



<http://researchspace.auckland.ac.nz>

ResearchSpace@Auckland

Copyright Statement

The digital copy of this thesis is protected by the Copyright Act 1994 (New Zealand).

This thesis may be consulted by you, provided you comply with the provisions of the Act and the following conditions of use:

- Any use you make of these documents or images must be for research or private study purposes only, and you may not make them available to any other person.
- Authors control the copyright of their thesis. You will recognise the author's right to be identified as the author of this thesis, and due acknowledgement will be made to the author where appropriate.
- You will obtain the author's permission before publishing any material from their thesis.

To request permissions please use the Feedback form on our webpage.

<http://researchspace.auckland.ac.nz/feedback>

General copyright and disclaimer

In addition to the above conditions, authors give their consent for the digital copy of their work to be used subject to the conditions specified on the Library Thesis Consent Form.

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

A C K N O W L E D G E M E N T S

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.

Many of the staff and graduate students of the Department of Psychology, Auckland University, assisted me. Mr Rhys Owen wrote the computer programs used in the experiment described in Chapter 4 and Miss Rachel McLaren helped in the collection of control data for that experiment. Mr Frank Bielby made various pieces of equipment for me, always beautifully constructed and finished. The electronic workshop technicians serviced equipment for me, Mr Tom Attwood wrote many order forms and often made helpful suggestions about how best to get things done in a hurry, and Ms Alison Thompson, Mrs Lydia Brooks and Ms Philippa Brooks willingly typed various letters, data forms and articles for me. I also thank Mrs Gail Korving of Crisis Secretarial Services for the care and efficiency with which she put my thesis onto the word processor. Mr Gregory O'Leary and Mr Bill Cole made a professional job of the dichotic tapes used in the experiments described in Chapter 5. Mr Bill Cole also knew just the right Shakespearean quote for the thesis. The assistance given to me by visitors to the Auckland University Open Day who acted as control subjects for the experiments reported in Chapter 4 and 5 is appreciated.

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.

A very important element in the completion of this thesis was the practical and emotional support provided by my husband, John, and my children, Caroline, Jonathan, Josie and Joachim. My very special thanks go to them for their patience and understanding, especially during those times when I was feeling tired and disheartened after spending time with patients who were very ill, and in the last few months when I was writing my "mixed-up chapter book" as the thesis was dubbed by Joachim.

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.

A B S T R A C T

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.

T A B L E O F C O N T E N T S

	Page
1. GENERAL INTRODUCTION	
1.1 ORIENTATION AND HISTORICAL BACKGROUND	3
1.2 DEFINITIONS AND PHENOMENOLOGY OF HEMINEGLECT AND UNILATERAL EXTINCTION	7
1.21 Visual Hemineglect	8
1.22 Auditory Hemineglect	10
1.23 Tactile Hemineglect	10
1.24 Hemiasomatagnosia	11
1.25 Emotional and Behavioural Phenomena Associated with Hemineglect	12
1.26 Unilateral Extinction on Double Simultaneous Stimulation	15
1.3 NEUROPATHOLOGY	21
1.31 The Evidence for a Right-Left Hemispheric Difference in Incidence of Hemineglect	21
1.32 Locus of Lesion Producing Hemineglect and Extinction	24
1.4 EXPERIMENTAL EVIDENCE FROM HUMAN STUDIES	28
1.41 Descriptive Studies	28
1.42 Experimental Studies on Hemineglect Patients	30
1.43 Experimental Studies on Normal Subjects and Patients Without Neglect	33
1.5 EXPERIMENTAL EVIDENCE FROM ANIMAL STUDIES	36
1.6 THEORIES OF HEMINEGLECT AND EXTINCTION	41
1.61 Sensory and Perceptual Hypotheses	41
1.62 Interhemispheric Hypotheses	42
1.63 Hypotheses of Defective Attentional Activation	45
1.64 Hemineglect; A Mutilated Representation of Space?	47
1.7 ORGANIZATION OF THE THESIS	50
2. GENERAL METHODOLOGY	
2.1 THE NEUROPSYCHOLOGICAL INVESTIGATION	54

	Page
2.2 MAXIMIZING PERFORMANCE LEVELS ON NEUROPSYCHOLOGICAL TESTS	57
2.21 Test Anxiety and Emotional Factors Related to Illness and Hospitalization	58
2.22 Fatigue, Distractibility, Depression, Frustration and Decreased Attention Span	60
2.3 THE EFFECTS OF DRUG THERAPY ON TEST PERFORMANCE	63
2.4 THE PATIENT SAMPLE	65
2.5 THE LESIONS	68
2.51 The Significance of Active Lesions of Recent Etiology for the Study of Hemineglect	68
2.52 The Diagnosis and Measurement of Lesions	69
2.53 General Descriptions of the Types of Lesions Sustained by the Patients Included in These Studies	70
(a) Neoplasms and Cerebral Abscesses	71
(b) Lesions Resulting From Cerebrovascular Diseases and Head Injuries	73
2.6 NON-NEUROLOGICAL CONTROL SUBJECTS	78
2.7 THE TESTS	79
3. ANTERO-POSTERIOR INTERHEMISPHERIC DIFFERENCES IN THE LOCI OF LESIONS PRODUCING VISUAL HEMINEGLECT	
3.1 INTRODUCTION	83
3.2 METHOD	86
3.21 Subjects and Categorization of Lesions	86
3.22 Neurological Examination	89
3.23 Neuropsychological Examination	89
3.3 RESULTS	95
3.31 Incidence of Hemineglect	95
3.32 Age and Sex of Patients with Visual Hemineglect	97
3.33 Visual Field Defects (V.F.D.)	97
3.34 Type and Size of Lesion and Hemineglect	98
3.35 Locus of Lesion and Hemineglect	99
3.36 Severity of Hemineglect	100

	Page
3.37 Extinction on Double Visual Simultaneous Stimulation	105
3.38 Ipsilateral Neglect	106
3.4 DISCUSSION	108
3.41 Incidence of Visual Hemineglect	108
3.42 Anterior-Posterior Interhemispheric Differences in Incidence	109
3.43 Severity of Hemineglect	110
3.44 Ipsilateral Neglect	111
4. THE UNILATERAL NEGLECT OF AN IMAGE	
4.1 INTRODUCTION	115
4.2 METHOD	119
4.21 Subjects	119
4.22 Procedure	120
4.3 RESULTS	129
4.31 'Static' Condition	129
4.32 'Dynamic' Condition	129
4.4 DISCUSSION	131
4.41 Hemineglect of Images in Brain-Damaged Patients	131
4.42 Implications for Normal Imagery Processes	133
5 IPSILATERAL AUDITORY EXTINCTION	
5.1 GENERAL INTRODUCTION	138
EXPERIMENT 1 - Auditory Extinction on a Dichotic Digits Task	143
5.2 METHOD	143
5.21 Subjects	143
5.22 Location and Size of Cerebral Lesions	144
5.23 Assessment of Visual Field Defects, Visual Hemineglect and Tactile Extinction	145
5.24 Test Procedure	146
5.3 RESULTS AND DISCUSSION	150
5.31 Comparison Between the S.D. and D.D. Tests	150

	Page
5.32 Results of Patients with Auditory Extinction on 'Shadowing Digits'	151
5.33 Case Studies of Patients with Ipsilateral Auditory Extinction on 'Shadowing Digits'	154
5.34 Results of Patients Who Did Not Demonstrate Auditory Extinction on 'Shadowing Digits'	168
5.35 Ear Extinction and Size of Lesion	168
5.36 Ear Extinction and Spatial Hemineglect	169
5.37 Dichotic and Diotic Listening	170
5.4 DISCUSSION	172
EXPERIMENT 2 - Further Studies of Auditory Extinction Using a Non-Verbal Dichotic Task	177
5.5 INTRODUCTION	177
5.6 METHOD	179
5.61 Subjects	179
5.62 Test Procedure	180
5.7 RESULTS AND DISCUSSION	182
5.71 Control Group	182
5.72 Experimental Groups	182
5.8 DISCUSSION	184
5.9 GENERAL CONCLUSIONS	186
6. MULTIMODAL HEMINEGLECT	
6.1 INTRODUCTION	189
6.11 Overview	189
6.12 Hemineglect and Extinction	192
6.13 Ipsilateral Hemineglect and Extinction	196
6.2 METHOD	197
6.21 Subjects	197
6.22 Test Procedure	198
6.3 RESULTS	202
6.31 Incidence, Sex and Age	202
6.32 Locus and Side of Lesion	203
6.33 Correlations between the Different Modalities of Neglect	206

	Page
6.4 DISCUSSION	209
6.41 A Single, Underlying Cause for Multimodal Neglects?	209
6.42 The Association of Auditory, Tactile and Body Neglect with Visual Neglect	212
6.43 Summary	214
6.44 An Illustrative Case Study	215
7. SUMMARY AND CONCLUSIONS	
7.1 RELATIVE INCIDENCE AND SEVERITY OF HEMINEGLECT FOLLOWING LEFT- AND RIGHT-HEMISPHERIC LESIONS	220
7.2 NEGLECT VERSUS EXTINCTION	228
7.3 THE SPECIAL CASE OF IPSILATERAL EXTINCTION	230
7.4 IS HEMINEGLECT A UNITARY PHENOMENON?	232
7.5 GENERAL THEORIES OF NEGLECT	234
REFERENCES	239
APPENDIX	257

L I S T O F T A B L E S

	Page
CHAPTER 3	
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.	88
3-2 Comparisons between LBD and RBD groups on the incidence of visual hemineglect for each neuropsychological test.	96
3-3 The presence and absence of VFDs in RBD and LBD patients with and without visual hemineglect.	98
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.	99
3-5 Numbers of patients* in the different lesion locality groups with and without visual hemineglect.	101
3-6 Comparisons between LBD and RBD hemineglect groups on the severity of hemineglect.	104
3-7 Numbers of patients with and without visual hemineglect and visual extinction and patients not able to be assessed for extinction.	106
CHAPTER 4	
4-1 Neurological Characteristics and Laterality Quotients of Brain-Damaged Patients.	127
4-2 Laterality Quotients of Control Subjects.	128
CHAPTER 5	
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.	151
5-2 S.D. and D.D. laterality quotients and nature and location of lesion for patients with auditory extinction in Experiment 1.	153
5-3 S.D. and D.D. laterality quotients and nature and location of lesion for LBD patients without auditory extinction in Experiment 1.	166

CHAPTER 5 (cont'd)

- 5-4 S.D. and D.D. laterality quotients and nature and location of lesion for RBD patients without auditory extinction in Experiment 1. 167
- 5-5 Laterality quotients for the earphone and speaker versions of the 'Shadowing digits' task for four patients with left-hemispheric lesions. 171
- 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. 183

CHAPTER 6

- 6-1 Patients with right-hemispheric lesions and hemineglect and extinction in different modalities. 204
- 6-2 Patients with left-hemispheric lesions and hemineglect and extinction in different modalities. 205
- 6-3 Phi correlation coefficients between neglects in different modalities for the RBD and LBD groups. 208
- 6-4 Tetrachoric correlation coefficients between neglects in different modalities for the RBD and LBD groups. 208

L I S T O F F I G U R E S

	Page
CHAPTER 3	
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.	93
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.	94
CHAPTER 4	
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.	122
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.	123
4-3 Computer printouts of responses for the 'Static' condition of the Imagery experiment.	126
4-4 Computer printouts of responses for the 'Dynamic' condition of the Imagery experiment.	126
CHAPTER 5	
5-1 CASE 8 - A glioma of the left 'posterior paraventricular' region that resulted in ipsilateral auditory extinction.	158
5-2 CASE 14 - A glioma of the right 'posterior paraventricular' region that resulted in left ear auditory extinction.	158

CHAPTER 5 (cont'd)

- | | | |
|-----|---|-----|
| 5-3 | CASE 1 - A glioma of the left anterior basal ganglia that resulted in ipsilateral auditory extinction. | 159 |
| 5-4 | CASE 2 - A meningioma of the left frontal lobe that resulted in ipsilateral auditory extinction. | 160 |
| 5-5 | CASE 3 - A meningioma of the left frontal lobe that resulted in ipsilateral auditory extinction. | 161 |
| 5-6 | CASE 5 - A meningioma of the anterior-superior aspect of the left parietal lobe that resulted in ipsilateral auditory extinction. | 162 |
| 5-7 | CASE 6 - An infarct of the left frontal lobe that resulted in ipsilateral auditory extinction. | 163 |
| 5-8 | CASE 9 - A meningioma of the left fronto-parietal region that resulted in ipsilateral auditory extinction. | 164 |
| 5-9 | CASE 10 - A glioma of the left fronto-parietal region that resulted in ipsilateral auditory extinction. | 165 |