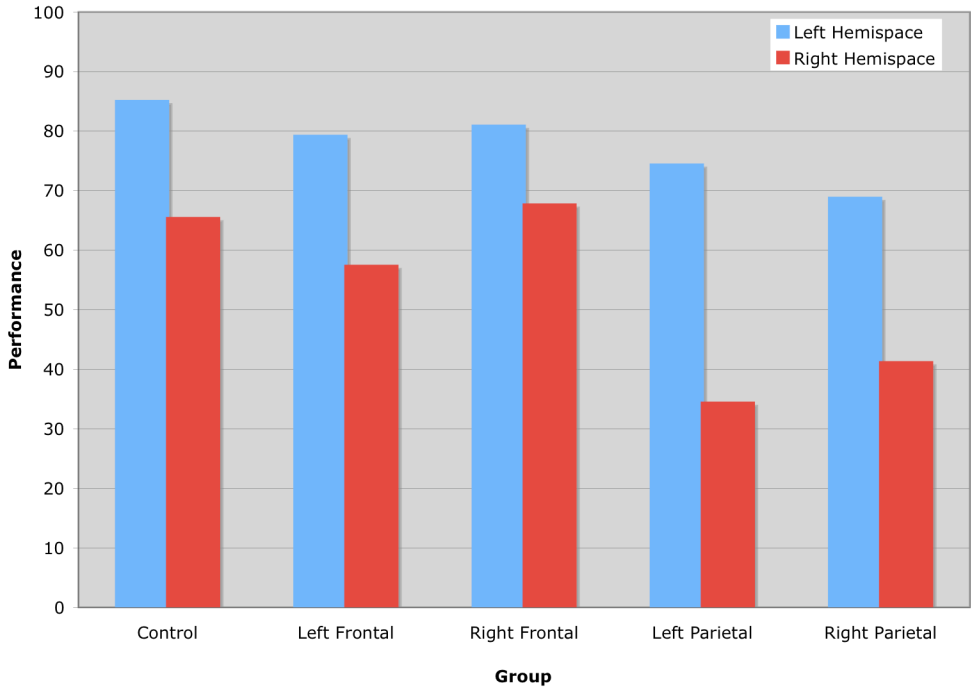

Figure 8. Monitoring Task: Performance by Hemispace Across Subgroups

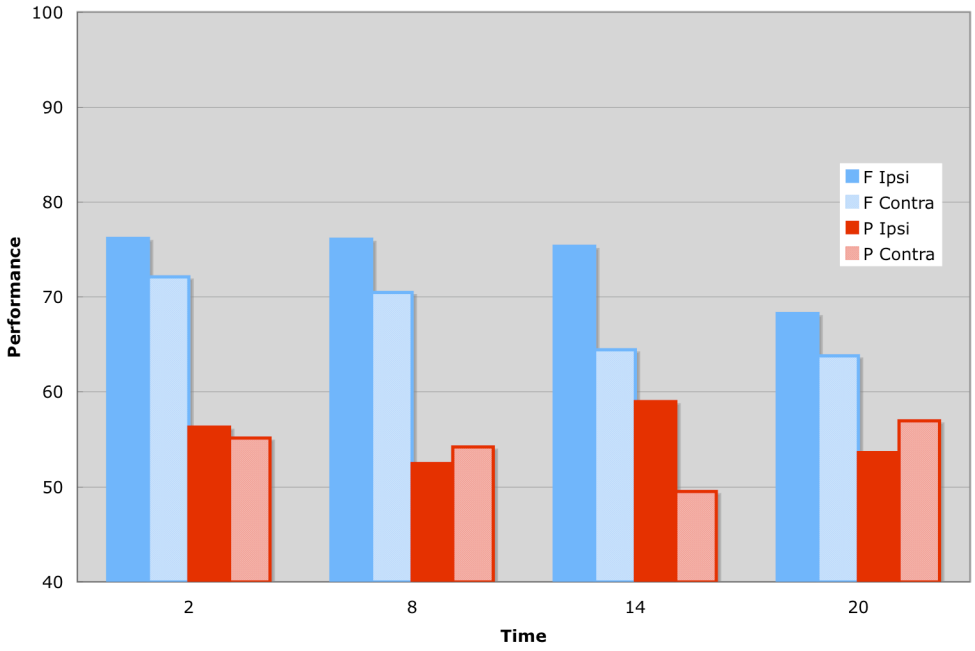

Figure 9. Monitoring Task: Frontal (F) and Parietal (P) Ipsilesional (Ipsi) and Contralesional (Contra) Performance Across Time

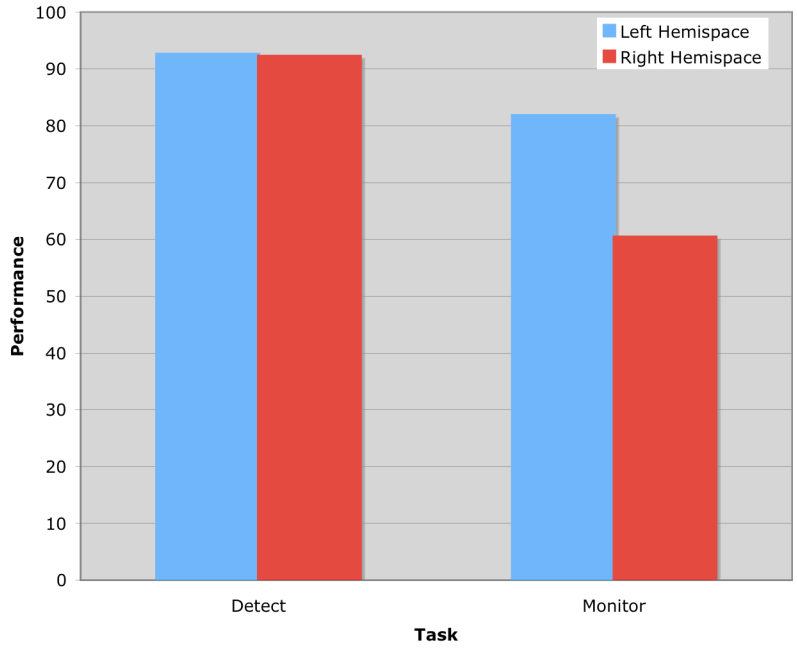

Figure 10. Detection and Monitoring Tasks

VITA

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Papers

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D'Esposito, M., Postle, B., Ballard, D., & **Lease, J**. (1999). Maintenance and manipulation of information held in working memory: An Event-Related fMRI study. *Brain and Cognition, 41*, 66-86.

Kimberg, D.Y., Aguirre, G.K., **Lease, J**., & D'Esposito, M. (2001). Cortical effects of bromocriptine, a D-2 dopamine receptor agonist, in human subjects, revealed by fMRI. *Human Brain Mapping, 12*, 246-257.

Kable, J.W., **Lease-Spellmeyer, J**., Chatterjee, A. (2002). Neural substrates of action event knowledge. *Journal of Cognitive Neuroscience, 14(5),* 795-805.

Selected Abstracts

D'Esposito, M., Tang, J., Zarahn, E., **Lease, J**. & Aguirre, G.K. (1996). Prefrontal function in normal human aging: A functional MRI study. *Neurology, 48*, A168-169. (American Academy of Neurology.)

Kimberg, D.Y., **Lease, J.F**., Caggiano, D.M., & D'Esposito, M. (2000). An fMRI study of the cortical effects of the dopamine agonists bromocriptine and pergolide during working memory performance. (Cognitive Neuroscience Society.)

Lease-Spellmeyer, J., Sancar, F., & Chatterjee, A. (2001). Spatial attention to color and shape following right brain damage. (Cognitive Neuroscience Society.)