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OCULOMOTOR BEHAVIOR AND VISUAL HEMI-NEGLECT

*City University of New York*

Ph.D. 1983

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**OCULOMOTOR BEHAVIOR AND VISUAL HEMI-NEGLECT**

**by**

**Cris W. Johnston**

**A dissertation submitted to the Graduate  
Faculty in Psychology in partial fulfillment  
of the requirements for the degree of Doctor  
of Philosophy, The City University of New York.**

**1983**

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1983

This manuscript has been read and accepted for the graduate Faculty in Psychology (the Neuropsychology subprogram) in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

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## **Abstract**

### **OCULOMOTOR BEHAVIOR AND VISUAL HEMI-NEGLECT**

by

**Cris W. Johnston**

**Advisor: Professor Leonard Diller**

The present study sought to determine if the severity of visual hemi-neglect (VHN) is reflected in the degree of abnormality of oculomotor (OM) behavior, and also to investigate the effect responding to stimuli in different spatial fields has on OM behavior. The dependent variables employed consisted of the gross measure Exploration Time, which yields a value based on the amount of time a subject spends exploring different areas of a visual display, as well as the components of exploratory behavior, i.e., Saccadic Latency, Saccadic Accuracy, and Fixation Stability. Ten adult right hemisphere stroke patients with marked VHN, 10 with mild VHN, and 10 neurologically unimpaired adults were studied. Although all variables distinguished the control subjects from those with marked VHN, other group differences varied with the different OM measures. Fixation Stability was the only variable that

demonstrated a spatial field effect. Furthermore, the direction of eye movement and the presence of a sensory visual field deficit were found to be important factors in interpreting the findings. It was concluded that the relationship between VHN and OM behavior is complex, and that sorting out the complexities should aid in the development of programs designed to remediate neglect.

### Acknowledgements

As I reflect on the number of people who played a significant role in the development and/or execution of this research it is with some incredulity that I consider what might (or might not!) have transpired without their involvement. Among these people are mentors and professors, administrators and program directors, friends and fellow students, and of course all those who served as subjects. I am indebted to them all.

A very special note of thanks is extended to my advisor, Leonard Diller, for his unfailing support at every turn.

The person who is by far the most deserving of my appreciation is Marcia, my wife. Her contributions, indeed her sacrifices, were so numerous that I would be remiss if I did not underscore my gratitude by dedicating this work to her.

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## CHAPTER I

### INTRODUCTION

The purpose of the present research was to investigate the nature and extent of abnormal oculomotor (OM) behavior manifested by right hemisphere brain-damaged individuals who display visual hemi-neglect (VHN). Abnormal OM behavior is defined as that which is significantly deviant from the average OM performance of a group of neurologically unimpaired control subjects. Using a gross measure of OM behavior, Chedru, Leblanc, & Lhermitte (1973) have shown such a difference to exist for patients with marked VHN, but not for those with mild VHN. Using more refined measures of OM performance, it was the present author's expectation to demonstrate abnormal OM behavior in mild as well as marked cases of VHN, and by the nature of the results to assess a recently proposed mechanism and theory (Heilman, 1979) purported to underlie VHN.

VHN is manifested by the failure of a person to acknowledge visual stimuli occurring on the side opposite his or her cerebral lesion (Heilman, 1979). Although neuropsychological literature abounds with reports dealing with the phenomenology and postulated underlying mechanisms of VHN (e.g., see reviews by Heilman & Valenstein, 1979a,b;

Lynch, 1980; and Weinstein & Friedland, 1977), to date little is known about the OM performance of those who demonstrate this lateralized symptom. The only controlled OM/VHN study known to the present author (Chedru et al., 1973) suggested that marked, but not mild cases of VHN are associated with abnormal ocular exploration of the area contralateral to the cerebral lesion. The dependent OM variable of concern in that study was a gross measure (Exploration Time), and severity of VHN was only vaguely defined. Indeed, most of the data analyses performed compared groups of patients with visual field defects to those without, rather than using the presence or absence of VHN as the grouping variable. In discussing the findings of their study, the authors suggested that more elementary components of OM behavior need to be systematically studied.

In response to these criticisms, the present study examined the relationship of severity of VHN not only to Exploration Time, but more importantly to fundamental parameters of the OM activity found in exploratory behavior (cf., Russo, 1978). These parameters are termed Fixation Stability, Saccadic Latency, and Saccadic Accuracy. Fixation Stability refers to the capacity to maintain fixation on some intended target without premature distraction away

from it. Saccadic Latency is another way of saying eye movement reaction time. Saccadic Accuracy means correctness (in terms of coordinates) of eye movement, with poor accuracy often referred to as dysmetria. The stimuli that were used to study these parameters consisted of individual crosses to stare at in the case of Fixation Stability, the onset of a light for Saccadic Latency, and two dots separated in the horizontal meridian to be alternately and repeatedly fixated for the measure of Saccadic Accuracy. In comparing operationally defined groups (i.e., Mild VHN and Marked VHN), it was hypothesized that the degree of abnormality of the individual OM parameters would be smaller for the Mild VHN group, but abnormalities would nevertheless be demonstrated for this group when compared to a group of normal control subjects without VHN.

The idea that an OM cycle (the lowest level of a single, complete unit of Exploration Time) may be subdivided into several time related components (namely Saccadic Latency, Saccadic Accuracy, and Fixation Stability) which may be found to yield abnormal scores when each component is individually studied, yet result in a normal score when the components are obscured by resorting to such a gross summary measure as Exploration Time, receives support from OM research in other areas including individ-

ual differences (Johnston & Pirozzolo, 1981), and reading (Rayner & Inhoff, 1981). Furthermore, there is little question that complex neuropsychological abilities (e.g., verbal communication) rely on functional systems, within which a subtle interruption in the integrity of individual aspects (e.g., word retrieval, phonemic understanding) may result in unimpaired global outcomes when evaluated with a screening test, but produce quite a different picture under more detailed examinations (e.g., compare the Aphasia Screening Test with the Boston Diagnostic Aphasia Examination). This functional systems conceptualization was first promulgated by Anokhin (1935) and recently popularized by Luria (1973). Using an example germane to the present field of study, if one considers a case of mild VHN in which the global measure of OM Exploration Time is within normal limits, it may be possible that the VHN which is present could be shown to be associated with impaired subcomponents of Exploration Time (e.g., anomalous Fixation Stability) but that the subcomponents would not significantly affect, either singly or collectively, the grosser measure Exploration Time. In other words, it may be the subtler aspects of exploration that may be affected.

In a further attempt to characterize the nature of abnormal OM behavior, the research design employed also

permitted an analysis of data for independent spatial fields. Left and right spatial fields are determined with reference to the sagittal head or body midline (Heilman 1979), and thus have boundaries that remain constant with respect to eye movements. By contrast, visual fields are determined by one's line of sight and shift as direction of gaze shifts. The spatial field in which stimuli and responses occur has been demonstrated as one possible mechanism underlying the asymmetric extremity motor performance of patients with hemi-neglect (Heilman & Valenstein, 1979b). Heilman (1979) postulates that each hemisphere is responsible not only for mediating contralateral sensory and motoric activity per se, but that each hemisphere may also be responsible for mediating this activity in the opposite spatial field, regardless of the hemisphere with which the afferent or efferent nervous information is directly associated. If this is a valid hypothesis, then OM performance should vary according to the spatial field in which it occurs, i.e., be more impaired in the spatial field contralateral to the lesion. However, research of spatial field effects has not, prior to the present study, been experimentally extended to the OM domain.

The scientific merit of studying OM behavior and VHN

as described is, therefore, two-fold. One aim is to discover whether a breakdown of exploratory ocular activity into components provides a more definitive account of abnormal OM behavior and VHN than does using a gross measure such as Exploration Time. The second aim is to test the spatial field theory Heilman (1979) has offered to explain unilateral visual neglect.

Before describing the details and experimental design of the present study, the following three sections will expand on the major areas of research relevant to this investigation. These areas are the phenomenology of VHN and experimental variables of interest, previous eye movement and eye fixation research related to the issues at hand, and the mechanisms thought to underlie this disorder, with particular emphasis on spatial field effects.

## CHAPTER II

### VISUAL HEMI-NEGLECT

Hemi-neglect refers to the "...failure of patients to report, respond, or orient to stimuli presented to the side contralateral to a cerebral lesion," (Heilman, 1979, p. 268). Although the present research is concerned specifically with visual hemi-neglect (VHN), neglect phenomena may occur in any modality or combination of modalities, and be accompanied, as well, by a variety of neuropsychological and neuropathological conditions. The synonyms for VHN reflect the various concerns of different investigators. Some authors prefer the term "inattention" to "neglect" (Weinstein & Friedland, 1977), certain others are opposed to both, suggesting "imperception" as more appropriate (Gianutsos, Glosser, Elbaum, & Vroman, 1981), while still others (as indicated by Heilman, 1979) prefer the term "agnosia". As substitution for the "visual-hemi" part of the term one may find "unilateral spatial", "hemispatial", or "visuospatial" (Heilman, 1979). This varied nosology suggests some of the putative neuropsychological and neuropathological factors involved. Nomenclature notwithstanding, this chapter will focus on those factors considered to be relevant as far as the present study is

concerned. That is, the particular topics to be discussed are the clinical tests used to elicit VHN, the role of the visual sensory system, the duration of neglect symptomatology, and the laterality, locus, and etiology of the underlying lesion.

Depending on the constraints one places on the definition of VHN, the original description of this disorder may be attributed to either Holmes (1918), or Ridoch (1935), although it was experimentally produced in animals (Bianchi, 1895) before it was readily recognized by clinicians. With the burgeoning of case reports and controlled group studies that attempted to define the important aspects of VHN better, it soon became apparent that a consensus could be reached only in terms of general phenomenology (see the review by Critchley, 1966). For example, one patient with VHN may tend not to notice food to his or her left, whereas another may have difficulty finding the beginning of the next line while reading. Although common general behavior patterns may be identified, the causal neuropsychological mechanisms considered to underly VHN are as numerous as the investigators who do research in this area. The various notions of the pathogenesis of VHN will be discussed in the chapter titled Spatial Field Effects.

Although a general consensus exists for what one

observes in those who display VHN, there is no single, standard test used by all who investigate this phenomenon. Since the operational definition of VHN is an important factor in the design of any study in the area, the rationale for selecting the specific instruments that were used in this study will be presented.

A variety of paper and pencil tasks have been employed to elicit impairments specific to VHN. Copying and spontaneous drawing exercises have shown that patients with VHN may omit, or produce a disorganized or incomplete representation of the side of a figure contralateral to the side of their cerebral lesion. This is especially evident when symmetrical figures such as a person, or daisy are to be drawn (Lezak, 1976). Cancellation exercises demonstrate VHN when performance yields relatively more omissions on the side of the page contralateral to the lesion than on the side which is ipsilateral. Such exercises have included crossing out lines according to the method of Horenstein (Heilman, 1979), or specific letters (or numbers, words, or geometric figures) from among a field of a mixture of them (Diller, Ben-Yishay, Gerstman, Goodkin, Gordon, & Weinberg, 1974). Simple line bisection tests have been used to indicate VHN when patients show a tendency to bisect at a point to the side of the real midline

that is ipsilateral to their lesion (Heilman & Valenstein, 1979b).

Visual searching and match-to-sample tests have also been used to demonstrate VHN. These tests are scored for the presence of a response bias (rather than total errors) in selecting items occurring ipsilateral to the side of lesion as being characteristic of patients with VHN. Some investigators have used well known psychometric tests, such as Raven's Colored Progressive Matrices, to get this information (e.g., Gainotti, 1968), while others have developed some very refined matching tests which are capable of identifying even quite mild cases of VHN (e.g., Piasetsky, 1981). The latter test is particularly successful, probably due to the variety of demands it places on the examinee. This test consists of four conditions: straightforward matching, and matching from immediate recall both figures that are familiar (e.g., shoes), as well as ones that are unfamiliar (nonsymbolic geometric shapes).

Of these various methods for determining the presence and extent to which one manifests VHN, the present research used a combination of the letter cancellation tests (Diller et al., 1974; see Appendix A for an example), and the matching test of Piasetsky (1981; see Appendix B for an example). This decision was based on Piasetsky's findings

comparing the success of his test in identifying patients with VHN to the incidence of neglect obtained using five other commonly used instruments. In a group of right brain-damaged subjects (n=51) his test was the most sensitive to the presence of neglect, indicating a significant response bias whenever any other single test also indicated the presence of neglect. Only three subjects were not identified by the matching test. However, they did reach criterion for neglect on either a single letter cancellation task, or a double letter cancellation task. Therefore, it was considered that the combined use of the matching and cancellation tests would allow for both the most sensitive detection of VHN, and assessment of its severity.

The integrity of the visual sensory system is of concern in the present study since stimuli are visual. However, it has been shown that neither defects in visual acuity (Brain, 1941), nor defects in visual fields (McFie, Piercy, & Zangwill, 1950) are necessary predisposing factors for the occurrence of VHN. Although homonymous hemianopic field defects are often associated with neglect, VHN appears to occur just as frequently in the presence of an intact visual sensory system. However, in order to exclude the possibly confounding effects of deficits in the

visual sensory system, i.e., to ensure a certain minimal sensory capacity is present (obviously a legally blind individual would be expected to experience difficulty on any visual task), visual fields and visual acuity were measured. Test procedures and specific criteria (for visual acuity) will be discussed in the Research Design chapter.

The laterality of brain-damage is important to the present study for a variety of reasons. The patients that Holmes (1918) discussed who showed neglect had bilateral lesions. It eventually became clear that unilateral lesions were sufficient to produce VHN (Brain, 1941), but it is here that clarity fades. Although it is generally agreed that VHN is more common in right hemisphere lesions than in left hemisphere lesions the proportions vary with different authors (Friedland & Weinstein, 1977). One possible explanation for this situation is discussed by Battersby, Bender, Pollack, and Kahn (1956). They point out that left brain-damaged patients are at a decided disadvantage in many tests used to demonstrate the presence of neglect since these patients may have difficulty understanding task demands because of aphasia. Another ramification of this problem is that some studies comparing lateralization differences may, by excluding individuals

with comprehension deficits (lesions in and around Wernicke's area), exclude people with posterior left hemisphere lesions, thus possibly distorting estimates of prevalence of VHN relating to lateralization of damage since such patients may also demonstrate neglect.

In order to avoid these problems, the present research was confined to an investigation of patients with unilateral right hemisphere brain-damage. Furthermore, a certain minimal degree of verbal cognitive ability was required of all participating subjects. This was based on performance on the Vocabulary subtest of the Wechsler Adult Intelligence Scale (WAIS), as well as on the digits forward portion of the Digit Span subtest from the WAIS. These measures reflect verbal receptive, memory, cognitive, and expressive ability (Lezak, 1976). Specific selection criteria involving these tests are given in the Research Design chapter.

It was felt that a more discrete breakdown of locus of lesion might provide some useful information for a post hoc analysis of performance. Animal research has shown neglect can be produced by lesions from the upper brainstem to the cortex (Friedland et al., 1977). Suction (Welch & Stuteville, 1958) of specific brain structures, and the destruction of chemical pathways (Ungerstedt, 1973), have

been successfully used in animals to produce VHN. These various studies have emphasized somewhat different neglect phenomena. In people neglect has not only been observed accompanying parietal lobe damage (e.g., Critchley, 1966), but also with lesions in the dorsolateral frontal lobe, the cingulate gyrus (Heilman & Valenstein, 1972), the neostriatum (Heir, Davis, & Richardson, 1977), and the thalamus (Watson & Heilman, 1979). The vast majority of reports indicate VHN is most commonly seen associated with post-Rolandic lesions (Heilman, 1979). The nature and severity of neglect may be related to various of the proposed underlying mechanisms, which in turn are thought to reflect different anatomical/functional substrata (Lynch, 1980; and Mesulam, 1981). Therefore, data were obtained for each patient regarding the locus and extent of lesion for the purpose of performing post hoc analyses of individual cases, and subgroups, if possible. The data collected included neurological examinations and laboratory test information such as computerized tomographic (CT), radiographic, angiographic, electroencephalographic, and brain scan reports.

Of possible additional relevance to the present research, aside from locus, is the nature of the underlying brain pathology. Heilman (1979) has noted differences in

the occurrence and persistence of neglect related to differences in the etiology of brain-damage. He states that in people the most common cause of neglect from sub-cortical lesions is intracerebral hemorrhage, while thrombotic or embolic infarcts are the most common causes of cortically produced neglect. Insidious pathology is less likely to produce neglect; thus VHN may also be seen with rapidly growing tumors (e.g., glioblastomas), but seldom with slowly growing tumors or degenerative diseases. Neglect syndromes may occur during the post-ictus of seizures; however, the duration of neglect in these episodes is typically quite short. In order to invoke some degree of control over this multiplicity of variables, only those patients with brain-damage due to cerebral vascular disorders were studied. These disorders included hemorrhages (usually aneurysm-related), and thrombotic or embolic infarcts.

The length of time following the cerebral insult was considered to be an important factor for the design of the present research insomuch as the first three weeks after a brain lesion are often marked by a high degree of general symptom instability (Kertesz, 1979). Systematic studies of the natural recovery from VHN are scarce. Campbell and Oxbury (1976) examined performance of patients with neglect

at one and six months after stroke. The second testing revealed continued impairment on visuospatial tasks, despite resolution of neglect. It should be noted that presence of neglect was based on performance on drawing tasks, not as sensitive a measure as some other types of tasks (cf., Piasetsky, 1981). There are reports of neglect persisting up to 12 years after onset (Kertesz, 1979). Lawson (1962) stresses that lack of a patient's awareness regarding his or her neglect retards improvement, and Johnston and Diller (1983) have recently underscored the importance of this factor in a group of VHN patients who showed performance deficits one year post-stroke, despite the fact that they had received perceptual-cognitive retraining. Heilman (1979) suggests that while monkeys typically show no signs of neglect after one month following a lesion, humans may never recover. Given the above findings, the decision was made to use brain-damaged subjects who were at least four weeks post cerebrovascular attack, and hospitalized for rehabilitation. This determination not only served to minimize the general symptom instability encountered immediately following an acute stroke, but also ensured that all brain-damaged subjects would be contained in a similar environment at the time of testing.

### CHAPTER III

#### OCULOMOTOR BEHAVIOR

Since the purpose of the present study was to investigate the nature and extent to which right hemisphere brain-damaged people with VHN also manifest abnormal OM behavior, a review of relevant eye movement and eye fixation research is appropriate. This chapter will focus on the rationale behind choosing the particular OM parameters employed in the present study (i.e., Fixation Stability, Saccadic Accuracy, and Saccadic Latency).

Technological developments (for a review see Young & Sheena, 1975) have facilitated objective, detailed investigations of the relationship between OM behavior and psychological functioning (Fisher, Monty, & Senders, 1981; Monty & Senders, 1976; Senders, Fisher, & Monty, 1978; and Yarbus, 1967). While the great majority of this research has been on non-medical populations, the value of studying subjects with neuropsychological dysfunction is being increasingly appreciated. For example, referring to their work with children, Boersma and Muir (1975, p.77) stated, "...if the retardate is deficient in the ability to direct his visual attention, objective evidence of the nature of the deficit should precede attempts directed toward improv-

ing his attentional behavior. This logically leads to the study of eye movements as a means of objectively describing visual behavior." The same enjoiner should apply to investigations of "attentional deficits" (to use the term loosely) in adults. But curiously, only a handful of scattered attempts have been made to study VHN using eye monitoring technology.

The present author is aware of only one experimentally rigorous group study of OM behavior and VHN (Chedru et al., 1973) published in English. These investigators used electro-oculography, and match-to-sample tasks taken from Popplereuter (1917) to study the OM behavior of 36 normal control, and 115 focally lesioned brain-damaged subjects. The brain-damaged subjects were divided into four groups based on whether the left or right hemisphere was affected, and according to the presence or absence of visual field defect. Two levels of severity of VHN were distinguished for each group. Major VHN was determined to be present when the "patient's daily activities" (p. 95) were affected. Minor VHN existed when "omissions or displacements in tests of copying sketches or of writing" (p. 95) were present. No further clarification of severity was presented, this being a serious shortcoming of this study (see the discussion of assessing the severity of VHN in the

chapter titled Phenomenology). Twenty-seven patients with right hemisphere lesions were found to have neglect. Seven of them did not have a visual field cut (four of these had major VHN, three had minor VHN). Twenty did have a field cut (10 with major VHN, and 10 with minor VHN).

Chedru et al. (1973) analyzed their data for the most part with respect to laterality of lesion and presence or absence of visual field defect. They found that the mean search time and fixation duration of normal subjects was significantly shorter than that of the brain-damaged subjects. Those who had visual field defects had mean search times more than twice as long as the subjects without field defects. Search times were significantly longer for targets situated in the contralesional hemispace than in the ipsilesional space (except for the finding of no difference in the right hemisphere brain-damaged group who did not have a visual field defect). In contrast to all other subjects, hemianopics made significantly more short saccadic movements. This measure was based on a subjective rating of scan paths. Finally, they concluded that marked left unilateral neglect was found to be associated with defective exploration of that field; however, minor signs of neglect were not associated with disturbed searching behavior. The dependent variable was

amount of time spent exploring each hemifield.

The Chedru et al. (1973) study sets the stage for two questions important to understanding the relationship between OM behavior and VHN. The first is, why should only patients with marked VHN (vs. those with mild VHN) have asymmetrical searching behavior? Secondly (and perhaps intimately related to the first question), can the gross measure of exploratory behavior be broken down to reveal a more refined relationship between OM behavior and severity of neglect?

In attempting to explain why subjects with mild VHN do not show significant asymmetries in exploration, Chedru et al. (1973) suggested that the neglect symptomatology of such subjects may be caused by a combination of hemianopic defect and constructional apraxia. In reviewing the data presented by Chedru et al. (1973), it is evident that even the mild group demonstrated the trend of OM asymmetry. Furthermore, their suggestion does not account for neglect behavior in patients without visual field defects. Heilman (Note 1) has unpublished data that indicate that patients with hemi-neglect make hypometric saccades when moving their eyes to the neglected side, in addition to the finding that during a line bisection task they do not explore the contralesional side of the line. Heilman does not

consider these findings to be dependent on a concomitant visual field defect. It is plausible to suspect that the relevant OM parameter of asymmetry for the mild VHN subjects in the Chedru et al. (1973) study may have been "washed out" by being embedded in such a gross measure as Exploration Time.

Thus, the present study investigates the hypotheses that certain fundamental OM parameters of visual exploratory behavior (such as Saccadic Accuracy) are abnormal in patients with VHN (i.e., vary with severity of VHN but still are abnormal in mild as well as severe cases), and also show an asymmetry according to the field in which the ocular activity occurs. The basis for these hypotheses derives not only from the Chedru et al. (1973) study, but also from research and models of OM behavior and cognitive functioning (Johnston & Pirozzolo, 1981; Rayner & Inhoff, 1981; Russo, 1978), other research on OM behavior and brain-damage (Belleza, Rappaport, Hopkins, & Hall, 1979; Cohn, 1972; Gassel & Williams, 1963; Karpov, Luria, & Yarbus, 1968; Messert & Barron, 1973; Sundqvist, 1978; Van Allen, 1973), and from studies concerning one of the mechanisms (i.e., the spatial field effect) considered to underlie the manifestation of hemi-neglect (Heilman, 1979; Heilman & Valenstein, 1979b; Heilman, Note 2).

The remainder of this chapter will discuss relevant OM models and research on OM behavior of brain-damaged people. The spatial field effect and its theoretical foundation will be dealt with in the subsequent chapter.

A consideration of functional OM behavior modelling reveals how it is possible that mild VHN patients may in fact have abnormal and/or asymmetric OM performance. As previously mentioned, the measure Exploration Time is a gross one. By reducing Exploration Time to its component elements it would not be inconsistent with the Chedru et al. (1973) data to discover abnormal performance on individual parameters. In cases where the degree of abnormality of single elements and/or the number of abnormal scores on various parameters is very great, a gross measure such as Exploration Time would undoubtedly be affected. In less extreme cases this would not necessarily be so. This result is obtained when the gross measure is of such a high order that another level of measurement exists between the summary variable and the more discrete parameters. In the context of the present study that middle level link may be termed an eye movement cycle. Thus, a continuum of normality-abnormality may be operating in the OM behavior of patients with VHN, yet be obscured by referring to a high level summary variable.

Russo (1978) has summarized the components of a complete eye movement cycle from the viewpoint of a cognitive theorist. The components are as follows:

- (1) Determine the location of the next fixation.
- (2) Transmit the movement command to the motor system.
- (3) Move the eye to the new position.
- (4) Transmit the new visual stimulus to the cortex.
- (5) Decode the received stimulus.

He states that the typical duration for a complete cycle is 230 msec., with a minimum duration of 70 msec. With the proper experimental manipulations these components may be operationally defined in terms of Fixation Stability, Saccadic Latency (or depending on the paradigm, Fixation Duration), and Saccadic Accuracy (cf. Rayner & Inhoff, 1981). These are the parameters used in the present study.

Such a componential view is also shared by theories of neuropsychological functioning (e.g., Luria, 1973) as well as supported by studies of OM behavior during performance on cognitive tasks (e.g., Johnston & Pirozzolo, 1981). Luria (1973) has developed a view of brain-behavior relationships in which functional systems exist, any component of which may be disturbed to produce a specific deficit, without significantly interfering with more general behaviors. Johnston and Pirozzolo (1981) have presented

evidence suggesting that OM behavior during cognitive tasks may also operate according to a componential principal. They believe a trade-off may exist for certain OM parameters such that marked differences in specific parameters could exist between individuals but that by applying particular formulas to these data a global (comprehensive) measure may be created that would not distinguish different individuals.

There are no other published reports of OM behavior in patients with VHN known to the author. Since it is a fairly common symptom in people with right hemisphere brain-damage (at least 60 percent as reported by Diller & Weinberg, 1977), it is instructive to consider research on OM behavior in such unilaterally brain-damaged subjects. The studies to be noted are only those which have a bearing on the OM parameters under examination in the present investigation.

Two studies suggest that right hemisphere brain-damaged subjects, and by inference patients with VHN, have difficulty with Fixation Stability (Bellaza et al., 1979; Messert & Barron, 1973). In patients who demonstrated drawing impairment Bellaza et al. (1979) noted significantly shorter fixations, and a greater number of saccades, compared to normals. Although they found

scanning patterns for opposite peripheral fields were often asymmetric, an analysis of dependent variables was not performed according to laterality or locus of lesion. Messert and Barron (1973) reported the case of a patient with a dense left homonymous hemianopia who exhibited continuous, smooth eye movements approximately 15 to 20 degrees into the affected field. These movements were about 1.6 seconds in duration, followed by a quicker movement returning to the previous fixation. The patient was unaware of the field defect and the movements. As noted earlier, visual field cuts may be correlated with VHN (Chedru et al., 1973).

Continuing to follow the association between unilateral brain-damage and VHN, there are also two studies that indicate that accuracy of eye movements may be negatively affected (Gassel & Williams, 1963; Karpov et al., 1968). Karpov et al. (1968) studied the gaze behavior of a 42 year old male with parietal-occipital brain disease who had a left sided hemianopia. The patient was unable to shift his direction of gaze accurately from one point to another located to the left of the first point, even though both targets were close enough to be simultaneously visible to him. Therefore, the field cut could not account for this dysmetria. Gassel and Williams (1963) studied the OM

performance of 35 patients with homonymous hemianopia. Seventeen of the patients demonstrated a tonic oculomotor imbalance with deviation of the eyes to the hemianopic side. Six patients had some difficulty on a line tracing task and in jumping to each corner of a square. Twenty-one patients had relatively irregular and inaccurate movements to command to the hemianopic side. The authors mentioned that the worst performance was obtained in patients who had, among other disturbances, evidence of "some complex perceptual disorder such as ignoring the left half of space" (p.35).

Finally, the Saccadic Latency of unilaterally brain-damaged subjects has also been shown to be abnormal, and to vary according to laterality of lesion (Cohn, 1972; Sundqvist, 1978; Van Allen, 1973). Van Allen studied 13 patients' responses to a 500 Hz. tone presented approximately 2.2 seconds after a higher warning tone. In response to the stimulus tone subjects were to look 40 degrees to the right or left to a target light which came on concurrently with the warning tone. Brain-damaged subjects were 45 percent slower, on the average, than a group of nonbrain-damaged hospitalized control patients. Rightward saccadic latencies of brain-damaged patients were significantly slower than left-going movements, regardless

of hemisphere involved. In response to 10 degree left and right peripheral visual stimuli, Sundqvist (1978) found saccadic latencies for responses to the hemisphere opposite the side of lesion were significantly longer than those to the same side of the lesion. Two patients were right hemisphere damaged, four were left hemisphere damaged. Considering saccadic latencies to the same side of the lesion, two-thirds of the patients were still slower than one standard deviation below the mean saccadic latency of a group of normal control subjects. In a number of cases of homonymous hemianopia Cohn (1972) observed that while staring straight ahead, if double simultaneous visual stimuli were presented bitemporally, patients would deviate their gaze toward the normal field of vision. The movement itself generally took about 300 msec. to complete. Less definitive findings occurred in cases where the homonymous defect was for the right half field, with repeated testing, and as the duration of the lesion became more chronic.

These three areas of research suggest that patients with VHN would have difficulty with maintaining gaze at a specified target (Fixation Stability), with quickly moving their eyes laterally in response to visual information (Saccadic Latency), and with accurately moving their eyes to specific locations (Saccadic Accuracy). Furthermore, it

is the author's contention that the number and severity of these deficits is related to the severity of VHN to the extent that even mild cases of VHN will perform abnormally on at least one of these parameters. If such a result should be forthcoming, then the Chedru et al. (1973) finding of normal ocular exploratory behavior in patients with mild VHN would have to be qualified, and speculations based on their data would have to be modified. In order to provide a check (i.e., a replication of sorts) on the Chedru et al. (1973) finding that is being questioned, the present investigation also included a stimulus task that enabled the measure they term Exploration Time to be taken.

## CHAPTER IV

### SPATIAL FIELD EFFECTS

This section provides the background and rationale for studying OM behavior in independent spatial fields. Just as little in general is known about OM behavior in people who have VHN, this author is not aware of any study that has addressed the relationship between OM behavior and spatial field in those who have VHN. The knowledge gained from such a study could serve to support, or refute a recently proposed theory that has been tendered by Heilman (1979) to explain the finding of spatial field effects in extremity-motor hemi-neglect research (e.g., Watson, Miller, & Heilman, 1978; Heilman & Valenstein, 1979b). The discussion that follows will briefly review the various postulated mechanisms of hemi-neglect, with particular emphasis on spatial field effects and Heilman's theory.

A variety of mechanisms have been suggested to explain the bases for VHN symptomatology. A review of studies in the area suggests that no researcher believes a single mechanism could be responsible for all the manifestations of VHN (e.g., Critchley, 1966; Mesulam, 1981; Heilman, Note 2). Furthermore, it is becoming apparent that lesions in fairly localized areas of the brain may produce neglect phenomena for a number of different reasons, many of which

traditionally have not been given much credence. A quote from a recent excellent treatise (Lynch, 1980, p. 485) illustrates this trend in thinking for the brain region most often associated with VHN: "...it is proposed that the sensory association model of posterior parietal function is inadequate to account for the complexities of the present evidence. Instead, it now appears that many diverse neural mechanisms are located in part in parietal cortex, that some of these mechanisms are involved in sensory processing and perceptual functions, but that others participate in motor control, and that still others are involved in attentional, motivational, or emotional processes."

Furthermore, a recent investigation of right hemisphere damage and subsequent deficits found that not only did spatial neglect occur with lesions in a variety of locations, but that in contrast to widely held beliefs, this symptom was found more often with frontal and central lesions than with parietal involvement (Kertesz & Dobrowolski, 1981; 1982). It is clear there is yet much controversy over the structural and functional substrates for VHN.

Thus, different investigators have placed different degrees of emphasis on sensory-perceptual, spatial-motoric, and attention-arousal hypotheses in accounting for hemi-

neglect. Brain (1941), Critchley (1966), and more recently Heilman and Watson (1977a), have viewed neglect as primarily an attention-arousal disorder. The latter group suggested this to be an alerting or orienting defect produced by a lesion in the cortico-limbic-reticular loop. Accordingly, damage to either cortical or subcortical structures could produce the syndrome. Interhemispheric imbalance has been variously considered to play an important role. Heilman and Watson (1977b) suggest that the cerebral hemispheres are bilaterally hypoaroused with the lesioned side being the most affected. Kinsbourne (1972), on the other hand, believes this imbalance may consist of a more active than normal non-lesioned hemisphere resulting from lack of inhibitory influence from the lesioned hemisphere.

Various levels of sensory-perceptual explanations have also been offered. Battersby, Bender, Pollack and Kahn (1956) considered decreased sensory input superimposed on generally lowered cognitive function to be responsible for neglect behavior. Denny-Brown and Banker (1954) applied the term amorphosynthesis, or disturbance in the spatial summation of multiple sensory stimuli in explaining neglect phenomena. Eidelberg and Schwartz (1971) proposed that quantitatively asymmetrical afferent (sensory) input to the

cerebral hemispheres was the root of the problem (in essence an inter-hemispheric imbalance theory).

A relatively new theory, and a primary focus of the present research, is the interesting spatial, or frame of reference mechanism posited by Chedru et al. (1973), and developed by Heilman (1979), and Heilman and Valenstein (1979b). The latter group notes that performance on various tasks is improved by moving the task entirely into the hemispatial field ipsilateral to the side of the lesion. They suggest an akinesia occurs for any task which must be performed in the contralesional spatial hemifield.

Heilman's (1979) interpretation of "stimulus response compatibility" research emphasizes the spatial aspect of the spatial akinesia phenomenon discussed above. Stimulus response compatibility is a term that, in standard usage, refers to an interesting finding in reaction time studies in normals (Anzola, Bertolini, Buchtel, & Rizzolatti, 1977). Namely, responses to laterally presented stimuli are fastest for the hand that is in the same lateral field, regardless of whether one is testing crossed, or uncrossed hands. According to Heilman (1979) two explanations have been tendered to account for this. One hypothesis is that this reflects a "natural tendency" and therefore somehow a better response with the hand already in the hemispatial

field containing the stimulus (Craft & Simon, 1970). The alternative explanation offered is that, in addition to various other lateralized functions, each hemisphere is responsible for the general mediation of behavior occurring in the contralateral hemispatial field (Heilman, 1979).

Recently, Bowers and Heilman (1980) undertook to determine the extent to which a hemispace-based model could account for similar effects observed on a tactile line bisection task. In this study a group of normal dextrals had to find the middle of a number of sticks of various lengths using only the sense of touch. The experimental design consisted of a 2X3 matrix of conditions, i.e., the right or left hand was used in each of three spatial conditions; stimuli were presented at body midline, to the space left of midline, and to the space right of midline. Regardless of which hand was used, the task was performed most accurately in the left hemispace. Thus, there was no significant main effect for hand alone, but there was for spatial field. This finding was replicated by Bruder (Note 3).

The spatial field effect has also been studied in brain-damaged animals and humans. In an animal study, Watson et al. (1978) trained monkeys to move the extremity contralateral to a stimulus, then unilaterally lesioned one

hemisphere of the animals' brains. Following the operation, the animals were determined to have normal strength in all extremities, and apparently normal sensation. When stimulated on the side contralateral to the lesion, they performed normally (as trained) with the ipsilesional arm. However, when stimulated on the ipsilesional side they did not use the contralateral arm (as trained). The authors suggested that the akinesia observed was related to the mediation of any acts that might be performed in the contralesional hemispace, rather than due to the use of the contralesional extremity per se.

To test this hypothesis in man, a group of patients with left-sided neglect was evaluated on a graphomotor line bisection task with stimuli presented in the left, middle, and right spatial fields (Heilman & Valenstein, 1979b). As a control for the possibility of a left-sided visual sensory defect confounding performance due to patients not seeing the entire line, letters were placed at the ends of each line and the patients had to read the letters before doing the task. Despite this precaution, performance was found to be significantly better for lines placed in the right spatial field compared to the left.

These data, in conjunction with what is known about OM behavior in people with VHN, led the present author to

speculate that OM performance may also be poorer in the spatial field contralateral to a brain lesion. Furthermore, if OM performance is thus affected, an argument may be made that certain of the extremity motor findings are influenced by inadequate visual sensory information. In order to study the relationship between OM behavior and spatial field, all fundamental OM tasks (those which provide measures of fixation stability, and saccadic latency and accuracy) were presented in each of three spatial fields: the central, left, and right.

## CHAPTER V

### RESEARCH DESIGN

This chapter presents an overview of the purpose and rationale of the present investigation, details of the research design, and the specific hypotheses addressed.

The general goal of this study was to further examine the Chedru et al. (1973) finding that ocular Exploration Time is abnormal for patients with marked VHN, but not for those with mild VHN, when compared to a neurologically normal group. To accomplish this, the OM behavior of these three groups was investigated for performance on four measures of ocular activity: (1) Exploration Time, (2) Fixation Stability, (3) Saccadic Latency, and (4) Saccadic Accuracy (detailed operational definitions of terms are given in the Methods section of this chapter). The rationale for this approach was derived from a confluence of research indicating that (a) employing a gross measure to demonstrate differences in the performance of individuals with varying degrees of deficit in an area of complex neuropsychological functioning is apt to fail, while using a breakdown of the components thought to make up the gross measure is apt to succeed (after Johnston & Pirozzolo, 1981; Luria, 1973), and (b) Exploration Time (as used by

Chedru et al., 1973) is a gross measure, and may be broken down into the time bound parameters of eye movement and eye fixation activity noted above, i.e., measures "2", "3", and "4" (after Russo, 1978).

Although visual field status is not necessarily associated with VHN, in their study of OM behavior and hemi-neglect Chedru et al. (1973) found significant differences in OM performance between groups of brain-damaged subjects based on presence of visual field defects (but without concurrently considering the severity of VHN). Therefore, in the present study visual field defects were measured in order to determine their effect, if any, on the dependent variables. Integrity of the visual fields was measured using Amsler's Grid (Note 4) for central field assessment, and confrontation testing (Forster, 1973) for peripheral field assessment. Confrontation testing was performed for single and double simultaneous stimuli presented randomly, three times in each visual quadrant, according to the method of Forster (1973). This method has the examiner sit facing the subject, approximately two feet away, and extend both arms in the upper or lower peripheral fields. As the subject fixates the examiner's nose, s/he is required to indicate which of the examiner's hands have wriggling fingers. Although this is a rather imprecise

measure that has been shown to miss less than gross visual field deficits (Trobe, Acosta, Krischer, & Trick, 1980), limitations in available resources obviated the use of more exacting measures.

In order to further characterize the OM performance of patients with VHN, and to enhance our understanding of the underlying mechanisms of VHN, the present study also investigated the effect placing tasks in different spatial fields has on OM performance. This procedure was followed for the three components of Exploration Time listed above. The rationale for this approach was derived from Heilman's (1979) notion that neglect arises from unilateral brain-damage which interferes with the mediation of sensory and/or motor activity occurring in the contralateral spatial field.

Thus, the present study was designed to test (using non-directional statistical procedures) the following specific null hypotheses (Ho):

Ho 1 - Regarding severity of VHN and OM performance--

(A) No differences will be found for OM performance

(based on measures of Fixation Stability, Saccadic Latency, Saccadic Accuracy, and Exploration Time) between a Marked VHN group, a Mild VHN group, and a Normal Control group.

The alternative hypothesis is that differences will be found such that the Marked VHN group will perform significantly worse than the Mild VHN group and the Normal Control group, and that performance by the Mild VHN group on the above stated OM measures will be between the other two groups, i.e., worse than the Normal Control group but better than the Marked VHN group.

Based on the arguments advanced earlier, it is predicted that  $H_0 I(A)$  will be rejected for each group comparison of performance on each OM variable except for Exploration Time between the Mild VHN group and the Normal Control group; in this one case  $H_0 I(A)$  will not be rejected.

- (B) The presence of visual field defects will not significantly affect performance on OM variables beyond the effects that may be attributed to severity of VHN.

The alternative hypothesis is that the presence of visual field deficits will adversely affect performance on the OM variables, in addition to effects shown to be related to severity of VHN. Based on the preceding arguments, it is predicted that  $H_0 I(B)$  will not be rejected.

Ho 11 - Regarding OM performance by spatial field--

No differences will be found for OM performance (based on measures of Fixation Stability, Saccadic Latency, and Saccadic Accuracy) between the left, center, and right spatial fields across the three groups (Marked VHN, Mild VHN, and Normal Control). The alternative hypothesis is that differences will be found such that performance will be worst in the left spatial field compared to either of the other two fields for the Marked and Mild VHN groups, but best in the left spatial field compared to either of the other two fields for the Normal Control group.

Based on the preceding arguments, it is predicted that Ho 11 will be rejected.

### Subjects

Three groups of subjects were studied consisting of a total of 20 neurologically impaired right hemisphere stroke victims undergoing rehabilitation, and 10 normal controls. The stroke victims were dichotomized on the basis of severity of VHN (discussed below). All neurologically impaired subjects were obtained from the New York University Institute of Rehabilitation Medicine, and Goldwater Memorial

Hospital. The normal control group was obtained from volunteer members of special interest organizations at Northshore University Hospital, and staff members of the Department of Psychology at Queens College. The neurologically impaired subjects consisted of individuals who had suffered a right hemisphere (unilateral) stroke at least four weeks prior to their participation in the present study. The four week interval was required because of the general lack of symptom stability during the first one to three weeks following cerebral injury (Kertesz, 1979). In order to limit variability effects produced by age, and because of the limited age group in which strokes (and hence, VHN) tend to occur, only subjects between 50 and 80 years of age were included. Because of the paucity of subjects with VHN available to the investigator sex, ethnic background, education, and socioeconomic status could not be readily controlled. However, an attempt was made to obtain equal ratios of males to females for the normal control group compared to the brain-damaged group as a whole, and data regarding the other demographic variables were collected in order to determine their effect, if any, on the dependent variables.

Visual field and visual acuity testing were performed on each subject, but only the latter of the two was used to

screen subjects for participation in the present study. The examination for visual field defects was described above. Visual acuity was required to be at least 14/84 (American Optical standard reading distance rating expressed in inches), without correction. This is equivalent to being able to indicate the orientation of the symbol "W" (actual size), or to read 18 point size print (similar in size to the above symbol). Although reading distance acuity technically should not be converted to the commonly used measure made at the distance of twenty feet, the above criterion is roughly comparable to 20/100 using this more familiar distance. This apparently lax criterion was necessary since there is a higher incidence of presbyopia in the aged, and because the eye monitoring apparatus did not permit the use of corrective lenses. Even though these constraints necessitated accepting subjects with the above noted acuity, a number of subjects were rejected using this cut-off.

Two subtests of the Wechsler Adult Intelligence Scale (WAIS) were used to establish the basal level of verbal understanding and memory ability required of the subjects. These were the Vocabulary and Digit Span subtests. In order to be included in the study each subject had to obtain a score equal to or better than one standard devia-

tion below average for age (equivalent to a scaled score of at least 7). A prorated Digit Span scaled score was used for the memory test since it is known that right hemisphere brain-damage may selectively affect the digits reverse portion of this test, and that this deficit is probably not one of verbal memory per se (Lezak, 1976). Normally, a person is able to repeat one digit less in reverse than in the forward direction (Lezak, 1976). Therefore, to obtain an age scaled score of memory ability the expected digits reverse value corresponding to the individual's digits forward score was used, rather than the obtained value.

Unilaterality of lesion was established by satisfaction of a number of criteria. With the qualification just mentioned, the WAIS Vocabulary and Digit Span subtests are relatively insensitive to right hemisphere brain-damage, and relatively sensitive to left hemisphere brain-damage (Lezak, 1976). Thus, they provide a neuropsychological check on the laterality of the lesion, especially when compared to WAIS subtests considered to bear the opposite relationship. For this reason the WAIS Block Design subtest was also given. This subtest is the single best measure from the WAIS of visuo-spatial (right hemisphere) functioning (Lezak, 1976). No neurologically impaired subject was included who had a Block Design scaled score

five points higher than that obtained on either of the other two WAIS subtests. This criterion was based on what is considered to constitute significant subtest scatter (see Matarazzo, 1976). Additional evidence of unilateral-ity was obtained from records of neurological examinations and laboratory test reports (e.g., CT scans). Subjects who may possibly have suffered bilateral or diffuse lesions were excluded.

Unstructured interview, observation, and (in the case of patients) review of available records were used to determine that the following additional criteria were met by all subjects:

- (1) No subject could have a history of previous cerebral neurological disease.
- (2) No subject could have a history of debilitating psychiatric illness or drug abuse of any kind.
- (3) No subject could have strabismus.

### Methods

Once the subject had undergone the above evaluation and met the necessary criteria for inclusion in the study, the tests for determining the severity of VHN were administered. These tests, plus the above evaluation, lasted from two to three hours, and were completed in two to three

sessions. A final, separate session was required for administering the OM tests. That session lasted approximately one hour.

Presence and severity of VHN was assessed with the short form of Piasetsky's (1981) test, and two letter cancellation tests (Diller et al., 1974). Performance on these tests was summed to yield an index of VHN such that the higher (the worse) the score, the greater the magnitude of VHN. Equal weight was given to each type of test by converting raw scores to scaled scores. Based on previous work (Diller et al., 1974; Piasetsky, 1981), it was expected that this method would produce an overlap of approximately the highest 20% of the normal controls (the worst scores for this group) with approximately the lowest 20% of the neurologically impaired (the best scores for this group). Score distribution was expected to be approximately normal. No normal control subject was expected to obtain a score at or above the mean of the neurologically impaired subjects. By using a median split, the marked VHN group was formed from the 10 worst scoring neurologically impaired subjects (the upper half of the distribution). The mild VHN group consisted of the 10 best scoring neurologically impaired subjects (the lower half of the distribution). See Chapter VI - Results for additional details

on the calculation of VHN scores.

A non-invasive photo electric technique was used to monitor OM behavior (Gulf & Western, Note 5). The apparatus used was a limbus tracking device worn by the subject, and consisted of an infrared light source and photodiodes mounted on a pair of spectacle frames (without lenses). Nothing touched the eyes. Resolution of this apparatus is  $\pm 0.25$  degrees in the horizontal axis, and  $\pm 0.5$  degrees in the vertical axis. In accordance with standard application of this apparatus (Johnston & Hakerem, 1982), and due to squint and blink artifacts obtained from the vertical recording channel (Johnston & Hutton, 1979), only data from the horizontal channel were used to provide values for the OM parameters of interest.

Visual stimuli for OM testing were presented via a head mounted scene holder (Johnston & Hutton, 1979) worn by the subject. This apparatus consists of a lightweight headgear (680 grams) to which stimulus scenes are attached. The scenes themselves were mounted on lightweight artist's tagboard. Onset of scene exposure was controlled by removal of a drop cloth suspended immediately in front of the subject's eyes. Removal of the drop cloth completed a photo-cell circuit, thus providing a marker pulse which coincided with scene exposure. Mounted in the scene holder

were tri-color light emitting diodes (LEDs) used for the purpose of OM reaction time (Saccadic Latency). The fixation LED was always yellow. When it was turned to red or to green an LED located 8 degrees to the right or left simultaneously came on in the same color as the fixation LED in order to provide an actual visual target. The investigator controlled both the color and the location of the light stimuli. This enabled the possibility of providing a choice reaction time paradigm simply by informing the subject of the relationship (e.g, if the fixation light turns red look right, if it turns green look left). A square wave pulse output was provided to mark the exact time of color change.

All signals were recorded on a seven channel FM instrumentation tape recorder (Ampex FR-1300) for off-line analysis. The tape recorded data were played back in real time through direct current driver-amplifiers (Grass model 5E) interfaced to a multi-channel ink writing oscillograph (Grass model 5DWC 12 PA) for hand measurement of the various signals. Chart speeds used were 6 and 60 mm./sec. The latter speed was used only for saccadic latency measures, providing a resolution of 16.7 msec./mm.

Four separate OM tasks were administered. These tasks provided measures of Fixation Stability, Saccadic Latency,

Saccadic Accuracy, and Exploration Time. The tasks were presented in the following order for all subjects:

- (1) First block of Fixation Stability task
- (2) Exploration Time task
- (3) Saccadic Latency task
- (4) Saccadic Accuracy task
- (5) Second block of Fixation Stability task

Fixation Stability stimuli consisted of a 3 X 3 grid of 1 cm. sized crosses which were also used for calibration of the eye monitoring apparatus. The subject was instructed to fixate the center of each cross for a period of 10 seconds, and to attempt to maintain gaze at the center of the cross as steadily as possible. Subjects were instructed to point to each cross to ensure proper localization prior to beginning an individual trial. Grid positions were numbered consecutively from left to right, from 1 to 9 (the numbers did not appear on the actual scene). Position 5 was always tested first, randomly followed by the order 2, 8, or 8, 2. These positions were randomly followed by the order 4, 6, 1, 3, 7, 9, or 6, 4, 3, 1, 9, 7. Thus, all points in the central spatial field were always tested first, followed by alternating positions in the left and right spatial fields, sometimes beginning on the left, sometimes beginning on the right. The entire

fixation stability procedure was done once at the beginning of the OM session, and in the same order at the end of the OM session. This procedure was followed primarily for convenience, since the same scene was used for equipment calibration at the beginning and end of the recording session. Each 10 second epoch of analog eye position data from the strip chart recorder was traced with a digital plotter (an electronic stylus used to measure distance). This yielded a single value for each epoch indicating the summed extent of eye movement for that epoch. The greater the extent of eye movement, the poorer was the subject's performance considered to be. To summarize the previously stated hypotheses being tested, it was predicted that severity of VHN would be positively correlated with poorness of Fixation Stability performance, and that such performance would also vary by spatial field.

Saccadic Latency was obtained using the tri-color LEDs discussed above. The paradigm was choice reaction time, randomized between subjects for the relationship of color to direction of eye movement. That is, for a given subject red always meant look to the left, green to the right; the reverse being true for a different subject. Stimulation was always foveal, negating certain problems thus caused by using a peripheral stimulus in subjects with a peripheral

field sensory defect. A verbal warning signal of "Ready" preceded each stimulus. A stimulus was not triggered until the subject was observed to be fixating the fixation light, and not blinking. The stimulus order (left vs. right) was randomized for eight trials per direction per block, with three blocks administered. The first block was always performed in the central spatial field, the remaining two blocks randomized among subjects for left spatial field or right spatial field occurring first. In the lateral spatial field conditions the fixation light was 8 degrees to the left or right of the cephalo-sagittal midline. Extra stimuli were provided at the end of each block to replace known artifactual responses occurring during the original block. A minimum of four "practice trials" were given in order to explain the procedure. Pilot work had shown that despite the apparent simplicity of the task, some subjects need more examples than others in order to understand exactly what is required of them. Understanding was considered present when the subject was able not only to repeat the task demands, but also actually to perform the task at least twice in each direction. The Saccadic Latency measurement was taken by hand from the strip chart recording obtained off-line. This was done by measuring the distance between the stimulus marker pulse and the

corresponding eye movement, then converting this value using the appropriate time scale (i.e., 1 mm = .0167 sec.). The longer the latency, the worse the performance was considered to be. The same predictions applied for this measure as for Fixation Stability.

Saccadic Accuracy was obtained from a stimulus used by this investigator and others on a variety of brain-damaged populations (Gassel & Williams, 1963; Hutton, Johnston, Shapiro, & Pirozzolo, 1979). The stimulus consisted of a square, with two dots placed peripherally in the horizontal meridian, one to either side of the square. The scene was exposed twice in each spatial field. On the first exposure the subject was told, "Trace the square from corner to corner as quickly and as accurately as you can until I say stop." On the second exposure the subject was told, "Look back and forth from dot to dot as quickly and as accurately as you can until I say stop." Exposure time in both cases was 10 seconds. The task was presented in the central spatial field first, and randomized among subjects for whether it next occurred in the left or right spatial field. The accuracy score was obtained by counting the number of saccades (from the strip chart recording) necessary to fixate each dot before moving to the next one. The greater the number of saccades, the poorer was a sub-

ject's performance considered to be. The purpose of the square was to serve as an interference in the "ground" of the figure. This method has been shown to elicit abnormal OM behavior in brain-damaged populations, while simply looking between two points may not (Hutton et al., 1979). The same predictions applied for the measure Saccadic Accuracy as for the above OM measures.

Exploration Time was obtained using a modified letter cancellation task (Diller et al., 1974). An eight line by 26 letter array of capital letters (24 point size) was presented with the task being to identify all the letter "Rs" that could be found (there were 50). Identification was indicated by means of pressing a hand held button which provided a marker pulse that was tape recorded concurrent with the eye monitor signals. The measure of Exploration Time was a proportional one calculated according to the method of Chedru et al. (1973), i.e., by determining the percentage of time spent exploring the left half of the stimulus scene. This value was obtained only for performance with the task in the central spatial field. This was done because of the skewing which occurs with this particular eye monitoring apparatus for signals located further and further in the periphery (Johnston & Hutton, 1980). One trial was considered sufficient based on the stability

of repeat performances on this task, even as long as four weeks after the first administration (Hibbard, Note 6). The same predictions applied to this as the other measures, however; spatial field effects were not (could not be) tested.

#### Data Analyses

The present study incorporated three separate groups, four separate OM measures, and three separate fields for three of the four OM measures. Initially, the OM data were collapsed across spatial field and analysis of variance (ANOVA) performed for pooled data. This was followed by a repeated measures ANOVA that included spatial field as a primary factor. Newman-Kuels tests were done to make pairwise group comparisons. To examine the degree of relationship between severity of VHN and the various independent or control variables (e.g., visual acuity, age) correlational procedures were applied to the data. ANOVA with covariance adjustments analyses were performed on the OM data to reveal the influence of visual field defect on OM performance.

## CHAPTER VI

### RESULTS

In general, hypotheses put forward regarding severity of VHN and OM performance were supported; those regarding OM performance broken down into spatial fields were not. Before presenting a detailed description of those results, analyses regarding demographic, neurologic, psychometric, and VHN data will be presented.

#### VHN Scores: Marked, Mild, Normal Control Groups

A VHN score was calculated for each subject according to the following formula:

VHN Score =  $(2.916 A + B + C) / 280$ , where

2.916 = Weighting factor,

A = Bias from Piassetzky's Matching Test,

B = Bias from Cancellation H,

C = Bias from Cancellation C & E, and

280 = Divisor to reduce the dividend to a value between 0.00 and 1.00.

Thus, the VHN score may range from .00 to 1.00 with a score of .50 signifying no neglect (no bias). As scores increase in magnitude (from .50), the more the subject neglects the left (i.e., favors or shows a bias for the right), and vice

versa as scores decrease in magnitude.

As noted above, and described in Chapter V - Research Design, three tests were used to contribute to the VHN score. On each test the extent to which a subject responds to stimuli on the right versus those on the left is the variable of interest, and this is what is meant by bias. Based on the above formula, a subject who chooses more selections on the right side than the left in the matching test, and who finds more letters on the right than the left in the cancellation tests would have a strong bias for the right (neglect for the left), and the calculated VHN score would clearly be above .50.

The two general types of tests (i.e., matching and cancellation) were given equal weights in determining the VHN score. In other words, 1/2 of an individual's VHN score was determined by performance on the matching test, and 1/4 by performance on each of the two cancellation tests. Although there is evidence that the matching test may be a better measure of VHN than are cancellation tests (Piasefsky, 1981), equal weight was given each type of test because of the more common use of the latter. To arrive at values for A, B, and C, raw bias scores for each test were converted to positive values by adding a constant for that particular test. Since raw bias scores for the matching

test could range from -24 to +24, the constant for conversion of that test score was 24, thereby converting the range to 0 to 48. Raw bias scores on each of the cancellation tests could range from -35 to +35, therefore, the constant for those tests was 35, changing the range to 0 to 70. The weighting factor of 2.916 (rather than just "2") was applied to the matching test because the possible range of converted raw scores was less on the matching test than on the cancellation tests.

Consider the following example. For the values A, B, and C, the worst scores a subject could obtain would be 48, 70, and 70, respectively. By giving equal weight to the matching test as to the sum of the cancellation tests (i.e., by multiplying 48 by 2.916) the obtained matching score is 140. The grand total is then 280. For convenience of score reporting, this value is divided by 280 to obtain a VHN score of 1.00, indicating the most marked left hemi-neglect possible for these visual tasks.

Figures 1 and 2 present the distribution of VHN scores thus calculated. The distribution for all subjects combined is slightly positively skewed, representing the effect of twice as many brain-lesioned subjects as normal controls. All brain-lesioned subjects obtained a VHN score greater than .50, indicating some degree of left hemi-

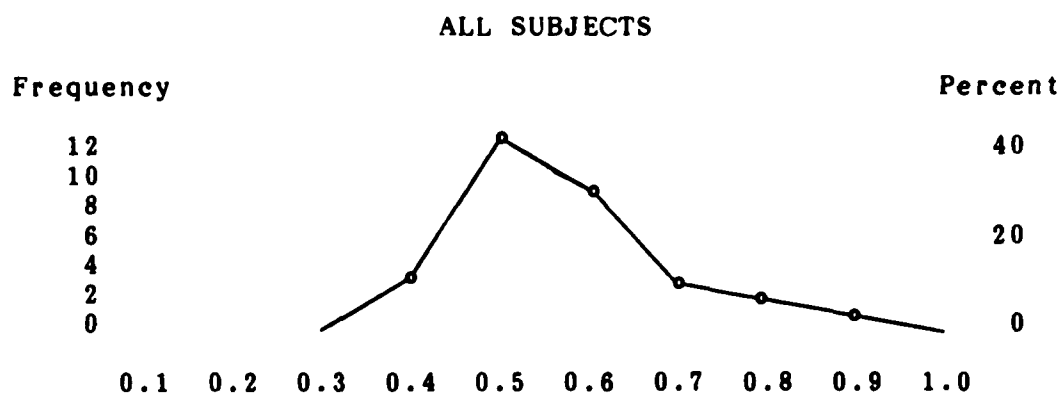


**Figure 1. Distribution of VHN scores.**  
Circles identify interval midpoints.

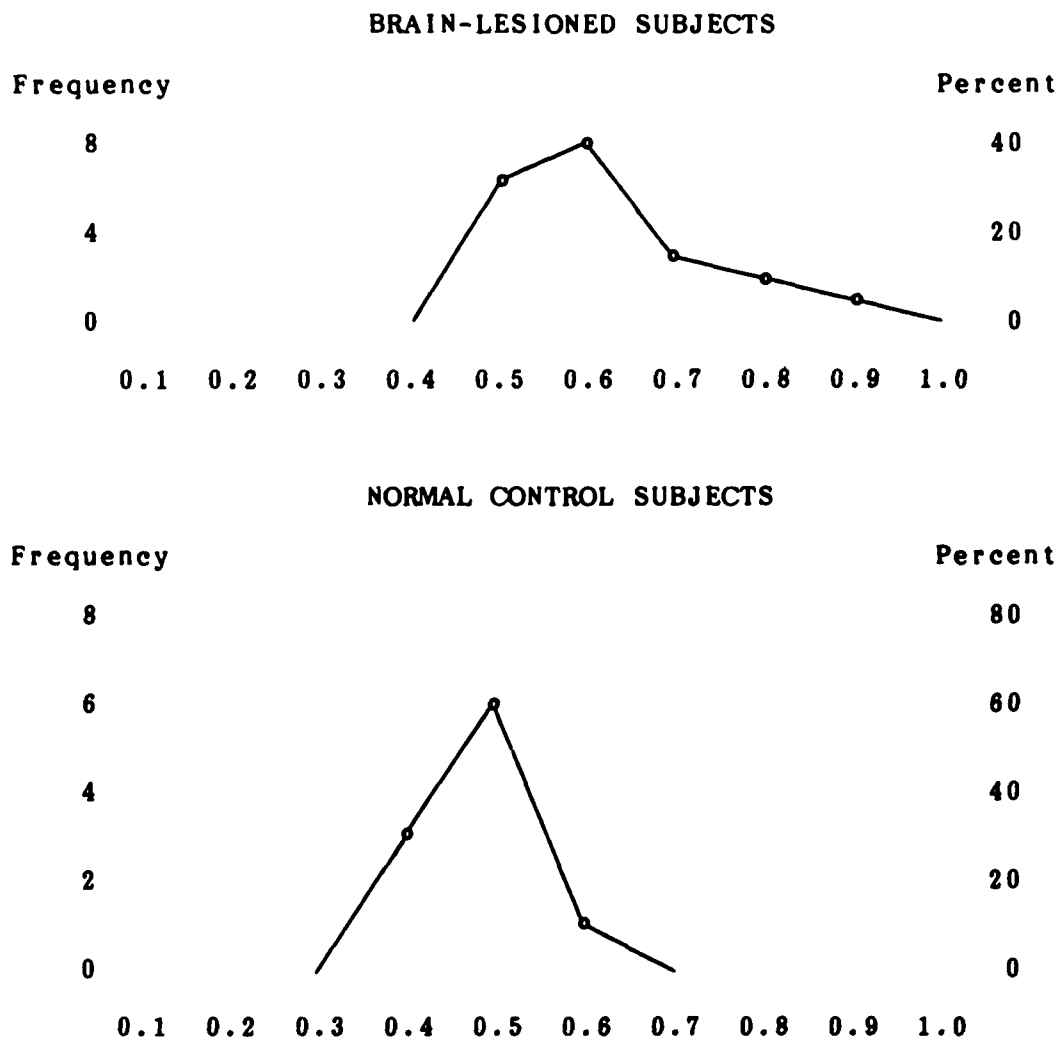


Figure 2. Distribution of VHN scores by subgroup. Circles identify interval midpoints.

neglect for each subject. Scores for the normal control group cluster closely around .50, indicating no neglect or bias in either direction for the group as a whole. No control subject obtained a VHN score equal to or greater than the mean VHN score for the brain-lesioned group as a whole, which was .667.

Although severity of VHN is clearly a continuous variable, in order to establish a Marked VHN group and a Mild VHN group a median split was performed based on the VHN scores of the neurologic subjects. The median value was .638. Each of the two groups thus created was equal in size to the Normal Control group ( $n=10$ ). Table 1 reveals that significant group differences were obtained for VHN scores when these data were subjected to a one-way analysis of variance (ANOVA  $p < .001$ ). The Marked group's mean VHN score was found to be significantly different from each of the other groups when Student-Newman-Keuls pairwise tests were performed ( $p < .05$ ). The group mean scores for the Mild and Normal Control groups were not significantly different from each other. Hereafter, "groups" will refer to the three groups: Marked VHN, Mild VHN, and Normal Control subjects.

TABLE 1

ANALYSIS OF GROUP (MARKED, MILD, NORMAL CONTROL)<sup>a</sup>  
 DIFFERENCES IN VHN SCORES: ONE-WAY ANALYSIS OF  
 VARIANCE AND STUDENT-NEWMAN-KEULS PROCEDURE

## Analysis of Variance

| SS              | MS              | df | F-ratio    |
|-----------------|-----------------|----|------------|
| .2748 (between) | .1374 (between) | 2  | 28.7486 ** |
| .1290 (within)  | .0048 (within)  | 27 |            |

## Student-Newman-Keuls

| Marked Group<br>$\bar{x}$ / s.d. | Mild Group<br>$\bar{x}$ / s.d. | Normal Control<br>$\bar{x}$ / s.d. |
|----------------------------------|--------------------------------|------------------------------------|
| .7494/.1026                      | .5827/.0414                    | .5233/.0459                        |
|                                  |                                |                                    |

<sup>a</sup>n=10 per group, \* p < .05, \*\* p < .001, n.s. = not significant

### Analyses of Demographic and Psychometric Data

A series of one-way analyses of variance were performed to determine if overall group differences existed for demographic variables, and for performance on psychometric tests. Table 2 presents descriptive statistics for each of these variables, and Table 3 presents the ANOVA results. No significant group differences were found for age, visual acuity, visual field defect, or occupation. Data from Amsler's Grid are not presented since no variability was demonstrated in central visual fields using this test; all subjects reported uncompromised foveal vision. Significant group differences were found for education ( $p < .05$ ), and on all three subtests administered from the WAIS: Digit Span ( $p < .05$ ), Vocabulary ( $p < .01$ ), and Block Design ( $p < .001$ ).

In order to determine the extent of the relationship of VHN scores to demographic variables, and to performance on psychometric tests, the data were subjected to Pearson Product Moment correlational analyses. The purpose of these analyses was not to differentiate Marked from Mild groups but rather to examine performance on a continuum. Therefore, the brain-lesioned group was not subdivided. It was expected that the presence (or absence) of brain-damage would be substantially reflected in certain correlations

TABLE 2  
DESCRIPTIVE STATISTICS FOR GROUP  
(MARKED, MILD, NORMAL CONTROL)<sup>a</sup> PERFORMANCE  
ON DEMOGRAPHIC AND PSYCHOMETRIC VARIABLES

| Variable               | Marked Group<br>$\bar{x}$ / s.d. | Mild Group<br>$\bar{x}$ / s.d. | Normal Control<br>$\bar{x}$ / s.d. |
|------------------------|----------------------------------|--------------------------------|------------------------------------|
| Age <sup>b</sup>       | 65.3 / 9.6                       | 69.6 / 5.7                     | 64.6 / 7.9                         |
| Educ. <sup>b</sup>     | 13.3 / 3.9                       | 10.6 / 3.0                     | 14.3 / 1.8                         |
| Occup. <sup>c</sup>    | 4.1 / 1.9                        | 5.2 / 1.1                      | 3.9 / 0.6                          |
| Acuity <sup>d</sup>    | 4.0 / 1.7                        | 3.2 / 1.1                      | 3.0 / 1.5                          |
| VF Defect <sup>e</sup> | 2.0 / 2.7                        | 1.0 / 2.0                      | 0.0 / 0.0                          |
| WAIS DS <sup>f</sup>   | 10.8 / 2.6                       | 11.7 / 3.3                     | 14.4 / 2.1                         |
| WAIS Voc. <sup>f</sup> | 11.7 / 2.2                       | 10.9 / 2.5                     | 14.0 / 1.4                         |
| WAIS BD <sup>f</sup>   | 5.3 / 3.4                        | 5.9 / 3.6                      | 11.2 / 2.4                         |

<sup>a</sup>n=10 per group, <sup>b</sup>expressed as years, <sup>c</sup>expressed as position on a seven point scale (from 1=executives and major professionals to 7=unskilled workers), <sup>d</sup>expressed as position on American Optical ratio scale (as value increases acuity decreases), <sup>e</sup>expressed severity on a six point scale for responses to confrontation testing (from 0=no impairment to 6=dense visual sensory deficit), <sup>f</sup>expressed as age scaled score

TABLE 3

ANALYSES OF GROUP (MARKED, MILD, NORMAL CONTROL)  
DIFFERENCES FOR PSYCHOMETRIC AND DEMOGRAPHIC  
VARIABLES: ONE-WAY ANALYSES OF VARIANCE

| Analyses of Variance |         |     |        |     |    |           |
|----------------------|---------|-----|--------|-----|----|-----------|
| Variable             | SS      |     | MS     |     | df | F-ratio   |
| Age                  | 146.47  | (b) | 73.23  | (b) | 2  | 1.18 n.s. |
|                      | 1680.94 | (w) | 62.26  | (w) | 27 |           |
| Educ.                | 73.27   | (b) | 36.63  | (b) | 2  | 4.01 *    |
|                      | 246.60  | (w) | 9.13   | (w) | 27 |           |
| Occup.               | 9.80    | (b) | 4.90   | (b) | 2  | 2.79 n.s. |
|                      | 47.40   | (w) | 1.76   | (w) | 27 |           |
| Acuity               | 5.21    | (b) | 2.60   | (b) | 2  | 1.26 n.s. |
|                      | 55.69   | (w) | 2.06   | (w) | 27 |           |
| VF Defect            | 20.00   | (b) | 10.00  | (b) | 2  | 2.65 n.s. |
|                      | 102.00  | (w) | 3.78   | (w) | 27 |           |
| WAIS DS              | 70.20   | (b) | 35.10  | (b) | 2  | 4.74 *    |
|                      | 200.10  | (w) | 7.41   | (w) | 27 |           |
| WAIS Voc.            | 51.80   | (b) | 25.90  | (b) | 2  | 5.88 **   |
|                      | 119.00  | (w) | 4.41   | (w) | 27 |           |
| WAIS BD              | 210.87  | (b) | 105.43 | (b) | 2  | 10.52 *** |
|                      | 270.60  | (w) | 10.02  | (w) | 27 |           |

\*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ , b=between, w=within,  
n.s.=not significant

(e.g., visual field defect). Table 4 presents the results of those analyses with corresponding two-tailed t-tests for significance. While WAIS Digit Span and Block Design scores were correlated with VHN scores for all subjects combined ( $r = -.444$ ,  $p < .05$ ; and  $r = -.584$ ,  $p < .05$ ), neither the brain-lesioned subjects nor the control group alone yielded significant correlations on any of the demographic and psychometric variables.

#### Neurological Data for Brain-Lesioned Subjects

All 20 brain-lesioned subjects carried a diagnosis of right hemisphere cerebrovascular accident (CVA). Neurologic examination supported this diagnosis in all 20 cases. Table 5 presents data on the neurologic status of each case. All subjects manifested unilateral left hemiparesis or left hemiplegia. Thirteen manifested unilateral left-sided disturbances in tactile sensation. Nine demonstrated mild dysarthria. Review of records supported the diagnosis of CVA in 15 cases based on adjunctive CT scan, brain scan, or angiogram findings. At the time of this study the records of only nine cases contained detailed enough descriptions of laboratory findings that would allow some degree of localization and identification of the lesion in the right hemisphere. On the basis of laboratory findings

TABLE 4

PEARSON PRODUCT-MOMENT CORRELATIONS ( $r$ ) OF VHN SCORES WITH  
DEMOGRAPHIC AND PSYCHOMETRIC VARIABLES

|            | All Ss<br>VHN Score<br>(n=30) | Brain-lesioned Ss<br>VHN Score<br>(n=20) | Control Ss<br>VHN Score<br>(n=10) |
|------------|-------------------------------|------------------------------------------|-----------------------------------|
| Age        | -.084 n.s.                    | +.346 n.s.                               | -.352 n.s.                        |
| Education  | -.025 n.s.                    | +.368 n.s.                               | +.218 n.s.                        |
| Occupation | +.030 n.s.                    | -.129 n.s.                               | -.468 n.s.                        |
| Acuity     | +.181 n.s.                    | -.191 n.s.                               | +.146 n.s.                        |
| VF Defect  | +.084 n.s.                    | -.162 n.s.                               | .000 n.s.                         |
| WAIS DS    | -.444 *                       | -.386 n.s.                               | -.205 n.s.                        |
| WAIS Voc.  | -.286 n.s.                    | +.022 n.s.                               | +.020 n.s.                        |
| WAIS BD    | -.584 *                       | -.117 n.s.                               | -.364 n.s.                        |

Note: significance testing is based on two-tailed t-tests

\*  $p < .05$ , n.s.=not significant

TABLE 5

## NEUROLOGIC DATA OF RIGHT-HEMISPHERE BRAIN-LESIONED SUBJECTS

| Subject <sup>a</sup> | Type of CVA<br>(if known)        | Impairments on Neurologic Exam |       |            |
|----------------------|----------------------------------|--------------------------------|-------|------------|
|                      |                                  | Tactile                        | Motor | Dysarthria |
| P.B.                 | n.a.                             | no                             | yes   | yes        |
| R.Bi.                | n.a.                             | no                             | yes   | no         |
| R.Bo.                | n.a.                             | no                             | yes   | yes        |
| L.G.                 | n.a.                             | no                             | yes   | no         |
| M.G.                 | MCA aneurysm                     | yes                            | yes   | no         |
| J.M.                 | MCA infarct                      | yes                            | yes   | no         |
| S.R.                 | PCA aneurysm                     | yes                            | yes   | no         |
| R.S.                 | infarct basal<br>ganglia area    | no                             | yes   | no         |
| Y.S.                 | MCA infarct                      | yes                            | yes   | yes        |
| L.T.                 | n.a.                             | yes                            | yes   | yes        |
| L.B.                 | n.a.                             | no                             | yes   | yes        |
| T.B.                 | MCA infarct                      | yes                            | yes   | yes        |
| J.D.                 | infarct internal<br>capsule area | yes                            | yes   | no         |
| M.D.                 | MCA infarct                      | yes                            | yes   | no         |
| W.F.                 | n.a.                             | yes                            | yes   | no         |
| J.H.                 | n.a.                             | yes                            | yes   | no         |
| J.P.                 | n.a.                             | yes                            | yes   | yes        |
| R.R.                 | n.a.                             | yes                            | yes   | no         |
| T.S.                 | n.a.                             | yes                            | yes   | yes        |
| G.T.                 | infarct internal<br>capsule area | no                             | yes   | yes        |

<sup>a</sup>The first 10 Ss had mild VHN, the last 10 had marked VHN.

CVA = cerebral vascular accident

MCA = middle cerebral artery

PCA = posterior cerebral artery

n.a. = type of CVA is unknown

three subjects suffered right internal capsule or basal ganglia infarcts, four suffered infarcts in the distribution of the right middle cerebral artery, one had a hemorrhage due to an aneurysm in the right posterior cerebral artery, and one suffered a hemorrhage due to an aneurysm in the right middle cerebral artery. These data were not statistically analyzed due to the small number of subjects in each group.

#### OM Performance For Data Collapsed Across Spatial Fields

Repeated measures analyses of variance tests were applied to each OM variable under study to determine the existence of group differences in performance. The expectation was that the Marked VHN group would perform the worst, the Normal Control group would perform the best, and the Mild VHN group would perform better than the former but worse than the latter group on all OM variables except Exploration Time. For this dependent measure it was expected that no difference would be found between the Mild and Normal Control groups. Table 6 presents the results of those tests (see Table 7 for descriptive statistics for each OM variable). Statistically significant overall group differences were found for each variable: Fixation Stability ( $p < .05$ ), Saccadic Latency ( $p < .01$ ), Saccadic

TABLE 6

ANALYSES OF GROUP (MARKED, MILD, NORMAL CONTROL)  
 DIFFERENCES IN OCULOMOTOR PERFORMANCE ACROSS  
 SPATIAL FIELD: ONE-WAY ANALYSES OF VARIANCE  
 WITH REPEATED MEASURES

|    | SS      |     | MS      |     | df | F-ratio |
|----|---------|-----|---------|-----|----|---------|
| ET | 2770.62 | (b) | 1385.31 | (b) | 2  | 5.87 ** |
|    | 6375.67 | (w) | 236.14  | (w) | 27 |         |
| SL | 2322.69 | (b) | 1161.34 | (b) | 2  | 6.72 ** |
|    | 4665.78 | (w) | 172.81  | (w) | 27 |         |
| SA | 4.66    | (b) | 2.33    | (b) | 2  | 8.24 ** |
|    | 7.64    | (w) | .28     | (w) | 27 |         |
| FS | 63.40   | (b) | 31.70   | (b) | 2  | 4.83 *  |
|    | 177.04  | (w) | 6.56    | (w) | 27 |         |

\*  $p < .05$ , \*\*  $p < .01$ , b=between, w=within,  
 ET=Exploration Time, SL=Saccadic Latency,  
 SA=Saccadic Accuracy, FS=Fixation Stability

TABLE 7

ANALYSES OF GROUP (MARKED, MILD, NORMAL CONTROL)<sup>a</sup>  
 DIFFERENCES IN OCULOMOTOR PERFORMANCE ACROSS  
 SPATIAL FIELD: STUDENT-NEWMAN-KEULS PROCEDURE

|    | Marked Group<br>$\bar{x}$ / s.d. | Mild Group<br>$\bar{x}$ / s.d. | Normal Control<br>$\bar{x}$ / s.d. |
|----|----------------------------------|--------------------------------|------------------------------------|
| ET | 30.07 / 21.54                    | 46.08 / 14.17                  | 53.02 / 6.59                       |
|    |                                  | *                              | n.s.                               |
| SL | 625.94 / 361.93                  | 399.49 / 113.48                | 270.43 / 26.67                     |
|    |                                  | *                              | n.s.                               |
| SA | 2.41 / .56                       | 2.48 / .68                     | 1.59 / .27                         |
|    |                                  | n.s.                           | *                                  |
| FS | 10.26 / 2.54                     | 9.13 / 3.56                    | 6.78 / .76                         |
|    |                                  | n.s.                           | n.s.                               |

<sup>a</sup>n=10 per group, \* p < .05, n.s.=not significant

ET=Exploration Time (expressed as percent of time spent  
gazing in left spatial field)

SL=Saccadic Latency (expressed in msec.)

SA=Saccadic Accuracy (expressed as number of saccades to  
fixate a new target)

FS=Fixation Stability (expressed as degrees of eye  
movement)

Accuracy ( $p < .01$ ), and Exploration Time ( $p < .01$ ), thus supporting the first aspect of the above hypothesis.

To test for significant differences between the various combinations of group pairs (i.e., Marked vs. Mild, Marked vs. Control, and Mild vs. Control) Student-Newman-Keuls procedure was applied to the data for each OM variable. Table 7 presents those results. On every variable the performance of the Marked group was significantly different from the Normal Control group ( $p < .05$ ). For the variable Fixation Stability, this was the only statistically significant paired-group difference. Only for the variable Saccadic Accuracy, was there an additional significant difference between the Mild and Control groups ( $p < .05$ ). And only for the variables Saccadic Latency and Exploration Time were statistically significant group differences found between the Marked and Mild groups ( $p < .05$ ). For each OM variable except Saccadic Accuracy, the Mild group performed somewhere between the Marked and Normal Control groups (although statistically significant different performance was not always demonstrated). For the variable Saccadic Accuracy, even though the Mild group performed worse than the Marked group, this difference was not significant. Thus, these findings generally support the second aspect of the above hypothesis (regarding

specific relationships in performance between groups).

#### Extended Analyses: Eye Movement Vectors

Another way of addressing the relationship between VHN and OM behavior which might account for some of the results obtained thus far is to consider the different vectors of eye movement for those OM variables for which such a breakdown can be made. This is possible for the variables Saccadic Latency and Saccadic Accuracy. That is, for these two variables the data may be separated on the basis of whether the eyes move in a leftward or rightward direction in response to the stimuli presented. Two-way analyses of variance with repeated measures were performed for Saccadic Latency and Saccadic Accuracy measures, with group membership and direction of eye movement as the two factors. Table 8 presents the results of those analyses (see Table 9 for descriptive statistics for OM variables based on direction of eye movement). As noted earlier, group effects were significant. These additional analyses reveal that a significant effect also exists for direction of eye movement (Saccadic Accuracy  $p < .05$ , Saccadic Latency  $p < .01$ ). A significant interaction exists only for Saccadic Latency ( $p < .01$ ). These findings reveal that VHN and dynamic OM behavior (that which involves movement) has

TABLE 8

ANALYSES OF GROUP (MARKED, MILD, NORMAL CONTROL)  
DIFFERENCES IN OCULOMOTOR PERFORMANCE FOR DIRECTION OF  
EYE MOVEMENT (LEFT, RIGHT): TWO-WAY ANALYSES OF VARIANCE  
WITH REPEATED MEASURES

|    | Source      | SS      | MS      | df | F-ratio   |
|----|-------------|---------|---------|----|-----------|
| SL | Group       | 4645.37 | 2322.69 | 2  | 6.72 **   |
|    | Error       | 9331.55 | 345.61  | 27 |           |
|    | Direction   | 3460.04 | 3460.04 | 1  | 11.60 **  |
|    | Interaction | 3440.28 | 1720.14 | 2  | 5.77 **   |
|    | Error       | 8053.09 | 298.26  | 27 |           |
|    |             |         |         |    |           |
| SA | Group       | 9.33    | 4.66    | 2  | 8.24 **   |
|    | Error       | 15.29   | 0.57    | 27 |           |
|    | Direction   | 1.36    | 1.36    | 1  | 6.68 *    |
|    | Interaction | 0.51    | 0.26    | 2  | 1.25 n.s. |
|    | Error       | 5.50    | 0.20    | 27 |           |
|    |             |         |         |    |           |

\*  $p < .05$ , \*\*  $p < .01$ , n.s.=not significant  
SL=Saccadic Latency, SA=Saccadic Accuracy

TABLE 9

DESCRIPTIVE STATISTICS FOR GROUP  
(MARKED, MILD, NORMAL CONTROL)<sup>a</sup> PERFORMANCE ON OCULOMOTOR  
VARIABLES ACCORDING TO DIRECTION OF EYE MOVEMENT

|                           | Marked Group        | Mild Group          | Normal Control      |
|---------------------------|---------------------|---------------------|---------------------|
|                           | $\bar{x}$ /<br>s.d. | $\bar{x}$ /<br>s.d. | $\bar{x}$ /<br>s.d. |
| SL <sup>b</sup> Left EMs  | 926.85 /<br>685.30  | 465.93 /<br>186.37  | 280.39 /<br>30.17   |
| SL <sup>b</sup> Right EMs | 322.73 /<br>106.02  | 331.57 /<br>139.03  | 259.55 /<br>31.17   |
| SA <sup>c</sup> Left EMs  | 2.65 /<br>0.76      | 2.65 /<br>1.09      | 1.62 /<br>0.28      |
| SA <sup>c</sup> Right EMs | 2.17 /<br>0.48      | 2.26 /<br>0.40      | 1.57 /<br>0.26      |

<sup>a</sup>n=10 per group

SL=Saccadic Latency, SA=Saccadic Accuracy, EM=Eye Movement  
bexpressed in msec.

cexpressed as number of saccades to fixate a new target

a relationship that is dependent upon whether the movement is toward the neglected field or away from it, and in some cases also depends on the severity of VHN.

In order to further assess the nature of the interaction between direction of eye movement and severity of neglect for the variable Saccadic Latency, separate one-way analyses of variance were performed for leftward and rightward eye movements (see Table 10). Significant group differences were found only for leftward movements ( $p < .01$ ). As Table 11 shows, Student-Newman-Keuls group comparisons revealed significant differences between the Marked and Mild groups and between the Marked and Control groups, but not between the Mild and Control groups ( $p < .05$ ).

#### OM Performance For Data Collapsed Across Spatial Fields and Covaried For Visual Field Defect

In noting that the nature of three out of four OM variables required eye movements into the blind field of those who had visual field deficits, it was considered prudent to investigate the role played by visual field deficits in the OM variables under study. Thus, the data for each OM variable were collapsed across spatial field and a series of one-way analyses of covariance was performed, with the co-

TABLE 10

ANALYSES OF GROUP (MARKED, MILD, NORMAL CONTROL)  
DIFFERENCES IN OCULOMOTOR PERFORMANCE ACROSS SPATIAL FIELD  
FOR SACCADIC LATENCY RESPONSES SEPARATED ACCORDING TO  
DIRECTION OF EYE MOVEMENT: ONE-WAY ANALYSES OF VARIANCE

|              | SS                              | MS                            | df      | F-ratio   |
|--------------|---------------------------------|-------------------------------|---------|-----------|
| SL Left EMs  | 7974.3707 (b)<br>16362.6805 (w) | 3987.1853 (b)<br>606.0252 (w) | 2<br>27 | 6.58 *    |
| SL Right EMs | 111.2827 (b)<br>1021.9654 (w)   | 55.6413 (b)<br>37.8506 (w)    | 2<br>27 | 1.47 n.s. |

\*  $p < .01$ , n.s.=not significant, b=between, w=within  
SL=Saccadic Latency, EM=Eye Movement

TABLE 11

ANALYSES OF GROUP (MARKED, MILD, NORMAL CONTROL)<sup>a</sup>  
DIFFERENCES IN OCULOMOTOR PERFORMANCE ACROSS SPATIAL FIELD  
FOR SACCADIC LATENCY RESPONSES<sup>b</sup> OF LEFT EYE MOVEMENTS ONLY:  
STUDENT-NEWMAN-KEULS PROCEDURE

| Marked Group<br>$\bar{x}$ / s.d. | Mild Group<br>$\bar{x}$ / s.d. | Normal Control<br>$\bar{x}$ / s.d. |
|----------------------------------|--------------------------------|------------------------------------|
| 926.85 / 685.30                  | 465.93 / 186.37                | 280.39 / 30.17                     |
|                                  | *                              | n.s.                               |

<sup>a</sup>n=10 per group

<sup>b</sup>expressed in msec.

\* $p < .05$

variate being Visual Field Defect. This test was performed on each OM variable--including direction of eye movement--but only for comparison of the Marked with the Mild group. This was required as a consequence of the limitation that equality of slopes (for the covariate) could not be tested (Dixon, 1981) for the Normal Control group relative to the others, due to the fact that the variance-covariance matrix of the Normal Control group is singular (i.e., no control subject had any visual field defect). Table 12 presents the results of those analyses (see Tables 7 and 9 for descriptive statistics regarding these OM variables).

As can be seen from Table 12, covarying for Visual Field Defect results in no significant group differences being obtained on any OM variable when comparing the Marked and Mild groups only. This finding is contrary to what was expected. Significance at the .05 level is approached, however, for the variables Exploration Time (obtained  $p = .077$ ), and Saccadic Latency for leftward eye movements (obtained  $p = .081$ ). This finding of an effect for Visual Field Defect cannot be explained by a high correlation between VHN and visual field deficits, since a high correlation between these two variables does not exist in the present study (see Table 4).

Table 12

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ANALYSES OF GROUP (MARKED, MILD) DIFFERENCES IN OCULOMOTOR  
PERFORMANCE ACROSS SPATIAL FIELD AND COVARIED ON VISUAL  
FIELD DEFECT: ONE-WAY ANALYSES OF COVARIANCE

|                    | SS         | MS        | df | F-ratio     |
|--------------------|------------|-----------|----|-------------|
| Exploration Time   |            |           |    |             |
| Adjusted means     | 1245.8951  | 1245.8951 | 1  | 3.5387 n.s. |
| Zero slope         | .0462      | .0462     | 1  | .0001 n.s.  |
| Error              | 5985.3708  | 352.0806  | 17 |             |
| Equal. of slopes   | 508.1309   | 508.1309  | 1  | 1.4843 n.s. |
| Error              | 5477.2399  | 342.3275  | 16 |             |
| SL for Left EMs    |            |           |    |             |
| Adjusted means     | 3143.8113  | 3143.8113 | 1  | 3.4357 n.s. |
| Zero slope         | 777.4819   | 777.4819  | 1  | .8497 n.s.  |
| Error              | 15555.6422 | 915.0378  | 17 |             |
| Equal. of slopes   | 228.2062   | 228.2062  | 1  | .2382 n.s.  |
| Error              | 15327.4359 | 957.9647  | 16 |             |
| SL for Right EMs   |            |           |    |             |
| Adjusted means     | 6.7120     | 6.7120    | 1  | .1237 n.s.  |
| Zero slope         | 68.2400    | 68.2400   | 1  | 1.2578 n.s. |
| Error              | 922.3098   | 54.2535   | 17 |             |
| Equal. of slopes   | 11.7949    | 11.7949   | 1  | .2073 n.s.  |
| Error              | 910.5148   | 56.9072   | 16 |             |
| SA for Left EMs    |            |           |    |             |
| Adjusted means     | .0002      | .0002     | 1  | .0002 n.s.  |
| Zero slope         | .0026      | .0026     | 1  | .0028 n.s.  |
| Error              | 15.9251    | .9368     | 17 |             |
| Equal. of slopes   | .0046      | .0046     | 1  | .0047 n.s.  |
| Error              | 15.9204    | .9950     | 16 |             |
| SA for Right EMs   |            |           |    |             |
| Adjusted means     | .0335      | .0335     | 1  | .1614 n.s.  |
| Zero slope         | .0068      | .0068     | 1  | .0328 n.s.  |
| Error              | 3.5245     | .2073     | 17 |             |
| Equal. of slopes   | .1260      | .1260     | 1  | .5930 n.s.  |
| Error              | 3.3986     | .2124     | 16 |             |
| Fixation Stability |            |           |    |             |
| Adjusted means     | 3.6000     | 3.6000    | 1  | .3849 n.s.  |
| Zero slope         | 12.8688    | 12.8688   | 1  | 1.3759 n.s. |
| Error              | 159.0059   | 9.3533    | 17 |             |
| Equal. of slopes   | 2.0197     | 2.0197    | 1  | .2058 n.s.  |
| Error              | 156.9863   | 9.8116    | 16 |             |

SL = Saccadic Latency, SA = Saccadic Accuracy  
EM = Eye Movement, n.s. = not significant

### OM Performance According to Spatial Field

Repeated measures two-way analyses of variance were performed on the OM data to determine the presence of an effect for the spatial field in which the stimuli and responses occurred. It was expected that an overall group effect would be found for each variable, and furthermore that performance would be worst in the left spatial field for both brain-lesioned groups, but best in that field for the Normal Control group. Table 13 presents the two-way ANOVA results (see Table 14 for corresponding descriptive statistics for OM variables according to spatial field). As noted in Chapter V - Research Design, scores for the variable Exploration Time were derived from performance only in the center spatial field due to technical limitations, and so spatial field effects could not be tested for this variable. Of the remaining three variables, a significant spatial field effect was found only for Fixation Stability ( $p < .001$ ). There were no significant interactions found between group and spatial field for any OM variable.

It is noteworthy that the spatial field effect that was found was not in the predicted direction (as stated above), yet significant for each possible paired comparison ( $p < .05$  for left vs. right and center vs. right,  $p < .01$  for

TABLE 13

ANALYSES OF GROUP (MARKED, MILD, NORMAL CONTROL)  
DIFFERENCES IN OCULOMOTOR PERFORMANCE BY SPATIAL FIELD  
(LEFT, CENTER, RIGHT): TWO-WAY ANALYSES OF VARIANCE WITH  
REPEATED MEASURES

|    | Source      | SS       | MS      | df | F         |
|----|-------------|----------|---------|----|-----------|
| SL | Group       | 6854.26  | 3427.09 | 2  | 6.51 **   |
|    | Error       | 14214.48 | 526.46  | 27 |           |
|    | Sp. Field   | 156.02   | 78.01   | 2  | 1.66 n.s. |
|    | Interaction | 215.74   | 53.93   | 4  | 1.15 n.s. |
|    | Error       | 2540.07  | 47.04   | 54 |           |
| SA | Group       | 13.99    | 6.99    | 2  | 8.24 **   |
|    | Error       | 22.93    | 0.85    | 27 |           |
|    | Sp. Field   | 0.41     | 0.20    | 2  | .73 n.s.  |
|    | Interaction | 0.34     | 0.09    | 4  | .31 n.s.  |
|    | Error       | 14.94    | 0.28    | 54 |           |
| FS | Group       | 190.19   | 95.10   | 2  | 4.83 *    |
|    | Error       | 531.12   | 19.67   | 27 |           |
|    | Sp. Field   | 59.28    | 29.64   | 2  | 10.70 *** |
|    | Interaction | 20.41    | 5.10    | 4  | 1.84 n.s. |
|    | Error       | 149.51   | 2.77    | 54 |           |

\*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ , SL=Saccadic Latency  
SA=Saccadic Accuracy, FS=Fixation Stability  
n.s.=not significant

TABLE 14  
 DESCRIPTIVE STATISTICS FOR GROUP  
 (MARKED, MILD, NORMAL CONTROL)<sup>a</sup> PERFORMANCE ON OCULOMOTOR  
 VARIABLES ACCORDING TO SPATIAL FIELD

|                                       | Marked Group<br>$\bar{x}$ / s.d. | Mild Group<br>$\bar{x}$ / s.d. | Normal Control<br>$\bar{x}$ / s.d. |
|---------------------------------------|----------------------------------|--------------------------------|------------------------------------|
| <b>Saccadic Latency<sup>b</sup></b>   |                                  |                                |                                    |
| Lt. Field                             | 657.30/472.76                    | 436.25/174.87                  | 264.89/32.67                       |
| Ctr. Field                            | 663.47/386.74                    | 381.24/106.69                  | 280.39/32.67                       |
| Rt. Field                             | 547.78/282.22                    | 392.58/140.36                  | 264.72/29.34                       |
| <b>Saccadic Accuracy<sup>c</sup></b>  |                                  |                                |                                    |
| Lt. Field                             | 2.58/1.02                        | 2.53/1.06                      | 1.60/0.28                          |
| Ctr. Field                            | 2.40/0.66                        | 2.44/0.88                      | 1.55/0.23                          |
| Rt. Field                             | 2.24/0.42                        | 2.37/0.60                      | 1.63/0.43                          |
| <b>Fixation Stability<sup>d</sup></b> |                                  |                                |                                    |
| Lt. Field                             | 12.15/4.11                       | 10.07/5.84                     | 7.06/1.56                          |
| Ctr. Field                            | 8.94/2.24                        | 8.04/1.93                      | 6.35/0.14                          |
| Rt. Field                             | 9.74/1.78                        | 9.29/3.12                      | 6.94/0.75                          |

<sup>a</sup>n=10 per group

<sup>b</sup>expressed in msec.

<sup>c</sup>expressed as number of saccades to fixate a new target

<sup>d</sup>expressed in degrees

left vs. center). This is presented in Table 15. It was found that performance was best in the center field, and worst in the left field for all groups. Thus, the hypothesized spatial field effect was poorly supported in terms of there being an overall influence of spatial field on each OM variable, and entirely unsupported in terms of the specific nature of the single spatial field effect that was found.

#### Extended Analyses: OM Variable Intercorrelations

In order to examine the interrelationship among OM scores a Pearson Product Moment intercorrelation matrix was obtained for the OM variables for data collapsed across spatial fields. Table 16 presents those results with corresponding two-tailed t-tests for significance. As with the other correlational analyses presented, the present analyses were performed for all subjects combined, and for the subgroups consisting of brain-lesioned subjects only, and non-lesioned subjects only. Separate tests were run on lesioned and non-lesioned groups, due to consideration for the effects of brain-lesion per se.

The highest correlation in each analysis was obtained between Exploration Time and Saccadic Latency ( $r = -.698$ ,  $p < .05$  for all subjects,  $r = -.642$ ,  $p < .05$  for brain-

TABLE 15

ANALYSIS OF SPATIAL FIELD (LEFT, CENTER, RIGHT)  
 DIFFERENCES ACROSS GROUPS<sup>a</sup> FOR FIXATION STABILITY<sup>b</sup>:  
 STUDENT-NEWMAN-KEULS PROCEDURE

| Left Field<br>$\bar{x}$ / s.d. | Center Field<br>$\bar{x}$ / s.d. | Right Field<br>$\bar{x}$ / s.d. |
|--------------------------------|----------------------------------|---------------------------------|
| 9.76 / 2.56                    | 7.78 / 1.32                      | 8.65 / 1.50                     |

Diagram illustrating the Student-Newman-Keuls procedure for fixation stability across three spatial fields (Left, Center, Right). The mean values ( $\bar{x}$ ) and standard deviations (s.d.) are shown for each field. The Center Field (7.78 / 1.32) is significantly different from both the Left Field (9.76 / 2.56) and the Right Field (8.65 / 1.50), indicated by double asterisks (\*\*). The Left Field and Right Field are not significantly different from each other, indicated by a single asterisk (\*).

<sup>a</sup>n=30, <sup>b</sup>expressed in degrees of eye movement  
 \*  $p < .05$ , \*\*  $p < .01$

TABLE 16  
INTERCORRELATION MATRICES FOR OCULOMOTOR VARIABLES  
ACROSS SPATIAL FIELD

|                             |    | ET       | SL       | SA       | FS    |
|-----------------------------|----|----------|----------|----------|-------|
| All Ss<br>(n=30)            | ET | 1.000    |          |          |       |
|                             | SL | -.698 *  | 1.000    |          |       |
|                             | SA | -.557 *  | +.608 *  | 1.000    |       |
|                             | FS | -.392 *  | +.462 *  | +.346 ns | 1.000 |
| Brain-lesioned Ss<br>(n=20) | ET | 1.000    |          |          |       |
|                             | SL | -.642 *  | 1.000    |          |       |
|                             | SA | -.477 *  | +.497 *  | 1.000    |       |
|                             | FS | -.252 ns | +.316 ns | +.048 ns | 1.000 |
| Control Ss<br>(n=10)        | ET | 1.000    |          |          |       |
|                             | SL | -.510 ns | 1.000    |          |       |
|                             | SA | +.244 ns | -.187 ns | 1.000    |       |
|                             | FS | -.061 ns | +.161 ns | +.481 ns | 1.000 |

ET=Exploration Time, SL=Saccadic Latency  
SA=Saccadic Accuracy, FS=Fixation Stability  
\*  $p < .05$  (two-tailed tests), ns = not significant

lesioned subjects, and  $r = -.510$ ,  $p = \text{n.s.}$  for control subjects). No significant intercorrelations were obtained for the Control group. For brain-lesioned subjects Fixation Stability was not correlated with any other OM variable.

## CHAPTER VII

### DISCUSSION

This chapter returns to the specific hypotheses listed in Chapter V - Research Design to discuss the finding that certain predictions were supported by the present study, and others were not. Each hypothesis will be discussed in the original order of presentation.

#### Severity of VHN and OM Performance Across Spatial Field

The first hypothesis stated that differences would be found for OM performance (based on measures of Fixation Stability, Saccadic Latency, Saccadic Accuracy, and Exploration Time) between the Marked VHN, Mild VHN, and Normal Control groups, and furthermore, that of all possible pairwise group comparisons the only instance of no difference would occur for Exploration Time between the Mild VHN and the Normal Control groups. The prediction of overall group differences was supported for each dependent measure. But, while pairwise group comparisons also supported the prediction of no difference as stated for Exploration Time, other findings of no difference were obtained for the remaining OM variables under certain group comparisons.

Three important points may be derived from this outcome. One point is that the Chedru et al. (1973) finding regarding the inability of Exploration Time to distinguish Mild VHN and Normal Control groups was replicated. A second point is that overall group differences were found on all dependent OM measures, despite the existence of large differences in group variances (see Table 7) on two of the measures: Exploration Time, and Saccadic Latency. The third point is that breaking down the gross measure Exploration Time into the components consisting of Saccadic Accuracy, Saccadic Latency, and Fixation Stability does provide a more complete account of the relationship between OM behavior and severity of VHN, as long as all three components are considered; no single component is satisfactorily discriminating at all levels of severity.

The replication of the Chedru et al. (1973) finding that Exploration Time does not distinguish subjects with mild VHN from those without VHN is of value for empirical as well as theoretical reasons. Their finding was based on a searching task that involved data accumulated and averaged from a series of trials (displays) where the purpose was to locate a single target on each display (after Popplereuter, 1917). The present study used a single trial where the purpose was to locate a large number of appear-

ances of the same target from only one display (after Diller et al., 1974). The same result was obtained in both studies, thus corroborating the limited discriminability of Exploration Time. This finding supports, in turn, the viewpoint of the present research emphasizing the need to break down this gross measure into its various components (Saccadic Latency, Saccadic Accuracy, and Fixation Stability) for further study.

The finding of overall group differences for each OM measure (based on ANOVA testing) is especially noteworthy given the magnitude of intergroup variance differences for certain variables. Levene's Test for Equal Variances (Brown & Forsythe, 1974) revealed that two OM measures were not associated with significantly different group variances, while two were, viz., Exploration Time ( $F=6.90$ ,  $p<.01$ ), and Saccadic Latency ( $F=6.22$ ,  $p<.01$ ). Although this may have been due in part to the fact that scores for Exploration Time and Saccadic Latency had much greater potential ranges than did the scores for the other two variables, it is likely that a neuropsychological factor (in addition to this statistical one) was also influential. One such factor is the clinically observed tendency of patients with marked VHN simply not to direct their gaze to the neglected side even when instructed to, or to do so

only after such great delay that one is not sure the response is actually a result of the command (e.g., Weinstein & Friedland, 1977).

The possibility of obtaining differences in group variances was considered early in this study on the basis of pilot work done regarding the variable Saccadic Latency. The results of preliminary research revealed that the potential existed for brain-lesioned subjects in certain cases to make no response, or a very delayed response to a stimulus signalling a leftward eye movement. It follows to reason that the range of response latencies in a group containing such patients could be quite great.

This non-responding behavior is also seen in patients with neglect on tests of double-simultaneous stimulation, often referred to as DSS (Deutsch, Tweedy, & Lorinstein, 1980). On such tests while the patient correctly responds to a single stimulus, if that stimulus is concurrently accompanied by another one of the same nature (e.g., wriggling fingers in the left and right visual fields at the same time), only the stimulus most proximal to the subject, or ipsilateral to the damaged hemisphere will elicit a response. Recall that the Saccadic Latency stimulus was the onset of a foveally presented light that was simultaneously accompanied by the onset of an appropriately

located peripheral light to which the subject was to direct his/her gaze. The similarity between this and the DSS paradigm is readily apparent. The use of a single peripherally placed stimulus was not employed because it was felt that doing so would automatically place at a disadvantage those subjects with peripheral visual field sensory deficits.

Despite its relatively common observation in the clinic, why such behavior--sometimes termed an extinction response (Bender, 1952)--should occur at all remains a mystery. As discussed earlier (see pp. 31-35), the numerous explanations that have been postulated to explain symptoms such as this may be divided into sensory-perceptual (e.g., Denny-Brown & Banker, 1954), spatial-motoric (e.g., Heilman, 1979), and attention-arousal (e.g., Kinsbourne, 1972) hypotheses. Of particular relevance in accounting for the specific phenomenon known as extinction, Birch, Belmont, and Karp (1967) have shown that in a DSS paradigm this symptom may be reduced somewhat by stimulating the abnormal side before the intact side, and made more pronounced by stimulating the intact side first. They suggest that the damaged cerebral hemisphere is subject to interference from the intact hemisphere since the lesioned side is presumably slower to process information.

Another plausible neuropsychological explanation, given the present paradigm, is that the anatomical substrate necessary for planning and/or initiating the correct motor program has been damaged, and therefore no response is produced, or the response is greatly delayed or abnormal in some other way. This explanation is supported by the observation that in cases where such behavior does occur, when the patient is asked what s/he is supposed to do the correct verbal response is given. Yet the patient is unable to perform correctly (see also, Johnston & Diller, 1983; Konow & Pribram, 1970; Luria, Pribram, & Homskaya, 1964). This did in fact happen in the present investigation. Furthermore, a significant effect was shown for direction of eye movement on those variables for which this factor could be tested, i.e., Saccadic Latency and Saccadic Accuracy responses were worse for left-going eye movements than for right-going eye movements.

To omit no-response trials from the analyses, or to use some average response time as substitute for such trials would produce a distortion of the results. To avoid this it was decided that the best way to handle occurrences of no-response would be to substitute those particular trials with that subject's longest latency response. This was done for eight subjects, three of whom belonged to the

Marked VHN group, and the other five to the Mild VHN group; none were members of the Normal Control group. This procedure served to increase the likelihood that significant differences in group variances would be obtained. But when differences in group variances were taken into account through the application of one-way analyses of variance tests for within-group variances not assumed to be equal (Welch, and Brown-Forsythe statistics; see Brown & Forsythe, 1974), significant differences were still shown in the overall mean group scores for Exploration Time (Welch  $F=5.46$ ,  $p<.05$ ; Brown-Forsythe  $F=5.87$ ,  $p<.05$ ) as well as Saccadic Latency (Welch  $F=10.21$ ,  $p<.01$ ; Brown-Forsythe  $F=6.72$ ,  $p<.05$ ).

An interesting sidelight from the standpoint of explaining this non-responding behavior is that only one subject manifesting the phenomenon was determined to have a sensory visual field deficit. On the other hand, four subjects who showed non-responding behavior for Saccadic Latency trials also demonstrated the extinction response when tested with visual DSS as described. This suggests that subjects in the present study who did not respond to stimuli for leftward eye movement were behaving so for some other reason than the presence of a visual sensory deficit for the left visual field.

It was predicted that the specific nature of inter-group differences in OM performance would be such that each OM measure would differentiate each group from each of the other groups, except that no difference in performance would be found between the Mild VHN and Normal Control groups for the measure of Exploration Time. While the exception was in fact supported, it was not to be the only exception.

The underlying construct here was that Exploration Time is such a gross measure that it would not be as sensitive to group differences as would individual components of Exploration Time. This viewpoint stems in particular from the functional systems orientation in neuropsychology (Anokhin, 1935; Luria, 1973). Intercorrelations of Exploration Time with each of the other OM variables were found when all subjects were combined, but not when the control subjects and brain-lesioned subjects were considered separately. One might expect this to be the case if it is true that an individual component would be more discriminating than the gross measure Exploration Time, particularly when that component is related to a specific aspect or severity level of the VHN phenomena. The important point here, which is a finding somewhat divergent from the initial thesis, is that no individual component was found

to differentiate each group from every other group.

That such a pattern should occur is not actually inconsistent with the underlying hypothetical construct as it is stated above. In fact, it extends previous knowledge (i.e., Chedru et al., 1973) by demonstrating that a group of subjects with Mild VHN can be discriminated from a group without VHN when the subcomponent Saccadic Accuracy is the dependent variable, but not when the grosser measure Exploration Time is used. What is required is modification of the construct to state that subcomponents of Exploration Time may be differentially sensitive to different severity levels of VHN. Thus, while the component Saccadic Accuracy does not differentiate Marked from Mild VHN, Saccadic Latency does (and this latency effect is due to differences in leftward but not rightward eye movements). At a lower level of severity of VHN (i.e., comparing the Mild VHN group with the Normal Control group) Saccadic Accuracy holds significant discriminating power while the other components do not.

Finally, the predicted hierarchy of group performance on OM measures suggesting that the Marked VHN group would perform the worst, the Normal Control group would perform the best, and performance by the Mild VHN group would lie somewhere in between the other two groups was upheld with

one exception. That exception was for Saccadic Accuracy, whereupon it was shown that the Mild VHN group performed very slightly (nonsignificantly) worse than the Marked VHN group.

A project for continued research along these lines might be to develop a regression equation that would take into consideration the relative contribution of these (and perhaps other) OM variables (after Johnston & Pirozzolo, 1981) not only to predict the severity of VHN, but also to characterize it "symptomatically" for the development of individualized remediation programs.

#### The Influence of Visual Field Defect

The second hypothesis which stated that the presence of visual field deficits would have minimal effects on OM data beyond those accounted for by severity of VHN was not supported. When data analyses were employed that treated visual field defect as a covariate, group mean differences that otherwise were significant fell to levels that did not quite reach significance. No group differences increased in magnitude as a result of this statistical treatment.

Only the Marked and Mild VHN groups were considered in these analyses. The reduction in the significance of group differences was, therefore, relevant only to the OM vari-

ables Exploration Time and Saccadic Latency, since they were the only ones for which a significant difference was found between the two groups compared before covarying. In fact, the obtained  $p$  values did approach significance at the .05 level after covarying (approximately .08 in both cases). Based on the concepts of power analysis (Cohen, 1977), it is estimated that a significance level of .05 would have been reached with a sample size of 17 in each of the two groups (see sample size tables for F-Tests on Means in the Analysis of Variance and Covariance, p. 384).

It is not surprising that covarying for visual field defect should produce the result in the direction that it did. Although the premise was that severity of VHN, and not visual field deficit is strongly related to OM performance, certainly a task requiring directing gaze into a blind visual field should be affected to some extent by the presence of a visual sensory deficit.

The magnitude of the effect of covarying for visual field defect is surprising, however. It is puzzling to note that covarying for visual field deficits served to reduce the significance of different group means for OM variables to non-significance when there were no significant group differences for the variable Visual Field Defect. This may indicate that the presence of a visual

sensory deficit has a greater effect on patients with Marked VHN than on those with Mild VHN, in the context of OM performance. This notion is supported by Chedru et al. (1973) at a descriptive level for the variable Exploration Time. Statistical procedures were not applied to their finding in this regard, however.

#### OM Performance In Different Spatial Fields

The prediction corresponding to the second major hypothesis stated that differences would be found for OM performance between the left, center, and right spatial fields such that the worst performance for the Marked and Mild VHN groups would be in the left field, while the worst performance for the Normal Control group would be in the right field. This prediction was based primarily on the work of Bowers & Heilman (1980), and Heilman (1979) discussed in Chapter IV - Spatial Field Effects. Results of the present study demonstrated a spatial field effect only for the variable Fixation Stability, and that the nature of the effect was not as predicted. That is, in all groups performance was worst in the left field, almost as poor in the right field, and best in the center field. Although no interaction between group and spatial field was found, performance was worst for the Marked VHN group and

best for the Normal Control group. Overall performance (collapsed across all fields) has already been discussed. The following discussion addresses the limited and unexpected findings regarding spatial field effects.

In attempting to account for the demonstrated spatial field effect only for Fixation Stability, three explanations may be considered. One holds that the difference in area covered by one spatial field vs. another was not great enough to produce performance differences on certain OM measures. Another explanation is that the nature of the OM performance itself is the significant factor, i.e., that regardless of how distant one spatial field is from another, for certain OM variables (i.e., those involving ballistic movement) significant differences will never be obtained based on spatial field. The third possibility is an extension of the second that considers the different neuroanatomical substrata that may be involved in different types of OM behavior, and the consequently different degrees of dependence on unilateral vs. bilateral systems.

The unusual nature of the spatial field effect that was found may also be considered in light of two different possible explanations. One explanation is an offshoot of the neuroanatomical considerations just mentioned, with the other possibility being that a pre-existing phenomenon

observed in neurologically intact subjects is merely accentuated in brain-lesioned subjects. These various possibilities will now be explored.

Concerning the distance between spatial fields, the midpoints of adjacent fields in the present study were separated by 8 degrees for Saccadic Accuracy and Saccadic Latency, while targets for Fixation Stability stimuli of adjacent fields were separated by 16 degrees. The distance between spatial fields in studies of extremity motor performance (Bowers & Heilman, 1980; Heilman & Valenstein, 1979b; Reed, 1982) were even greater. For example, in the study by Reed (1982) rods were to be bisected by sense of touch. The center of the rods placed in each lateral spatial field were 30 cm. from the midpoint of the center spatial field. The relatively small distance from midline used in the present study may account for the limited OM/spatial field findings. Furthermore, of the three OM variables examined for a spatial field effect, the one that did demonstrate an effect was the one with the greatest distance between fields.

It may also be of significance that the only variable in the present study for which a spatial field effect was shown was one that demanded sustained inhibition of movement, as opposed to others which were dynamic and non-

sustained. Jenkyn, Walsh, Culver, and Reeves (1977) have shown that lateral gaze impersistence is often demonstrable in patients with brain-damage, and is a good predictor of the degree of accompanying cerebral dysfunction. Although their patients had diffuse brain disease, it has been reported that unilateral cerebral lesions are also associated with certain types of motor impersistence (Fisher, 1956). In these studies the patients could initiate the activity (e.g., stare in a certain direction) but could not maintain their performance. The nature of the eye movements observed in the present study that represent oculomotor impersistence were small saccades (not physiological nystagmus), with a few large saccades interspersed. It is not unreasonable to speculate that, as has been found in this study, unilateral lesions could produce gaze impersistence (poor Fixation Stability) and VHN that would vary according to spatial field.

An important qualification to the logic of this theory is that the nature of the spatial field effect obtained was inconsistent with general clinical observations of the manifestation of VHN (consider the term "hemi-"), and inconsistent with what one would expect from a lateralized lesion. From a neuroanatomical point of view, it is difficult to reconcile how a unilateral lesion could manifest

bilateral peripheral impairments in performance while relatively sparing performance in a central field. Perhaps the solution to this riddle lies with considering the system (pathways) involved that relate to particular types of motor activity.

It is important to note that in other studies of spatial field effects the dependent variables required some form of measurement of extremity movement (Anzola et al., 1977; Bowers & Heilman, 1980; Bruder, Note 3; Heilman & Valenstein, 1979b; Watson et al., 1978). In the present study eye movement was required on certain tasks, and inhibition of eye movement on certain others. Pathways of efferent signals traverse the pyramidal system for cortically initiated extremity movements, and extrapyramidal routes for cortically initiated eye movements; but more important than the route per se is the fact that different collateral input is involved with the different routes (Barr, 1974). In the OM system it has long been known that horizontal vectors of eye movement are, for the most part, mediated by a unilateral arrangement of neuroanatomical connections (e.g., Prevost, 1868), and that movements which are primarily vertical require the concerted involvement of bilateral hemispherical systems (e.g., Schaefer, 1888). Although pathways mediating horizontal eye movements have

been reasonably well defined, those for vertical movements are as yet not very well known (Bender, 1980). In extremity movement the primary underlying neuroanatomical system is unilateral, regardless of whether movement is horizontal or not (Barr, 1974). Furthermore, unlike left and right limbs of the extremity motor system, the left and right eyes of the OM system are yoked in movement through all planes (including horizontal) by virtue of internuclear neurons and reciprocal connectivity of left and right OM nerve nuclei (Bender, 1980).

This may have considerable bearing on the understanding of a spatial field mechanism as it relates to OM behavior. Heilman's (1979) spatial field theory de-emphasizes the importance of motor systems in suggesting that the each hemisphere is responsible for mediating activity in the contralateral spatial field, regardless of the laterality of the sensory-motor system involved. Thus, a consistent spatial field effect is shown, irrespective of the extremity used (e.g., Bowers & Heilman, 1980). When the more complexly innervated OM system with bilateral and synergistic pathways is studied, spatial field effects do not appear for certain components of OM behavior, and are atypical--relative to those found using extremity motor dependent measures--when such effects are found. Only when

the pathways for all vectors of eye movement, and all components of OM behavior have been elucidated, will it be possible to unravel this problem from a neuroanatomical point of view.

Finally, consideration must be given to the notion that pre-existing differences (i.e., of neurologically unimpaired individuals) in performance in different spatial fields may be accentuated by the effects of cerebral lesions and VHN. Thus, the Normal Control group demonstrated the same spatial field effect as did the brain-damaged groups, but to a lesser extent, and the effect was greatest for the Marked VHN group. This is similar to an idea favored in explaining other kinds of neuropsychological sequelae to brain injury (e.g., personality changes as discussed in Levin, Benton, & Grossman, 1982).

In summary, the following conclusions may be drawn from the present study:

- (a) Differences in OM performance are demonstrable between individuals with VHN and those who do not show VHN.
- (b) Differences in OM performance are demonstrable within different levels of severity of VHN.
- (c) Breaking down the OM behavior found in exploratory ocular activity into components (Fixation Stability, Saccadic Latency, and Saccadic Accuracy) provides a more

accountable relationship between abnormal OM behavior and VHN than does using a gross measure such as exploration time.

(d) Visual field deficit may have a greater effect on the OM behavior of patients with marked VHN than those with mild VHN.

(e) Dynamic OM behavior is related to left VHN such that eye movements in a leftward direction are more abnormal than those in a rightward direction, and the degree of abnormality is positively related to severity of VHN.

(f) A spatial field effect exists for OM behavior requiring inhibition of movement (Fixation Stability) but not for dynamic OM behavior, and the nature of the effect is different than that observed in extremity motor paradigms. That is, OM performance is best in the center spatial field and worst in the left spatial field.

This information has implications for the development of perceptual-cognitive retraining (PCR) programs for patients with VHN (cf., Diller & Weinberg, 1977). Various PCR programs have related success in retraining to severity of deficit, and noted that different subtypes of patients may require slightly different approaches in PCR (Ben-Yishay, Diller, Gerstman, & Gordon, 1970; Carter, Caruso, Languirand, & Berard, 1980).

The present study demonstrates that severity of VHN is related to differences in OM performance, and gives some insight into the nature of that relationship. This author suspects that an underlying defect in OM behavior has been assumed to be present in certain patients by those who develop PCR programs. The use of a scanning machine by Diller et al. (1974) in patients with marked VHN indicates that this is so. But the decision to use an exercise requiring development of smooth-pursuit ocular activity (as the scanning machine does) rather than--or in addition to--saccadic eye movement activity was not based on the direct study of OM performance in patients with VHN. In fact, smooth-pursuit OM activity is seldom required in our everyday lives. Exploratory eye movements (i.e., the saccadic system) are what we most often rely on to derive meaning from our visual environment. It is interesting that PCR program development has not been preceded by any rigorous investigation of ocular activity of patients with VHN.

Knowing that patients with marked VHN, relative to those with mild VHN, have significantly more difficulty moving their eyes quickly to a specified target should encourage the development of different PCR programs for these different types of patients. The finding that accu-

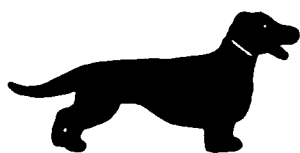
racy of eye movement is equally problematic for both types of patients is similarly noteworthy. Furthermore, the fact that spatial field does not appear to be important for either of these parameters of OM behavior, but direction of eye movement does, should further guide program development.

It is not premature to suggest that in certain individuals a causal relationship may exist between severity of abnormal OM performance and VHN. This notion relies on the realization that what the eyes are not directed to will not be appreciated. At a different level, if the eyes are directed only approximately to the target, or get there too late, or leave the target too soon, a distorted appreciation may develop as the information gleaned from that target is assimilated with information from preceding and succeeding targets. A necessary assumption to such a causal relationship is that impaired OM control mechanisms precede the clinical manifestation of VHN, and not vice versa. This remains an assumption. Conversely, Heilman (Note 2) has suggested an "intentional" deficit may exist in which a primary deficit of the ability to carry out intended activity forms the basis for such secondary effects as OM dysfunction. It remains to be seen which view will be supported by research.

Appendix A:  
Cancellation Stimulus

F E G I C H E I C B D A C H F B E D A C D A F C I H C F E B A F E A C F C H B D C F G H E  
D C F E H B F C A D E H A E I E G D E G H B C A G C I E H C I E F H I C D B C C F D E B A  
E H F A E F E G C H G D E H B A E G D A C H E B A E D G C D A F C B I F E A D C B E A C G  
  
F B C A F E A B F C H D E F C G A C B E D C F A H E H E F D I C H B I E B C A H C D E F B  
E H A C A F C I C A B E G F B E F A E A B G C G F A C D B E B C H F E A D H C A D E F E G  
G E A D F E B E I G A C G E D A C H G E D C A B A E F B C H D A C G B E H C D H E H A I E

## Appendix B: Piasefsky Test Stimulus



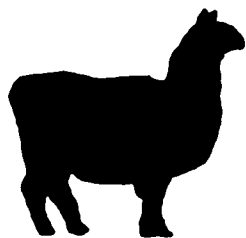
1



2



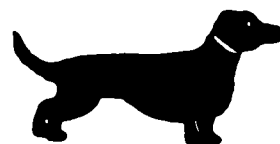
3



4



5



6

### Reference Notes

1. Heilman, K. Personal communication, 12/16/81.
2. Heilman, K. Personal communication in unpublished, untitled manuscript, 3/5/82.
3. Bruder, G. Personal communication, 4/20/82.
4. Ansler's Grid, Hamblin Ltd., 31 Cavendish St., London W1M 7RL.
5. Gulf & Western, Applied Science Laboratories, 335 Bear Hill Road, Waltham, Massachusetts 02154.
6. Hibbard, M. Personal communication, 2/10/82.

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### Autobiographical Career Statement

I was born 32 years ago in La Crosse, Wisconsin. In 1975 I obtained an Associate in Applied Science Degree in Electroencephalography (EEG) from Western Wisconsin Technical Institute, and a Bachelor of Science degree in Psychology from the University of Wisconsin. In 1978 I received a Master of Arts degree in Psychology from Queens College-CUNY. I am board registered in EEG, and licensed in psychology in the state of Minnesota.

The impetus to return to graduate school in 1980 in quest of a doctorate resulted from my affiliation with Thomas Hutton, a behavioral neurologist who had the good fortune to study under A.R. Luria, and who thereby provided me with a vicarious exposure to Soviet neuropsychology. The development of my career has also been particularly influenced by training and supervision from three eminent neuropsychologists, Manfred Meier, Barbara Wilson, and my dissertation advisor, Leonard Diller.

The combination of formal education with a background of published research, college teaching, and broad-based clinical neuropsychological service has culminated in my present positions. I am currently Neuropsychologist for the University of Minnesota Hospitals Division of Child & Adolescent Psychiatry, and Consulting Neuropsychologist for the Minneapolis Speech-Language-Audiology Clinic.