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OUT OF MIND, OUT OF SIGHT:

UNILATERAL SPATIAL DISORDERS IN BRAIN-DAMAGED PATIENTS

JENNIFER ANN OGDEN

A thesis presented to the University of Auckland in partial fulfilment of the requirements for the degree of Doctor of Philosophy December, 1983.
"Now o'er the one half-world
Nature seems dead, and wicked dreams abuse
The curtained sleep."

Macbeth II i
ACKNOWLEDGEMENTS

I thank my supervisor, Professor Michael Corballis, for his time, ideas, helpful discussions and constructive suggestions. I am particularly grateful for the way in which he read, annotated and returned everything I wrote within 24 hours. His interest in my research and his unwavering faith and confidence in my ability, helped maintain my enthusiasm, even when doing statistical analyses!

I also thank Dr Dorothy Gronwall, who taught me the principles of clinical neuropsychology. Her advice on clinical aspects of the research were particularly helpful.

Dr Jonathan Simcock, Senior Neurologist, and Mr Philip Wrightson, Senior Neurosurgeon, acted as my Medical Consultants. Without their support and generous permission to test their patients, this thesis would not have been possible. Dr Simcock proved to be a stimulating teacher and was always willing to discuss medical and clinical aspects of my research with me. Added to this, Dr Simcock and Dr K. Eyre cheerfully shared their hospital office with me and my equipment.

The entire medical and nursing staff of the Neurology and Neurosurgery Wards of Auckland Hospital assisted me in numerous ways. All the medical staff agreed to my testing any of their patients, and the nursing staff often re-arranged their duties around my testing program.
The hostesses and the secretaries assisted by finding files of past patients and arranging times for testing patients with busy schedules. My thanks also go to the Radiologists and their staff who took, processed and analysed the C.T. scans that formed an essential part of this research.

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I am also grateful for the suggestions made by four unknown reviewers who reviewed versions of Chapter 3 and 5 when they were submitted as articles for publication.

While carrying out this research I was supported by a U.G.C. Postgraduate Scholarship, a Winifred Gimblett Scholarship and Grant No. 81/4 from the N.Z. Neurological Foundation Incorporated to M.C. Corballis and D. Gronwall. The Neurological Foundation Inc. grant and Grant No. 141 Psychology 33 from the University of Auckland Research Fund to M.C. Corballis also paid in part for equipment and working expenses.

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Finally, and most sincerely, I thank all the patients who so willingly and cheerfully copied drawings, listened to endless digits and tones and concentrated on meaningless patterns on a video screen. Not a single patient I asked declined to participate in my study, even although many of them must have been going through a difficult and frightening time. Many patients were genuinely interested in what I was doing and very eager to help me in any way they could. Not only did I learn much
more about human neuropsychology than I could possibly report in this thesis, but these patients also taught me a great deal about courage, determination and acceptance. I would like to dedicate this thesis to all these patients, many of whom are well again, and sadly, many of whom have since died, in the hope that the time and effort they put into this research will, in some positive way, add to our knowledge and increase our understanding of brain functions and disorders and rehabilitation methods for neurological patients.
ABSTRACT

Hemineglect and unilateral extinction on double simultaneous stimulation in humans are neuropsychological disorders which sometimes follow a lesion to the cortex, subcortex or basal ganglia of one cerebral hemisphere. The main symptom is that the patient appears to neglect or be unaware of stimuli which impinge in one half of space relative to the patient's body. The side of space neglected is usually the side contralateral to the brain lesion. This thesis comprises a collection of studies on various aspects of these disorders. Experimental subjects were patients in the Neurology and Neurosurgical Wards of Auckland Hospital. All had clearly defined solitary unilateral brain lesions confirmed by Computerized Tomography.

Chapter 1 provides a historical review of research in the area, defines concepts, reviews recent human and animal research on hemineglect and extinction, and outlines the different theories which have been proposed in order to explain hemineglect.

Chapter 2 describes the methods used in neuropsychological testing, the criteria used in the selection of subjects, the etiologies of the different types of lesions sustained by the patient sample, and the neuropsychological tests used in the various studies.

Chapter 3 is a study of the incidence and severity of visual hemineglect in a group of 56 patients with left-
hemispheric lesions and 45 patients with right-hemispheric lesions. Five paper and pencil tests designed to measure the presence and severity of visual hemineglect were given to these patients. The incidence of hemineglect in the two groups did not differ significantly (50% and 44% in the left and right brain-damaged groups respectively). However, visual hemineglect was found to be more severe after right- than after left-hemispheric lesions. The two groups were found to differ significantly with respect to the loci of lesions most likely to result in hemineglect. In the right brain-damaged group most patients with hemineglect had posterior lesions, and in the left brain-damaged group most patients with hemineglect had anterior lesions. Possible reasons for this are discussed in terms of the effects the lateralization of language representation might have on the representation of spatial functions.

Chapter 4 describes an experiment designed to determine whether patients with unilateral cerebral lesions neglect the contralesional sides of their mental images of the external world. This phenomenon has previously been observed for patients with left-sided visual neglect following right-hemispheric lesions. Twenty control subjects and 16 patients with right- and left-hemispheric lesions were involved in the experiment reported in this chapter. In the 'static' condition they viewed pairs of complex shapes displayed, one shape at a time, on a video-screen controlled by a computer. In the 'dynamic' condition the pairs of shapes apparently moved, one at a
time, behind a narrow vertical slit. In both conditions the subject had to decide whether the two shapes of each pair were the same or different. In fact, some were the same, while some differed on the right and others on the left. In the 'dynamic' condition subjects had to construct spatial images from non-spatial external stimuli before they could make a same/different response. Both right and left brain-damaged groups demonstrated a significant neglect of the contralesional sides of their images of the shapes in that they often gave 'same' responses when the shapes actually differed on their contralesional sides. This has implications for normal imagery processes. It appears that at some advanced stage our images are mapped onto our hemispheres in an analogue fashion. That is, objects or parts of objects that we imagine to be on our left are mapped onto our right hemispheres, and those parts we imagine to be on our right are mapped onto our left hemispheres. If one hemisphere is damaged at a site which is essential to this imagery process, the contralateral half of the external stimulus that is being imagined will be degraded or neglected.

Chapter 5 is a study of auditory extinction in unilaterally brain-damaged patients. In particular the phenomenon of ipsilateral auditory extinction is investigated in detail. Previous researchers have found ipsilateral auditory extinction for dichotically presented verbal stimuli following lesions only of the posterior left hemisphere. It has been hypothesized that a lesion in this area disconnects a posteriorly routed interhemispheric
auditory pathway and that as a consequence, left-ear verbal input is unable to reach the left (speech) hemisphere. In Experiment 1, I tested 53 brain-damaged patients for extinction of digits on a dichotic listening task and found that patients with lesions wholly anterior to the central sulcus in the left hemisphere exhibited ipsilateral extinction as well as patients with posterior left-hemispheric lesions. This finding poses problems for the above hypothesis that relies on a posterior inter-hemispheric pathway, and alternatives to this hypothesis are discussed. In Experiment 2, I tested 16 patients for extinction of dichotically presented non-verbal material (tones) to ascertain whether ipsilateral extinction is restricted to verbal tasks. Ipsilateral extinction was not found on this task for either left or right brain-damaged patients. This suggested not only that ipsilateral auditory extinction is related to the disconnection or disruption of left-ear verbal input from the left (speech) hemisphere, but that the right hemisphere is not essential for the decoding and processing of non-verbal material. That is, I did not find right-ear ipsilateral extinction for non-verbal input in patients with right-hemispheric lesions.

Chapter 6 is the study of multimodal hemineglect and extinction in patients with right- and left-hemispheric lesions. Clinical observations suggest that multimodal neglect may be a single disorder with a common underlying cause, and that the number of modalities affected is dependent upon the severity of the underlying deficit. For
example, patients with hemiasomatagnosia (body hemineglect) are often observed to exhibit visual hemineglect and tactile extinction as well. I tested 50 patients for contralesional visual hemineglect, auditory extinction, tactile extinction and hemiasomatagnosia and computed phi correlation coefficients for pairs of disorders to see if there was any basis for supposing they were caused by the same underlying deficit. As the proportion of patients with hemineglect varied widely from modality to modality, possibly as a result of varying sensitivities of the tests used to measure the different forms of neglect, I also computed tetrachoric correlation coefficients. This measure corrects for varying proportions on the assumption that a normal distribution underlies each dichotomy. The results were inconclusive as the phi-coefficients were generally low and the tetrachoric coefficients very high. Because of the extreme difference between the two forms of correlation it was not possible to come to any conclusions about the 'true' correlations. It may be that at least some forms of hemineglect and extinction are independent of one another and are often found together in the same patients because the lesions overlap modality-specific areas, rather than because they result from the same underlying cause.

Chapter 7 summarizes the studies described in Chapters 3, 4, 5 and 6, and the results are reviewed in the light of some of the more important theories of hemineglect.
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1.7 ORGANIZATION OF THE THESIS
1.1 ORIENTATION AND HISTORICAL BACKGROUND

The importance of space in human perception and behaviour is unequivocal. It not only pervades our every activity when we are relating to our external world, but it also plays an essential role in our perceptions of our own bodies.

Research into spatially oriented behaviour in humans has, in the past, consisted largely of the study of patients who exhibited spatial deficits following brain-damage. While many of these deficits such as constructional apraxia (Benton, 1967) and loss of topographical orientation (Paterson and Zangwill, 1945) concerned a global inability to order space, there were occasional reports of a bizarre disorder in which patients appeared to ignore or neglect only that half of space opposite to the brain lesion. For example, in 1876 John Hughlings Jackson reported a patient who tried to read by starting at the lower right-hand corner of the page and proceeding backwards. Among other symptoms she also had difficulty dressing and finding her way around. At autopsy she was found to have a glioma of the right posterior hemisphere. Jackson dubbed this disorder "Imperception" and since then disorders with a similar phenomenology have variously been known as unilateral inattention, unilateral spatial neglect and hemineglect.

Hemineglect followed brain lesions that were restricted to one hemisphere, and patients neglected the stimuli in the side of space contralateral to that
hemisphere. While the disorder appeared to be most marked in tasks requiring the visual exploration of space or visual manipulospatial skills (Brain, 1941) some patients also neglected their contralesional limbs (Cobb, 1947) or contralateral tactile (Critchley, 1949) or auditory (Bender and Diamond, 1965) stimuli.

Subsequent to Jackson's paper in 1876, occasional articles were published on spatial disorders (Badal, 1888; Head and Holmes, 1911; Holmes, 1918, 1919; Holmes and Horrax, 1919; Pick, 1908; Zingerle, 1913) but there was not a great deal of interest displayed in this type of disorder. At the time, spatial deficits possibly seemed insignificant in comparison with the discoveries that were being made about the aphasias (Broca, 1861, 1865; Dax, 1865; Wernicke, 1874). These discoveries also served to concentrate research on lesions of the left hemisphere and away from the right.

In the 1940s an interest in spatial disorders was rekindled, partly as a result of a paper published by Brain in 1941. Brain reported three cases of patients with large right parieto-occipital lesions whom he suggested suffered from a deficit of "spatial orientation...inattention to or neglect of the left half of external space" (p.257). Brain was particularly prominent in noting the relation of the awareness of one's body to the awareness of external space. As he wrote in 1941, "Since each half of the body is a part of the corresponding half of external space, it is not surprising to find that perception of the body and perception of external space are closely related and
subject to similar disorders." (p.263). By this time the importance of the posterior cortex of the right hemisphere to disorders of space was well established (Brain, 1941, 1945; Critchley, 1949, 1953; Kleist, 1922; Pick, 1908; Pineas, 1931; Scheller and Seidermann, 1931). In fact, the association of hemineglect of the body with right parietal lesions was noted by Pick as early as 1908. Brain (1941) also emphasized the importance of the right parietal lobe to the unawareness of the contralateral half of the body and space, although he was also the first to suggest that the low incidence of right-sided neglect after left-hemispheric lesions could be due to a sampling bias in that many patients with left-hemispheric lesions were excluded from studies of hemineglect because of aphasia (Brain, 1945).

Hemineglect was so frequently found in association with lesions of the right parietal cortex (Critchley, 1953) that many neurologists considered it to have localizing value. However, more recent research has thrown some doubt on this point of view as a number of patients have been reported who have a right-sided neglect disorder following a left-hemispheric lesion (Albert, 1973). Other patients have been found to have hemineglect following frontal and basal ganglia lesions (Damasio, Damasio and Chang Chui, 1980; Heilman and Valenstein, 1972a, 1972b).

Another unilateral disorder of space which is considered by many researchers to belong to the same "family" of disorders as hemineglect (Critchley, 1949; Denny-Brown, 1963; Heilman and Valenstein, 1972a, 1972b) is
the unilateral extinction of a stimulus on double simultaneous stimulation (DSS). According to Benton (1956) the method of DSS was first used in 1884 by Jacques Loeb. In 1885, Oppenheim used it to measure tactile extinction and in 1899 Anton used it in the assessment of visual defects. Poppelreuter (1917) referred to the unilateral extinction of a visual stimulus on DSS as "visual inattention". Neurologists "rediscovered" the technique of DSS after it was brought to their attention by Critchley (1949) and Bender (1952). Visual and tactile extinction are now commonly assessed by DSS in the course of a standard neurological examination.

Since the 1940s, there has been a steady build-up in the literature of individual cases and group studies of unilaterally brain-damaged patients with unilateral spatial disorders, and these have been complemented by experimental studies of hemineglect and extinction in both animals and humans. Experimental studies of split-brain patients have provided additional information, as have animal studies using sophisticated single cell recording techniques. As a result of this ever increasing literature, a number of theories have been proposed in an attempt to explain hemineglect and extinction. However, after more than 100 years of observation and experimentation these disorders are still far from understood.
1.2 DEFINITIONS AND PHENOMENOLOGY OF HEMINEGLECT AND UNILATERAL EXTINCTION

Hemineglect and extinction on double simultaneous stimulation are both spatial disorders which follow unilateral brain lesions. While extinction occurs in many patients who demonstrate hemineglect, the relationship of extinction to hemineglect is a matter of some dispute.

Hemineglect (also known as hemi-inattention, unilateral inattention and unilateral spatial neglect) is the failure by a patient with a unilateral lesion to respond or orient to a stimulus presented to the side contralateral to the cerebral lesion. That is, the patient appears to neglect or be unaware of stimuli impinging in the contralesional hemispace, or to the side of the body opposite to the lesion, even in the absence of a sensory or motor defect.

The term 'hemispace' refers to the extracorporeal space to the left or right of the body and head midline. It is distinct from visual field, and from the hand or ear receiving sensory input. For example, if a person places their right arm across their body, their right hand will now be in their left hemispace. Only in the situation where a person aligns body and head and visually fixates directly ahead, do the left and right visual fields and left and right visual hemispaces coincide. If the head or eyes are moved to the left or right, the visual fields are displaced accordingly. The visual hemifields are not, however, tied to eye movements and therefore will no longer
coincide with the visual fields. Whether an individual divides space into right and left hemispheres depending upon the orientation of the head or the body is not clear. There is some experimental evidence that suggests that in normal subjects both head and body orientation contribute to the perception of hemispace (Bradshaw, Nathan, Nettleton, Pierson and Wilson, in press, a; Bradshaw, Nettleton, Nathan and Wilson, in press, b; Pierson, Bradshaw and Nettleton, in press) and when the two are not aligned, the right and left hemispheres are not clearly separated.

Hemineglect can occur in any modality, and in one or more modalities in the same patient. In some patients different behavioural manifestations can be seen at different times. The disorder can be more or less severe, and in some patients it appears to spontaneously resolve while in others it does not. Hemineglect is commonly at its most severe in the early stages of a brain lesion and the most striking symptoms of neglect often diminish as the brain stabilizes in the weeks and months following a non-progressive lesion.

1.21 Visual Hemineglect

Hemineglect is most frequently observed in the visual modality, and while many patients with visual hemineglect also have a hemianopia, many do not. Patients with visual hemineglect may display bizarre behaviours such as eating the food on only one half of their plates and then
complaining that they are hungry, or their neglect may be so unobtrusive that it can only be picked up on drawing tests. Because of the importance of vision to so many every day activities, visual hemineglect is often a debilitating disorder. While moving about either by walking or in a wheelchair the patient will constantly collide with objects on the side contralateral to the brain lesion, often suffering bruises as a consequence. When reading, the patient will miss the words on the contralateral side of the page and will sometimes even miss the letters on one side of a word. Occasionally such a patient will confabulate and fill in the missing words with words of his or her own. Writing is also affected by neglect, and friends must often be puzzled when they receive letters with all the writing squashed into one side of the page. On tasks which involve pointing or searching for objects, patients with visual neglect may entirely miss the objects on their neglected side or take a long time to find them.

Drawing and copying tasks are often used in the assessment of visual hemineglect. While most patients simply fail to complete the contralesional half of the object they are drawing, a few patients neglect to draw the contralesional half of one object yet still draw one half of another object which is situated on the neglected side of the first object (Gainotti, Messerli and Tissot, 1972).
1.22 Auditory Hemineglect

The strict definition of auditory hemineglect is the lack of orienting or response to auditory stimuli which originate in the hemispace contralateral to the patient's cerebral lesion in the absence of a competing stimulus in the opposite hemispace. However, this has to my knowledge, never been reported in the literature. A related disorder which has repeatedly been reported is alloacusis, in which sounds emanating from contralesional hemispace are incorrectly located in ipsilesional hemispace (Wortis and Pfeffer, 1948; Denny-Brown, Meyer and Horenstein, 1952; Battersby, Bender, Pollack and Kahn, 1956; Schott, Jeannerod and Zahin, 1966; Heilman and Watson, 1977).

1.23 Tactile Hemineglect

Tactile hemineglect is also rarely reported in the literature, probably because specialized techniques are required in order to demonstrate it. De Renzi, Faglioni and Scotti (1970) when using a tactile finger maze found that some unilaterally brain-damaged patients failed, or took a long time, to find a marble when it was placed in one of the contralateral corners of the maze. Chedru (1976) asked blindfolded patients to tap the keys all over a teletype keyboard as quickly as possible and in any manner they wished. Patients with right-hemispheric lesions showed a significant preference for right-sided keys. Both of these experiments possibly demonstrate a
true tactile hemineglect, although this is a little
doubtful in the case of Chedru's experiment as it did not
demand an exploration of space but simply required the
tapping of keys independently of their location.

1.24 Hemiasomatagnosia

Hemineglect of the body (hemiasomatagnosia) is often the most dramatic form of hemineglect and can produce some bizarre behaviours. Patients with hemiasomatagnosia almost always have a hemiparesis or a hemianaesthesia, and their body neglect is expressed by their apparent lack of interest in their contralateral body half and their unwillingness to incorporate it into their activities. For example, they might not use or dress their contralateral limbs or shave or apply makeup to the contralateral half of their faces. There are many other clinical phenomena related to the defective awareness of one side of the body (Critchley, 1965), some of which may be manifestations of hemiasomatagnosia. One of the more common forms is anosognosia, which is a denial of hemiplegia. One patient with this disorder, when asked how his arm was replied "It is fine, perhaps just a little tired, but there is nothing wrong with it that a bit of exercise wouldn't cure".
1.25 Emotional and Behavioural Phenomena Associated with Hemineglect

Anosognosia may also be viewed as a hysterical reaction to hemiplegia (Anton, 1899; Corballis, 1983; Schilder, 1935; Weinstein and Kahn, 1955). The elaborate confabulations given by some patients in order to 'explain' their hemiparesis certainly possess a hysterical quality. One example given by Weinstein, Cole, Mitchell and Lyerly (1964) is that of a right-sided hemiparetic patient with hemineglect and dysphasia who said that he had been riding in an open truck in the winter when his right side became frozen. He explained that he tried to call to the driver inside the cab to stop, but the driver could not hear him because of the noise of the engine! Some patients refer to their paralysed limbs in the third person (e.g. "he doesn't want to move" rather than "I can't move my arm") and others use derisive names such as "useless Martha" and "dead meat" when talking about their limbs (Critchley, 1974; Weinstein and Friedland, 1977). In another form of denial patients may believe their paralysed limbs belong to someone else, often the doctor or neuropsychologist! For example, when I asked one patient if she could feel me touching her paralysed left arm, she replied "It must be very difficult for you to write with that sore hand". Critchley (1953) dubbed this 'autoheterosyncasis'.

Patients with hemineglect will occasionally react differently toward people depending on the side they are standing on. One patient of mine seemed very reluctant to
attempt my drawing tests and commented rather sullenly that he "couldn't spare the time for this sort of thing". As I had found him very pleasant and co-operative only the day before, I was rather surprised and asked him if he would rather I sat on his other side. He agreed that he would feel better if I moved to his other side and when I did this, his attitude changed immediately. He said he enjoyed doing my tests and didn't know what had come over him before! I had of course initially been sitting in his neglected hemispace, but it is important to note that this should not have caused him any physical difficulty as he had neither a hemianopia nor a hemiplegia.

Another 'ploy' of patients with hemineglect is to use humour as a vehicle for justifying their hemineglect. One woman who totally denied that she had a paralysed arm and also denied the seriousness of her illness (a right parietal glioma), would attempt to dress herself while hopping around on her right leg, even although she was quite capable of supporting herself on her left leg as well. She would chuckle loudly every time she lost her balance and say "It's a good job I used to be an acrobat in a circus because it helps me put my knickers on when I'm standing on one leg!" This same patient, when asked why she did not put her cardigan on her left arm, said "I'm starting a new off-the-shoulder fashion!"

A paradox in the behaviour of many patients with hemineglect is that while they may deny that they are hemiplegic or have a visual field defect or are ill at all, they will remain in hospital and willingly agree to
examinations, X-rays, laboratory and neuropsychological tests and even surgery (Weinstein and Kahn, 1955). While the patient persists in neglecting one side of space and body, he/she often represents it either verbally or in gesture (Weinstein and Friedland, 1977). When I asked a patient why he did not copy the tree on the left of a scene in one of my visual hemineglect tests (see Chapter 3, test 5) he replied "Well I can see that it is there, but I can't be bothered with it just now." Another patient, when asked why she did not copy the fence to the left of the house in the same test, said "I will draw it if you really want me to, but it is sure to blow down in the next wind!"

It is important to note that many of the more extreme behaviours mentioned here are only found in a minority of patients who have hemineglect. While some symptoms of visual hemineglect can be found in nearly 50% of patients with recent unilateral lesions of the cortex and subadjacent white matter (see Chapter 3, this thesis) only a small proportion of these would exhibit the more bizarre behaviours just described. On the other hand, most patients who demonstrate drawing hemineglect, if questioned, will give some sign that they are not interested in, or would rather not do, the contralateral side of the drawing.

There appears to be little doubt that patients with hemineglect are aware at one cognitive level of the stimuli that they neglect on another level.
1.26 Unilateral Extinction on Double Simultaneous Stimulation

Sensory extinction is the failure of a unilaterally brain-damaged patient to respond to a contralateral stimulus when it is presented simultaneously with a stimulus on the opposite, ipsilateral side. It has been observed in the visual (Anton, 1899; Poppelreuter, 1917), tactile (Loeb, 1885; Oppenheim, 1885) and auditory (Bender and Diamond, 1965; Denny-Brown et al, 1952; Heilman and Valenstein, 1972a) modalities. The disorder, which is commonly termed 'extinction to double simultaneous stimulation' (D.S.S.) can be elicited using touch, pin-prick, auditory, visual, gustatory, vibratory and barognostic stimuli (Bender, 1952; Critchley, 1949; Head and Holmes, 1911). Cross-modal extinction is sometimes observed in patients with organic mental impairment (Bender, 1970; Bender and Diamond, 1970). This form of extinction can be tested using visual-tactile, visual-auditory or auditory-tactile stimuli. Bender (1977) gives an example of a tactile stimulus being extinguished by a visual stimulus. He touches a patient with a severe organic syndrome on the hand and at the same time touches his own face with his other hand. While the patient is looking at him he asks "Where did I touch you?" The patient answers "On my face" and touches his face. If the examiner touches only the patient's hand, he will perceive it correctly. Another form of extinction usually found only in patients with organic mental impairment is
'exosomesthesia' (Shapiro, Fink and Bender, 1952). In this disorder, the patient displaces one of the two stimuli into extrapersonal space. Extinction differs from hemineglect in that it can only be elicited when two stimuli are simultaneously presented, one in each hemifield or on each side of the body. When a stimulus is presented alone in either hemifield, the patient responds correctly. Unlike hemineglect, this disorder does not have associated with it bizarre behavioural manifestations such as denial and confabulation. It appears to be a much less complex disorder than hemineglect. The main feature it has in common with hemineglect is that the stimulus which is not responded to is that which appears in the hemispace contralateral to a unilateral hemispheric lesion. Neither hemineglect nor extinction are dependent upon a sensory deficit, and in fact it is pointless to test a patient for extinction if he/she has a sensory deficit severe enough to account for a lack of response to the stimulus. Occasionally a patient will demonstrate extinction of the ipsilateral stimulus. However, the underlying deficit of ipsilateral extinction may be different from that which underlies contralateral extinction (see Chapter 5 and 6, this thesis). Visual extinction is usually assessed in the following fashion. The examiner sits directly in front of the patient and the patient is asked to fixate by maintaining eye contact with the examiner. The examiner holds up a finger in each of the patient's (and his/her
own) visual fields. The patient is asked to say or point to the finger or fingers moving. If he/she is correct whenever the examiner moves one finger only in either visual field, but responds to only the ipsilesional finger when both fingers are moved simultaneously, he/she is said to exhibit contralateral extinction. Obviously this test can only be carried out on patients who do not have a hemianopia. In some cases when a patient has severe visual hemineglect it is difficult to tell whether he/she has a visual field defect or is exhibiting contralateral neglect when stimulated by D.S.S. Patients with hemineglect will often blink if the examiner suddenly moves his/her hand into the contralateral visual field, whereas a patient with a hemianopia will not.

Tactile extinction is also commonly assessed by D.S.S. The patient is asked to close his or her eyes and to indicate which hand (or cheek, or other part of the body) is being touched. The examiner either lightly touches both hands simultaneously, or one hand alone. If the patient correctly responds to the touch on each hand when it is stimulated in isolation from the other, but frequently fails to respond to the contralateral hand touch on D.S.S., he/she is said to exhibit tactile extinction. D.S.S. can be applied to any opposite and corresponding parts of the body although the hands and cheeks are used most often. It can also be applied to two opposite and different body parts such as the right cheek and the left hand. Some patients will also extinguish the contralateral stimulus under these conditions. In another form of
tactile extinction, patients touched on the side contralateral to their lesion may report that they have been touched in a homologous site on the ipsilateral side of their body. This has been termed 'al aesthesia' or 'allochiria' (Obersteiner, 1881).

According to Schwartz, Marchok and Flynn (1977) and Schwartz, Marchok, Kreinick and Flynn (1979), assessing tactile extinction with the standard D.S.S. technique is unreliable in that it produces many false negatives, and a number of patients who actually exhibit tactile extinction are not picked up. They developed a test called the 'Quality Extinction Test' which, they claimed, was much more sensitive to tactile extinction than D.S.S. The test material consisted of boards covered with different textured materials. The patient was blindfolded and each board was moved across both palms of the patient's hands. The patient's task was to identify the material which he had previously learnt to identify using one hand only. On the practice trials the material was the same on both halves of the board, but on the test trials each half of the board was covered with a different material so that each palm was stimulated by a different texture simultaneously. The patient was not told that he/she might feel two textures. If he/she identified only one of the materials, he/she was said to exhibit tactile extinction of the hand stimulated by the unidentified material. Schwartz et al (1977, 1979) did identify more patients with left and right tactile extinction using this test than when they used the standard D.S.S. technique. However, some of their
results were puzzling in that they found a number of patients with left frontal lesions who exhibited tactile extinction of their left, ipsilateral hands. They suggested this was dependent on the verbal response required, and that a disconnection between the left-hand input and the left hemisphere speech areas was the cause. This ipsilateral tactile extinction exhibits some parallels with the ipsilateral auditory extinction reported in Chapter 5 of this thesis, and it will be discussed in more detail in that chapter as well as in Chapter 6.

The assessment of auditory extinction using D.S.S. is usually carried out in a rather crude fashion where the examiner stands behind the patient and produces sounds either singly or simultaneously on either side of the patient’s head. The auditory stimuli used have included rubbing or snapping of fingers, jangling coins or voice sounds made by two people standing on either side of the patient (Bender and Diamond, 1965; Denny-Brown et al, 1953; Diamond and Bender, 1965; Heilman and Valenstein, 1972a; Heilman, Watson and Schulman, 1974). Another more sophisticated technique is to transmit sounds binaurally or monaurally over earphones. In this thesis (Chapters 5 and 6) I assessed auditory extinction using dichotically presented digits. This differs from the usual method of D.S.S. in that the patient is hearing different stimuli (i.e. different digits) simultaneously in each ear, rather than the same stimuli. In normal subjects a slight left-ear extinction (or right-ear advantage) has been found for dichotically presented verbal material (Kimura, 1961)
and a slight right-ear extinction for some musical and non-verbal sounds (Bever and Chiarello, 1974; Gates and Bradshaw 1977; Kimura, 1964; King and Kimura, 1972). This has been associated with left- and right-hemispheric specialization for verbal and non-verbal stimuli respectively. Patients who have had commissurotomies (split-brain patients) exhibit extinction of left-ear digits when digits are presented dichotically (Milner, Taylor and Sperry, 1968). This is also probably a result of the disconnection of the left-ear verbal input from the left (speech) hemisphere.

However, unilateral damage to a hemisphere, even in areas outside the auditory cortex, seems to result in an over-riding of the normal consequences of hemispheric specialization in dichotic tasks. In these cases, there is often some suppression of material presented to the contralesional ear relative to the material presented to the ipsilesional ear (Milner, 1962; Oxbury and Oxbury, 1969). The phenomenon of auditory extinction has proved to be more complex than its counterparts in the visual and tactile modalities, and it will be discussed in detail in Chapter 5.
1.3 NEUROPATHOLOGY

1.3.1 The Evidence for a Right-Left Hemispheric Difference in Incidence of Hemineglect

The single, most important claim relating to the neuropathology of hemineglect, is that it appears to be more frequent and more severe after right- than after left-hemispheric lesions. If this is true then hemineglect is of special interest because it does not belong uniquely to either of the two main categories of effects related to unilateral lesions. One category consists of deficits reflecting hemispheric specialization but unrelated to one or other side of space; thus lesions to the left hemisphere typically produce aphasia or apraxia, and lesions to the right hemisphere may produce global spatial disorders. The other category reflects hemispheric representation of the contralateral side of space or the contralateral side of the body, but typically does not involve differential hemispheric specialization. For example, damage to the right motor cortex disrupts activities of the left limbs and damage to the left motor cortex disrupts the right limbs. Hemineglect, according to the claim, is a hybrid: it is more frequent and severe following right- than following left-hemispheric lesions, implying hemispheric specialization, yet it affects the contralesional side of space.

Studies which have found a significantly higher incidence of hemineglect following right brain damage
include those by Hecaen and Angeleurgues (1963) who found
hemineglect in 34% of right brain-damaged patients and in
only 2% of left brain-damaged patients, and Gloning,
Gloning and Hoff (1968) who found hemineglect in 31% of
right brain-damaged patients and 2% of left brain-damaged
patients. Both of these studies were carried out on very
large groups of patients with unilateral brain damage.
Weinstein and Cole (1963) found that right-hemispheric
hemineglect outnumbered left-hemispheric hemineglect by 22
to 3, Cohn (1961) found a right-left ratio of 3 to 1, Zarit
and Kahn (1974) found a right-left ratio of 2 to 1, but
Albert (1973) found no significant difference in incidence
of visual hemineglect between left and right brain-damaged
patients.

Why should there be such a variation in incidence of
right- and left-sided neglect in different studies? A
methodological argument which has been put forward by a
number of researchers is that some studies of hemineglect
exclude many patients who have left-hemispheric lesions
because they are aphasic. If all such patients were
included then the incidence of hemineglect after left-
hemispheric lesions might well be higher. (Brain, 1941;
Battersby et al, 1956; Oxbury, Campbell and Oxbury, 1974;
Zarit and Kahn, 1974). Nevertheless, in many studies which
assessed the incidence of hemineglect in an unselected
sample of unilaterally brain damaged patients and in which
simple tests were used in order to reduce the problem of
exclusion of aphasic patients, a higher incidence of
hemineglect was still found after right-hemispheric lesions
(Arrigoni and De Renzi; 1964; Benton, 1969; Costa, Vaughan, Horwitz and Ritter, 1969; Chedru, 1976; Colombo, De Renzi and Faglioni, 1976; De Renzi et al., 1970; Gainotti, 1968; Gainotti et al., 1972; Oxbury et al., 1974). However, the incidence of hemineglect elicited by any test is dependent upon the sensitivity of that test to the disorder. One possibility is that some aspects of the hemineglect disorder are more easily compensated for by patients with left-hemispheric damage if he or she is given time to remedy the deficit. Some evidence in support of this comes from De Renzi et al.'s (1970) study in which patients explored a tactile finger maze in order to find a marble. Patients in both hemispheric groups took longer to find the marble when it was in a corner contralateral to the side of the lesion. However, right brain-damaged patients sometimes failed to find the marble at all when it was in a contralateral corner.

Another possible reason why different frequencies of hemineglect are found in different studies is that the etiology of the lesions and the recency of the lesions may be different. Given that the more striking symptoms of hemineglect often diminish in the weeks directly following a brain lesion (Campbell and Oxbury, 1976; Gainotti, 1968) and assuming that neglect following left-hemispheric lesions tends to be less severe anyway than that following right-hemispheric lesions (Albert, 1973), then one might expect a greater right-left difference in incidence as the time interval between the sustaining of the lesion and the
test session increases. These issues are discussed further in Chapter 3.

1.32 Locus of Lesion Producing Hemineglect and Extinction

In both humans and animals the posterior parietal lobe has been implicated more frequently in neglect than any other cerebral area. In humans the most convincing evidence comes from neurosurgical patients and patients who have undergone computerized tomography (C.T.). Lesions which involved the parieto-occipital and the parieto-temporal-occipital junction were found to be associated with hemineglect more often than lesions in other areas by Bisiach, Luzzatti and Perani (1979), Hecaen and Angelergues (1963), Hecaen, Penfield, Bertrand and Malmo (1956) and Heilman and Watson (1977).

There have been a number of animal studies in which lesions and ablations to specific areas of the cortex have been made in an attempt to produce a neglect syndrome comparable to that found in humans. However, a number of problems arise when comparing animal neglect to human neglect. The disorder which follows lesions to the posterior parietal cortex in monkeys is unilateral extinction on double simultaneous stimulation rather than a neglect of objects presented singly in the contralateral field (Denny-Brown and Chambers, 1958; Ettlinger and Kalsbeck 1962; Heilman, Pandya, Karol and Geschwind, 1972; Schwartz and Eidelberg, 1968). Unlike human neglect, extinction in animals follows lesions to either hemisphere
with equal frequency (Mountcastle, Lynch, Georgopoulos, Sakata and Acuna, 1975) and the disorder is transient (Orem, Schlag-Rey and Schlag, 1973). There are also significant anatomical differences between the parietal lobes of the human and the monkey. Brodmann's areas 39 and 40 do not exist in the monkey, and unlike those of the monkey, the right and left parietal lobes of the human differ in shape.

The frontal cortex has also been associated with hemineglect in man (Damasio et al 1980; Gloning, 1965; Heilman and Valenstein, 1972b; Jenkner and Kutschera, 1965; Silberpfennig, 1941; Stein and Volpe, 1983; Van Der Linden, Seron, Gillet and Bredart, 1980) and extinction in animals (Bianchi, 1895; Kennard, 1939; Latto and Cowey, 1971a, 1971b; Watson, Miller and Heilman, 1978; Welch and Stuteville, 1958). However, De Renzi (1982) comments that most of the human cases of frontal lobe neglect are doubtful and even those cases where the neglect is more convincing (i.e., Silberpfennig, 1961; Van Der Linden et al, 1980) the hemineglect symptomatology is much less pronounced than the neglect found after parietal lobe lesions.

On the other hand, the data from ablation and lesion studies of animals are consistent and convincing, although of course the disorder in these cases is extinction rather than hemineglect. In animals with frontal lesions, the contralateral extinction is thought to be a consequence of the ablation of the frontal eye fields. There is ample evidence from single cell studies that the frontal eye
fields are a visuomotor area as well as an oculomotor area (Mohler, Goldberg and Wurtz, 1973; Wurtz and Mohler, 1976b). It appears that neurons in this area (area 8) can analyse incoming visual messages in terms of their psychological relevance for the animal. In this sense they have a similar function to neurons in area 7 (of the parietal lobe) in the monkey. These also are sensitive to psychologically significant stimuli in the contralateral environment and, according to Robinson, Goldberg and Stanton (1978) and Yin and Mountcastle (1977), could provide a physiological basis for attentional mechanisms.

Human neglect has also been observed after lesions involving the cingulate gyrus (Heilman and Valenstein, 1972b), the basal ganglia (Damasio et al., 1980; Heir, Davis, Richardson and Mohr, 1977) and the thalamus (Watson and Heilman, 1979). In monkeys, the right cingulate gyrus has been ablated with a resultant tactile extinction of the left leg on double simultaneous stimulation (Watson, Heilman, Cauthen and King, 1973). Unilateral lesions of the mesencephalic reticular formation have also resulted in contralateral extinction in the monkey (Reeves and Hagamen, 1971; Watson, Heilman, Miller and King, 1974; Watson et al, 1978), and unilateral lesions of the lateral hypothalamus give rise to a transient extinction disorder in the cat for all sensory modes (Marshall, Turner and Teitelbaum, 1971; Marshall, Richardson and Teitelbaum, 1974; Orem et al., 1973). Unilateral lesions of the superior colliculus in cats and monkeys have been shown to produce trimodal
extinction (Denny-Brown et al., 1952; Sprague and Meikle, 1965).

To summarize, in humans hemineglect has been reported most often following posterior parietal lesions of the right hemisphere. There have also been a number of reports of neglect following frontal lesions and lesions of the cingulate gyrus, the thalamus and the basal ganglia. Hemineglect has been reported after lesions to most of these areas in the left hemisphere as well, although these cases are much less frequent. In animals a similar distribution of lesions emerges with the addition of superior collicular and mesencephalic reticular formation lesions, but the resulting disorder is extinction rather than neglect. Extinction follows left- and right-hemispheric lesions with equal frequency in both animals and humans. A number of related theories of hemineglect have been built around the pattern of human and animal lesions which have been associated with neglect and extinction. The substance of these theories is that hemineglect is the result of a disruption in a cortical-reticular network which mediates arousal and directed attention (Heilman and Valenstein, 1972b; Heilman and Watson, 1977a, 1977b; Mesulam, 1981; Watson et al. 1973). The plausibility of these theories depends on the demonstration that pathways do exist between these various lesion sites and that the entire 'network' forms a functional system. These theories will be described in more detail later in this chapter.
1.4 EXPERIMENTAL EVIDENCE FROM HUMAN STUDIES

Studies on humans which have implications for the understanding of the hemineglect disorders fall into three general categories. There are single case studies and studies of groups of patients that are primarily descriptive; there are experimental studies designed to elucidate particular aspects of the hemineglect disorder; and there are experimental studies on normal subjects and on brain-damaged patients who do not have hemineglect (e.g. split-brain patients) that provide data on brain function of relevance to the study of hemineglect.

1.41 Descriptive Studies

The first descriptions of hemineglect came from reports of individual patients (e.g. Brain, 1941; Damasio et al., 1980; Gloning, 1965; Jackson, 1876; Jenkner and Kutschera, 1965; Silberpfeenning, 1941; Valenstein and Heilman, 1979; Van Der Linden et al., 1980), and individual case studies continue to provide an important source of data. Studies of large and small groups of unselected unilaterally brain-damaged patients have produced a great deal of important, yet often contradictory information on the incidence and severity of hemineglect in different groups of patients, the loci of lesions which result in hemineglect, and the different forms the disorder can take (e.g. Albert, 1973; Battersby et al., 1956; Chain, Leblanc, Chedru, and Lhermitte, 1979; Gainotti et al., 1972; Gloning
et al, 1968; Hecaen and Angelergues, 1963; McFie and Zangwill, 1960). These studies usually test large groups of patients with one or more tests designed to elicit hemineglect. In more recent studies the locus of each patient's lesion is often documented either as a result of observations during neurosurgery or from the appearance of the lesion on C.T. scans (e.g. Bisiach, et al, 1979). The studies reported in Chapters 3 and 6 of this thesis are primarily descriptive.

Two other types of descriptive studies concern the natural history of the neglect disorders (e.g. follow-up studies on hemineglect patients) and the treatment and rehabilitation of hemineglect patients. Unfortunately, there have been very few studies in either of these areas. With regard to follow-up of patients, Gainotti (1968) found hemineglect present in 45% of vascular patients examined within a month after a stroke. This percentage reduced to 20% in a group of patients who were tested one month to three years after their stroke. Campbell and Oxbury (1976) retested at six months a group of six patients who were found to have hemineglect one month post-stroke. While all six patients still showed a preference for right-sided responses on a visual recognition test, only two of the patients still showed neglect on drawing tests. Diller and Weinberg (1977) described a rehabilitation program for patients with hemineglect in which patients were given cues in order to promote the turning of the head and eyes to the neglected side. They concluded that this could achieve a long-term improvement, but as their study included only two
individual cases, it can be considered to have heuristic value only.

1.42 Experimental Studies on Hemineglect Patients

A number of researchers have carried out experiments on particular aspects of the hemineglect disorder using patients who have already demonstrated that they have neglect. These types of experiments can have a number of purposes. They can be simply exploratory with the aim of finding out more about a particular aspect of hemineglect. For example, Gainotti et al. (1972) examined the drawings of patients with hemineglect to see if there were any qualitative differences in the drawings of right and left brain-damaged patients. They concluded that hemineglect following right brain damage was more severe than that following left brain damage, and that while in copying tasks some right brain-damaged patients neglected the left side of a number of individual objects in the same drawing, left brain-damaged patients tended to neglect only objects on the right side of the drawing, and then only if they were small or otherwise insignificant in comparison with the rest of the drawing.

The usual reason for experimenting with patients with hemineglect is to collect data that might provide support for a particular hypothesis of hemineglect. For example, Heilman and his colleagues have designed a number of experiments in an attempt to provide support for their theory that hemineglect is an unilateral attention-arousal
defect. They used electroencephalographic techniques (EEG) to demonstrate that most patients with unilateral neglect have a diffuse ipsilateral slowing (i.e. a decrease in arousal) whereas only a small proportion of aphasic patients demonstrated ipsilateral slowing (Watson, Andriola, and Heilman, 1978). Heilman also found that the visual reaction times of neglect patients were very slow, even when the stimulus was delivered to both fields and the ipsilesional hand performed the response. Warning stimuli failed to reduce this reaction time (Heilman, 1978) even although a warning stimulus normally decreases reaction time by increasing arousal (Lansing, Schwartz, and Lindsley, 1959). A galvanic skin response was also used as a measure of arousal. Hemineglect patients showed a minimal or no galvanic skin response in comparison to aphasic controls even when the electrical stimuli and the response were given to and recorded from the ipsilesional hand (Heilman, Schwartz, and Watson, 1978).

Patients with hemineglect, when asked to bisect a line will often bisect it on the side ipsilesional to the mid-point. If neglect is a deficit in attention they should improve on this task if their attention is first drawn to the end of the line which falls in their neglected hemispace. On the other hand, if neglect is the result of a unilateral akinesia for any act which must be performed in the contralesional hemispace, then drawing the patient's attention to that hemispace should not improve performance on the task. However, performance should be better for lines which are entirely on the non-neglected side of the
patient. Heilman and Valenstein (1979) found support for the unilateral akinesia hypothesis in that hemineglect patients who before bisecting a line read a letter at the end of the line which fell in the neglected hemispace, did not improve on the task. Performance was significantly better, however, when the line was on their non-neglected side. Another possible explanation for this is that patients with neglect have a unilateral memory deficit and even although they attend to the whole line, they 'forget' the neglected end of the line as soon as they start exploring the other end. Heilman, Watson and Schulman (1974) demonstrated that patients with auditory extinction did indeed show a unilateral auditory memory deficit for consonant trigrams presented monotonically. The studies reported in Chapter 5 of this thesis were designed in order to test hypotheses about the deficits underlying ipsilateral auditory extinction.

A third reason for experimenting with hemineglect patients is in order to gain insights into normal cognitive processes. Probably the most significant group of studies which fulfill this purpose are those demonstrating that patients with visual hemineglect neglect the contralesional side of their internal representations of their external world (Bisiach, Capitani, Luzzatti and Perani, 1981; Bisiach and Luzzatti, 1978; Bisiach et al, 1979; De Renzi et al, 1970).

The results of these experiments imply that at some point in our formation of visual images, they are mapped in an analogue fashion across our cerebral hemispheres, at
least with respect to their left and right sides. These experiments also provided a basis for an interesting theory of hemineglect; that it is a 'mutilated representation of space' (De Renzi et al, 1970).

1.43 Experimental Studies on Normal Subjects and Patients Without Neglect

There are many studies on normal subjects which have relevance for the study of hemineglect. These include studies which demonstrate that even normal subjects suppress stimuli lateralized to one side under certain conditions. The most obvious examples of these are the dichotic-listening studies in which normals are shown to have a right-ear advantage for verbal stimuli (Kimura, 1967; Milner, 1962) and a left-ear advantage for some non-verbal stimuli (Kimura, 1964; King and Kimura, 1972). Kimura (1961, 1967) has suggested that this is the result of the suppression of the ipsilateral auditory pathways relative to the contralateral auditory pathways under dichotic conditions. Therefore, verbal stimuli from the right ear are more directly routed to the left (speech) hemisphere than left ear verbal stimuli, and non-verbal stimuli from the left ear are more directly routed to the right hemisphere (which is thought to be dominant for the processing of some non-verbal stimuli) than right ear non-verbal stimuli. However, a number of experiments have shown that there might be a hemispheric advantage for some types of stimuli dependent upon the hemispace they come
from rather than on the particular side of the body the receptive sense organ is located on.

Bowers and Heilman (1980) asked blindfolded subjects to perform a tactile 'line' bisection task, using rods. The best performance was when the left hand was in the left hemispace and the worst when the right hand was in the right hemispace. However, overall the subjects were more accurate when either hand performed the task in the left hemispace. They concluded that both hemisphere-hemispace and hemisphere-hand connections contributed to laterality effects and that the right hemisphere is dominant over the left in the performance of tactile tasks.

Bradshaw et al (in press, a), measured response latencies to vibrotactile stimulation delivered to the forefinger of the left or the right hand placed in the left or the right hemispace. While the two hands did not differ on response latency, both performed better when in the right hemispace. However, when the subjects performed the task with their heads rotated 90° to the right or the left so that their right-left body hemispace was different from their right-left head hemispace, this right hemispace advantage was greatly reduced. A further experiment suggested that the right hemispace advantage was dependent on the motor response and not the sensing of the vibrotactile stimulation. Another study in the auditory modality carried out by the same group (Pierson et al, in press) also incorporated the technique of dissociating body space from head space by having the subjects turn their heads. They found a significant right ear and right
hemispace advantage for vocal shadowing latencies to unilaterally presented competitive verbal stimuli from a single earphone or speaker. However, when the head was rotated 90° so that the auditory stimuli were presented either laterally or front-back with respect to the body, the right ear and right hemispace advantages vanished, indicating that the head and body need to be in alignment in order to obtain right ear and hemispace advantages. When verbal stimuli were played diotically through speakers to the front and back of the subject and a 'dummy' speaker was placed either to the right or left of the subject to create the illusion that the auditory stimuli were coming from all three speakers, a significant right side advantage (i.e. when the dummy speaker was placed on the right) was obtained. This suggested that it was the perceived source and not the actual source of the sound which resulted in an asymmetry. (See also Morais, 1975).

Data from studies of brain-damaged patients who have not been assessed specifically for hemineglect can also provide useful information. Howes and Boller (1975) found that patients with right-hemispheric lesions had slower reaction times than patients with left-hemispheric lesions, and that the slowest reaction times were from patients with right posterior parietal lesions. The wealth of data which has resulted from the study of split-brain patients can also provide useful insights into the nature of hemineglect, although it should not be forgotten that the brain of a candidate for commissurotomy may be abnormal, possibly grossly so. Nevertheless, the study of these
patients can provide information which has relevance for disconnection hypotheses of hemineglect and extinction. For example, split-brain patients demonstrate extinction of left ear verbal stimuli on a dichotic listening task. (Milner, Taylor and Sperry, 1968). A similar phenomenon has been found in patients with left posterior and frontal lesions (see Chapter 5 of this thesis). One possibility is that in both cases this left ear extinction is due to a disconnection of the left ear input from the left-hemispheric language areas (Damasio and Damasio, 1979; Sparks, Goodglass and Nickel, 1970).

1.5 Experimental Evidence from Animal Studies

Bianchi (1895) was the first to produce a hemineglect disorder in animals (monkeys and dogs) by lesioning the prefrontal cortex. This resulted in an ipsiversive rotation lasting for one to two weeks and an apparent disinclination to use the contralesional limb except when the other limb was immobilized. This was accompanied by an unresponsiveness to contralateral visual stimuli which lasted up to three weeks. Since then, hemineglect, or more correctly extinction on double simultaneous stimulation, has been produced in cats, dogs, rats and monkeys many times as a result of unilateral ablations of the parietal and frontal cortex, the cingulate gyrus, the mesencephalic reticular formation, the lateral hypothalamus and the superior colliculus (see Section 1.32, this chapter, for details of these studies).
Many important contributions to the understanding of hemineglect and extinction have been made by researchers who use animals in order to demonstrate the connections between different areas of the cerebrum. These types of data are often used in support of various hypotheses of hemineglect. In particular the attention-arousal corticolimbic loop hypotheses depend upon the demonstration of interconnections between the different areas which have been associated with hemineglect. Pandya and Kuypers (1969) have shown that the inferior parietal lobule, the dorsolateral frontal lobe and the cingulate gyrus all have prominent corticocortical interconnections. All three areas are connected directly or indirectly to more than one sensory modality and all have prominent connections with the brainstem reticular formation (Astruc, 1971; French, Hernandez-Peon, and Livingston, 1955; Nauta, 1964; Segundo, Naguet, and Buser, 1955). The superior colliculus also projects to the mesencephalic reticular formation, and the dorsolateral frontal lobe projects to the colliculus (Astruc, 1971). The substantia innominata and the lateral hypothalamus receive fibres from the mesencephalic reticular formation and project to the cortex (Kievit and Kuypers, 1975).

In recent years it has become possible to record electrophysiological activity from single neurons in the cortex of animals while they are performing previously learned behaviours of appropriate types. Mountcastle and Lynch and their colleagues (Lynch, Acuna, Sakata, Georgopoulos and Mountcastle, 1973a; Lynch, Sakata,
Georgopoulos and Mountcastle, 1973b; Lynch, Mountcastle, Talbot and Yin, 1977; Mountcastle, 1978; Lynch, 1980) and Robinson and Goldberg and their colleagues (Goldberg and Robinson, 1977; Robinson and Goldberg, 1977; Robinson, Goldberg and Stanton, 1978) have studied the neural activity of several thousands of cells in the superior and inferior parietal lobules of macaque monkeys. While it is clear that many diverse neural mechanisms are located in part in the parietal cortex, the function of some classes of neurons in these areas are particularly relevant to hemineglect. In area 5 (superior parietal cortex) were a group of cells comprising about 10% of the total, that apparently had no sensory input but fired only if the movement of the contralateral arm satisfied some appetitive drive such as grooming, picking up food, or tripping a switch that brought a liquid reward. In area 7 (inferior parietal lobule) the largest single class of cells, the 'visual fixation neurons' which comprised more than 50% of the total studied by Mountcastle's group, were continuously active as long as the monkey looked at an interesting visual target such as food when the animal was hungry. In order to be effective, the stimulus of interest had to be presented within the animal's reach. The majority of these cells were 'partial-field' fixation cells and these were more active when the target was in one-half or one-quarter of the visual field. Two other general classes of cells in the inferior parietal lobule are called 'visual tracking neurons' and 'saccade neurons'. Both these classes of cells are also dependent upon attentional or motivational
factors. Mountcastle and his colleagues proposed that these three classes of cells provide a mechanism for the generation of action as well as for the processing of sensory information. They suggested that parietal cells were directing visual attention by commanding eye movements.

Goldberg and Robinson (1977), while agreeing with many of Mountcastle's findings, disagreed about the command function of the parietal neurons. Rather, they proposed that attention is a process of stimulus selection for behaviour that will probably, but not necessarily, result in the animal's orientation to an attended stimulus. In other words, whereas Mountcastle sees a causal (command) relationship between attention and movement, Goldberg and Robinson believe attention is dissociable from movement. They relate this to visual hemineglect by suggesting that parietal lesions can result in an inability to select from the environment a stimulus of psychological significance.

Hemineglect and extinction resulting from frontal lobe lesions also finds support from single cell studies, in this case of the frontal eye fields. Mohler et al., (1978) recorded from an area anterior to the arcuate sulcus and around the caudal end of the sulcus principalis, and found neurons that discharge when a visual stimulus is presented in a definite sector of the contralateral visual field. If the stimulus becomes a target for a saccadic eye movement, the cells show an increased discharge (Wurtz and Mohler, 1976b). In this they are similar to cells in the superficial layers of the superior colliculus (Wurtz and
Mohler, 1976a) and also to cells in the inferior parietal lobule (Robinson et al, 1978). Cells in the striate cortex do not have this property.

While animal studies can suggest mechanisms which might underlie neglect in humans, there are a number of problems which must be taken into account when making inferences about human neglect from studies of animals with experimentally produced extinction. Lesions made by suction or freezing cannot be identical to neoplasms or vascular diseases in humans. Added to this, the neural organization of animals is different from that of humans, particularly with respect to the parietal lobes and the limbic representation in the cortex. The very fact that unilateral lesions usually produce only a transient extinction disorder in animals, whereas in humans they can produce long-term hemineglect, is reason enough to proceed cautiously when making comparisons between animal and human neglect disorders.
1.6 THEORIES OF HEMINGLECT AND EXTINCTION

1.6.1 Sensory and Perceptual Hypotheses

There have been several theories of neglect which relate it to defective sensation. Brain (1941) believed that the parietal lobes contained the body schema, and that since spatial perception was also mediated by the parietal lobes a patient with a parietal lesion would fail to recognize not only half of his/her body but also half of space. Denny-Brown and Banker (1954) ascribed the neglect syndrome to a defect in spatial summation (amorphosynthesis). They thought that the parietal lobes were important in cortical sensation, and damage to them resulted in an inability to synthesize more than a few properties of a sensory stimulus at any one time.

Battersby et al. (1956) enlarged the sensory deprivation hypothesis of hemineglect to include a general mental deterioration. That is, they proposed that hemineglect was the result of a unilateral deprivation of sensations superimposed upon a background of mental deficiency. Sprague, Chambers and Stellar (1961) provided some support for this when they induced extinction in cats by lesioning the lateral portion of the mesencephalon. As such lesions also disrupt the ascending sensory pathways, Sprague et al. (1961) concluded that extinction was caused by a loss of patterned sensory input to the neocortex. Eidelberg and Schwartz (1971) more recently proposed that extinction was a passive phenomenon caused by a quantita-
tively asymmetrical sensory input to the two hemispheres or by differences between the two hemispheres in the functional mass of the area which mediate somatic sensation.

However, while these hypotheses of hemineglect and extinction based on sensory deficits may provide partial explanation for some forms of extinction, they are inadequate as explanations for hemineglect. Hemineglect implies a defective scanning of space and not simply disordered perceptions. The well established finding that many patients with visual hemineglect do not have a hemianopia (De Renzi, 1982) contradicts a simple sensory hypothesis. Most patients with hemianopias quickly compensate by learning to turn the head and eyes towards the affected side. However, the hemianopic patient with hemineglect does not learn to compensate and behaves as if one half of space simply does not exist.

1.62 Interhemispheric Hypotheses

Most of the hypotheses in this category have been proposed to explain extinction rather than hemineglect. In 1945, Bender and Furlow suggested that a defect in sensation in one of their patients was increased by the phenomenon of rivalry and dominance by the intact hemisphere. Furmanski (1950), Nathan (1946) and Reider (1946) explained extinction by postulating that the normal side was suppressing the abnormal side. Birch, Belmont and Karp (1967) proposed that the damaged hemisphere is much
slower at processing information than the undamaged hemisphere and as a consequence it is subject to interference from the normal side. They demonstrated in support of this that stimulation of the normal side before stimulation of the abnormal side induced extinction, whereas when the abnormal side was stimulated first, extinction was reduced.

Oculomotor disorders are frequently associated with neglect. Schott et al. (1966) and Kinsbourne (1970, 1977) postulated that neglect was the consequence of an imbalance between the control centres for head and eye turning which are present in both hemispheres. When one of these centres is suddenly inactivated it results in the disinhibition of the healthy hemisphere and an involuntary turning of the head and eyes to the opposite side. While this phenomenon only lasts a few days or weeks, the patient still displays preference for looking towards the ipsilesional side, particularly when confronted with multiple displays.

Kinsbourne (1974) extended this hypothesis to include a non-motor imbalance of attention in the absence of any gaze shift. That is, unilateral cerebral damage would drive attention to the normal hemisphere (i.e. to the side of space contralateral to it) even in situations where no oculomotor activity was involved (e.g. dichotic listening, tasks). Kinsbourne attempted to explain the prevalence of left-sided neglect by suggesting that tests for neglect activated the left hemisphere because of the verbal set adopted by the patient when interacting with the tester or doctor. This activation of the left hemisphere would cause
the patient's gaze to turn to the contralateral right side. Conversely, if the patient were engaged in non-verbal visuospatial tasks, the right hemisphere would be activated resulting in a shift of attention to the left. That is, left-sided neglect is more frequently observed because the patient is normally engaged in some verbal activity which will enhance a right shift of attention in patients with right-hemispheric damage, and counteract any left shift of attention caused by left-hemispheric damage. However, this theory is unconvincing as in many studies patients have been tested for neglect with visuospatial non-verbal tasks yet the deficit is still apparent (De Renzi, 1982).

Kinsbourne's (1970, 1977) reciprocal inhibition model also implies that patients should be hyperattentive to ipsilesional stimuli as the normal hemisphere is released from inhibition by the damaged hemisphere. However, Heilman and Watson (1977b) point out that hemineglect patients often appear to be generally hypokinetic, and they have carried out a number of experiments which suggest just this (Heilman, 1978; Heilman et al, 1978; Watson et al, 1978; see Section 1.32, this chapter). They think that neglect is due to the decreased activity of the lesioned hemisphere rather than to the increased activity of the normal hemisphere, and that patients are hypokinetic because they have bilateral arousal deficits which are more severe on the lesioned side than on the unlesioned side.
1.63 Hypotheses of Defective Attentional Activation

A deficit of attention has long been implicated in the hemineglect disorders (Brain 1941; Critchley, 1949; Poppelreuter, 1917). More recently, Heilman and his colleagues have expanded and strengthened this hypothesis by supporting it with anatomical and physiological data from both animal and human studies. They propose that hemineglect results from a disruption of a cortiolimbic-reticular loop (similar to that proposed by Sokolov, 1963) that when intact activates the orienting reflex in response to novel or meaningful stimuli (Heilman and Watson, 1977a). This results in hypoarousal and the animal or human is unable to deal with sensory events occurring in the contralateral half of space. The main support for this hypothesis derives from neuroanatomical and neurophysiological animal studies that show that the cortical and subcortical lesions associated with extinction and hemineglect are interconnected in a functional network (Mesulam, 1981) and that neurons in the inferior parietal lobule are specialised for encoding the psychological impact of sensory events occurring in the contralateral part of extrapersonal space (Lynch, 1980; Mountcastle et al., 1975; Robinson et al., 1978). These studies are described in Section 4.1 of this chapter.

Mesulam (1981, 1983) collated these data and the evidence from animal and human studies of extinction and hemineglect into a comprehensive and persuasive "cortical network for directed attention and unilateral neglect".
He postulated that the three major cerebral areas in this cortical network each had a particular role to play in unilateral neglect. The inferior parietal cortex may contain a sensory template of the extrapersonal world; the frontal cortex, including the frontal eye fields may contain a motor map for the distribution of scanning, orienting and exploration within the extrapersonal world, and the cingulate cortex and surrounding areas may contain a motivational map for the distribution of interest and expectancy. The arousal level of each of these areas is regulated by input from the reticular formation. While each of these three representations (sensory, motor and limbic) is mostly responsive to the contralateral hemispace, ipsilateral representation is also present. For the effective distribution of directed attention in extrapersonal space, all three cortical areas, and the reticular formation, must be intact. If one area is damaged, or the connections between the areas are disrupted, contralateral neglect may result. Damage to a particular area may result in a particular clinical form of hemineglect. Damage to more than one area might result in a more severe clinical form of hemineglect (Mesulam, 1983). In order to explain the more severe, and possibly more frequent occurrence of hemineglect after right- than after left-hemispheric lesions in humans, Heilman and Van Den Abell (1980) and Mesulam (1983) have postulated that the right hemisphere is dominant for attention. Data from EEG and evoked potential studies of normal humans (Desmedt, 1977; Heilman and Van Den Abell, 1980) and reaction times
of patients with unilateral lesions (Howes and Boller, 1975) have suggested that the right hemisphere directs attention to both sides of space although the dominant tendency is to direct attention to the contralateral hemispace, whereas the left hemisphere directs attention almost exclusively to the right side of space. Therefore, lesions confined to the left hemisphere are less likely to result in contralateral neglect because the intact right hemisphere can utilize the neural mechanisms for directing attention to the right side. However, as the left hemisphere does not have the ability to direct attention to the left side, when the right hemisphere is lesioned a severe left-sided neglect results.

1.64 Hemineglect; a Mutilated Representation of Space?

De Renzi et al (1970) were the first to suggest that hemineglect may be the result of a "mutilated representation of space". They noted that some patients with right-hemispheric lesions, when exploring a tactile maze for a marble, appeared to be unaware that the left side of space existed. This idea was supported by Bisiach and his colleagues who demonstrated that patients with left-sided visual neglect neglected the left side of images retrieved from memory (Bisiach et al, 1981; Bisiach and Luzzatti, 1978) and the left sides of images constructed from immediate external input (Bisiach et al, 1979). These studies are discussed in detail in Chapter 4 of this thesis.
The notion that contralateral neglect also occurs in internal representations of the external world implies that at some level of processing in the intact brain, images are represented analogically. That is, when we form visual images we do so by mapping our interpretations or memories of the left side of the external world onto our right hemispheres, and our interpretations or memories of the right side of space onto our left hemispheres.

When there is unilateral damage to the cortical areas involved in this level of the imagery process, the image mediated by that hemisphere is disrupted. That is, the image of the contralesional half of space is 'mutilated'. Alternatively, the disruptions may occur during a mental scanning of an analogical representation. The patient with unilateral cerebral damage may still be able to form a complete visual 'picture' of the external world by mapping it in analogue fashion across the two hemispheres, but if the lesion interferes unilaterally with the ability to scan internal representations, the patient's recall of the image of the contralesional side of external space may be disordered.

While this data from hemineglect patients provide support for an analogue theory of imagery, a propositional theory of imagery is not ruled out (Anderson, 1978). For example, there is no reason to suppose images are encoded or stored analogically. This part of the imagery process may equally well be carried out in propositional form. In the clinical literature there has been at least one case study reported that implies that there are both
propositional and analogue stages in visual imagery. Basso, Bisiach, and Luzzatti (1980) described a patient with a left-hemispheric lesion who appeared to have 'lost' the ability to form visual 'pictures' and had to rely solely on propositional information when he was constructing internal representations.

The "mutilated representation of space" notion of hemineglect has the implication that hemineglect transcends specific modalities and that all modalities which are represented in the neglected side of the image are likely to be disrupted. However, although the original idea was first stimulated by a tactile maze experiment (De Renzi et al, 1970), to date the only convincing data have come from studies involving visual imagery.
1.7 ORGANIZATION OF THE THESIS

Chapter 2 deals with the general methodology used in the various studies reported in this thesis, with a particular emphasis on the criteria used for the selection of brain-damaged patients. Chapters 3 and 4 are both concerned with visual hemineglect. Chapter 3 is primarily a descriptive study which provides data on the incidence and severity of visual hemineglect in a large sample of patients. It also provides data on the loci of lesions which produce hemineglect, and these show an unexpected anterior-posterior interhemispheric difference in the cerebral areas which result in neglect. Chapter 4 is the report of an experiment on the neglect of visual images. The experimental design follows that of Bisiach et al (1979), but whereas that study only involved patients with right-hemispheric lesions, this study also involved patients with left-hemispheric lesions.

Chapter 5 is concerned with auditory extinction, and in particular the phenomenon of ipsilateral auditory extinction which is exhibited by some patients with left-hemispheric lesions. Possible mechanisms which might underlie this disorder are discussed.

Chapter 6 is a study of the incidence of multimodal hemineglect and extinction. It examines whether multimodal neglect is the result of independent neglect disorders in different modalities which occur together in the same patient, or whether it is a single disorder caused by the same underlying deficit.
Chapter 7 is a summary of the studies and their results and some conclusions are drawn about the ways these results might add to general theories of hemineglect.

Chapters 3 and 5 (Experiment 1) are presented essentially in the forms in which they were submitted and revised for publication. Therefore there is some repetition, particularly in the 'Method' sections of these chapters.
2. GENERAL METHODOLOGY

2.1 THE NEUROPSYCHOLOGICAL INVESTIGATION

2.2 MAXIMIZING PERFORMANCE LEVELS ON NEUROPSYCHOLOGICAL TESTS
   2.21 Test Anxiety and Emotional Factors Related to Illness and Hospitalization
   2.22 Fatigue, Distractibility, Depression, Frustration and Decreased Attention Span

2.3 THE EFFECTS OF DRUG THERAPY ON TEST PERFORMANCE

2.4 THE PATIENT SAMPLE

2.5 THE LESIONS
   2.51 The Significance of Active Lesions of Recent Etiology for the Study of Hemineglect
   2.52 The Diagnosis and Measurement of Lesions
   2.53 General Descriptions of the Types of Lesions Sustained by the Patients Included in These Studies
      (a) Neoplasms and Cerebral Abscesses
      (b) Lesions Resulting From Cerebrovascular Diseases and Head Injuries
The aim of this chapter is to outline the various clinical and methodological considerations which are important in the design of a study involving a clinical population.

2.1 THE NEUROPSYCHOLOGICAL INVESTIGATION

Hemineglect and extinction are neurological disorders in that they are undoubtedly the result of neurological damage. However, it is becoming increasingly apparent that while standard neurological techniques may be appropriate for the assessment of extinction, neurological methods alone are not always sufficient to assess either the presence or severity of hemineglect. Added to this, patients with hemineglect often display complex behaviours which may supply clues to the cognitive deficits underlying hemineglect, and these behaviours are more appropriately assessed by psychological methods.

The neuropsychological investigation combines neurological and psychological techniques and theories and is aimed at obtaining data on changes in human behaviours which occur specifically as a result of cerebral lesions. This information, when used in conjunction with information obtained from neurological and radiological investigations, provides the most appropriate data base for studying human hemineglect.

There are two major components in the neuropsychological investigation; neuropsychological tests which provide quantitative as well as qualitative data,
and observations of patient behaviours which provide qualitative data. Both these components form an essential part of any neuropsychological assessment, and this is particularly true in the case of hemineglect. Many patients with hemineglect have unilateral sensory deficits which must be assessed and differentiated from hemineglect. A complicating factor is that a sensory deficit may often exacerbate the neglect. In the case of visual and auditory hemineglect and extinction there may be no sensory deficit at all. However in the case of tactile extinction and hemiasomatognosia a sensory or motor deficit is often present while not being causal.

Neuropsychological tests can provide information about both quantitative and qualitative aspects of neglect. For example, Albert (1973) designed a test in which the patient was asked to cross out lines scattered apparently randomly over a page. The number of lines left uncrossed on one side of the page relative to the number of lines left uncrossed on the other side of the page can be used as a severity measure. The style in which patients copy a drawing can also provide information about the deficit. When copying a number of individual objects displayed on one page, some patients may neglect to copy all the objects on one side of the page while others may copy half of each object.

The every day behaviour of patients as well as their behaviour while they are performing tests can also supply valuable qualitative data. Some patients may neglect to eat the meal on one half of their plates but remedy this
when it is pointed out. Others may not eat the other half until the plate is rotated 180° so that the uneaten half is on their non-neglected side. The conversation of patients with hemineglect can also be instructive. For example, one patient, when asked why she did not draw the fence to the left of a house when copying a drawing, replied that she would copy it if she must but it would probably blow down in the next wind! Comments like this suggest that the patient is aware of the stimuli on the neglected side but does not 'wish' to respond to them.

Both group studies and single case studies provide useful information about hemineglect. While group studies can tell us what symptoms patients with hemineglect have in common as well as provide statistical data on incidence of hemineglect and its association with lesions in different cerebral locations, individual case studies can supply us with a wealth of qualitative data and information about the different forms hemineglect can take. Single case studies often have heuristic value in that they stimulate ideas about the possible nature of hemineglect that can then be translated into hypotheses and tested on groups of patients.
2.2 MAXIMIZING PERFORMANCE LEVELS ON NEUROPSYCHOLOGICAL TESTS

There are a number of guidelines that can help to maximize the chances of obtaining valid test results from neurological patients. While neuropsychological tests are psychological in the sense that they are measuring behaviour, their purpose is not to measure psychological disturbance but to measure neurological disturbance. In order to fulfill this purpose the patient should be encouraged to perform to the best of his or her ability so that any deficit revealed is likely to be a direct result of the brain lesion uncontaminated by the effects of a threatening emotional climate, environmental distractions or fatigue (Lezak, 1976). Added to this the test should ideally be presented in a standardized form so that scores obtained in different test administrations can be compared. This can be difficult to adhere to if test instructions are complicated or long, or if tests are timed. Many patients with brain damage may have difficulty fully comprehending verbal instructions and the test performance of patients with left-hemispheric lesions in particular may be constrained by their inability to comprehend verbal instructions. Timed tests should be avoided as brain damage in general increases response time and patients may be slow to perform a test as a result of a motor rather than a cognitive deficit (Lezak, 1976).
In the neuropsychological investigations which form the basis of this thesis, a number of procedures were carried out in an attempt to maximize each patient's performance and to minimize the number of patients who had to be excluded because they could not cope with the test conditions.

2.21 Test Anxiety and Emotional Factors Related to Illness and Hospitalization

Tests of any kind often cause anxiety, and psychological tests are no exception. Neurological patients who are aware that they are in hospital because of a brain lesion, may find requests to perform psychological tests very threatening. From their point of view the very fact that they are being asked to do 'mental' tests tends to confirm their worst fears that they are 'going crazy'. Added to this many neurological patients have already undergone a number of often frightening procedures such as angiography. Even non-invasive procedures such as computerized tomography can be overwhelming for some patients who are unaccustomed to hospitals. Most patients share a room with a number of other patients, many of whom may be severely debilitated as a result of their brain damage. Even interactions with other patients who are behaving quite normally can be disturbing at first if those patients have shaven heads with the operation site exposed, or if their heads are heavily bandaged. The impact of such a disturbing, and for the patient,
threatening environment can be very deleterious to the patient's test performance. High levels of anxiety can result in scrambled or blocked words, memory failure and the inability to think or respond quickly (Rabin, 1965; Wrightsman, 1962).

In an attempt to help patients relax, I always spent some time talking with them before asking them if they would be willing to help me by doing some simple drawings. Initially I introduced myself to the patient and explained that I was a PhD student and was hoping they would be able to help me with my research. This invariably produced a positive response, and as they were helping me rather than the reverse, this possibly made the situation less threatening. I then spent 10 to 30 minutes talking to them on any topic of conversation that came up. This conversation often included other patients in the room, as well as the nurses and anyone else who came by. This time not only gave the patients an opportunity to get to know me, but also allowed me to observe their behaviour in a non-test situation. During this conversation period I explained in general terms what a neuropsychologist does and in particular tried to allay any fears that brain damage means 'going crazy'. I also explained that I was giving many patients my tests, although some would have no trouble with them. This often stimulated patients to tell me the various neurological problems they had been having lately, and this information often proved very useful.

By the time I actually presented the paper and pencil tests described in Chapter 3, the patient was usually
quite eager to 'have a go'. These tests took no longer than 5 to 10 minutes. If the patient was still motivated and not tired, I then gave the tactile extinction tests (see Chapter 6) and one or more of the auditory tests described in Chapter 5. More often than not I left the auditory tests until the next morning, as these take quite a long time and require the patient's constant attention. I always spent some time talking with the patient about the test results when I had completed all the tests. At this point I made an effort to explain to the patient, at a level he or she could understand, about any specific neuropsychological problems I had observed or they were worried about. Talking about their deficits in an objective fashion seemed to be helpful in allaying unfounded fears about other deficits.

2.22 Fatigue, Distractibility, Depression, Frustration and Decreased Attention Span

Brain-damaged patients tend to tire easily and while many will be aware of this and tell the tester when they feel tired, some will not be aware of it. Therefore, I was always alert for signs of fatigue such as restlessness, increased distractibility, increased droop on the paralyzed side of the patient's face or a slowing down of responses. Patients were always tested in the morning on the assumption that they should be more rested at this time. Distractibility is also a frequent problem. To minimize extraneous stimulation I usually pulled the
curtain around the bed when actually testing, and if the patient was mobile, he or she was taken to a quiet room away from the main ward for the auditory tests. The computer tests described in Chapter 4 were always given in a quiet room separate from the ward with only the patient and myself present.

Some patients became depressed or frustrated when they were unable to perform tests to their satisfaction. This was avoided to a large extent by frequently encouraging and praising the patient, especially when he or she performed a test satisfactorily. Depression and frustration could often be alleviated by encouraging the patient to talk about his or her feelings and demonstrating understanding and empathy.

The attention span of many patients is often shorter than normal and is further decreased by fatigue and distractibility. The best way to deal with this was to be sensitive to it and break test sessions up into smaller sessions whenever necessary. Test instructions were kept simple and short, and when there were a large number of trials, the instructions were repeated at appropriate intervals throughout the test. While the ideal is to have standardized instructions, there must be room for flexibility with brain-damaged patients. The instructions may need to be reframed in different words for some patients. It is also much less threatening if instructions are given in a conversational manner rather than read word for word. The 'Shadowing Tones' test described in Chapter 5 required quite complex instructions and these were
recorded on the test tape in my voice. After every instruction the patients were asked, via the tape, if they understood, and if they looked puzzled or responded that they did not understand, the cassette tape was stopped and the instructions were given again, this time 'in person', and if necessary using different words and appropriate hand signals.
2.3 THE EFFECTS OF DRUG THERAPY ON TEST PERFORMANCE

The vast majority of neurological patients with recent unilateral lesions are on drugs. Many patients are having corticosteroid therapy for cerebral oedema, and a few patients are also on diuretics. At the time of neuropsychological testing, most patients on corticosteroid drugs have only been taking them a matter of days. Therefore side effects are likely to be minimal. Most patients with tumours, abscesses or infarcts are on anticonvulsant drugs. Drowsiness, headache, dizziness, fatigue and ataxia can occur as side effects of these. Patients with cerebrovascular disease are less likely to be on anticonvulsants but are usually taking B-adrenergic receptor blocking drugs or other antihypertensives and diuretics. Some of these can also have side effects of drowsiness, headache, dizziness, nausea and vomiting.

Because the effects of these drugs on an individual's test performance cannot be assessed as a pre-drug baseline is not obtainable, any effects must be accepted as a constant factor across the majority of patients. In order to avoid the more obvious effects of drugs or of the lesion itself on test performance, testing was postponed if the patient had a headache, or felt nauseous or generally unwell, fatigued or lethargic. Patients were always asked specifically about these symptoms before testing, and asked to inform me if any of them occurred during testing so that we could stop and complete the tests another day.
While the test performance of some patients may be globally depressed by some drugs (particularly anticonvulsants) they are unlikely to influence the expression of hemineglect. Hemineglect is expressed as a difference in responses to stimuli on each side of the patient, and while a global depression or hypoarousal of the central nervous system may slow down responses in general, it is unlikely to have a significant effect on the differential between responses to left- and right-sided stimuli.
2.4 THE PATIENT SAMPLE

All patients who took part in the studies reported in this thesis were in-patients at some time over an 18 month period of the Neurology and Neurosurgery Wards of Auckland Hospital, New Zealand. Neuropsychological testing was carried out on the wards or in a quiet room adjacent to the main wards. All the neurologists and neurosurgeons connected with the wards agreed to my testing any of their patients. Participation in the study was entirely voluntary and all patients were made aware of this. However, every patient who was approached agreed to participate. Therefore, the withholding of permission for testing by either the medical practitioner or the patient was not a factor in exclusion of patients from the studies.

Factors which did influence selection were:
(a) the type of cerebral lesion the patient had, and
(b) the mental status of the patient.

Only lesions that were solitary, confined to one hemisphere, and focal, were considered suitable. Patients were only tested if they were generally alert and showed no signs of any generalized intellectual deterioration. Patients with a known or suspected subnormal premorbid I.Q. were also excluded. Patients were usually in hospital because they were exhibiting neurological symptoms as a result of their cerebral lesion and therefore their lesions could be said to be 'active'. The testing of patients with 'active' lesions, usually of relatively recent etiology, is of significance to these studies and therefore will be
discussed in detail in the next section.

Within these criteria the patient sample was unselected. That is, every patient who was mentally alert and had a solitary, clearly definable unilateral active lesion involving the cortex, subadjacent white matter or basal ganglia, had an equal chance of inclusion in the studies. As the number of patients in the Neurology and Neurosurgery Wards is very large at any one time (a maximum of 51 neurosurgical patients and 28 neurological patients), it was not always possible for me to test all suitable patients. However, I did not select patients to test from among the suitable patients but simply tested as many of the patients as time permitted in the order in which they came to my notice and were available for testing.

I used various sources of information to pinpoint suitable patients. I attended the weekly 'imagery conference' where C.T. scans and angiographs of current patients were shown and discussed by the neurologists, neurosurgeons and radiologists. I went through the Ward notes of all current patients at least twice a week and took note of the daily operating schedules which specified the type of neurosurgical operation to be performed and the location of the lesion. Nursing and medical staff often informed me about new patients they thought might be suitable.

Reports kept as part of the Ward notes and written by neurologists, neurosurgeons, radiologists, pathologists, nursing staff, physiotherapists, occupational therapists and social workers, were all taken into account in my
overall assessment of the diagnosis of the cerebral lesion, the treatment of the patient and the behaviour of the patient. For every patient I recorded this information and other information I obtained from my own observations and testing of the patient on standardized forms which can be found in the Appendix. These forms are called 'Subject information - general', 'Subject information - neuropsychological 1' and 'Subject information - neuropsychological 2'. On these forms the patient was identified only by the side of his or her lesion and a number (e.g. LBD 16; RBD 23).
2.5 THE LESIONS

2.5.1 The Significance of Active Lesions of Recent Etiology for the Study of Hemineglect

Hemineglect is a disorder which is usually at its most severe stage in patients with active or unstable lesions and lesions of recent or traumatic etiology. Many patients demonstrate some degree of recovery from hemineglect in the first few weeks or months following their lesion, and some recover completely (Campbell and Oxbury, 1976; Gainotti, 1968). It follows from this that if one wishes to assess the potential for hemineglect following lesions to different areas of the brain and not recovery rates from it, patients should be assessed when they are most vulnerable to the disorder. Therefore, patients with recent or active lesions were selected for study on the assumption that this group would exhibit the highest frequency of hemineglect, as well as the most severe and therefore unmistakable symptomatology.

There are, however, a number of problems associated with the study of patients with recent, unstable lesions. The boundaries of certain types of lesions (e.g. gliomas) are often difficult to define although the general cerebral area affected can usually be estimated by the appearance of the lesion on C.T. scan. Transient symptoms often result from brain oedema and pressure from enlarged ventricles, and these can resolve rapidly as a result of drug (e.g. corticosteroid) treatment or the insertion of a
ventricular shunt. With space-occupying lesions, brain herniations may occur, and mid-line shift and vascular spasm can result in cerebral areas remote from the actual lesion site being affected.

2.52 The Diagnosis and Measurement of Lesions

Lesions of all patients were located by Computerized Tomography (C.T.) and in some cases by angiography as well. C.T. was performed in a Delta 2020 Scanner and consecutive cuts oriented parallel to the superior orbitomeatal line were obtained. Patients with lesions which on C.T. scan or at operation showed any indication of involvement of the other hemisphere were excluded. Patients with more than one lesion were also excluded, and this included patients who on angiography were found to have two or more aneurysms, even although only one appeared to be symptomatic. Neuropsychological testing was usually carried out within one to three days of the C.T. scan being taken, and therefore any oedema present on C.T. scan was included in estimates of the size and site of the lesion. Lesions were estimated as large, medium or small depending on a subjective estimate of the size of the lesion as seen on C.T. scan. As a general guide lesions which in cross-sections on at least one C.T. section involved one-quarter or more of one hemisphere were graded as large, those involving one-fifth to one-quarter were graded as medium, and those involving less than one-fifth of the hemisphere were graded as small. All lesions were
diagnosed by neurologists or neurosurgeons and in many cases by pathologists following excision or biopsy of brain or tumour tissue. The site of the lesion was inferred from its position on C.T. scan.

The cortical and subcortical areas of the brain were divided up differently depending on the aims of the particular study. When assessing the incidence of visual hemineglect following lesions to different cerebral loci (see Chapter 3), each lesion was classified as being either anterior (wholly anterior to the central sulcus), anterior/posterior (on both sides of the central sulcus), or posterior (wholly posterior to the central sulcus). In Chapter 5, the cerebral locations associated with ipsilateral auditory extinction, were being assessed. In this case it was important to know if the lesion involved the auditory cortex or pathways. Therefore, lesions were defined more precisely as temporal, anterior frontal, posterior frontal, frontal, fronto-parietal, anterior parietal, posterior parietal, parietal, parieto-occipital, occipital, anterior basal ganglia and posterior basal ganglia.

2.53 General Description of the Types of Lesions Sustained by the Patients Included in These Studies

All 113 patients included in these studies had lesions confined to the cortical or subcortical matter of one hemisphere. Lesions included neoplasms (gliomas, meningiomas and solitary metastases), lesions resulting
from cerebrovascular disease or accident (infarctions and intracerebral haemorrhages) and cerebral abscesses. In the group of patients with right-hemispheric lesions 72% had neoplasms and 28% had lesions of cerebrovascular origin. In the group of patients with left-hemispheric lesions, 73% had neoplasms, 24% had lesions of cerebrovascular origin and 3% had cerebral abscesses.

(a) Neoplasms and Cerebral Abscesses

Neoplasms or tumours of the brain occur in many forms, and produce a variety of neurological symptoms because of their size, location and invasive properties. They usually destroy the tissues in which they are sited and cause displacement and oedema of tissues around them. They often result in increased intracranial pressure and are frequently fatal.

Benign tumours (meningiomas) arise from the meninges and often grow to a large size before they cause symptoms as a result of intracranial pressure and atrophy of the adjacent brain tissue. They do not invade the brain substance and can often be successfully removed surgically. Of the 113 patients I tested, 27% had meningiomas. Primary malignant tumours included gliomas and astrocytomias in this group of patients. These tumours invade brain tissue and can rarely be removed fully by surgery because of the extent to which they invade the surrounding tissue. A biopsy is often performed to confirm diagnosis, and the patient is commonly treated with corticosteroids. This
often results in a rapid decrease in some of the neurological symptoms because of the effectiveness of corticosteroids in reducing the oedema surrounding the tumour. Many patients are given a course of radiation therapy in an attempt to arrest tumour growth. Secondary neoplasms (metastatic carcinomas) result from the detachment of cells from malignant tumours in other parts of the body (particularly the lung and the breast) which are carried via the blood stream to the brain to begin independent growth there. Of the 113 patients I tested, 38% had gliomas or astrocytomas and 8% had metastases. According to Adams and Victor (1981), most patients with gliomas die within 12 months and survival beyond two years is exceptional. Patients with low grade astrocytomas can live for much longer periods. Patients with metastatic carcinomas rarely survive longer than weeks or months after diagnosis. Most patients with solitary, benign or malignant tumours involving the cortex, or basal ganglia were considered suitable subjects for hemineglect testing. The main reason for exclusion of such patients was a general intellectual deterioration.

Cerebral abscesses can result in a symptomatology similar to that of a neoplasm. Although they are usually accompanied by signs of an infection, a diagnosis often depends upon an exploratory operation. Brain abscesses are usually secondary to infection elsewhere in the body and only about 10% result from infection being introduced from outside (e.g. from a skull fracture or intracranial operation). Only patients with abscesses involving the
cortex were tested and testing only took place after antibiotic treatment was well established and the patient no longer had a fever. When antimicrobial treatment (for bacteria or fungi) does not reduce intracranial pressure promptly, the abscess is usually aspirated, drained or excised. Patients who survive can be left with neurological deficits and focal epilepsy. Only two of the 113 patients tested (2%) had abscesses.

(b) Lesions Resulting from Cerebrovascular Diseases and Head Injuries

Cerebrovascular lesions are very common in the neurological population but they are represented in relatively low proportions (25%) in the group of patients I studied. This was the result of two factors. Firstly, I tested only 10 patients over the age of 65, and most patients were under 56 years of age. This was because elderly patients tended not to be admitted to the Neurology and Neurosurgery Wards unless they were physically fit and mentally alert, and otherwise healthy. One of my criteria for selection of patients was that they should have no symptoms of general intellectual deterioration and no signs of cortical atrophy on C.T. scan. Added to this, older patients with brain lesions tend to have more symptoms of headache and general unwellness and therefore cannot be tested. Because a significant proportion of patients with vascular lesions in the general neurological population are over the age of 65, the proportion of patients with
vascular lesions in my study was reduced.

The second reason for the low proportion of patients with vascular lesions was that many lesions resulting from cerebrovascular disease or accident do not result in clearly defined focal damage to the cortex, white matter, or basal ganglia of one hemisphere. For example, the rupture of a saccular aneurysm into the subarachnoid space does not often result in unilateral focal damage. Primary intracerebral haemorrhage can often cause unilateral focal damage but patients with haemorrhage into the cerebellar hemispheres or the brain stem were excluded from this study. Patients who had a long history of transient ischemic attacks preceding an atherothrombic infarction were excluded because of the gradual onset of neurological deficits and the possibility that they may have other minor atherothrombic infarctions in other cerebral areas. In my sample of 113 patients, 12% had infarctions and 13% had haemorrhages involving intracerebral tissue.

A cerebral infarction is dead brain tissue caused by a loss of blood supply to the tissue. This can be an atherothrombotic infarction where a clot blocks an artery already narrowed by arterial disease, or an embolic infarction where an artery is suddenly occluded by embolic material which in most cases has broken away from a thrombus in the heart. The neurologic deficits which result from these infarctions often resolve over time. However, there is a danger of further infarctions at any time. Patients with embolic infarctions in particular, are often suitable subjects for inclusion in these studies.
Intracerebral haemorrhage is bleeding into the brain tissue causing damage to the tissue and displacement and compression of adjacent brain tissue. Spontaneous intracerebral haemorrhage is usually associated with elevated blood pressure and is probably caused by the rupture of a penetrating artery (Adams and Victor, 1981) or the rupture of small pathological dilations (Charcot-Bouchard aneurysms) on the internal arteries (Walsh, 1978). Once bleeding has been arrested it does not usually occur from the same site again. A haemorrhage of this type is almost always subcortical and is unlikely to rupture through the cerebral cortex. Neurological deficits which result are unlikely to resolve to any marked extent. Patients with spontaneous intracerebral haemorrhages are often suitable subjects for hemineglect studies.

Another common cause of haemorrhage is the rupture of a saccular (berry) aneurysm. These are small (22mm to 3cm in diameter) thin-walled blisters protruding from the Circle of Willis, which lies in the subarachnoid space, or its major branches. According to Adams and Victor (1981), they are probably formed over the years on the basis of a developmental or acquired arterial defect. Occasionally a large unruptured berry aneurysm will cause neurological symptoms as the result of pressure, often on the optic nerve or chiasm or on a cranial nerve, but usually they are asymptomatic until rupture. On rupture, blood is forced under high pressure into the subarachnoid space. The immediate result of this varies. Most patients experience a severe generalized headache, often, but not always,
followed immediately by a loss of consciousness which can last a matter of minutes or hours or days, and in some cases leads to death. Patients who regain consciousness often remain drowsy and confused for some days and suffer from amnesia, a severe headache and a stiff neck. In most cases, gross lateralizing signs do not occur and if they do, they are often transitory. In many cases, bleeding recurs in the following days and weeks. Patients who sustain subarachnoid haemorrhages from the rupture of a berry aneurysm or any other cause, only rarely have focal cerebral damage, usually due to an intracerebral clot or ischemia in the territory of the artery bearing the aneurysm. Therefore these patients form a very small proportion of the sample of patients studied in this thesis.

The other type of cerebrovascular lesion which occasionally renders a patient suitable for inclusion in this study, is the presence or rupture of an arteriovenous (A.V.) malformation. An A.V. malformation consists of a tangle of dilated vessels between the arterial and venous systems, and while it is a developmental abnormality, the mass enlarges with growth and time. Focal neurological symptoms can occur as the result of compression of neighbouring structures by a large A.V. malformation, or as the result of a rupture into the surrounding cerebral tissue. Once ruptured, there is a risk of rebleeding. A small proportion of patients suitable for these studies had ruptured A.V. malformations or were tested post-operatively after a block dissection of an A.V. malformation.
Treatments for cerebral infarctions and haemorrhages commonly include bed rest, anti-hypertensive and anticoagulant drug treatments, drug therapy to decrease cerebral oedema, and surgical measures which include removal of a haematoma, clipping, wrapping or ligating of aneurysms, and block dissection of A.V. malformations or embolization or ligation of the arteries feeding the A.V. malformation.

Another group of patients who are very occasionally suitable for study of neurological deficits caused by focal damage are those who sustain damage as a result of craniocerebral trauma. Patients who sustain blunt head injuries are hardly ever suitable, as diffuse or multiple brain damage and concussion are frequent concomitants. However, in some cases of penetrating wounds, a solitary focal infarct may result, and even after blunt head injury a single, unilateral intracerebral haemorrhage may occur.
2.6 NON-NEUROLOGICAL CONTROL SUBJECTS

Because most of the tests used to assess hemineglect and extinction were such that normal subjects would (and do) obtain perfect scores, formalized control studies were often unnecessary. However, normative scores were necessary for the computer tests for visual imagery described in Chapter 4, and for some of the auditory tests described in Chapter 5. Control subjects for these tests were self-selected from a large group of people who visited on Open Day at the University and agreed to participate in the study. Motivation to perform well on the tests appeared to be high, probably because volunteering subjects were given information about hemispheric specialization and were provided with a measure of their laterality on the various tasks. The general information obtained about these subjects included sex, age, level of formal education attained, handedness (assessed by the Edinburgh Handedness Inventory; Oldfield, 1971) and dexterity and sinistrality of parents, siblings and children. Before testing, volunteers were asked about previous or current neurological disorders such as head injuries, strokes or epilepsy, and about hearing defects. If they had any of these or appeared to be of below normal intelligence, they were still tested but their data were excluded from any analyses of normative data.
2.7 THE TESTS

All patients were given the battery of paper and pencil tests described in Chapter 3. Other behavioural information possibly relating to hemineglect was also noted. These behaviours included reading one side of a word or paragraph, eating from one side of a plate, colliding with objects on one side and ignoring people who were on one side of the patient. None of this information was used in the final assessment of hemineglect because (a) it was difficult information to quantify in a standard form, and (b) patients who exhibited any of these behaviours also exhibited visual hemineglect on one or more of the five paper and pencil tests which were used in the assessment of visual hemineglect. Patients who dressed one side of their body were noted and this was included in the information used to assess whether or not a patient had hemiasomatagnosia (body hemineglect). The five paper and pencil tests and the form used for summarizing visual hemineglect can be found in the Appendix. The data from the computer visual imagery tests were printed out by the computer and filed with the other information relating to that patient.

Visual field defects were tested independently by myself and a neurologist by confrontation at the bedside, in the following manner. One of the patient's eyes was covered and the other looked directly into the corresponding eye of the examiner (the patient's right eye and the examiner's left). A target consisting of a
white disk 1/2cm in diameter mounted on a black rod was then moved from the outside toward the centre of the visual field from different points around the circumference of each half field. With the target at an equal distance between the examiner's and the patient's eyes, the patient's fields were compared with those of the examiner. The patient's blind spot was aligned with the examiner's and its size determined by moving the target outward from the blind spot until it was seen.

All patients were assessed for deficits in tactile sensation by using a light touch of the fingertips unilaterally to each hand and cheek. Patients who exhibited no deficits on these were tested with the method of tactile double simultaneous stimulation (see Chapter 6). Patients were also tested on their ability to name unseen objects placed in each hand, but these data were not used in the studies reported in this thesis. The form used for summarizing the tactile hemineglect data can be found in the Appendix.

The majority of patients were given the 'Shadowing digits' test described in Chapter 5, and many of these were also given the 'Dichotic digits' test. All patients who were given the 'Shadowing digits' test were also given an auditory location test where they were asked to locate (point to the ear) in which they heard a series of three digits, played over earphones. The digits could be in one ear only or in both ears simultaneously. These results are not reported in this thesis. In the latter half of the 18 months over which patients were tested, some patients were
also tested on the 'Shadowing Tones' test which is described in Chapter 5. Data forms and a summary form for all these auditory tests can be found in the Appendix.

Qualitative data relating to hemiasomatagnosia were recorded under 'Behavioural observations/psychiatric signs' on the 'Subject information - neuropsychological 2' form (see Appendix) and if necessary additional data were recorded on a separate page.
3. ANTERO-POSTERIOR INTERHEMISPHERIC DIFFERENCES
IN THE LOCI OF LESIONS PRODUCING VISUAL HEMINEGLECT

3.1 INTRODUCTION

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3.4 DISCUSSION

3.41 Incidence of Visual Hemineglect
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3.1 INTRODUCTION

Hemineglect, also known as unilateral inattention, is a spatial disorder which is quite frequently observed following focal hemispheric lesions in humans, monkeys and cats. The main symptom is an apparent unawareness or neglect of stimuli in the side of space contralateral to the lesion. While the disorder may appear in any modality (De Renzi, 1982), it has been most often reported in the visual modality. Most researchers view visual hemineglect as predominantly a result of right-hemispheric damage (Arrigoni and De Renzi, 1964; Benton, 1969; Brain, 1941, 1945; Chedru, 1976; Colombo et al., 1976; De Renzi et al., 1970; Hecaen, 1962, 1969; McFie and Zangwill, 1960; Oxbury et al., 1974; Piercy, Hecaen and Ajuriaguerra, 1960, Schenkenberg, Bradford and Ajax, 1980) although a minority have found no significant differences in the frequencies of the disorder after right- and left-hemispheric lesions (Albert, 1973; Battersby et al., 1956). Some researchers point out that the populations tested for hemineglect often exclude patients who have left-hemispheric damage resulting in severe dysphasia, and that this could give rise to an underestimate of the incidence of hemineglect following left brain damage (Albert, 1973; Battersby et al., 1956; Brain, 1945; De Renzi, 1982).

One point on which most researchers agree is that right-hemispheric lesions usually result in visual hemineglect of greater severity than do left-hemispheric lesions (Albert, 1973; Denes, Semenza, Stoppa, and Lis,
1982; Gainotti et al, 1972). Costa et al (1969) provide one exception to this. They found the severity of hemineglect was similar in both their right- and left-hemispheric lesioned groups although the frequency of hemineglect was higher after right brain damage. A well established finding is that while patients with visual hemineglect often have a visual field defect, either symptom can exist without the other. Therefore, a visual field defect, while possibly exacerbating visual hemineglect, is not a direct cause of it (Albert, 1973).

The lesion site which have been associated with hemineglect in humans include the parietal lobes (Bisiach et al, 1981; Bisiach et al, 1979; Critchley, 1953; Hecaen et al, 1956; Heilman and Watson, 1977a), the frontal and cingulate cortex (Damasio et al, 1980; Gloning, 1965; Heilman and Valenstein, 1972b; Van der Linden et al, 1980), and the basal ganglia (Damasio et al, 1980; Watson and Heilman, 1979). Right parietal lesions, particularly of the inferior lobule, are by far the most frequent in published cases of hemineglect.

This study was an attempt to arrive at a more representative assessment of the incidence, severity and neuropathology of hemineglect by testing as many patients as practicable who presented over a 12 month period to the Neurological and Neurosurgical wards of a major public hospital. All patients had solitary, well-defined, unilateral lesions involving the cortical and/or subcortical matter of the cerebral hemispheres or the basal ganglia. The hemineglect test battery included tests
simple enough for most dysphasic patients and patients with hemiplegia to comprehend and attempt. While it may be dubious to infer brain-behaviour relationships from the testing of patients with space-occupying lesions because of possible effects of oedema, compression and infiltration on other parts of the brain, these could well be the lesions that are most likely to result in hemineglect. It is a common clinical impression that severe symptoms of hemineglect are most often found in patients with large and/or unstable lesions (De Renzi, 1982) and that these symptoms usually diminish over time (Campbell and Oxbury, 1976; Gainotti, 1968). Therefore, a more accurate assessment of incidence should be gained by testing all patients, whatever the nature of their lesion. In particular this should increase the likelihood of observing hemineglect in patients with left-hemispheric lesions, who, as mentioned previously, tend to suffer less severely from hemineglect anyway and possibly recover more rapidly than those with right-hemispheric lesions (Denes et al, 1982).

While the patients in this study were tested for auditory and tactile extinction and hemiasomatagnosia as well as visual hemineglect, only the results for visual hemineglect are reported in this Chapter. This is to enable comparisons to be made with other studies which assessed incidence, as these in the main tested only for visual hemineglect.
3.2 METHOD

3.21 Subjects and Categorization of Lesions

All 101 subjects were patients in the Neurological and Neurosurgical wards of Auckland Hospital, Auckland, New Zealand, and had a well-defined, unilateral lesion of the cerebral cortex or basal ganglia confirmed in all cases by Computerized Tomography (C.T.) and in many cases by carotid angiography as well. The patients with left-hemispheric lesions included 16 patients with lesions involving the cortex anterior to the central sulcus (A), 14 patients with lesions involving the cortex both anterior and posterior to the central sulcus (A/P), and 20 patients with lesions wholly posterior to the central sulcus (P). A further three patients had lesions of the left basal ganglia and three had lesions confined to the left temporal lobe. The patients with right-hemispheric lesions included 10 patients in the 'A' category, 12 in the 'A/P' category, and 18 in the 'P' category. A further two patients had right basal ganglia lesions and three had right temporal lobe lesions. In all cases great care was taken to ensure that the lesion was confined strictly to one hemisphere and that there were no other brain lesions. In the case of gliomas, the tumour area was defined as clearly as possible with radiological techniques, and any oedema shown on C.T. at the time of testing was included in the lesioned area. Mass effects were noted. All subjects were cleared of any other brain disorder in the past or present, and did not
show any symptoms of a general intellectual deterioration. The two groups designated Right Brain Damaged (RBD) and Left Brain Damaged (LBD) were compared statistically and found not to differ significantly with respect to the number of patients with lesions in the different localities ($X^2 = 0.65; df = 4; N.S.$)$^1$, nor on the types or sizes of brain lesions. The numbers of patients in each of these categories are given in Table 3-1. Tumours were the most frequent type of lesion followed by Intracerebral haemorrhages (ICH) and infarcts. The category 'others' included solitary metastases, abscesses and arteriovenous malformations (AVM). All lesions had resulted in some clinical symptoms. Lesions were designated as large, medium or small depending on a subjective estimate of the size of the lesion as seen on the C.T. scan. As a general guide lesions which in cross section on at least one C.T. section involved one-quarter or more of one hemisphere were graded as large, those involving one-fifth to one-quarter were graded as medium, and those involving less than one-fifth of the hemisphere on a C.T. section were graded as small.

The two groups did not differ significantly with respect to age ($t = 0.72; df = 99; N.S.$) and sex ($X^2 = 0.04; df = 1; N.S.$)$^2$. In the RBD group the average age for females ($N = 23$) was 41.96 years with an age range of 8-74

---

1. $X^2$ is used for the test statistic in chi-square tests since most modern, advanced treatments of the analysis of categorical data use this rather than $X^2$.

2. All chi-squares based on 2 x 2 tables throughout the thesis have been corrected for continuity.
years, and the average age for males (N = 22) was 47.64 years with an age range of 17-76 years. In the LBD group the average age for females (N = 27) was 44.74 years with an age range of 9-65 years and the average age for males (N = 29) was 49.62 years with an age range of 11-73 years.

**TABLE 3-1** Comparisons between the LBD and RBD groups on the numbers of patients with different types and sizes of lesions and with or without visual field defects.

<table>
<thead>
<tr>
<th>Type of Lesion</th>
<th>N for each Hemispheric Group</th>
<th>Statistical Comparisons between Groups (p = .05)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBD</td>
<td>RBD</td>
</tr>
<tr>
<td>Meningioma</td>
<td>18</td>
<td>11</td>
</tr>
<tr>
<td>Glioma</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>I.C.H.*</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Infarct</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Others</td>
<td>12</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Size of Lesion</th>
<th>N for each Hemispheric Group</th>
<th>Statistical Comparisons between Groups (p = .05)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBD</td>
<td>RBD</td>
</tr>
<tr>
<td>Large</td>
<td>28</td>
<td>21</td>
</tr>
<tr>
<td>Medium</td>
<td>18</td>
<td>15</td>
</tr>
<tr>
<td>Small</td>
<td>10</td>
<td>9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Visual Field Defect</th>
<th>N for each Hemispheric Group</th>
<th>Statistical Comparisons between Groups (p = .05)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>Absent</td>
<td>37</td>
<td>30</td>
</tr>
</tbody>
</table>

* I.C.H. = Intracerebral Haemorrhage
3.22 Neurological Examination

On admission all patients were given a complete neurological examination by a neurologist and relevant findings were taken into account when assessing the patients neuropsychologically. Visual field defects (V.F.D.) were independently assessed by confrontation and in some cases by perimetry by a neurologist and myself. Visual field assessment was not possible for two RBD patients and six LBD patients either because of severe visual neglect or because they were unable to fixate. The two groups did not differ significantly with respect to the presence/absence of V.F.D.'s (see Table 3-1). Symptoms of dysphasia were noted by both the neurologist and myself. One patient with a right-hemispheric lesion was excluded because she demonstrated expressive dysphasia suggesting that her left hemisphere was not dominant for speech.

3.23 Neuropsychological Examination

PROCEDURE: Each patient was tested early in the day, and only when he/she was feeling alert and co-operative, had a normal temperature, no headache and was not feeling nauseous. Visual neglect tests usually took no longer than 10 minutes. In all tests visual hemineglect was considered to be the failure to respond, in a free viewing situation, to stimuli on the side contralateral to the cerebral lesion.
Tests and Scoring Procedures

Five pencil and paper tests were used to assess visual hemineglect. As patients are often variable in the ways in which they display neglect, by using five tests I was more likely to discover evidence of neglect that might have been missed had I used a single test. Each test has been used in some form in previously published research on visual neglect. Patients held the pencil in their dominant hands when possible. Those who had to use their non-dominant hands were sometimes unable to attempt the more difficult drawings. In all tests the patient was not permitted to tilt the page but was permitted to move the page horizontally from one side to the other.

A patient was considered to have visual hemineglect if he/she demonstrated neglect on any one of the tests. Each patient was given a severity score for each test attempted, and a separate severity score was also computed by summing the tests on which neglect was demonstrated. The five tests are described below.

1. Crossing Lines
This test was designed by Albert (1973) and requires the patient to draw lines across forty 2.5cm long lines dispersed apparently randomly over a page. There were 18 lines on each side of the page and four lines in the centre. The examiner demonstrated the desired response by drawing a line through one line in the centre of the page. When the patient had finished the examiner said, "Check
that you have crossed all the lines". Albert designated the patient as having visual neglect if he/she missed one or more lines. He found a similar incidence of neglect on the test following both right- and left-hemispheric lesions (i.e. 37% after RBD and 30% after LBD). A similar scoring system was used here. The patient received a score equal to the number of lines missed (out of 18) on the side of the page contralateral to his/her lesion. Missed centre lines were not scored. If patients also missed lines on the ipsilateral side of the page, these were subtracted from the contralateral hemineglect score. Patients who demonstrated more ipsilateral than contralateral neglect on this test or on any of the other four tests were noted but were not classified as having hemineglect on that test for the purpose of this study.

2. Clock numbers:
The patient was asked to draw in the numbers on a clock face, the '12' already being in position. If the numbers were squashed into three-quarters of the clock face, the vacant quarter being on the contralateral side, the patient was given a score of 1, and if the numbers were squashed into half or less of the ipsilesional side of the clock face, the patient was given a score of 2 (Fig. 3-1, A and D).

3. Necker cube:
The patient was asked to copy a drawing of a Necker cube and was given a score of 1 if the drawing was only
partially completed on the contralesional side, and 2 if the contralesional half was completely missing (Fig. 3-1 B and E).

4. Five-pointed star:
The patient was asked to copy a drawing of a five-pointed star and was given a score of 1 if the drawing was only partially completed on the contralesional side, and 2 if the contralesional half was completely missing (Fig. 3-1, C and F).

5. Scene:
The patient was asked to copy a drawing of a tree, fence, house and tree (similar to that used by Gainotti et al, 1972). If only the chimney or the contralesional window was missing a score of 1 was given; if the contralesional half of a tree or the house was missing a score of 2 was given; if the contralesional tree was completely missing a score of 3 was given; and if the contralesional tree plus more of the drawing on the same side was missing, a score of 4 was given (Fig. 3-2, A-C). (See Appendix for test forms and visual hemineglect summary form).

Visual Extinction:
All patients without a V.F.D. were tested with double simultaneous stimulation (D.S.S.) (Bender, 1952). This was not used as a test of visual neglect and results are given separately.
FIGURE 3-1   Drawings A, B and C are those of patients with posterior right-hemispheric tumours, and D, E and F, those of patients with tumours of the left-hemisphere. The patient who drew D had a posterior tumour, the patient who drew E an anterior tumour, and the patient who drew F a basal ganglia tumour. Hemineglect severity scores of 2 were given to A, B and F, and scores of 1 to C, D and E.
FIGURE 3-2  Drawing A is the one copied by the patients. Drawing B was by a patient with a right basal ganglia tumour and drawing C by a patient with a tumour of the left frontal lobe. Both drawings received hemineglect severity scores of 4.
3.3 RESULTS

Incidence of Hemineglect:

In the LBD group 28 out of 56 patients (50%) had visual hemineglect on at least one of the five tests, compared with 20 out of 45 patients (44.44%) in the RBD group\(^3\). These proportions were not significantly different \(X^2 = 0.16; \text{df} = 1; \text{N.S.})\).

Chi-square tests comparing the RBD and LBD groups on the incidence of hemineglect on each individual test demonstrated no significant differences between the groups on any of the tests. However, test 5 (scene) showed the highest incidence overall of hemineglect (i.e. whatever the side neglected) followed by test 1 (crossing lines), test 3 (cube), test 4 (star), and test 2 (clock), in that order. These results are presented in Table 3-2.

3. Two out of the 45 RBD patients (4%) and five out of the 56 LBD patients (9%) were classified as not having hemineglect but were only able to complete three or four of the five tests. Therefore, these seven patients may have been incorrectly classified. One RBD patient had an anterior lesion and the other six patients had anterior/posterior or posterior lesions.
### TABLE 3-2
Comparisons between LBD and RBD groups on the incidence of visual hemineglect for each neuropsychological test.

<table>
<thead>
<tr>
<th>Test</th>
<th>Hemispheric Group</th>
<th>Total N</th>
<th>No. of patients with neglect</th>
<th>Statistical comparisons between groups (p = .05)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Crossing lines</td>
<td>LBD</td>
<td>53</td>
<td>14</td>
<td>$X^2 = 0.05; df = 1; N.S.$</td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>41</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>2. Clock</td>
<td>LBD</td>
<td>50</td>
<td>4</td>
<td>$X^2 = 1.69; df = 1; N.S.$</td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>38</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>3. Cube</td>
<td>LBD</td>
<td>46</td>
<td>8</td>
<td>$X^2 = 1.17; df = 1; N.S.$</td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>34</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>4. Star</td>
<td>LBD</td>
<td>52</td>
<td>9</td>
<td>$X^2 = 0.61; df = 1; N.S.$</td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>40</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>5. Scene</td>
<td>LBD</td>
<td>49</td>
<td>17</td>
<td>$X^2 = 0.05; df = 1; N.S.$</td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>40</td>
<td>15</td>
<td></td>
</tr>
</tbody>
</table>
3.32 Age and Sex of Patients with Visual Hemineglect

There were 14 females and 14 males with hemineglect in the LBD group and 11 females and 9 males with hemineglect in the RBD group. The age range in the LBD hemineglect group was 9 to 73 years and the mean age 49.70 years. For the RBD hemineglect group the age range was from 17 to 76 years and the mean age 54.48 years. A t-test showed that the two groups did not differ significantly in age ($t = 0.99; \text{df} = 46; \text{N.S.}$), and a chi-square test showed they did not differ significantly with respect to sex ($X^2 = 0.09; \text{df} = 1; \text{N.S.}$).

3.33 Visual-Field Defects (VFD)

A tabulation of patients according to side of lesion, presence or absence of hemineglect, and presence or absence of VFDs, is shown in Table 3-3. Excluded are the eight patients who could not be assessed for VFDs (three LBD patients with hemineglect and three without, and two RBD patients with hemineglect). The three-way contingency was broken down into three two-way interactions and one three-way interaction according to the procedure described by Winer (1971, p.857-858). Only the three-way interaction proved significant ($X^2 = 6.07, \text{df} = 1, p < .025$). Accordingly, two-way contingencies between hemineglect and VFDs were calculated separately for each hemispheric group. For the LBD group this contingency was not significant ($X^2 = 0, \text{df} = 1, \text{N.S.}$), indicting that LBD patients with
hemineglect were no more likely to have VFDs than those without. For the RBD group, by contrast, the contingency was highly significant ($X^2 = 9.17$, df = 1, p < .01), showing that those patients with hemineglect were much more likely to have VFDs than those without.

3.34 Type and Size of Lesion and Hemineglect

Lesions were categorized with respect to their type and size according to the criteria described in the 'Subjects and Categorization of Lesions' section. Chi-squared tests were carried out on the RBD- and LBD-hemineglect groups combined and separately for each of these lesion parameters and no significant differences were found (see Table 3-4).

**TABLE 3-3** The presence and absence of VFDs in RBD and LBD patients with and without visual hemineglect.

<table>
<thead>
<tr>
<th></th>
<th>RBD</th>
<th></th>
<th>LBD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+ VFD</td>
<td>- VFD</td>
<td>+ VFD</td>
</tr>
<tr>
<td>+ HN</td>
<td>10</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>- HN</td>
<td>3</td>
<td>22</td>
<td>7</td>
</tr>
</tbody>
</table>
TABLE 3-4  Comparisons between the LBD and RBD groups on the numbers of patients with visual hemineglect and with different types and sizes of lesions.

<table>
<thead>
<tr>
<th>Type of Lesion</th>
<th>N for each Hemispheric Group</th>
<th>Statistical Comparisons between Groups (p = .05)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBD</td>
<td>RBD</td>
</tr>
<tr>
<td>Meningioma</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>Glioma</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>I.C.H.*</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Infarct</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Others</td>
<td>5</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Size of Lesion</th>
<th>N for each Hemispheric Group</th>
<th>Statistical Comparisons between Groups (p = .05)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBD</td>
<td>RBD</td>
</tr>
<tr>
<td>Large</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>Medium</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Small</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

* I.C.H. = Intracerebral Haemorrhage

3.35 Locus of Lesion and Hemineglect

Patients were divided into subgroups according to the A, A/P or P locus of the lesion. Patients with lesions to the basal ganglia or the temporal lobes were excluded from this sample as such patients cannot be clearly classified as having lesions restricted to A, A/P or P areas. However, all five patients with basal ganglia lesions had
hemineglect while none of the six patients with temporal lobe lesions did.

The relation of hemineglect to locus and side of lesion is shown in Table 3-5. The three-way contingency was again analysed according to the procedure described by Winer (1971, p.857-858), yielding insignificant two-way interactions and a significant three-way interaction ($X^2 = 9.39$, $df = 2$, $p < .01$). Because of this three-way interaction, separate two-way interactions were computed for each hemispheric group, and for patients with and without hemineglect.

In the LBD group there was a significant contingency between hemineglect and locus ($X^2 = 6.77$, $df = 2$, $p < .05$), with hemineglect associated predominantly with anterior lesions. In the RBD group the contingency was again significant ($X^2 = 6.60$, $df = 2$, $p < .05$), but in this case hemineglect was more frequent among those with posterior lesions. Among all patients with hemineglect there was a significant contingency between side and locus of lesion ($X^2 = 6.60$, $df = 2$, $p < .05$), again reflecting the association with anterior lesions in the LBD group and posterior lesions in the RBD group. Among patients without hemineglect, however, the association between locus and side was not significant ($X^2 = 2.85$, $df = 2$, N.S.).

3.36 Severity of Hemineglect

The main problem with making comparisons between patients on severity measures is the number of patients who
### TABLE 3-5
Numbers of patients* in the different lesion locality groups with and without visual hemineglect.

<table>
<thead>
<tr>
<th>Hemispheric Group</th>
<th>Visual Hemineglect</th>
<th>Number of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present</td>
<td>A</td>
</tr>
<tr>
<td>RBD</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>8</td>
</tr>
<tr>
<td>LBD</td>
<td>Present</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>4</td>
</tr>
</tbody>
</table>

* NOTE: Patients with basal ganglia lesions and lesions restricted to the temporal lobes are excluded from this Table.

were not assessed on all five tests. Among the patients who were classified as having hemineglect (i.e. demonstrated hemineglect on at least one of the five tests), nine RBD patients and three LBD patients could not complete all of the tests. All of these patients had A/P or P lesions. With RBD patients the most common reason for this was that their drawings were indecipherable, often a problem with patients who have parietal lesions. LBD patients also have a problem if they have to use their non-dominant hands for drawing. In all the statistical tests comparing severity of visual hemineglect, patients who did not demonstrate neglect on any of the tests were excluded. This should not influence the results as the LBD
and RBD groups did not differ significantly on incidence of neglect. The two groups were compared on two different measures of severity, a within-test measure and a between-test measure.

Within-test severity

A within-test severity score was assigned on each test as explained under 'Tests and Scoring Procedures'. The two groups were compared on these scores for each of the five tests separately. Separate comparisons were needed because severity scores could not be equated across tests. That is, a score of two on one test was not necessarily equivalent to a score of two on another test. Only patients who demonstrated neglect on the test being compared (i.e. received a score of one or greater), were included in these comparisons. The patients demonstrating hemineglect in the LBD and RBD groups were compared using Mann-Whitney U tests when the range of scores was greater than two, and Fisher's exact test of significance for 2 x 2 tables when only two scores were possible (Table 3-6). Although the RBD group yielded the higher scores on all but one of the tests (clock), none of the five tests reached significance on a two-tailed test. The 'crossing lines' test yielded a difference that was significant (p < .05) according to a one-tailed test, however, which may be taken as confirmation of Albert's (1973) finding that RBD patients show a greater degree of neglect than LBD patients on this test.
Between-test severity

The range or breadth of neglect was calculated by counting the number of different tests on which each patient demonstrated neglect. This gave a range of scores from 1 to 5. Two comparisons were made, the first between patients with hemineglect who had been assessed on all five tests, and the second between all patients demonstrating hemineglect, regardless of the number of tests they were assessed on. Since more RBD patients than LBD patients were unable to complete all of the tests, this second comparison should be, if anything, biased against the RBD group. On both comparisons, however, the scores of the RBD group were significantly higher than those of the LBD group. That is, if the number of tests showing neglect is a valid measure of severity, then RBD patients are significantly more impaired than LBD patients. These severity results are tabulated in Table 3-6.

To see whether patients with hemineglect and lesions in different locations (A, A/P and P) differed on their between-test hemineglect scores, Mann-Whitney U tests were computed across the RBD and LBD groups for A, A/P and P lesioned patients separately, and within each hemispheric group each lesion location group was compared with each of the other two lesion groups. The only significant difference was between the RBD and LBD posterior lesioned groups when all hemineglect patients were included (U = 26; \( n_1 = 9, n_2 = 12; \ p = .05 \)), the RBD 'P' group having the more severe neglect. However, this comparison did not reach significance when hemineglect patients with
TABLE 3-6 Comparisons between LBD and RBD hemineglect groups on the severity of hemineglect.

<table>
<thead>
<tr>
<th>Test</th>
<th>Hemispheric Group</th>
<th>N</th>
<th>Range of Scores</th>
<th>Mean Score</th>
<th>Statistical Comparisons between Groups (p = .05)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBD</td>
<td>14</td>
<td>1-18</td>
<td>2.64</td>
<td></td>
</tr>
<tr>
<td>Crossing Lines</td>
<td>RBD</td>
<td>12</td>
<td>1-18</td>
<td>6.25</td>
<td></td>
</tr>
<tr>
<td>Clock</td>
<td>LBD</td>
<td>4</td>
<td>1-2</td>
<td>1.25</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>7</td>
<td>1-2</td>
<td>1.14</td>
<td></td>
</tr>
<tr>
<td>Cube</td>
<td>LBD</td>
<td>8</td>
<td>1-2</td>
<td>1.38</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>10</td>
<td>1-2</td>
<td>1.90</td>
<td></td>
</tr>
<tr>
<td>Star</td>
<td>LBD</td>
<td>9</td>
<td>1-2</td>
<td>1.22</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>10</td>
<td>1-2</td>
<td>1.40</td>
<td></td>
</tr>
<tr>
<td>Scene</td>
<td>LBD</td>
<td>17</td>
<td>1-4</td>
<td>2.71</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>15</td>
<td>1-4</td>
<td>3.00</td>
<td></td>
</tr>
<tr>
<td>A: Tests out of 5 showing hemineglect</td>
<td>LBD</td>
<td>25</td>
<td>1-5</td>
<td>1.72</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>11</td>
<td>1-5</td>
<td>2.82</td>
<td></td>
</tr>
<tr>
<td>B: Tests showing hemineglect</td>
<td>LBD</td>
<td>28</td>
<td>1-5</td>
<td>1.75</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RBD</td>
<td>20</td>
<td>1-5</td>
<td>2.65</td>
<td></td>
</tr>
</tbody>
</table>

A: Excludes patients who were not assessed for hemineglect on all five tests.

B: Includes all patients who demonstrated hemineglect however many tests they were assessed on.
incomplete test batteries were excluded (U = 29; n₁ = 8, 
n₂ = 9; N.S.). Because of the anterior-posterior 
difference in incidence of hemineglect between the LBD and 
RBD groups, the LBD 'A' group and the RBD 'P' group were 
compared with the Mann-Whitney U test on their between-test 
hemineglect scores. All patients with hemineglect were 
included, whether or not they had completed the test 
battery. This comparison just failed to reach significance 
at the p = .05 level on a two-tailed test (U = 38; n₁ = 12, 
n₂ = 12; N.S.).

3.37 Extinction on Double Visual Simultaneous Stimulation

A chi-square test on all patients without VFDs 
demonstrated that the LBD and RBD groups did not differ 
significantly with regard to extinction. However, 
approximately one-third of the patients in each of the 
hemispheric groups could not be assessed for extinction 
because they either had VFDs, did not respond to unilateral 
visual stimulation, or could not fixate. Therefore, only 
limited conclusions can be reached about the relationship 
of extinction to visual hemineglect. The numbers of 
patients with and without extinction and hemineglect as 
well as the numbers who could not be assessed are given in 
Table 3-7.

One result is unambiguous; in both hemispheric groups 
there are a number of patients who demonstrate visual 
neglect on the drawing tests but who do not exhibit 
extinction.
TABLE 3-7  Numbers of patients with and without visual hemineglect and visual extinction and patients not able to be assessed for extinction.

<table>
<thead>
<tr>
<th>Hemispheric Group</th>
<th>Visual Hemineglect</th>
<th>With Extinction</th>
<th>Without Extinction</th>
<th>Unable to assess</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBD</td>
<td>Present</td>
<td>0</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>2</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>LBD</td>
<td>Present</td>
<td>4</td>
<td>15</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>2</td>
<td>17</td>
<td>9</td>
</tr>
</tbody>
</table>

3.38 Ipsilateral Neglect

Because it is not clear whether ipsilateral neglect can be considered the same disorder as contralateral neglect, patients who neglected on the ipsilateral side on Tests 2 to 5 or left uncrossed more ipsilateral lines than contralateral lines on Test 1, were treated as non-neglecting patients on those tests. This is unlikely to have influenced the results as only six LBD patients demonstrated ipsilateral neglect and five of these also demonstrated contralateral neglect on other tests (and were therefore included in the neglect incidence figures).

The 'crossing lines' test, the 'cube' test and the 'clock' test were all neglected ipsilaterally by two or three patients with left-hemispheric lesions. None of the right brain damaged patients exhibited ipsilateral neglect. On the 'crossing lines' test one LBD and two RBD patients
left lines uncrossed on the ipsilateral side, but as they missed many more lines on the contralateral side they were scored as having contralateral neglect.
3.4 DISCUSSION

3.41 Incidence of Visual Hemineglect

The finding that left-hemispheric lesions are as likely to result in visual hemineglect as right-hemispheric lesions is not entirely unexpected, as some researchers have found this to be so in previous studies (Albert, 1973; Battersby et al., 1956), while others have commented that this was a possibility (Brain, 1945; De Renzi, 1982). In this study it was found that while there was no significant difference in the numbers of LBD and RBD patients exhibiting neglect on any one test, individual RBD patients neglected on more tests than LBD patients. Therefore, using a wider range of tests is more likely to pick up left-hemispheric neglect than using a single test. Perhaps this partially explains why more RBD than LBD patients are observed to have neglect in assessment programs where only one test is used. The method of testing nearly all patients with clearly defined unilateral cortical and basal ganglia lesions also increases the chances of finding neglect in left-hemispheric patients, particularly given that such patients are more likely to have anterior rather than posterior lesions. Finally, the fact that the lesions of this group of patients were predominantly tumoral rather than vascular as in most previous studies could have contributed to the higher incidence of hemineglect found in LBD patients.
3.42 Anterior-Posterior Interhemispheric Differences in Incidence

While it is well established that posterior lesions are those most likely to result in hemineglect in patients with right-hemispheric lesions (Bisiach et al, 1979; Hecaen et al, 1956) the finding that anterior lesions are significantly more likely to result in visual hemineglect in patients with left-hemispheric lesions is unexpected. If the five LBD and two RBD patients who were classified as not having neglect but who could not be assessed on all five tests were, in fact, classified incorrectly, there is a possibility that this could influence the significant anterior-posterior difference in incidence between the groups. However, this seems unlikely as these patients comprised only 9% (LBD) and 4% (RBD) of the total hemispheric samples. Added to this, each of these patients was assessed on at least three of the tests and yet demonstrated no neglect.

The asymmetry with respect to hemineglect following anterior and posterior lesions suggests that the representation of visual space is relatively anterior in the left hemisphere, and posterior in the right. This meager representation of space in the posterior portion of the left hemisphere may well be due to the invading presence of language representation in that region (cf. Corballis and Beale, 1976). Le Doux, Wilson and Gazzaniga (1977) argue similarly that right-hemispheric specialization is most pronounced for manipulospacial
skills, and that this specialization depends critically on the region of the parieto-temporal junction. This region is homologous to Wernicke's area in the left cerebral hemisphere. Le Doux et al. infer that the mediation of manipulospatial skills by the left parieto-temporal region is drastically curtailed by the invading presence of language representation in that area. This could also provide an explanation for the finding that in the left-hemisphere, anterior lesions are more likely to result in hemineglect than posterior lesions, at least when the tasks require manipulospatial skills. That is, as a consequence of language representation in the left parieto-temporal region, spatial representation has become more anterior.

It is conceivable that the battery of tests used in this study biased the results in favour of a form of visual hemineglect dependent on manipulospatial skills, particularly as three of the five tests involved drawing. However, the anterior-posterior interhemispheric difference still reaches significance (p < .05) for the patients who demonstrated neglect on Albert's (1973) crossing lines test. As this does not require manipulospatial skills, this argues against a bias due to the tests used.

3.43 Severity of Hemineglect

Most previous studies have found that right-hemispheric damage results in a more severe neglect disorder than does left-hemispheric damage. The between-test severity measure used in this study corroborates this
finding. While the difference between LBD and RBD groups on severity scores for individual tests only reached significance for one out of five tests, the RBD scores were the higher on all but one of the tests.

Why should hemineglect be more severe after right- than after left-hemispheric lesions? One possibility is that each hemisphere primarily represents the contralateral side of space, but also to a lesser degree represents the ipsilateral side of space. If, as a consequence of the lateralization of language, this ipsilateral representation is further diminished in the left hemisphere relative to the right, this would result in a more severe neglect of contralateral space after right- than after left-hemispheric damage.

3.44 Ipsilateral Neglect

While it is not surprising that damage to either hemisphere results in neglect of the stimuli in the contralesional space, the occasional occurrence of ipsilateral visual neglect is puzzling. It is, in fact, difficult to be sure whether ipsilateral visual neglect is a true phenomenon or simply an artifact of other visuo-spatial problems. For example, if a patient draws the numbers on a clock face very close together, he/she might fit all 12 numbers into the right side of the clock and stop there. Whether this can be called ipsilateral neglect in a patient with a left-hemispheric lesion is questionable. It is also not difficult to see how a
patient might miss the occasional line on either side of the page on the 'crossing lines' test. While being aware of these problems, it is nevertheless interesting to note that in this study only LBD patients appear to neglect ipsilateral visual stimuli. Ipsilateral tactile extinction has been documented in a number of patients following left-hemispheric lesions involving the frontal lobes but not following right-hemispheric lesions (Schwartz, Marchok and Flynn, 1977). A number of researchers have found ipsilateral auditory extinction in patients with left-hemispheric lesions of the cortical and subcortical area posterior to the posterior aspect of the lateral ventricle (Damasio and Damasio, 1979; Damasio, Damasio, Castro-Caldas and Ferro, 1976; Rubens, Johnson and Speaks, 1978; Sparks et al, 1970) and I have have also found ipsilateral auditory extinction in many patients with lesions restricted to the left frontal lobe (see Chapter 5). The only patients with ipsilateral auditory extinction following right-hemispheric lesions appear to be those with speech represented in the right hemisphere (Damasio and Damasio, 1979). In my own research I tested a right-handed patient with speech on the right (i.e. she became dysphasic and dyslexic after a right-hemispheric stroke) who demonstrated both right-ear extinction for dichotically presented digits and neglected lines only on the right of the page in the 'crossing lines' test.

It would seem that ipsilateral visual neglect and ipsilateral tactile and auditory extinction sometimes follow damage to the hemisphere dominant for speech, but do
not follow damage to the hemisphere non-dominant for speech. A disconnection hypothesis has been proposed to explain ipsilateral auditory extinction for verbal stimuli after left-hemispheric damage (Damasio and Damasio, 1979; Sparks et al., 1970), and as patients showing ipsilateral tactile extinction used a verbal response on the test, these results might also be explained in a similar fashion. However, the drawing tasks used in this study did not involve verbal stimuli or verbal responses. It is therefore difficult to see how a hypothesis dependent upon the involvement of the hemisphere dominant for speech could explain ipsilateral visual neglect on these tasks, unless perhaps patients tended to mediate their performance by the use of verbal self-instruction.

To summarize, this study finds an equal incidence of visual hemineglect following left- and right-hemispheric lesions which are predominantly of tumoural etiology. However, the neglect following right brain-damage tends to be more severe than that following left brain-damage. The most interesting result was that while patients with right brain damage and neglect most often had posterior lesions, patients with left brain damage and neglect most often had anterior lesions.
4. THE UNILATERAL NEGLECT OF AN IMAGE

4.1 INTRODUCTION

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   4.21 Subjects
   4.22 Procedure

4.3 RESULTS
   4.31 'Static' Condition
   4.32 'Dynamic' Condition

4.4 DISCUSSION
   4.41 Hemineglect of Images in Brain-Damaged Patients
   4.42 Implications for Normal Imagery Processes
4.1 INTRODUCTION

One of the most intriguing theories proposed to explain hemineglect is that it is the result of a 'mutilated representation of space'. This idea was first suggested by De Renzi et al., (1970) in an attempt to explain the data they obtained in an experiment in which unilaterally brain-damaged patients and control subjects were asked to locate a marble placed in one of four lateral arms of a tactile finger maze. The subjects could not see the maze which was hidden by a curtain, and they were timed while they searched for the marble. The forefinger of the ipsilesional hand was used by the brain-damaged patients, and an equal number of controls used the right or left hand. While both left and right brain-damaged patients took longer to locate the marble when it was in one of the contralesional arms of the maze than when it was in one of the ipsilesional arms, many more patients with right brain-damage failed to locate the marble at all when it was in one of the contralesional arms, within the 90 seconds allowed for each trial. De Renzi et al., suggested that since neither oculomotor deficits nor motor-sensory deficits of the hand used to explore the maze could affect the task, the exploration of the maze was likely to be guided by the subject's imagination of the spatial layout of the maze and his/her movements in it. The finding that the patients with right brain damage appeared to be unaware that there was any left space to explore, was taken to imply that these patients had a mutilated representation of space.
In 1978, Bisiach and Luzzatti reported two cases of patients with left-sided visual neglect that provided some support for this idea. These patients, when asked to recall the buildings situated on the two long sides of the Piazza del Duomo, the cathedral square in Milan, recalled only the buildings to their right when they imagined they were standing on the far side of the square facing the Duomo, yet recalled only the buildings on the other side, but again to their right, when they imagined they were standing with their backs to the Duomo. That is, those buildings which were recalled in one imagined situation were rejected immediately afterwards, given the opposite imagined perspective. In 1981, Bisiach, Capitani, Luzzatti and Perani reported similar results for a larger group of patients with left-sided visual hemineglect. Bisiach and his colleagues concluded that patients suffering from hemineglect of perceived scenes also neglect the contralesional sides of their imagined representations of the external world.

Another case study along similar lines was reported by Mesulam and Geschwind in 1977. Their patient demonstrated hemineglect following a stroke, and also suffered visual hallucinations due to alcoholic withdrawal. Apparently his hallucinations were confined to one half of space he was aware of in his external world! It seems that even visual images over which we have no voluntary control, are affected by unilateral neglect.

These case studies demonstrate that patients with visual hemineglect also neglect the contralesional sides
of images constructed from memory. In 1979, Bisiach, Luzzatti, and Perani carried out an experiment to see if patients with visual hemineglect would also neglect the contralesional sides of images constructed from immediate input rather than from memory. Patients with left-sided visual hemineglect and control subjects were required to decide whether pairs of cloud-like patterns were the same or different. Each pattern was presented for two seconds and was followed one second later by the second pattern. The pairs of patterns had a common central sector and in two-thirds of the trials the lateral sectors differed either on the left or on the right. There were two conditions, a 'static' condition and a 'dynamic' condition. In the 'dynamic' condition the patterns moved behind a slit so that the subject could only see a section of the pattern at any time. Half the pairs moved from left to right and half from right to left. Not only did the patients with hemineglect make more errors than control patients in discriminating differences on the left but not the right of the patterns in the 'static' condition, but they also made significantly more left-sided than right-sided errors in the 'dynamic' condition. The direction in which the patterns were moving made no significant difference to the results. As all the information regarding the shape of each pattern came from the same spatial locus in the vicinity of the vertical centre, Bisiach et al, (1979) argued that this could not be a deficit of perception or scanning ability but pointed to a specific inability to construct the left side of spatial images.
As Bisiach et al. (1979) used only right brain-damaged patients and controls in their experiment, I decided to repeat a version of the experiment to see if patients with left-hemispheric damage also neglected the contralesional sides of images. As I reported in Chapter 3, visual hemineglect occurs with an equal frequency after right- and left-hemispheric lesions, and most patients with left-sided visual neglect have posterior right-hemispheric lesions, whereas most patients with right-sided visual hemineglect have anterior left-hemispheric lesions. Given this interhemisphere difference in the loci of lesions which result in hemineglect, the possibility of qualitative differences in left and right visual hemineglect should be explored. One possibility is that neglect of the contralesional side of an image is not common to patients with left- and right-hemispheric lesions.
4.2 METHOD

4.21 Subjects

Three groups of subject, Control (C), left brain-damaged (LBD) and right brain-damaged (RBD) performed the experiment.

Control subjects were 20 visitors to a University Open Day who volunteered to take part in the experiment. There were 11 women and 9 men in the age range 17 to 60 years (mean age 38.9 years). Their formal education ranged from two years secondary schooling to an undergraduate university education.

Experimental subjects were patients with clearly defined unilateral cerebral lesions who were in the ward during the period the experiment was being run. Only patients who were right-handed, had no severe motor disability of the right hand, and felt generally well (e.g. no headache, nauseous feelings or fatigue), were asked to participate in the experiment. There were seven men and two women in the LBD group with an age range of 11 to 66 years and a mean age of 49.8 years. In the RBD group were also seven men and two women, with an age range of 23 to 55 years and a mean age of 41.6 years. Visual hemineglect was demonstrated on at least one of the five tests in the test battery described in Chapter 3 by seven LBD patients and three RBD patients. Unilateral cortical and subcortical lesions were sited in anterior, anterior/posterior and posterior locations as follows. Anterior (A) lesions were
wholly anterior to the central sulcus, anterior/posterior (A/P) lesions were on both sides of the central sulcus, and posterior (P) lesions were wholly posterior to the central sulcus. Table 4-1 specifies the neurological characteristics of the brain-damaged subjects.

4.22 Procedure

The stimuli were presented on a 19cm x 12cm colour video screen controlled by an Apple II Plus microcomputer. Immediately in front of the midline of the screen was a small response panel with two red buttons 1cm apart and directly below them one larger grey button. The subject was seated with his/her eyes approximately 40cm from the screen and had freedom of head and eye movement. (Fig.4-1 shows a photograph of a subject seated at the equipment).

There were two experimental conditions; a 'static' condition and a 'dynamic' condition. In each condition the subject's task was to decide whether pairs of shapes were the same or different. Each shape measured 4.5cm horizontally and 2.2cm vertically and the shapes were coloured white on a purple background. The two shapes of each pair were presented successively, and there was a five second interval between the two shapes. A warning bleep immediately preceded each shape. The subject pressed either of the two red buttons for a 'different' judgement and the grey button for a 'same' judgement. The buttons were labelled appropriately. The 18 pairs included six pairs of shapes which differed on the right only, six pairs
which differed on the left only and six pairs which were identical. For each pair of shapes a mirror image pair was also used. Therefore a pair which was different on the left became different on the right in the mirror image form. Drawings of nine of the pairs of shapes can be seen in Figure 4-2. The other nine pairs were the mirror images of these. In both the 'static' and 'dynamic' conditions the pairs of shapes were presented according to a fixed, random schedule with the restriction that trials differing on the right or trials differing on the left would not cluster. The 'static' condition was always given first to ensure that the subject could perform the simpler of the two conditions. Each shape was presented in the centre of the screen for one second and each of the 18 pairs was presented once. In the 'dynamic' condition the shapes appeared to move behind a central vertical slit, 1.4cm wide, each shape taking two seconds to move past the slit.

In this condition, each of the 18 pairs was presented twice, once moving from left to right and once moving from right to left.

These presentation conditions differed slightly from those of Bisiach et al, because of limitations imposed by the computer. While in Bisiach et al's experiment the interstimulus internal for each pair of patterns was one second, in this experiment it was five seconds. In both experiments each pattern took two seconds to move past the slit, but because the projected patterns of Bisiach et al's task were about four times the size of the computerized patterns in this experiment, Bisiach et al's patterns
FIGURE 4-1  A control subject seated at the Apple II Plus computer. The video screen displays a shape from the 'Static' condition of the Imagery experiment.
FIGURE 4-2  Drawings of the nine pairs of shapes used in both the 'Static' and 'Dynamic' conditions of the Imagery experiment. The top three pairs differ on the left, the middle three pairs differ on the right, and the bottom three pairs are identical. The mirror images of these nine pairs were also used.
actually moved past the slit at a much higher speed.

On completion of each condition, a table of results appeared on the screen and these were explained to the subject. The table presented the numbers of same and different responses for each of the pair types (same(S), different on left (DL), and different on right (DR)) and in the 'dynamic' condition these were further divided with respect to direction of movement (See Figs. 4-3 and 4-4 for examples of these Tables). Several brain-damaged subjects had to be excluded from the 'dynamic' condition because they were not able to complete the experiment. Reasons for this were usually that they said it made them dizzy, or gave them a headache or that they could not 'see' the shapes at all (i.e. form images of the shapes) and asked if they could stop.

In order to measure left or right response bias, Laterality Quotients were calculated using the correct responses (CR) for pairs which differed on the right (DR) and the correct responses for pairs which differed on the left (DL). In the 'dynamic' condition there was no consistent trend for any of the subject groups to make more errors when the shapes were moving in a particular direction, so pairs of shapes which differed on the same side but moved in opposite directions were grouped
together. The Laterality Quotient was calculated as follows:

\[
L.Q. (CR) = \frac{CR_{DR} - CR_{DL}}{CR_{DR} + CR_{DL}} \times \frac{100}{1}
\]

Therefore, a positive \( L.Q. (CR) \) indicated neglect of the left side and a negative \( L.Q. \) indicates neglect of the right side. Laterality Quotients for each of the brain-damaged subjects for the two conditions are included in Table 4-1, and for the Control subjects in Table 4-2.
FIGURE 4-3  Computer printouts of responses for the 'Static' condition of the Imagery experiment.

(A) Correct responses (Control subject)
Laterality Quotient = 0

(B) Responses of an RBD patient
Laterality Quotient = + 33.3
(Left-sided neglect)

FIGURE 4-4  Computer printouts of responses for the 'Dynamic' condition of the Imagery experiment.

(C) Correct responses (Control subject)
Laterality Quotient = 0

(D) Responses of an RBD patient
Laterality Quotient = + 28.6
(Left-sided neglect)
<table>
<thead>
<tr>
<th>Subject</th>
<th>Lesion Location</th>
<th>Hemiplegia of Contra-Lesional Hand</th>
<th>Visual Field Defect</th>
<th>Visual Hemineglect (Test Battery)</th>
<th>L.Q. Static Condition</th>
<th>L.Q. Dynamic Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M 54</td>
<td>A</td>
<td>Mild</td>
<td>No</td>
<td>Yes</td>
<td>-50</td>
<td>-40</td>
</tr>
<tr>
<td>M 54</td>
<td>A/P</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>- 9.1</td>
<td>- 5.3</td>
</tr>
<tr>
<td>M 66</td>
<td>P</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>-25</td>
<td>-20</td>
</tr>
<tr>
<td>M 54</td>
<td>P</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>+ 9.1</td>
<td>+ 23.1</td>
</tr>
<tr>
<td>M 54</td>
<td>A</td>
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<td>No</td>
<td>Yes</td>
<td>-20</td>
<td></td>
</tr>
<tr>
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<td>A/P</td>
<td>Mild</td>
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<tr>
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<tr>
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<td>No</td>
<td>No</td>
<td>- 9.1</td>
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<td>RBD</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>M 23</td>
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</tr>
<tr>
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<td>A/P</td>
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<tr>
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<td>P</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>+ 9.1</td>
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</tbody>
</table>

**KEY:**
- **LBD** = left brain damage
- **RBD** = right brain damage
- **A/P** = anterior/posterior
- **P** = posterior
- **A** = anterior
- **L.Q.** = laterality quotient

(A positive L.Q. indicates left-sided neglect and a negative L.Q. indicates right-sided neglect.)
<table>
<thead>
<tr>
<th>Control Subject</th>
<th>L.Q. Static Condition</th>
<th>L.Q. Dynamic Condition</th>
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</thead>
<tbody>
<tr>
<td></td>
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</tr>
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</tr>
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<td>M</td>
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</table>
4.3 RESULTS

4.31 'Static' Condition

The mean L.Q. for the LBD group (N = 9) was -19.86, for the RBD group (N = 9) was +2.29 and for the Control group (N = 20) was -0.5.

An analysis of variance showed that the effect of groups was significant (F = 3.475; df = 2, 35; p < .05). The Scheffe method was then used to compare the groups two at a time. The Control group and the LBD group differed significantly (t = 2.38; df = 35; p < .05), as did the LBD and RBD groups (t = -2.36; df = 35; p < .05). However, there was no significant difference between the Control group and the RBD group (t = -0.34; df = 35; N.S.).

To see if the subjects within each group demonstrated significant hemineglect, their L.Q. scores were compared with scores of zero. The L.Q. scores of the LBD group did differ from zero (t = -2.92; df = 35; p < .01), but those of the RBD and Control groups did not.

4.32 'Dynamic' Condition

The mean L.Q. for the LBD group (N = 4) was -22.1, for the RBD group (N = 5) was +31.78, and for the Control group (N = 20) was -4.16.

An analysis of variance showed that the effect of groups was significant (F = 9.69; df = 2, 26; p < .01). The Scheffe method was then used to compare the groups two at a
time. Significant differences were found between the Control and RBD groups (t = -3.69; df = 26; p < .01), and between the LBD and RBD groups (t = 4.13, df = 26; p < .001), but not between the Control and LBD groups (t = 1.68; df = 26; N.S.).

To see if the subjects within each group demonstrated significant hemineglect, their L.Q. scores were compared with scores of zero. The L.Q. scores of the LBD group did differ from zero (t = -2.27; df = 26; p < .05), as did the L.Q. scores of the RBD group (t = 3.65; df = 26; p < .01). However, the Control group's L.Q. scores did not differ from zero.
4.4 DISCUSSION

4.41 Hemineglect of Images in Brain-Damaged Patients

The results of this experiment confirmed Bisiach et al's (1979) finding that subjects with unilateral cerebral lesions of the right hemisphere tended to neglect the left sides of images. In addition, they show that patients with left-hemispheric unilateral lesions also tend to neglect the contralesional sides of images, although in this case, of course, it is the right sides of images they neglect.

The three groups were found to differ significantly in both the 'static' and 'dynamic' conditions although when groups were compared in pairs in the 'static' condition, the RBD group did not differ significantly from the Control group, while in the 'dynamic' condition it was the LBD group which did not differ significantly from the Control group.

When individual subject L.Q.s were examined for each group and each condition, it was clear that patients were much less likely to consistently neglect the contralesional side of the shapes in the 'static' condition than they were in the 'dynamic' condition. While all patients who performed the 'dynamic' condition displayed contralesional neglect on it, only seven out of nine LBD patients and four out of nine RBD patients displayed contralesional neglect in the 'static' condition. This possibly means that the 'dynamic' condition provides a more sensitive test of visual hemineglect than does the 'static' condition. Given
the additional complexity that must be involved in the constructing of images of shapes over that of simply perceiving shapes, it would be surprising if a deficit of the awareness of space did not disrupt the complex process more than the simpler process.

In Bisiach et al's (1979) experiment, all of the brain-damaged subjects had a "more or less severe left visual field defect" (p.611), 18 out of the 19 had a severe left hemiparesis and all patients demonstrated visual hemineglect on a test of crossing lines. In my experiment, even if we look only at the patients who performed the 'dynamic' condition and who all demonstrated contralesional neglect, only a minority have a visual field defect or a severe hemiparesis, and only two out of the five right brain-damaged patients and three out of the four left brain-damaged patients displayed visual hemineglect on paper-and-pencil tests of hemineglect. That is, hemineglect of a visual image is neither directly nor indirectly related to a visual field defect or a hemiparesis, and hemineglect tests of a more traditional nature (e.g. paper and pencil tests) may not pick up visual hemineglect in all patients.

This could be important in the rehabilitation of patients. For example, a patient with a unilateral lesion who has no field defect and no motor or sensory deficit, and who does not demonstrate visual hemineglect in his/her everyday behaviour or on drawing and construction tests, would probably be permitted to drive a car. However, if he/she does, in fact, have a mild hemineglect disorder
which would have been demonstrated on the imagery task, when driving he/she may well neglect moving objects (e.g. other vehicles, people, animals) which travel in and out of his/her contralesional hemispace. Clearly this is a potentially dangerous situation.

4.42 Implications for Normal Imagery Processes

The finding that some patients with unilateral cerebral lesions actually neglect the contralesional half of an image has implications for the normal cognitive processes involved in imagery. It seems that at some advanced stage in the visual imagery process our images are mapped onto our cerebral hemispheres in an analogue fashion, at least with respect to their left and right sides. That is, objects or parts of objects that we imagine to be on our left are mapped onto our right hemispheres and objects or parts of objects that we imagine to be on our right are mapped onto our left hemispheres. This is not to say that an image reconstructed from past experience (e.g. a map of our childhood home) is necessarily encoded and stored in an analogue form. Indeed storage in terms of an abstract propositional code, according to Pylyshyn (1973), requires less storage capacity and is thus more efficient than is storage in analogue or pictorial terms. Pylyshyn also argues that when memory images are incomplete, whether because of forgetting or because of imperfect registration, the missing information is propositional rather than analogue
in character; an incomplete memory image of some event does not resemble a torn photograph.

Furthermore, it seems reasonable to postulate, at least on the grounds of efficiency and parsimony, that when we encode a spatial map (e.g. the Piazza del Duomo in Milan), we do so in terms of absolute space. That is, the map is encoded and stored as a nonegocentric spatial framework within which objects are located relative to that stationary framework (O'Keefe and Nadel, 1978). However, when we wish to view that map from a particular egocentric position, we can utilize the information contained in the propositional code of the absolute map to construct (imagine) the desired perspective, which is mapped across the cerebral hemispheres in an analogue fashion. So to conclude the example of the patients who neglected the left side of the Piazza del Duomo relative to their imagined perspective, these patients clearly had available to them a complete absolute map of the Piazza del Duomo but their egocentric maps were incomplete because they neglected the imagined space to their left, presumably because their right-hemispheric analogue was missing or 'mutilated' in some way.

One final point raised by this experiment is the distribution of lesion locations which result in the neglect of the contralesional image. Not only do patients with left-hemispheric lesions demonstrate neglect of an image as often as patients with right-hemispheric lesions, but in the left brain-damaged group, the patient who demonstrated the most severe right-sided neglect of images
had an anterior lesion while the right brain-damaged patient with the most severe left-sided neglect of images had a posterior lesion. While the latter case squares with Bisiach et al's (1981) suggestion that the parietal association cortex is the most likely locus for a 'display screen' or 'scratch-pad' for analogue representations, the anterior lesion of the left brain-damaged patient does not. It does, however, conform to the finding reported in Chapter 3 that in left brain-damaged patients, anterior lesions result in visual hemineglect more often than lesions in other locations.

This experiment on the imagery deficits of brain-damaged patients provides an example of how such a study cannot only add to our understanding of a neurological disorder and benefit the rehabilitation of patients with that disorder, but can also provide insights into normal cognitive processes.
5 IPSILATERAL AUDITORY EXTINCTION

5.1 GENERAL INTRODUCTION

EXPERIMENT 1 - Auditory Extinction on a Dichotic Digits Task

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5.23 Assessment of Visual Field Defects, Visual Hemineglect and Tactile Extinction
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5.5 INTRODUCTION

5.6 METHOD

5.6.1 Subjects

5.6.2 Test Procedure

5.7 RESULTS AND DISCUSSION

5.7.1 Control Group

5.7.2 Experimental Groups

5.8 DISCUSSION

5.9 GENERAL CONCLUSIONS
5.1 GENERAL INTRODUCTION

Patients with unilateral brain lesions, when tested under conditions of double simultaneous auditory stimulation, sometimes fail to respond to the contralesional stimulus. This has been called auditory extinction and it is thought to be a form of hemineglect, especially when it occurs in patients who also exhibit visual and sometimes tactile hemineglect (Bender and Diamond, 1965; Diamond and Bender, 1965; Heilman and Valenstein, 1972a; Heilman et al., 1974; Chapter 6, this thesis). This form of auditory extinction implies a deficient function in an auditory hemifield rather than in an ear, and has to date been associated in the human with lesions of the non-dominant parietal and frontal lobes (Heilman and Valenstein, 1972a; Heilman et al., 1974) and the dominant frontal lobe (Damasio et al., 1980; Heilman and Valenstein, 1972a). Another form of auditory extinction related to perceptual processing rather than hemineglect, is associated with lesions of the temporal lobes. Under dichotic stimulation (Broadbent, 1954; Kimura, 1961a) patients with temporal lobectomies may demonstrate a partial suppression of the verbal material arriving in their contralesional ears (Milner, 1962; Oxbury and Oxbury, 1969; Sparks et al., 1970). This is more likely to occur in patients with left-temporal lobectomies and is more marked if Heschl's gyri are also excised (Kimura, 1967; Oxbury and Oxbury, 1969). In patients with right temporal lobectomies it occurs only in cases where Heschl's gyri are removed
(Oxbury and Oxbury, 1969). Deficits of this sort are not associated with hemineglect in other modalities.

In the above examples, the deficit applies to the ear contralateral to the lesion. There are also examples, however, of auditory extinction of material presented to the ear ipsilateral to the lesion, a phenomenon dubbed "paradoxical ipsilateral ear extinction" (Sparks et al, 1970). So far, this has been demonstrated only in the case of left-ear extinction for verbal material following left-hemispheric lesions (Damasio and Damasio, 1979; Damasio et al, 1976; Rubens et al, 1978, Sparks et al, 1970). Since commissurotomized patients also exhibit virtually complete suppression of left ear verbal material (Milner et al, 1968; Sparks and Geschwind, 1968), a possible interpretation of ipsilateral auditory extinction is that the lesion disrupts the interhemispheric route from right to left hemisphere. Under dichotic presentation, it appears that the ipsilateral auditory pathways are somehow suppressed (Kimura, 1961a; Sparks and Geschwind, 1968), so that auditory information reaches the cortex only via the contralateral pathways. Verbal material presented to the left ear would be projected to the right hemisphere, and could then access the speech areas of the left hemisphere only by way of an interhemispheric pathway.1 If this

1. Studies by Morais and Bertelson (1975), Morais (1975), and Pierson et al (in press) provide evidence against this hypothesis. Their data suggest that the hemispheric asymmetry for verbal material may in part depend on the hemispace the auditory stimuli are perceived to come from, rather than their actual position in space or proximity to an ear. These experiments are discussed in Chapter 1.
pathway were disrupted either by a callosal section or by a lesion in the extracallosal portion of the pathway in either hemisphere, the patient would then have difficulty in reporting the material (Sparks et al., 1970).

Damasio and Damasio (1979) have attempted to corroborate this 'disconnection' hypothesis by using Computerized Tomography (C.T.) to locate the lesions of patients with auditory extinction, and so infer the route taken by this interhemispheric pathway. Their sample included four patients with left-hemispheric lesions, speech represented on the left and ipsilateral auditory extinction, one patient with a right-hemispheric lesion, speech represented on the right and ipsilateral auditory extinction, and four each of left- and right-lesioned patients with speech represented on the left and contralateral auditory extinction. A further seven patients exhibited normal dichotic listening patterns. Their conclusions were as follows:

1. Left-hemispheric lesions which resulted in ipsilateral ear extinction always involved the white matter lateral and posterior to the posterior aspects of the lateral ventricle (the posterior paraventricular region). These lesions did not involve the auditory cortex, the geniculocortical pathways or the medial geniculate bodies. Lesions in these latter regions of either hemisphere resulted in contralateral auditory extinction.

2. Right-hemispheric lesions of the posterior paraventricular region also resulted in left-ear
extinction, presumably because these lesions also
damaged the interhemispheric auditory pathway.

(3) Lesions in the frontal or occipital lobes which did not
encroach on the posterior paraventricular regions or
auditory cortices of either hemisphere were not
associated with abnormal dichotic-listening patterns.

Damasio and Damasio (1979) proposed, therefore, that
an interhemispheric auditory pathway does indeed travel in
the splenium of the corpus callosum having first travelled
from each auditory cortex "backward and upward to arch
around the lateral ventricles at the level of the trigone,
joining the corpus callosum in its posterior region."
(p.653). Research on Rhesus monkeys has shown that an
interauditory pathway exists in the splenium of the corpus
callosum, immediately anterior to the intervisual
connections and posterior to the connections that link the
somesthetic areas (Pandya, Hallett and Murkherjee, 1969).
While this provides some evidence in support of Damasio and
Damasio's proposed interauditory pathway, I observed that
some patients with frontal lesions of the left hemisphere
appeared to exhibit ipsilateral auditory extinction on a
dichotic digits test. Such a finding, if reliable, would
not readily conform to Damasio and Damasio's (1979)
disconnection interpretation.

The aim of this study, therefore, is to test an
unselected group of patients with solitary unilateral
cerebral lesions for ipsilateral auditory extinction using
a dichotic digits task. The location of the lesion of each
patient is estimated by its appearance on C.T. scan and the
association of different lesion sites with the presence or absence of ipsilateral auditory extinction is examined. If a number of patients with frontal lesions are found to exhibit ipsilateral auditory extinction, this will be evidence against Damasio and Damasio's proposed posterior interhemispheric auditory pathway.

Experiment 1 tests extinction on a dichotic digits task and is therefore similar to Damasio and Damasio's (1979) study, and Experiment 2 tests extinction on a dichotic tones (non-verbal) task to see if ipsilateral auditory extinction is a general auditory phenomenon or only applies to verbal material.
EXPERIMENT 1
Auditory Extinction on a Dichotic Digits Task

5.2 METHOD

5.21 Subjects

Subjects were patients in the Neurology and Neurosurgical Department of Auckland Hospital, Auckland, New Zealand. All patients who were tested had well-defined focal unilateral cerebral lesions as inferred from clinical data and in all cases confirmed by C.T. scans taken at the time of testing. Patients were excluded from the sample if they were younger than 15 years or older than 69 years or had any hearing defects or asymmetries, previous cerebral disorders, a subnormal I.Q., or a cognitive or communicative disorder such that they were unable to fully understand or respond to the test instructions. Of the 53 patients tested, 18 males and 13 females had left brain damage (LBD), and 13 males and 9 females had right brain damage (RBD). These two groups did not differ significantly with respect to age, sex, nature of lesion or lesion site within the affected hemisphere. The average age for the LBD group was 47.4 years with a range of 15 to 69 years, and for the RBD group was 43.5 years with a range of 17 to 62 years. All patients were left-cerebrally dominant for speech as inferred from handedness and symptoms resulting from their brain lesions.
This group overlapped with, but was not the same as, the group of patients described in Chapter 3.

5.22 Location and Size of Cerebral Lesions

C.T. was performed in a Delta 2020 Scanner and consecutive cuts oriented parallel to the superior orbitomeatal line were obtained. The location of each lesion was estimated and agreed upon by the radiologists, neurologists and neurosurgeons on the basis of its location on C.T. scan and in some cases also on the basis of additional information provided by angiography and sighting during neurosurgery. All lesions were solitary and unilateral and lesion locations were defined as anterior frontal, posterior frontal, frontal, fronto-parietal, anterior parietal, posterior parietal, parietal, parieto-occipital, occipital, temporal, anterior basal ganglia and posterior basal ganglia. Because of the particular significance of the auditory cortex in assessment of auditory deficits, any lesion which involved a temporal lobe was classified as 'temporal' even if it also involved adjacent areas. A 'frontal' lesion involved both anterior and posterior areas of the frontal lobe and a 'parietal' lesion involved both anterior and posterior areas of the parietal lobe. The only lesions that involved cerebral tissue on both sides of the central sulcus were those designated 'fronto-parietal'. All other lesions (except for temporal and basal ganglia lesions) were either wholly anterior or wholly posterior to the central sulcus.
When lesions involved the cortex, this was noted. Using this system of lesion location, the only patients who had lesions possibly involving the area labelled by Damasio and Damasio (1979) as the 'posterior paraventricular region' were those with 'parietal', 'posterior parietal', 'parieto-occipital' or 'posterior basal ganglia' lesions.

Lesions were designated as large, medium or small, depending on an estimate of the size of the lesion as seen on C.T. scan. As a general guide, lesions which in cross section on at least one C.T. section involved one-quarter or more of one hemisphere were graded as large, those involving one-fifth to one-quarter were graded as medium, and those involving less than one-fifth were graded as small. The location and size of each patient's lesion and whether or not it involved the cortex can be found in Tables 5-2, 5-3 and 5-4.

5.23 Assessment of Visual Field Defects, Visual Hemineglect and Tactile Extinction

Visual field defects were independently assessed by confrontation and in some cases by perimetry by a neurologist and myself.

Visual hemineglect was assessed by a battery of five paper and pencil tests. These included Albert's (1973) test in which the patient crosses lines scattered apparently randomly over a page, inserting the numbers on a clock face, copying a Necker cube and a five-point star, and copying a drawing of a tree, house, fence and tree.
Free head and eye movements were permitted. If a patient failed to complete the contralesional half of any one of these, he or she was considered to have visual hemineglect.

Tactile extinction was assessed by double simultaneous stimulation (DSS); that is, light fingertip touch to both hands or to both cheeks simultaneously. If the patient responded correctly to unilateral touch on both sides but failed to respond to the contralesional touch on DSS at least four out of six times on either the hands or cheeks, he or she was considered to exhibit tactile extinction.

5.24 Test Procedure

The stimulus material used for testing auditory extinction was a version of Kimura's (1961a, 1961b) dichotic digits tape. Before testing with dichotic digits, patients were first tested on a series of 48 monaurally presented digits, spoken in groups of three by a male voice and played on the same equipment subsequently used for the dichotic digits. Within each group of three digits the side of presentation shifted randomly from one ear to the other. The patient listened to the digits with cushioned ear phones and repeated the digits after each group of three. All patients who proceeded to the dichotic digit tests made no errors on this task. This test was also used as a check that the volume was perceived by the patient as being the same in each ear and allowed him/her to become
accustomed to the equipment and the task of repeating digits back after hearing groups of three.

The stimulus material used in the two following tests consisted of a cassette of 60 pairs of dichotically presented digits (1 to 9) spoken by a male voice. Groups of three pairs of digits were separated by a silence of 6.8 seconds which gave most patients time to respond. However, when necessary, the tape was stopped to give the patient an unlimited response time. Each group of three pairs of digits was spoken at a rate of 0.66 seconds per digit pair. Each group of three pairs appeared twice; once in the first half of the tape and again in reversed ear order in the second half of the tape. The cassette tape was played on a Sanyo portable stereo radio cassette recorder and the patient and experimenter listened to it in a quiet room each with cushioned stereo earphones. Earphones were reversed for half the patients in each of the LBD and RBD groups. The stimuli were played at a volume which was comfortable for the patient and well above threshold.

TEST 1 Shadowing Digits (S.D.)

In this test it was explained to the patient that he/she would hear pairs of digits, that is, digits spoken two at a time, one in each ear, and that these would be presented in groups of three pairs. The patient was then instructed as follows. "I want you to listen to the numbers in one ear only; this, your right (or left) ear" - at this point the experimenter touched the right (or left) ear of the patient - "and in the gap after the three pairs
I want you to repeat back only the three numbers you heard in this ear. Remember, repeat only the numbers you hear in your right (or left) ear and ignore the numbers in your other ear." If the patient appeared not to understand after the first six pairs, the tape was rewound and the instruction repeated. At the halfway point (after 30 pairs) the tape was stopped and the patient was instructed to listen to and repeat only the digits in the other ear. Once again, the appropriate ear was named and touched by the experimenter. Half of the patients in each of the RBD and LBD groups were instructed to listen to the right ear first and the other half to the left ear first.

TEST 2 Dichotic Digits (D.D.)

The same dichotic digit tape was used as for the 'Shadowing digits' test, but this time the patient was instructed to repeat as many of the digits as possible from both ears and in any order. If necessary, the tape was rewound and the instructions repeated after the first six pairs. (See Appendix for test forms).

For both tests the results were recorded as a laterality quotient \( \frac{R-L}{R+L} \times 100 \) where \( R \) is the number of correct right ear responses and \( L \) the number of correct left ear responses. An incorrect response on the 'Shadowing digits' test can either be no response for the ear being shadowed, or the repeating of digits from the opposite ear when the patient clearly believes he is repeating digits heard in the ear he has been instructed to listen to. In Test 1, the maximum number correct in each
ear is 30, whereas in Test 2 it is 60. A quotient of zero indicates no ear advantage (NEA), a positive quotient a right ear advantage (REA) and a negative quotient a left ear advantage (LEA). While the great majority of normal subjects make no errors on 'Shadowing digits', most show a small REA on 'Dichotic digits'. There does not appear to be any criteria for claiming a REA or LEA for individual subjects on the 'Dichotic digits' test, so in this study patients who had a between ear difference of equal to or greater than 20% were considered to show extinction of the ear with the lower score. That is, only those patients who had laterality quotients of greater than +10 were considered to show extinction of their left ears, and those with laterality quotients of less than -10 extinction of their right ears. In the beginning stages of the study, patients were given the 'Shadowing digits' test only. Later on, the 'Dichotic digits' test was added in order to provide a basis for comparison with other studies of auditory extinction (Damasio and Damasio, 1979; Damasio et al, 1976; Rubens et al, 1978; Sparks et al, 1970). In all, 14 patients were given only the 'Shadowing digits' test and 39 patients were given both the 'Shadowing digits' and 'Dichotic digits' tests.
5.3 RESULTS AND DISCUSSION

5.31 Comparison Between the S.D. and D.D. Tests

To ascertain whether the 'Shadowing digits' and 'Dichotic digits' tests were measuring the same dimension, a correlation coefficient was computed between the two sets of scores for the 39 patients who performed both tests, and a high positive correlation was found ($r = 0.74$; $df = 36$; $p < .001$). While all patients who scored a laterality quotient of greater than +10 or less than -10 on 'Shadowing digits' likewise scored values more extreme than ±10 on 'Dichotic digits', many patients who scored greater than +10 or less than -10 on 'Dichotic digits' had a laterality quotient of 0 or close to 0 on 'Shadowing digits'. One reason, therefore, for preferring the 'Shadowing digits' test is that it appears to provide a much more conservative measure of ear advantage (or conversely, ear extinction) than does the 'Dichotic digits' test. Another reason is that normal subjects virtually always score zero on the 'Shadowing digits' test since they can easily report all digits from either ear. Hence the 'Shadowing digits' quotient is more clearly indicative of cerebral pathology than is the 'Dichotic digits' quotient. Therefore while both 'Shadowing digits' scores and 'Dichotic digits' scores (where available) will be given, patients with 'Shadowing digits' laterality quotients of greater than +10 (left ear extinction) or less than -10 (right ear extinction) can be
considered to demonstrate an extreme and undoubtedly pathological degree of auditory extinction.

5.32 Results of Patients With Auditory Extinction on 'Shadowing Digits'

Table 5-1 gives the number and percentages of patients in the RBD and LBD groups with normal 'Shadowing digits' scores, right ear extinction and left ear extinction.

TABLE 5-1 The number and percentage of patients in each of the right and left brain damaged groups in Experiment 1 who had normal S.D. scores, right ear extinction and left ear extinction.

<table>
<thead>
<tr>
<th></th>
<th>RBD No.</th>
<th>%</th>
<th>LBD No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal S.D.</td>
<td>17</td>
<td>77.3%</td>
<td>17</td>
<td>54.8%</td>
</tr>
<tr>
<td>Right ear extinction</td>
<td>0</td>
<td>0%</td>
<td>3</td>
<td>9.7%</td>
</tr>
<tr>
<td>Left ear extinction</td>
<td>5</td>
<td>22.7%</td>
<td>11</td>
<td>35.5%</td>
</tr>
</tbody>
</table>
All RBD patients who demonstrated extinction on 'Shadowing digits' had extinction of the ear contralateral to their lesion (22.7%). In the LBD group only 9.7% demonstrated extinction of the contralesional ear whereas 35.5% demonstrated paradoxical ipsilateral extinction. The 'Shadowing digits' and 'Dichotic digits' scores and laterality quotients for the 19 patients who had S.D. laterality quotients of greater than +10 or less than -10 are given in Table 5-2.

Of the 11 cases with left-hemispheric lesions that resulted in paradoxical ipsilateral left ear extinction (on the S.D.) only four involved the posterior parietal lobe (Cases 4, 7, 8, 11), thereby corresponding with Damasio and Damasio's (1979) results. Figure 5-12 provides an example of a left-hemispheric lesion sited in the area labelled by Damasio and Damasio (1979) as the "posterior paraventricular region" (Case 8). The remaining seven patients in this group had anterior lesions which did not involve the temporal lobe or the posterior parietal region. None of these seven patients had a visual field defect which provides further evidence for non-involvement of the posterior areas of the hemisphere.

The three patients with left-hemispheric lesions and contralateral auditory extinction all had lesions involving the temporal cortex and therefore were typical of many previously reported cases (Kimura, 1967; Milner, 1962; Oxbury and Oxbury, 1969).

2. All C.T. sections are oriented so that the frontal lobes are to the top of the page and the right hemisphere to the right of the page.
### TABLE 5-2  S.D. and D.D. laterality quotients and nature and location of lesion for patients with auditory extinction in Experiment 1.

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Nature of Lesion</th>
<th>Size of Lesion</th>
<th>Dichotic Right Ear</th>
<th>Dichotic Left Ear</th>
<th>Quotient</th>
<th>Shadowing Right Ear</th>
<th>Shadowing Left Ear</th>
<th>Quotient</th>
<th>Location of Lesion</th>
<th>Cortical Damage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>15</td>
<td>G</td>
<td>M</td>
<td>28</td>
<td>15</td>
<td>+30</td>
<td>19</td>
<td>4</td>
<td>+65</td>
<td>Anterior basal ganglia</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>69</td>
<td>Me</td>
<td>L</td>
<td>51</td>
<td>38</td>
<td>+14</td>
<td>30</td>
<td>16</td>
<td>+30</td>
<td>Frontal</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>63</td>
<td>Me</td>
<td>L</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>26</td>
<td>9</td>
<td>+49</td>
<td>Frontal</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>54</td>
<td>Me</td>
<td>L</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>30</td>
<td>24</td>
<td>+11</td>
<td>Parietal</td>
<td>+</td>
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<tr>
<td>5</td>
<td>M</td>
<td>17</td>
<td>Me</td>
<td>S</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>23</td>
<td>12</td>
<td>+31</td>
<td>Anterior parietal</td>
<td>+</td>
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<td>44</td>
<td>I</td>
<td>S</td>
<td>49</td>
<td>22</td>
<td>+38</td>
<td>30</td>
<td>16</td>
<td>+30</td>
<td>Anterior frontal</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>58</td>
<td>SM</td>
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<td>11</td>
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**KEY:** Sex:  M = male, F = female.  
Size of Lesion:  L = large M = medium S = small  
Nature of Lesion:  I = infarct, Ab = abscess, G = glioma, Me = meningioma, ICH = intracerebral haemorrhage, SM = solitary metastasis.  
Cortical damage:  + = present, - = not present.  
Dichotic digits:  - = not assessed.
The five patients with right hemispheric lesions and contralateral auditory extinction corresponded well with the results of Damasio and Damasio as all had involvement of either the auditory cortex or the posterior parietal area or the posterior basal ganglia. Figure 5-2 is a C.T. scan of a patient with a right posterior parietal glioma who demonstrated left ear extinction (Case 14). This patient also had severe left-sided visual hemineglect and tactile extinction.

5.33 Case Studies of Patients with Ipsilateral Auditory Extinction and Anterior Lesions of the Left Hemisphere

In order to provide examples that will enable the reader to make comparisons with Damasio and Damasio's (1979) results (also cf Figs. 5-1 and 5-2), those C.T. brain scans of patients who had lesions involving the left anterior cortex or anterior basal ganglia and who demonstrated ipsilateral auditory extinction on 'Shadowing digits' are reproduced here in full (Figs. 5-3 to 5-9). Descriptions of these lesions and the clinical symptoms of the patient follow. In each of Figs. 5-3 to 5-9 the scan in the top left-hand corner is the most inferior cut and consecutive cuts follow from left to right so that the most superior cut (through the superior plane of the frontal and parietal lobes) is in the bottom right hand corner.
CASE 1 (Fig. 5-3) is a 15 year old girl with a glioma of the left anterior basal ganglia. On biopsy it was noted that the tumour did not reach the cortex and did not invade the ventricles. The patient had a right-sided hemiparesis with some loss of sensation and mild anomia. She suffered from severe visual and tactile hemineglect of the right side but demonstrated a severe extinction of her left ear. Her laterality quotient on the S.D. was +65.

CASE 2 (Fig. 5-4) is a 69 year old man with a left frontal lobe meningioma. He was remarkably free of symptoms except for some lack of insight (i.e. no anxiety about his brain surgery the next day), a mild right-sided hemineglect and auditory extinction of his left ear. His laterality quotient on the S.D. was +30.

CASE 3 (Fig. 5-5) is a 63 year old woman with a recurrent left parasagittal frontal meningioma. Removal of a meningioma in the same position three years previously had been considered macroscopically complete. (Note in the C.T. scan the surgical clips from the previous surgery). The patient had a mild right hemiparesis and intermittent expressive dysphasia, and demonstrated extinction of her left ear. Her laterality quotient on the S.D. was +49.

CASE 5 (Fig. 5-6) is a 17 year old boy with a small meningioma situated in a parasagittal position in the anterior-superior aspect of the left parietal lobe. His only clinical symptoms other than auditory extinction of
his ipsilesional ear (laterality quotient on the S.D. of +31) was a weakness of his right arm and leg.

CASE 6 (Fig. 5-7) is a 44 year old woman who has an infarct of the left frontal lobe as the result of a subarachnoid haemorrhage. Her presenting symptoms were headaches, vomiting and drowsiness and some transient episodes of expressive dysphasia. At the time of testing, she had recovered well except for tactile neglect of her right side and an auditory extinction of her ipsilesional ear. She had a laterality quotient on the S.D. of +30.

CASE 9 (Fig. 5-8) is a 65 year old woman who has a large fronto-parietal meningioma. She complained of a poor memory and confusion over the six to twelve months prior to her admission. She had poor balance but no leg or arm weakness, no seizures and no visual field defect. She had severe right visual neglect and auditory extinction of her ipsilesional ear. Her laterality quotient on the S.D. was +67.

CASE 10 (Fig. 5-9) is a 48 year old man. The scan reproduced here was taken prior to his having a left frontal glioma removed three years before testing. His most recent scan taken at the time of testing (after a recent grand mal seizure) is not reproduced here as it shows only a small area of enhancement at the left frontal pole and apart from some signs of atrophy of the left frontal lobe, appears relatively normal. The patient at
the time of testing had a mild right hemiparesis and occasional anomia. He demonstrated extinction of his left ear with a laterality quotient on the S.D. of +46. A comment made by this patient when he was attempting the D.D. test (on which he received a laterality quotient of +88) was, "I can hear them all but I can't repeat them."
FIG. 5-1  CASE 8 - A glioma of the left 'posterior paraventricular' region that resulted in ipsilateral auditory extinction.

FIG. 5-2  CASE 14 - a glioma of the right 'posterior paraventricular' region that resulted in left ear auditory extinction.
FIG. 5-3 CASE 1 - A glioma of the left anterior basal ganglia that resulted in ipsilateral auditory extinction.
FIG. 5-4 CASE 2 - A meningioma of the left frontal lobe that resulted in ipsilateral auditory extinction.
FIG. 5-5  CASE 3 - A meningioma of the left frontal lobe that resulted in ipsilateral auditory extinction.
FIG. 5-6  CASE 5 - A meningioma of the anterior-superior aspect of the left parietal lobe that resulted in ipsilateral auditory extinction.
FIG. 5-7 CASE 6 - An infarct of the left frontal lobe that resulted in ipsilateral auditory extinction.
FIG. 5-8  CASE 9 - A meningioma of the left fronto-parietal region that resulted in ipsilateral auditory extinction.
FIG. 5-9  CASE 10  - A glioma of the left fronto-parietal region that resulted in ipsilateral auditory extinction.
5.34 Results of Patients Who Did Not Demonstrate Auditory Extinction on 'Shadowing Digits'

The S.D. and D.D. scores and laterality quotients for the 17 LBD patients and 17 RBD patients who did not exhibit auditory extinction on the 'Shadowing digits' test are given in Tables 5-3 and 5-4 respectively. Of these patients 13 LBD and 12 RBD patients were given the 'Dichotic digits' test as well as the 'Shadowing digits' test. In the main the 'Dichotic digits' results confirm the finding that lesions of the left posterior frontal lobe and the posterior parietal areas of both hemispheres result in some degree of left ear extinction, while right frontal lesions are not associated with auditory extinction at all. The one exception to this was Case 40. This patient had a right frontal glioma with extensive oedema. His lateral ventricles were greatly enlarged which may have resulted in some compression of the posterior regions of either or both hemispheres. In agreement with previous findings (Kimura, 1967; Milner, 1962; Oxbury and Oxbury, 1969) more patients with left temporal lesions demonstrated contralesional auditory extinction (either on the D.D. or on both the D.D. and S.D.) than patients with right temporal lesions.

5.35 Ear Extinction and Size of Lesion

Chi-squared tests revealed no significant association between presence and absence of auditory extinction and size of lesions in either the LBD or RBD groups separately.
**TABLE 5-3** S.D. and D.D. laterality quotients and nature and location of lesion for LBD patients without auditory extinction in Experiment 1.

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Nature of Lesion</th>
<th>Size of Lesion</th>
<th>Dichotic Digits</th>
<th>Shadowing Digits</th>
<th>Location of Lesion</th>
<th>Cortical Damage</th>
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**KEY:**  
Sex: M = male, F = female.  
Size of Lesion: L = large M = medium S = small  
Nature of Lesion: I = infarct, Ab = abscess, G = glioma, Me = meningioma, ICH = intracerebral haemorrhage, SM = solitary metastasis.  
Cortical damage: + = present, - = not present.  
Dichotic digits: - = not assessed.
TABLE 5-4  S.D. and D.D. laterality quotients and nature and location of lesion for RBD patients without auditory extinction in Experiment 1.

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<tr>
<th>Case</th>
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<th>Size of Lesion</th>
<th>Dichotic Digits</th>
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KEY:  Sex:  M = male,  F = female.
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Cortical damage:  + = present,  - = not present.
Dichotic digits:  - = not assessed.
or when both groups were combined. That is, larger lesions are no more likely to produce auditory extinction than are smaller lesions.

5.36 Ear Extinction and Spatial Hemineglect

All patients were tested for visual hemineglect and tactile extinction as part of the wider study, and the possibility that auditory extinction might be an auditory form of spatial neglect was considered. Chi-squared tests revealed no significant association between the presence and absence of auditory extinction and the presence and absence of visual hemineglect and/or tactile extinction in either the LBD or RBD groups separately, or when both groups were combined (see also Chapter 6). This was the case for contralateral auditory extinction alone and when contralateral and ipsilateral auditory extinctions were combined. That is, auditory extinction does not appear to be especially associated with the 'syndrome' of spatial hemineglect. This statistical finding does not, however, exclude the possibility that for some patients contralateral auditory hemineglect is also present and cannot be discriminated from contralateral auditory extinction by dichotic tests. In extreme cases auditory hemineglect can be demonstrated by the patient's lack of response to free field sounds in the space contralateral to his/her lesion. This is usually accompanied by neglect of contralateral visual and sometimes tactile stimuli (De Renzi, 1982).
5.37 Dichotic and Dotic Listening

Given that a disconnection interpretation of ipsilateral auditory extinction on dichotic stimulation requires the suppression of the ipsilateral auditory pathways, one question which arises is whether such a suppression occurs only on dichotic stimulation when each ear is stimulated in isolation from the other or also under conditions of dotic stimulation (Morais and Bertelson, 1973, 1975), when both ears hear the digits but from opposing auditory half-fields.

Four LBD patients who had ipsilateral auditory extinction on 'Shadowing digits' but who were not included in the main part of Experiment 1, were retested for auditory extinction on the 'Shadowing digits' task, but this time the task was dotic as the pairs of digits were played through two speakers on each side of the patient's head, six feet from each ear. The task was otherwise the same. Three of the patients still demonstrated ipsilateral auditory extinction. That is, they exhibited extinction of digits from the left speaker even although they had been instructed to attend to it. The fourth patient demonstrated no marked extinction of the digits from either speaker. The laterality quotients for the earphone and speaker versions of the 'Shadowing digits' task are given for these four patients in Table 5-5.

There are two possible explanations for these results. The first is that dotic listening is very similar to dichotic listening as far as the anatomical
TABLE 5-5  Laterality quotients for the earphone and speaker versions of the 'Shadowing digits' task for four patients with left-hemispheric lesions.

<table>
<thead>
<tr>
<th>Case</th>
<th>Location of Lesion</th>
<th>Laterality Quotients for 'Shadowing digits'</th>
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</tbody>
</table>

pathways are concerned. If this is the case, possibly the suppression of the ipsilateral auditory pathway is not all-or-none, and dichotic stimulation suppresses it more than diotic stimulation. While this might explain the data of the one patient who demonstrated ipsilateral auditory extinction on the earphone task but not on the speaker task, it should also be noted that this could be a manifestation of regression toward the mean. The second possible explanation is that the left-ear extinction is fundamentally a 'hemineglect' disorder in that it is the hemispace which is important rather than the ear of entry. If this were the case the disorder should also be apparent when a non-verbal task is used, as there is no reason to suppose a spatial disorder should be restricted to verbal stimuli or to a task requiring a verbal response. This is tested in Experiment 2.
5.4 DISCUSSION

These results do not agree with those of Damasio and Damasio (1979) who found ipsilateral auditory extinction only for patients with deep lesions in the left 'posterior paraventricular' region. My data does correspond with Damasio and Damasio's data in that included are patients with right-and left-hemispheric posterior parietal lesions and left ear extinction and patients with temporal lobe lesions and contralesional ear extinction. However, I also found a significant number of patients with left frontal or left anterior basal ganglia lesions who demonstrated extinction of their left ears. Damasio et al (1980) also found severe left-sided visual neglect in a patient with a left basal ganglia infarct. They were unable to explain this.

Two other studies demonstrating ipsilateral extinction with left frontal lesions were carried out by Schwartz et al (1977, 1979) using a tactile task. They found that patients with left or right parietal lesions demonstrated tactile extinction when using their contralesional hands but that a significant proportion of patients with left frontal lesions demonstrated tactile extinction when using their left hands. Schwartz et al, (1977, 1979) commented that this would seem to be related to the function of the speech hemisphere as their tactile extinction task required a verbal response. They hypothesized that the tactile information from the left hand follows a diffuse path via the frontal lobes and the
anterior callosum to the left speech centres and is therefore weaker than the signal from the right hand. Damage to this diffuse pathway will weaken the left hand tactile signal even further relative to the right, resulting in left hand tactile extinction. Note, however, that according to this hypothesis, damage to either left or right frontal lobe should result in left hand tactile extinction. Schwartz et al. (1979) did in fact find a number of patients with right frontal lesions and left tactile extinction. In my study, patients with right frontal lesions did not exhibit auditory extinction of their left ears.

Another right-handed patient I tested, who was not included in this sample because she had speech represented either bilaterally or in her right hemisphere (i.e. she suffered expressive aphasia after having a small aneurysm on the dorsal stem of the right middle cerebral artery clipped), also exhibited ipsilateral auditory extinction (S.D. quotient of -29). Some spasm of the right anterior and middle cerebral arteries was observed and no vascular abnormalities were seen in the left hemisphere. While it is possible that the right posterior parietal area may have been affected given the diminished flow on the right, it is more likely that this was a fronto-parietal lesion. If so, this suggests that ipsilateral auditory extinction may follow anterior lesions to the hemisphere dominant for speech, whether it is left or right.

These cases of ipsilateral auditory extinction following anterior lesions to the hemisphere dominant for
speech (usually the left) pose a problem for Damasio and Damasio's (1979) disconnection hypothesis. Their proposed 'pathway' is perhaps too specific to explain ipsilateral auditory extinction. Another structural interpretation couched in more general terms may fit the data better. Such an interpretation is given in the 'General Conclusions' section of this chapter.

However, one possible way to reconcile the data with Damasio and Damasio's hypothesis is to suppose that left anterior lesions disrupt an extension of a posteriorly routed interhemispheric pathway. That is, the interhemispheric pathway, having crossed from the right to the left temporal lobe, might course anteriorly to Broca's area. A difficulty with this interpretation, however, is that one might expect this extended pathway to affect both ipsilateral and contralateral input to Broca's area so that disruption to the pathway should produce contralateral as well as ipsilateral extinction.

There are two possible answers to this objection. One is that ipsilateral input, having been relayed from the right to the left hemisphere, is registered more weakly than contralateral input, which is received directly by the left hemisphere. Added to this, verbal stimuli registered initially in the left (speech) hemisphere may have an intrinsic advantage over verbal stimuli registered initially in the hemisphere non-dominant for speech. Consequently, lesions in this pathway produce a greater disruption to the left ear input. Alternatively, one might suppose that there are distinct pathways to Broca's area.
for ipsilateral and contralateral input, and that only the pathway for ipsilateral input is disrupted by left anterior lesions.

Whatever the pathway for ipsilateral input, it does not seem to involve the arcuate fasciculus, normally considered the route from Wernicke's area to Broca's area in the left hemisphere. Lesions of the arcuate fasciculus typically result in conduction aphasia (Geschwind, 1969), yet none of the seven patients with ipsilateral extinction displayed any symptoms of conduction or receptive aphasia. Four of the seven did exhibit some degree of expressive dysphasia or mild anomia.

A patient who was not included in this sample because he was only 11 years old, nevertheless provided some interesting data. He sustained a large intracerebral haemorrhage in the fronto-parietal region of his left hemisphere. As a result he had a total expressive aphasia and a severe right-sided hemiplegia at the time of his initial testing on the 'Shadowing digits' test. He performed the test by pointing to digits on a paper having first satisfied me that he was able to comprehend complex instructions and point correctly to digits when he heard them. On this occasion he received an S.D. quotient of +74. That is, he demonstrated ipsilateral auditory extinction. He was later able to express himself vocally and at a subsequent testing received a similar S.D. quotient (+71) using a vocal response. If the right hemisphere is mute, but can comprehend and respond non-vocally to verbal material (Zaidel, 1978; Zaidel and
Peters, 1981), then this case raises some questions as to whether a disconnection hypothesis can explain ipsilateral auditory extinction at all. Another query is raised by Damasio et al's (1979) report of a patient with a left anterior basal ganglia infarct and severe ipsilateral visual neglect. Perhaps ipsilateral auditory extinction is not linked to a deficit in perceptual processing as has been supposed. One alternative possibility is that in some cases at least it may be a form of neglect, but why some lesions should result in neglect of stimuli in the ipsilesional rather than the contralesional space is a puzzle. A 'neglect' interpretation would also require an explanation for the finding that some lesions result, in the same patient, in ipsilesional neglect in one modality and contralesional neglect in another modality.
EXPERIMENT 2
Further Studies of Auditory Extinction
Using a Non-Verbal Dichotic Task

5.5 INTRODUCTION

If a disconnection explanation is true, then ipsilateral auditory extinction in patients with lesions of the dominant hemisphere for speech should only occur for tasks with a verbal component. That is, such patients should not demonstrate ipsilateral auditory extinction for non-verbal tasks as there is no necessity for the left-ear input to travel via the corpus callosum from the right to the left hemisphere for decoding or for a non-verbal response. However, this has not yet been demonstrated. On the other hand, it does seem possible that some patients with right-hemispheric lesions might demonstrate right-ear auditory extinction for a non-verbal dichotic task. Patients with right 'posterior paraventricular' lesions who demonstrate left-ear extinction on the 'Shadowing digits' task should, according to Damasio and Damasio's (1979) hypothesis, also have a disconnection of the pathway between the right and left temporal lobes. These patients could therefore demonstrate ipsilateral auditory extinction for non-verbal stimuli if it is necessary for the right-ear input to travel via the proposed interhemispheric pathway from the left to the right hemisphere for decoding. In order to test these possibilities I developed a dichotic
task which used both non-verbal stimuli and a non-verbal response.
5.6 METHOD

5.61 Subjects

A control group consisted of 13 normal right-handed subjects and five normal left-handed subjects who visited a University Open Day and volunteered to take part in the experiment. Their mean age was 32.94 years (age range 17 to 60 years). Handedness was tested using the Edinburgh Handedness Inventory (Oldfield, 1971). The right-handers had Handedness laterality quotients of +67 to +100, and the left-handers had Handedness laterality quotients of -50 to -100.

The experimental groups consisted of nine RBD patients and seven LBD patients with cortical and subcortical unilateral lesions, selected according to the criteria used in selecting subjects for the previous experiment. These patients were not, however, included in Experiment 1. All patients used their right hand for writing and most other tasks. Patients with temporal lobe lesions were not included in this group because of possible damage to the auditory cortex which could result directly in the suppression or extinction of sounds from the contralateral ear (Milner, 1962; Oxbury and Oxbury, 1969; Sparks et al, 1970). The mean age of the RBD group was 40.8 years (age range 20 to 62 years) and of the LBD group was 37.6 years (age range 18 to 59 years).
5.62 Test Procedure

The Shadowing Tones Test (S.T.)

Four non-verbal stimuli were used and these consisted of the same note (middle C) played on four different instruments (piano, recorder, guitar, organ). These were identified for the subject as four different tones, and never identified by the name of the instrument on which they were played. Patients first listened to each of the tones played binaurally over earphones and then listened to a target tone (the recorder note). They then listened to a series of 12 single tones played either through the left earphone, the right earphone or both earphones. They were instructed to raise a finger every time they heard the target tone. If they could do this correctly they were then given a demonstration of each pair of tones played dichotically. The target tone was then repeated and they were instructed to listen to one ear only and ignore the other, and raise a finger each time the target tone was heard in the specified ear. The ear was both identified as the 'right' or 'left' ear, and touched by the tester to ensure that the subject understood which ear to attend to. The subject then listened while two blocks of 28 dichotic pairs of tones were played over the earphones. For the first block of trials he/she was instructed to listen to one ear and for the second block of trials the other ear. Half of the subjects listened to the right ear first and half to the left ear first. The instructions and the target tone were repeated between the two blocks of trials.
Pairs of tones were balanced with respect to ear within each block and across the two blocks. Laterality quotients were then calculated according to the formula $\frac{R-L}{R+L} \times \frac{100}{1}$ where $R$ and $L$ are the number of trials correct out of the 28 trials in the block in which the subject was instructed to listen to the right and left ears respectively. (See Appendix for data forms and script for the 'Shadowing Tones' test).
5.7 RESULTS AND DISCUSSION

5.71 Control Group

The right-handed subjects demonstrated a significant left-ear advantage on 'Shadowing tones' (t = -2.89; df = 12; p < .02) while the left-handed subjects demonstrated no ear advantage (t = -0.43; df = 4; N.S.). The right- and left-handed groups also differed significantly on 'Shadowing tones' (t = 3.15; df = 16; p < .01). These results imply a right-hemispheric dominance for these particular non-verbal stimuli when they are presented dichotically (Kimura, 1964).

5.72 Experimental Groups

The RBD group had a mean right-ear advantage (L.Q. = +3.94) but did not differ significantly from the right-handed control group (t = 1.94; df = 20; N.S.). The LBD group had a mean left-ear advantage (L.Q. = -3.74) but did not differ significantly from the right-handed control group (t = 0.97; df = 18; N.S.). That is, patients with cortical or subcortical unilateral lesions which excluded the temporal lobes but did in some cases include the anterior cortex or the posterior paraventricular white matter, did not demonstrate ipsilateral auditory extinction for non-verbal stimuli whichever hemisphere was lesioned. The five patients who had left-hemispheric lesions and demonstrated ipsilateral auditory extinction for digits,
either demonstrated a small left-ear advantage for the non-verbal stimuli, or no ear advantage (see Table 5-6). In other words, these patients did not differ from normal subjects who also demonstrate a small left-ear advantage or no ear advantage for musical stimuli (Kimura, 1964). Schuloff and Goodglass (1969) and Sparks et al (1970) also found a falling off in efficiency of the right ear for tonal sequences in patients with left-hemispheric damage.

**TABLE 5-6** Laterality quotients on the 'Shadowing digits' (S.D.) and 'Shadowing tones' (S.T.) for those patients with left-hemispheric lesions who demonstrated ipsilateral auditory extinction on 'Shadowing digits' in Experiment 2.

<table>
<thead>
<tr>
<th>Case</th>
<th>Location of Lesion</th>
<th>Laterality Quotients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>S.D.</td>
</tr>
<tr>
<td>55</td>
<td>Parietal</td>
<td>+ 76</td>
</tr>
<tr>
<td>58</td>
<td>Frontal</td>
<td>+ 46</td>
</tr>
<tr>
<td>59</td>
<td>Frontal</td>
<td>+ 30</td>
</tr>
<tr>
<td>60</td>
<td>Frontal</td>
<td>+ 13</td>
</tr>
<tr>
<td>61</td>
<td>Parieto-occipital</td>
<td>+ 45</td>
</tr>
</tbody>
</table>
5.8 DISCUSSION

The finding that LBD patients who demonstrate left-ear ipsilateral auditory extinction for digits do not demonstrate ipsilateral auditory extinction for the tones supports the disconnection hypothesis. Conversely, it provides evidence that ipsilateral auditory extinction for digits is not a form of hemineglect because if it were one might expect to find ipsilateral extinction for non-verbal stimuli as well. That is, hemineglect is not dependent on the type of stimuli received but rather on the hemispace from which the stimuli originate.

Patients with right-hemispheric lesions also did not demonstrate ipsilateral auditory extinction of the tones, even if the lesion was in a 'posterior paraventricular' location. There are three possible explanations for this. The first is that the proposed interhemispheric pathway which connects the two temporal lobes (Damasio and Damasio, 1979; Sparks et al, 1970) and serves to transfer verbal information from the right to the left hemisphere, does not serve to transfer non-verbal information from the left to right hemisphere. The second possibility is that the transfer of verbal and non-verbal auditory information between the hemispheres is not restricted to a single pathway and that in fact such a pathway may not exist. The third possibility is that non-verbal stimuli can be processed in the hemisphere contralateral to the ear of input whether it be the right or left hemisphere, and a non-verbal response can be correctly made. This latter
explanation seems the most likely as it is well established that while a small left ear advantage is often found for some types of non-verbal stimuli such as melodic patterns (Kimura, 1964; King and Kimura, 1972) such stimuli are not as clearly lateralized to the right hemisphere as verbal stimuli are to the left (Bever and Chiarello, 1974; Gates and Bradshaw, 1977).
5.9 GENERAL CONCLUSIONS

It appears that ipsilateral auditory extinction is indeed restricted to patients with lesions of the hemisphere dominant for speech, and that it is only demonstrable on dichotic or diotic tasks which involve verbal stimuli and/or responses. An equivalent deficit has not been found for non-verbal stimuli in patients with lesions of the hemisphere non-dominant for speech.

A tentative explanation for the deficit is that the ipsilateral auditory pathways are suppressed under conditions of dichotic or diotic stimulation and therefore verbal input to the left ear must travel to the left hemisphere for decoding or processing via the corpus callosum. While Damasio and Damasio (1979) have postulated that left-ear ipsilateral auditory extinction is the result of a disconnection of a posterior interhemispheric pathway by a 'posterior paraventricular' lesion, it is difficult to see how such a disconnection could readily explain why both 'posterior paraventricular' and anterior left-hemispheric lesions should result in left-ear extinction. A more parsimonious explanation is that the damaged left hemisphere, particularly if lesioned in one of the areas involved in the mediation of language, is less able to decode or process the left-ear verbal input which, even in an undamaged brain, is weaker than the right-ear verbal input. This weak signal from the left ear is a result of the indirect route it must travel (i.e. via the corpus callosum) in order to reach the left hemisphere. While
the right hemisphere does demonstrate some degree of dominance for some types of non-verbal and musical stimuli, this dominance is not as marked as the left-hemispheric dominance for verbal material. Therefore, the converse result is not found for patients with right-hemispheric lesions on a dichotic task which uses non-verbal stimuli. Presumably such stimuli can be decoded and processed in either hemisphere.

Given that ipsilateral auditory extinction appears to be restricted to tasks which involve a verbal component, it seems that a 'neglect' interpretation for ipsilateral auditory extinction is not feasible, in spite of the occasional finding of ipsilateral neglect in other modalities (Damasio et al., 1980; Schwartz et al., 1977, 1979). However, as many patients who exhibited contralateral auditory extinction on the 'Shadowing digits' test also made more errors on the non-verbal stimuli arriving at the contralateral ear on the 'Shadowing tones' test, it is possible that for these patients the deficit is a true 'hemineglect' disorder. In the case of contralateral extinction, it is not possible on the basis of these dichotic tests to discriminate a 'neglect' interpretation, which probably involves the disruption of higher cognitive processes, from a simpler structural interpretation.
6. MULTIMODAL HEMINEGLIGENCE

6.1 INTRODUCTION

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6.1 INTRODUCTION

6.1.1 Overview

So far in this thesis I have discussed research on two neuropsychological deficits, visual hemineglect and auditory extinction. These are both unilateral disorders of space, but whether the unilateral neglect of a stimulus and the unilateral extinction of a stimulus are different forms of the same disorder or conversely have quite separate underlying causes, is unknown given our present state of knowledge.

If, for the moment, it is assumed that neglect and extinction are closely associated and are grouped together as a single 'hemineglect' disorder, the next question which must be asked is whether visual and auditory hemineglect are the result of the same spatial deficit. Most researchers in the field have suggested that they are (Bisiach et al, 1978, 1979, 1980; De Renzi et al, 1970; Friedland and Weinstein, 1977; Heilman and Valenstein, 1972a; Mesulam, 1981; Weinstein and Friedland, 1977). De Renzi et al's (1970) suggestion that hemineglect is a 'mutilated representation of space' (see Chapter 4) also implies that hemineglect transcends particular modalities, and therefore it should not be surprising to find a single patient displaying hemineglect of visual, auditory and tactile stimuli and ignoring one half of his/her body as well. Such patients certainly do exist, although in humans visual hemineglect is by far the most frequently observed
form of neglect. This may reflect the dominance of vision over the other spatial senses in humans (De Renzi, 1982).

The study of the multimodal nature of hemineglect has usually been secondary to studies of neglect in modalities other than vision, and the results of these suggest that auditory and tactile hemineglect and extinction are almost always found in association with visual hemineglect (Bender and Diamond, 1965; Denny-Brown et al., 1953; De Renzi et al., 1970; Diamond and Bender, 1965; Heilman et al., 1972; Heilman and Valenstein, 1972a, 1972b). Hemiasomatagnosia (neglect of the contralateral half of the body) and anosognosia (denial of a contralateral hemiplegia) are not so clearly associated with visual hemineglect. For example, Cutting, (1978) found that visual hemineglect occurred in only 52% of patients who had anosognosia. However, anosognosia may not be a neglect disorder, and there have been very few studies carried out in hemiasomatagnosia which is a much rarer disorder and has a more definite 'neglect' quality about it. Clinical observations certainly suggest that patients who have an unambiguous neglect of the body almost always have a severe visual hemineglect, and usually a tactile neglect as well (Frederiks, 1969).

So while these studies tell us that when hemineglect is multimodal, visual hemineglect is almost always present, they do not tell us the relative frequencies of multimodal and unimodal hemineglect. Another shortcoming of these studies is that they look almost exclusively at patients with right-hemispheric damage. Given the finding reported
in Chapter 3 that the locations of the lesions most likely to result in visual hemineglect are posterior in the right hemisphere and anterior in the left hemisphere, it is possible that neglect following left brain damage may not follow the same modality 'pattern' as neglect following right brain damage.

In an attempt to remedy these gaps in our knowledge, I tested a large group of right and left unilaterally brain-damaged patients for hemineglect or extinction in the visual, auditory and tactile modalities as well as for hemineglect of the body (hemiasomatagnosia). Because of probable differences in the sensitivity of the various tests used to assess neglect and extinction in the different modalities, and because I tested for extinction only in the auditory and tactile modalities, the resulting frequencies of multimodal hemineglect are, at best, an estimate of the minimum numbers of patients with multimodal hemineglect. That is, if I had equally sensitive tests for neglect in each of the modalities I might have identified more cases of multimodal hemineglect. However, what this study can show are the relative frequencies of multimodal hemineglect in left and right brain-damaged patients, given that both hemispheric groups were tested with the same tests.

In designing a study like this, two problems of definition arise. Firstly, there is the problem mentioned earlier of whether to categorize hemineglect and extinction as a single disorder or as separate disorders. Secondly, there is the problem posed by ipsilateral hemineglect and
extinction. Are these variants of contralateral hemineglect and extinction or do they comprise a different disorder altogether?

6.12 Hemineglect and Extinction

The behavioural *distinction* between hemineglect and extinction is that patients with the former neglect objects and stimuli presented in the contralesional hemispace alone, whereas patients and animals with the latter, fail to respond to a stimulus presented in the contralesional hemispace only when a second stimulus is presented simultaneously on the other side. It is, however, a fine line which divides neglect from extinction in many situations. For example, when a patient copies a star or crosses lines distributed over a page and fails to complete one side (see Chapter 3), this is traditionally called hemineglect. But perhaps this could also be termed extinction, as the patient is receiving similar stimulation simultaneously from both hemispheres.

However, it is still necessary to decide whether hemineglect and extinction should be classed as one or two disorders. A satisfactory solution to this problem does not seem possible at present as there is evidence for and against either possibility. Both deficits always involve a unilateral unawareness of, or lack of response to a stimulus and a number of workers in this field believe extinction to be an attenuated form of neglect (De Renzi, 1981; Heilman and Valenstein, 1972a). Many animal
Researchers go so far as to equate extinction with neglect (Denny-Brown and Chambers, 1958; Heilman, Pandya and Geschwind, 1970; Schwartz and Eidelberg, 1968; Welch and Stuteville, 1958). This view that extinction is simply a milder form of hemineglect implies that patients who demonstrate hemineglect on traditional tests should also display extinction on double simultaneous stimulation. However, as I reported in Chapter 3, a number of patients who demonstrated visual hemineglect on the hemineglect test battery did not exhibit visual extinction. While this is evidence against extinction being an attenuated form of hemineglect, it does not exclude the possibility that extinction is a form of hemineglect which some patients demonstrate and others do not, regardless of the severity of the underlying hemineglect disorder. There is no doubt that patients with hemineglect are very variable in the neglect behaviours they display. For example, even within the paper and pencil tests described in Chapter 3, some patients would neglect the 'Scene' test but not the 'cube' or 'star', while others would do the opposite. Individual patients will even neglect different drawings on different days. So it would not be particularly incongruous if some patients with hemineglect also displayed extinction, while others did not.

One objection to considering neglect and extinction as a single disorder is the frequent finding that hemineglect in humans is associated primarily with right-hemispheric lesions, whereas extinction is equally frequent after damage to either hemisphere (Weinstein and
This objection is weakened by the data of Chapter 3, which also show an equal frequency of hemineglect following left- and right-hemispheric lesions. It was not clear, however, whether the unusually high incidence of hemineglect following left-hemispheric lesions was due to the presence of aphasic patients, who are normally excluded, or whether it was due to the patients being tested during the early or 'active' phases of their lesions. If the latter were the case, then the inter-hemispheric imbalance in the incidence of neglect may well hold for patients with long-term stable or 'passive' lesions, and the objection may still carry some weight.

Nevertheless, most research on unilateral spatial deficits in the auditory modality use the extinction paradigm (Bender and Diamond, 1965; Denny-Brown et al., 1952; Diamond and Bender, 1965; Heilman and Valenstein, 1972a, Heilman et al., 1974). This is probably because auditory hemineglect has proven difficult to define and test. Patients are usually said to have the disorder if they answer to the right when addressed from the left (or vice versa) even if, for example, the person they turn to is a man and they were addressed by a woman's voice. However, this is not strictly hemineglect as the sounds are perceived and correctly responded to but their source is shifted to the ipsilateral side (De Renzi, 1982). This type of mislocation, or alloacusis, has been reported a number of times and has only been observed in patients with severe visual hemineglect (Wortis and Pfeffer, 1948; Denny-Brown et al., 1952; Battersby et al., 1956; Schott

Auditory extinction on bilateral simultaneous stimulation is often considered a mild form of hemineglect, and Heilman and Valenstein (1972a) in their article on auditory hemineglect in man clearly state that auditory hemineglect and auditory extinction are one and the same disorder, and all their patients with auditory extinction (or neglect as they called it) also had visual and tactile hemineglect. Research on unilateral spatial deficits in the tactile modality is also usually based on the extinction paradigm (Critchley, 1949; Gainotti, Caltagirone, Lemmo and Micelli, 1975; Heilman and Valenstein, 1972b; Schwartz, Marchok and Flynn, 1977) although a study by De Renzi et al., (1970) described in detail in Chapter 4 of this thesis, did assess tactile hemineglect rather than tactile extinction.

The authors of the few studies which do provide some data on multimodal hemineglect tend to take the point of view that extinction and hemineglect are associated to some degree at least (Heilman and Valenstein, 1972a; Weinstein and Friedland, 1977).

Therefore, in this study I 'equate' extinction and hemineglect and look at the associations between visual hemineglect, tactile and auditory extinction, and behaviours that imply hemineglect of the body.
6.13 Ipsilateral Hemineglect and Extinction

Hemineglect refers to the neglect of stimuli in the contralateral hemispace, and therefore patients with unilateral lesions who demonstrate a neglect or extinction of stimuli in the ipsilateral hemispace cause a problem. It seems logical to suppose that the perceptions and representations of each hemispace are predominantly a function of the contralateral hemisphere in the same way that sensory and motor functions are mediated by the contralateral hemisphere. It is therefore difficult to see how ipsilateral hemineglect or extinction can fit into the hemineglect disorder, even although it is also a unilateral neglect of space.

In this study, ipsilateral hemineglect only became a significant problem when I was testing for auditory extinction. For the above reason and others which I will explain in the 'Method' section, I included only contralateral auditory extinction in this analysis of multimodal hemineglect. That is, in this study I look at the association of visual, tactile, body and auditory neglect and extinction of stimuli in the contralesional hemispace in a group of right and left brain-damaged patients who were assessed for all of these.
6.2 METHOD

6.21 Subjects

There were 19 women and 19 men with lesions confined to the right hemisphere (RBD). The mean age of the women was 44.84 years (range 8 to 74 years) and the mean age of the men was 46.00 years (range 17 to 76 years). In the group of patients with left-hemispheric lesions (LBD) were 20 women and 23 men. The mean age of the women was 45.85 years (range 15 to 65 years) and of the men, 48.22 years (range 11 to 69 years). All patients were selected according to the criteria described in Chapter 2 with the additional criterion that patients were right-handed and patients with severe sensory loss to either hand or cheek were excluded on the grounds that they could not be tested for tactile extinction. Patients with a hearing loss were also excluded because they could not be tested for auditory extinction. This sample of patients was not identical to either of the samples used in the studies reported in Chapters 3 or 5, although they overlapped to a large extent. In fact, 75 patients were common to this study and that reported in Chapter 3, and 50 patients to this study and that reported in Chapter 5. Some patients described in Chapters 3 and 5 could not be included in this study because they were not tested for neglect or extinction in all four modalities or because they had severe sensory loss. On the other hand, this study includes patients who were tested after the data for the studies reported in Chapter 3 and 5 had been analysed.
6.22 Test Procedure

All patients were given the same tests for visual hemineglect, tactile extinction, body hemineglect and auditory extinction. These tests are described below.

Visual hemineglect

Patients were given the test battery described in Chapter 3 and were designated as having visual hemineglect if they demonstrated contralateral neglect on at least one of the five tests in this battery. Note that visual extinction on bilateral simultaneous stimulation is not included as 26 patients (32%) could not be tested for it because of a visual field defect.

Tactile extinction

Patients were not tested for tactile hemineglect (for examples of this see De Renzi et al., 1970; and Chedru, 1976), but were tested for tactile extinction using the standard procedure of bilateral simultaneous stimulation to the hands and to the cheeks. Patients who consistently perceived unilateral touch to each hand and cheek, but who failed to respond to touch on the contralateral hand (or cheek) at least four out of six times when a second touch was applied simultaneously to the other side, were considered to demonstrate tactile extinction. Schwartz, Marchok and Flynn (1977) and Schwartz, Marchok, Kreinick and Flynn (1979) have developed a much more exacting test for tactile extinction, the 'Quality Extinction Test'. However, their results showed a number of parallels with those of the auditory extinction test I used, and therefore
raised the same doubts. In both cases, ipsilateral extinction was often demonstrated by patients with left frontal lobe lesions and this appeared to be influenced not only by deficits of spatial functioning but also by factors related to the verbal response required (see Chapter 5).

Hemineglect of the body (Hemiasomatagnosia)

This was defined rather more subjectively than was visual hemineglect or tactile extinction. A patient was classified as having body hemineglect if he/she behaved as if uninterested in the contralesional side of the body by, for example, frequently leaving half the body outside the bed covers, or allowing an arm or leg to fall off the wheelchair armrests and footrests even after this was pointed out, or 'forgetting' to dress contralesional limbs. Such patients, when asked about these behaviours, remained apparently unmotivated or unconcerned to do anything about them. One of my patients with body hemineglect hopped around on her ipsilesional leg, even although she had only a mild weakness of her contralesional leg. When asked why she did this she asked in return, "Well, how else am I to get around?"

Auditory extinction

This was tested using the 'Shadowing digits' test (S.D.) which is described in detail in the 'Test Procedure' section of Chapter 5. All patients who were given this test had normal hearing according to Weber's test and Rinne's test (Adams and Victor, 1981). A patient was
considered to exhibit auditory extinction if his/her laterality quotient \( L.Q. = \frac{R-L}{R+L} \times \frac{100}{1} \) was greater than +10 or less than -10.

Both the test paradigm I used to test auditory extinction and the data it produced departed from those of previous studies of auditory extinction (Bender and Diamond, 1965; Denny-Brown et al., 1952; Diamond and Bender, 1965; Heilman and Valenstein, 1972a, Heilman et al., 1974). The essential difference in this test paradigm was the use of earphones to deliver the bilateral simultaneous auditory stimuli, rather than delivering them bilaterally in a free acoustic field. However, dichotic listening does not seem to differ very much from diotic listening, according to the data reported in Chapter 5. That is, patients who demonstrated extinction of the digits in a particular ear on the 'Shadowing digits' test when it was played over earphones, tended to demonstrate extinction of the digits in the same ear for the 'Shadowing digits' test played over bilaterally placed speakers. A more important difference between this auditory extinction test and the tests used by other researchers, concerned the responses to the test given by some patients with lesions of the hemisphere dominant for speech. The ipsilateral auditory extinction demonstrated by many of these patients suggested that this form of auditory extinction may not be one of the hemineglect disorders but instead a disconnection deficit that is dependent on the verbal nature of the task. As
previous researchers have used non-verbal stimuli such as clicks and jangling coins, this may be the reason they have not found ipsilateral auditory extinction. By contrast, the contralateral extinction demonstrated by all patients with right-hemispheric lesions and by a small proportion (22%) of patients with left-hemispheric lesions who had a deficit on this test, is likely to be the same disorder reported by other researchers (Bender and Diamond, 1965; Denny-Brown et al., 1952; Diamond and Bender, 1965; Heilman and Valenstein, 1972a; Heilman et al., 1974) and identified by them as auditory extinction. Although it is possible to apply the same disconnection hypothesis used to explain ipsilateral auditory extinction to contralateral auditory extinction in certain cases when the patient has a 'posterior paraventricular' lesion (see Chapter 5), an equally likely hypothesis is that contralateral auditory extinction is a true hemineglect disorder in the sense that it is, like visual hemineglect, the neglect of stimuli in the contralesional hemispace. Therefore, for the purposes of this analysis of multimodal hemineglect, only patients with contralateral auditory extinction are considered to have the hemineglect disorder in the auditory modality and patients with ipsilateral auditory extinction are categorized as not having an auditory hemineglect deficit.
6.3 RESULTS

6.31 Incidence, Sex and Age

There were 15 women and 9 men in the RBD group who had hemineglect in one or more of the four modalities. The mean age of the women was 47.53 years (range 8 to 74 years) and of the men, 44.78 years (range 17 to 76 years). Of these 24 patients, 14 had multimodal hemineglect. Three of these had hemineglect in all four modalities, five had hemineglect in three modalities and six had hemineglect in two modalities. Eight patients had visual hemineglect alone, two patients had auditory extinction alone, but no patients had either tactile extinction or body hemineglect alone. Two patients had multimodal hemineglect without visual hemineglect and five patients had multimodal hemineglect without auditory extinction.

There were 10 women and 16 men in the LBD group who had hemineglect in one or more of the four modalities. The mean age of the women was 46.4 years (range 15 to 65 years) and of the men, 53.38 years (range 11 to 69 years). Of these 26 patients 10 had multimodal hemineglect. One of these had hemineglect in all four modalities, three had hemineglect in three modalities and six had hemineglect in two modalities. Eleven patients had visual hemineglect alone, four patients had tactile extinction alone, one patient had body hemineglect alone and none had unimodal auditory extinction. Two patients had multimodal
hemineglect without visual hemineglect and six patients had multimodal hemineglect without auditory extinction.

6.32 Locus and Side of Lesion

The patients were also classified according to whether the lesion was anterior (A), anterior/posterior (A/P), or posterior (P). Chi-square tests failed to reveal significant differences between RBD and LBD patients in the distribution across these lesion categories, regardless of whether all patients with some form of neglect were included in the comparison, or whether separate comparisons were undertaken for each form of neglect. Nevertheless, in the case of visual neglect, there were anterior-posterior differences comparable to those observed in Chapter 3, although the comparison did not reach significance \( (X^2 = 5.17, df = 2, .05 < p < 10) \): of the LBD patients with visual neglect six had anterior and six posterior lesions, whereas only one RBD patient with visual neglect had an anterior lesion while 10 had posterior lesions.

RBD and LBD patients were also compared, regardless of lesion location, with respect to whether they had multimodal or unimodal neglect. There was no significant difference. Tables 6-1 and 6-2 give the individual patient data on multimodal hemineglect for the RBD and LBD hemineglect patients respectively. Included in these tables is the location of each patient's lesion and whether or not the patient had a visual field defect. Also
<table>
<thead>
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<th>Sex of Subject</th>
<th>Age</th>
<th>Lesion Location</th>
<th>Visual Field Defect</th>
<th>Visual HN</th>
<th>Tactile Extinction</th>
<th>Body HN</th>
<th>Auditory Extinction</th>
</tr>
</thead>
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<td>P</td>
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<td>X</td>
<td>X</td>
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</tr>
</tbody>
</table>

KEY: M = male  F = female  P = posterior lesion  
A/P = anterior/posterior lesion  A = anterior lesion  
B.G. = basal ganglia lesion  + = present  
X = not present  HN = hemineglect  I = ipsilateral  
C = contralateral
TABLE 6-2  Patients with left-hemispheric lesions and hemineglect and extinction in different modalities

<table>
<thead>
<tr>
<th>Sex of Subject</th>
<th>Age</th>
<th>Lesion Location</th>
<th>Visual Field Defect</th>
<th>Visual HN</th>
<th>Tactile Extinction</th>
<th>Body HN</th>
<th>Auditory Extinction</th>
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</table>

KEY: As for Table 6-1

NOTE: The last four patients have ipsilateral auditory extinction only.
included are LBD patients who had ipsilateral auditory extinction.

6.33 Correlations Between the Different Modalities of Neglect

The four modalities of hemineglect were inter-correlated, using the phi-coefficient, separately for RBD and LBD patients (see Table 6-3). Although all positive these correlations are all very low, and only that between body and tactile hemineglect in the RBD group is significant ($X^2 = 4.33$, df = 1, $p < .05$). With this exception, there is therefore little evidence that the overlap between different modalities is due to anything but chance. Nevertheless the low correlations can be attributed in part to the fact that different proportions of patients showed neglect on the different tests, which would have attenuated the values of the phi-coefficient. If we can suppose that these different proportions are due to varying sensitivities of the tests rather than to intrinsic differences between modalities in the incidence of neglect, then a more realistic estimate of the correlations may be provided by tetrachoric correlations shown in Table 6-4. The tetrachoric correlation corrects for varying proportions on the assumption that a normal distribution underlies each dichotomy, and provides an estimate of the product-moment correlation between these assumed normally distributed variates. These estimates must be considered rough, especially in cases where the
dichotomies are extreme. Nevertheless they are considerably higher than the corresponding phi-coefficients, which may therefore substantially underestimate the "true" correlations.
TABLE 6-3  Phi correlation coefficients between neglects in different modalities for the RBD and LBD groups.

<table>
<thead>
<tr>
<th></th>
<th>Visual Hemineglect</th>
<th>Auditory Extinction</th>
<th>Tactile Extinction</th>
<th>Body Hemineglect</th>
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<td>Body</td>
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LBD

TABLE 6-4  Tetrachoric correlation coefficients between neglects in different modalities for the RBD and LBD groups.

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<tr>
<th></th>
<th>Visual Hemineglect</th>
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6.4 DISCUSSION

6.41 A Single, Underlying Cause for Multimodal Neglects?

This study generally confirms previous reports that hemineglect is often multimodal and when it is visual hemineglect is usually present. These conclusions hold not only for patients with right brain damage, the group most often reported in the literature, but also for patients with left brain damage. The two hemispheric groups did not differ significantly on the proportions of patients who had multimodal rather than unimodal hemineglect, although patients with multimodal hemineglect in the RBD group tended to demonstrate neglect across more modalites than did LBD hemineglect patients. For example, of the patients with hemineglect, eight RBD patients demonstrated hemineglect in three or four modalities and six in two modalities, whereas in the LBD group five patients demonstrated neglect in three or four modalities and 12 in two modalities. Ten RBD patients and 13 LBD patients had unimodal hemineglect.

The observation that many patients do have hemineglect in more than one modality does not necessarily mean that there is a single underlying cause for hemineglect. The very small phi-correlation coefficients and the finding that only one of these correlations is significant suggests, in fact, that there may not be a common basis for hemineglect in the different modalities. That is, with the possible exception of body and tactile
hemineglect in the right brain-damaged group the overlap between the different modalities can be attributed to chance. Even the slight tendency toward positive correlations can be attributed to lesions overlapping different modality-specific areas.

On the other hand, the high tetrachoric correlations point to the possibility that there may be a common underlying cause for some of the neglect if the variations in the proportion of patients showing neglect in the different modalities are due to the varying sensitivities of the tests. The tetrachoric correlation coefficient assumes however that a normal distribution underlies each dichotomy, and this may be dubious in the context of hemineglect.

Assuming for the moment that the tetrachoric coefficients are a more valid indicator than the phi-coefficients of the correlations between the different modalities of neglect, it could by hypothesized that there is a common underlying cause for neglect. The fact that visual hemineglect is much more frequent than other forms of neglect could be explained by the notion that vision is the dominant modality used by humans in the exploration of space, and that the severity of the underlying neglect disorder is the deciding factor in how many other modalities are affected. What is more difficult to explain is the occasional occurrence of unimodal hemineglect in a modality other than vision. This is much more readily explained by the theory that each form of hemineglect is, to a large extent, independent of other
forms although the areas which produce neglect when lesioned are topographically close so that they often overlap.

A counter argument to this is that hemineglect-producing lesions are found in a number of cortical and subcortical areas, which according to one theory comprise a cortico-limbic reticular loop which mediates attention and arousal (Heilman and Valenstein, 1972b, Watson, et al., 1973, 1974). The suggestion is that when this loop is damaged it causes a predominantly contralateral disruption of attention-arousal which results in a hemineglect disorder which presumably should transcend specific modalities.

The truth probably lies somewhere between these two extremes, one being that there is a single underlying cause for all the neglects, and the other that neglect in each modality is independent of neglects in other modalities. For example, one possibility would be that there is an attention-arousal deficit caused by a disruption of a cortico-limbic reticular loop, but that the modality through which the hemineglect disorder is expressed depends upon the particular cortical or subcortical area lesioned. This idea can certainly be entertained in the case of body hemineglect which is usually found only in hemiparetic patients implying that it is associated with specific lesions.

In this study the only significant phi-correlation was between body and tactile hemineglect in RBD patients and this suggests that these two neglects at least might have some unitary underlying cause. These neglects are
similar in that they are both concerned with the concept of personal space whereas visual and auditory neglect usually concern extrapersonal space. Body neglect is also the least frequently observed form of neglect which might also be explained by its greater dependence upon lesions of specific areas, relative to visual neglect. The majority of patients with body hemineglect have been found to have lesions of the parietal lobe and the subadjacent white matter (Roth, 1949) although a few cases with thalamic lesions have also been reported (Carreras, De Risio and Visintini, 1968). In this study, in both right- and left-hemispheric groups, patients with body hemineglect had basal ganglia lesions, posterior lesions or anterior/posterior lesions. None had anterior lesions. Visual hemineglect has been observed after lesions in a variety of areas, including the anterior cortex (Bisiach et al, 1981; Critchley, 1953; Damasio et al, 1980; Gloning, 1965; Heilman and Valenstein, 1972b; Stein and Volpe, 1983; Vande Linden et al, 1980; Watson and Heilman, 1979).

6.42 The Association of Auditory, Tactile and Body Neglect with Visual Neglect

While auditory and tactile extinction are usually considered to occur only with visual hemineglect (Heilman and Valenstein, 1972a, 1972b), in this study both were found to occur alone in a few patients. Even more interesting is the unimodal occurrence of body hemineglect in an LBD patient and its occurrence without visual
hemineglect in two further LBD patients and one RBD patient. Clinical observations give a strong impression that body hemineglect only occurs within the framework of a conspicuous hemineglect "syndrome" (De Renzi, 1982; Frederiks, 1969) and added to this it is considered to be much more frequent following right brain damage than following left brain damage (Cutting, 1978; Hecaen and Angelergues, 1963). In fact there have only been four case reports of right-handed patients with left-hemispheric lesions and anosognosia, which is a denial of hemiplegia rather than a strict body neglect (Nathanson, Bergman and Gordon, 1952; Von Hagen and Ives, 1937; Weinstein and Kahn, 1955; Welman, 1969), and no reports of hemiasomatognosia (body hemineglect) in patients with left hemispheric lesions. In this study however, I found one LBD patient with unimodal body hemineglect and a further five LBD patients who had body hemineglect as well as one or more other forms of neglect. In the RBD group I found eight patients with body hemineglect, all of whom had one or more other neglects.

Body hemineglect was nevertheless still found to be a fairly uncommon disorder relative to visual hemineglect. In this unilaterally brain-damaged population, 44% of LBD patients had visual hemineglect while only 14% had body hemineglect. In the RBD group 52.6% had visual hemineglect and 21% had body hemineglect. These proportions for visual and body hemineglect in RBD patients are nearly identical to those found by Cutting (1978) in his study of 48 unselected RBD patients examined, like mine, at an early
stage of the disease. In his sample, 50% had visual hemineglect and 21% had body hemineglect. This suggests that my criteria for body hemineglect were similar to those used by Cutting and therefore my figures for body hemineglect after left-hemispheric lesions should be equally valid.

6.43 Summary

Overall, this study, while pointing to some interesting data relating to the possibility of finding a unimodal or multimodal hemineglect disorder which does not include visual hemineglect, is inconclusive on the question of whether the neglects are independent of one another or derive from a common underlying deficit. What is clear is that spurious conclusions about the unitary nature of the neglect disorders can be arrived at on the basis of clinical observations of a few patients who have a multitude of neglects. Before such conclusions can be reached it is also necessary to examine the nature of hemineglect in patients who have a less conspicuous neglect disorder and are perhaps likely to be overlooked.

While the inconclusive findings are probably primarily due to the problems of definition and varying test sensitivities as outlined in the introduction, many further difficulties arise because of the idiosyncrasies of the patients. While this is a problem for most human neuropsychological studies presumably because every human brain is, at some level, unique, the problem is multiplied
when a number of different symptoms or behaviours are assessed and an attempt is made to correlate them. To conclude this discussion, therefore, I shall describe a case study which illustrates some of the difficulties which can arise in the assessment and interpretation of multimodal neglect.

6.44 An Illustrative Case Study

J.S. is a 23 year old man who suffered an embolic infarction of the middle cerebral artery territory of his right hemisphere. As a consequence he became mute and sustained a dense hemiplegia of his left arm and leg and a left homonymous hemianopia. Both the hemiplegia and hemianopia gradually resolved over the following four weeks although he was left with some residual weakness of his left leg and a more severe weakness of his left arm. His speech returned spontaneously eight days after his infarction occurred. Immediately following his embolic infarction he demonstrated a severe neglect of the left side of his body. For example, he would pull himself upright in his bed and swing himself around so he was sitting on the bed with his right leg over the side. However, he would invariably leave his left leg lying on the bed at right angles to his right leg, in spite of the fact that he was capable of pulling it around next to his right leg by using his right arm and hand, and had been shown how to do this. When asked why he did not do this, he answered, "I guess it looks pretty silly lying on the
bed by itself, but I'm not really interested in shifting it." He demonstrated tactile extinction on bilateral simultaneous stimulation to his cheeks, but his hands were not tested as he had some sensory loss in his left hand. On the "shadowing digits" test he demonstrated severe extinction of the digits delivered to his left ear (i.e. he had a laterality quotient of +67) although when asked to repeat groups of three digits delivered via headphones monaurally in a random order to each of his ears, he made no errors. In this condition he was not instructed to listen to any particular ear but just to repeat the digits he heard. That is, he had no way of knowing in which ear he would hear each digit. Paradoxically, he showed no signs of visual hemineglect on any of the five tests of the visual hemineglect battery, even in the first week after his stroke when his body hemineglect was very severe. Three weeks after his infarction he was able to do the computer-controlled imagery task described in Chapter 4 and although he made a number of errors on matching shapes on both the static and dynamic conditions, there was no unilateral bias in his error pattern.

One further experiment was carried out in order to see whether J.S.'s auditory extinction extended to digits presented diotically in a free auditory field rather than via headphones. The results of this experiment may provide a possible explanation for auditory extinction in some patients with severe body hemineglect. It was found that J.S. could perform the task of repeating digits delivered monaurally from each of two speakers placed at distances of
six feet on either side of his head. However, when digits were diotically presented in groups of three through the two speakers and the patient was asked to listen to one speaker only and repeat the digits after each group of three he always repeated the digits from the right speaker. He did this both when he was instructed to repeat the digits from the speaker on his right and when he was asked to repeat the digits from the speaker on his left. He also demonstrated contralesional auditory extinction for dichotically or diotically presented tones (see Chapter 5 for a description of the "shadowing tones" test) and digits when they were delivered via headphones or via speakers and a non-verbal finger-raising response was required (on hearing a particular tone or digit in a particular ear) rather than a verbal response.

Over a period of four weeks his hemiplegia became less dense and, possibly because of constant reminding, he gradually stopped neglecting the left side of his body. Simultaneously he began to report increasingly more digits and tones from the left speaker on three weekly repetitions of the "shadowing digits" and "shadowing tones" tests. By the time his body neglect had completely recovered, so had his auditory extinction. At this point he could move his left arm a little and walk on his left leg, although they were far from normal strength. It was almost as if this patient had been neglecting sounds in his contralesional hemispace because they were closest to his neglected body half, and in particular his left ear. That is, he may not have been demonstrating auditory hemineglect per se but
rather as a side effect of his body hemineglect. This idea finds support in the high tetrachoric correlation (+.81) between auditory extinction and body hemineglect in the group of LBD patients, especially as J.S., although having a right-hemispheric lesion, had speech on the right. As the tetrachoric correlation between auditory extinction and body hemineglect for the RBD group is not nearly so high (+.38) perhaps auditory extinction is a direct result of body hemineglect in patients with lesions of their speech dominant hemispheres only. In patients with lesions of the non-dominant hemisphere, auditory extinction may be independent of body hemineglect.

Here then is a case of an RBD patient who has speech mediated either by his right hemisphere or bilaterally, and who at no time appeared to have a visual hemineglect disorder yet demonstrated an unequivocal neglect of the left side of his body. He also exhibited a severe extinction of sounds in his left hemispace, but whether this was a true neglect of an auditory hemispace or whether it was simply an extension of his body hemineglect is not known. The finding that his body hemineglect resolved in parallel with his hemiplegia could mean either that the same cerebral location mediated both his hemiplegia and his hemineglect or that his body hemineglect was to some extent dependent upon his hemiplegia.
7. SUMMARY AND CONCLUSIONS

7.1 RELATIVE INCIDENCE AND SEVERITY OF HEMINEGLECT FOLLOWING LEFT- AND RIGHT-HEMISPHERIC LESIONS

7.2 NEGLECT VERSUS EXTINCTION

7.3 THE SPECIAL CASE OF IPSILATERAL EXTINCTION

7.4 IS HEMINEGLECT A UNITARY PHENOMENON?

7.5 GENERAL THEORIES OF NEGLECT
The four studies reported in this thesis (Chapters 3-6) each relate to different aspects of hemineglect and extinction. Each of the four studies has its own discussion and therefore will not be discussed again in detail here. Rather, in this final chapter I will give a brief summary of these studies and their results and speculate on how they might support or expand the more important theories which have been proposed to explain hemineglect.

7.1 RELATIVE INCIDENCE AND SEVERITY OF HEMINEGLECT FOLLOWING LEFT- AND RIGHT-HEMISPHERIC LESIONS

One of the primary aims of this thesis was to establish the incidence of hemineglect and the loci of lesions which are associated with hemineglect, in a large group of patients with unilateral left- and right-hemispheric lesions. The main impression gained from reading a general synopsis of the hemineglect disorders in a neurological or neuropsychological textbook is that it is a unilateral spatial disorder associated primarily with posterior parietal lesions of the right hemisphere.

As one delves more critically into the extensive literature on hemineglect and extinction it becomes increasingly clear that this general impression is by no means firmly rooted in fact. In particular the figures relating to the incidence of hemineglect following right-
and left-hemispheric lesions vary widely from study to study. While most studies have reported a much higher incidence of hemineglect following right-than following left-hemispheric lesions (e.g. Gloning et al, 1968; Hecaen and Angelergues, 1963; Weinstein and Cole, 1963) in other studies the differences between the two hemispheric groups is greatly diminished (Cohn, 1961; Zarit and Kahn, 1974), and in a study by Albert (1973), no difference in incidence was found between left and right brain-damaged patients.

There are also many exceptions to the common observation that hemineglect results from lesions to the posterior parietal cortex. An increasing number of studies are now being published that report human hemineglect following lesions to the frontal cortex (Damasio et al, 1980); Gloning, 1965; Heilman and Valenstein, 1972b), the cingulate gyrus (Heilman and Valenstein, 1972b), the basal ganglia (Damasio et al, 1980; Heir et al, 1977) and the thalamus (Watson and Heilman, 1978). Animal studies on extinction confute the issue further. Not only can extinction, and in rare cases, hemineglect be produced by ablating all of the areas equivalent to those which are associated with hemineglect in humans, but extinction also follows lesions to the superior colliculus and mesencephalic reticular formation in animals (Denny-Brown et al, 1952; Sprague and Meikle, 1965). What is more, extinction in animals and in humans follows lesions to the right and left hemispheres with equal frequency (Mountcastle et al, 1975; Weinstein and Friedland, 1977),
and this suggests that the deficit that underlies extinction may not be the same as that underlying hemineglect.

The vast bulk of the literature on human hemineglect relates to visual hemineglect. This may be a direct result of the fact that visual neglect is indeed a more frequent disorder than tactile, auditory or body neglect. Another possibility is that it is a result of the lack of sensitive tests developed for assessing auditory, tactile and body neglect as opposed to the many tests developed for testing visual hemineglect.

I attempted to improve on previous studies by designing a test battery for visual hemineglect which was simple enough for most patients to perform, thereby decreasing the numbers of patients which must be excluded because, for example, they were aphasic or hemiplegic. The exclusion of aphasics (with left-hemispheric lesions) from samples of patients assessed for hemineglect has been proposed by a number of researchers as a possible reason for the low incidence of hemineglect found in the left brain-damaged population (Albert, 1973; Battersby et al, 1956; Brain, 1945; De Renzi, 1982). I also attempted to assess tactile, auditory and body hemineglect or extinction in most of these patients.

Another possibly significant departure from previous studies was that my patient sample included a high proportion of patients with lesions of tumoural etiology, whereas the few previous large group studies in which the lesion etiology of the patient sample was made explicit
(e.g. Bisiach et al, 1979, 1981; Oxbury et al, 1974) tended to have a higher proportion of patients with lesions of vascular etiology. I tested the patients at the time of their hospitalization for diagnosis and treatment of their cerebral lesion, and therefore their lesions were usually of recent etiology and almost always "active" or unstable. As some patients recover either completely or partially from hemineglect in the first few weeks or months following the onset of the lesion (Campbell and Oxbury, 1976; Gainotti, 1968) the type of patient sample I assessed is more likely to exhibit a high incidence of hemineglect than a sample of patients with, say, long-term infarcts resulting from past vascular disease or trauma.

The study reported in Chapter 3 differed from most previous studies in that it demonstrated that visual hemineglect did follow right- and left-hemispheric lesions with an equal frequency in a large group of patients with active lesions. It did however concur with the frequent reports that visual hemineglect is more severe after right- than after left-hemispheric lesions (Albert, 1973; Denes et al, 1982; Gainotti et al, 1972). This difference in severity could provide one possible explanation as to why a patient sample with active lesions of recent etiology demonstrates an equal incidence of hemineglect following right- and left-hemispheric lesions, and a patient sample with more stable, longer-term lesions demonstrates a higher incidence of hemineglect following right- than left-hemispheric lesions. One could safely assume that the less severe the hemineglect is when it is first exhibited, the
sooner it will resolve completely. Consequently the longer the time period between the sustaining of the lesion and testing (with the possible exception of high grade malignant tumours) the less likely one will be to find hemineglect in left brain-damaged patients relative to right brain-damaged patients. Clearly the more sensitive the test is to hemineglect, the more likely one will be to pick up the deficit in left brain damaged patients who only have it in a mild form.

Another possible explanation for the change in incidence over time, could be that the right hemisphere is eventually able to "take over" the representation of the right side of space in the event of the left hemisphere being damaged. However, when the right hemisphere is damaged, the left hemisphere is not able to compensate and take over the representation of the left side of space. There is evidence from evoked potential and EEG studies that the right hemisphere mediates attention to both sides of space although the dominant tendency is toward the left side, whereas the left hemisphere mediates attention almost exclusively to the right side of space (Desmedt, 1977; Heilman and Van Den Abell, 1980).

A definitive study would be to test a group of unilaterally brain damaged patients in the early stages of their lesions and again some months later when their lesions were no longer in an "active" phase. If an equal incidence of hemineglect was found in the two hemispheric groups in the initial testing but at follow-up a higher incidence was found in the right brain-damaged group, this
would support the theory that left brain-damaged patients recover more quickly either because the right hemisphere gradually takes over its spatial functions, or because left-hemispheric hemineglect is less severe than right-hemispheric hemineglect in the first place, and recovery mediated by other mechanisms (e.g. resolution of oedema, compensation by other areas of the left hemisphere, formation of new neural pathways) is therefore more likely to occur. Such a finding would diminish the possibility that a low incidence of hemineglect in left brain-damaged patients is simply a consequence of the exclusion of aphasic patients.

I attempted a follow-up study 10 to 18 months after the initial hospitalization of 15 patients who at that time demonstrated severe visual hemineglect on one or more of the five tests described in Chapter 3. I was forced to abandon this follow-up because I was only able to retest two of the 15 patients. Eight of the group had since died (four with gliomas, one with a meningioma, one with a solitary metastasis, one with an embolic infarction and one with an intracerebral haemorrhage). Of the remaining five patients, three had deteriorated to the extent that they were now unable to do the tests, and two could not be located. One of the patients who was retested had had a large left frontal meningioma excised 15 months earlier and at that time had exhibited neglect on four of the visual hemineglect tests described in Chapter 3 (Tests 1, 2, 3 and 5). Fifteen months later she had made a complete recovery from all her neurological symptoms (headaches, drowsiness,
unsteady gait, mild expressive dysphasia and grand mal seizures) and demonstrated no signs of visual hemineglect at all. The second patient who was retested had a parieto-occipital glioma of left hemisphere resulting in grand mal seizures, unsteady gait and Wernicke's aphasia. His neurological condition had deteriorated over the 11 month period since he was first tested and the visual neglect which he had demonstrated on the "copying a star" test had neither resolved nor worsened.

One thing that can be concluded from this attempt at a follow-up is that severe hemineglect during the early stages of a cerebral lesion may indicate a poor prognosis. It would be interesting to conduct a follow-up study on patients who suffered a spontaneous intracerebral haemorrhage and as a result had unambiguous neurological deficits and exhibited visual hemineglect. According to Adams and Victor (1981) such patients do not usually recover from their neurological deficits, nor does bleeding recur from the same site. Therefore if their hemineglect resolved and their neurological deficits did not, this would suggest that hemineglect is not simply the result of damage to specific cerebral areas or pathways as are many neurological deficits, but is perhaps associated with the general disturbance and disorganization which occurs in a hemisphere when part of it is damaged. Once the lesion is stable (i.e. no longer changing) perhaps the undamaged parts of the hemisphere are able to resume optimal functioning and a general reorganization takes place around the lesioned area. That is, the ability to deal
successfully with each half of space might in part be a function of the overall stability of the contralateral hemisphere and not entirely due to specific cortical areas or neural pathways being damaged.

The most interesting result that emerged from the study described in Chapter 3 was that visual hemineglect was most likely to follow posterior lesions in right brain-damaged patients, but was most likely to follow anterior lesions in left brain-damaged patients. One possible reason for this is that as a consequence of language representation in the posterior parieto-temporal area of the left hemisphere, some aspects of spatial representation have become more anterior in that hemisphere. Why has this anterior-posterior interhemispheric difference in the loci of lesions producing visual hemineglect not been observed before? It could be that researchers have not looked for hemineglect in patients with left anterior lesions before, especially as it is commonly believed that posterior lesions cause hemineglect, whichever hemisphere is lesioned. Another possibility is that some patients with left anterior lesions have been excluded from previous assessments because they have expressive aphasia. I also found that patients with left-hemispheric lesions did not exhibit hemineglect on as many of the five visual neglect tests as did the patients with right-hemispheric lesions. This suggests that when only one test is used as has been the case in most previous studies, fewer left brain-damaged patients with hemineglect will be picked up.
7.2 NEGLECT VERSUS EXTINCTION

The problem of whether to treat hemineglect and extinction on double (bilateral) simultaneous stimulation as separate disorders or as forms of the same disorder remains unsolved. It is nevertheless a very important issue, especially as animal research on extinction is commonly used as a model for hemineglect in humans. One point in favour of hemineglect and extinction being treated as the same disorder is that hemineglect usually occurs in the context of bilateral stimulation even although hemineglect is defined as the neglect of a contralesional stimulus irrespective of the presence of a stimulus on the ipsilesional side. Therefore in most test situations it is not easy to define the point at which stimulation can be said to be present in one hemispace only, at least in the visual modality.

In the auditory modality, hemineglect has rarely, if ever been reported in the literature. In its place, patients are tested for auditory extinction (Heilman and Valenstein, 1972a; Heilman et al, 1974). It is difficult to know whether such a disorder as auditory hemineglect even exists as I know of no studies which have even attempted to test for it. Occasionally I have observed patients with severe visual hemineglect or hemiasomatagnosia who appear to ignore questions spoken by a person on their neglected side. However this could be a result of their visual hemineglect in that they are ignoring the person rather than the voice.
One practical consideration when testing for hemineglect and extinction is that a number of patients must be excluded from extinction tests because they have a unilateral sensory deficit. For this reason visual extinction was not included in the test battery designed to assess visual hemineglect. On the other hand the assessment of hemineglect need not be hampered by a sensory deficit and is therefore in some ways a more suitable test of unilateral spatial disorders.

The main "evidence" against hemineglect and extinction being different forms of the same disorder is the observation that extinction is a very "simple" disorder in that it does not appear to have associated with it any of the bizarre and often idiosyncratic behaviours that sometimes accompany severe cases of hemineglect. The complexity of hemineglect labels it as a higher cognitive disorder. It would be stretching the point to place a similar label on extinction.
7.3 THE SPECIAL CASE OF IPSILATERAL EXTINCTION

As pointed out in Chapter 3 six patients with left-hemispheric lesions demonstrated ipsilateral visual hemineglect on one of the tests in the test battery but five of these patients also demonstrated contralateral neglect on other tests in the test battery. Albert (1973) suggested that while patients with neglect most often neglect contralesional stimuli, they were liable to neglect stimuli anywhere in space.

The phenomenon of ipsilateral auditory extinction discussed in Chapter 5 may be an entirely different disorder from extinction on double simultaneous stimulation as it is usually defined. Because it occurred only in patients with lesions of the hemisphere dominant for speech and appears to be associated with verbal stimuli only, I did not include it in my assessment of multimodal hemineglect and extinction (Chapter 6).

Previous investigators have found ipsilateral auditory extinction following posterior left-hemispheric lesions (Damasio and Damasio, 1979; Damasio et al., 1976; Rubens et al., 1978; Sparks et al., 1970) and have postulated that it was the consequence of a disconnection between the left-ear verbal input and the language areas of the left hemisphere. This disconnection is attributed to a disruption of a posterior interhemispheric pathway connecting the two temporal lobes. However, my finding that patients with left frontal lesions also exhibit ipsilateral auditory extinction for verbal stimuli (but
not for non-verbal stimuli) causes problems for this disconnection hypothesis. An alternative hypothesis is that damage to the left hemisphere in any one of the areas mediating language renders it less able to decode or process the weaker verbal signal from the left ear.

Having come to the tentative conclusion that ipsilateral auditory extinction is not a "true" hemineglect or extinction disorder, one must nevertheless note the similarity in the data relating to the loci of lesions associated with visual hemineglect (Chapter 3) and ipsilateral auditory extinction (Chapter 5). In both cases the frontal lobe of the left-hemisphere is implicated in a spatial task. More extensive testing of spatial abilities and deficits of patients with left-hemispheric lesions may provide clues as to whether this is coincidence or is associated with some underlying spatial function which is mediated by the left frontal lobe.
7.4 IS HEMINEGLECT A UNITARY PHENOMENON?

The clinical impression that multimodal hemineglect is a single syndrome was not borne out by the data presented in Chapter 6. It is a common trap to conclude that because a constellation of signs and symptoms are often observed in the same patient, they are therefore the result of the same disease process or brain dysfunction (Walsh, 1978). As Kinsbourne (1971, p.291) wrote: "Pending validation by appropriate testing, the clinically observed 'syndrome' represents an educated guess at a relationship which has value in generating hypotheses and experimentation."

The incidence of multimodal hemineglect (e.g. hemineglect or extinction in more than one modality) was higher in the right brain-damaged group than in the left brain-damaged group. Of the patients with hemineglect in at least one modality, 58% of right brain-damaged patients and 38% of left brain-damaged patients had neglect in two or more modalities. However, no firm conclusions could be reached about the unitary nature of multimodal hemineglect because while the phi-correlation coefficients between the different forms of neglect were very small, some of the tetrachoric correlation coefficients which corrected for the different proportions of hemineglect in the different modalities, were very high. It was the extreme difference between these two forms of correlation coefficient which made it difficult to estimate the 'true' correlation. Certainly on the basis of these data one cannot make the
claim that the same spatial deficit underlies hemineglect in all modalities. Given that the only significant correlation was that between body hemineglect and tactile extinction in right brain-damaged patients, a more likely explanation is that multimodal hemineglect can be attributed to lesions overlapping different modality-specific areas.

However, it may be that the correlations were low not because of a difference in the deficits underlying the different modalities of hemineglect but rather because hemineglect and extinction are different disorders. If I had been able to assess auditory and tactile hemineglect instead of auditory and tactile extinction, perhaps higher phi-correlations would have been obtained across the modalities. Against this is the fact that the only significant correlation was between body hemineglect and tactile extinction.
7.5 GENERAL THEORIES OF NEGLECT

One of the most complete and convincing theories of hemineglect is that proposed by Heilman and Watson (1977a) and their colleagues (Heilman and Valenstein, 1972b; Watson et al, 1973, 1974).

Their theory depends upon the existence of a corticolimbic-reticular loop which connects the various cerebral areas associated with hemineglect and extinction in humans and animals. The function of this loop is to maintain general arousal levels and to activate an orienting response to meaningful stimuli. When the loop is disconnected by a lesion, the organism becomes generally hypoaroused and the ability to orient to stimuli in the contralesional hemispace is disrupted. Hemineglect or extinction results.

One problem not entirely overcome by this hypothesis is the apparently unequal incidence of hemineglect following right- and left-hemispheric lesions in humans. One way of explaining this is that the right hemisphere, which is more specialized for visuospatial functions than the left, is able to mediate attention to both sides of space whereas the left hemisphere is equipped to attend only to the right side of space. However, the finding reported in Chapter 3 that visual hemineglect follows right- and left-hemispheric lesions with an equal frequency, provides a better fit to Heilman and Watson's (1977a) hypothesis. The greater specialization of the right hemisphere for visuospatial functions and its
particular ability to attend to both sides of space can then be called upon to explain the less extreme differences in hemineglect between the right and left hemispheres; i.e. the fact that left-hemispheric neglect is less severe and possibly recovers more rapidly than right-hemispheric neglect.

The hypothesis proposed by De Renzi et al., (1970) and Bisiach et al., (1979, 1981) that hemineglect is a "mutilated representation of space" is supported by their experiments which demonstrated that patients with left-sided hemineglect and right-hemispheric lesions neglected the left sides of their internal representations of the external world (Bisiach and Luzzatti, 1978; Bisiach et al., 1979, 1981). The experiment reported in Chapter 4 extended that to include patients with left-hemispheric lesions. These patients were also found to neglect the contralesional (right sides) of their images. The implication of this is that at some advanced level of processing, visual images are mapped across the two hemispheres in an analogue fashion, at least with respect to their left and right sides. Bisiach et al., (1981) likened the posterior parietal cortices to a 'scratch pad' (page 549), and developed this metaphor by suggesting that if one half of the scratch pad is damaged, the part of the spatial image mapped onto it will be mutilated.

One departure I made from Bisiach et al's (1979) study was that not all of the unilaterally lesioned patients I tested for the neglect of an image (Chapter 4) displayed visual hemineglect on the visual hemineglect battery described in Chapter 3. Yet, in spite of this,
they still neglected the contralesional sides of images. While this suggests that the 'visual image' test is more sensitive to hemineglect than more traditional paper and pencil tests, it also raises the possibility that all patients with unilateral lesions, with the possible exception of temporal lobe lesions, have some degree of hemineglect, at least in the early stages of their lesion, and it is a function of the insensitivity of the tests used to assess it that results in it being missed in some patients. It is interesting to note that the left brain-damaged patient who displayed the most severe contralesional neglect of images had an anterior lesion, while the right brain-damaged patient with the most severe hemineglect of images had a posterior lesion. This suggests that the 'scratch pad' might extend more anteriorly in the left hemisphere.

It could be that hemineglect is in part a consequence of the general disorganization of the hemisphere immediately following a lesion to one part of it. This would fit with the high incidence of hemineglect I found in both right and left brain-damaged patient groups when they were assessed in the early stages of cerebral damage. It would also tie in with the fact that even patients who do not display neglect on traditional tests do so on tests which require a more complex level of cognitive processing. Bisiach et al's (1981) notion of a scratch pad can be taken further to incorporate the hemisphere as a whole rather than just the parietal cortex. This is not to say that the scratch pad itself extends over the entire cortex but
rather than in order for it to be fully functional it requires that the rest of the hemisphere be undisturbed. It does not seem too far fetched to suppose that a clear, unbroken scratch pad relies upon a disciplined, stable hemisphere.

These three hypotheses do not need to be mutually exclusive. Hemineglect is very likely to be the result of a number of different deficits at different levels of perceptual and cognitive processing (see also Mesulam, 1981, 1983). A unilateral decrease in arousal and attention as proposed by Heilman and Watson (1979) might be one component. A general (and often transient) disorganization of one hemisphere may be a second component. A third component might relate to the specific cerebral area damaged, and this could perhaps explain the fact that hemineglect in different modalities does not always coincide. When a particular area is damaged this could result in an inability to carry out one of the processes necessary for the cognition of space. For example, damage to the frontal eye fields may result in a unilateral scanning deficit (Mohler et al, 1973; Wurtz and Mohler, 1976b) and damage to the posterior parietal cortex might result in an inability to integrate spatial information from different modalities, a mutilation of visual images because of a disrupted 'scratch pad' (Bisiach et al, 1981) and/or a disruption of the neurons that are specialized for encoding the psychological impact of sensory events occurring in the contralateral hemispace (Lynch, 1980; Mountcaste et al, 1975, Robinson et al,
1978). This bewildering variety of functions possibly mediated by the parietal lobe may explain why lesions to the inferior parietal cortex are so often associated with hemineglect. To encompass the finding that anterior lesions of the left hemisphere result in a higher incidence of visual hemineglect than do anterior lesions of the right hemisphere, it must be supposed that one or more of the visuospatial functions (the 'scratch pad'?%) mediated by the right parietal cortex is mediated more anteriorly in the left hemisphere as a result of the specialization of its posterior cortex for language.


Bradshaw, J.L., Nettleton, N.C., Nathan, G., and Wilson, L. (In press, b). Head and body space to left and right front and rear - II Visuotactual and kinesthetic studies and left-side underestimation. Neuropsychologia.


Pierson, J.M., Bradshaw, J.L., and Nettleton, N. (In press). Head and body space to left and right, front and rear - I. Unidirectional competitive auditory stimulation. Neuropsychologia.


Sparks, R., Geschwind, N. (1968). Dichotic listening in man after section of neocortical commissures. Cortex, 4, 3-16.


## APPENDIX

<table>
<thead>
<tr>
<th>Subject Information Forms</th>
<th>Number of Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>1</td>
</tr>
<tr>
<td>Neuropsychological</td>
<td>2</td>
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<table>
<thead>
<tr>
<th>Visual Hemineglect Forms</th>
<th>Number of Pages</th>
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<tbody>
<tr>
<td>Paper and pencil tests</td>
<td>3</td>
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<tr>
<td>Summary</td>
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<tr>
<th>Auditory Extinction Forms</th>
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<tr>
<td>Dichotic digits/Shadowing digits data</td>
<td>3</td>
</tr>
<tr>
<td>Summary</td>
<td>1</td>
</tr>
<tr>
<td>Shadowing tones data</td>
<td>2</td>
</tr>
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<td>Shadowing tones script</td>
<td>4</td>
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<table>
<thead>
<tr>
<th>Tactile Extinction Form</th>
<th>Number of Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Summary</td>
<td>1</td>
</tr>
</tbody>
</table>
SUBJECT INFORMATION - GENERAL

NEUROLOGICAL PATIENTS

SUBJECT NUMBER: ________  CODE NUMBER: ________

DATE FIRST TESTED: ______________

SEX: ________  AGE: ________  BIRTHDATE: ________

EDUCATIONAL LEVEL:

PRESENT OCCUPATION:

PAST OCCUPATIONS:

I.Q. PREMORBID ESTIMATE:

PRESENT:

HANDEDNESS:

DOCTOR/SURGEON:

WARD:

DATE OF RELEASE:

HOME CONTACT:
SUBJECT INFORMATION - NEUROPSYCHOLOGICAL 1.

SUBJECT NUMBER:_________  CODE NUMBER:_________

EXPERIMENTAL GROUP:

DATE OF ADMISSION:

PREVIOUS ADMISSIONS FOR NEUROLOGICAL DISORDERS:

LESION:

ETIOLOGY:
SUBJECT INFORMATION - NEUROPSYCHOLOGICAL 2

SUBJECT NUMBER:__________  CODE NUMBER:__________

MEDICAL PROCEDURES/DRUGS:

SURGICAL PROCEDURES:

NEUROLOGICAL SIGNS:
Sight:
Hearing:
Kinesthetic sense:
Tactile sense:
Paralysis:
Aphasia:
Other:

BEHAVIOURAL OBSERVATIONS/PSYCHIATRIC SIGNS:
TEST 2

DRAW IN THE NUMBERS ON THE CLOCK FACE

TEST 3
COPY THIS CUBE IN THE SPACE BELOW

TEST 4
COPY THIS STAR IN THE SPACE BELOW
COPY THIS DRAWING IN THE SPACE BELOW
SUBJECT INFORMATION

VISUAL HEMINEGLECT

SUBJECT NUMBER__________ CODE NUMBER__________

DATE:__________

VISUAL FIELD DEFECTS:

EXTINCTION WITH SIMULTANEOUS BILATERAL STIMULATION:

CLOCK:

CUBE:

STAR:

HOUSE/FENCE/TREES:

XXXXXXXX:

CROSS OUT LINES:

READ ONE SIDE OF WORD:

READ ONE SIDE OF PARAGRAPH:

DRESS ONE SIDE:

EAT FROM ONE SIDE OF PLATE:

COLLIDE WITH OBJECTS ON ONE SIDE:

IGNORE PEOPLE ON ONE SIDE:

ESTIMATED DEGREE OF SEVERITY OF VISUAL HEMINEGLECT:
(SEVERE/MODERATE/MILD)
<table>
<thead>
<tr>
<th>Correct Response</th>
<th>Subject Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>R 246</td>
<td></td>
</tr>
<tr>
<td>L 641</td>
<td></td>
</tr>
<tr>
<td>921</td>
<td></td>
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<tr>
<td>729</td>
<td></td>
</tr>
<tr>
<td>852</td>
<td></td>
</tr>
<tr>
<td>642</td>
<td></td>
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<td>64</td>
<td></td>
</tr>
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<td>274</td>
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<td></td>
</tr>
<tr>
<td>358</td>
<td></td>
</tr>
<tr>
<td>614</td>
<td></td>
</tr>
<tr>
<td>853</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Series Two. Point to location in space.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digits</td>
</tr>
<tr>
<td>--------</td>
</tr>
<tr>
<td>721</td>
</tr>
<tr>
<td>612</td>
</tr>
<tr>
<td>944</td>
</tr>
<tr>
<td>762</td>
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<tr>
<td>819</td>
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<tr>
<td>524</td>
</tr>
<tr>
<td>917</td>
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<td>583</td>
</tr>
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<td>869</td>
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<tr>
<td>357</td>
</tr>
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<td>175</td>
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<tr>
<td>368</td>
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<tr>
<td>672</td>
</tr>
<tr>
<td>918</td>
</tr>
<tr>
<td>354</td>
</tr>
</tbody>
</table>

Subject No. 001: Series One. Repeat all digits. 028: Series Two.
Subject No. _____ SHADOWING DIGITS  (Headphones correct)

Repeat digits in **RIGHT EAR**

<table>
<thead>
<tr>
<th>RIGHT</th>
<th>LEFT</th>
<th>SUBJECT'S RESPONSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>745</td>
<td>291</td>
<td>SHADOWING</td>
</tr>
<tr>
<td>389</td>
<td>462</td>
<td>DICHOTIC</td>
</tr>
<tr>
<td>485</td>
<td>627</td>
<td></td>
</tr>
<tr>
<td>916</td>
<td>725</td>
<td></td>
</tr>
<tr>
<td>342</td>
<td>965</td>
<td></td>
</tr>
<tr>
<td>216</td>
<td>734</td>
<td></td>
</tr>
<tr>
<td>983</td>
<td>657</td>
<td></td>
</tr>
<tr>
<td>512</td>
<td>837</td>
<td></td>
</tr>
<tr>
<td>147</td>
<td>238</td>
<td></td>
</tr>
<tr>
<td>462</td>
<td>538</td>
<td></td>
</tr>
</tbody>
</table>

Repeat digits in **LEFT EAR**

<table>
<thead>
<tr>
<th>RIGHT</th>
<th>LEFT</th>
<th>SHADOWING</th>
<th>DICHOTIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>657</td>
<td>983</td>
<td></td>
<td></td>
</tr>
<tr>
<td>291</td>
<td>745</td>
<td></td>
<td></td>
</tr>
<tr>
<td>627</td>
<td>485</td>
<td></td>
<td></td>
</tr>
<tr>
<td>837</td>
<td>512</td>
<td></td>
<td></td>
</tr>
<tr>
<td>538</td>
<td>462</td>
<td></td>
<td></td>
</tr>
<tr>
<td>725</td>
<td>916</td>
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<td>734</td>
<td>216</td>
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<td>965</td>
<td>342</td>
<td></td>
<td></td>
</tr>
<tr>
<td>238</td>
<td>147</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\[
L.Q.\text{ (Shadowing Digits)} = \]

\[
L.Q.\text{ (Dichotic Digits)} = \]
Subject No. ______ SHADOWING DIGITS (Headphones reversed)

Repeat digits in LEFT EAR

<table>
<thead>
<tr>
<th>LEFT</th>
<th>RIGHT</th>
<th>SUBJECT'S RESPONSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>745</td>
<td>291</td>
<td>SHADOWING</td>
</tr>
<tr>
<td>389</td>
<td>462</td>
<td></td>
</tr>
<tr>
<td>485</td>
<td>627</td>
<td></td>
</tr>
<tr>
<td>916</td>
<td>725</td>
<td></td>
</tr>
<tr>
<td>342</td>
<td>965</td>
<td></td>
</tr>
<tr>
<td>216</td>
<td>734</td>
<td></td>
</tr>
<tr>
<td>983</td>
<td>657</td>
<td></td>
</tr>
<tr>
<td>512</td>
<td>837</td>
<td></td>
</tr>
<tr>
<td>147</td>
<td>238</td>
<td></td>
</tr>
<tr>
<td>462</td>
<td>538</td>
<td></td>
</tr>
</tbody>
</table>

Repeat digits in RIGHT EAR

<table>
<thead>
<tr>
<th>LEFT</th>
<th>RIGHT</th>
<th>SHADOWING</th>
<th>DICHOTIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>657</td>
<td>983</td>
<td></td>
<td></td>
</tr>
<tr>
<td>291</td>
<td>745</td>
<td></td>
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</tr>
<tr>
<td>627</td>
<td>485</td>
<td></td>
<td></td>
</tr>
<tr>
<td>837</td>
<td>512</td>
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<td></td>
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<tr>
<td>538</td>
<td>462</td>
<td></td>
<td></td>
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<tr>
<td>725</td>
<td>916</td>
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<td>734</td>
<td>216</td>
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<td></td>
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<tr>
<td>462</td>
<td>389</td>
<td></td>
<td></td>
</tr>
<tr>
<td>965</td>
<td>342</td>
<td></td>
<td></td>
</tr>
<tr>
<td>238</td>
<td>147</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

L.Q. (Shadowing Digits)

L.Q. (Dichotic Digits)
SUBJECT INFORMATION

AUDITORY EXTINCTION

SUBJECT NUMBER___________ CODE NUMBER___________

DATE:_______________

EXTINCTION WITH SIMULTANEOUS BILATERAL STIMULATION:

IGNORES SOUND/SPEECH FROM ONE SIDE:

SERIES ONE:
Digits in right ear:

Digits in left ear:

SERIES TWO:
Digits in right ear:

Digits in left ear:

Digits in both ears:

SHADOWING DIGITS
Repeat digits in right ear:

Repeat digits in left ear: L.Q. =

DICHOTIC DIGITS
Right ear

Left ear L.Q. =
<table>
<thead>
<tr>
<th>Series 1. Raise finger on R.</th>
<th>Series 2. Dichotic Raise finger on R.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stimulus</td>
<td>Rgt</td>
</tr>
<tr>
<td>1</td>
<td>G</td>
</tr>
<tr>
<td>2</td>
<td>R</td>
</tr>
<tr>
<td>3</td>
<td>P</td>
</tr>
<tr>
<td>4</td>
<td>G</td>
</tr>
<tr>
<td>5</td>
<td>O</td>
</tr>
<tr>
<td>6</td>
<td>R</td>
</tr>
<tr>
<td>7</td>
<td>G</td>
</tr>
<tr>
<td>8</td>
<td>R</td>
</tr>
<tr>
<td>9</td>
<td>O</td>
</tr>
<tr>
<td>10</td>
<td>O</td>
</tr>
<tr>
<td>11</td>
<td>R</td>
</tr>
<tr>
<td>12</td>
<td>P</td>
</tr>
</tbody>
</table>

Correct =

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Stimulus</td>
<td>Rgt</td>
</tr>
<tr>
<td>1</td>
<td>G</td>
</tr>
<tr>
<td>2</td>
<td>P</td>
</tr>
<tr>
<td>3</td>
<td>R</td>
</tr>
<tr>
<td>4</td>
<td>O</td>
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<tr>
<td>5</td>
<td>R</td>
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<td>6</td>
<td>O</td>
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<td>7</td>
<td>P</td>
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<td>8</td>
<td>R</td>
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<td>11</td>
<td>P</td>
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<tr>
<td>25</td>
<td>O</td>
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<td>26</td>
<td>G</td>
</tr>
<tr>
<td>27</td>
<td>R</td>
</tr>
<tr>
<td>28</td>
<td>P</td>
</tr>
</tbody>
</table>

Correct = Incorrect =

Laterality =

Series 3/4 =
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Stimulus</td>
<td>Response</td>
</tr>
<tr>
<td>Rgt</td>
<td>Lft</td>
</tr>
<tr>
<td>1</td>
<td>G</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>P</td>
</tr>
<tr>
<td>4</td>
<td>G</td>
</tr>
<tr>
<td>5</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>R</td>
</tr>
<tr>
<td>7</td>
<td>R</td>
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<tr>
<td>8</td>
<td>-</td>
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<tr>
<td>9</td>
<td>R</td>
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<tr>
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<td>-</td>
</tr>
<tr>
<td>11</td>
<td>-</td>
</tr>
<tr>
<td>12</td>
<td>P</td>
</tr>
</tbody>
</table>

Correct= 18

<table>
<thead>
<tr>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Stimulus</td>
<td>Response</td>
</tr>
<tr>
<td>Rgt</td>
<td>Lft</td>
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<tr>
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<td>R</td>
</tr>
<tr>
<td>2</td>
<td>O</td>
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<td>3</td>
<td>P</td>
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<td>R</td>
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<td>7</td>
<td>G</td>
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<td>8</td>
<td>R</td>
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<td>9</td>
<td>P</td>
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<tr>
<td>10</td>
<td>O</td>
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<td>12</td>
<td>P</td>
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<td>13</td>
<td>R</td>
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<tr>
<td>14</td>
<td>O</td>
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<tr>
<td>15</td>
<td>O</td>
</tr>
<tr>
<td>16</td>
<td>P</td>
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<td>17</td>
<td>R</td>
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<td>18</td>
<td>G</td>
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<td>P</td>
</tr>
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<td>20</td>
<td>R</td>
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<td>21</td>
<td>R</td>
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<td>22</td>
<td>O</td>
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<td>23</td>
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<td>R</td>
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<tr>
<td>25</td>
<td>P</td>
</tr>
<tr>
<td>26</td>
<td>R</td>
</tr>
<tr>
<td>27</td>
<td>O</td>
</tr>
<tr>
<td>28</td>
<td>R</td>
</tr>
</tbody>
</table>

Correct= 27

Incorrect= 11

Laterality = left ear

Series 3/4
Script for "Shadowing Tones" Tape. Stereo as indicated.

**BOTH**

"Listen to the following sounds. Each is made by a different musical instrument"

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIANO 1 second</td>
<td>(timer cue 6 on master)</td>
</tr>
<tr>
<td>ORGAN 1 second</td>
<td>(timer cue 10 on master)</td>
</tr>
<tr>
<td>GUITAR 1 second</td>
<td>(timer cue 12 on master)</td>
</tr>
<tr>
<td>RECORDER 1 second</td>
<td>(timer cue 14 on master)</td>
</tr>
</tbody>
</table>

**NOTE** the last four entries are repeated many times hereafter and are scripted thus... PIANO P ORGAN O GUITAR G RECORDER R

"Now you are going to hear a series of these sounds. Sometimes you will hear a sound in your right ear, sometimes in your left ear and sometimes in both ears. Every time you hear this sound R

I want you to raise your finger. Remember, listen to all the sounds and raise your finger **only** when you hear this R

Do you understand what to do? **Start**"

**NOTE** here follows first block of trials. The duration of the stimulus is to be 1 second and the inter-stimulus interval is 2 seconds.

<table>
<thead>
<tr>
<th>RIGHT EAR</th>
<th>LEFT EAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>G</td>
<td>-</td>
</tr>
<tr>
<td>-</td>
<td>R</td>
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**BOTH**

"Good. Now for a more difficult task. This time you will hear the sounds two at a time, one in each ear. They will sound something like this......."
Shadowing Tones  Page 2

R       O
P       G

BOTH

Once again, every time you hear this sound
R       R

I want you to raise your finger. Do you understand what to do? "
2 second pause........"Start"

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"Now you will hear a similar series of sounds, two at a time,
one in each ear. This time, however, I want you to listen to
one ear only"
2 second pause........"and to raise your finger only when you
hear this sound"

RIGHT EAR
R

"in this ear" (Touch patient's ear.)

BOTH

"Do you understand what to do?" 2 second pause........
"Start"
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Now listen only to the sounds in the other ear PAUSE FOR 2 SECONDS .......and raise your finger when you hear this sound

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in that ear.

BOTH

Start.
SUBJECT INFORMATION

TACTILE EXTINCTION

SUBJECT NUMBER _______ CODE NUMBER _______

TACTILE SENSE: DATE: _______

RIGHT SIDE: _______

LEFT SIDE: _______

EXTINCTION WITH SIMULTANEOUS BILATERAL STIMULATION:

(a) BILATERAL STIMULATION TO SAME PARTS OF BODY:

(b) BILATERAL STIMULATION TO DIFFERENT PARTS OF BODY:

ABILITY TO IDENTIFY BY HANDLING:

(a) Safety Pin.
   R.H. L.H.

(b) Ball Point Pen.
   R.H. L.H.

(c) Teaspoon.
   R.H. L.H.

(d) Pencil Eraser.
   R.H. L.H.