AN INFORMATION PROCESSING APPROACH TO COGNITIVE RECOVERY FOLLOWING CLOSED HEAD INJURY

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A thesis submitted in partial fulfillment of the requirements of the Council for National Academic Awards for the degree of Doctor of Philosophy

January 1991 Polytechnic South West REFERENCE ONLY

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ACKNOWLEDGEMENTS

My thanks to all of the patients who participated in these studies for generously giving their time to allow testing across a number of follow-up sessions.

I am grateful for the support shown by my wife, Sandy, and daughters. Sarah and Chloe, over the years it has taken to prepare this thesis: I have tried their patience to unreasonable lengths, and promise to be a better person from now on.

Thanks are also due to Dr Tony Carr, my advisor on the thesis. He has maintained the delicate balance between an understanding of the problems experienced by a clinician attempting a PhD thesis and subtle reminders of the need to make progress.

Finally, I wish to thank my secretary. Caroline Bell, for her assistance in preparing graphs and appendices for inclusion in the thesis.

ABSTRACT

The aim of this thesis was to investigate cognitive recovery following closed head injury within an information processing approach. Reasons why Clinical Neuropsychology has neglected the potential contribution from experimental psychology were outlined. Relevant head injury variables were reviewed, including the cognitive deficits often associated with such damage and their recovery.

A pilot study confirmed that head-injured people, even soon after injury, can attempt tasks with a high information processing load. The study covered the first six months post-injury using mild/moderate and severe head-injured subjects (total n=12), the findings indicating slower performance in severe subjects and their greater susceptibility to interference from irrelevant information.

The central focus of the thesis was Sternberg's Memory Scanning Paradigm and this was described in detail. The relevant literature was discussed in depth, including both general and clinically-relevant studies. Although pertinent studies are scarce, brain damage appears to slow memory scanning speed, differential effects being

suggested according to severity of damage. In the main sample of head-injured subjects (n=42) study a was followed-up longitudinally at 1, 3, 6, 12, 24, and 36 A second patient sample (n=10) was months post-trauma. also tested at 24 and 36 months after injury, to allow a long-term follow-up "back-up" in case of excessive drop-out. A control sample (n=10) of normal volunteers was also tested. In addition to memory scanning performance patient subjects were also tested number of other clinical memory tests (Rey AVLT, digit span, WMS), and subjective memory questionnaire data were also obtained.

Findings pointed to a slowing of memory scanning ability after head injury, the degree of dysfunction being most marked in subjects who had sustained an extremely severe head injury. Evidence of cognitive recovery was noted some patients beyond 12-24 months post-injury. Significant associations between memory performance and other memory measures were observed, and a number of clinical variables were also examained. findings were discussed in detail, and a (primarily attentional) model was proposed to describe memory scanning and its dysfunction in head injury.

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CHAPTER 1

BRIEF HISTORICAL INTRODUCTION

1.1 ORIGINS OF CLINICAL NEUROPSYCHOLOGY

can be argued that due to its origins Clinical Neuropsychology has failed to achieve its potential contribution to the development of models and theory in the study of brain-behaviour relationships. The discipline has evolved from a variety of specialties, including Behavioural Neurology, Clinical Psychology, and Experimental Psychology. The relative influences of these have tended to determine the topics for investigation and the research methods employed in Clinical Neuropsychology. The impact of background specialties is outlined below.

1.1.1 Behavioural Neurology

Researchers in the fields of Medical and Surgical Neurology have long welcomed the involvement of Psychologists in behavioural (or higher functions) been that Clinical Neurology. The hope has Neuropsychologists can provide quantitative data to profile the deficits observed in a particular patient The taxonomic/classification approach group. Neurology has led some investigators in Clinical Neuropsychology to focus upon a specific disease or syndrome in order to describe it in detail. Neurology's preoccupation with acute diagnostic medicine has created interest amongst researchers in trying to discriminate between different diagnoses on the basis of neuropsychological test results.

of Neurology and Neurosurgery have had a The needs constricting influence upon the theorising of Clinical Neuropsychologists. Instead of spending some time in increasing their understanding of the cognitive deficits they have noted, many Neuropsychologists have expended their energy in developing neuropsychological measures purely to aid the process of diagnostic discrimination or syndrome description. The most refined. thorough, example of this approach is provided by the Halstead-Reitan Neuropsychological Test Battery (HRNTB), originally constructed 40 years ago (see Reitan Davison, 1974). The HRNTB was constructed by combining tasks which had been clinically validated against brain lesions, both localised and diffuse. Ιt included psychometric instruments such as the Wechsler-Bellevue (or more recently the Wechsler Adult Intelligence Scale :WAIS; see Matarazzo, 1972). Much clinical research time has been devoted to relating the HRNTB to site and type of brain lesion, and the work continues (eg. Hom and Reitan, 1984).

Although the HRNTB provides Chinical Neuropsychologists with a well-proven "diagnostic" instrument, researchers in the USA (Golden, 1981) have recently extended the "standardised battery" approach to Luria's work to develop the Luria-Nebraska Neuropsychological Battery (LNNB). It is claimed that the LNNB has clinical validity, detecting the presence of brain damage, lateralising the damage, and providing localisation information. The development of the LNNB must have required an enormous effort, in terms of "man hours", given that validity and reliability studies have been performed, hundreds of patients in various diagnostic categories have been assessed using the battery, and a large volume of test materials has been produced. Leaving aside the question of whether another standardised neuropsychological test battery is necessary for diagnostic purposes, the human research resources which have been invested in the LNNB's development and promotion are enormous (see 1.1.2).

1.1.2 Clinical Psychology

The psychometric approach to assessment traditionally favoured by Clinical Psychologists has played a major part in the development of Clinical Neuropsychology.

Davison (1974) stated that "Clinical Neuropsychology...

has roots in Academic Psychology, Behavioural Neurology, and, especially, the mental measurement or psychometric in Psychology" (page 3). He viewed Clinical Neuropsychology as "emphasising psychological tests with norms and cutting scores" and characterises Clinical Neuropsychologists as those who "measure intellectual deficits, and relate these to brain lesions.." (page 3). The influence of psychometrics, particularly in the USA, has also fostered the test battery approach and the "diagnostic" links with psychometric instruments (e.g. WAIS) have been investigated. The penchant of American Psychologists for large test batteries and multivariate statistical analysis has led to good characterisation of various patient groups, although the concomitant aim of understanding the differences between groups in terms of neuropsychological functioning has often been overlooked. The focus upon psychometric properties has opportunities for theorising and the limited the generation of models to explain particular forms cognitive dysfunction.

The most striking example of this preoccupation with psychometrics is the inappropriate development of the LNNB. Luria's method of investigation rejected the concepts of standardisation of test items, cutting scores, norms, etc. His philosophy was based upon

individual clinical examinations of patients' neuropsychological functioning, using/devising test materials which he thought specifically appropriate for This non-standardised, the particular person. approach οf Luria would have gualitative made psychometric development almost impossible. However. Christensen unwittingly helped Golden to develop LNNB by devising (with Luria's agreement) some standard test materials (Christensen, 1975). Subsequently, Golden and his co-workers proceeded to provide psychometric data on the LNNB via studies on validity, discriminative power, and the effects of age educational background (see Golden, 1981, for review). Production of the LNNB has led to a long-running argument in the scientific journals between those who view the battery as a violation of Luria's methodology with psychometric "dificulties" (e.g. Adams, 1984), and those who seek to defend it and demonstrate that it can compete with the HRNTB (eq. Golden, 1981). Through its promotion as an alternative to the HRNTB, researchers have spent thousands of hours in testing hundreds of patients to prepare many papers on the characteristics of the LNNB (recently reviewed by Stambrook, 1983).

neglecting the Experimental Psychology literature relating to cognitive functioning in non-brain-damaged people, the clinical researcher's hypothesising has been necessarily limited. Instead of pursuing this line of research. Clinical Neuropsychology has tended towards increasing refinement of psychometric and clinicallyvalidated traditional test batteries, producing improved norms by investigation of the effects of variables such as age, sex and educational background. The 'Handbook of Research Methods in Clinical Psychology' (Kendall and 1982) contains a chapter entitled Multidimensional Perspective on Clinical Neuropsychology Research" (Filskov and Lochlear, 1982). Although the chapter begins by presenting a three-dimensional model of research issues which includes an experimentalclinical axis, there is virtually no subsequent reference to experimental methods.

1.1.3 Experimental Psychology Methods

Although the Experimental Psychology tradition of theorising and data gathering from normal subjects has not been totally overlooked, its influence has appeared minor until recently. Where clinical researchers have drawn upon the experimental literature to help them understand cognitive deficits in their patients,

theoretical and clinical advances have often resulted. The area of alcohol-induced amnesia is a prime example, where paradigms provided by Experimental Psychology have assisted clinical examinations and understanding. Butters (1984) has discussed the contribution made by experimental studies of amnesia and dementia to comprehension of memory disorders. He pointed out, for instance, that differences between memory impairments in Huntington's disease and Korsakoff's disease are obvious from psychometric memory assessment. Similarly. an experimental approach to developmental disability has advanced knowledge and has led to models of the disorder which include concepts of 'surface' and 'deep' dyslexia, and to a wealth of hypothesis-testing studies (Ellis, 1984). Also, there are signs of the widening appreciation of the value of experimental psychology methods in Clinical Neuropsychology. For example, the recent book edited by Hannay (1986) specifically addresses the use of experimental techniques in Clinical Neuropsychology.

As in other branches of Clinical Psychology, British and European Clinical Neuropsychologists originally gravitated towards the psychometric tradition in assessment. However, over the last 10 years more varied research strategies have emerged in the UK and Europe.

for example, Shallice (1979), Marshall & Newcombe (1984), and Wilson (1987) have argued strongly in favour of the single-case approach in helping to understand cognitive deficits.

Principal theorists in dyslexia research are based in the UK, and many prominent workers in the field of experimental studies of amnesia are resident in this country. A positive aspect of Clinical Neuropsychology beginning to move closer to Experimental Psychology is the increasing cooperation between workers in the two fields (eg. Baddeley and Wilson, 1983).

1.2 CLINICAL NEUROPSYCHOLOGY AND HEAD INJURY

The cognitive consequences of head injury are reviewed in chapter 2, though relevant investigation methods will be introduced here. As in other fields, research into head injury has been influenced by the specialties from which Clinical Neuropsychology has evolved. Although studies on the cognitive deficits attributable to head injury have been carried out for 50 years (see, for example, the collected papers of Russell, 1971), the large majority have not employed experimental cognitive tasks. Most studies have drawn upon a relatively small

psychometric tests such as the WAIS number of Mandleberg and Brooks, 1975) and the Wechsler Memory Scale (eg, Brooks, 1976), or on the standardised HRNTB Boll, 1974). As will be discussed in the next chapter, psychometric evaluation οf cognitive functioning after head injury has underestimated the range and severity of the impairments; psychometric insensitive in detecting cognitive tests can be deficits, particularly if the assessment is performed more than 12 months after the head injury occurred.

The increase in the knowledge base about head injury and sequelae has probably also been slowed from the medical viewpoint. Neurologists are particularly concerned with acute diagnostic medicine. Few cases of head injury present a neurological 'challenge', or offer differential diagnostic problem to the neurologist clinician: head injury produces diffuse damage which is impossible to delineate clearly as with a "clean" discrete lesion, the limits of which can be resolved using CT (Computerised Tomography) brain scanning. Similarly, the Neurosurgeon may not see an intellectual challenge in head injury. Most head-injured patients suffer too-mild an injury to be referred Neurosurgeon; of those who are referred, the large majority require no surgical intervention, but rather conservative intensive care and good nursing.

Although they are a minority, some researchers in Clinical Neuropsychology have utilised models and methods taken from Experimental Psychology in their studies. For example, an 'early' study by Miller (1970) investigated cognitive functioning after head injury using a reaction time paradigm. Brooks (1974) employed signal detection theory to analyse memory performance following head injury, as did Richardson (1979). Hannay, Levin and Kay (1982) employed a tachistoscope in their research. Of particular importance have been the studies of van Zomeren and his co-workers (van Zomeren and Deelman, 1978; van Zomeren, Brouwer, & Deelman, 1984). These, and other relevant studies on the cognitive effects of head injury will be reviewed in subsequent chapters.

1.3 SUMMARY

During its evolution Clinical Neuropsychology has been particularly influenced by Behavioural Neurology and Clinical Psychology. To date their influence has outweighed that from Experimental Psychology, tending to restrict Clinical Neuropsychology's contribution to

theory and model-building. Medical and Surgical Neurology have sought assistance from the discipline in the areas of diagnostic discrimination and the profiling of intellectual impairments.

Much energy has been expended in devising and clinically validating neuropsychological test batteries for detecting brain damage and lateralising/localising lesions. The psychometric tradition, so strong in the development of Clinical Psychology, has supported the "test battery" approach, and the use of clinical instruments which may be atheoretical (eg, Wechsler Scale) rather than Memory tests developed Experimental Psychology. Only a minority of clinical neuropsychological studies have included tasks derived from Experimental Psychology. Clinical Neuropsychology can improve its contribution to the development of theory through a closer relationship with Experimental Psychology.

CHAPTER 2

REVIEW OF RELEVANT HEAD INJURY VARIABLES

2.1 DEMOGRAPHIC CHARACTERISTICS

Head injury is very common. In more than two-thirds of road accidents in the USA a head injury is sustained, this being the cause of death in about 70% of fatalities (Rimel and Jane, 1984). Work carried out by Lewin between 1967 and 1970 (quoted in the Field Report, 1976) indicated that the incidence of severe head injury, defined as a period of post-traumatic amnesia (PTA) longer than 24 hours, in England and Wales is 7,500 (150 per million). A Health District of 200,000 population could expect an incidence of approximately 30, 6 of whom could be left with a major permanent disability precluding return to ordinary work, and 2 who would require permanent nursing care. In terms prevalence, this size of Health District would contain about 112 people showing considerable disability following head injury. A recent survey of all head injury admissions for 1982 to a District General Hospital (DGH) in a district offering neurosurgical facilities (Skilbeck, Langton-Hewer and Skilbeck, 1986), noted 79 cases (11%) with a PTA longer than 24 hours (although the "catchment" population was only 215,000).

The probability of suffering a head injury is influenced by age, sex, lifestyle and other factors. Most studies indicate that head injury is 2 or 3 times more frequent in males than females (Rimel & Jane, 1984; Field, 1976; Skilbeck et al, 1986), although some (e.g. Kerr, Kay & Lassman, 1971) have reported an even higher ratio.

Age is a key variable: Rimel and Jane (1984) noted the highest incidence in the 15-19 years old age group, as did Kerr et al (1971) and Skilbeck et al (1986). Field (1976) reported this 5-year span, and 0-4 years, as the ages of highest incidence. Table 2.1 details hospital admissions for head injury, by age, in a number of large studies, demonstrating considerable agreement in the UK Rimel and Jane (1984) noted a relatively high research. incidence of head injury amongst those on low salaries (particularly students), and the unemployed. The relationship between lower socioeconomic status and increased risk of head injury does not just reflect the effect of "dangerous" lower-paid industrial occupations, as only 8% of head injuries occur at work according to the work of Rimel & Jane. This finding is supported by the Canadian work of Klonoff & Thompson (1969) who noted 10%-11% of head injuries in adults due to industrial accidents, and by Kerr et al (1971) and Skilbeck et al (1986) in the UK, who recorded 14% and 11% of cases from

TABLE 2.1: AGE & HOSPITAL ADMISSION FOR HEAD INJURY

			STUD	Y
,	Karlsbeek	Kerr	Field	Skilbeck
	et al	et al		et al
AGE (yr)	1980, USA	1971, UK	1976, UK	1986, ⊍K
0-15	23%	*	38%	32%
15-24	35%	20%	24%	29%
25-44	15%	20%	17%	18%
45-64	13%	17%	12%	18%
64+	14%	9%	9%	11%

^{*} Prorated study: no patients under 15 years included.

this cause respectively. These 2 groups of workers, and Field (1976), commented on the under-representation of social class 1 & 2 and the over-representation of social class 4 & 5 in the UK head injury data.

The evidence from a number of centres is highly consistent in identifying road traffic accidents (RTAs) as the major cause of head injury: usually about 50% of all injuries result from RTAs. This finding is again age-dependent, being associated with young adults. An unusual strength of the Rimel & Jane work was the obtaining of blood alcohol levels on 86% of their

sample. They noted 52% of their subjects as "legally intoxicated" (blood level 0.1%, or higher), and 25% reported having received treatment for alcohol abuse. The work of these authors is valuable given the dearth of relevant research, although their population may not be typical given their base in a University centre with a large (100 miles radius) rural catchment area.

2.2 MECHANISMS OF INJURY

A number of good reviews of the pathophysiology of head injury are available (e.g. Teasdale & Mendelow, 1984; Miller, 1984). The physical factors determining outcome following head injury are the premorbid brain condition, the immediate (primary) damage to the brain and subsequent (secondary) damage produced because of intracranial systemic sequelae of the injury.

2.2.1 Primary Damage

This occurs at the time of injury as a result of mechanical factors and is usually not treatable. Primary damage delivers two different types of lesion: contusion and white matter shearing. Contusions represent localised haemorrhages, often in the cerebral cortex, which may be large enough to form a clot.

Contusion under the site of impact is rare, unless a depressed skull fracture is present, this type of damage being most frequent on the under surfaces of the frontal lobes and the poles of the temporal lobes. The latter found because primary damage is determined by the relationship between a rigid skull, whose internal surface is irregular, and a non-rigid/non-compressible The mechanics are that a head injury causes the brain. brain to move within the skull, rotating and scraping against its inner surface. The maximal damage to the fronto-temporal region is caused by its relative movement against the sphenoid wing of the skull. Teasdale & Mendelow (1984) have provided a more detailed description.

The postulated importance of the contre coup mechanism, whereby damage is caused to the brain at a point opposite to the site of injury is not supported by the above finding, nor by research which indicates that when skull fracture occurs contusional damage is more frequent on the side of the brain where the fracture occurred.

The shearing of nerve axons in the white matter of the brain is now considered to be the most important process causing primary damage. The shearing arises rotational forces. which includes the movement of different brain areas in relation to each other. The discovery of this tearing process is relatively recent because of the difficulty in detecting its presence (short of post-mortem). Teasdale & Mendelow (1984)pointed out that even extensive axonal tearing may be difficult to see on the brain surface, or in section. Microscopic examination is often necessary, a process which has confirmed the tendency for shearing damage to include the corpus callosum and brainstem, although this by lesions of the cerebral always accompanied hemispheres.

It is now held that the degree of axonal damage relates to the length of unconsciousness following head injury. Long, deep comas tend to be associated with severe, widespread axonal damage. The exact mechanism by which the person is rendered unconscious is still not certain: it has been proved that brainstem damage can produce unconsciousness, but whether this can arise purely from damage sustained at the cerebral hemisphere level is unclear. Contusions at a cortical level are now regared as less significant than previously. It would seem that

they usually do not cause unconsciousness even when severe, although they may yield temporary clinical signs particularly when associated with swelling and oedema. Related focal areas of ischaemia reflect permanent damage, which may subsequently produce epilepsy.

2.2.2 Secondary_Damage

presence of this type of damage may be suspected The when loss of consciousness is delayed for some time after head injury, or when depth of coma increases. Intracranial (e.g. haematoma, brain swelling, hydrocephalus, infection) and extracranial (hypotension, hypoxia) events can lead to secondary damage. Whatever the specific factor(s) involved, the underlying hypoxic/ischaemic mechanism is either brain or compression (Teasdale & Mendelow, 1984).

Intracranial bleeding following trauma produces a clot (haematoma) in approximately 40% of comatose patients Blood clots within (Miller, 1984). the cortex haematoma) and those outside (intracerebral the brain substance but within the dural membrane (subdural than extradural clots. haematoma) are more common latter generally produces good Evacuation of the results, though removal of intracerebral and subdural

haematomas is often less successful because of their association with primary damage. Brain swelling may result from an increase in the amount of tissue fluid in the brain (oedema), or from a rise in cerebrovascular volume (itself often a secondary result of constriction of cerebral veins due to oedema). Oedema can produce a shift in brain tissue and/or raised intracranial pressure (ICP), producing ischaemic damage. Excess fluid in the brain, elevating ICP, can also occur because of malabsorption of cerebrospinal fluid (CSF). Other secondary factors, such as infection, form rare complications of head injury.

Extracranial events can also lead to secondary brain dysfunction, these events often being linked to difficulties in respiration (eq, air or blood in the pleural cavity of the lungs). In these cases insufficient oxygen is available to be carried in the vascular system to the brain, resulting in hypoxic damage. Because of shock and blood loss hypotension in the cerebral circulation can give rise to ischaemic damage (Teasdale & Mendelow, 1984).

2.3 MEASUREMENT OF SEVERITY OF HEAD INJURY

A small number of useful indicators of trauma severity are available, particularly length/depth of coma and duration of post-traumatic amnesia (PTA).

2.3.1 Coma

Any head injury which involves no, or only brief (minutes), loss of consciousness is likely to be very mild. Exceptions to this rule include those cases in which secondary brain damage is acquired because of intracranial bleeding, even though no loss of consciousness occurred at the time of injury. For those cases where some depression of consciousness persists at least until admission to hospital, it is important to have a method for characterising the depth of coma. The most widely-used scale for this purpose is the Glasgow Scale (GCS: Table 2.2), which defines level of Coma consciousness in terms of the patient's verbal, motor eye-opening responses (Teasdale & Jennett, 1974). The lower the score, the deeper the coma. Rimel & Jane (1984) noted that 25% of their patients were 'comatose', having a GCS score of less than 9. These authors noted 'minor' head injuries (GCS 12-14) in 49% of their sample although 93% of patients reported losing consciousness

at the time of injury (42% were comatose on admission). study duration of unconsciousnes was this often confounded by alcohol intake. Given the high reported of unconsciousness. Rimel & Jane seem to have included a relatively high proportion of serious head injuries. This suggestion is supported by the findings al (1986), who noted Skilbeck et а loss consciousness in less than 50% of their patients and GCS scores of 12-14 in 85% of their population.

Introduction of the GCS has helped to standardise coma as an indicator measurement of of head injury severity. Its strengths include a high inter-rater reliability (Teasdale, Knill-Jones & Sande. probable pood cross-cultural reliability because language does not confound its use, and it requires no special expertise or training for its use. The capacity of the GCS to predict outcome after head injury suggests offers a satisfactory measure of initial severity. For example, Jennett, Teasdale & Braakman (1979) noted that 87% of their patients with GCS scores of 3-4 died became vegetative, whereas only 12% of those with scores of 10+ suffered these outcomes. Similarly, only patients with these low scores made a left with a moderate disability, recovery or were compared with 87% of those scoring 10+ (see table 2.4).

TABLE 2.2: THE GLASGOW COMA SCALE

<u>Item</u>	<u>Score</u>	Response
Eye Opening	1	never
ı	2	to pain
	3	to sound
	4.	spontaneously
Best Motor Response	1	none
	2	extension
	3	flexion
	4	localises pain
	5	normal
Best Verbal Response	1	none
	2	incomprehensible
	3	inappropriate
	4	confused
•	5	orientated

Skilbeck et al (1986) found a 54% death/'vegetative' rate amongst patients with GCS scores of 3-4, and a 1% death rate for scores of 11-14. Unlike Jennett and his co-workers, Skilbeck and his colleagues noted that 39% of patients with poor GCS scores either made a good

recovery or were left with only a moderate disability, the corresponding figure for those with GCSs of 11-14 being 98%.

2.3.2 Post Traumatic Amnesia (PTA)

PTA can be defined as the period extending from the moment of head injury until the re-establishment of continuous memory. During PTA 'islands' of memory may form, but the period of amnesia is not at an end until continuous day-to-day consolidation of events into long-term memory has been achieved. PTA as an indicator of severity may be thought less useful than depth of coma, given that it can be difficult to determine its exact length (often dependent upon patient report), and that it is an index which may not be available immediately after a head injury. However, even given these possible drawbacks PTA has proved to be the most sensitive indicator of severity of head injury, particularly in relation to cognitive outcome (see 2.5 below).

PTA was proposed as a severity index 50 years ago by Russell (see Russell,1971). He suggested the scaling shown in table 2.3. As this table indicates, the large majority of head injuries are very mild.

TABLE 2.3: LENGTH OF PTA & SEVERITY OF HEAD INJURY

Length of PTA	Severity	Skilbeck
		et al, 1986

0-60	min	mild	84%
1-24	hr	moderate	5%
1-7	day	severe	5%
7+	day	very severe	6%

2.4 MEASUREMENT OF OUTCOME: PRELIMINARY CONSIDERATIONS

In common with many other clinical problems, the study of head injury has tended to concentrate upon the acute stage (diagnostic and initial management features). However, once beyond the immediate, potentially life—threatening consequences of the injury, families are more interested in the degree of recovery and the 'quality of life' of the patient. The clinical research position has changed over the last 10 years and interest has developed in studying outcome, its prediction, and the rehabilitation needs of patients and their families. After preserving life, the most important aspects of outcome relate to self-care and independence: cognitive, emotional, social and occupational functioning.

A number of simple global outcome scales have been devised; the most popular being the Glasgow Outcome Scale (GOS; Jennett & Teasdale, 1981). The most useful version of the scale has 5 points (table 2.4). The poorest outcome is death, with vegetative state ('condition of non-sentient survival', Jennett & Teasdale, 1981) being the next poorest: patients can show wakefulness without any associated meaningful cognitive activity.

TABLE 2.4: THE GLASGOW OUTCOME SCALE

Category	Description					
5	dead					
4	vegetative					
3	severely disabled					
2	moderately disabled					
1	good recovery					

The GOS 'severely disabled 'category includes those patients who have regained consciousness but who are dependent upon others for some activities of daily living. In the worst cases, patients may be severely physically disabled and also suffer a marked handicap in communication. Severe physical problems will always be

associated with gross cognitive deficits, although some patients will be classified as having a severe disability on the basis of their cognitive problems alone: the degree of their cognitive impairment is such as to make them dependent upon others for some of their daily needs, or for supervision. Severely-disabled people often become residents of an institution, though sometimes even those who are highly dependent can be cared for at home if domestic circumstances allow.

Those with a 'moderate disability' are disabled but capable of independent living, and may return to some form of work. Most patients in this category will show some cognitive deficits and/or personality problems. Patients showing a 'good recovery' may not fully regain their pre-morbid status. Although they may have mild deficits detectable via neuropsychological assessment, they are able to undertake a normal social life and to return to work.

The prediction of GOS grades from initial data on severity of injury has been attempted in a number of studies. As mentioned in 2.3.1, Jennett et al (1979) noted that the outcome for 87% of patients with initial GCS scores of 3-4 was death or a vegetative existence, whereas this was the outcome for only 12% of patients

with a GCS of 11-14. The corresponding results for Skilbeck et al (1986) were 54% and 1%. Similarly, length of PTA and outcome has been investigated. Table 2.4 indicates that in the Jennett & Teasdale (1981) study no patient with a PTA of less than 14 days was classed as severely disabled at 6 month follow-up (and 83% had made a good recovery), whereas 30% of patients with a PTA longer than 1 month were severely disbled (only 27% were judged to have made a good recovery). In the Skilbeck study 47% of patients with a PTA longer than 1 month made a good recovery.

prognostic significance of a number of other been investigated. variables has also Jennett & Teasdale (1981) reported a clear linear relationship between age and GOS score, such that many children (approximately 50%) make better recoveries compared with less than 10% in those aged 60 years or over. The study by Jennett et al (1979) suggested that the presence of an intracranial haematoma increased the probability of a poor GOS outcome (death/vegetative state), in younger However, these authors noted little prediction value from skull fracture, type of injury occupation-related), side of (RTA, assault, fall, or maximal brain damage or occurrence of a major chest injury.

2.5 PSYCHOLOGICAL OUTCOME: COGNITIVE FUNCTIONS

The psychological consequences of head injury are generally of greater long-term significance than physical injuries (Yishay & Diller, 1983). Because head injury is a pathological process which produces diffuse damage to the brain, the range of cognitive functions which may show deficits is large. These include memory, attention, and spatial organisation abilities (Yishay & Diller, 1983). Although specific cognitive deficits often occur together, it is convenient to consider them separately particularly as researchers have tended to focus upon one type of deficit.

That head injury can cause impaired cognitive. functioning is well documented, dating back to the 1930s. For example, Conkey (1938) compared a sample of mild head injury patients with control subjects over the first year post-injury. Her findings indicated that the patients showed deficits in perception, motor speed, memory and learning. She interpreted her findings as suggesting that permanent cognitive deficits were probably only acquired in relation to more complex functions.

Although a small number of studies appeared in the 1930s 1940s, major research interest in cognitive functions and other psychological sequelae of head injury only revived in the 1970s. Brooks (1984a) has provided a good general review of cognitive deficits following head injury. Brooks, Deelman, van Zomeren, van Dongen, van Harskamp and Aughton (1984) considered the methodological and practical problems in measuring cognitive recovery after head injury. These authors identified the testing schedule, functions to be assessed and type of control group as relevant variables, and emphasised the importance of achieving as high a follow-up rate as possible. Their review indicated that most studies have ceased follow-up by 12 months post-injury, or sooner, usually on the assumption that cognitive recovery has reached a plateau. However, with more severely-injured patients an extended followup may be justified, and "even 1 or 2 years may not be enough to fully record the natural history of the recovery" (Brooks et al, 1984, p.74).

The schedule of follow-up may be considered in terms of the specific cognitive functions under investigation. Brooks and his co-workers suggested that more complex functions should be followed for a longer period, citing the work of Mandleberg (1975) who observed changes in

performance IQ up to 2 years post-trauma, and van Zomeren & Deelman (1978) who reported gains in choice reaction time in the second year after injury.

et al (1984) pointed out Brooks that different researchers have resolved the question of subjects in a variety of ways. For example, Brooks & Aughton (1979a) used non head-injured hospital patients, Gronwall & Wrightson (1974) used a mild head-injured comparison for a more severely injured experimental group, and Levin, Grossman, Sarwar & Meyers (1981) used normal healthy working subjects to form their control group. Others have employed no control group, leaving it to already-available normative data to provide the basis against which to compare their experimental group.

Brooks and his co-workers also reviewed the problem of distinguishing practice effects from natural recovery. They concluded that serial testing of head-injured and control subjects is generally satisfactory, though even with this design it could be that head-injured subjects differentially benefit from practice on the test due to possible interaction effects between level of performance and gain from practice. One solution to this potential problem is to compare the scores of a

serially-tested group of patients with those tested only once at the same (final) point; for example, one group might be tested at 3, 6, and 12 months post-trauma and the second group only at the 12 months point. Using this type of procedure, Brooks et al (1984) reported some evidence of possible practice effects for Raven's Progressive Matrices (Raven, Court & Raven, 1977), and cautioned that conventional psychometric tests are often those most prone to practice effects. However, Mandleberg and Brooks (1975) failed to note such effects in an earlier study.

al (1984) pointed out that the Brooks alternate forms of a test may not avoid the problem of practice effects, partly because of 'learning to learn' carry-over effects between conceptually-similar material (in addition to the difficulty of ensuring equivalence between so-called parallel versions of recommended selection of test). They measures intrinsically unaffected/little affected by practice, which they felt removed the need for a control group. Amongst these meaures they cited the complex information processing tasks involving reaction time utilised by van Zomeren & Deelman (1978), and encouraged their use.

2.5.1 Memory

This area has received most attention from Neuropsychologists investigating the effects of head injury. Schacter & Crovitz (1977) provided an excellent review, covering PTA, the nature of memory deficits observed and their recovery time course.

A variety of memory deficits may be apparent after a significant head injury. Soon after the trauma patients may show disturbances in their day-to-day memory. At this stage they are said to be "in PTA" (see section 2.3.2). Patients may also demonstrate recall difficulties for events immediately preceding the trauma. This so-called retrograde amnesia usually covers a short period (minutes/hours) and tends to 'shrink' with the passage of time, so that recall for some events just prior to injury returns.

Many studies have shown that once the period of PTA has ended, impairments in memory and learning may still persist (Schacter & Crovitz, 1977). As might be expected, severity of memory impairment seems to be related to the 'severity' indices of coma and PTA, the association being much stronger for the latter. Tooth (1947), Dikmen, Machamer, Temkin, & Mclean (1990), and

Teasdale & (1974)Jennett noted a non-significant tendency for memory disturbance to be positively associated with length of coma, with Levin, Grossman, & Teasdale (1979)observing a significant between coma duration and poor GOS score relationship (see section 2.4), and between GOS scores and memory or learning scores. A number of studies have reported a significant relationship between length of increasing severity of memory deficit (eg. Tooth, 1947; Smith, 1961; Brooks, 1976; Brooks & Aughton, Russell & It is worthy of note that the Wechsler Memory 1979a,b). Scale (WMS; Wechsler, 1945) figures very prominently in the examination of memory after head injury. example, Brooks (1976) noted poor performances by headinjured subjects on subtests of the WMS up to 2 years after injury.

Russell & Smith (1961) noted a clear association between length of PTA and the probability of developing a memory or calculation deficit (although they did not specify the nature of the testing, nor the time post-trauma when testing took place). They observed that 11% of patients with a PTA of 1-24 hours, 29% of patients with a PTA of 1-7 days and 56% of patients with longer PTAs developed such deficits. In their review, Schacter and Crovitz (1977) concluded that the evidence was somewhat

inconsistent with regard to the relationship of PTA duration to subsequent memory impairment. Time of testing seems important in that studies generally show this relationship to be strong when testing has occurred within 12 months of the trauma, whereas the evidence for the association at longer periods is more equivocal. Schacter & Crovitz (1977) concluded that "future studies should examine the relationship between PTA duration and specific features of memory as revealed by objective testing"(p.161).

Attempts have also been made to relate other clinical features to observed memory impairment after head injury, Brooks (1984a) reviewed this aspect of literature, including possible efects of presence/site of skull fracture, persisting/severe neurological signs, presence of subdural haematoma, and age. He concluded that most of these factors had little bearing upon the severity of memory impairment, particularly when the confounding effect of length of PTA was taken consideration. Clinical signs which may correlate with severity of memory deficit include early hemiparesis or abnormal motor findings (Levin, Grossman, Rose Teasdale, 1979; Dye, Milby and Saxon, 1979).

Little work has addressed the questions of rate and extent of recovery of memory deficits after head injury. Gronwall and Wrightson (1974) reported that patients with a PTA of under 1 hour on average took 27 days to return to normal performance on the Paced Addition Serial Task, whereas the corresponding figure for those with a PTA of 1-24 hours was 41 days. Methodological problems encountered in attempting such work, including practice effects and high drop-out rate, have been mentioned above. Brooks & Aughton (1979b) noted that patients failed to attend for follow-up. Similarly, Conkey's (1938) experiment involved 5 testing sessions for subjects in the first year post-injury. Although she assessed 25 patients initially, only 4 attended all follow-ups. Brooks (1984a) provided a review of studies employing the sequential testing of memory functions. These studies included a variety of re-test intervals and followed their subjects for 1-3 years.

Brooks (1984a) commented on the difficulty of comparing different studies, given variations such as the number/type of patients investigated, types of tests utilised, and method of statistical analysis employed.

However, he did conclude that studies on simple memory (digit span, WAIS) have produced results indicating good recovery (often a return to normal level) within 3 years or much sooner. Verbal learning appears to show a slow recovery curve, with marked deficits being noted at least 1 year after injury. In their review Schacter and Crovitz (1977) noted that memory performance following closed head injury does improve with time, although an insufficient number of post-trauma assessment times have been employed to allow a detailed description of the time course of recovery.

Only in the last 15 years have studies appeared in any number which have investigated the nature of the memory deficit associated with head injury. Writing in 1977 Schacter & Crovitz addressed the question of whether the memory impairment could be characterised as a storage or retrieval deficit. This approach, given the diffuse damage inflicted upon the brain in a significant closed injury may appear too specific, however correct scientifically. Schacter & Crovitz found the available evidence inconclusive in this respect, and Richardson's (1978) description of a "generalised impairment function, observable in free recall, recognition memory, and paired-associate learning, with both pictorial and verbal material, and with both unrelated words and

connected narrative" (p.700) is probably a better approximation of the real (clinical) world. Schacter & Crovitz did, however, offer one useful conclusion — that increasing the period for which the patient has to hold on to information before retrieval differentially penalises head—injured patients compared with control subjects. These authors also pointed out that among the areas which have as yet received little attention is the relationship between memory impairment and other cognitive deficits.

Clinicians have occasionally queried the extent to which memory test findings in the hospital will be paralleled in everyday life; ie, is it safe to presume that test findings will generalise to a patient's life out in the community? Sunderland, Harris & Baddeley (1984) recently reviewed this issue and questionnaires designed to more directly reflect patients' everyday memory functioning after head injury via self-report relatives' ratings. This 'subjective report' approach carries a number of risks, given the nature of the data obtained, and Morris (1984) discussed the central problem of validity. His opinion was that correlation between subjective and objective test) report is generally low either because tests do not reflect real-life performance, or because the former

accurately assess memory impairment: it may be false to expect meaningful correlations between the two Morris pointed out that in using subjective questionnaires, the self-report relies upon the patient 'appropriate' memory failure: having the an questionnaire items may be too specific to be relevant to the respondent. In addition, patients must first recognise that they have a memory deficit before being able to classify it, and must remember the failure in order to report it. There is also the risk patients will become sensitised to 'normal' failures, which are common to all, and will report these acquired deficits. Schacter & Crovitz dismissed the use of subjective reports of functioning, seeking instead to promote more detailed objective assessment.

Morris (1984) discussed the confounding factors acquiescence and social desirability which may operate questionnaires. subjective memory Although defended their of additional use as а source information. he did not feel they could replace the testing of actual memory performance. In their study, Sunderland et al (1984) noted significant correlations between memory test results and subjective estimates of memory functioning produced by head-injured patients and their relatives. The highest correlations were noted between short-story recall and relatives' reports (questionnaires: r=.72, p<.01; checklist: r=.58, p<.01), with weaker associations being observed for patient responses (questionnaire: r=.50, p<.01; checklist: r=.36, p<.05). The issue of degree of corresponence between subjective and objective measures of cognitive performance requires further research. A useful approach (Wilson, Cockburn, Baddeley, & Hiorns, 1989), is the development of behavioural memory tests which may help to reconcile the two methods of measurement.

2.5.2 Attention

Van Zomeren, Brouwer & Deelman (1984) provided a review of theories of attention, including those by Broadbent, Triesman, Shiffrin & Schneider, and also outlined the concepts of alertness, selectivity, and speed of information processing. The present study particularly involves investigations of the latter, and its detailed consideration will be undertaken in chapter 3. Van Zomeren and his co-workers remarked on the long history of references to attentional deficits in the literature. They cited the work of Meyer in 1904 which referred to patients being "unable to concentrate their attention".

However, as these authors indicated, very often studies mentioning attentional difficulties are merely reporting clinical impression, proposed to account for poor psychological test performance.

Dencker & Lofving (1958) tried to test for impaired attention using monozygotic twins, one of whom in each pair had sustained a head injury. The sample was also unusual in that at the time of testing the posttraumatic period averaged 10 years, and approximately two-thirds had suffered a mild injury (PTA of 1 hour, or In their experiments stories were read less). subjects, whilst interfering information was presented (a number of simultaneous conversations). Subsequently, subjects were asked story questions. Dencker & Lofving's findings indicated no differences in recall performance between the headinjured and control groups, which may not be surprising given the time since injury and the mild nature of most of the head injuries sustained.

A more recent study by Gronwall & Sampson (1974) also examined subjects who had suffered a mild head injury. They employed a dichotic listening procedure within 24 hours of injury, and again failed to detect any interference effects upon attention. Van Zomeren et al

(1984) criticised these 2 studies on the grounds that discrimination needed to sustain attention to the relevant message against interference was not difficult; in the Dencker & Lofving study the message (story) was read aloud to subjects and the interference was recorded, and Gronwall & Sampson consistently presented the message to only one ear in their dichotic task. Another study which yielded negative findings was that of Miller & Cruzat (1981) who employed a card-sorting task (relevant stimuli being the letters 'A' and 'B') in an experiment including irrelevant information (0,1,4,8 additional letters). This study, discussed in greater detail in chapter 3, only indicated slower performance in the severely head-injured group.

However, more recent RT research on milder head injury yielded significant results in relation to attentional processes. Gentilini. Nichelli. (1985) studied patients who had Schoenhuber, et al suffered a mild head injury (defined as a period of unconsciousness of less than 20', initial GCS of 13-15, and length of hospitalisation less than 3 days). particularly well controlled, via study was matching, and included 50 patients. The results obtained at 1 month post-injury failed to reveal significant differences between patients and control subjects on Raven's PM and a number of memory tests, although a significant ANOVA finding (p<.05) was noted using a test of selective attention.

McMillan and Glucksman (1987), within 1 week of their 24 head-injured patients with PTAs of trauma, examined between 1 24 hours and and a brief period unconsciousness. They employed a range of tests, including the PASAT, and used a control group othopaedic patients. All intellectual and memory test variables failed to distinguish between the patient and control groups, the only significant finding (PK.01) being obtained from the PASAT. This significant result was noted in relation to a fast presentation of digits, being no significant differences between the 2 groups with a slower rate of presentation. McMillan and Glucksman concluded that their findings pointed to head injury affecting the rate of information processing in association with difficulty of task, rather than just reflecting a reduction in processing ability per se.

Van Zomeren et al (1984) also reported on 2 studies in which the Stroop test (Stroop, 1935) was used with negative results. In their own work van Zomeren and his colleagues utilised a visual Choice Reaction Time (RT) paradigm to investigate interference effects. They

studied 20 patients over a wide severity range, at 3-12 months post-injury, and a normal control group. The stimuli comprised 4 buttons which, when lit. provided the response device. After running trials with no interference, irrelevant button stimuli were added to array (1 per response button). These irrelevant lights which were situated close to the relevant S-R buttons lit up in concert with their stimulus 'twin', so Van distracting subjects. Zomeren et al's results demonstrated that although interference occurred for both groups, the irrelevant stimuli had a significantly greater (p<.001) distractibility effect upon headinjured subjects.

latter finding is supported by the results of Stuss, Ely, Hugenholtz, Richard, LaRochelle, Poirer & (1985) who noted a highly-significant (p<.0001) Bell difference between a group of 20 head-injured patients, of mixed severity (65% severe/very severe) tested at 5 months post-injury, and well-matched control subjects in terms of recall performance under Brown-Peterson interference conditions (Brown, 1958). The Significance levels obtained were lower WMS for measures, and no WAIS comparisons reached significance.

MacFlynn, Montgomery, Fenton, and Rutherford (1984)concentrated on investigating RT performance in minor head injury (PTA< 24 hours) against that of case matched controls. Patients were tested on a 4-choice procedure within 48 hours of their injury, at 6 weeks, and at 6 months post-trauma. Using t-test analyses these authors noted significantly poorer RT performance in the patient group at their first 2 follow-up points, but not at 6 months after injury. An unexpected finding was the significantly faster (P<.05) RTs in the patients compared with the controls at the latter follow-up. authors faiil to account for this satisfactorily, referring to possible practice effects despite the 4.5 month interval between the sessions 2 and 3.

The work of Van Zomeren and his colleagues on attention after head injury not only showed patients' proneness to interference, but also examined recovery of information-processing capacity. The time course plotted by van Zomeren (1981) suggested that severely head-injured people may continue to recover beyond 2 years on high information-processing capacity tasks (choice RT).

Development of the concept of a Supervisory Attentional System (SAS) by Norman and Shallice (Shallice, 1988) is important in the context of head injury, given it has

been linked to frontal lobe functions. The SAS is viewed as significant in the initiation of voluntary actions, and is necessary where the routine behaviour selection is inadequate to deal with novel situations, or where the environment presents dangers. Shallice indicated that when the SAS malfunctions 'frontal' disorders can be observed. As its name implies, the SAS has a modulating, rather than a directing/dictating, role in relation to psychological processing.

Posner and his colleagues (Posner, Cohen, & Rafal, 1982) postulated a more specific visuospatial attentional control mechanism. They investigated the concept in relation to left-side visual neglect. They noted that with a left-side target stimulus and the provision of an simultaneous invalid visual cue (an almost directing attention to the right) patients with neglect usually failed to detect the target at all. However, with the introduction of a 50 msec. delay between the invalid cue and the onset of the target stimulus, these patients responded to the target although they took longer than control subjects to do so. The 50 msec. cue-target interval is too short to allow eye movement, and Posner's group viewed the findings as showing that a neglecting patient's damaged attentional system longer (ie, 50 msec.) to re-orientate to the left side.

2.5.3 <u>IQ</u>

A number of studies have been carried out to assess the effects of closed head injury upon IQ. Often workers have employed the WAIS (Wechsler, 1955); for example, Mandleberg (1976), Mandleberg & Brooks (1975), and Levin et al (1979).

Generally, researchers have reported that verbal IQ recovers well, approximately to premorbid level, with performance IQ showing both greater deficit initially and often a prolonged period of impairment. performance functions appear to show permanent deficits, particularly after a severe head injury. Mandleberg & Brooks (1975) conducted serial testing on a group of severely-injured patients, their results showing no significant improvement in any verbal WAIS scale when the scores of patients at 4-6 months follow-up were compared with those at 13 months follow-up. However, significant gains (p<.05) were noted for all performance subtests except picture completion, and performance IQ improvement was significant at p<.01. group comparisons of patients against a control group (neurotic psychiatric patients), the former scored significantly lower at the 0-3 month follow-up for verbal IQ (p<.01) and performance IQ (p<.001), at the 46 month follow-up for verbal IQ (p<.05) and performance IQ (p<.001) and at the 7-12 month point for performance IQ alone. No significant IQ comparisons were noted at follow-up beyond this point. The only WAIS subtest to offer significant results for comparison of the 2 groups at every follow-up was digit span, although digit symbol and picture arrangement yielded significant differences at all except the final one.

As mentioned in section 2.5.2. Stuss et al (1985)impressive findings, failing to note obtained even less significant differences between head-injured (all of whom were at least 5 months patients postmatched controls on and any WAIS scale. trauma) However, it should be pointed out that Stuss's patients tended to have suffered milder injuries (35% of sample had a PTA less than 1 day). IQ tests do not appear particularly sensitive general indicators of cognitive functioning when compared with corresponding results obtained from assessing memory and attention.

Brooks (1984a) pointed out that a number of hypotheses have been advanced to account for the different post-injury course seen in verbal and performance IQs, including the suggestion that performance tasks require sustained effort, involve a speed component, or are

intrinsically more complex in nature. Verbal WAIS items, in contrast, usually require a simple response.

Attempts have been made to relate the intellectual deficit observed to indices of severity of injury. Whilst duration of coma does not help to predict subsequent intellectual performance, increasing length of PTA is associated with greater intellectual impairment, especially for performance IQ (Brooks, 1984a). Brooks (1984a) concluded that severity of injury does not affect rate of recovery: severely-impaired patients recover at the same rate as mildly-damaged patients, but as the former are very likely to show a lower initial intellectual level they will achieve lower final plateaux.

2.6 PSYCHOLOGICAL OUTCOME: SOCIAL ASPECTS

Whereas a sizeable literature concerning cognitive outcome following closed head injury has accumulated, especially over the last 20 years, the number of available studies relating to social factors is relatively small and tends to be more recent. Oddy (1984) and Brooks (1984b) have provided good reviews of the general area and this section will focus more upon studies examining return to work after head injury.

early investigation by Rowbotham, MacIver, Dickson An and Bousfield (1954)reported on the postal questionnaire responses of 236 patients at 3-4 years injuries of varying severity. Their results indicated that less than 5% had failed to return to work after head injury, although a further 12% had either not regularly or had taken 'light' jobs. worked Oddy's review concluded that even with severe cases, (1984)80%-90% are able to return to work. Studies involving very severe injuries, including those in which patients were unconscious for 3 weeks or more, suggest a 60%-75% rate of return to work although this rate may be reduced by pre-existing alcoholism and in older patients (see Oddy, 1984).

A number of studies have pointed to the importance of psychological deficits, both cognitive and personality, in determining return to work, including those by Fahy, Irving & Millac (1967), Bond (1975) and Roberts (1976). The work of Oddy and his co-workers is of particular value, given the length of follow up achieved. Their original paper (Oddy, Humphrey & Utley, 1978) reported on 50 severely head-injured patients and an age-matched 'orthopaedic' control group. Whereas 97% of the control group and 71% of patients with a PTA of 7 days or less had returned to full/part-time work by the 6 months

follow-up, only 50% of the very severe patients had achieved this. By 12 months post-trauma, 96% of the severe and 73% of the very severe patients had returned More pessimistic findings were reported in a subsequent paper (Oddy, Coughlin, Tyerman & Jenkins, 1985), in which another group of very severe patients were followed at 2 years and 7 years post head-injury. Occupational data was available on 43 patients at both points. At 2 year follow-up 48% had returned to work, a figure which was virtually unchanged at the 7-year At this latter follow-up all of those who were point. unemployed at 2 years were still unemployed, number of patients had improved their status from "fulllower level" to return to "former time work at a job/normal career progression".

More recently, Brooks, McKinlay, Symington, Beattie, and Campsie (1987) pointed out the wide divergence in estimates of frequency of return to work after head injury. This variability stems not only from severity of injury, but also from length of follow-up. Brooks et al followed 134 of their severely head-injured patients for 7 years after injury. Whilst 86% of their sample had been in employment before head injury, only 29% had a job post-trauma. Brooks and his colleagues also examined cognitive outcome, and obtained information on

emotional and behavioural outcome, as well as Follow-up assessments personality ratings. were conducted at various times post-injury, which allowed calculation of changes in employment rate over time. These authors noted no clear evidence of an increase in employment rate beyond 2 years post head injury. data did suggest, however, that patients professional/managerial occupations had a higher chance of returning to work, as did those under 45 years of age. Multiple regression predictions of return to work showed a significant contribution from verbal memory and PASAT score. Those returning to work tended also to be rated as having been more 'energetic' in their premorbid state, to show less evidence of changeable depressed mood after injury, and to have better anger control post-traumatically.

In both of these studies cognitive difficulties appeared to play a part in determining return to employment, although some caution may be necessary before accepting subjective reports in this area (see section 2.5.1). Oddy et al (1978) noted that memory problems were the most frequently reported symptom at 6 months post-injury by both patient (38%) and relative (44%). The picture is enhanced at the 7-year follow-up (Oddy et al, 1985) when both patients (53%) and relatives (79%) indicated

that memory problems were, by far, the most frequent complaint. At that point "concentration difficulties" was reported as the second most frequent problem by both patients (46%) and relatives (50%). Reviewing the progress of patients in rehabilitation, Oddy (1984) concluded that their results suggested "an interaction between severity of closed head injury and the effects of personality and cognitive deficits on ability to return to work....both were strongly related to delay in returning to work" (p.115).

The Glasgow group of researchers have produced similar findings (McKinlay, Brooks, Bond, Martinage & Marshall, Brooks, 1984b). The study by Mckinlay and his colleagues observed frequent reports of personality and cognitive deficits amongst relatives of severely-injured patients. For cognitive deficits, in the 3-12 month follow-up period the frequency of reporting slowness varied between 86%-67%, and memory problems between 73%-69%. Brooks (1984b), in his review, concluded that a high degree of memory and personality impairment was associated with a loss of working capacity and disruption in both family relationships and activities. Findings on the importance of cognitive deficits and their persistence are not restricted to UK studies. For example, van Zomeren & van den Burg (1985)

followed-up 57 severely head-injured patients for years. They noted 54% of their sample reporting memory difficulties, 33% poor concentration and 33% slowness. all, 84% of patients reported some residual cognitive/personality difficulties. These authors demonstrated that slowness (r=.36, p<.05), and inability to handle two tasks simultaneously (r=.56, p<.05), correlated with level of return to work. A Principal Components Analysis yielded 2 factors, one of which showed high loadings from PTA (.80), return to work (.70), forgetfulness (.63), slowness (.66) and inability cope with two tasks simultaneously (.62).

indicated at the beginning of this section, As comprehensive review of social variables is beyond the scope of this thesis. The available studies may be summarised generally reflecting considerable as personality/emotional disturbance in patients following head injury. Table 2.5 presents data from a number of studies on the more common symptoms reported. The large variations in reported disturbance may result differing follow-up points, type of respondent (relatives tend to report disturbances more often than patients) and particular questionnaire/checklist used. One depressing aspect of the table is that the work of McKinlay et al (1980) provides little evidence that

these social and emotional problems resolve across the first 12 months after trauma. Indeed, these authors' results suggest that problems may intensify during this period. Using a different index of social functioning, the study by Oddy et al (1978) revealed that 33% of severely-injured patients at 6 month follow-up felt that their leisure activities had been adversely affected by their head injury, with the corresponding figure for very severely-injured (PTA 7+ days) being 42%.

TABLE 2.5: FREQUENCY (%) OF SOCIAL/EMOTIONAL PROBLEMS

Senior	Oddy		McKinlay			van	Zomeren	0	Oddy	
Author:	1978		1981			1985		1	1985	
	<u>(n=50)</u>		<u>(n=55)</u>			-	(n=57)	<u>(n</u>	(n=34)	
Follow-up:	6 m		3,6,12 m			2	24 m	7	yr	
Sample :	Pt.	Rel.	Relative			Pá	atient	ent Pt. Rel		
			Зт	6m	12m					
Bad Temper	35	33	48	56	67		_	31	-	
Easily Tired	33	38	82	69	69		30	_	43	
Low Drive	21	_	-	-	_		23	28	43	
Impatient	29	35	36	69	71		39	_	43	
Depressed	_	-	57	52	57		19	-	-	
Anxious	_	_	57	66	58		18	_	-	

The research conducted by van Zomeren and van den Burg on psychological variables in head injury (1985)revealed 2 main factors in the data. One, discussed in section 2.5.2, related to severity of injury and The second factor, which showed cognitive deficits. negligible loadings from PTA and 'return to work', recorded high loadings from a number of social/emotional variables, such as 'irritability' (r= 59), 'fatique' (.68), and 'loss of initiative' (.51). Van Zomeren & van den Burg's analyses demonstrated that these subjective non-cognitive factors did not relate to the main index of injury severity (PTA), nor to return to Cognitive and social/emotional psychological variables generally did not intercorrelate highly their study, though undoubtedly the frequency of these social 'symptoms' must reflect a high level of stress for both patient and relatives, and must place a great burden upon family relationships.

Epilepsy after head injury can be viewed as a medical or psychological (both cognitive and social) consequence. Because of its potentially-major effect upon psychological functioning, it is probably best viewed in the latter category. The incidence of post-traumatic epilepsy is well documented (Jennett, 1975), and approximates 5% (Skilbeck et al, 1986). Dodrill (1981)

has provided a comprehensive review of the psychological problems for patients with epilepsy, including social stigma. Beyond social difficulties, the epileptic patient is likely to have to cope with the cognitive problems caused by his or her anticonvulsant medication (Trimble & Thompson, 1981).

2.7 SUMMARY

Severe head injury is relatively common, with the average UK health district accumulating approximately 30 new cases each year. At greatest risk are teenage males, with low socioeconomic status also being an important factor. The most common cause of head injury is a RTA. The primary damage, contusion and nerve axon shearing, arises at the time of trauma with secondary damage (hypoxic/ischaemic, or brain compression) occurring subsequently, if at all.

Depth of coma and length of PTA offer useful indices of the severity of head injury. Most people sustaining injury do not lose consciousness, but the development of the GCS has helped to standardise measurement of coma. Both GCS and PTA can be used to predict outcome, the latter more accurately. Although studies have often concentrated upon the acute medical aspects, in the

longer term degree of recovery and quality of life are more important. The GOS provides a simple, if crude, measure of level of recovery.

Given that the psychological consequences, rather than the physical damage sustained, are more significant for patients and their families (except in the very short literature has developed which addresses term) cognitive deficit after head injury. This memory, attention, and IQ. Studies assessing social/emotional outcome are both fewer in number and tend to have appeared more recently. Although there are methodological and practical difficulties in charting cognitive recovery, it is now well-established that memory functions are often impaired as a result of head injury. The degree of impairment can be related to the severity of the injury sustained, and recovery is often than for other cogntive abilities. slower relationship between subjective reports of memory disturbance and objective test results has yet to be fully explored.

Attentional deficits have recently also been investigated, results to date suggesting that recovery may be detectable beyond 2-year follow-up. General intellectual functioning has often been studied,

researchers usually reporting that verbal IQ recovers quickly and fairly completely, so that approximate premorbid level may be achieved by 6-12 months post-trauma. The time course of recovery for performance IQ and some of its subtests appears longer. Within the WAIS, the subtests which reflect continuing improvement for the longest period are digit span, digit symbol and picture arrangement. IQ tests are less sensitive indicators of cognitive recovery than attentional and memory tasks.

Most studies examining the social/emotional aspects of head injury have appeared within the last 10 years. A number of investigations report that return to work relates to initial severity of head injury. The available evidence for very severely-injured people is somewhat conflicting, varying between a 73% rate at 12 months in one study and a 50% rate, approximately, at 2 years and 7 years after injury in another. Cognitive status appears important in determining return to work.

High rates of social and emotional distress after head injury are reported by patients and their relatives. There is some evidence to suggest that social/emotional difficulties do not resolve within the first 12 months, and relatives report a significant frequency of personality disturbance as long as 7 years post-trauma.

CHAPTER 3

THE STUDY OF MEMORY SCANNING

This chapter focusses upon memory scanning research, the foundation for which is located within the information-processing literature. The large majority of studies in this literature utilise the senstive, accurate measures offered by reaction time indices.

3.1 INFORMATION PROCESSING: REACTION TIME STUDIES

For an appropriate response to be made to a stimulus:

- (a) A sense organ must detect a stimulus and transmit this information to the brain.
- (b) The stimulus must be identified.
- (c) Organisation/selection of the appropriate response must occur.
- (d) The response must be produced.

Welford (1980a) pointed out that the stages (a) and (d) require very little time, with stimulus identification and response selection taking longer. As he indicated, much experimental work is still required before a comprehensive RT model, accounting for all data, can be formulated. Hick (1952) proposed an information theory law which stated that under choice reaction time (RT) conditions a subject gains information at a constant rate.

He proposed the following formula:

Mean choice $RT = K \log (n+1)$

Where the number of possible stimuli is n, and K is a This formula represents Hick's constant. law. The resulting graph, plotted by Hick produced a straight line passing through the origin. Using logarithms to the base 2 (i.e. units of "bits"), then log 2(n+1) = 1when there is one stimulus and K provides the simple RT. The formula includes (n+1), rather than n. because on each stimulus presentation the subject also has to decide whether a stimulus has occurred at all, in addition to deciding which stimulus.

Some elaboration on Hick's Law has occurred. For example, the amount of information transmitted under choice RT conditions will be reduced if all stimuli are not equiprobable. The amount of information relating to uncertainty constitutes the sum of the information from of the number stimuli weighted according to the probability of each's occurrence: Unequal stimulus probabilities reduce uncertainty and this leads faster RTs. Predictable relationships in the sequence of stimulus presentations also reduces uncertainty and hence the amount of information transmitted. Errors. too, reduce the amount of information gained and erroneous RTs tend to be quicker. Welford (1980a) has provided a more detailed consideration of factors influencing the operation of Hick's Law, and included discussion of serial versus simultaneous processing models to describe choice RT.

An interesting application of Hick's Law was described by Crossman (1953), whose chosen task was the sorting of stimuli allowed consideration of playing cards. Such the RT performance of subjects according to, for example, the colour (red/black) which involves one "bit" of information, suit which involves two bits, or numbers (court cards removed) which involves approximately three bits of information. Crossman's results approximated Hick's Law well, as did those of Crossman and Szafran (1956) who examined the performance of subjects different age groups (20-40 years, 41-60 years, In a much later study using the same playing years). Skilbeck confirmed card stimuli, (1970) the applicability of Hick's Law using a sample of sports 20-50 years), whilst noting referees (age range However, this latter author did strong age effects. observe age-related slowing (affecting subjects 40-50 years age range) using a simple RT task.

McNicol and Stewart (1980) have provided a general review of the usefulness of RT experiments in the study of memory. In addition to outlining Sternberg's contribution (discussed in 3.2 below), these authors summarised a number of models used to describe retrieval from memory. McNicol and Stewart concluded that Sternberg's exhaustive serial scanning model fitted the data well for error-free RTs, though it was difficult to extend it to error-prone performance.

Welford (1980b) provided a useful review of stress, age and sex variables in relation to RT. Slowing in response latency has often been detected under central nervous system (CNS) fatigue (as opposed to peripheralmotor fatigue). Prolonged on-task testing tends to slowing, but also not only increasingly produce irregular performance. This yields a skewed distribution of RTs with variance rising in association with mean score. Welford (1980b) reported Bills' (1931) concept that this irregularity arises from intermittent "blocking", defined occasional, short as gaps otherwise fast RTs. The frequency of these blocks is said to rise when the task is prolonged. Welford indicated that response latency would be longer, and the probability of errors would rise, immediately prior to These the appearance of a block. features would disappear immediately following a block. Welford was unable to offer a good explanation for blocking. In considering stress, Welford included the concept of raising/lowering a subject's level of arousal, invoking the 'inverted-U hypothesis'. According to the latter, on any particular task performance will improve with rising arousal (from a low level) until an optimum is achieved. Increasing arousal level beyond this point becomes counterproductive and quality of performance deteriorates.

Welford (1980b) when reviewing age effects concluded that simple and choice RT begin to slow gradually between 20-50 years of age, and thereafter more rapidly. he pointed out, these findings relate more to CNS changes, rather than to the marginal effects produced by slower sense organ processing or nerve conduction speed, Welford also indicated that there or motor activation. evidence that older people monitor is good closely and are more cautious, more therefore attend less to new incoming stimuli: tend to trade-off speed for accuracy. Findings relation to sex are consistent across tasks and studies (Welford, 1980b) in noting faster RTs in males (except in the age group 10-14 years). Although the reason for this is unclear, it is presumed to be biological.

his review of the effects of impaired brain functioning upon response latency, Nettelbeck (1980) supported the suggestion that RT can be regarded as an index of brain efficiency, particularly as this variable is open to very precise measurement and is relatively by social/cultural factors. unaffected Nettelbeck concluded that "virtually all psychopathological conditions are accompanied by slower and more variable RT (whether simple or choice tasks are employed), and irrespective of the modality of either stimulation or response. Furthermore, the extent of slowing covaries with clinical estimates of the condition's severity" indicated that people with a mental (p.356). Не handicap show slower RTs which are more variable. variability takes the form of an increased positive skew distribution, although in addition the of the RT quickest RTs achieved by these subjects are poorer than those noted in undamaged people.

These features of generally slower and more variable performance are consistently found in studies comparing brain-damaged people with normal subjects, with severity of damage being a good index of the degree of disturbance in RT performance. These conclusions have been shown to hold in the case of localised cerebral lesions, epilepsy, and Parkinsonism. Frontal cerebral

damage seems more important in determining the extent of the RT slowing (Nettelbeck, 1980). In their study on localised hemispheric lesions. Dee and Van Allen (1973) employed an RT paradigm involving 1-4 stimuli. Their results obeyed Hick's Law in that mean RT was a linear function of the number of stimulus possibilities, and they also noted that left hemisphere damage produced steeper RT slopes (and more errors) than was seen in right hemisphere damaged subjects and normals.

An interesting study was that carried out by Miller (1970) using simple and choice (2-4-8 items) RT with head-injured subjects, all of whom were severely injured 7+ days). His sample only involved 5 subjects. with a further 5 normal control subjects also tested. However, his results demonstrated slower RTs in the patient group (p<.05), the discrepancy in performance being greater with increasing information Plots for both groups showed load (p<.001). linearity, with very similar zero intercepts. The latter suggests that the RT findings do not stem from motor difficulties between the groups, and Miller drew a parallel between the adverse effects upon CNS functioning of normal ageing and of head injury.

In a subsequent experiment, Miller and Kruzat (1981) tested 2 groups of head-injured patients, each with 15 In the "severe" group, the median PTA was 9 subjects. days and in the "mild" it was 20 minutes. Also studied was a control group of 15 members of the hospital staff. The task employed was a simple card-sorting procedure, consisting of 20 cards containing either the letter 'A', In one condition only these letters were depicted on the cards, whereas in three other packs additional irrelevant letters (1, 4 or 8) were also included. The subjects task was to sort each pack into two piles (A, B) as quickly as possible. Miller and Kruzat's results showed that the inclusion of the irrelevant information had a major effect upon the RTs all subjects (p<.001), and severely head-injured subjects generally produced slower RTs than either of the mild or control subjects (p<.001). Interestingly, Miller and Kruzat did not detect the significant interaction which would have been expected if headinjured subjects were finding it difficult to cope with the irrelevent information because of poor selective attention.

Finally, mention should be made of the work of Van Zomeren (1981). His detailed study of RT and attention after head injury included one experiment in which 57

head-injured patients were followed for up to 2 years post-injury. Van Zomeren's work is, therefore, rare in injury research, in that it both employed head experimental psychology approach (study of RT) and included repeat testing of subjects for a long period The results of an ANOVA, with after head injury. repeated measures, based upon approximately two-thirds his sample (between 5 and 24 months post injury) indicated significant effects on severity of head injury (mild, moderate, severe), information load, and time (all p<.01). Significant interaction terms also reflectd different recovery times to asymptote according severity of head injury, and the factor asymptote was delayed according to increasing information load.

3.2 STERNBERG'S PARADIGM

As indicated in the last section, a traditional idea in the study of reaction times (RT) is that the time between the presentation of a stimulus and the production of the relevant response is taken up by a train of processes (mental operations). These processes are presumed to be non-overlapping, and their summation determines the RT. As Sternberg (1969a) pointed out, if

it were possible to work out the component times of each of these processes this would then answer key questions about the mental operations that they represent. Donders (1868) was the first to use RT measures to study stages in information processing. He employed a subtraction method to separate out RT components; for example we might presume that time between stimulus and response involves:

- (a) Stimulus detection
- (b) Stimulus identification
- (c) Response organisation

If so, a useful experiment to conduct is one which has the following two conditions: In the first there is just one stimulus and one response, and in the second there are multiple stimuli and multiple responses. Donders considered that differences in the total RTs between these two conditions would reflect the duration of stages (b) and (c).

The above approach was originally very popular, although early in this century two specific criticisms were advanced. First, that differences in mean RTs between subjects, and between experiments, were often large. In retrospect, these differences may have arisen in part

instructions because of differences in task and differences between the particular tasks employed, which failed to control the processing strategy employed by subjects. Second. subjects' reports suggested that the introduction of an additional stage into a task might also change the processing in other stages; for example, in stimulus identification processing could influence response organisation too. If true, invalidate the assumption that RTsubtraction methodology can provide clear evidence on the stages of information processing. These two criticisms reduced the number of RT "fractionation" studies for some time, although interest in RT per se has grown again over the last 20 years. Sternberg (1969b) claimed that modern experimental control and analysis procedures make to overcome these earlier criticisms. possible Sternberg's own work has focussed on memory search inretrieval when learning processes involved retention are essentially perfect.

Sternberg's method a small number of Ιn question memorised. the subject is then asked a these items, the subject responds referring to quickly as possible, and response latency is measured. One goal is error-free performance. RT is investigated according to the question asked, the number of items in the memorised set, and other variables. In a Sternberg study, the memorised list constitutes the "positive set", the remaining items in the same set (same category) form the "negative set". For example, if the experiment involves digits and the subject is asked to memorise the items '2-5-6' (positive set), then the numbers 0,1,3,4,7,8,9 comprise the negative set. Within this item-recognition paradigm a number of different procedures are possible. With regard to the positive set, the items contained may be "fixed" or "variable". In the example above, if the digits 2-5-6 constituted the positive set on every trial, they would represent the fixed set. However, if the three digits chosen to form a positive set changed trial by trial, a varied-set procedure was being employed. In the typical experiment subjects are asked to hold the positive set in memory (e.g. '2-5-6'), then a stimulus (probe) is presented. If the target belongs to the positive set (eg '5') then the subject presses a button as quickly as possible. However, if the target is a negative set item (eg '8') then the subject presses another button, again as fast as possible.

Sternberg (1969b) reported some typical data for itemrecognition study. He concluded that:

- 1. A linear relationship exists between RT and positive set size.
- 2. The zero intercept for the positive set RT is approximately 400 msec.
- 3. Positive and negative RTs increase at about the same rate with increasing information load (approximately 40 msec per item in memory).
- 4. By manipulating the relative frequency of presenting positive and negative items the relationship between the two mean RTs can be altered (but not the slopes of their plots).

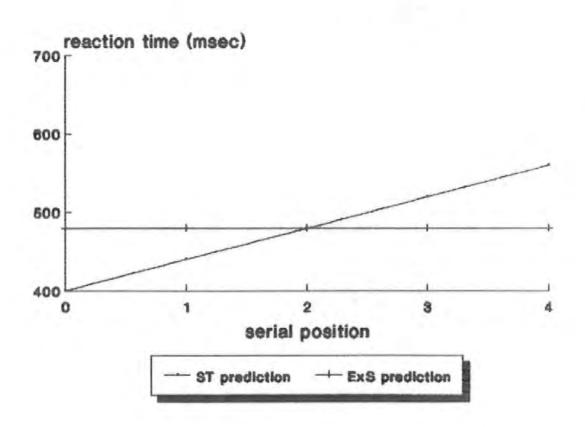
Sternberg (1969b) also discussed the process by which items in memory are presumed to be searched in a serial manner when subjects are asked to attempt a match with the probe stimulus. In searching, subjects may scan the items, one-by-one, until they find a match (if exists), and then stop (called a self-terminating serial search). If no match exists (i.e. the probe belongs to the negative set) all positive items will be searched. Alternatively, subjects may compare the target with all items successively and only then produce a response (exhaustive serial search); the response will positive if a match has been found, and negative if not. The first strategy is not necessarily the best (ie, the fastest) if, as Sternberg (1969b) arqued, a selfterminating search might involve a check for a 'match' after each item is scanned, whereas an exhaustive search might need this check only after all items have been scanned.

Although both search strategies assume a rising response latency with increasing positive set size, they predict different findings under certain conditions. example, according to the exhaustive search hypothesis, the rate of RT slowing with increasing set size is the same for the positive and negative responses (because all the items are scanned before a positive or negative response is produced): the slope of positive RT functions is, therefore, parallel. negative Ιn contrast, the self-terminating search hypothesis predicts that the two slopes will diverge as size of positive set rises (because, on average, a match with the probe is obtained half-way through scanning the list when the target belongs to the positive set); Response latency for positive items, therefore, rises at half the rate of that for negative items.

Another difference between these two search hypotheses relates to the serial position of positive items. The prediction from exhaustive search (ExS) theory is that the serial position of the positive set items is

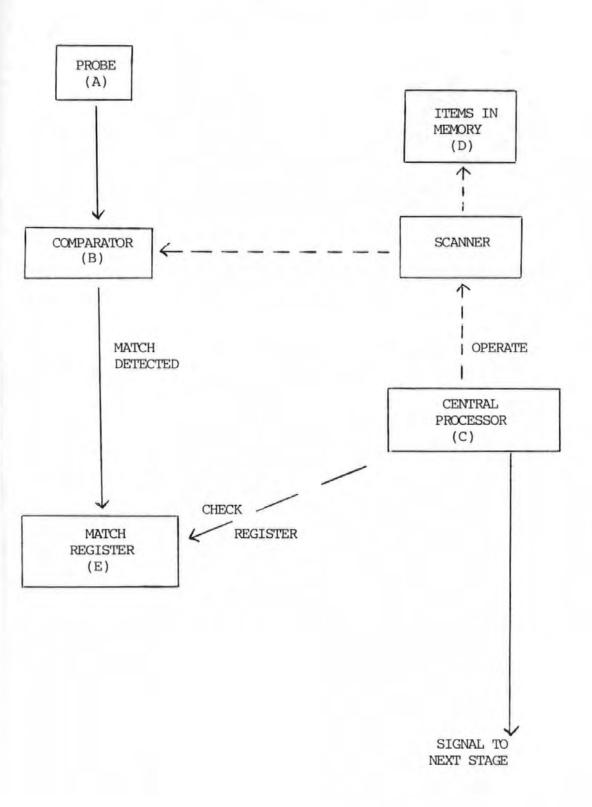
immaterial to the observed RT, as all items are scanned before responding. With a self-terminating (ST) search framework, however, if scanning commences with item 1 and subsequent items are processed serially, then the RT noted increases linearly according to the serial position of the target match (figure 3.1). Also, the latter model will hold irrespective of positive set size. Only a self-terminating search strategy which scanned items randomly would produce the flat RT curve predicted by the exhaustive scan hypothesis.

FIGURE 3.1: RT FOR AN ITEM ACCORDING TO SERIAL POSITION



The results reported by Sternberg (1969b) using small samples of subjects (n=6-8) supported the ExS model, and pointed to people's ability to scan items at high-speed (a rate of 25-30 digits per second). Sternberg (1969b) also reviewed some of the evidence suggesting serial scanning of information in memory is not material-specific (ie, the results discussed above in relation to digit item recognition were not obtained because of the sequentially-related nature of material). He concluded that serial high-speed scanning memory is not dependent upon material being very familiar to subjects. Serial search appears to be demonstrated even when alternative "association" strategies, such as shared physical characteristics of positive of the set items, orsemantic relationships between these items offered alternative search mechanisms.

As indicated above, ExS on average involves more comparisons than ST searching, which might argue against it's validity on the grounds of inefficiency. It appears maladaptive to continue attempts at matching after a matched item has been located. However, if the cognitive processing involved in memory searching is that depicted in figure 3.2, the exhaustive procedure might be more efficient.



The model envisages a representation of the stimulus or probe (A) being introduced into a comparator (B). The central processor (C) uses a scanner to examine positive items in memory (D) and compares each with that in the comparator, one by one. If a match is detected, is sent to the match register (E). a signal The most important concept in this system is that the central processor cannot both drive the scanner and check the match register simultaneously, and alternating between these operations takes time. Sternberg (1969b) argued that if the switching time is relatively long compared with scanning rate (25-30 items/sec.), and size of the positive set is small, then ExS may be quicker (ie, more efficient) because it involves checking the match register only once.

Sternberg pointed out that one drawback of this proposed system is that probably little information would still be available after ExS without further scanning of the items in memory. For example, information regarding the position within the list of the matched item might not be available. Sternberg predicted that this kind of information was not preserved by the high-speed ES process, and asking subjects to provide it would require them to adopt an alternative strategy which would be slower, and might be self-terminating.

Sternberg reported a small-sample study to test these predictions, noting that scanning was indeed slower when subjects were asked only to report the serial position of the matched item (all test stimuli belonged to the positive set). Instead of about 25 items/sec., results obtained suggested a scanning Plotting an RT serial position approximately 4/sec. graph also demonstrated that an ST search was employed by subjects, although differences between subjects in terms of whether they began to search at item 1 in the list, or randomly, were observed. Sternberg noted high error rates with longer memory lists (approximately 5% items, 10% with 6 items, and 25% with 7 items). He questioned whether this error rate might differential learning level amongst lists of different length, and whether they could be partly responsible for slowed RT.

Additional experimentation, designed to improve learning level of the memory list via repetition, supported the first hypothesis (errors dropped by a factor of 3), although RT was not faster as a result. As experiment involved recall (of the item's list position) recognition, Sternberg than just conducted a further experiment to ensure that the findings did not arise because of differences in the

response mechanism. To achieve this he employed a visual display of 3-6 digits, presented sequentially and subsequently displayed a pair of digits together from the display as the test stimulus. Subjects were asked to decide whether the two digits had originally been presented in the same left-right order. involved two levers (representing "same" and response Although this was a recognition task no "different"). single item matching was involved. The results obtained this context-recall experiment were from linear. supporting the use of scanning process, with the additional linear results according to serial position of the stimulus pair within the memory list suggesting an ST strategy.

As indicated above, at first sight ExS might appear less efficient than an ST search procedure. However, if one accepts that the rate of scanning is very rapid (gauged by Sternberg to be 25 items/sec. or faster), and that to stop the search process after each item is examined to check for a match adds significant time to the search process, then ExS can appear the best strategy: all items are scanned without "pause" and only then is a check for match carried out. Using this view of ST versus ExS memory searching, the relation between rate of scanning and individual item matching time is very

If scanning were a slow process then the important. for matching might not item-by-item check add "significant" amount of time to the search time, and memory searching under these circumstances ST could be more efficient than an exhaustive approach. Sternberg (1975) re-examined the findings of earlier research by other workers, categorising results of their subjects into "exhaustive scanners" (RT slope ratio of positive and negative plots approaching 1.0) and "selfterminator scanners" (RT slope ratio approximately 0.5). The former had scanning rates which were 50%-89% faster, so supporting this argument for the relative efficiency of ExS when scanning rate is rapid.

3.3 BRIEF REVIEW OF THE GENERAL LITERATURE

In his major review of memory scanning, Sternberg (1975) again outlined some of the arguments for employing RT methods when researching memory. In particular, he pointed out that the traditional methods of studying memory by examining its failures (errors) involves the theoretical quagmire of learning versus retention versus retrieval processes. The examination of memory via determination of processes' times in paradigms which yield very low error rates avoids some of these

difficulties by concentrating upon information held in short- or long-term memory. Also, he pointed out that the findings that RT functions are approximately linear, and show similar positive and negative response slopes. have been demonstrated by a wide variety of researchers using different stimulus material (eq, visual auditory digits, shapes, facial photographs, colours). Altering the relative probability with which a positive or negative set probe appears does not change response characteristics, although the RT intercepts are different (the difference between the negative zero intercept and the positive increases with the increasing relative frequency of the positive stimulus). Sternberg concluded that the available evidence suggested that error rates up to approximately 10% do not affect response characteristics under speed/accuracy trade-off instructions.

Results from various age groups and diagnostic samples tend to present the same essential response characteristics, although older subjects and subjects with a mental handicap (reviewed below) show steep response slopes and higher intercepts. The latter is observed in young children (Harris and Fleer, 1974), although the slopes are very similar to those of young adults. Findings from studies investigating practice

effects (reviewed by Sternberg, 1975) are reassuring from a clinical testing point of view: whilst RT functions flatten with extended practice on a fixed set of items over a number of days, if sets are changed "from session to session ... and stimuli are not consistently assigned to particular responses, extended practice seems to have virtually no effect on the phenomenon" (Sternberg, 1975; p.9).

However, when the positive set consists of 2 subsets of items, and a subject is not alerted to their presence, RT slope is reduced, but only by 25% (a 50% reduction would be expected if search was restricted to only the relevant subset items). Two types of explanation have been advanced to account for this finding (Sternberg, 1975). The first suggests that irrelevant items are searched at twice the rate of relevant ones. The second hypothesis is that there are 2 storage "bins" for the 2 categories (subsets) of positive items. Access to these bins is not selective, and items in each are searched exhaustively at the normal rate. However, when the bins containing the relevant item is entered this 'recognised' and the search ends after the contents of this bin have been scanned. The latter process would precisely explain the 25% slope reduction, because the irrelevant bin has a probability of 0.5 of being searched, and the relevant bin a probability of 1.0. Thus irrelevant items add, on average, half as much time as relevant items to the search process. The second hypothesis appears intuitively plausible, and very neatly explains the 25% slope reduction observed. Support for this explanation is also provided by findings which show that this 25% reduction effect disappears if the 2 subsets of items are intermingled in the positive set (ie, not obviously categorised into 2 separate bins).

There have been occasional attempts to link RT memory scanning paradigms to more traditional concepts memory functioning, including those employed in clinical For example, Cavanagh (1972) argued that as both response latency measures and their associated errors suggested that recall and recognition processes may have a common memory (Freund, Brelsford & Atkinson, 1969; Sternberg, 1969a), then scanning rate and immediate span may be related and their relationship could offer some insight into this memory system. published work on adult subjects, Cavanagh noted that greater the memory span for a particular type of stimulus material (eg, words, digits, shapes), the faster was the scanning rate reported in studies using that type of material. Cavanagh pointed out that only

group data were published and it would be useful to gain within-subject results. Cavanagh's 'size' hypothesis suggests that short-term memory offers a fixed "space" can hold only a limited number of items. stimulus recognition requires feature-testing the stored target, then the processing time per item is number of features proportional to the per Similarly, on average, the greater number of features per item to be tested, the fewer stimuli will be needed to fill the available memory space. Processing rate is, therefore, related to the reciprocal of memory span. suggested that Cavanagh's results Sternberg (1975)should be confirmed in studies designed to investigate memory scanning and memory span in the same subjects.

Okada (1971) conducted Burrows and an experiment investigate the conditions under designed to which position effects in high speed memory scanning observed. They hypothesised that might be conditions of fast presentation (inter-trial interval of seconds, with .5 second warning signal) serial position effects were more likely to be observed than under slow (inter-trial interval 1.2 seconds, warning signal 1.2 seconds) presentations. Their experiment involved 6 University subjects who were investigated under both slow and fast conditions. Their results

produced similar error rates under the two conditions, similar linear functions for both positive negative slopes. They also noted that serial position effects were observed (increasing RT with position, except for the final item), though under the slow condition there was much weaker evidence for position effect. In both conditions fast RTs serial were observed for the final item in the positive set, suggesting that a recency phenomenon may have been Burrows and Okada argued that it is still operating. possible to have an exhaustive scan and note serial positioning effects if it is assumed that the total scan can be completed more rapidly if the target is placed in favourable serial position. This hypothesis seems impossible to disprove, and also implies unequal distribution of memory capacity across items. The latter may be plausible given that serial position effects have been described in other areas of memory However. Okada offered research. Burrows and explanation as to why the fast condition should produce a more noticeable serial position effect.

Finally, Biederman & Webb Stacy (1974) investigated set size and stimulus probability, pointing out that studies often confound set size with the probability of an item's occurrence. It is thus it difficult to decide,

under these circumstances, whether increasing RT relates to increasing set size per se, or is observed as a function of reduced probability of an item as set size increases. Biederman and Webb Stacy manipulated the probability of occurrence of positive set items, making this explicit to subjects. Their results did not support the hypothesis that increasing RT resulted from a reduced probability (thereby supporting Sternberg's hypothesis), nor did they provide strong evidence of an interaction between set size and probability.

3.4 CLINICALLY-RELEVANT STUDIES

Age is often an important variable in clinical research. number of studies have addressed this factor performance, though few have relation to RT published which directly relate to Sternberg's Paradigm. One such study was that of Anders, Fozard and Lillyquist (1972), who investigated the memory scanning performance of subjects whose ages ranged from 20-68 years. These authors employed a varied-set procedure, using digits 1-9 and positive set sizes of 1, 3, 5 Positive negative set stimulus probes and equiprobable. The results for the 3 age groups (young. mean age 20 years; middle, mean age 38 years; old, mean

68 years) all suggested that subjects employed a age serial search procedure, and also supported Sternberg's hypothesis that the process is exhaustive by showing similar response latency slopes for positive negative items. Significant (p<.05) age differences were noted in terms of rate of memory scanning, younger subjects' performances being superior to those noted in the other two groups. Older subjects showed significantly higher (p<.05) intercepts than either young or middle age subjects. Errors were rare for the three groups, averaging 0.6%-1.4%.

Similar, though not identical, findings were noted by Eriksen, Hamlin and Daye (1973) using positive sets of 1, 2 or 4 digits. These workers observed significant age effects (p<.01) in terms of RT, positive set size, and positive versus negative latencies, as well between age and set size. latter interaction The finding was produced by the 50-55 year subjects (the others being 20-25 and 35-45 years), whose RTs were generally slower and were differentially penalised (steeper slope) by increasing positive set size. As in the Anders et al study, no significant slope differences between positive and negative RT plots were observed. Erikson's findings also replicated Sternberg's results to support the serial, ExS hypothesis, and confirmed the Anders finding of a higher intercept for older subjects.

of studies have investigated memory A small number scanning in 'clinical' samples". For example, Pharr and (1980) examined the performances of chronic schizophrenic patients, acute schizophrenic patients and They found that the mean RT of the normal individuals. patients was longer than that of the chronic sample, which was in turn longer than the normal significant (p<.05) subjects (p<.05). Α interaction between group and set size was also noted, with the RT slopes for chronic and acute patients being larger than that for normals. errors were low (1%-4%) and Mean tended to occur trials with longer response on latencies.

Stuss. Kates. Poirer, Hylton, Humphreys, Keene Lafleche (1987) examined the memory scanning performance patients with the muscle-wasting disease Myotonic This is a multisystemic disorder, and in Dystrophy. some patients cerebral functioning is affected. and his colleagues noted support for Sternberg's ExS hypothesis in both patients and normal controls, though no significant differences between the 2 groups in terms of speed of memory scanning or slope were observed.

Warren, Hubbard and Knox (1977) compared the scanning performances of normal individuals with those of people Their research was carried out because 3 with aphasia. earlier studies had divided 2:1 in terms of supporting exhaustive versus self-terminating memory scanning (all previous studies (Carson, Carson and 1968; Tikofsky, 1971; Swinney and Taylor, 1971) observed slower scanning in people with aphasia. Warren and his co-workers, too, observed slower RTs in the latter 11.5 items per (average scan rate second), aphasic subjects also showing higher intercepts and steeper RT slopes.

Warren et al (1977) found the expected linear plots for RT and set size and flat serial position plots. error rates were 2.7% and 7.4% for the normal sample and the aphasic individuals, respectively. Out of the 10 aphasics tested, 6 had visual memory spans smaller than the largest positive set size employed in the experiment and were, therefore, engaging in supra-span scanning on trials where the set sizes were larger than immediate span. For these subjects, memory scanning time per item for positive (59 msec) and negative items (110 msec) yielded a negative plot almost twice as steep the positive, providing some evidence that these subjects may have been using a self-terminating strategy. The equivalent values for the 4 aphasic people with immediate memory spans of 6+ averaged 41.7 msec and 41.5 msec, respectively. However, an alternative explanation for these findings (Murdock, 1971) is that with supra-span scanning subjects tend to re-check the negative items.

Also of importance is a check for recency effects (Warren et al, 1977), given that when the retention interval between the presentation of a positive variedset and the probe stimulus is 1 second, or less, fast responses can occur if the target is the last positive item. This recency effect is more marked with supraspan searching (Corballis and Miller, 1973). Warren et al (1977) used a 3-second retention interval to avoid this confounding problem, and noted no recency phenomenon. Swinney and Taylor (1971) used a mean retention interval of .7 seconds, and if they employed supra-span searching then this may account for their findings; in fact, these authors did not check their subjects' span, and so it is impossible to be sure of the correct interpretation.

The applicability of the serial exhaustive model to the memory scanning performances of people with a mental handicap was investigated by Harris and Fleer (1974).

These workers compared the results of normal individuals with two samples of subjects with a familial handicap (pre-natal, peri-natal) and a sample of people who had suffered anoxic encephalopathy. Their design employed in set sizes 1-4, both positive and items being equiprobable. Subjects were tested sessions, 4 months apart. The results of Harris and Fleer demonstrated that people with a mental handicap made more errors at the first testing session, but not Response latencies on negative items were the second. significantly longer (p<.01), though both positive and negative plots were linear with parallel slopes, and no interactions between groups and set size were observed. The RT slopes for the normal subjects were significantly smaller (p<.01) than for the two groups of subjects familial handicap, with the steeper slope showing a being seen in the anoxic encephalopathic (significantly different to the other handicapped groups: p<.01).

Overall, therefore, Harris and Fleer's results indicated that the serial exhaustive model fits the memory scanning performances of people with a mental handicap. The parallel and linear plots of the positive and negative functions relating to set size, and the lack of serial position effects upon RT for all samples involved

the study, supports Sternberg's hypothesis. in Ιt appears that people with a mental handicap process information in the same qualitative way as normal individuals, though the differences RT in slopes suggests that this processing was less efficient.

Kaszniak, Klawans and Garron (1980) observed Wilson, that patients with Parkinson's disease were slower than age-matched control subjects in scanning the contents of their memory, noting also a steeper slope with increasing set size in the patient sample. Hart and Kwentus (1987), investigating elderly depressed found that this group performed more slowly patients, than control subjects, although slope weights were virtually identical. In the same experiment these authors discussed the results from 3 patients with Friedreich's Ataxia whose memory-scanning mean RTs were not only slower than the other 2 groups, but also showed much higher slope weights.

A very recent study by Rao, St Aubin-Faubert and Leo (1989) employed memory scanning with Multiple Sclerosis patients, using fixed, positive set sizes of 1, 2, or 4 digits. Their findings supported Sternberg's ExS hypothesis. These authors noted not only a higher zero intercept (expected on the basis of motor symptoms), but

also a significantly higher slope factor (p<.02) for the patients compared with normal age-matched controls. Rao et al (1989) also found a significant correlation (.36; p<.05) between slope value and length of neurological symptoms in patients. Examination of patient subgroup data on the basis of taking psychoactive medication or, not, provided only negligible results.

Stokx and Gaillard (1986) attempted to study the stages in Sternberg's information processing model, using headinjured patients more than 2 years after their trauma. Their experiment was linked with driving skills examine the power of RT results to predict driving ability. Although patients were generally slower than control subjects. Stokx and Gaillard's results did not identify any one stage and its experimental manipulation (Stimulus-Response compatibility and time uncertainty, Stimulus encoding and visual field effects, Memory set size and Response-Stimulus interval, Response-Stimulus distraction were examined) interval and as being differentially vulnerable to head injury. There was a .69 correlation between RT and driving test data.

Shum, McFarland, Bain, and Humphreys (1990) also researched the effects of head injury upon attentional processes via an information processing stage analysis.

These authors criticised Stokx and Gaillard's study on grounds that the stages were investigated in the experiments, rather than together, separate therefore could not be verified as being additive (as required by Sternberg model). Shum and his colleagues examined a different pool of head-injured subjects to Stokx and Gaillard, including a severely-injured subgroup tested within 1 year of trauma, a severelyinjured subgroup tested at least 1 year after trauma, and a mildly-injured subgroup tested within 1 year of their injury. Shum et al's results indicated that the different head injury subgroups showed deficits different information processing stages: severelyinjured subjects tested at 1 year, or later, showed an impairment only in terms of response selection and response execution stages, whereas severely head-injured patients tested within 1 year of trauma showed a deficit the these stages and also at stage of stimulus indentification. The mildly head-injured subjects any information processing showed no impairment at stage.

3.5 SUMMARY

Findings from the use of RTs in information processing research have generally approximated. Hick's Law. The slowing effect of age upon RT appears gradual until the sixth decade, although sex is a major determinant of RT for nearly all age ranges. The critical factor in RT performance differences between normals and patient groups appears to be CNS functioning.

In clinical samples, the findings consistently reflect slower and more variable RT scores, irrespective of specific diagnosis, the extent of this abnormality correlating with severity of condition.

Sternberg's paradigm examines memory search procedures. In the typical experiment, a subject memorises a number of items, termed the positive set. Remaining items in the same category constitute the negative set. A probe stimulus is presented and the subject has to respond as quickly as possible to indicate whether the probe matches a positive, or a negative, set item. Findings which generally hold include a linear relationship between RT and positive set size, that the zero intercept for positive set RT is about 400 msec, and that the rise in RT of approximately 40 msec per item

applies to both positive and negative trials. Sternberg viewed these findings as supporting his exhaustive scanning (ExS) hypothesis of memory searching, although number of studies have observed results small suggesting that under certain conditions subjects will scan the contents of their memory using a self-The lack of evidence for terminating (ST) strategy. serial position effects in memory scanning argues for the ExS hypothesis. Unless extended, daily, testing with a fixed set of items is undertaken, practice effects are not noted.

A small number of researchers have carried out studies of "clinical" relevance using Sternberg's paradigm. For example, Cavanagh (1972) commented on the fact scanning rate appears to correlate with immediate memory span, and the effects of age upon memory search rate have been investigated by a number of authors. Pharr Connor (1980) noted slowed scanning and RTs in schizophrenic subjects, with these patients showing a penalty with increasing memory load. findings have been observed in aphasic patients, those with Friedreich's Ataxia, those with Multiple Sclerosis, and in people with a mental handicap. The latter group do not provide evidence of an interaction between RT and set size. Only patients with acquired brain damage

appear to show such an interaction.

Memory scanning RT findings appear relatively stable across a range of studies from experimental psychology. Sternberg's paradigm offers a potentially-sensitive method for detecting changes in cognitive functioning following acquired brain damage. Interesting questions relating to the memory-scanning strategy adopted by head-injured subjects and differential effects according to severity of damage, can be investigated by employing the paradigm.

CHAPTER 4

PILOT STUDY: INFORMATION PROCESSING AND HEAD INJURY

4.1 AIMS

Before carrying out the main study, it was thought to conduct pilot investigation, desirable a studies of information processing experimental the abilities of head-injured people are available. A major aim of the pilot research was to check whether any constraints would apply to the design of the main study, for example in terms of an inability to respond by severely-damaged subjects soon after injury. Another aim was to confirm suggestions from some earlier investigations that experimental tasks can be sensitive cognitive recovery following head injury. Specifically, it was hypothesised that:

- Severely head-injured subjects would show slower and more variable RTs than those with a mild/moderate injury.
- Increasing information load would differentially penalise the RTs of 'severe' subjects.
- 3. The addition of irrelevant information would differentially adversely affect the RTs of 'severe' subjects.
- 4. Subjects would show recovery in RT over time.

4.2 SUBJECTS

Subjects were patients admitted to the Regional Neurological Centre, Newcastle General Hospital with a diagnosis of head injury. Given the pressure unpredictable nature of acute clinical work Neuropsychology Department in a Teaching Health District, subjects constituted a random sample of headinjured patients admitted to the Centre, but were not consecutive attenders: they were entered into the study as time allowed, over a 6-month period.

The target sample size for attendance at all 3 testing sessions was 10. No geographical exclusion criterion (ie, place of residence within the Northern region) was operated, and to try to allow for drop-out over the 6 months study period it was planned to recruit 20 subjects into the study. These would comprise 10 mild/moderate head-injury patients (PTA=<24 hours) and 10 patients with a severe head injury (PTA>24 hours). In the event, 5 subjects from the severe group and 3 from the mild/moderate group failed to keep one or more of their 3 follow-up appointments, leaving sub-samples of 5 and 7 respectively (appendix A1.1). Of these 12, 4 were resident in Newcastle upon Tyne.

4.3 PROCEDURE

Subjects identified from Regional Neurological Centre notes were tested approximately 1, 3 and 6 months post The information-processing task employed head injury. similar to Rabbit's (1964) procedure selected by Miller (1970). This task was chosen because it involves different levels of information load (1, 2, bits) and includes a varying number of irrelevant stimuli (0, 4, 8 elements). It was thought to be a good test of the 'robustness' of RT measures obtained from severely head-injured subjects close to their trauma. The design, therefore, involved a 3-factor experiment (Kirk, 1982) involving severity of head injury (severe, mild/moderate), information load (1, 2, 3 bits), and irrelevant information (0, 4, 8 elements), with repeated measures (1, 3, 6 months post head injury).

All subjects were tested in the Neuropsychology Department of the Regional Neurological Centre. Stimuli were 1-cm high letters stencilled on to Tachistoscope cards using black fibre tip pen. The same fixed order of stimulus presentation was used for each subject, in a quasi-random sequence. The order was manipulated so that no particular stimulus could appear on more than 4 successive trials, to avoid the risk of subjects'

hypothesising unequal stimulus probabilities. Subjects were seated comfortably in front of the tachistoscope on a height-adjustable chair. Stimuli were presented via an Electronic Developments Tachistoscope and responses were recorded using a plunger response device and millisecond timer.

The procedure was that each stimulus was preceded by an auditory warning, the stimuli appearing approximately 2 seconds later. Subjects were under instruction to locate a target letter and release the plunger device as soon as possible, and then to verbally report which target stimulus had been presented. The next trial then The experiment was carried out in three blocks of 30 trials each, according to the information load of one bit (letters 'A', 'B'), two bits ('A'-'D'), or 3 bits ('A'-'H'). Each block contained 12 trials each of the 3 irrelevant information conditions: irrelevant information took the form of non-target presented simultaneously with the target stimulus. trials where an error response was produced the RT was disregarded and an additional trial was added to the end of the block. The order of presentation of the 3 blocks was randomised across subjects, with each subject receiving the same sequence over the 3 testing sessions.

4.4 RESULTS

4.4.1 Clinical Background

Background clinical and other data on subjects are provided in appendix A1.

Of the initial 20 subjects, 8 failed to attend all 3 follow-up assessments over the 6-month period of the experiment, leaving data on 12 for analysis. Given that subjects might live anywhere in the Northern region, this drop-out rate may primarily reflect geographical problems in maintaining the sample.

The age range of subjects was 17-54 years (table A1.1). Subjects in the mild group were significantly younger (t=2.305; p<.05), although the explanation of this finding is unclear. The 2:1 sex ratio in favour of males is typical of that reported for head injury. The cause of head injury was RTA for 75% of subjects, which is higher than the approximately 50% often quoted.

The mean length of PTA for the mild/moderate (M/M) group was approximately 2 hours, and all but one of the subjects were unconscious for 'minutes', at most (appendix table A1.2). For severely head-injured

subjects (S) the mean PTA was 13 days with mean duration of unconsciousness being 7 days. No skull fractures were confirmed in the M/M group, although 2 were noted (1 depressed) in the S. Haematoma were observed in 3 S subjects (2 subdural, 1 subarachnoid), and 1 subdural haematoma in the M/M group.

4.4.2 Reaction Time Data

Appendix A2 provides the raw data for each subject in terms of mean, standard deviation and median Because of the typically skewed nature of RT data, statistical analysis concentrated upon median and scores (Hays, 1963; Dunn & Master, 1982). A 3-factor with repeated measures (Kirk, 1982). was performed on median RTs a summary of which is shown in table 4.1. As the table shows, there were highly significant main effects (p<.001) from head severity, information load, and presence of irrelevant information. A similarly significant effect was also noted from the passage of time, and its interactions with severity and irrelevant information. Interactions of irrelevant information with severity and information load also attained this level of significance. interaction between severity and information load, whilst being weaker, was also significant (p<.05) and

TABLE 4.1: ANOVA SUMMARY, MEDIAN RTs

So	urce	df	MS	F Ratio	Siq.Level
1	A: SEVERITY	1	14085415	80.17	***
2	C: IRREL.INFO	2	4929195	28.06	***
3	D: INFO.LOAD	2	5840056	33.24	***
4	AC	2	12160936	69.22	***
5	AD	2	550068	3.13	*
6	CD	4	6636809	37.77	***
7	ACD	4	108302	0.62	n.s.
8	SWG	90	175698		
9	B: REP.MEASUR	. 2	3188496	36.30	***
10	AB	2	958462	10.91	***
11	BC	4	5639527	64.20	***
12	BD	4	109673	1.25	n.s.
13	ABC	4	41350	0.47	n.s.
14	ABD	4	136161	1.55	n.s.
15	BCD	8	68954	0.73	n.s
16	ABCD	8	1157215	13.17	***
17	B x SWG	180	87849		

^{*=}p<.05; ***=p<.001;

the 4-way interaction was highly significant (p<.001). Figure 4.1a plots the recovery in RT over time, demonstrating the significant interaction (AB) between severity of head injury and time post-injury.

FIGURE 4.1a: RECOVERY IN MEDIAN RT & TIME SINCE HEAD INJURY, BY SEVERITY GROUP

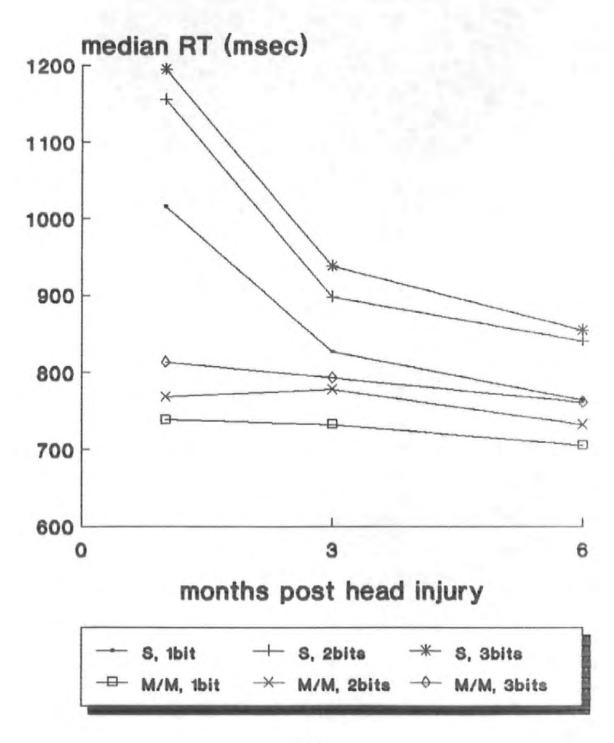


FIGURE 4.1b: EFFECTS OF IRRELEVANT INFORMATION UPON MEDIAN RT, BY SEVERITY GROUP

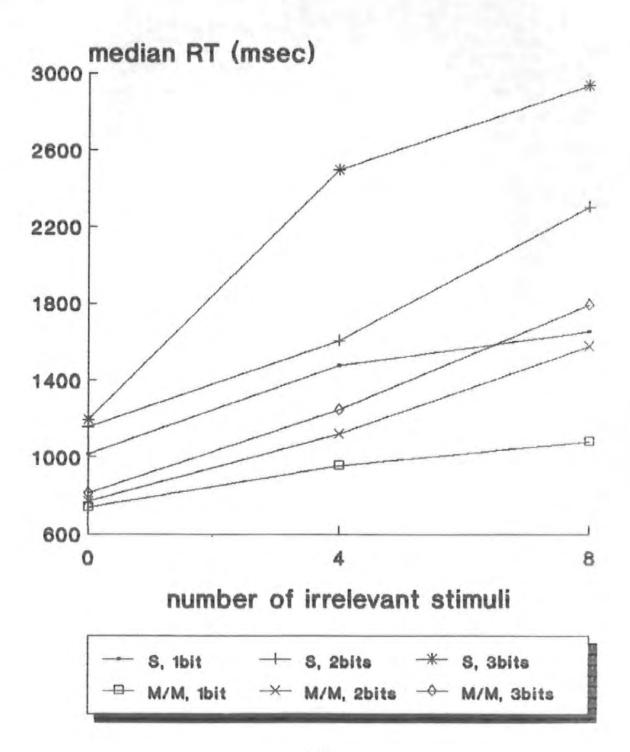


Figure 4.1b graphs the interaction between amount of irrelevant information and median RT for each severity group at 1-month post-trauma. The figure shows that a high level of irrelevant information (8 items) slowed RT in both the M/M group and the S group when processing either 2 or 3 bits of relevant information. In addition, in the '3 bit' condition the S group showed slowing with only 4 irrelevant elements.

Table 4.2 provides the median RT data for the M/M and S groups in each of the experimental conditions, and ttest comparisons, performed following ANOVA (in table 4.1). Given the directional nature of the hypothesised differences in RT according to severity of head injury, t-test values utilised 1-tailed comparisons. table indicates, all of the mild/moderate (M/M) versus severe (S) comparisons were significant at the 1-month follow-up. These 1-month comparisons also showed a generally higher significance level with greater information-processing load (3 bits). Comparisons at 3 months and 6 months after head injury continued to show significant differences, though often at a lower level. It should be borne in mind, however, that the risk of obtaining a significant finding by chance rises using multiple t-tests, there being 9 M/M versus S comparisons examined at each follow-up point.

TABLE 4.2: MEDIAN RT & t-VALUES FOR SEVERITY GROUPS

(ONE BIT	TWO BITS	THREE BITS
1/12 FU <u>0</u>	4 8	0 4 8	0 4 8
M/M (7): 739	.957 1081	769 1119 1579	813 1248 1795
S (n=5):1016	1478 1655	1156 1607 2306	1195 2499 2938
t-value:3.64	2.77 2.15	2.34 2.54 2.24	3.75 3.95 3.74
***	*** *	** ** **	*** *** ***
3/12 FU			
M/M : 733	889 1016	778 1069 1176	793 1214 1581
S : 827	1096 1334	898 1391 1927	938 1609 2361
t-value:1.94	1.83 2.37	1.95 1.98 2.18	2.97 2.55 1.90
*	* **	* * *	*** ** *
6/12 FU			
M/M : 706	850 923	733 918 1128	762 1119 1515
S : 764	1036 1247	841 1220 1636	855 1537 2060
t-value:1.54	2.20 2.35	2.59 2.43 4.14	1.48 2.09 1.94
	* **	** ** ***	* *
*=p<.05;	**=p<.025;	***=p<.01	

Table 4.3 summarises the within-group t-tests of median RTs, for M/M and S subjects, based upon the scores presented in table 4.2. The Table shows significant improvement in median RT between 1 and 6 months posttrauma for most of the t-test comparisons in the S group, with more than 50% of the M/M group comparisons

TABLE 4.3: t-TESTS, MEDIAN RT WITHIN SEVERITY GROUPS

Information-Processing Load

<u>FU:</u>	<u>1</u>	v 3/12	3 v 6/1	12 1	v 6/12
<u>M/M:</u> 1 bit,	O ':,	<1	<1		< 1
(n=7)	4.:	1.05	<1		<1
	8;	<1	1.83	k	2.37**
2 bit,	0:	<1	1.20		1.09
	4:	<1	1.82	k	2.28**
	8:	2.05*	<1		2.26**
3 bit,	0:	<1	<1		2.31**
	4:	<1	1.10		1.58
	8:	1.90*	<1		1.88*
<u>S:</u> 1 bit,	0:	2.28*	1.23		3.24***
(n=5)	4:	1.82	<1		2.28*
	8:	1.10	<1		1.40
2 bit,	0:	1.51	<1		1.89*
	4:	<1	<1		1.84
	8:	<1	<1		2.36**
3 bit,	0:	1.15	<1		2.26*
*=p< .05;		**=p<.025;	***=	=p< .01	

achieving statistical significance. Less frequent significant t-values were noted for comparisons of the 1- and 3-month median RTs within the 2 severity groups.

Even with small sample sizes, the predictablity of recovery and of the effects obtained by increasing the information-processing load are interesting questions. Using median RTs, linear regression equations were generated for the M/M and S groups, using data from the 1-, 3-, and 6-month follow-ups (see table 4.4).

TABLE 4.4: LINEAR REGRESSION, MEDIAN RT 1-6 MONTHS
MILD HEAD INJURY GROUP

	ONE BIT			5	rwo bi	ITS	THREE BITS		
	0	4	8_	0	4	8_	0_	4	8
W t.:	-7	-21	-32	-8	-41	-84	-10	-26	-53
Int. :	727	890	1007	760	1035	1294	791	1194	1630
Corr.:	98	97	-1.0	83	99	86	-1.0	99	92
SEVERE HEAD INJURY GROUP									

		(ONE BIT			rwo B	ITS	THREE BITS		
		0	4	8	0	4	8	0	4	8
Wt	:	-48	-83	-78	-60	-76	-131	-65	-179	-170
Int.	:	869	1203	1412	965	1406	1956	996	1882	2453
Corr.	:	93	88	91	90	99	99	93	85	96
Wt.	=	slope	weigh	nt for	month	ns pos	st-inj	jury		

Int. = Intercept; Corr. = correlation coefficient

The correlations provided in table 4.4 generally indicate high linearity in predicting recovery curves for the M/M group (the majority of correlations

coefficients exceeded .97, and therefore accounted for 95%+ of the variance). Recovery for the S group in the various information-processing conditions was somewhat less linear, with fewer than 25% of coefficients exceeding .97. The equations in table 4.4 also show higher intercepts in the S group for each information condition. For both M/M and S groups the weights and intercepts rose with increasing information load, these rises being more marked for the latter.

Supplementary analyses were conducted to investigate the relationships between RT and other variables. Table 2, appendix A3, provides the correlation coefficients for median RTs and PTA at the 1-and 6-month assessments, in each of the experimental conditions. Nearly all of these correlations, which ranged from .51 to .89, were significant at the 5% level though 2, involving an information load of 3 bits, attained the 1% level. Correlation coefficients were weaker at the 6-month follow-up only one being significant at the 5% level.

To examine any effects from age, Pearson product-moment correlation coefficients with median RTs at the 6 month point were calculated for the two severity groups. No coefficient was large enough to achieve statistical significance in the M/M group, although coefficients

calculated for the two highest information-processing conditions (3 bits, 4 and 8 irrelevant stimuli) attained significance (p<.05) for the S group (appendix table A3.1.

4.4.3 Standard Deviation of RT

Additional analyses using the standard deviations of subjects' RT responses were conducted. The SD measure may be particularly appropriate given an hypothesis that a major component in the poorer cognitive performance of head-injured patients is an inability to sustain attention. According to this argument, more severely damaged subjects might be expected to show increased variability of RT responses.

Table 4.5a offers the summary of the ANOVA, conducted using SD data, involving severity, information load, level of irrelevant information, and time since head injury (repeated measures). The table shows highly-significant effects from the first 3 of these factors, with changes over time being significant at a lower level. Table 4.5 presents the SD data for M and S subjects in each experimental condition, with associated t-test values carried out following ANOVA, and appendix A2 provides the raw data for each subject.

TABLE 4.5a: ANOVA SUMMARY, SD OF RTs

<u>So</u>	urce	<u>df</u>	<u>MS</u>	F Ratio	Sig.Level
1	A: SEVERITY	1	5676298	10.95	**
2	C: IRREL.INFO	2	11897803	22.95	***
3	D: INFO.LOAD	2	7152672	13.80	***
4	AC	2	763753	1.47	n.s.
5	AD	2	268183	<1.00	n.s.
6	CD	4	1950059	3.76	· **
7	ACD	4	242022	<1.00	n.s.
8	SWG	90	518365		
9	B: REP.MEASUR	. 2	337408	4.25	*
10	AB	2	314250	3.96	*
11	BC	4	354900	4.45	* *
12	BD	4	102382	1.29	n.s.
13	ABC	4	26834	<1.00	n.s.
14	ABD	4	118170	1.49	n.s.
15	BCD	8	44273	<1.00	n.s
16	ABCD	8	87743	1.106	n.s.
17	B x SWG	180	79322		

Table 4.5 provides little evidence of significant differences between the 2 groups in relation to SD by the 6-month point. However, at both 1- and 3-months post-injury approximately half of the comparisons proved significant.

SD data in table 4.5 reflects the significant CD interaction (involving irrelevant information and information load) depicted in table 4.5a: the addition of irrelevant information to the target stimulus increases SD differentially, according to the information load (larger numbers of irrelevant items and higher information loads lead to higher SDs). The significant BC interaction is more complicated: no improvement in the 'zero irrelevant items' condition between 3 occurs and 6 months post-trauma, after generally marked improvements between the 1- and 3-month follow-ups. With 4 irrelevant items (and to some degree with 8 irrelevant items) lttle evidence is noted oof improvement between the 1- and 3-month points, although for most levels of information load improvement observed between 3 and 6 months after head injury.

TABLE 4.5: T-TESTS, SD OF RT FOR SEVERITY GROUPS

	ONE BIT TWO BITS				T	HREE 1	BITS		
1/12 FU	0	4	8_	0	4	8_	0	4	8
M/M :	79	154	234	80	261	629	69	397	974
s :	99	356	573	148	377	1113	231	1586	1679
t-value:	<1	3.17	1.63	1.40	1.65	1.82	2.17	5.96	2.13
		**					*	***	*
3/12 FU									
M/M :	49	152	195	70	234	390	58	644	1226
S :	51	334	369	71	679	1029	84	1114	1443
t-value:	<1	1.96	1.76	<1	2,05	2.33	1.56	1.42	< 1
		*			*	**			
6/12 FU)									
M/M :	58	168	205	72	192	293	99	546	1088
s :	61	259	416	82	348	817	93	567	1295
t-value:	<1	1.34	1.39	<1	1.77	6.24	< 1	< 1	<.1
	·					* * *			
*=p<.05;		**=p	(.01;	,	***=p<	(.001;			

Linear regression equations were generated in relation to increasing information-processing load. The correlations, weights and intercepts for these equations at 1- and 6-month follow-up are shown in table 4.6, for both M/M and S groups.

TABLE 4.6: LINEAR REGRESSION, RT SD & INFORMATION LOAD

<u>M/M</u>		Ol	NE MOI	NTH	S	IX M	ONTHS
		0	4	88_	0	4	:8
Wt.	:	-5	122	370	21	189	442
Int.	:	86	28	-128	35	-76	-354
Corr.	:	83	. 99	. 99	. 98	.89	.90
<u>s</u>							
Wt.	:	66	615	553	16	154	440
Int.	:	27	-457	16	47	83	-36
Corr.	;	. 98	. 87	. 99	. 98	. 97	. 99

Wt. = weight for months post injury

Int. = Intercept; Correl = Correlation coefficient

Half of the correlations in the M/M group exceeded .97, and all but one in the S group attained this value. For M/M subjects linearity fluctuated between the 2 follow-up points, whereas in the S group linearity remained unchanged for 2 equations and improved for the other.

The relationship of SD to severity was also examined via correlations with PTA (table 4.7). The results indicate a clear relationship between RT variability and severity of head injury at 1-month follow-up: most coefficients were significant, almost half at the 1% level. The 6-month coefficients were all non-significant, though as

TABLE 4.7: CORRELATION OF PTA & SD, PILOT STUDY

		<u>Correlation</u>	Coefficient
Information	<u>Irrelevant</u>	1/12 FU	<u>6/12 FU</u>
Load	<u>Stimuli</u>	(n=11)	(n=12)
1bit	0	. 374	055
	4	.876**	. 367
	8	.642*	.127
2bit	0	. 496	. 025
	4	. 695*	. 333
	8	.508	. 531
3bit	0	.793**	. 183
	4	.803**	. 202
	8	. 298	. 51,1
* / OF	** -4 01		

table 4.7 shows, with higher levels of irrelevant information there was a tendency for SD to be related to length of PTA.

4.5 DISCUSSION

4.5.1 Drop-Out

Subjects in this pilot study were recruited from the whole of the Northern region. Perhaps as a result the drop-out rate was high: of the 8 patients who failed to

complete attendance at 3 follow-up testing sessions only 2 were domiciled in Newcastle. Severity of head injury may also have been a factor in drop-out, as 5 severely-injured subjects were lost to the study compared with 3 in the M/M group. Given that S subjects who completed the study were older than those in the M/M group, a check on the age of the drop-out severe subjects was conducted. No evidence was obtained of a relationship between age and drop-out (t<1; df: 8, ns).

It is difficult to judge whether the drop-out rate for the present study is typical of that observed in similar experimental psychological investigations of headinjured patients, as drop-out/refusal information often not reported in studies (e.g. Miller, 1970; Miller and Cruzat, 1981). As noted in chapter 2, Brooks and Aughton (1979b) commented that drop-out rates for headinjured patients were considerable. Whilst Van Zomeren appeared to maintain approximately 80% of his (1981)sample of head-injured patients for testing on 4 occasions over a 19 month period, Conkey (1938) managed to obtain only a 16% rate for attendance at 5 follow-up sessions in the first year after head injury.

4.5.2 Median RT

The pilot study fulfilled its main aim in demonstrating that even severely head-injured subjects, close to the time of trauma, can respond to an experimental task which manipulates the level of information processing addition of irrelevant information. The study also confirmed the hypothesised sensitivity of this type of task to severity of head injury: the ANOVA summarised in table 4.1 indicates a highly-significant main effect from severity upon response time (table 4.2). The latter also demonstrated the differential effects upon the 2 groups by reflecting values of greater significance (p<.01) for comparisons in the high (3 bit)information condition (thereby supporting hypothesis 2). This result supports Miller (1970) who noted a very similar finding using a choice RT paradigm with severe head injury and control subjects.

Results from the present experiment also indicate that the median RT differences between the 2 severity groups persisted, with about half of the relevant t-test comparisons yielding significant values at the 6-month follow-up. The fact that this finding was obtained with very small groups points to the sensitivity of RT measures to severity of head injury, and suggests (at

least in the severe group) that further recovery would be necessary to achieve the presumed premorbid level of functioning. Inspection of table 4.2 confirms the hypothesised trend for median RTs to become faster between these 2 follow-up points, this finding applying to both groups in each of the 9 information conditions.

The ANOVA conducted indicated a significant effect from adding irrelevant information to the task. This finding clearly reflected in table 4.2, where median RT is increased according to the number of irrelevant stimuli within each information condition, at every follow-up, for both M/M and S groups. Miller and Cruzat (1981) also noted that the addition of irrelevant information to a processing task (card sorting) significantly slowed subjects' response times (p<.001). However, these authors did not obtain an interaction between groups (mild head injury, severe head injury, control subjects) and amount of irrelevant information, and concluded that the presence of a selective attention deficit in head injured subjects was not, therefore, supported. and Cruzat then went on to suggest that the negative interaction finding probably arose because experiment had "not tapped the right aspect of selective attention" (p.70). In this regard, Miller and Cruzat cite one of the possible flaws in their study as being

that the relevant stimuli appeared in regular, predictable positions. In the present experiment the irrelevant stimuli appeared in unpredictable positions on the tachistoscope card (as did the target stimulus), which may support their analysis as a significant groups x irrelevant information condition interaction was observed (to support hypothesis 3). This interaction finding accords with clinical observation that severely head-injured patients in the months after their trauma manifest poor attentional control and appear to be distractable.

Additional evidence of differences between severely and mildly head-injured subjects is provided by the finding length of PTA and median RT significantly in nearly all information-processing conditions at the 1-month follow-up. This association showed a marked reduction as recovery occurred, so that by 6 months post-trauma only 1 coefficient attained statistical significance. The results presented table 4.4 suggest that recovery in visual informationprocessing ability for the early post-trauma months may be predictable and linear. This finding is necessarily of limited value, given that the study covers only the first 6 months following head injury.

4.5.3 Standard Deviation of RT

The present study also included some analyses using SD measure of RT variability. Using t-test comparisons, this index provided less evidence significant differences between the 2 severity groups. Significant associations between SD and PTA were noted correlation analyses using at 1-month follow-up, (offering partial support for hypothesis 1) although this relationship weakened by 6 months post-injury. linear However. rises in SD under conditions increasing information load were noted, these changes being more predictable in the severe group.

Although data only covers the first 6 months cognitive recovery after head injury, improvement information-processing speed, as reflected by median RT, appears to be predictable using linear equations. The fit is better for the M/M group, with S subjects also showing higher intercepts and steeper recovery curves. Even though the predictability covers only the early post-trauma, the results obtained do raise the interesting possibility that longer-term cognitive recovery may be open to prediction. If this were possible, then the clinical implications could be great: it might become feasible to advise when, for example, a head-injured patient was likely to be able to return to work or education. Similar research in the field of stroke (Skilbeck, Wade, Langton-Hewer and Wood, 1983) has enabled the prediction of functional outcome in Activities of Daily Living areas.

Finally, although interpretation of the finding is complicated by the fact that the M/M group was significantly younger, correlations between age and median RT were significant 6 months after head injury for those conditions offering the highest information-processing load.

4.6 SUMMARY

The pilot study was designed to investigate whether head-injured subjects could cope with tasks involving the processing of high levels of information. This question has been answered satisfactorily, and severely head-injured subjects soon after trauma are able to handle a high information-processing load. No evidence to suggest design constraints upon the main study has been noted.

Results from the present experiment lend support to the hypothesis that severely head-injured subjects process information more slowly. They also indicate that the presence of irrelevant information has a differentially adverse effect upon response speed in severe subjects. Increasing the information load differentially slows RT in severe subjects. Some evidence of greater RT variability in severely head-injured subjects was observed.

Another aim was to seek evidence that informationprocessing tasks can detail cognitive recovery following head injury. The results provided in section 4.5 support this suggestion. On data covering only first 6 months of cognitive recovery after head injury. information-processing improvement in speed, reflected by median RT, appears to be predictable using Increase in RT variability, linear equations. measure by SD, also seems linear and predictable under conditions of increasing information-processing load.

CHAPTER 5

MAIN STUDY: STERNBERG'S PARADIGM

& COGNITIVE RECOVERY

FOLLOWING HEAD INJURY

5.1 AIMS

The results of the pilot study described in chapter 4 demonstrated that an information-processing approach may be applied to the investigation of cognitive recovery following head injury.

A primary aim of the main study was to describe one aspect of cognitive disturbance arising from head injury, and its recovery, in terms of a specific paradigm drawn from experimental psychology. The selected procedure, Sternberg's paradigm, offers number of theoretical aspects and research has already been published on its use with a wide range of subject groups (reviewed in chapter 3). It was predicted that the selection of a sensitive indicator (based upon millisecond timing of patients' responses) would be able both to reflect differential cognitive deficits according to severity of head injury, and would also allow for the detection of any continuing recovery occurring between 12-24 months, or longer, after injury.

A second aim of the study was to relate the findings from using Sternberg's paradigm to those obtained from a range of other cognitive tasks that are more widely used in clinical neuropsychological practice. These tasks

include both traditional clinical measures of memory such as are provided by the Wechsler Memory Scale (WMS; Wechsler, 1945), and a task designed for experimentalclinical neuropsychological use - the Rey AVLT Lezak, 1983). Also included was a subjective measure of memory performance (Bennett-Levy & Powell, 1980). measures used are specified in more detail below. In addition, the study aimed to examine the relationship between clinical variables (such as length of PTA, length of unconsciousness, neurosurgical intervention, etc), and an estimate of premorbid IQ (largely based upon the National Adult Reading Test (NART; Nelson, A small number of demographic variables were also available for investigation.

From these aims, and the review of the literature, a number of specific hypotheses were generated:

- Using Sternberg's paradigm it would be possible to detect cognitive recovery 12-24 months after head injury, or even later.
- The level of disturbance in memory scanning performance assessed soon after head injury would relate to severity of head injury.
 Welford (1980b) viewed age slowing as being caused primarily by changes in the Central Nervous System.

RT can be viewed as an index of brain efficiency covarying with severity, so that the slowing of Sternberg RTs would be predicted to be more marked in more severely head-injured subjects. It was hypothesised that the 'final' (recovered) memory scanning results would remain abnormal in those sustaining extremely severe head injuries.

- 3. Disturbance in Sternberg performance (cf the performance of non-brain-damaged people), and its subsequent recovery, would be reflected in:
 - a. Median RT. The slowing of Sternberg RTs would be marked in more severely head-injured subjects.
 - b. Standard deviation of RT. Greater 'blocking' would be seen in patients (linked to increasing severity), because of their reduced attention—sustaining ability. This would be reflected in a larger variability in performance and, therefore, in larger SDs. Blocking is usually only seen in normals under of prolonged on—task testing.
 - c. The slope weight. It was predicted that the increase in information-processing load stemming from a larger positive set size would differentially penalise the more severely-damaged subjects. This would be reflected in a larger slope value associated with the linear regression lines.

A number of other hypotheses were generated from existing research (reviewed in chapter 3) using the Sternberg paradigm:

- d. Error responses would be faster because they reduce the amount of information gained.
- e. Male RTs would be faster than those of females.
- f. Greater damage to the left hemisphere would lead to additional error responses and a steeper RT slope.
- g. Parallel positive and negative RT slopes would be observed.
- h. Practice effects would not occur, as extended daily testing with fixed stimuli was not employed.

5.2 SUBJECTS

The present experiment aimed to study cognitive recovery over an extended period of time — up to 3 years — after trauma. The problems encountered in trying to maintain a sample across numerous follow—up test sessions, distributed over a long period, are great (discussed in chapter 2). In particular, Conkey (1938) and Brooks & Aughton (1979b) commented on very high drop—out rates. It was decided, therefore, to include two clinical samples in the current experiment. The principal sample consisted of patients scheduled to be tested at 1, 3, 6, 12, 24, and 36 months post—injury (Sample A). Given a

probably-high attrition rate, and that a particular aim of the study was to investigate long-term recovery (24 months post-trauma, and longer), it was decided to construct a second sample of patients tested at 24 and 36 months post-injury (Sample B). Equipment variables such as screen luminosity and type of response device may influence the specific RT values obtained. Given this, 'normal' data was obtained for the specific hardware configuration employed in the study, using a sample of young volunteer hospital workers (Sample C).

The planned intake into the study for sample A was 10 patients in each of the 4 severity groups (M/M, S, VS, ES), making a total of 40 subjects. However, due to initial misclassification of 2 patients' severity, it was necessary to recruit an extra 2 subjects to meet the criterion of 10 patients per severity group. Sample A, therefore, consisted of 42 subjects. The initial target size for sample B was 15 subjects, and for sample C was 10. Sample B lost 5 subjects because 2 subjects did not attend at the 24 month follow-up, 2 did not attend at 36 months, and 1 because of a prior history of head injury.

All patients in sample A were hospitalised in Frenchay Hospital, either by direct admission or by transfer from another hospital to receive specialised neuroscience management. Suitable subjects were either identified randomly selected from the wards, or were Book. Hospital Admissions The latter was necessary to include in sample A sufficient patients who had suffered mild head injuries; such patients are often only hospitalised overnight for neurological observation, and would be difficult to recruit to the study if Admissions Book was not consulted. The method recruitment was by personal approach, via twice-weekly visits to the wards, if the patient was still letter if the hospital and patient had by been-The patients comprising sample B were discharged. identified from Psychology Department records, being patients who had previously been routinely referred for neuropsychological evaluation by neuroscience consultants at Frenchay Hospital. These patients were approached by letter.

Exclusion criteria for subjects in these clinical samples were:

Geographical. The South-West Regional Health
 Authority covers a very large narrow region which is
 250 miles from north to south. Only patients who
 lived in the northern part of the region (Somerset,
 and northwards) were included, plus those patients
 who, although they were resident outside of the area

- covered by the SW RHA, lived within 1 hour travel time of Frenchay Hospital. The latter covered, for example, people living in Bath and Cardiff.
- 2. Prior History. Any potential subject with a history of previous head injury or neurological involvement was excluded from the study.
- 3. Age. As reviewed in chapter 3, there is considerable evidence that a number of aspects of RT performance change significantly in subjects over the age of 50 years. Similarly, RT performance in young children may differ from that seen in older children and adults. The current study, threfore, only accepted subjects in the age range 10-50 years.

The period of intake covered approximately 18 months. between February 1981 and August 1982. The normal subjects were all volunteer employees of Frenchay HA.

5.3 PROCEDURE

Once they had been identified, and their agreement to participate in the study obtained (or that of their families'), arrangements were made with sample A subjects to test them at approximately 4 weeks posttrauma. As will be noted in the results section, some of the more severely-injured patients were untestable at this one-month follow-up as they were still in PTA. The

intention for all sample A subjects was to carry out cognitive assessments at 1, 3, 6, 12, 24, and 36 months after their head injury. Attendance at each of these follow-ups entailed testing with Sternberg's memory scanning paradigm, assessment of WAIS digit span (Wechsler, 1955), and completion of a parallel version of the Rey AVLT (see Lezak, 1983). The parallel forms are reproduced in appendix B1. The sequence of their presentation to subjects was randomised.

The Sternberg procedure employed positive set sizes of 1-4 items (see chapter 3), fixed for any one run, and was presented using a Commodore 'PET' microcomputer. Attached to the micro via its parallel user port was a 'button press' response device. The Sternberg software was jointly written by the author and Dr. David Norris. Scientist based in the Medical Computer Department of Frenchay Hospital. Dr. Norris' particular contribution related to the insertion of a millisecond timing routine into the program. Four versions of the software were written, according to positive set size. As an example, the program covering set size 2 is listed in appendix B2. The sequence of presentation of the 4 positive set size runs was determined randomly for each subject. For testing, subjects were seated comfortably in a height-adjustable chair in front of a table on

which were placed the microcomputer visual display unit and response device. The latter was positioned according to the subject's preference.

Each Sternberg run presented 45 trials to the subject. The first 5 were regarded as practice (Hamsher & Benton, 1977), and the remainder offered 20 positive set and 20 negative set trials in a quasi-random sequence: each run was constructed to balance positive and negative trials with a maximum-allowable sequence of 4 positive or 4 negative trials. The latter feature was included to avoid subjects developing a false probability judgement about the relative frequency of occurrence of positive or negative items. Contained within each program was sufficient data for 5 runs, to allow repeat testing.

Running any version of the Sternberg program first required the insertion of a datafile name for storage of data at the end of the run. After entering information covering date, run name, positive set size, and data set, the VDU displayed instructions to described in chapter 3, the subject is the subject. As asked to hold in memory a small number of digits, which form the 'positive set'. With a positive set size of 1, only 1 digit is kept in memory, and with a positive set of 4 items, 4 digits (eg, 1-3-7-8) are held in memory.

this example, all other digits (ie, 0,2,4,5,6.9) constitute the negative set. The subject is instructed to respond as quickly as possible to a probe stimulus (ie, digit) presented via the VDU by pressing a red button if the probe belonged to the positive set, and a black button if it belonged to the negative set. experimenter ensured that the subject understood the his/her fingers resting instructions, had the buttons, and then initiated the run. The subject was then presented with the 45 visual probes, one-by-one. Following a response, the VDU cleared for 2 seconds and then presented the message 'get ready' for approximately 1.5 seconds before onset of the next probe stimulus. card was attached to the response panel, above response buttons, to remind subjects of the positive set Patients responded using their dominant hand, except in the few cases where physical damage had affected the dominant hand or arm (either peripheral injury, or hemiparesis/hemiplegia). In this situation, the non-dominant hand was used to respond at all follow-ups. Data on handedness and response hand are provided in appendix table C5.1.

As it was running, each Sternberg program recorded the RT in milliseconds for each of the 45 trials, and its accuracy. It seemed possible that 2 subject behaviours

might interrupt the smooth running of the program. First, after making a response a subject might hold down a response button, so preventing the program proceeding to the next trial. The program was designed to check for this, so that in the event of a failure to release a response button the subject was asked, via the VDU, to release the button. Second, a subject might fail to make a response to a probe stimulus. In this case subjects were reminded of the instructions, again through the VDU. After displaying this reminder for 10 seconds, the program moved on to the next trial. At the end of the run the program stopped, awaiting input from the experimenter to provide hard-copy of results (an example printout is provided in appendix B3).

The program then proceeded to store these results on floppy disk within the datafile named at the beginning of the run. One complete Sternberg run took less than 5 minutes, and the total memory scanning assessment for the 4 positive set sizes required approximately 20 minutes. To reduce boredom or fatigue, Sternberg runs were interspersed with other test material and interview.

Estimates of premorbid intellectual level were gained for most patients using the National Adult Reading Test (NART; Nelson, 1982). Development within the department during 1982 of a microcomputer-administered version of the Subjective Memory Questionnaire (SMQ; Bennett-Levy & Powell, 1980) allowed most sample A subjects to rate their own memory ability, usually at 24 or 36 months This program was written by Mr. post-trauma. Olive, Psychology Technician in the author's department. The SMQ was included to allow asociations with Sternberg findings, and with other memory tasks to be investigated. Data was gathered on the Wechsler Memory Scale (WMS; Wechsler, 1945), and on a shortened WAIS (Wechsler, 1955). Due to time constraints and possible subject fatigue, data on the NART, SMQ, WMS, and WAIS were only gained on some occasions (rather than at all follow-ups).

Sample B received the same set of test procedures as described above for sample A, at 24 and 36 months after head injury. Sample C completed the 4 Sternberg runs (positive set sizes 1-4), Rey AVLTs, and provided digit span data at each testing session. The schedule for sample C subjects was 4 test sessions, spaced at two-week intervals to provide a rigorous check for any possible practice effects which may have been operating.

5.4 RESULTS

5.4.1 Clinical & Demographic Data

Given the relationships between clinical aspects of head injury and cognitive performance, reviewed in chapter 2, relevant data were recorded whenever possible for subjects in samples A and B. The clinical variables chosen included neurosurgical intervention, occurrence of fits, CT brain scan results, etc (see tables 5.1a, and 5.2a). The raw data for these variables are shown in appendix C4, tables C4.1 and C4.2. Additional background information on subjects relating to age, sex, time to return to work/school, and other variables is also presented in appendix C, tables C5.1 and C5.2.

Table 5.1a provides data on clinical variables for samples A and B, and other background information on these subjects is shown in table 5.1b. Using a severity categorisation based upon duration of PTA (table 2.3), sample A contained 11 mild/moderate subjects (M/M), 10 severe (S), 10 very severe (VS), and 11 extremely severe (ES). In sample B no subject had suffered a mild head injury, 3 had sustained a moderate injury, 1 a severe. 3 a very severe, and 3 an extremely severe injury. Tables 5.2a and 5.2b presents the clinical and background data on the sample A subjects, by severity group.

5.4.2 Memory Scanning Data: Recovery in Median RT

a. <u>Introduction</u>. Given that a potentially enormous amount of data was available for analysis, some decisions concerning the statistical focus were necessary. As was pointed out in chapter 4, RT data is typically skewed. Therefore, although summary tables include presentation of group mean scores, statistical analyses were carried out using the median (as recommended by Hays, 1963; Dunn & Master, 1982) and standard deviation as measures of performance.

TABLE 5.1a: CLINICAL DATA, SAMPLES A & B

<u>Variable</u>		Sample A	Sample B
		(n=42)	(n=10)
GCS score	: Median	7	8
	Mean	7.4	8.1
	SD	3.7	4.6
Duration of Coma	: Median	39	72
(hours, n=39)	Mean	199.3	126.8
	SD.	321.3	163.1
Length of PTA	: Median	7	11
(days)	Mean	19.2	14.7
	SD	25.9	14.3
Number undergoing neu	rosurgery	7	5
Number undergoing oth	er surgery	2	1
Number abnormal skull	X-ray	19	7
Number abnormal CT sc	an	26	7
Number with fits, in	hospital	8	4
Number with fits, pos	t-discharge	2	2
Number on anticonvuls	ants	17	5
Signs of lateralisati	on, L/R	11/15	4/4

TABLE 5.1b: BACKGROUND INFORMATION, SAMPLES A & B

		Sample A	Sample B
Age	: Median	18	20
	Mean	22.6	20.2
	SD	9.8	5.8
Number of males		25	8
Number of social cl	ass : 1&2	12	2
	3	10	2
	4&5	11	3
	Student	8	2
	Unemployed	1	1
Educational level:	<=15/CSE	11	5
	'O' level	11	2
	'A' level	6	1
	Tertiary	8	1
St	ill at school	3	1
Cause of :	RTA, car	16	2
head injury	RTA, m/cycle	7	2
	RTA, ped.	9	0
	Occupational	1	0
	Sport	2	2
	Home/other	4	1
Time to return to	: Median	5	9
work/school (months) Mean	23.1	23.0
(n=35, sample A)	SD	33.6	32.7
Handedness	: Left/Right	4/37	1/9

TABLE 5.2a: CLINICAL DATA, SAMPLE A SEVERITY GROUPS

				•
	mild/		very	extrem
	mod	severe	severe	severe
	(n=11)	(n=10)	(n=10)	(n=11)
GCS score : Median	11	7	7	4
Mean	10.6	8.2	7.3	3,9
SD	3.0	2.9	3.6	0.7
Coma duration : Median	0.3	14	48	744
(hours) Mean	11.0	25.5	75.2	609.9
SD	21.1	35.2	106.1	343.1
Length of PTA : Median	1	5	14	42
(days) Mean	0.8	4.7	15.4	52.4
SD	0.4	2.2	6.6	22.5
Neurosurgery no.	2	2	3	0
Other operations no.	1	0	0	1
Abnormal skull X-ray no	. 4	5	6	4
Abnormal CT scan no.	4	7	5	10
Fits in hospital no.	3	0	2	3
Fits post-discharge no.	1	0	0	1
No. on anticonvulsants	3	3	4	4
No. with Signs of :Left	2	2	2	5
lateralisation : Right	: 2	4	5	4

TABLE 5.2b: BACKGROUND INFORMATION, SAMPLE A GROUPS mild/mod severe v.sev. ex.sev. (n=11) (n=10)(n=10)(n=11)AGE : Median 17 19 20 18 Mean 20.0 19.1 25.7 25.5 10.6 SD 6.7 4.6 12.7 No. of Males 5 5 8 7 No. of social class 1&2 5 1 2 3 1 1 5 3 4&5 2 3 2 4 Student 3 1 2 2 Unemployed 1 0 Ω 0 Educat. level <=15/CSE 2 4 2 4 'O' level 2 5 1 3 'A' level 2 1 2 1 Tertiary 2 1 3 2 Still at school 1 1 2 1 2 5 Cause of RTA, car 4 5 head RTA, m/cycle 2 2 2 1 RTA, cycle injury 1 1 1 0 2 RTA, ped 3 0 4. Other 1 3 2 1 Time to return : Median 3 4 4 23

25.9

36.2

13.0

26.6

5.5

2.7

44.2

38.3

Mean

SD

to work/school

(months)

A second decision concerned the type of response data which should be analysed - all memory scanning RTs, or only those involving correct RTs? One aim of the Sternberg paradigm is to study errorless performance, suggesting that only correct responses should analysed. Also, it is impossible to be sure of what has occurred, in information processing terms, on any trial where incorrect response is the final an product. Although Sternberg (1975) indicated that the literature suggested that error rates of up to 10% do not alter response characteristics, it was thought appropriate in the current study to concentrate statistical analyses upon those RTs gained from correct responses. comments, however, will be offered in relation to the RT differences between 'correct' and 'error' responses.

Data from the 'severity' groups of sample A were analysed longitudinally between follow-up points, and cross-sectionally at each follow-up. Sample B's results were analysed at its two follow-up points, including investigations of effect of initial severity of head injury. The severity groups' averages for mean RT, SD, and median RT (msec) in sample A at each follow-up are shown in table 5.3. Similar data for samples B and C are included in tables 5.4 and 5.5. More comprehensive raw data is tabulated in appendices C1-C3.

TABLE 5.3: SAMPLE A AVERAGE MEDIAN, SD, & MEAN RT

ONE-MONTH FOLLOW-UP

		Pos	itive	Set		Nega	Negative Set		
		1	2	3	4	1	2	3	4
A (n=23)		•						
Median	:	938	795	818	884	836	796	833	960
SD	:	200	205	230	286	237	222	252	251
Mean	:	992	845	897	7 938	921	835	906	992
<u>M/M(8)</u>									
Median	:	463	534	565	597	491	553	618	684
SD	:	121	127	135	195	121	136	168	162
Mean	:	485	546	593	636	525	575	659	706
<u>s (7)</u>									
Median	:	670	733	843	937	716	821	885	1094
SD	:	128	218	252	340	191	257	233	273
Mean	:	669	775	901	1017	756	895	932	1106
VS (6)				-					
Median	: .	1932	1265	1095	1140	1430	1035	998	1149
SD	:	385	272	323	316	398	262	315	307
Mean	: 2	2094	1370	1280	1213	1623	1030	1134	1182
<u>ES (2)</u>									
Median	:			-	_		_	_	_
SD	:	235	309	300	383	361	338	500	394
Mean	:	972	974	1142	1187	1130	1175	1231	1263
		A=sa	mple	A ;	M/M=	mild/mod;	S=s	evere;	

VS=very severe; ES=extremely severe

TABLE 5.3: SAMPLE A AVERAGE MEDIAN, SD, & MEAN RT

THREE-MONTH FOLLOW-UP

		Pos	itive	Set		Nega			
·		1	2	3	4	1	2	3	4
A (n=27	<u>)</u>								
Median	.:	641	662	764	807	630	715	777	833
SD	:	173	197	215	263	177	213	201	242
Mean	:	662	700	785	846	668	747	808	889
<u>M/M(5)</u>									
Median	:	349	408	533	489	423	477	563	568
SD	:	70	95	126	142	113	102	170	139
Mean	:	354	422	535	525	437	490	595	583
<u>S (7)</u>									
Median	:	579	785	851	880	627	794	849	866
SD	:	159	231	231	235	182	231	241	186
Mean	:	597	792	879	887	676	839	898	894
VS (9)									
Median	:	415	453	492	580	478	545	581	683
SD	:	111	114	142	177	112	94	115	245
Mean	:	430	477	535	608	500	551	590	765
ES (6)									
Median	:	1296	1121	1365	1429	1033	1148	1244	1321
SD	:	367	415	415	576	320	512	331	420
Mean	:	1343	1249	1356	1536	1104	1231	1287	1411
		-			34 /34		~		

A=sample A; M/M=mild/mod; Sev=severe;

VS=very severe; ES=extremely severe

TABLE 5.3: SAMPLE A AVERAGE MEDIAN, SD, & MEAN RT
SIX-MONTH FOLLOW-UP

		Posi	tive	Set		Nega			
		1	2	3	4	1	2	3	4
A (n=41	<u>)</u>								
Median	:	522	603	731	713	569	657	735	780
SD	:	128	163	208	222	140	178	194	236
Mean	;	513	623	768	772	592	690	772	839
M/M(11)									
Median	:	413	541	573	607	458	570	617	664
SD	:	124	190	156	148	106	190	169	163
Mean	;	442	573	595	625	468	607	649	691
S (10)									
Median	:	544	587	683	673	5.78	632	695	739
SD	:	112	133	180	207	143	134	154	185
Mean	:	557	597	699	741	602	638	729	783
<u>VS (9)</u>									
Median	:	392	421	469	528	444	485	516	586
SD	:	97	98	113	147	97	103	86	147
Mean	:	404	447	487	573	464	502	526	621
<u>ES (11)</u>									
Median	:	717	828	1108	1005	776	908	1036	1091
SD	:	172	215	350	370	206	265	333	429
Mean	:	746	840	1192	1111	811	974	1101	1217
		A=sa	mple	A ;	M/M=	mild/mod;	Sev	≔sever	e:

VS=very severe: ES=extremely severe

TABLE 5.3: SAMPLE A AVERAGE MEDIAN, SD, & MEAN RT

TWELVE-MONTH FOLLOW-UP

		Posi	tive	Set		Negative Set			
		1	2	3	4	1	2	3	4
A (n=39)	<u>)</u>								
Median	;	459	495	616	630	516	555	650 ⁻	686
SD	:	109	110	186	167	124	124	170	198
Mean	:	476	502	658	658	550	574	684	732
<u>M/M(10)</u>									
Median	:	446	511	588	574	482	557	614	652
SD	:	133	123	173	165	137	139	199	206
Mean	:	471	506	613	610	522	577	668	681
<u>S (8)</u>									
Median	:	404	456	495	533	461	494	552	593
SD	:	70	88	112	108	76	122	109	119
Mean	:	414	459	519	542	478	526	566	610
<u>VS (10)</u>									
Median	;	366	432	488	526	429	511	5.75	552
SD	:	84	97	142	130	66	92	128	112
Mean	:	386	448	516	544	434	519	589	577
ES (11)									
Median	:	594	580	869	847	667	658	832	905
SD	:	139	127	301	246	200	146	230	325
Mean	:	609	595	956	890	733	675	887	1009
		A=sa	mple	A ;	M/M=	mild/mod;	Sev	-sever	e;

VS=very severe, ES=extremely severe

TABLE 5.3: SAMPLE A AVERAGE MEDIAN, SD, & MEAN RT

TWENTY-FOUR-MONTH FOLLOW-UP

		Posi	tive	Set		Negative Set			
		1	2	3	4	1	2	3	4
<u>A (n=26</u>	<u>)</u>								
Median	:	447	491	581	59 3	506	552	635	680
SD	:	149	144	200	191	123	132	186	177
Mean	:	479	525	629	634	529	571	672	703
M/M(7)		-							
Median	:	429	467	555	604	503	522	605	652
SD	:	124	191	220	262	131	129	224	153
Mean	:	452	524	597	672	512	538	675	664
<u>S (5)</u>									
Median	:	392	400	394	481	425	475	499	562
SD	:	266	97	81	97	102	88	98	164
Mean	:	452	422	420	494	449	482	522	582
<u>VS (8)</u>									
Median	;	397	435	454	515	439	482	523	570
SD	:	197	105	107	121	95	111	94	160
Mean	:	442	455	484	533	460	491	544	588
<u>ES (6)</u>									
Median	:	508	564	732	651	553	626	743	766
SD	:	129	168	302	201	118	162	245	189
Mean	:	536	596	821	698	583	653	778	799
		A=sa	mple	A ;	M/M=i	mild/mod:	Sev	=sever	;

VS=very severe: ES=extremely severe

TABLE 5.3: SAMPLE A AVERAGE MEDIAN, SD, & MEAN RT

THIRTY-SIX MONTH FOLLOW-UP

		Posi	tive	Set		Negat	ive S	et		
A (n=10	<u>)</u>	1	2	3	4	1	2	3	4	
Median	:	371	441	464	486	441	463	509	536	
SD	:	151	141	192	232	161	159	177	304	
Mean	:	417	481	525	572	476	519	5,84	654	
TAB	LE	<u>5.4</u> :	SAMP	LE B	AVERAGE	MEDIAN,	SD,	& MEAN	RT	
TWENTY-FOUR MONTH FOLLOW-UP										
Positive Set Negative Set										
B (n=10	<u>)</u>	1	2	3	4	1	2	3	4	
Median	:	853	664	597	1198	655	706	656	918	
SD	:	214	399	151	333	248	285	179	360	
Mean	:	964	837	621	1377	726	926	692	1049	
			THIR	TY-S	X MONTH	FOLLOW-	UP			
		Posi	tive	Set		Negat	ive S	et		
B (n=10	<u>)</u>	1	2	3	4	1	2	3	. 4	
Median	:	533	612	686	741	577	608	762	753	
SD	:	132	143	170	247	163	180	237	200	

Mean: 549 629 715 799 613 664 803 796

TABLE 5.5: SAMPLE C AVERAGE MEDIAN, SD, & MEAN RT a. FIRST FOLLOW-UP (n=10) Positive Set Negative Set 3 4 : 358 Median 391 413 447 : 73 94 110 SD : 386 400 437 477 419 481 Mean b. SECOND FOLLOW-UP (n=10) Positive Set Negative Set 2 3 : 328 364 418 Median SD : 100 82 74 114 73 : 363 388 430 418 436 Mean c. THIRD FOLLOW-UP (n=10) Positive Set Negative Set 1 2 1 2 Median : 324 : 49 70 SD : 334 374 Mean FOURTH FOLLOW-UP (n=6) Positive Set Negative Set Median : 312 335 SD

Mean : 322 380 411 428

b. Median RT. The first major analysis employed median RT data, gathered longitudinally from sample A subjects during the first 24 months post-injury. A 3-way ANOVA with repeated measures was used (Kirk, 1982): severity of head injury (4 levels: M/M, S, VS, ES), positive set size (4 levels: 1-4 items), type of set (2 levels: positive, negative).

To include the maximum number of subjects, the analysis was performed on data from the 3-24 month follow-up points; 9 of the 11 subjects who had sustained ES head injuries were not testable at the 1-month point. Even so, a number of subjects were non-attenders at more than one follow-up and had to be excluded from tha analysis, leaving a sample of 26 patients. Of these, 3 in M/M group, 3 in the S group, and 2 in the ES group did not provide data at the 3-month point. Scores for these subjects were constructed by interpolation of the appropriate severity group median score at 3 months. At the 6-month point data was missing for 1 VS subject, and at 12 months for 1 S subject.

The summary of this ANOVA is shown in table 5.6, the results indicating significant main effects from the repeated measures factor (time since head injury; p<.001), severity of head injury (p<.001), and positive

TABLE 5.6: ANOVA SUMMARY, MEDIAN RT

Source	<u>SS</u>	<u>d</u> f	<u>MS</u>	<u>F-ratio</u>	<u>Sig.</u>
1. A: SEVERITY	13260522	3	4420174	29.551	***
2. C: +/- SET	362796	1	362794	2.425	n.s.
3. D: SET SIZE	3575631	3	1191877	7.968	* *:*
4. AC	60129	3	20043	<1.000	n.s.
5. AD	203201	9	22579	<1.000	n.s.
6. CD	25958	3	8653	<1.000	n.s.
7. ACD	111767	9	12419	<1.000	n.s.
8. S.W.G	30514116	204	149579		
9. B	6971141	3	2323714	147.885	***
10.AB	8659804	9	962200	61.236	***
11.BC	140704	3	46901	2.985	**
12.BD	50850	9	5650	<1.000	n.s.
13.ABC	4088004	9	454223	28.908	***
14.ABD	655389	27	24274	1.545	n.s.
15.BCD	89279	9	9920	<1.000	n.s.
16.ABCD	21457129	27	794709	50.577	***
17.B x S.W.G.	9616498	612	15713		
* = p<.05;	** = p	(.01;	***	= p<.001;	

set size (p<.001). The results obtained from set (positive, negative) just failed to attain the 5% level of statistical significance. Table 5.6 also displays significant interactions between time and severity (p<.001), time and set (p<.01), time-set-severity

(p<.001), and the interaction of all 4 factors (p<.001). The highly significant results involving severity and time will be investigated further below, but figures 5.1a-d reflect the significant interaction of these variables with set. The latter reflected the fact that S and ES groups showed steeper recovery curves, and that median RTs on negative trials were faster than their positive equivalents at the 3-month point.

Although demonstrating significance is difficult with small samples, following the significant ANOVA findings presented in table 5.6 t-test analyses were conducted using all subjects who attended adjacent follow-ups to further examine recovery in RT. All t-test results reported in this thesis (excepting demographic clinical variables) 1-tailed, hypotheses being are directional and related to a priori planned comparisons (Kirk, 1982). There is, of course a statistical risk in carrying out a large number of t-tests: the larger the nuumber of t-test values computed, the larger the probability that a statistically significant result will be obtained by chance. Interpretation of findings will take account of this risk. Appendix table C6.1 shows the t-test values comparing adjacent follow-up points for each severity group and set size/type, and provides only occasional evidence of significant recovery in RT.

FIGURE 5.1a: RECOVERY IN MEDIAN RT. POSITIVE & NEGATIVE TRIALS, M/M GROUP

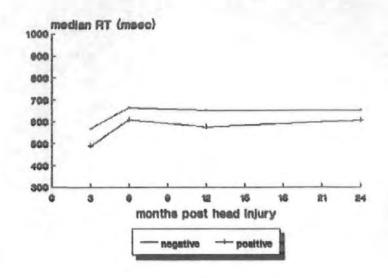


FIGURE 5.1b: RECOVERY IN MEDIAN RT,
POSITIVE & NEGATIVE TRIALS, S GROUP

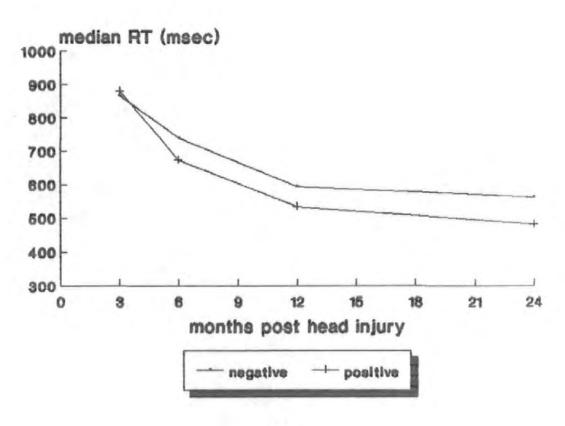


FIGURE 5.1c: RECOVERY IN MEDIAN RT, POSITIVE & NEGATIVE TRIALS, VS GROUP

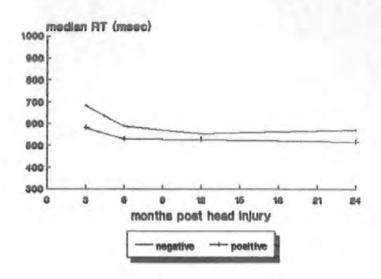


FIGURE 5.1d: RECOVERY IN MEDIAN RT,
POSITIVE & NEGATIVE TRIALS, ES GROUP

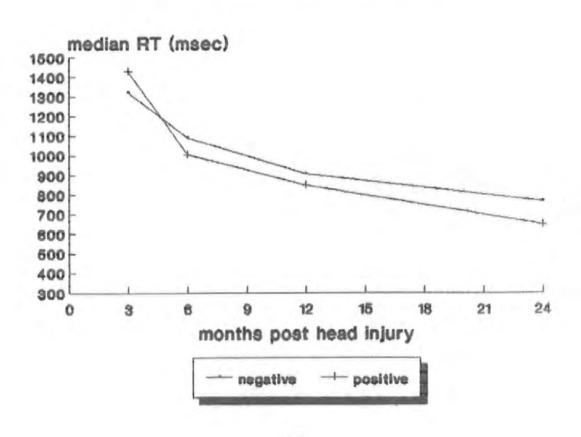


TABLE 5.7: SIGNIFICANT RECOVERY IN RT AFTER 6 MONTH FU

FU Period	Group	<u>n</u>	Set/Size	t-value	Sig.level
6-12/12	A	38	+ , 2	1.778	*
	S	8	+ , 1	1.853	*
	ES	11	- , 2	2.722	,* * *
	ES	11	+ , 2	3.171	***
12-24/12	٧s	5	+ , 3	2.231	*
6-24/12	A	27	+ , 1	1.871	*
	A	27	+ , 2	2.221	**
	A	27	+ , 4	1.780	* *
	A	27	- , 2	2.010	**
	A	27	- , 4	1.692	*
	S	6	+ , 1	2.488	* *
	S	6	+ , 2	2.242	**
	S	6	+ , 3	1.879	*
	S	6	+ , 4	2.077	*
	S	6	- , 1	1.864	*
	S	6	- , 2	2.029	*
	ES	8	+ , 2	2.556	**
	ES	8	+ , 4	1.885	*
	ES	8	- , 1	2.101	*
	ES	8	- , 2	2.384	**
	ES	8	- , 4	1.934	*
*=p<.05;	**=p<.	025;	***=p<	(.01;	****=p<.005;

However, table 5.7 does demonstrate that for the S and ES groups RT recovery occurred beyond the 6-month point (and for sample A overall). The lack of significant values in relation to the M/M and VS groups underlines the significant severity-time interaction presented in 5.6. The most surprising finding reflected in Figure 5.1 is that the median RT for subjects in the VS group were faster than for those in the M/M and S This is a difficult finding to account for groups. satisfactorily. Severity of injury was gauged on the basis of length of PTA (table 2.3), and inspection of table 5.2a suggests that the VS group (mean PTA: 15.4 days) is appropriately placed above the M/M (0.8 days) and S (4.7 days) groups. In addition, the table shows that VS subjects had, on average, poorer initial GCS scores than these groups and longer periods of coma. There is also no evidence from the other signs of subjects severity of injury, such as number of undergoing neurosurgery or with abnormal skull X-ray/CT findings, to suggest that the VS group was actually 'milder' than might be judged solely from length of PTA. However, some data in table 5.1b can be viewed supporting the idea that the VS subjects did actually make a faster recovery: the mean time to return work/school for VS subjects was shorter than for other groups. The data on educational level contained

table 5.4 also shows a tendency for more VS subjects to progress beyond 'O' level than in other groups.

The change in median RT for sample A over the 3 years following head injury can be illustrated graphically, as can the recovery for each severity group using all the subjects available at any one follow-up. Figures 5.2a-e provide the positive plots for the total sample A and each severity group, for each information condition (all based on the data provided in table 5.3). These graphs suggest an early recovery for sample A, followed by plateaux between 12 and 24 months, then further improvements (figure 5.2a); however, the sample included only 10 subjects at the 36-month follow-up so the latter group 'recovery' should be interpreted with caution.

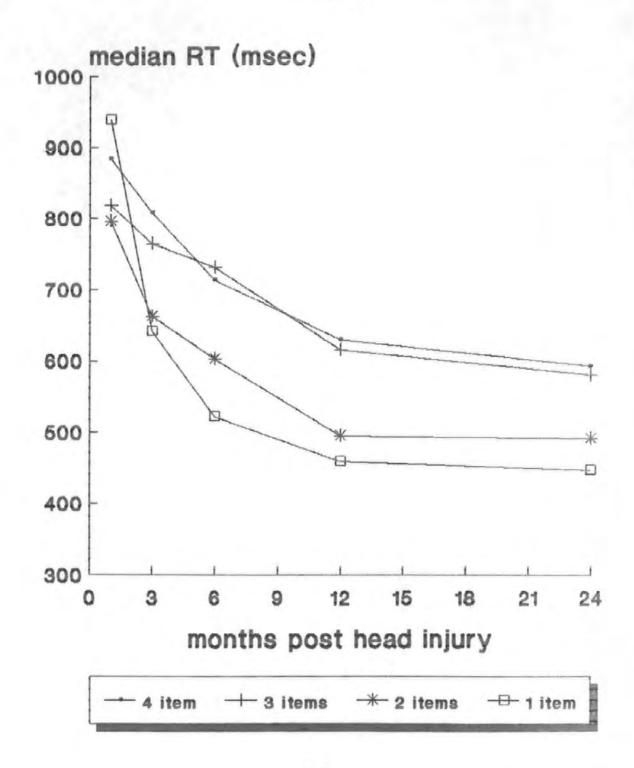
No evidence of median RT recovery was noted for M/M (figure 5.2b), and a strange pattern for VS (figure 5.2d) involving a very early, rapid recovery in RT followed by plateaux. The S subjects (figure 5.2c) early improvement, showed no consistent but recover between 3 and 24 months after head appeared to injury. Insufficient data was available on the ES group to gauge very early recovery, but figure 5.2e suggests clear improvements in median RT between 3 and 24 months post-injury.

Whatever the situation up to the 24-month point, t-test analysis provided no evidence of sample A recovery in median RT between 24 and 36 months. This finding might have arisen because the sample size was reduced to 10 subjects by the 36-month follow-up, half of whom were M/M in severity. Given this predictable large loss of sample 3 years after head injury, sample B was included to allow further group examination of RT. The results for these subjects, too, were non-significant in terms of recovery between 24 months and 36 months (all t values proving less than 1.000).

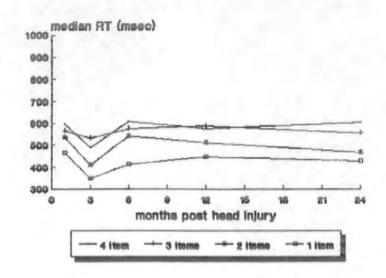
Recovery in median RT was also investigated via the nonparametric binomial test (Siegel, 1956). Using adjacent follow-up points, binomial Z values were computed from the observed frequency of improving (ie faster) median RTs between the points compared with that expected by Table 5.8 provides these values chance alone. sample A: the majority of Z values for the various information conditions between 1-3 months post-trauma, 3-6 months, and 6-12 months were significant, most at the .025 or .01 level. Comparisons carried out between the 12- and 24-month points showed a reduction number of significant values observed, and for the 24-36 month no t-value attained statistical period significance.

FIGURE 5.2: RECOVERY IN MEDIAN RT POSITIVE SET SIZES 1-4

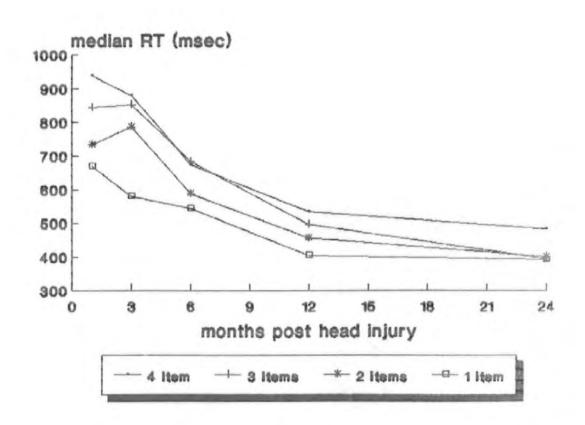
a. SAMPLE A



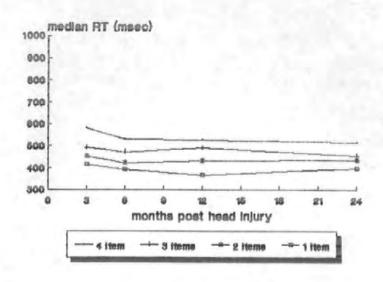
b. M/M GROUP



c. S GROUP



d. VS GROUP



e. ES GROUP

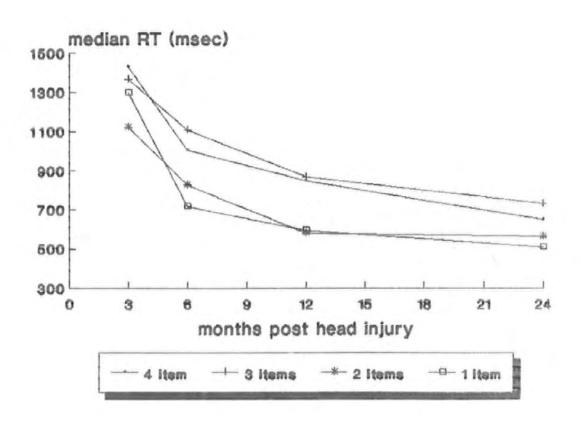


TABLE 5.8: FREQUENCY OF IMPROVEMENT IN MEDIAN RT
Follow-up Points

<u>Sample</u>	<u> </u>	<u>1-3m</u>	<u>3-6m</u>	6-12m	<u>12-24m</u>	<u>24-36m</u>		
1 item	+ve Z=	2.25**	0.20	2.43***	0.98	1.33		
	-ve Z=	2.25**	1.37	0.16	2.16**	0.67		
2 item	+ve Z=	2.58***	2.00*	2.17**	1.60*	0.00		
	-ve Z=	1.55	2.00*	2.17**	280***	0.00		
3 item	+ve Z=	2.07**	2.50***	1.01	0.98	1.33		
	-ve Z=	2.58***	2.50***	1.69*	177*	0.00		
4 item	+ve Z=	2.58***	1.60*	3.62***	0.59	0.67		
	-ve Z=	1.55	2.00*	2.76***	0.20	0.67		
*=p<.05;		**=p<.0	**=p<.025;		***=p<.01;			

In group studies, unless sample sizes are large, between-subject variability can make it difficult to demonstrate statistical significance underlying differences between groups. Indeed sensitivity to within-group variability between subjects is a major contributor to the robustness of some parametric tests, such as the t-test. In the case of head-injury studies, arguments in favour of restricting statistical analyses to those based on groups have to be set against the fact that the concept of head-injury severity based upon length of PTA is an arbitary one: the division into M/M, S, VS, and ES (table 2.3) does not have an objective logic.

It has become increasingly acceptable over the last 10 years to report data from individual cases separately, rather than just combining them into a 'group'. This approach appears particularly appropriate to a field such as head injury given that, for example, a 'S' group can include subjects whose PTA was as short as 1 day, or as long as 1 week. Similarly, an 'ES' group could contain subjects whose PTA was 8 days, 80 days, or longer. The imperfections of PTA as a severity measure argue for examination of individual subjects' scores, particularly in relation to the question of continuing cognitive recovery over a prolonged period.

In the present study, individual subject scores for sample A were examined at adjacent follow-up points to further check for evidence of continuing cognitive recovery. Similarly, sample B subject' scores at 24 and 36 months after head injury were also investigated. analysis employed was Biserial Point method of Correlation (see Garrett & Woodworth, 1958, for computation), often used in behavioural research to a subject's scores during baseline and intervention phases. In the current research at any particular follow-up point (equivalent to baseline) a subject's set of memory scanning RTs was compared with the set of RTs obtained at the next follow-up point, to check for evidence of significant change over the intervening period. The computer analysis program used generated biserial correlation coefficients (which are provided in appendix table C6.2a) and corresponding to values. The latter are displayed in table 5.8a, below, with levels of significance (a minus sign indicates a deterioration).

For comparisons of individual subjects' data at the 1and 3-month follow-ups only one ES subject accessible to testing, but as table 5.8a shows all patients achieved at least 1 significant improvement over the 8 sets (4 positive, 4 negative) of memory scanning RT data. For example, for the positive data sets, of the 15 subjects whose data were examined at 1 significant after injury, 13 showed and 3 months improvements on set size 1, 11 on set size 2, 7 on set size 3, and 11 on set size 4. Comparison of the 3- and for each subjects yields fewer 6-month data sets significant improvements, although all but 2 of the ES subjects showed significant improvements. Comparison of the 6- and 12-month data in table 5.8a shows that M/M subjects were producing fewer significant improvements performance, although some individuals (eg, case 27) yielded strong evidence of continuing gains.

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, ADJOINING FOLLOW-UPS

Positive	Trials	1.m v	Зm

		1		2		.3		4	
<u>Case</u>	<u>Gr.</u>	t-val	<u>siq</u>	<u>t-val</u>	siq	<u>t-val</u>	sig	<u>t-val</u>	siq
1	M/M	4.44	****	3.00	***	<1	ns	1.94	*
3	M/M	3.20	***	2.21	**	2.91	***	<1	ns
19	M/M	3.13	***	3.05	***	< 1	ns	<1	ns
34	M/M	3.63	***	4.61	****	3.29	***	3.01	***
42	M/M	2.23	**	2.30	**	1.61	ns	2.14	**
4	S	2.81	***	1.24	ns	<1	ns	1.92	*
5	S	3.50	****	4.48	****	2.00	*	3.04	***
6	S	3.07	***	2.75	***	<1	ns	<1	ns
9	S	5.51	****	4.06	****	4.31	****	4.00	***
10	S	<1	ns	<1	ns	<1	ns	<1	ns
2	VS	1.25	ns	3.32	***	3.10	***	2.60	***
7	vs	2.66	***	<1	ns	<1	ns	1.77	*
16	VS	5.28	****	5.09	****	4.88	***	5.26	****
23	VS	1.97	*	<1	ns	1.63	ns	2.61	***
14	ES	3.34	****	3.05	* * *	4.99	****	397	****

^{*=}p<.05; **=p<.025; ***=p<.01; ****=p<.001;

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Negative Trials, 1m v 3m

		1		2		3		4	
<u>Case</u>	<u>Gr.</u>	<u>t-val</u>	siq	<u>t-val</u>	siq	<u>t-val</u>	siq	<u>t-val</u>	siq
1	M/M	1.91	*	< 1	ns	<1	ns	1.31	ns
3	M/M	2.32	*,*	<1	ns	2.56	***	<1	ns
19	M/M	2.73	***	2.49	***	1.03	ns	2.43	***
34	M/M	2.91	***	3.22	***	2.77	***	3.01	***
42	M/M	1.59	ns	1.10	ns	3.03	***	3.02	***
4	S	1.25	ns	2.68	***	1.63	ns	<1	ns
5	s	2.21	**	4.23	****	1.04	ns	2.76	***
6	S	1.80	*	2.49	***	1.98	*	<1	ns
9	S	5.52	****	4.56	***	4.13	***	5.50	****
10	s	<1	ns	1.36	ns	<1	ns	2.03	**
2	vs	< 1	ns	1.71	*	3.67	****	< 1	ns
7	٧s	1.48	ns	3.32	***	< 1	ns	<1	ns
16	٧s	5.08	****	5.08	***	4.42	* * * *	5.58	****
23	vs	3.31	***	<1	ns	1.57	ns	2.51	***
14	ES	2.10	**	3.29	***	3.08	* *,*	2.72	***

^{*=}p<.05; **=p<.025; ***=p<.01; ****=p<.001;

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Positive	Trials	3m s	/ fim
1 00 1 6 1 0	aa away,	- JIII 1	

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	<u>siq</u>	t-val	sig	<u>t-val</u>	sig	<u>t-val</u>	siq
. 1	M/M	<1	ns	1.32	ns	2.09	**	1.32	ns
3	M/M	<1	ns	3.13	***	1,86	*	< 1	ns
19	M/M	2,39	**	2.30	**	<1	ns	<1	ns
34	M/M	<1	ns	<1	ns	1.58	ns	<1	ns
42	M/M	< 1	ns	<1	ns	<1	ns	<1	ns
4	S	<1	ns	1,19	ns	<1	ns	<1	ns
5	S	1.36	ns	4.24	****	3.46	***	4.12	***
6	S	3.98	***	1.20	ns	1.55	ns	<1	ns
9	S	<1	ns	<1	ns	1.52	ns	2.96	***
10	S	2.44	***	1.09	ns	2.16	**	< 1	ns
27	S	1.04	ns	244	***	2.57	***	<1	ns
36	S	1.86	*	1.71	*	2.15	**	3.56	****
2	VS	1.57	ns	1.05	ns	<1	ns	<1	ns
7	۷s	<1	ns	1.01	ns	<1	ns	1.14	ns
16	VS	1.92	*	229	**	3.33	***	3.22	***
20	VS	<1	ns	1.83	*	1.58	ns	2.77	***
23	vs	1.60	ns	<1	ns	<1	ns	3.42	****
29	VS	<1	ns	1.29	ns	M/E	M/E	3.62	****
35	vs	4.83	****	1.40	ns	1.42	ns	1.50	ns
*=p<.0	05;**	'=p<.02	25;***=	=p<.01;	****= <u>[</u>	o< .001	M/E=0	lata er	ror

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT IN SAMPLES A & B, AJOINING FUS (cont)

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	sig	<u>t-val</u>	siq	<u>t-val</u>	siq	<u>t-val</u>	siq
14	ES	<1	ns	<1	ns	1.36	ns	<1	ns
15	ES	M/E	M/E	4.26	****	3.46	****	<1	ns
18	ES	2.18	**	1.45	ns	<1	ns	1.57	ns
22	ES	4.30	****	4.38	****	2.13	**	3.90	****
28	ES	4.33	_***	<1	ns	1.81 -	_*	<1	ns

Negative Trials, 3m v 6m

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	siq	<u>t-val</u>	sig	<u>t-val</u>	siq	<u>t-val</u>	siq
1	M/M	1.55	ns	2.00	*	< 1	ns	< 1	ns
3	M/M	< 1	ns	<1	ns	<1	ns	1.68	ns
19	M/M	< 1	ns	2.07	**	1.49	ns	<.1	ns
34	M/M	<1	ns	2.94	***	2.40	**	<1	ns
42	M/M	2.39	**	<1	ns	1.42	ns	1.14	ns
4	S	2.25	**	2.95	***	1.28	ກຮ	3.29	***
5	S	<1	ńs	4.28	***	3.74	****	3.52	****
6	S	3.98	****	1.20	ns	1.55	ns	<1	ns
9	S	1.27	ns	1.08	ns	<1	ns	2.70	***
10	s	<1	ns	<1	ns	2.18	**	2.99	***
*=p<.05; **=p<.025; ***=p<.01; ****=p<.001; M/E=data error									

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Negative Trials, 3m v 6m (cont)	Negative	Trials.	3m v	бm	(cont)
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		1		2		3		4	
<u>Case</u>	<u>Gr.</u>	t-val	giq	t-val	sig	t-val	siq	<u>t-val</u>	<u>sig</u>
27	S	2.43	***	1.68	ns	2.27	**	<1.	ns
36	s	<1	ns	<1	ns	2.47	***	2.83	***
2	VS	<1	ns	<1	ns	2.11	**	< 1	ns
7	vs	1.51	ns	<1	ns	2.27	**	2.14	*.*
16	٧s	2.59	***	2.14	**	2.31	**	< 1	ns
20	٧s	1.37	ns	<1	ns	1.91	*	3.16	***
23	٧s	2.45	***	1.64	ns	1.50	ns	2.49	***
29	٧s	3.37	****	3.95	***	M/E	M/E	2.22	**
35	٧s	4.73	****	<1	ns	3.34	***	2.36	**
14	ES	<1	ns	<1	ns	1.42	ns	<1	ns
15	ES	M/E	M/E	2.76	***	4.93	***	1.71	*
18	ES	2.80	***	<1	ns	1.49	ns	1.75	*
22	ES	2.48	***	3.65	****	3.15	***	2.94	***
28	ES	3.09	_***	2.04	**	1.81	*	<1	ns
*=p<.05; **=p<.025; ***=p<.01; ****=p<.001; M/E=data error									rror

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT IN SAMPLES A & B, AJOINING FUS (cont)

Positive Trials, 6m v 12m

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	siq	<u>t-val</u>	siq	t-val	<u>siq</u>	<u>t-val</u>	sig
1	M/M	1.76	*	1.86	·*	1.31	ns	1.48	ns
3	M/M	1.63	ns	< 1	ns	<1	ns	<1	ns
13	M/M	2.58	***	2.10	**	<1	ns	1.96	*
17	M/M	<1	ns	2.24	**	2.17	**	1.27	ns
19	M/M	1.41	ns	<1	ns	1.07	ns	1.30	ns
24	M/M	1.84	*	2.63	* *,*	<1	ns	<1	ns
25	M/M	<1	ns	< 1	ns	2.74	***	<1	ns
27	M/M	4.65	***	2.89	***	4.09	***	3.50	****
34	M/M	<1	ns	<1	ns	<1,	ns	1.80	*
41	M/M	2.70	***	<1	ns	<1	ns	< 1	ns
42	M/M	<1	ns	1.08	ns	3.03	***	1.01	ns
4	S	<1	ns	2.40	**	<1	ns	<1	ns
5	S	5.38	***	5.34	***	5.17	***	4.26	***
6	S	<1	ns	3.12	***	2.43	***	2.89	***
11	S	3.64	***	3.27	***	2.22	**	1.88	*
26	S	1.61	ns	<1	ns	1.93	*	2.35	**
36	S	2.25	**	238	**	3.77	***	3.60	****
38	S	2.00	*	1.89	*	1.32	ns	<1	ns
*=p<.	05;	**=p<	.025;	***=p	<.01;	****=	p<.001	;	

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Positive Trials, 6m v 12m (cont)

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	sig	<u>t-val</u>	sig	<u>t-val</u>	siq	<u>t-val</u>	<u>sig</u>
2	۷s	<1	ns	<1	ns	1.15	ns	<1	ns
7	vs	<1	ns	2.81	* * *	<1	ns	<1	ns
8	VS	3.02	***	<1	ns	< ¹ 1	ns	1.61	ns
16	VS	<1	ns	1.98	_:*	4.08	_***	<1	ns
20	VS	<1	ns	<1	ns	1.64	ns	3.34	****
23	٧s	2.27	* *	<1	ns	<1	ns	4.01	****
29	VS	<1	ns	1.26	ns	M/E	M/E	1.58	ns
35	VS	2.21	**	<1	ns	3.18	***	2.01	*
39	۷s	<1	ns	<1	ns	1.03	ns	<1	ns
14	ES	1.02	ns	1.93	*	2.56	***	<1	ns
15	ES	4.60	***	5.91	***	2.72	***	2.46	***
18	ES	4.07	***	3.28	***	2.76	***	2.34	**
21	ES	4.17	***	<1	ns	<1	ns	190	*
22	ES	2.93	***	2.63	***	4.03	***	2.00	*
28	ES	5.50	****	M/E	M/E	M/E	M/E	4.97	***
30	ES	1.84	*	2.99	***	2.15	* *	1.27	ns
32	ES	1.56	ns	<1	ns	<1	ns	1.35	ns
37	ES	3.27	***	2.74	***	<.1	ns	2.09	**
40	ES	3.65	***	3.46	***	5.01	***	2.68	***
=p<.	05;	*=p<.0	25;***	=p<.01	, * * * * =	p< .001	; M/E=	data e:	rror

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Negative Trials, 6m v 12m

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	siq	<u>t-val</u>	sig	<u>t-val</u>	siq	<u>t-val</u>	siq
1 '	M/M	<1	ns	<1	ns.	1.08	ກຮ	1.62	ns
3	M/M	<1	ns	1.42	ns	1.59	ns	<1	ns
13 ·	M/M	<1	ns	3.36	****	<1	ns	<1	ns
17	M/M	<1	ns	<1	ns	<1	ns	1.00	ns
19	M/M	2.78	***	<1	ns	< 1	ns	2.00	*
24	M/M	<1	ns	<1	ns	2.00	*	2.50	*,* *
25	M/M	1.91	*	2.31	**	2.40	**	2.23	* -*,
27	M/M	2.51	***	2.66	* * *	2.25	**	1.27	ns
34	M/M	<1	ns	<1	ns	<1	ns	2.30	**
41	M/M	2.27	* *	1.13	ns	< 1	ns	<1	ns
42	M/M	1.10	ns	2.16	**	3.44	****	1.51	u'a
4	S	<1	ns	<1	ns	1.58	ns	<1	ns
5	S	5.11	****	5.00	***	4.73	****	4.70	****
6	S	1.41	ns	2.83	***	2,91	***	4.17	****
11	S	1.91	*	1.21	ns	3.02	***	<1	ns
26	S	3.76	****	<1	ns	1.08	ns	3.47	****
36	S	3.60	****	2.95	***	3.92	****	2.70	***
38	S	1.81	*	1.23	ns	1.18	ns	1.24	ns
*=p<.	05;	**=p<	.025;	***=p	<.01;	*.* * * =]	p< .001	;	

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Negative Trials, 6m v 12m (cont)

		1		2		3		4			
<u>Case</u>	<u>Gr.</u>	<u>t-val</u>	siq	t-val	<u>sig</u>	t-val	siq	<u>t-val</u>	sig		
2	VS	<1	ns	<1	ns	1.15	ກຮ	<1	ns		
7	٧s	<1	ns	2.81	***	<1	ns	<.1	ns		
8	VS	1.62	ns	<1	ns	1.03	ns	< 1	ns		
16	VS	<1	ns	2.85 -	_***	4.32 -	_***	1.61	ns		
20	vs	2.06	**	1.31	ns	3.30	***	4.48	****		
23	vs.	3.11	***	2.37	**	<1	ns	3.09	***		
29	٧s	<1	ns	<1	ņs	M/E	M/E	<1	ns		
35	VS	<1	ns	2.27	**	4,31	***	1.77	*		
39	۷s	3.56	***	<1	ns	<1	ns	<1	ทร		
14	ES	1.58	ns	2.61	***	2.67	**	1.29	ns		
15	ES	1.10	ns	4.67	***	3.84	***	1.73	*		
18	ES	1.67	ns	3.35	***	1.97	*	3.51	****		
21	ES	1.48	ns	4.10	***	1.58	ns	1.45	ກຮ		
22	ES	3.53	***	2.14	**	2.68	***	3.24	***		
28	ES	4.95	***	M/E	M/E	M/E	M/E	4.22	****		
30	ES	1.28	ns	2.80	***	2.85	***	2.01	*		
32	ES	< 1	ns	1.23	ns	<1	ns	<1	ກຮ		
37	ES	< 1	ns	3.77	***	1.02	ns	2.72	***		
40	ES	3.10	***	3.74	***	4.27	* * * *	2.71	***		
*=p<.	*=p<.05;**=p<.025;***=p<.01;****=p<.001; M/E=data error										

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Positive Trials, 12m v 24m

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	sig	<u>t-val</u>	siq	<u>t-val</u>	sig	<u>t-val</u>	siq
1	M/M	1.10	ns	<1	ns	1.86	*	<1	ns
3	M/M	2.90	***	3.31	***	1.03	ns .	1.09	ns
17	M/M	1.90	*	1.00	ns	2.73	***	<1	ns
19	M/M	1,36	ns	< 1	ns	2.09	**	1.51	ns
25	M/M	1.48	ns	1.27	ns	2.21	**	< 1	ns
27	M/M	<1	ns	1.66	ns	<1	ns	<1	ns
34	M/M	< 1	ns	1.27	ns	< 1	ns	1.65	ns
41	M/M	5.34	***	3.09	***	5.08	***	4.23	****
5	s	2.76	***	< 1	ns	1.60	ns	3.92	****
6	S	3.21	***	2.50	***	<1	ns	2.57	***
11	S	2.17	**	1.95	*	< 1	ns	1.85	*
26	s	<1	ns	1.03	ns	<1	ns	2.54	***
38	s	2.80	***	1.42	ns	3.11	***	1.18	ns
2	VS	<1	ns	2.16	**	4.06	****	2.29	**
7	٧s	1.89	*	2.51	***	<1	ns	1.34	ns
16	VS	< 1	ns	2.78	***	4.60	***	< 1	ns
20	VS	<1	ns	1.25	ns	2.77	***	2.86	***
23	vs	3.77	****	3.24	***	4.16	****	3.56	***
33	vs	2.24	**	<1	ns	2.36	**	1.45	ns
*=p< .(05;	**=p<	.025;	* * * = p<	(.01;	* * * * = 1	o< .001	;	

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Positive	m · _ 1	4.0	~ 4	
DOCTENDA	1201210	1 7 2 1 1	, '//Im	/ AANT I
FUSILIVE	111015.	1 /. 111 //	<i>-</i>	11.03111

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	siq	<u>t-val</u>	siq	<u>t-val</u>	sig	<u>t-val</u>	siq
14	ES	1.50	ns	1.86	*	1.49	ns	<1	ns
15	ES	1.56	ns	1.02	ns	₹1	ns	1.49	ns
18	ES	2.11	**	2.60	***	<1	ns	<1	ns
21	ES	<1	ns	1.71	*	1.81	*	<1	ns
22	ES	4.21	****	2.88	***	4.52	****	4.03	***
31	ES	4.50	****	M/E	M/E	1.97	*	2.15	* *
32	ES	1.84	*	<1	ns	1.76	*	183	*

Negative Trials, $12m \vee 24m$

		1		2		3		4	
Case	<u>Gr.</u>	t-val	sig	t-val	siq	<u>t-val</u>	siq	<u>t-val</u>	siq
1	M/M	1.24	ns	<1	ns	289	***	<1	ns
3	M/M	2.49	***	1.39	ns	1.40	ns	2.77	***
17	M/M	2.56	***	2.52	***	2.62	***	1.06	ns
19	M/M	<1	ns	<1	ns	1.39	ns	< 1	ns
25	M/M	1.03	ns	1.75	ns	<1	ns	2.39	**
27	M/M	<1	ns	1.50	ns	1.41	ns	< 1	ns
34	M/M	1.63	ns	1.33	ns	2.01	*	3.90	****
41	M/M	4.46	***	4.79	****	3.92	****	3.41	****
=p<.	05;	*=p<.0	25;***	=p<.01	; * * * * =]	p< .001	; M/E=	data en	rror

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Negative Trials, 12m v 24m (cont)

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	sig	<u>t-val</u>	sig	<u>t-val</u>	sig	<u>t-val</u>	sig
5	S	3.96	****	2.75	***	<1	ns	2.67	k**
6	s	1.93	*	< 1	ns	1.47	ns	<1	ns
11	s	2.53	***	2.19	**	< 1	ns	<1	ns
26	s	3.19	***	1.11	ns	1.29	ns	3.27	***
38	s	2.17	**	2.36	**	2.07	*	183	*
2	vs	1.25	ns	3.26	***	2.33	**	<1	ns
7	٧s	1.51	ns	<1	ns	1.31	ns	< 1	ns
16	vs	2.21	**	2.92	***	4.62	****	<1	ns
20	٧s	1.66	ns	1.67	ns	3.38	***	4.08	****
23	VS	4.89	***	3.39	****	4.11	***	4.83	***
33	vs	2.27	**	1.23	ns	3.17	***	2.53	* *.*
14	ES	1.61	ns	1.97	*	<1	ns	1.13	ns
15	ES	5.75	***	2.47	***	<1	ns	3.34	****
18	ES	2.35	**	2.76	***	<1	ns	2.50	***
21	ES	<1	ns	<1	ns.	<1	ns	2.69	***
22	ES	1.80	*	1.91	*	3.79	*.* *.*	1.23	ns
31	ES	5.12	****	M/E	M/E	<1	ກຮ	2.16	**
32	ES	2.03	*	1.04	ns	1.55	ns	2.55	***
=p<.	05;	*=p< .0	25;***	=p<.01	; * * * * <u>=</u>	p<.001	; M/E=	data en	rror

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Positive Trials, 24m v 36m

		1		2		3		4	
Case	<u>Gr.</u>	<u>t-val</u>	siq	t-val	siq	t-val	siq	t-val	siq
1	M/M	< 1.	ns	1.59	ns	<1	ns	1.89	*
3	M/M	1.54	ns	2.58	***	< 1	ns	2.46	***
17	M/M	1.11	ns-	1.14	ns	1.66	ns	2.03	*
34	M/M	1.49	ns	<1	ns	1.45	ns	2.32	**
5	S	2.80	***	1.26	ns	2.56	***	1,.49	ns
11	S	1.63	ns	<1	ns	1.28	ns	<1	ns
1.5	ES	<1	ns	<1	ns	3.23	***	<1	ns
18	ES	<1	ns	<1	ns	2.61	***	<1	ns
21	ES	1.02	ns.	<1	ns	2.13	**	< 1	ns
B 2	M/M	<1	ns	2.22	**	1.22	ns	<1	ns
В 9	M/M	2.40	* *	<1	ns	< 1	ns	2.56	***
В 7	S	4.35	_****	2.01	_*	3.43	_***	4.88	_***
B 1	٧s	2.97	***	1.49	ns	<1	ns	2.72	****
B 4	vs	1.52	ns	1.79	*	2.08	**	2.40	***
B 5	VS	2.05	* *	<1	ns	< 1	ns	< 1	ns
B 6	٧s	2,92	***	2.48	***	2.36	**	2.93	***
В 3	ES	1.74	*	1.18	ns	<1	ns	<1	ns
В 8	ES	3.49	***	1.99	*	M/E	M/E	3.33	***
B10	ES	3.61	***	3.21	***	<1	ns	4.02	****
=p<;(05;	*=p<.0	25;***	=p<.01	; * * *·*=	p<.001	; M/E=	data e:	rror

TABLE 5.8a: BISERIAL POINT ANALYSIS, FOR EACH SUBJECT

IN SAMPLES A & B, AJOINING FUS (cont)

Negative Trials, 24m v 36m

		1		2		3		4	
Case	<u>Gr.</u>	t-val	siq	<u>t-val</u>	siq	<u>t-val</u>	sig	<u>t-val</u>	siq
1	M/M	<1	ns	1.61	ns	<1	ns	1.34	ns
3 [.]	M/M	<1	ns	1.90	*	<1	ns	1.38	ns
17	M/M	2.20	**	1.52	ns	<1	ກຮ	<1	ns
34	M/M	3.19	***	< 1	ns	4.27	***	<1	ns
5	S	1.74	*	<1	ns	3.06	***	1.76	*
11	S	< 1	ns	< 1	ns	2.59	***	1.88	*
15	ES	2.60	***	1.09	ns	<1	ns	2.71	***
18	ES	1.46	ns	3.47	****	1.79	*	<1	ns
21	ES	<1	ńs	<1	ns	2.62	***	<1	ns
B 2	M/M	<1	ns	<1	ns	1.81	*	<1	ns
B 9	M/M	<1	ns	<1	ns	<1	ns	<1	ns
B 7	S	3.91	_***	2.45	_***	3.03	_***	4.23	_***
B 1	VS	1.36	ns	2.23	**	1.15	ns	<1	ns
B 4	VS	1.40	ns	2.62	***	2.24	**	3.02	***
B 5	VS	2.19	**	<1	ns	1.78	* .	1.21	ns
B 6	VS	3.84	***	1.87	*	4.23	****	2.20	* *
вз	ES	1.77	*	1.37	ns	<1	ns	< 1	ns
B 8	ES	1.28	ns	1.59	ns	M/E	M/E	1.40	ns
B10	ES	1.69	ns	4.10	***	<1	ns	4.03	***

*=p<.05; **=p<.025; ***=p<.01; ****=p<.001; M/E=data error

Between 6-12 months after trauma S subjects continued to show large gains, including the high information conditions (eq. cases 5, 6, 36). The performances of VS subjects were less impressive, and variable: case 2 provided no evidence of significant improvement between 12 months, case 16 showed significant deterioration, whereas cases 20 and 23 performed well. More ES subjects were available for the 6-12 month interval, and strong evidence of individual recovery is reflected in the data in table 5.8a (eg, cases, 15, 18, 22. 28. 40).

Between 12-24 months Table 5.8a shows that evidence of significant recovery was noted for most M/M individuals, with case 41 being particularly impressive. Similarly, in the S patients case 5 produced some large gains, as did case 23 in the VS group, and case 22 in data in table 5.8a for the 12-24 The group. interval offers good support for significant improvement in individual subjects' RT performance occurring during this period. Similarly, although group analyses for the 24-36 month interval did not support continuing recovery, the data for individuals for the significant improvement suggests that was still Although an M/M subject, case 3 showed occurring. significant gains in RT memory scanning performance

between 24 and 36 months post-injury, as did case 5 from the S group. The ES cases 15 and 18 also produced some highly significant gains. Within the B sample of individuals, case B10 showed consistent evidence of deterioration, although other subjects generally produced significant improvements between 24 and 36 months.

Overall, the analysis of individual subjects' data yields much stronger evidence for continuing recovery beyond 12 and 24 months post-injury than the group data.

The effect of severity of head injury upon recovery was by examining median further assessed RT for available subjects at each follow-up in the severity groups. Table 5.9 summarises the t-analyses, and shows that the only consistent findings at 1 month (given that most ES subjects were still in PTA, and therefore not tested) were that S subjects produced slower median RTs than the M/M group. At the 3-month follow-up the ES group generally showed significantly slower RTs than the other groups. As pointed out above, the VS performed similarly to the M/M subjects, and the group's RTs were significantly slower than both. By 6 months post-trauma median RT differences between M/M

TABLE 5.9: MEDIAN RT. t-TESTS ON SAMPLE A

Positive Set

1/12 F	<u>U</u> :		<u>1</u>	<u>2</u>	<u>3</u>	4
M/M(8)	v	S'(7)	2.608**	2.864***	2.777***	2.626**
M/M	v	VS(6)	1.967*	1.328	1506	1.491
S	v	VS	1.569	<1	<1	<1
3/12 F	<u>U</u> :					
M/M(5)	v	S(7)	2.601**	2.072*	1.925*	2.481**
M/M	v	VS(9)	1.250	<1	<1	<1
M/M	v	ES (.6)	2.336**	3.879****	2.842***	3.27****
S	v	VS	2.059*	2.140*	2.882***	2.068*
S	v	ES	2.067*	1.316	1.842*	1.886*
VS	v	ES	2.400**	3.754****	3.121****	2.947***
6/12 F	<u>U</u> :					
M/M(11)·v	S(10)	1.740*	<1	<1	<1
M/M	v	VS (9)	<1	<1	<1	<1
M/M	v	ES(6)	3.128****	2.396**	3.004****	2.421**
S	v	VS	2.217**	2.365**	2.261**	2.283**
S	v	ES	1.647	2.555**	2.887***	2.523**
VS	v	ES	3.591****	5.555****	5.155****	4.07****
*=p<.0	5;	**	=p<.025;	***=p<.0	1; ****=	p<.005;

and S were smaller, though S group results were still significantly slower than those from the VS group (Table 5.9). Again, ES RTs were significantly poorer than those of the other groups. At 12- and 24-months

after injury ES RTs continued significantly slower than those of S and VS subjects.

TABLE 5.9: MEDIAN RT, t-TESTS ON SAMPLE A (cont)

Positive Set

						
12/12	FU	: .	1	<u>2</u>	<u>3</u>	<u>4</u>
M/M(10).v	S(8)	<1	<1	<1	<1
M/M	v	VS(10)	1.033	<1	<1	<1
M/M	V	ES(11)	1.670	<1	1.566	1.969*
S	v	VS	1.133	<1	<1	<1
S	v	ES	2970****	2.402**	2.488**	2.92****
VS	v	ES	4.069****	3.300****	2.799***	3.34***
24/12	<u>FU</u>	:				
M/M(7)	v	S(10)	<1	<1	1.605	1.331
M/M	v	VS(8)	< 1	<1	1.194	1.189
M/M	v	ES(7)	1.041	1.569	1.265	<1
s	v	VS	<1	<1	1.272	<1
S	v	ES	1.475	3.070****	2.460**	1.481
VS	v	ES	1.802*	2.557**	2.491**	1.485

Negative Set

<u>1/12 FU</u> :		<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
M/M(8) v	\$(7)	2.698***	3.920****	2.740***	1.985*
M/M v	VS(6)	2.005*	1.317	1.451	1.234
s v	vs	1.395	<1	<1	<1
*=p<.05;	**:	=p<.025;	***=p<.0	1; ****=	p<.005;

TABLE 5.9: MEDIAN RT, t-TESTS ON SAMPLE A (cont)

Negative Set

3/12 F	<u>U</u> :		<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
M/M(5)	٧	S(7)	2.489**	1.982*	2.091*	1.870*
M/M	v	VS(9)	< 1	<1	<1	< 1
M/M	v	ES(6)	3.175***	3,559****	3.156***	3.227***
S	v	vs	2.071*	1.765*	2.515**	1.173
S	v	ES	2.242**	1.504	1.841*	1.926*
VS	v	ES	3.202****	3.231****	3.246****	2.731***
6/12 F	Ŭ:					
M/M(11) v	S'(10)	1.589	<1	<1	<1
M/M	Ÿ	VS(9)	<1	<1 '	<1	<1
M/M	v	ES(6)	3.596****	2.722***	3.007****	2.906***
S	V	VS	2.069*	2.187**	2.511**	1.940*
S	٧	ES	2.143*	2.667***	3.429****	3.05****
VS	v	ES	4.291****	4.675****	6.063****	5.05****
12/12	<u>FU</u>	:				
M/M(5)	v	S(8)	<1	<1	<1	<1
M/M	v	VS(10)	<1	<1	<1	<1 .
M/M	v	ES (11)	2.264**	<1	1.535	1.652
S	v	VS	< 1	<1	<1	<1
S	v	ES	3.256****	3.646****	2.488**	2.328**
VS	v	ES	4.152****	3.092****	2.250**	4.27****
*=p<.0	5;	**:	=p<.025;	***=p<.0	1; ****=	p<.005;

TABLE 5.9: MEDIAN RT. t-TESTS ON SAMPLE A (cont)

Negative Set

24/12	FU:	:	1	2	3	4
M/M(7).	Ÿ	S (5)	<1	<1	1.054	1.023
M/M	v	VS(8)	<.1	<1	1.027	<1
M/M	v	ES(7)	<1	1.453	1.032	<.1
S	V,	vs	< 1	<1	<1	<1
S	v	ES	1.717	2.817***	1.920*	1.564
VS	v	ES	1.914*	3.176***	2.199**	5.05****
*=p<.0	5 ;	**:	=p<.025;	***=p<.	01; ***	**=p<.005;

Finally, the relationship between median RT and severity of head injury was investigated via its correlation with length of unconsciousness (U/C) and PTA duration. The values at each follow-up are summarised in Table 5.10. Although no significant correlations between RT and PTA, and between RT and U/C were noted 1 month after head injury, strong correlations (the large majority significant at the .01 level) between the 2 severity variables and RT were obtained at the 3-month and 6month follow-up points. These relationships, although significant, began to weaken by the still 12-month follow-up, and at the 24-month assessment no correlations achieved significance. However, at 36 months post-trauma, admittedly with a much reduced

TABLE 5.10: CORRELATIONS OF MEDIAN RT WITH LENGTH OF

UNCONSCIOUSNESS (U/C) & PTA, SAMPLE A

		F	ositive	Set		Neg	gative	Set	
<u>A</u>		1	2	3	4	1	2	3	4
1m	U/C:-	.13	11	.08	.07 -	09 -	07 -	07 -	. 09
	PTA:	. 22	. 11	.09	. 11	.22	.11	.08	. 08
Зm	U/C:	.78**	.63**	.67**	.69**	.82**	.63**	.64**	.66**
	PTA:	.53**	.47*	.51**	.52**	.60**	.48*	.49**	.50**
6m	U/C:	.46**	.44**	.11	.46**	.48**	.48**	.08	.48**
	PTA:	.44**	.40**	. 17	.45**	.44**	.44**-	02	.43**
12m	U/C:	.41*	.49**	.36*	.44**	.36*	.49**	.33*	.41*
	PTA:	. 35*	. 33*	. 31*	.39*	. 37*	.33*	. 28	. 37*
24m	U/C:	. 05	.12	.14	. 04	.01	.17	.14	. 05
	PTA:	. 17	.18	. 23	.07	.09	.21	.18	.12
36m	U/C:	. 38	.75*	.60	.62	.67*	.80**	.69*	.62
	PTA:	. 33	.71*	. 58	. 59	.65*	.79**	.68*	. 60
<u>B</u>									
24m	U/C:-	. 05	.28 -	05	.02	.01	. 23 -	06	. 20
	PTA:	. 31	. 46	. 31	. 34	. 35	. 41	. 31	. 37
36m	U/C:-	.18	.10 -	-,05	.11 -	13	.15	.09 –	. 04
	PTA:	.11	.31	. 15	. 24	. 18	. 38	. 35	. 15
*=p<	(.05;	y	*=p<.01	l;					

sample size, sample A subjects showed a majority of significant correlations with U/C and PTA. Sample B's 24-month median RT correlations with U/C were small and

similar to those observed in sample A, although the former's correlations with PTA were somewhat larger (though still non-significant). Differences between the 2 samples were very apparent at 36 months post-injury where sample B's RT correlations with both U/C and PTA were much lower.

How do the median RTs of subjects in samples A and B compare with those produced by non brain-damaged people? As described earlier in this chapter, a sample (C) of volunteer NHS staff was recruited. Besides offering some kind of 'calibration' for what might be considered 'normal' performance using the specific hardware and software configuration of the present study, sample C also allowed some investigation of possible practice effects which might be operating. Sample C was tested on 4 occasions, at approximately 2-week intervals, provide a rigorous test of the 'practice' hypothesis. The median RT data for this sample are provided in Table 5.5. The table shows that out of 10 subjects, only 6. attended for the final testing session, and for set sizes 3 and 4 at the first session the sample was only 9, due to experimenter error. A check for practice effects operating on RT yielded no evidence of phenomenon - the large majority of t-tests produced values under 1.000, with only 2 out of 48

significant. Given no systematic differences between the median RT data obtained from the 4 sessions, it was necessary to identify the results from one session to compare with those gained from the 2 patient samples. The RT data from session 4 was based on only 6 subjects, so this was excluded, and on the flip of a coin session 3 data was selected.

Table 5.11 reflects significantly slower RTs for the patients in sample A, tested against sample C subjects, for all comparisons conducted at the 24 month follow-up. None of the 24-month t-test comparisons involving the S group from sample A with sample C achieved significance, and only 2 of the 8 comparisons involving VS subjects sample C attained significance (.05 level). However, as table 5.11 indicates ES median RT scores were grossly slower than those obtained from sample C. and M/M median RTs at 24 months post-trauma were also generally significantly poorer. The equivalent results for sample B-C comparisons at this follow-up were less striking than those noted in relation to sample A, 36-month point all B-C comparisons although at the proved significant. Table 5.11 also shows that differences between A and C weaken by 36 although as figure 5.2f indicates both patient samples continue to perform below control subjects' level.

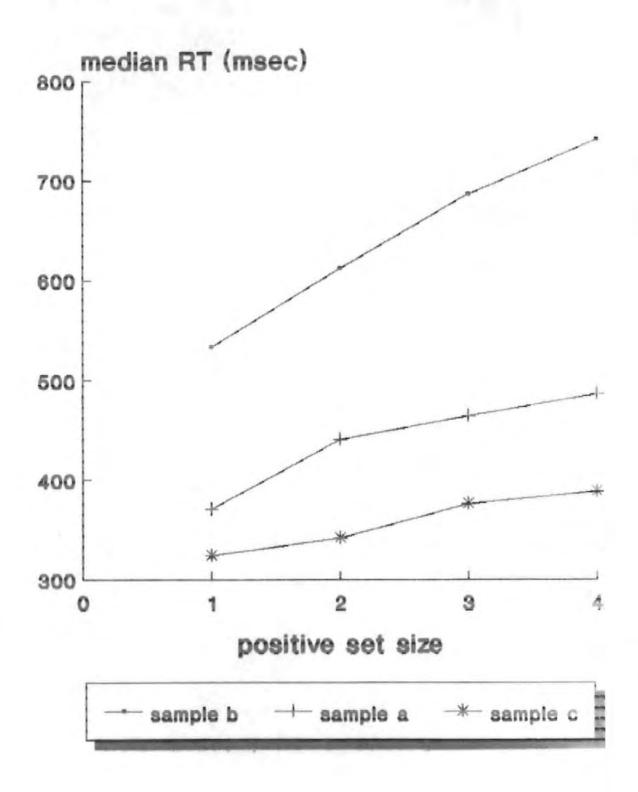


TABLE 5.11: MEDIAN RT, t-TESTS ON SAMPLES A, B & C

Positive Set

<u>24/12 FU</u> :	1	2	. 3	4				
A (27) v C(10)	2334**	2.448**	2.907****	2.358**				
B (10) v C(10)	1.398	2.726***	3.682****	1.654				
M/M(7) v C(10)	2.213**	1.425	2.256**	1.768*				
ES (7) v C(10)	3.924****	3.232****	3.512****	2.769**				
<u>36/12 FU</u> :								
A (10) v C(10)	1.642	2.121**	1.817*	1.300				
B (10) v C(10)	2.396**	2.634**	2.666***	2.737***				
Negative Set								
24/12 FU:	1	2	3	4				
24/12 FU: A (27) v C(10)	1							
	1 3.250****	2.939****						
A (27) v C(10)	1 3.250**** 1.757*	2.939**** 1.421	3.052**** 1.908*	3.371****				
A (27) v C(10) B (10) v C(10)	1 3.250**** 1.757* 2.850***	2.939**** 1.421 2.087*	3.052**** 1.908* 2.088*	3.371**** 1.539				
A (27) v C(10) B (10) v C(10) M/M(7) v C(10)	1 3.250**** 1.757* 2.850***	2.939**** 1.421 2.087*	3.052**** 1.908* 2.088*	3.371**** 1.539 2.937***				
A (27) v C(10) B (10) v C(10) M/M(7) v C(10) ES (7) v C(10)	1 3.250**** 1.757* 2.850*** 4.314***	2.939**** 1.421 2.087* 3.598***	3.052**** 1.908* 2.088* 3.644***	3.371**** 1.539 2.937***				
A (27) v C(10) B (10) v C(10) M/M(7) v C(10) ES (7) v C(10) 36/12 FU:	1 3.250**** 1.757* 2.850*** 4.314****	2.939**** 1.421 2.087* 3.598****	3.052**** 1.908* 2.088* 3.644****	3.371**** 1.539 2.937*** 4.211****				

In section 5.1 it was hypothesised that both positive and negative plots of median RT under increasing positive set size could be described via parallel linear functions, with the positive RTs being faster. The question of linearity is dealt with below (5.4.3), and

trials would be faster is supported by the data shown in figure 5.1 and presented in table 5.3. The latter shows that at the 1-month follow-up in only 4 (out of conditions) were the negative trials median RTs faster. Three of these were observed in the VS group, which presented a very disorganised profile at the first follow-up with no good evidence of a linear rise in median RT under increasing information load. The very long positive median RT for 1 item (1932 msec) in this group caused the sample A value (938) to exceed the With corresponding negative time (836). these few exceptions, all remaining positive median RTs were faster than their negative counterparts.

months post-trauma, 6 positive (of 20) median RTs the corresponding negative values. exceeded Half of these originated in the ES group, which both showed very long latencies and the absence of the expected linear relationship between set size and median RT. By the 6months point only 1 value was slower than its negative partner, a finding which also held for the 12-month follow-up. Αt 24 and 36 months post-trauma no positive median RT exceeded its negative counterpart in sample A, this finding being paralleled in the results obtained for the normal subjects in sample C.

Table 5.12 displays the mean differences between positive and negative median RTs. For those information conditions where positive trials produced faster responses the differences across information condition average out at about 50 msec for samples A (57 msec), B (47 msec) and C (49 msec).

TABLE 5.12: AVERAGE DIFFERENCES IN MEDIAN RT

BETWEEN POSITIVE & NEGATIVE TRIALS

Number of Items Scanned

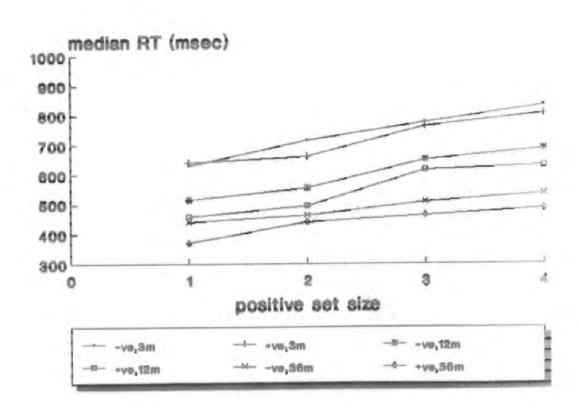
			1	2	3	4
<u>A</u>	1/12	FU:	-102	1	15	74
	3/12	FU:	- 11	53	13	26
	6/12	FU:	47	54	4	67
	12/12	FU:	57	60	34	56
	24/12	FU:	59	61	54	87
	36/12	FU:	34	29	50	84
<u>B</u>	24/12	FU:	-197	42	59	-280
	36/12	FU:	44	- 4	7.6	12
<u>C</u>	1st	FU:	2	6	44	23
	2nd	FU:	56	56	50	46
	3rd	FU:	48	76	50	69
	4rd	FU:	73	41	74	69

(nb: a minus sign indicates a faster negative trial)

A central tennet of the Exhaustive Scan hypothesis

(chapter 3) is that the positive and negative plots of RT against increasing number of items to be scanned will remain parallel (ie, self-terminating serial scanning of items will not occur). Inspection of table 5.12 and figure 5.3 provides no convincing evidence that the RT advantage on positive trials increases as the positive set size rises, and it therefore supports the exhaustive, rather than self-terminating, scanning position.

FIGURE 5.3: POSITIVE & NEGATIVE MEDIAN RT,
3-12-36 MONTH FOLLOW-UPS, SAMPLE A



Analysis of median RT and variability of RT concentrated upon correct responses only, for good theoretical There is evidence (Welford, 1980a) that error reasons. responses are faster, as subjects may have not processed the information fully. Table 5.13 below provides data errors according to type of set (+/-), and occurence of 'faster-than-median' errors. The data in table 5.13 does not relate to the frequency of observing errors per trial, but rather the number of runs (+/-), 20 trials, on which an error occurred. each of It can be seen that for all 3 subject samples, at all sessions, an error was more likely to occur on a positive set of trials.

Table 5.13 also shows that there was a tendency for the probability of an error on a run to be lower for the subjects in sample C. If errors occurred mainly through attenuated information processing by subjects so that they could produce faster responses, then the data for the frequency of error RTs faster than median RT should be higher than 50% (chance level). The data in table 5.13 offers no support for the hypothesis that error RTs would be faster than correct response RTs: the frequency of error RTs being faster than the median RT for sample A approximated chance level for both positive (45%) and negative (53%) sets, for sample B the values were less

than 50% for both (38%, 32%), and for sample C the rates were 53% and 37%, respectively. The frequency of errors

TABLE 5.13: OCCURRENCE OF ERROR(S) BY SET TYPE

		Type of	set where	Runs pr	oducinq
		error (s) occurred	faster	errors
<u>Gr</u> .	<u>FU</u>	pos.	nėg.	pos.	neg.
<u>A</u> :	1/12	37%	18%	55%	50%
	3/12	50%	38%	49%	46%
	6/12	39%	33%	44%	55%
	12/12	51%	34%	39%	50%
	24/12	54%	32%	43%	41%
	36/12	48%	32%	42%	75%
<u>B</u> :	24/12	64%	39%	36%	53%
a.	36/12	58%	23%	39%	11%
<u>C</u> :	1	34%	26%	69%	10%
	2	39%	15%	60%	33%
	3	36%	29%	50%	36%
	4	34%	14%	33%	67%

per se was extremely small: the probability of an error for sample A subjects was .03 (M/M, S, VS) to .04 (ES). In addition, where they did occur, the majority involved 1 or 2 error responses on any trial. For sample C the probability of an error was .02.

The above results indicate that the probability of an error was very small and that overall the reason for an error being produced was not related to a faster RT on that trial. The question remains as to why errors occur. One possibility is that as a subject's attention or concentration varies during a run, then 'flat spots' or fluctuations downwards, will be immediately followed by poorer information processing and the probability of an error will rise. If this explanation has validity, then it would be expected that longer-than-average RTs be noted for the 1 or 2 trials immediately would preceding the trial on which an error was produced. Alternatively, it might be that for the 1 or 2 trials preceding an error trial a subject is sustaining concentration at a particularly high level (with the attendant probability of faster-than-average RTs these trials). Using this explanation, the subsequent error trial represents the waning of the above-average attention. Subjects' raw data in samples A and C were examined to explore these explanations. Table 2 in appendix C6 displays the relevant results for the situation where only 1 error was produced on a (including runs with more errors could lead to problems of interpretation, particularly if the preceding trial had produced an error).

The data offers support for the idea that an error is more likely to follow a period of good concentration. For the pair of trials immediately preceding an error trial, in 43% of cases both RTs were faster than the median RT(s) for that run, in only 17% of cases were both RTs slower than the median(s), leaving 40% where one was faster and one slower. Support for this explanation was also provided by sample C subjects where 40% of errors followed a pair of RTs which were faster than the appropriate median(s), and only 12% were preceded by two slower-than-median RTs. This finding is quite tentative and the general issue of the production of errors and their prediction is a large topic beyond the scope of this thesis.

5.4.3 RT Regression Equations.

The work of Sternberg and others has suggested that memory scanning behaviour can be modelled as a straight line function. The predictive equation would then have the form:

RT = BX + C

where - B is the slope weight, C is the intercept, and

X is the number of memory items to be scanned

A potentially useful line of enquiry is the analysis of recovery in median RT in terms of the 'goodnes of fit'

of the data to a linear function using the correlation coefficient. Change over time can also be investigated for the weight and intercept variables in the equation. Raw scores for these variables are provided in appendix tables C7.1-C7.3, and group scores for the samples are shown in table 5.14 below.

Table 5.14 shows that between 3 months and 12 months the positive weight lay in the 65-68 msec range for sample A, falling to 52 msec and 44 msec at the 24 month and 36 month follow-ups, respectively. The negative weight fell in a more stepwise fashion between 3 months (78 msec.) and 24 months (60 msec.). From 3 months onwards the discrepancy between the positive and negative weights was never more than 10 msec. This nature of the positive confirms the parallel negative plots and indicates support for the Exhaustive Scan hypothesis. Table 5.14 illustrates that, once again, the VS group behaved very similarly to the M/M From the 3-month to the 24-month follow-up inclusive, the pattern for nearly all of the positive and negative weight values showed the highest were produced by the ES group. This feature is reflected in a number of significant t-test results when comparing the slope weights of ES subjects with those in other severity groups across the 3-24 month period (Table

5.15), supporting the hypothesis that the most severely head-injured subjects would show a differential penalty in RT with increasing processing load, and therefore a Over the same period the positive and steeper slope. negative intercepts invariably showed the ES group to have the largest values, as would be expected from the analysis of median RT and severity provided in section The t-values shown in table 5.15 support this 5.4.2. finding, particularly those carried out at 6, 12, and 24 months after head injury. After the 1-month point the correlation coefficients for linearity in sample A fell within the range +0.75 to +0.84. By 24 months the 'fit' for the S group was extremely good (+0.89/+0.93), was good for the M/M group (+0.84/+0.76), and was slightly lower for the ES group (+0.71/+0.79). The aberrant VS group showed a high correlation for negative set items (+0.89) and a poorer correlation for positive items (+0.64).

Figure 5.4a-d presents the linear regression-derived graphs for the M/M, S, and ES groups (positive plot) at follow-ups 3-24 months. The VS subjects are omitted, given the similarity of their results to those in the M/M group. Figure 5.4e provides the same plot for the patient samples at 36 months post-injury, and for the control subjects in sample C.

TABLE 5.14: MEDIAN RT REGRESSION VALUES FOR

SAMPLES A & B AT EACH FU, & C

		Positiv	ve Set		Negativ	ze Set	
1/12	? FU	Weight	Intercept	Corr.	<u>Weight</u>	Intercept	Corr.
<u>A</u>	mean:	34	186	. 68	79	629	. 78
(n=2	22)sd:	191	1364	. 49	80	552	. 24
<u>M/M</u>	mean:	43	433	. 55	68	419	,82
(8)	sd:	43	158	.50	46	122	.16
<u>s</u>	mean:	91	570	. 83	120	554	.76
(7)	sd:	61	91	.10	116	157	.16
<u>vs</u>	mean:	-103	353	. 55	57	948	. 65
(5)	sd:	353	76	.75	25	1044	. 38
3/12	? FU						
<u>A</u>	mean:	68	545	.76	78	540	. 81
(26)	sd:	114	543	, 31	72	280	. 17
<u>M/M</u>	mean:	54	309	. 87	52	377	. 81
(5)	sd:	37	82	.09	47	93	. 14
<u>s</u>	mean:	97	532	. 79	77	591	. 79
(7)	sd:	54	258	.14	50	202	. 21
<u>vs</u>	mean:	55	351	. 81	64	412	. 84
(9)	sd:	53	121	.13	59	89	.13
ES	mean:	62	1149	. 49	129	859	. 77
(5)	ed:	236	957	. 57	107	408	. 20

TABLE 5.14: MEDIAN RT REGRESSION VALUES FOR

SAMPLES A & B AT EACH FU & C (cont)

	Positi	ve Set		Negativ	e Set	
6/12 FU	Weight	Intercept	Corr	Weight	Intercept	Corr.
<u>A</u> mean	: 68	464	. 75	71	508	. 82
(n=41)sd	: 61	202	. 33	57	211	. 17
<u>M/M</u> mean	: 63	380	. 85	69	410	. 88
(11) sd	: 83	134	. 20	75	142	. 15
<u>s</u> mean	: 47	503	.68	54	525	. 76
(10) sd	: 26	206	. 36	27	169	. 22
<u>VS</u> mean	: 47	338	. 74	46	392	. 82
(9) sd	: 26	104	.51	16	76	. 16
<u>ES</u> mean	: 111	615	.73	114	694	. 81
(11) sd	: 56	207	. 18	60	263	. 11
12/12 FU						
<u>A</u> mean	: 65	399	. 78	63	454	. 78
(38) sd	: 56	162	. 30	60	149	. 26
<u>M/M</u> mean	: 46	413	. 60	5 <i>7</i>	435	. 73
(10) sd	: 60	241	. 50	73	180	. 22
<u>S</u> mean	: 46	396	. 87	46	412	. 93
(8) sd	: 19	119	.09	26	55	. 04
<u>VS</u> mean	: 58	314	. 87	43	409	. 79
(10) sd	: 32	49	.14	15	78	. 20
<u>ES</u> mean	: 103	546	.82	83	551	. 69
(10) sd	: 55	1.85	. 13	64	175	. 36

TABLE 5.14: MEDIAN RT REGRESSION VALUES FOR

SAMPLES A & B AT EACH FU & C (cont)

Positive Set

Negative Set

24/	12 FU	Weight	Interce	<u>ept</u>	Corr.	Weight	Intercept
Cor	<u>r</u> .					•	
<u>A</u>	mean:	52	396	. 77	60	442	. 84
(n=	26)sd:	40	122	. 27	44	145	5 .23
<u>M/M</u>	mean:	62	361	. 84	65	439	. 78
(6)	sd:	38	118	. 24	28	194	. 40
<u>s</u>	mean:	56	352	. 89	44	380	. 93
(5)	sd:	34	80	. 07	12	64	.06
<u>vs</u>	mean:	32	357	. 64	46	401	89
(8)	sd:	23	73	.40	10	71	11
<u>ES</u>	mean:	59	465	. 71	76	483	3 .79
(7)	sd:	49	103	. 19	63	102	. 15
<u>B</u>	mean:	146	612	. 82	91	554	. 87
(10) sd:	173	641	. 15	60	439	. 07
36/	12 FU						
<u>A</u>	mean:	48	366	. 79	65	380	. 88
(10) sd:	37	99	. 26	61	84	. 14
<u>B</u>	mean:	76	486	. 84	72	466	. 82
(10) sd:	53	258	. 22	29	344	. 20
<u>C</u>	mean:	33	302	. 86	35	349	. 88
(10) sd:	17	34	.12	17	44	1 .10

TABLE 5.15: MEDIAN RT REGRESSION t-TESTS FOR SAMPLES A, B & C

Positiv	e Set		Negative Set			
1/12 FU Weight	Interc.	Corr.	<u>Weight</u>	Interc.	Corr.	
M/M v S 1.781	2.015*	1.450	1.172	1.873*	<1	
M/M v VS 1.188	<1	< 1	<1	1,128	1.137	
S v VS 1.452	4.012***	* <1	1:297	< 1	<1	
3/12 FU						
M/M v S <1	2.139*	1.116	<1	2.454**	<1	
M/M v VS <1	<1	<1	<1	< 1	<1	
M/M v ES 1.159	1.957*	1.473	<1 .	<1	<1	
S v VS 1.534	1.713	<1	<1	2.179**	<1	
S v ES <1	1.407	1.361	1.139	1.517	<1	
VS v ES 1.144	1.857*	1.659	1.488	3.25***	<1	
<u>6/12 FU</u>						
M/M v S <1	1.638	1.355	<1	1.694	1.473	
M/M v VS <1	<1	<1	<1	< 1	<1	
M/M v ES 1.590	3.16****	1.479	1.554	3.15***	<1	
S v VS <1	2.163**	<1	< 1	2.167**	<1	
S v ES 315***	*1.241	<1	2.90***	*1.731*	<1	
VS v ES 3.15***	*3.89****	<1	3.45***	*3.48***	<1	
*=p<.05; **=	p<.025;	***=	p<.01;	****=p	< .005;	
Interc.= interc	ept;	Corr	.= corr	elation;		

TABLE 5.15: MEDIAN RT REGRESSION t-TESTS

FOR SAMPLES A, B & C (cont)

	Positiv	e Set		Negative Set			
12/12 FU	Weight	Interc.	Corr.	<u>Weight</u>	<u>Interc.</u>	Corr.	
M/M v S	<1	<1	1.499	< 1	<1	2.523**	
M/M v VS	S < 1 _.	<1	1.644	<1	< 1	< 1	
M/M v ES	2.215**	1.360	1.347	< 1	1.461	<1	
s v vs	3 <1	. <1	<1	<1	<1	1.938*	
s v Es	2.787**	*1.982*	<1	1.530	2.152**	1.865*	
VS V ES	3 2,236**	3.834***	* <1	1.924*	2.344**	< 1	
24/12 FU	1						
M/M v S	1.731	<1	<1	1.622	<1	< 1	
M/M v VS	1.128	<1	1.081	1.562	< 1	<1	
M/M v ES	3 <1	1.767	1.090	< 1	< 1	< 1	
s v Vs	1.037	<1	1.362	<1	<1	<1	
S v ES	1.252	2.147*	2.000*	1.275	2.150*	1.956*	
VS v ES	S < 1	2.314**	<1	1.220	1.800*	1.486	
A v E	3 1.674	1.047	<1	1.359	<1	< 1	
A v C	1.382	2347**	<1	1.676	1.941*	< 1	
Bvo	2.056*	1.527	<1	2.840**	1.469	< 1	
36/12 FU	<u>!</u>						
A v E	3:1.552	1.272	<1	< 1	< 1	< 1	
A v C	: <1	2.225**	<1	1.339	1.084	< 1	
B v (:2.443**	2.236**	<1	3.48***	*1.067	<1	
*=p<.05;	* *:	=p<.025;	***:	=p<.01;	****=	p<.005;	
Interc.=	interc	ept;	Cor	r.= corı	relation	;	

FIGURE 5.4a: POSITIVE REGRESSION PLOTS AT 3/12 FU
FOR M/M, S, & ES SEVERITY GROUPS

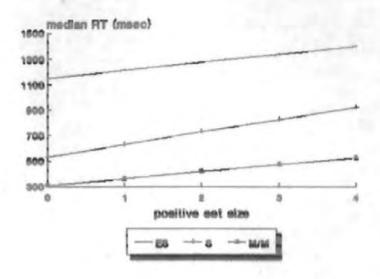


FIGURE 5.4b: POSITIVE REGRESSION PLOTS AT 6/12 FU
FOR M/M, S, & ES SEVERITY GROUPS

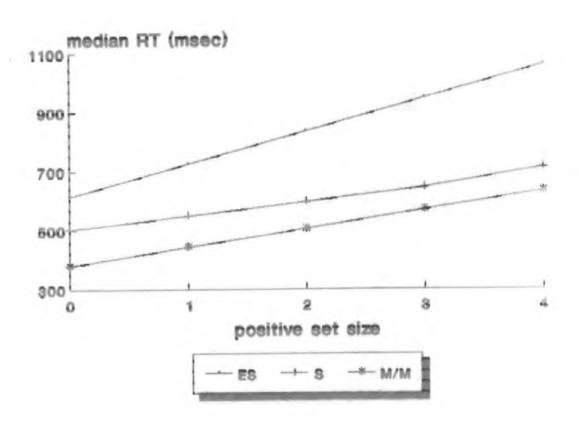


FIGURE 5.4c: POSITIVE REGRESSION PLOTS AT 12/12 FU
FOR M/M. S. & ES SEVERITY GROUPS

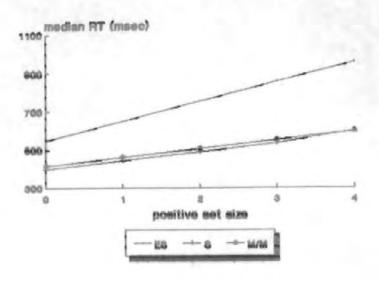


FIGURE 5.4d: POSITIVE REGRESSION PLOTS AT 24/12 FU

FOR M/M, S, & ES SEVERITY GROUPS

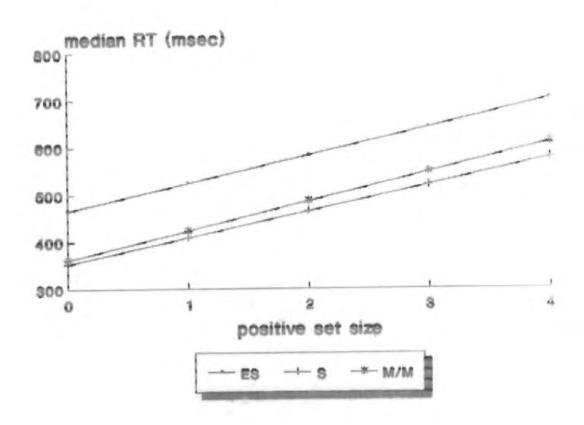


FIGURE 5.4e: POSITIVE REGRESSION PLOTS, SAMPLES

A & B AT 36/12 FU, & SAMPLE C

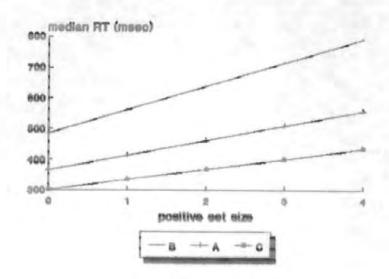
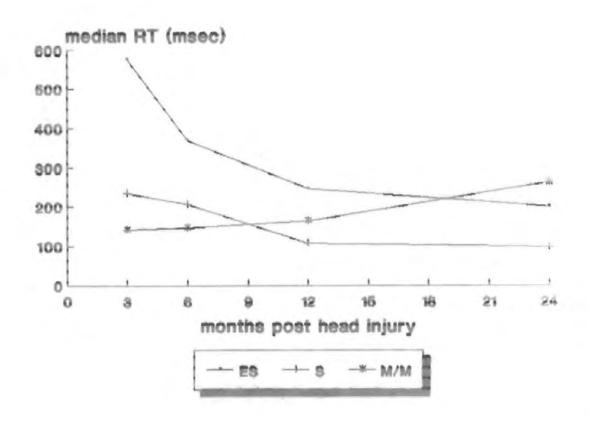


FIGURE 5.5: INTERACTION OF SEVERITY & RECOVERY, 4 ITEMS
M/M, S, & ES GROUPS, POSITIVE TRIALS



The significantly higher weight and intercept values for ES strengthened through the 3-6 month assessments, and were maintained at the 12-month point. However, at 24 months significant differences were only noted against the S and VS group. Examination of the 24- and 36-month patient data against that obtained from C revealed no significant differences between A and C groups for weights, though A showed higher intercepts. Sample B, in contrast, tended to show higher weights. Although differences were observed between the patient samples and the Controls on weights and intercepts, no evidence of poorer linearity was obtained (all correlation coefficients t-test values were less than 1.0).

5.4.4 Memory Scanning Data: Variability of RT.

As attentional factors have often been implicated in the cognitive dysfunction observed after head injury, the variability of subjects' memory scanning RT data was also examined. The most appropriate index of this is standard deviation (SD) of RT. It was hypothesised that size of SD would relate to severity of head injury and time post-trauma. Table 5.3 provides the average SD data for the samples of subjects at each follow-up, and more detail is provided in appendix table C6.3.

As with the median RT data, a 3-way ANOVA with repeated measures was performed on the SD scores. The results of this, shown in Table 5.16, indicate a highly-significant (p<.001) main effect from the severity variable, and a significant main effect from set size (p<.05). The type of set (positive/negative) main effect played no part in determining SD of subjects RTs. The highly-significant (p<.001) repeated measures factor reflects recovery in the variability of RT over time, and also provides a strong (p<.001) interaction with severity.

In addition, Table 5.16 indicates significant 3- and 4-way interactions, which appear to stem from the greater variability of positive RTs in the more severely-injured subjects at the 3-month point, followed by generally-greater SDs for negative RTs except for 4-item trials in M/M subjects at the 24-month point (and all trials in the ES group). The significant recovery over time and interaction with severity are illustrated in figure 5.5, using M/M, S, and ES plots for positive 4-item trials.

TABLE 5.16: ANOVA SUMMARY, SD OF RT

Source	<u>ss</u>	<u>df</u>	<u>MS</u> ,	<u>F-ratio</u>	<u>Sig.</u>
1. A: SEVERITY	1805496	3	601832	17.200	***
2. C: +/- SET	3716	1	3716	<.1.000	n.s.
3. D: SET SIZE	385601	3	128534	3.673	*
4. AC	8039	3	2680	<1.000	n.s.
5:. AD	238758	9	26529	<1.:000	n.s.
6. CD	25273	3	8424	<1.000	n.s.
7. ACD	3227394	9	358599	10.248	***
8. S.W.G	7138124	204	34991		
9. B	764623	3	254874	21.710	* *,*
10.AB	1174036	9	130448	11.111	***
11.BC	45092	3	15031	1.280	n.s.
12.BD	132555	9	14728	1.255	n.s.
13.ABC	102823	9	11425	<1.000	n.s.
14.ABD	413251	27	15306	1.304	n.s.
15.BCD	3230031	9	358892	30.570	***
16.ABCD	755098	27	27967	2.382	**
17.B x S.W.G.	7184980	612	11740		
* = p<.05;	** = p<	.01;	*** =	p<.001;	

No comparisons involving sample A at adjacent follow-up points were significant, except those for the interval 6-12 months. Table 4 in appendix C6 presents the t-test results for sample A and for comparisons of the severity groups at adjacent follow-up points, and the small number of significant t-values are displayed in Table 5.17, below. Table 5.17 indicates that the 6-12 month recovery in sample A arose from improvements in the performance of subjects in the S and ES groups (the significant results in relation to the VS group at 6 and 12 months post head injury actually represented poorer performances by these subjects).

Table 5.18 summarises the t-test analyses conducted on the severity groups at each point following the significant ANOVA finding in relation to severity. The table demonstrates that at each follow-up between 3 and 24 months post-injury a number of significant findings were observed, these findings generally suggesting greater variability in the performance of the S and ES groups compared with the VS group. However, after 3 months comparison of the S and ES groups with the M/M group yielded only non-significant results.

TABLE 5.17: SIGNIFICANT RECOVERY IN SD OF RT

FU Period	Group	<u>n</u>	Set/Size	<u>t-value</u>	Sig.level
1-3/12	M/M	5	+ , 1	2.005	*
3-6/12	ES	6	+ , 1	1.880	*
	ES	6	+ , 2	2.338	* *
6-12/12	Ą	38	+ , 2	1.891	*
	Α	38	+ , 4	1.803	*
	Α	38	- , 2	1.900	*
	S	8	+ , 4	2.071	*
	S	8	- , 1	2.546	**
	٧s	10	- , 3	-2.291	* *
	ES	11	+ , 2	2.788	***
	ES	11	- , 2	2.530	***
12-24/12	S	6	- , 2	3.023	* * *
	VS	5	- , 4	-1.967	*
6-24/12	S	6	+ , 1	2.181	*
	S	6	+ , 2	2.180	*
	S	6	- , 2	2.998	* * *
	٧s	5	+ , 3	2.272	*
	ES	8	- , 4	2.455	**
*=p<.05;	**=p	< .02	!5;	***=p<.01;	

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TABLE 5.18: SD OF RT, t-TESTS AT EACH FU

Positive Set 12 FU: 1 2

<u>1/12 F</u>	<u>U</u> :		1	2	3	4
M/M(8)	v	S(7)	<1	2.295**	2.129*	1.889*
M/M	v	VS(6)	1.480	<1	1.211	<1
S	v	VS	1.442	<1	<1	<1
3/12 F	<u>U</u> :					
M/M(5)	v	S(7)	2.170*	2.100*	1.518	1.748
M/M	v	VS(9)	1.091	<1	<1	<1
M/M	v	ES(6)	1.962*	2.754**	1.698	1.735
S	v	VS	1.395	2.496**	1.864*	1.639
S	V	ES	1.015	<1	<1	1.012
vs	v	ES	1.646	2.679***	1.790*	1.614
<u>6/12 F</u>	<u>U</u> :					
M/M(11) v	S(10)	<1	<1	<1	<1
M/M	v	VS(9)	1.070	1.348	<1	1.466
M/M	v	ES(11)	<1	<1	1.128	1.222
S	٧	VS	1.683	1.621	2.337**	2.363**
S	v	ES	<1	1.111	<1	<1
VS	v	ES	2.419**	2.573***	2.610***	1.811*
*=p<.0	5;		**=p<.025	;	***=p<.01;	

TABLE 5.18: SD OF RT, t-TESTS AT EACH FU (cont)

Positive Set

						
12/12	<u>FU</u>	:	1 .	2	3	4
M/M(10) v	·S((8)	1.,278	1.343	1.208	1.016
M/M	v	VS(10)	1.238	1.410	<1	1.245
M/M	Ÿ	ES (11)	<1	<1	<1	<1
S	v	VS	<1	<1	<1	<1
S	v	ES ,	2.099*	1.422	2.002*	2.180*
VS	v	ES	1.993*	1.470	<1	2.371**
24/12	FU	:				
M/M(7)	v	S(5)	<1	1.013	<1	<1
M/M	v	VS(8)	<1	1.131	1.740	1.422
M/M	v	ES(7)	<1	<1	<1	<1
S	v	VS	<1	<1	2.570**	1.798*
S	v	ES	<1	2.194*	1.095	<1
VS	v	ES	<1	2.891***	1.749	2.334**
A(26)	v	B(10)	<1	2.008*	1.019	1.265
A.(26)	v	C(10)	1.693*	2.059**	1.210	1.341
B(10)	Ÿ	C(10)	1.919*	2.720***	<1	2.077*
36/12	FU:	:				
A(10)	v	B(10)	1.065	<1	1.359	1.871*
A (10)	V	C(10)	2.139**	2.088*	<1	1.570
B(10)	v	C(10)	2.054*	2.198**	1.451	2.330**
*=p<.0	5;		**=p<.025;	***:	=p< .01;	

TABLE 5.18: SD OF RT, t-TESTS AT EACH FU (cont)

Negative Set

1/12 FU	:	1	2	3	4
M/M(8) \	/ S:(-7)	1.525	2.331**	1.101	1.948*
M/M	7 VS((6))	1.575	<1	<1	<1
s v	, VS	1.154	<1	<1	<1
3/12 FU	:				
M/M(5) \	s(7)	1.634	2.269**	1.142	1.094
M/M	/ VS(9)	<1	1.428	1.615	<.1
M/M v	ES(6)	1.277	1.899*	<1	1.864*
s v	, VS	2.157**	3.206****	2821***	<1
s v	, ES	<1	<1	<1	1.028
vs v	, ES	1.382	2.067*	1.642	1.653
6/12 FU	:				
M/M(11)	s(10)	1.672	<1	<1	<1
M/M \	/ VS(9)	<1	1.752	1.255	<1
M/M v	/ ES(11)	1.195	<1	<1	2.183**
s v	, VS	1.683	3.503****	2.573***	1.540
s v	, ES	<1	1.858*	<1	1.672
vs 、	, ES	1.290	3.939****	3.232****	2.835***
*=p<.05;	;	**=p<.025;	* * *	=p<.01;	

TABLE 5.18: SD OF RT, t-TESTS AT EACH FU (cont)

Negative Set

12/12 F	<u>'U</u>	:	1 :	2	. 3	4
M/M(10)	v	S(8)	1.401	< 1	1.426	<1
M/M	v	VS(10)	1.639	1.321	1.016	1.129
M/M	v	ES(11)	<1	<1	<1	<1
s	V	VS	<1	1.889*	1.026	< 1
s	v	ES	2.973****	<1	2.861**:	1.678
vs ·	v	ES	3.230****	1.950*	1.813*	2.431**
24/12 F	<u>'U</u> :	:				
M/M(7)	V	S(5)	<1	<1	<1	< 1
M/M	v	VS(8)	< 1	1.400	1.285	< 1
M/M	v	ES(7)	<1	<1	<1	< 1
S ,	v	VS	<1	1.027	1.531	< 1
s ·	v	ES	<1	<1	1.204	< 1
vs ·	v	ES	<1	2.259**	2.665***	<1
A(26)	v	B(10)	1.448	1.558	<1	1.733*
A(26) v	v	C(10)	<1	2.312**	1.983*	1.723*
B(10) v	v	C(10)	1.665	2.216**	2.305**	2.305**
36/12 FU	<u>U</u> :					
A(10) \	v	B(10)	<1	1.028	1.192	1.032
A(10)	v	C(10)	<1	1.937*	2.386**	< 1
B(10) \	v	C(10)	1.225	1.625	1.873*	1.744*
*=p<.05;	;	**=	•p<.025;	***=p<.01	L; ****	=p<.005

Comparisons of samples A and B at 24 months and 36 months produced only occasional significant findings to suggest less variable performance in the former. However, table 5.18 also indicates that the control subjects' SDs were much less variable than those observed in both patient samples at the 24- and 36-month follow-ups.

The SD data was also examined in terms of correlational relationships with unconsciousness (U/C), PTA, and median RT. Table 5.19 presents these values, showing no strong associations between sample A's SD and U/C or PTA at 1 month after head injury although correlation coefficients at 3 months with these variables were all significant. A majority of the values at 6 and 12 months showed significant associations between SD and the 2 indices of head injury severity. The association had weakened by 24 months post-trauma, although the reduced sample A available at the 36-month point showed

TABLE 5.19: CORRELATIONS OF RT SD WITH U/C, PTA, & RT

Po:	sitive Set	<u> </u>	Nega	ative S	Set	<u> </u>
		_				4
Sample A 1	2 3	4	1	2	3	4
<u>1m</u> U/C:10	0606	08	. 0,1	07	.01 -	01
PTA: .22	. 27 . 25	.23	. 36	. 2,6	. 39	.29
RT: .98*	* .88** .81**	.77**	.95**	.80**	.74**	.81**
<u>3m</u> U/C: .70*	* .70** .78**	68**	.78**	.82**	.63**	.63**
PTA: .49*	* .39*64**	.64**	.53**	.60*	.47*	.48*
RT: .94*	* .80** .81**	70**	.87**	.68**	.77**	.69**
<u>6m</u> U/C: .35*	.42** .20	. 27	.46**	.48**	.44**	.48**
PTA: .36*	.33* .14	. 20	. 44**	.44**	.40*	.44**
RT: .47*	* .50** .84**	.72**	.79**	.71**	.81**	.79**
<u>12m</u> U/C: .19	.30 .49**	.52**	.41**	.36*	.49**	.49**
PTA: .09	.25 .31*	.31*	. 35*	.37*	. 33*	.33*
RT: .63*	* .98** .73**	* .83**	.53**	.95**	. 28	.88**
24m U/C:11	01 .08	06	. 19	.14	. 17	.10
PTA:06	.01 .18	06 -	16	. 13	.17 -	01
RT: .18	.62** .87**	. 35	.82**	.86**	.72**	.78**
<u>36m</u> U/C: .38	.75* .60	.62	.67*	.80**	.69*	.62
PTA: .33	.71* .58	. 59	.65*	.79**	.68*	.60
RT: .56	.90** .95**	.78**	.95**	.88**	.90**	.87**
*=p<.05;	**=p<.01					

significant correlations for half of the coefficients computed. No coefficient for sample B's SD and the severity indices achieved significance at either 24 or 36 month follow-up (table 5.19b, below). All of the correlation coefficients calculated for sample A between SD and median RT at 1-12 months were significant, although a few non-significant values were noted at 24 and 36 months post-injury. Calculation of these coefficients for sample B generally yielded significant values, and for sample C most coefficients were sizeable, with approximately half being significant (see tables 5.19b-c, below).

TABLE 5.19b: CORRELATIONS OF RT SD WITH

U/C, PTA, & RT, SAMPLE B

	Positive Set				Neg	Set		
	<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>	<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
<u>24m</u>								
U/C:	.00	.11	. 08	16	. 16	18	03	. 0
PTA:	. 33	. 25	, 43	-,03	. 40	.06	.19	. 20
RT:	.99**	. 44	.97**	.77**	. 94**	.86*	* .92**	.87**
<u>36m</u>								
U/C:.	12 -	13	. 27	. 08	. 04	07	02	.07
PTA:	. 24	.17	. 54	. 50	. 38	. 27	. 29	. 27
RT:	.90**	.91**	.90**	. 59	94**	.92*	* .95**	.92**
*=p<.(05;	**=p<	.01;					

TABLE 5.19c: CORRELATIONS OF RT SD WITH RT, SAMPLE C

	Positive Set					Negative Set			
	<u>1</u>	<u>2</u> .	<u>3</u>	<u>4</u>	<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>	
RT:	.84**	. 24	.96**	. 46	.78**	.53	.99**	.91**	
*=p<	(.05;	**	=p<.01;						

Recovery in SD was also examined via investigation of frequency of improvement in SD between follow-ups. Unlike the findings for sample A median RT scores between follow-ups, Binomial test Z values provided in appendix table C6.6 offer little evidence of improvement in RT variability (SD) over time for sample A as a whole: for each follow-up interval only 1 significant result was noted (out of 8 information conditions), with the exception of the 6-12 month interval where 2 significant values were obtained. As table 5.17 indicates, recovery over time in SD was particularly associated with S and ES subjects.

5.4.5 Associations Between RT Data & Other Variables

a. Clinical & Demographic Variables.

Severity of head injury is, of course, the most important clinical variable, and this has been considered in previous sections. Other clinical factors of interest include the occurrence, of a neurosurgical operation, evidence of lateralisation of brain injury, the prescription of anticonvulsant medication, and the time taken to return to work/school. Relevant demographic and background variables include age, sex, and premorbid intellectual level. Raw data on these variables is included in appendices C4 and C5.

Neurosurgery following head injury was undergone by 7 sample A subjects, 2 received general anaesthetics as part of general surgery, and 33 subjects did not require any surgical intervention (appendix table C4.1). The t-test comparisons of the neurosurgery subgroup with those subjects who received neither neurosurgery nor general anaesthetic (table 6, appendix C6) provided no consistent evidence that the recovery of the latter was better in RT terms; the occasional significant results which were observed would be expected by chance. However, as figure 5.6a-d reflects, there was a tendency for the neurosurgery subgroup to show a faster recovery in the first 6 months

FIGURE 5.6: 'NEUROSURGERY' (N) & 'NO OPERATION' (NO)

SUBGROUPS, SAMPLE A, POSITIVE TRIALS

a. 3/12 Follow-up

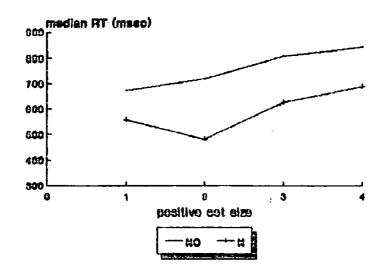


FIGURE 5.6: 'NEUROSURGERY'(N) & 'NO OPERATION'(NO)

SUBGROUPS, SAMPLE A, POSITIVE TRIALS

b. 6/12 Follow-up

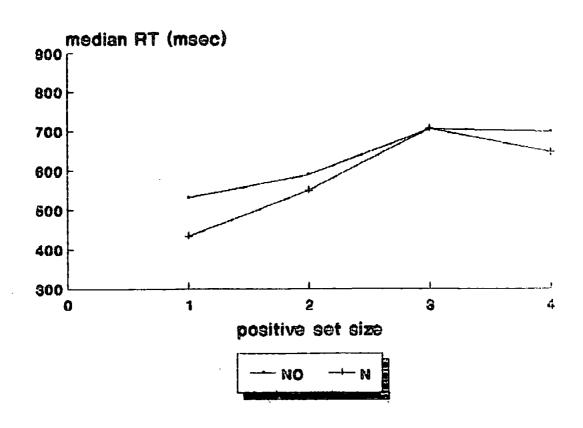


FIGURE 5.6: 'NEUROSURGERY'(N) & 'NO OPERATION'(NO)

SUBGROUPS, SAMPLE A, POSITIVE TRIALS

c. 12/12 Follow-up

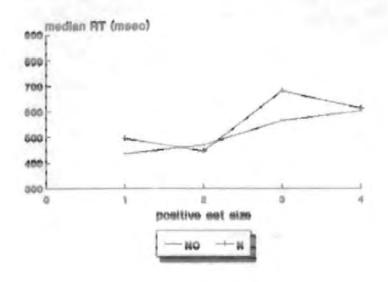
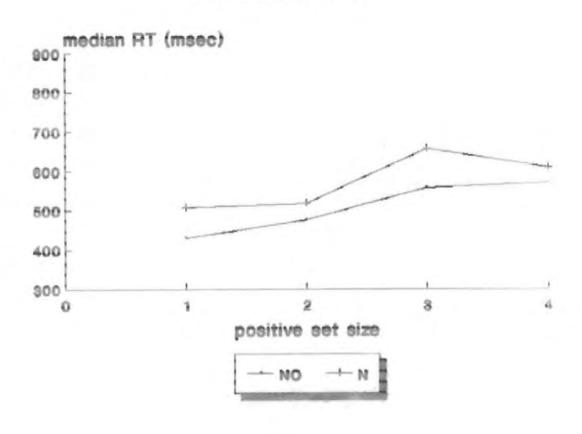


FIGURE 5.6: 'NEUROSURGERY'(N) & 'NO OPERATION'(NO)

SUBGROUPS, SAMPLE A, POSITIVE TRIALS

d. 24/12 Follow-up



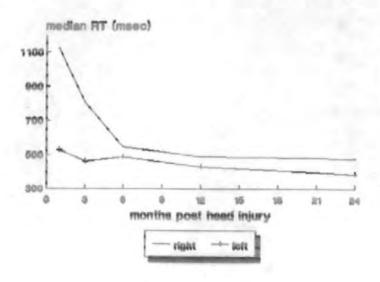
post-trauma, and for the 'no operation' subgroup to be performing marginally better at the 2-year follow--up.

Although closed head injuries produce diffuse damage, there is sometimes evidence of partial lateralisation of damage. study CT scan data and neurological In the present examination suggested partial lateralisation to the right hemisphere in 15 sample Α subjects and to the left hemisphere in 9 subjects. Comparison of these subgroups of median RT and SD, via t-test analyses, in terms generated no significant values: at the 1, 6, or 12 month follow-ups (though see figure 5.7, below). However, majority of the comparisons at the 3-month point and 50% of performed at 24 months post-injury yielded significant results (table 7, appendix C6). The findings favoured those for whom there was no evidence lateralisation to the right hemisphere. Figure 5.7a-breflects the tendency for those subjects with evidence of right hemisphere lateralisation to show a poorer recovery in RT. A similar picture was noted in relation to SD.

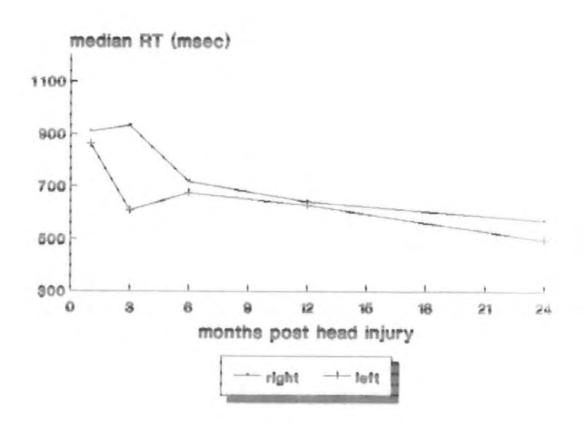
Of the 42 subjects in sample A, 2 were not in employment just prior to their head injury, 9 did not return to work during the period of the study (6 of these were in the ES group), and there was uncertainty with regard to 4, leaving 27 subjects for whom occupational/educational 'recovery'

FIGURE 5.7: RECOVERY IN MEDIAN RT AT EACH FOLLOW-UP

a. 1-item, Positive Trials



b. 4 items, Positive Trials



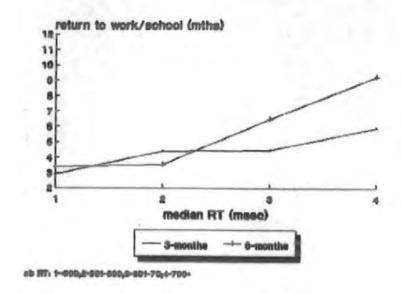
could be studied. In those subjects who achieved it, the time to return to work/school was 5.9 months (sd= Correlations of median RT and SD with time to return 5.0). to work/school were generally negligible 1 month after injury, and no values reached statistical significance at 3 months (although 50% of the coefficients exceeded +0.4). At the 6-month follow-up half of the 16 correlations were significant, 6 of these being noted in relation to median By 12 months all but one of the correlations of time to return to work/school with RT and SD were significant, there being some suggestion that the associations were stronger with increasing positive set size. At 24 months head injury most of the correlations remained statistical significant.

All of the above correlational findings are summarised in table 8 in appendix C6, and figure 5.8a-b depicts the relationship between time to return to school/work and median RT at follow-ups 3-24 months, using 4-item positive trials as the example. The clearest relationship between severity of head injury and time to return to work/school, however, was reflected in the significant correlations (both at the .05 level) with U/C (+0.41) and PTA (+0.39). This latter finding was observed even though 6 ES subjects did not return to work/school during the period of the study.

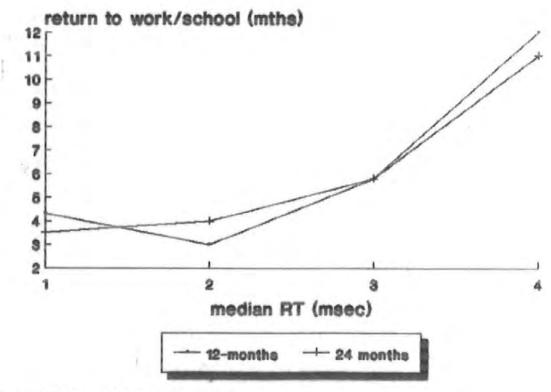
FIGURE 5.8: TIME TO RETURN TO WORK/SCHOOL & MEDIAN RT

FOR 4-ITEM POSITIVE TRIALS

a. 3- & 6-month FUs



b. 12- & 24-month FUs



nb RT: 1-4500;2-501-600;3-601-70;4-700+

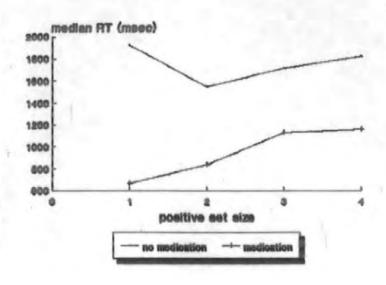
Eight subjects in sample A experienced fits in hospital, but only 3 suffered fits post-discharge (2 of whom had a single fit). With such small numbers it was impossible to examine the effects of fits upon cognitive performance.

effects of anticonvulsant medication upon RT indices were also difficult to investigate, partly due to the issue sample size and partly because patients' medication. was withdrawn by their doctors at various times postinjury. However, an attempt was made to address this aspect by 2 methods. First, the numbers of subjects who were/were not taking anticonvulsant medication prophylactically were ascertained. From these numbers it was possible to identify 2 subgroups of ES subjects who were (n=3), and were not (n=3), taking the medication at the 3-month follow-up, at the 6-month follow-up (n=6,5, respectively) and after 12 months (n=3,5). Similarly, subgroups of S subjects could be identified at 3 months (n=3,4), and 6 months (n=3,7). The within-group t-tests on median RT and SD are provided in appendix table C6.9. In spite of the very small sample numbers, table C6.9 shows that ES subjects taking anticonvulsant medication at 3 months performed significantly better than those not taking medication on half of the t-tests carried out. By 6 months the number of significant comparisons had reduced to 5 (out of 16), and at 12 months no significant t-values were

observed. The picture at 3 and 6 months post-injury is depicted in figure 5.9, below. The significant findings for the ES group were not based upon differing lengths of PTA in the 'medication' and 'no medication' subjects, although there was a non-significant tendency (t=1.697; df=4;ns) for the medication subjects to have experienced a shorter period of initial U/C. For the S group no significant results were noted in relation to anticonvulsant medication.

The second investigation of the effects of anticonvulsants upon RT involved examining results from 3 patients fortuitously assessed just prior to withdrawal of medication and then approximately 1 month later. The subjects studied were numbers 6 (withdrawal at about 6 months after head injury), 14 (10 months), and 33 (9 months). Their raw data, in appendix C1, table 4, provides no consistent evidence that removal of anticonvulsant medication produced specific changes in RT indices.

FIGURE 5.9: MEDIAN RT & ANTICONVÜLSANTS, ES GROUP
a. 3/12 FU



b. 6/12 FU

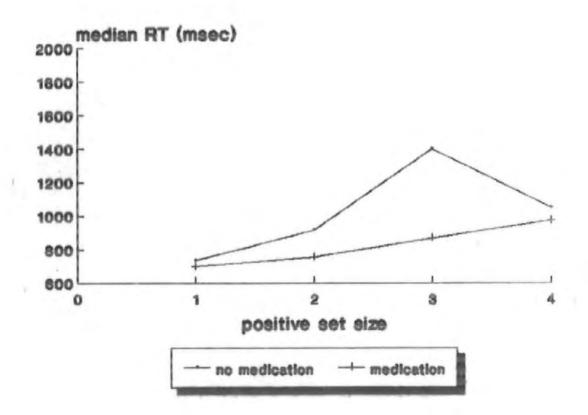


TABLE 5.2	20:	CORRELATIONS	OF	RT	VARIABLES	WITH	AGE
							

	positive					negative				
sample	<u>A</u>	1 .	2	3	4	1	2	3	4	
1/12	RT:	. 26 .	43* .	46*	.49*	. 37	. 47*	49*	. 48*	
(n=23)	SD:	, 33 .	43*	32	. 23	.31	. 34	. 34	. 30	
3/12	RT:	.10	52** .	53**	.53**	. 28	.53** .	54**	.53**	
(27)	SD:	.12 .	13 .	38	.42*	. 10	. 28	.52**	.53**	
6/12	RT:	. 20 .	21	06	. 20	. 25	.32* -	.06	. 23	
(41)	SD:	.03 .	17 .	.03	.13	. 20.	. 25	. 21	.32*	
12/12	RT:	.22 .	43** .	22	. 34*	.40*	.44**	20	.32*	
(39)	SD:-	01 .	43** .	41**	. 34*	.13	. 35*	.12	. 32*	
24/12	RT:	.19 .	14	09	.07	. 20	. 17	.04 -	,06	
(10)	SD:	.05	02	10 -	.09	.08	. 18	.03 –	. 08	
36/12	RT:-	10	18	12	.00 -	04 -	. 20 –	.05 –	. 10	
(10)	SD:-	01	28 -	16	.16 -	05	.02 –	. 17	. 15	
<u>sample B</u>										
<u>24/1</u> 2	RT:	.84** .	70* .	84**	.79**	. 78**	.66*	.84**	. 58	
(10)	SD:	.77* .	15 .	79**	. 34	.78**	. 46	.66*	. 29	
36/12	RT:	.62 .	71* .	60	. 45	.68*	. 68*	73*	. 42	
(10)	SD:	.79** .	68* .	73*	.61	.79**	. 75*	.81**	.66*	
sample C										
(10)	RT:	.09	15	37 -	.12 -	64* -	.50 -	.37 –	. 32	
	SD:	.03	01	36	.01 -	44 -	.16 -	.37 –	. 19	
*=p<.05;		**=p<.0)1;							

The relationships between age and the RT measures of median and SD were investigated via the correlations summarised in table 5.20. These show some interesting features. example, sample A showed good correlations between median at 1-and 3-months post-injury, and slightly weaker values when SD was examined in relation to age. However, at 6 months only 1 each of the correlations involving SD and median RT with age attained significance, although by the 12-month point the strong associations between the RT indices and age were again apparent. For sample A the significant associations of these variables age dissipated after 12 months and the coefficients became negligible. In contrast, the much smaller sample B showed strong correlations between median RT/SD and age at both 24- and 36-month follow-ups. For sample C only 1 of the 16 coefficients calculated reached statistical significance, which might be expected by chance, thereby providing no evidence of a significant association between the RT indices and age.

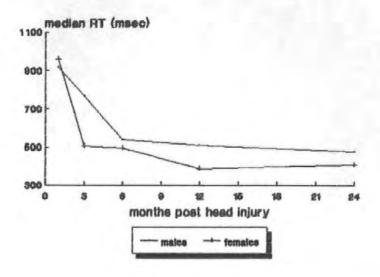
The sex ratio of sample A was 18 females and 24 males. Examination of possible sex differences in terms of median RT and SD was undertaken via t-test analyses at each follow-up, up to 24 months post head injury. The results are summarised in appendix C6 (table 10). They show that no significant differences were observed at the 1-month

point, 2 significant values were noted at 3 months, 3 at the 6-month point, and 2 at the 24-month follow-up. All of these 7 significant values involved negative set trials (5 for SD, 2 for median RT). The frequency of observing significant t-test results might just be regarded as approximating chance level, although it should be noted that the female group provided the better (ie, faster or less variable RTs) in all 7 cases. In addition, at the 12month follow-up 11 significant t-test comparisons were obtained (of 16 undertaken), with all of the significant results indicating better performance by the female group. The general tendency for female subjects to show faster RT recovery is reflected in the graphs provided in figure 5.10.

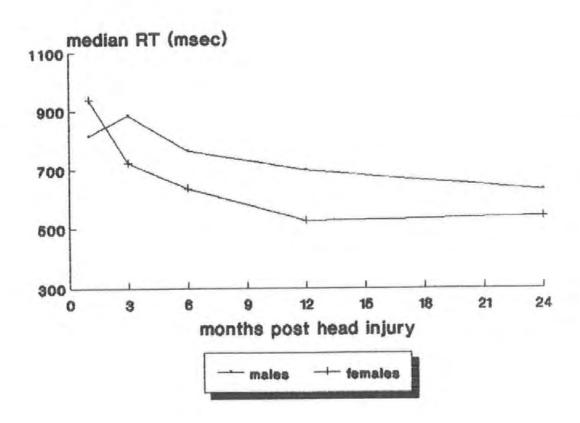
Further t-test analyses of the 2 gender groups involving comparisons of age (t=1.387; ns), length of unconsciousness (t=0.980; ns), and PTA (t=0.384;ns), offered little evidence that differences in initial severity of head injury, or of age, could account for the significant findings. However, the finding that the female subjects tended to take a shorter time to return to work/school (t=1.953; df=33; p<.10) suggests that the finding of female superiority in RT recovery might be genuine.

FIGURE 5.10: RECOVERY IN MEDIAN RT, MALES & FEMALES

a. 1-Item, positive Trials



b. 4-Item, positive Trials



The RT data were also considered in terms of estimated premorbid intellectual level. The National Adult Reading Test (NART: Nelson, 1982) was only introduced into routine in the author's department after the start of the current study, and data using it was only available on 27 in sample A (appendix table C8.4). remaining subjects a 'best estimate' was made from the available WAIS data (Wechsler, 1955), based upon age scale scores for 'hold' subtests. To ensure that these methods of estimating premorbid intellectual level did not yield significantly different values, t-tests were performed on verbal IQ (VIQ) and performance IQ (PIQ) using the two methods. The results for both VIQ (t=0.313; df=40; ns) and PIQ (t=0.123; df=40; ns) indicated that the data derived via the two methods were compatible.

Subsequently, estimated VIQ and PIQ were correlated with median RT and SD at each follow-up (these are depicted in table 11, appendix C6). Check correlations at the 6-month follow-up (largest sample point) confirmed no significant association between VIQ and U/C (r=-.17), and VIQ and PTA (r=-.10). The corresponding coefficients for PIQ with these variables were -.25 and -.12, respectively. Table C6.11 shows that coefficients calculated when correlating the IQ variables with median RT and SD at 1 and 3 months were nonsignificant though at the 6-month point both IQs

yielded significant results with SD and median RT in about 25% of the information conditions. At the subsequent 12-month follow-up only isolated significant correlations were observed, though at the 24-month follow-up approximately one-third of coefficients were statistically significant.

b. Other measures of Memory

Data was collected on the Rey AVLT (Lezak, 1983) and digit span (Wechsler, 1955) at each follow-up, and in addition subjects completed a Wechsler Memory Scale (WMS; Wechsler, 1945) at the 1-, 6-, and 24-month points. Subjects also provided responses on a subjective memory questionnaire (SMQ; Bennett-Levy & Powell, 1980). Individual raw scores on memory tests are presented in appendix C8, and group scores in appendix C9.

Table 5.21a provides Mean and SD scores for sample A at each follow-up point, on some Rey AVLT variables (A1, Total A, B, and Delayed A). Investigation of the Rey in terms of its sensitivity to severity of head injury was undertaken at each follow-up using t-tests. Rey data for samples A and B are shown in appendix tables C9.1a-b and C9.2a-b. Table 3 in appendix C9 provides no significant differences between severity groups at 1-month post-injury, though at 3 months the ES group was often performing significantly

TABLE 5.21a: SAMPLE A MEAN & SD SCORES FOR A1, TOTAL A, B, & DELAYED A TRIALS OF REY AVLT

FOLLOW-UP

Variable		1/12	3/12	6/12	12/12	24/12	36/12
A 1	Mean:	6.0	6.6	5.9	7.3	6.5	7.3
	SD:	2.1	1.6	1.6	1.9	1.9	2.7
TotA	Mean:	45.0	48.9	47.4	52.4	51.3	56.2
	SD:	11.4	12.0	11.1	10.3	10.1	10.0
В	Mean:	5.0	6.2	6.1	6.3	6.5	4.1
	SD:	1.6	2.8	2.2	2.7	2.4	2.0
DelA	Mean:	8.5	9.1	9.3	9.7	10.0	10.8
	SD:	3.5	4.2	4.3	3.8	3.8	4.1

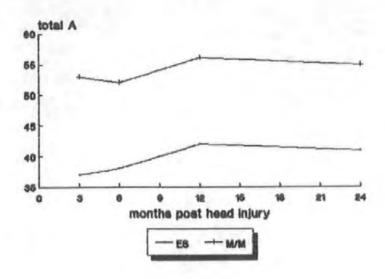
poorer than the M/M and VS subjects. This pattern continued at the 6-, 12- and 24-month follow-ups, the ES group generally showing poorer learning than the M/M, S, and VS groups. Table 5.21 provides example t-values for the comparison of the ES and M/M groups. Some Illustrations of poorer ES memory performance are provided in figure 5.11, where these subjects show lower learning scores and higher interference effects upon their total learning over list A trials. The finding of more impaired results in the ES subjects paralleled that noted in relation to median RT and SD, though the Rey results provide no evidence that the S group performed at a lower

level than the M/M and VS subjects (as was the case in relation to RT indices). Correlational analysis of Rey U/C and PTA at each follow-up (table scores with appendix C9) showed significant coefficients for recall developing at 3 months, becoming highlymeasures significant by 6 months and then almost disappearing at the 12-month point before returning to significance at 24 and 36 months post-trauma. Sample B, in contrast, showed no significant correlations between Rey scores and severity indices at 24 months, though a number were noted at the 36month follow-up (table 5, appendix C9).

Examination of the relationships between Rey variables and those of median RT and SD were also undertaken using correlations. Table 6, appendix C9 provides the large matrix, and table 5.22 presents an illustrative abstract of coefficients for some Rey variables. At 1 month after injury most coefficients were significant, and a number of features were apparent. First, the number and level of significance of correlations tended to be higher in relation to median RT, compared with SD.

FIGURE 5.11: REY PERFORMANCE AT EACH FU, M/M & ES GROUPS

a. Total A Learning Score



b. % Retroactive Interference

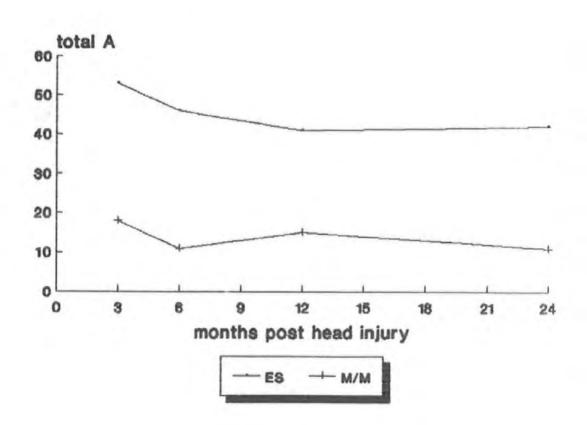


TABLE 5.21: t-TESTS, REY DATA, M/M v ES

Recall Scores on List A trials

		<u>A1</u>	<u>A2</u>	<u>A3</u> :	<u>A4</u>
3/12 FU	(n=5,4)	< 1	1.346	2348*	2.221*
6/12 FU	(11,10)	2.305**	2.300**	2.653***	3.919****
12/12FU	(10,9)	2.087*	2.894***	3.879****	3.645****
24/12FU	(7,7)	<1	2.568**	4.368***	3.945****
		Recall Sco	ores on List	s A & B	
		<u>A5</u>	Total A	<u>B</u>	A Delay
3/12 FU		2.612**	1.986*	1.365	2.409**
6/12 FU		4.104****	3.996****	2.327**	4.300****
12/12FU		3.288****	3.782****	1.168	3.691****
24/12FU		3.310****	3.469****	2.679**	3.875****
*=p<.05;	* * =	=p<.025;	***=p<.(01; **	**=p<.005;

Second, there was a trend towards the level of significance being higher with larger set sizes. The strongest correlations were seen with RT indices from the Rey recognition score, percentage retroactive interference, list 'A' score after interference and the summed score of 'A' across all 5 learning trials. Proactive interference and false positive scores showed no significant associations at all with RT measures at 1 month.

Correlations at 3 months again showed the tendency for more frequent/greater significance to be associated with median and larger set sizes. However, the frequency of significant results was much higher than at the 1-month point, and proactive interference and false positive scores showed significant coefficients with nearly all RT set size The frequency of significant findings was less months (still favouring median RT over SD), and this trend continued at 12 months. Ву 24 months, however, the significant results of rose again and good coefficients were generally maintained at 36 months postinjury (even with a small sample size, one-third were significant). (table 7, appendix C9), For sample B although none of the correlations reached significance at 24 months, approximately 50% did so at the 36-month follow-up.

Recovery in Rey recall variables over time was investigated using t-tests in sample A, and in the ES group (table 13, appendix C6). Sample A showed a significant improvement in Rey scores (3 variables) only between the 6 and 12 month points, and no t-test comparisons involving ES subjects between adjacent follow-ups reached significance.

TABLE 5.22: CORRELATIONS OF MEDIAN RT WITH SOME REY

VARIABLES AT EACH FOLLOW-UP, SAMPLE A

			<u>TotalA</u>	%Pro	%Retro	False+
1/12FU,	set	+1	40	18	.48*	04
(n=23)		+4	62**	22 ⁻	.67**	01
3/12FU,	set	+1	40*	.58**	.47*	. 30
(n=41)		+4	. 37	.71**	. 54**	.95**
6/12FU.	set	+1	71**	. 35*	. 29	.62**
(·n=41)		+4	61**	. 33*	. 23	.62**
12/12FU,	set	+1	44**	. 20	. 30	.07
(n=39)		+4	33*	. 28	.39*	. 19
24/12FU,	set	+1	51**	. 32	. 25	.53**
(n=26)		+4	39*	. 26	.01	. 29
36/12FU.	set	+1	62	35	03	14
(n=10)		+4	75**	33	. 20	.08
*=p<.05;		**=p<	<.01;			

Information on total digit span at each follow-up, in terms of Mean and SD, is provided in table 5.23a below. Table 2 in appendix C8 shows the raw data for digits at each follow-up, in terms of digits forward (DF), digits backward (DB), and digits total (DT), and table 9 in appendix C9 provides the t-test data comparing severity groups. The digits results were similar to the other memory test findings. Only 1 t-value was significant at 1 month, and 2 at 3 months. One of the latter involved the ES group.

TABLE 5.23a: SAMPLE A TOTAL DIGIT SPAN AT EACH FOLLOW-UP
FOLLOW-UP

<u>Variable</u>	1/12	<u>3/12</u>	<u>6/12</u>	12/12	24/12	36/12
DSpan Mean:	10.8	12.2	11.9	12.4	12.2	12.0
SD:	2.7	2.6	2.3	2.2	2.2	1.7

TABLE 5.23: t-TESTS, DIGIT SPAN, ES GROUP

		<u>F</u>	<u>'orward</u>	Back	<u>Total</u>
3/12 I	<u>:U</u> :				
M/M(5)	v	ES(4)	<1	1.327	1.128
S(7)	v	ES	<1	1.723	1.124
VS(9)	v	ES	<1	2.710**	1.683
6/12 I	<u>:U</u> :				
M/M(11)	v	ES(10)	< 1	2,538**	1.189
S(10)	v	ES	<1	1.735*	1.132
VS(9)	v	ES	<1	3.415***	2.487**
12/12 I	<u>. U</u> :				
M/M(10)	V	ES(9)	<1	< 1	<1
S(8)	v	ES	<1	1.587	1.691
VS (9)	v	ES	<1	1.990*	1.997*
24/12 I	<u>. U</u> :				
M/M(7)	v	ES(7)	<1	1.894*	1.459
S(10)	v	ES	2.607***	3.818***	4.007***
VS(8)	v	ES	<1	2.642**	1.990*
*=p<.05	5;	**=p	×:	**=p<.01;	

which at 6 months scored significantly lower on DB compared with each of the other severity groups. At 12 months only ES's comparison with VS subjects yielded significant findings, but by 24 months ES subjects again scored lower than subjects in the other severity groups. The t-values for comparisons involving the ES subjects are shown in table 5.23, above, and the plots of DB for ES and M/M subjects are depicted in figure 5.12.

Digit variables generally showed low correlations with U/C and PTA: none with PTA reached significance until the 24-month point (see table 5.24), and only 2 with U/C were significant before that follow-up. Sample B showed about 50% of significant correlations with U/C and PTA at both 24- and 36-month follow-ups.

Given the large number of subtests comprising the scale, and the fact that a stable factorial structure has been elicited (Skilbeck & Woods, 1980), examination of the Wechsler Memory Scale (WMS) concentrated upon the 3 main factors (learning, attention/concentration, and information/orientation). Table 3 in appendix C8 provides the sten scores for subjects using these WMS factors. Analysis of sten scores by severity group (very small samples) was carried out at the 6- and 24-month follow-ups (appendix C9, table 10). All of the

TABLE 5.24: CORRELATIONS OF DIGIT SPAN WITH U/C & PTA

AT EACH FOLLOW-UP, SAMPLES A & B

Sample A		Forward	Back	<u>Total</u>
1/12 FU:	U/C:	. 30	.22	. 28
(n=23)	PTA:	. 31	. 30	. 33
3/12 FU:	U/C:	34	18	. 25
(27)	PTA:	15	.04	. 32
6/12 FU:	U/C:	13	39**	31*
(41)	PTA:	06	27	22
12/12 FU:	U/C:	25	30	17
(39)	PTA:	09	16	03
24/12 FU:	U/C:	23	57**	47*
(26)	PTA:	15	41 *	33
36/12 FU:	U/C:	.01	67*	46
(10)	PTA:	07	73*	52
Sample B				
24/12 FU:	U/C:	67*	60	28
(10)	PTA:	86**	65*	39
36/12 FU:	U/C:	67*	67*	46
(10)	PTA:	70*	57	69*
*=p<.05;	* *	=p<.01;		

significant findings at 6 months involved ES subjects (see table 5.25, below). By the 24-month point the ES group was still performing significantly more poorly than the S and VS groups (factor 2 in both cases:

p<.005). Figure 5.13 graphs the factor sten scores for ES and M/M subjects at 6 and 24 months after head injury.

TABLE 5.25: t-TESTS, WECHSLER MEMORY SCALE, ES GROUP 6/12 FU: Factor 1 Factor 2 Factor 3 M/M(6) v ES(10) 3.464*** 1.939* 1.1553.314**** 1.570 2.487** S(10) v ES 2.909*** 3.895**** 3.596**** VS(9) v ES 24/12 FU: $M/M(6) \vee ES(5)$ 1.701 1.087 <1 S(3) v ES 1.206 5.353**** <1 VS(3) v ES 1.026 5.353**** < 1

TABLE 5.26: CORRELATIONS OF WMS FACTOR STEN SCORES
WITH U/C, & PTA AT 6/12 & 24/12 FU

6/12	Factor 1	Factor 2	Factor 3
(n=35) U/C:	66**	40*	53**
PTA:	60**	36*	36 *
24/12			
(n=19) U/C:	31	46*	08
PTA:	37	-,23	07
*=p<.05;	**=p<.01;		

FIGURE 5.12: DIGITS BACKWARDS FOR M/M & ES GROUPS 3-24 MONTH FOLLOW-UPS

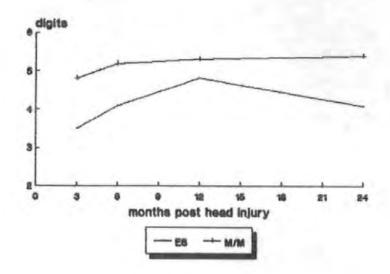
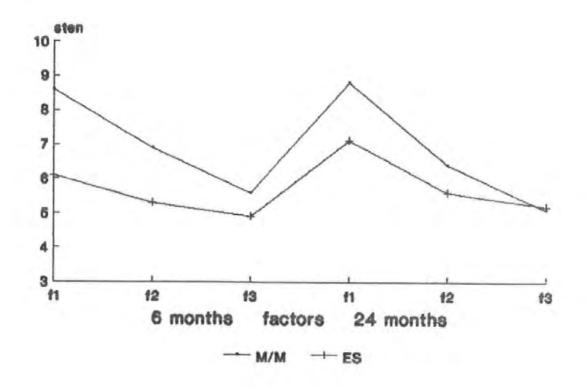


FIGURE 5.13: WMS FACTOR SCORES FOR ES & M/M GROUPS

AT 6/12 & 24/12 FU



correlational relationships between factor scores and severity indices are presented in table 5.26. At 6 months post-injury all factors correlated significantly with both U/C and PTA, with factor 1 showing strongest relationship. However, by 24 months the only significant finding related to factor 2 and U/C, although factor 1's correlations were still noteworthy. In terms of the RT measures at 6 months, factor 1 showed significant correlations with almost all of the SDs and RTs (see table 5.27). Factor 2 presented median similar picture, though in contrast factor 3 showed many fewer significant values with median RTs. By 24 months statistically-significant associations virtually all with RTs had disappeared (quite a number still exceeded -.3), though all 3 factors related significantly to some No evidence was noted of recovery between 6-24 months post-injury for sample A (all t-values less than 1.0), or for the ES group (t-values less than 1.0 for factors 1 and 2, and t=1.197 for factor 3).

Examination of relationships between Sternberg RT data the WMS can be achieved using factor scores, as described above. However, neuropsychologists often in their clinical employ only part of the WMS research work. The most frequently used WMS subtest is Logical Memory (LM). To facilitate comparison with other research findings table 5.27a provides the coefficients obtained when correlating LM with Sternberg RT and SD variables at 6 and 24 months post-trauma. The data in table 5.27a shows a majority of significant coefficients at the 6-month point, similar to the WMS factor results (table 5.27). No significant LM-RT correlations were noted at the 24 month follow-up, and only 25% of the coefficients involving SDs yielded significant findings. More significant values were observed using WMS factor scores (table 5.27).

It is very interesting, however, that the direction of the correlations is invariably negative; ie, higher LM scores are associated with faster RTs, and with smaller SDs, in all Sternberg conditions. This finding parallels that observed using WMS factor scores.

TABLE 5.27: CORRELATIONS OF WECHSLER MEMORY SCALE
WITH MEDIAN RT & SD, SAMPLE A

6/12 FU: Fact	<u>or 1</u>	Facto	or 2	Fact	or 3
(n=35) RT	SĎ	RT	SD	RT	SD
Set +1:68**	58**	60**	50**	51**	51**
-1:63**	38*	60**	55**	41*	33
+2:59**	32	50**	32	33	14
-2:63**	29	46**	42*	34	41*
+3:27	55**	22	42*	36	~.51**
-3:24	58**	19	41*	33	45**
+4:59**	66**	44**	36*	28	32
-4:56**	61**	49**	48**	37	40*
24/12 FU:					
(n=19) RT	SD	RT	SD	RT	SD
Set +1:20	34	37	51*	22	40
-1:19	15	38	40	32	43
+2:35	13	37	1º9	12	10
-2:31	40	37	44	16	19
+3:41	30	49*	54*	30	51*
-3:26	52*	45	55*	28	74**
+4:01	55*	21	49*	01	74**
-4: .01	14	29	25	04	-,14
*=p<.05;	**p=<.01	;			

TABLE 5.27a: CORRELATIONS OF WMS LOGICAL MEMORY
WITH MEDIAN RT & SD, SAMPLE A

	<u>6</u>	/12 FU	24	/12 FU
	,	n=35)	(n	=19)
	<u>RT</u>	<u>SD</u>	RT	SD
Set +1:	50**	28**	07	28
-1:	48**	31	20	13
+2:	33*	17	27	22
-2:	34*	40*	27	37
+3:	46**	41*	32	19
-3:	38*	44**	23	51*
+4:	26	36*	04	51*
-4:	29	35*	08	30
*=p<.05;		**=p<.01;		

Little change was noted in mean LM score between 6 months (11.3, SD:3.4) and 24 months (11.6, SD:4.3).

Subjective Memory Questionnaire (SMQ) data at 2 years post head injury were available on 21 of the subjects in sample A (appendix table C8.4). The correlations of SMQ with U/C (-.10) and PTA (-.28) were not significant, and only 2 (of 16) correlations with median RTs and SDs at 24 months yielded significant correlations (table 5.28). However, a majority of these coefficients with median RT at 6 months

were significant, most at the .01 level.

TABLE 5.28: CORRELATIONS OF 24/12 SMQ WITH MEDIAN RT

& SD AT EACH FU, SAMPLE A

FOLLOW-UP

Media	n R	Γ	1/12	3/12	6/12	12/12	24/12
			(n=15)	(15)	(21)	(20)	(21)
Set	1 +	⊦:	.02	49	64**	29	27
	-	- :	. 06	33	61**	53*	22
	2 +	+ :	.09	.08	67**	.08	43
	-	-:	. 11	.08	64**	.07	31
	3 +	⊦:	.01	.08	14	27	50*
	-	-:	.01	. 09	12	27	24
	4 +	+ :	. 10	. 08	61**	34	26
	-	~:	. 09	. 01	54*	40	22
SD							
Set	1 -	+ :	.02	58*	45*	21	02
	_	-:	.12	26	25	25	21
	2 -	+ :	02	35	47	.00	59**
	-	-:	Ó8	51	40	05	39
	3 +	+ :	08	18	30	14	24
	-	-:	01	.03	33	46*	26
	4 +	+ :	07	28	57**	48*	28
	_	-:	02	26	66**	48*	16
*/	Ω5.		**/	0.1			

5.5 SUMMARY

This main experiment aimed to describe the recovery of memory scanning ability following head injury. The study included 2 patient samples and a small number of normal control subjects. A number of hypotheses were tested, and memory scanning was investigated using median RT and SD. Follow-up assessments on subjects were conducted at 1, 3, 6, 12, 24 and 36 months post-injury. The results obtained were related to severity of head injury and to a range of variables from other memory tasks. The data was also examined in terms of other clinical variables, such as neurosurgical intervention, prescription of anticonvulsant medication, and time to return to work/school. Other variables examined included age, sex, and, intellectual level.

CHAPTER 6

DISCUSSION OF MAIN STUDY RESULTS

6.1 INTRODUCTION

The main experiment in this thesis included subjects with a severity of head injury, from mild to extremely severe. Sample A was constructed to provide a group of comprising approximately one-quarter each subjects with mild/moderate (M/M), severe (S), very severe (VS), and extremely severe (ES) head injuries. The literature, reviewed in section 2.5, suggests a significant relationship between severity of head injury and level of impairment, and recruitment cognitive of sample representative of the population of head-injured people for the current study would have produced a group in which 89% of subjects would have sustained a mild/moderate trauma, and only 6% a very severe or extremely severe injury (table the focus of the present experiment was λs examination of the relationship between one aspect cognitive functioning (memory scanning ability) severity of head injury it was appropriate to construct a sample 'biased' towards higher severity.

This greater severity is reflected in sample A's GCS scores (median: 7), duration of coma (median: 39hr), and length of PTA (median: 7 days) which lie at the boundary of the severe and very severe categories. Table 5.2a shows the average scores for the different severity groups on these

variables. Higher severity is probably also indicated by a frequency of any epileptic fit of 19% in the current study, compared with the 'population' expectation of 5% (Skilbeck et al, 1986).

In other ways sample A was a more typical sample. For example, the highest incidence of head injury is in the age range 15-19 years and in the present study the median age was 18 years. Typically, the ratio of males to females in head injury is 2:1, and in the present experiment it was about 3:1. It seems likely, however, that the educational level of sample A (table 5.1b) was higher than would be expected from a random sample of head-injured patients. Why this was so is not clear, although there is no evidence that the sampling procedure for the study was flawed.

The experiment aimed to test the memory scanning ability of sample A subjects at 1, 3, 6, 12, 24, and 36 months post-injury. This was achieved, though only approximately 25% of subjects attended at the 36-months point in sample A. The latter was partly due to the author moving post to another Region (at which point 12 subjects had not reached their 3-year follow-up), though a number just failed to attend the final follow-up (appendix C4), including 4 who moved to another part of the country. This latter point suggests that applying a 'geographical' criterion when

selecting subjects for long-term studies may not always be of assistance. Sample B was specifically included in the current study to support the examination of patients' recovery between 2 and 3 years post-injury, given the predicted difficulties in maintaining a sample (A) over a Other authors have commented on 3-year period. the problems in sustaining subject attendance over long-term follow-up (section 2.5). In the currents experiment 95%+ attendance was achieved at 6 and 12 months post-injury, with about two-thirds of the sample attending at 3 and 24 months. Testing subjects at the 1-month point (55% of sample) was restricted by the inaccessibility of 12 subjects who were still experiencing PTA. Attendance rates appear to have been quite successful in the light of the difficulties often noted in maintaining samples over extended periods; for example, Conkey (1938) managed to obtain less than a 20% rate for attendance at all 4 followups planned for the first year after head injury in her study.

6.2 MEMORY SCANNING DATA: RECOVERY IN MEDIAN RT

As was pointed out in chapter 4, RT data is usually skewed which complicates analysis of results by making direct reference to mean values in statistical analyses invalid. One solution is to base analyses on transformed RT scores

or log), although this can make it (reciprocal, more difficult for the reader to grasp the meaning of significant differences between values, and the individual data points lose a 'direct' relationship with actual RTs. The solution preferred by the author was to base analysis upon median RT values which offer a typical or average score for the subject and are meaningful to the reader. Master (1982) commended median RT as the single best descriptive index of response latencies.

major aim of the main study was to use Sternberg's The paradigm to illustrate cognitive recovery following head injury, and to investigate the relationship between memory scanning ability and severity of head injury. The first specific hypothesis was that, using memory scanning data, possible to identify continuing it would be cognitive recovery at 12. 24. or even 36 months post-injury. Inspection of the median RT values for sample A displayed in table 5.3 tends to support the argument that meaningful recovery took place after the 24 month follow-up. Although direct comparison of the two points is not totally valid due to the differences in sample size, it is illustrative of the 'improvement' in median RT between 24 and 36 months injury (the average change being about 100 msec). This tendency for continued recovery even after 24 months also reflected in figure 5.2a. However, statistical

analysis of this recovery tendency, using group data, fails to demonstrate significant recovery between the two points 1, appendix C6). Table 5.7 indicates statistically- significant recovery for sample A was achieved in comparing the 6-month data with that obtained the 12- and 24-month follow-ups in some information conditions, but not at all when comparing the 12-month data with that obtained at 24 months. Similarly, the data for S and ES subjects reflected significant improvement beyond the 6-month follow-up, but not beyond 12 months. Again, the available median RT data (table 5.3) for these severity groups appears to suggest (as it does for sample B) later improvement, but the within-group variability performance mitigates against demonstrating significant recovery with group data and t-tests. However, examining the data in terms of frequency of improvement in sample A median RT between follow-ups (table 5.8) offers some evidence of significant change between the 12- and 24-month points. No 'frequency of recovery' support is provided, though, for the 24-36 month interval.

The point concerning the 'swamping' effect of large SDs in group studies is well recognised, and over the last few years use of single subject designs in neuropsychology has been strongly advocated (eg Shallice, 1979; Marshall & Newcombe, 1984)). Statistical procedures based upon single

subject data are now 'respectable' in Neuropsychology. Single-case computer software is available, offering a range of programs, including the Point Biserial correlation used in this dissertation. The existence of quantitative techniques such as this for individual subject analysis, incorporated into routine research practice assist the neuropsychologist clinical researcher. conclusion from group results is that the specific hypothesis relating to detection of recovery at 12 months after head injury, and beyond, is only partly supported, statistical analysis (table 5.6) shows a strong effect of recovery over time, and certainly significant improvement median RT can be observed at the 12-month follow-up. The Binomial test findings also point to continuing recovery in the 12-24 month interval. However, individual case analysis lends much stronger support to the hypothesis that cognitive improvement following head injury can occur at 24 months or later.

The observation of evidence to suggest recovery beyond 12 and 24 months post-injury is a very valuable finding: most researchers into cognitive recovery following head injury have completed their follow-up by the 12-month point (Brooks et al, 1984), and there is little data available in the literature from which to gauge continuing recovery beyond this point. Notable exceptions are offered by the

Mandleberg (1975), who was investigating work of recovery, and Van Zomeren & Deelman (1978) who examined In both of these studies evidence was gained of choice RT. continuing cognitive recovery in the second year after head It seems unlikely that the observed changes in RT performance over time resulted from practice effects, given the nature of the task material compared with, traditional IQ and memory tests. Also, the inclusion of a control group (sample C) allowed examination of 'practice' hypothesis, and no evidence was forthcoming to support the operation of such effects. Given the dearth of studies examining long-term cognitive recovery following injury, the current noting of such recovery in the second year post-injury (and perhaps longer) represents a significant finding in the field.

Another specific hypothesis attached to the general aim of charting cognitive recovery using Sternberg's paradigm was that median RTs obtained from the memory scanning task would be differentially sensitive to severity soon after head injury; ie, that subjects who sustained more severe head injuries, as gauged from length of unconscious and PTA, would show slower median RT results. In addition, it was also predicted that this effect would be detectable over subsequent follow-ups, so that even 24 months post-trauma ES subjects would generate slower RTs. The data

obtained in relation to this hypothesis was convincing. Table 5.9 shows that at 1 month after head injury the M/M group produced significantly faster median RTs than subjects in either the S or VS groups. By 3 months a sufficient number of ES subjects were no longer in PTA, and were therefore included in analysis. From this point onwards this group's median RTs were generally slower than those obtained from subjects in other severity groups.

At most follow-up points S and ES subjects' RTs were significantly poorer than those of the M/M and VS groups, there being no great differences between the latter two groups after the first follow-up. With the passage of time, the finding of slower S group median RTs compared with the M/M and VS groups disappeared, so that by 12 no comparisons between these 3 groups were significant. The only unpredicted finding relating to severity groups was that which indicated better than expected performance from VS subjects. As was discussed in chapter 5. this finding did not arise from misclassification of subjects as determined by reference to GCS, duration of coma, or PTA. Although the finding of relatively good VS performance appears inexplicable, its validity is supported by the unexpectedly fast return to work/school of its subjects compared with those in other severity groups.

The strong association between severity of initial injury and median RT was also reflected in the correlations of median RTs in the various information conditions for sample A with the severity indices of U/C and PTA. As table 5.10 shows, these correlations were generally high at the 3-month point (most ES subjects were still in PTA at 1 month, and not tested), and then gradually weakened so that by 24 months post-trauma no coefficient attained significance. Similarly, no significant correlations were obtained from sample B at the 2-year follow-up.

The interpretation of these findings is that the effects of the head injury were clearly having a significant impact upon RT performance in the early months, these effects being proportional to initial severity. By the anniversary injury the process of natural recovery led to of subjects' RTs being determined to a lesser (though still significant) extent by initial severity. The association weakened as cognitive performance continued to recover over time, so that by 24 months after injury no significant relationship persisted between severity and median RT. interpretation is also supported by the significant correlations for sample B at 3 years but not by the surprising re-emergence significant associations between the two variables sample A at 3 years. The reappearance of significant correlations seems to have been a chance finding, perhaps particularly relating to the small sample size at that point (table 5.40).

Overall then, the hypothesis that median RT would sensitive to severity of head injury, both in terms of poor results from more severly damaged subjects soon after injury, and with longer-term follow-up, was strongly This finding is exciting, given supported. indicates that the memory scanning technique can become a very useful clinical tool. When coupled with the observation that the technique is also sensitive to longerterm recovery after head injury, there appears to be a strong case for developing the technique further so that it can be included in routine clinical neuropsychological Sternberg's paradigm has a considerable practice. theory, and the general field grounding in of performance and information processing has amassed a strong body of knowledge. In conjunction with this background, findings from the current investigation increase the probability that the paradigm will further our understanding of the nature of the cognitive dysfunction acquired as a result of head injury, and will be able to inform the subsequent rehabilitation process.

A number of hypotheses were generated in relation to patients' RT performance compared with non brain damaged subjects. It was predicted that even after 2, or 3, years of recovery the median RTs of the patient samples would be significantly slower than those of the control subjects in The t-test results provided in table C. confirm this for the 24-month point. Within sample A, only the M/M and ES groups produced significantly slower RTs than the normal subjects at that follow-up. Fewer significant t-values were noted when comparing samples A and B with C at the 3-year point. However, the prediction that patients' RT performance would remain abnormal even years after head injury was confirmed, with ES subjects providing the slowest RTs compared with the control sample, A major feature of the memory scanning process is that negative trial RTs should be longer than those for positive trials. This feature was generally observed in the present study, and is illustrated in figure 5.3.

It was also predicted that the regression lines of information load for patient subjects would show a larger slope variable than the control group, to reflect the increasing difficulty in processing the greater amounts of information. It was predicted (section 5.4.3) that the largest slope values would be observed in the ES group.

data depicted in table 5.15 t-test confirms the to the extent that the ES subjects showed prediction, significantly higher slope weights than those in all other severity groups at 12 months, and higher than the S and VS groups at 6 months. Even where the ES weights were not statistically different to those in the other severity groups, ES subjects generally showed higher slope values. Miller (1970) noted higher slopes in his five head-injured subjects compared with a normal sample, and in the current study the hypothesised greater slope values for samples A and B relative to sample C was partly confirmed: sample A showed non-significant larger weights at 24 and 36 months after injury, with significant t-values being observed when B and C were compared at both 24 and 36 months post-trauma. The patient samples did show, however, a similar degree of high linearity to that offered by sample C subjects (figure 5.4e). The results were, therefore, consistent with the view that the brain damage acquired from a severe head injury can reduce the speed of information processing per se, rather than just producing a general overall slowing.

An additional hypothesis tested in relation to RT latency involved the examination of error responses. As indicated in chapter 3, some doubt has been expressed on the inclusion of error trials in analyses given that they may have involved inadequate memory scanning/poor information

processing. Clinical studies on the topic are although low error rates (1%-4%) have been reported (Pharr & Connor, 1980) in schizophrenic patients, the error trials tending to show longer RTs. This finding goes against the prediction (Welford, 1980a) that faster RTs are more likely to result in errors. Warren et al (1978) noted higher error rates of approximately 7% in aphasic patients, with Hart & Kwentus (1987) reporting 6% for elderly depressed patients and 4% for their normal controls. In the current study, both patients (3%-4%) and controls (2%) demonstrated low error rates and, as was reported in the last chapter, the results obtained did not suggest that an error was more likely when a subject produced a faster-than-average RT. Although only a very superficial error analysis undertaken, the results obtained suggested that tended to follow fast, accurate responses. This might be interpreted as indicating that an error response represents a deterioration in attention from a relatively good level.

The main findings for median RT have been discussed above, and theoretical aspects of the RT results might be discussed at this point. However, as the sections below consider findings such as RT variability and relationships of RT indices to other memory tasks, theorising on the mechanism(s) of cognitive dysfunction and recovery is placed towards the end of this chapter (section 6.6).

6.3 VARIABILITY OF RT: STANDARD DEVIATION (SD)

Analyis of variability of RT, using SD as the index, was undertaken to help explore the putative relationship between attentional mechanisms and the production of RTs according to severity of head injury (section 5.1). Many of the basic findings obtained were similar to those noted For instance, significant median RT. in relation to SD occurred following head injury, this recovery in recovery being related to severity (table 5.16), and SD varied according to set size. Recovery in SD over time was particularly marked in the S and ES groups see figure 5.5). The overall correlations of SD with U/C and PTA were not significant 1 month after injury (when most ES subjects in the analysis), but very strong not included coefficients with U/C were obtained at 3 months (0.63-0.82) somewhat lower values (0.39-0.64), though still and significant, with PTA at that point. The size of correlations of SD with the two severity indices gradually reduced between 6 and 24 months post-trauma, so that by the latter point none were significant. However, as in relation to median RT remarked upon above findings, significant correlations re-emerged after 36 months, for to the subsample of patients who attended the final follow-up.

It is worth noting that SD generally showed very high

correlations with median RT at each follow-up. The finding those subjects who showed the slowest RTs also produced the most variable RTs tends to reinforce the arguments linking poorer attention with longer latencies: if patients' slower RTs stem from attentional dysfunction, then it would be predicted that both SD and median RT would adversely affected, the levels of impairment produced being correlated. Table 5.19 also suggests that the size of the association between median RT and SD was independent of set size. It would appear, therefore, that SD (like RT also) is able to offer a cognitive index which is sensitive both to severity of head injury and to recovery over time.

In general, the findings demonstrate the sensitivity of the memory scanning technique to severity of head injury and to When linked to its capacity to demonstrate recovery. persisting abnormality years after injury, these findings open up the possibility that memory scanning might be used in a large-scale manner as one factor in the prediction of longer-term recovery of patients, using data acquired soon after head injury. Parallel prediction work has carried out in the field of stroke recovery (Skilbeck, Langton-Hewer, & Wood, 1983). Developing reliable predictions of cognitive recovery would provide descriptive base against which the success of therapeutic interventions could be judged.

6.4 FINDINGS FROM OTHER VARIABLES

a. Clinical & Demographic Variables

Superficially, the finding that patients undergoing neurosurgery soon after head injury showed RT recovery which was as good as (and perhaps marginally better than) those who did not receive surgery is surprising: Jennett et a l (1979) found that the presence of an intracranial haematoma and its removal by neurosurgery was associated with a poorer outcome. However, in the current study only 3 (out of 7) subjects underwent neurosurgery to evacuate an intracerebral haematoma, and most subjects received neurosurgery to elevate depressed skull fractures. It has been suggested that occurrence of a skull fracture in head injury is actually a good prognostic sign, as some of the energy of the trauma to the head is absorbed by the skull rather than being directly transmitted to brain tissue. Also, neurosurgeons are somewhat wary about undertaking skull repair following head injury if the brain shows evidence of undue swelling: in such a situation the concern is that the brain will herniate through the hole created by bone removal during the repair. Because of this, the subjects who underwent neurosurgery soon after the injury present study probably showed only mild brain swelling. This may have operated as a selection criterion favouring mild brain damage, and in addition it could be

argued that the patients who underwent neurosurgery might have received higher quality medical care in terms of closer monitoring (by neurosurgeons rather than medical consultants) and better access to intensive care facilities. These features may have assisted the cognitive data on some patients who experienced neurosurgery.

A general point is that the above finding supports the argument for neurosurgeons assuming responsibility for a wider range of (ie, including less severe) head-injured patients. Although the finding of marginally-better early recovery in patients undergoing neurosurgery is very tentative, if confirmed in subsequent studies it would help to underline the value of using memory scanning testing in the assessment of head-injured patients.

showed evidence of patients who some additional lateralised brain damage, the choice RT literature offers a study (Dee & Van Allen, 1973) to suggest that left hemisphere lesions yield steeper RT slopes (ie, poorer informatioon processing speed in patients with this type of damage. Of course, in the current research no patient had damage restricted to only one cerebral hemisphere, but the data obtained provided no support for Dee and Van Allen's finding: patients with additional left hemisphere damage generally produced similar results to those who showed

extra right hemisphere involvement, and when significant differences were observed (at 3 and 24 months) they suggested better cognitive functioning in the 'left hemisphere' group. The hypothesis of poorer cognitive performance in this latter subsample was, therefore, not supported. Klatzky and Atkinson (1971) in their memory scan research obtained evidence to indicate a right hemisphere superiority for processing letter stimuli, their interpretation being that the letters would efficiently (ie, more speedily) processed using spatial, rather than verbal-acoustic, characteristics. It might be predicted, therefore, that subjects in the current study who sustained additional damage to the right hemisphere would produce poorer performances. The observation of a marginal superiority for the left hemisphere is consistent with this prediction.

Of the 40 subjects in sample A who were in work or at school prior to their head injury, 23% failed to return to work/school during the period of the study. This figure might seem high compared with those available from other studies (eg Rowbotham et al, 1954; Oddy, 1984), though it has to be remembered that the current research did not recruit a representative sample of hospitalised headinjured patients, but rather one deliberately biased towards greater severity. In fact, two-thirds of those who

failed to return to work/school were in the ES group. current data might be better compared with that observed by Oddy et al (1985) which indicated a 48% return by the 2. year follow-up. The review in section 2.6 pointed out that cognitive dysfunction appears to partly determine time to return to work, and the present findings offer some support for this position. Although no correlations of 'time to return' with RT indices were significant imediately after head injury, by the 3-month point 50% of the coefficients exceeded +0.4. The 6-month point is perhaps the most appropriate to examine the relationship between cognitive functioning and ability to return to work/school, given that the mean time to return was 5.9 months. At that point 50% of the computed coefficients were significant, mainly in relation to median RT. The suggestion that there is a 'lawful' association between severity of head injury and ability to return to work/school is supported by the significant (p<.05) correlation between time to return and both U/C and PTA.

This finding raises the possibility that the management of head-injured patients' recovery can be assisted by accurate prediction of the time required to return to work or school. The sensitivity of the memory scanning technique to severity of head injury and subsequent cognitive recovery could lead to its development as a predictive tool

in early post-trauma assessment. For this to occur, an emphasis upon outcome measures is necessary in future work.

Although the number of patient subjects experiencing posttraumatic epilepsy in the current research was too small to permit investigation, a limited attempt was made to examine medication upon effects of anticonvulsant cognitive performance. Earlier reviews (eg, Trimble & Thompson, 1981) have pointed to the potentially deleterious effects upon cognitive abilities of taking this medication, and there is case study evidence available in relation to memory scanning (Skilbeck, 1984) to suggest RT slowing from anticonvulsant medication. In the current experiment no data were gained from the small number of patients studied suggest that the taking of anticonvulsant medication negatively affected RT performance. The reason for this is clear, though only a small number of subjects were investigated and it may be that in the first year postinjury that the massive adverse cognitive effects of the acquired brain damage itself do not allow detection of more subtle influences upon cognitive functions which may be attributable to the medication.

Age effects upon memory scanning ability have been reported in earlier studies (eg Anders et al. 1972; Eriksen et al. 1973), and recent work in the general field of choice RT (Beringer, Wandmacher & Gortelmeyer, 1988: Hindmarch, 1988) has helped to confirm the asociation between response latency and age. Salthouse and Somberg (1982),in their comprehensive experiment on age, manipulated task complexity at the encoding stage (degraded stimuli), comparison stage (memory set size) and response separate finger digits). choice stage (Yes/No, investigated young and old subjects, and noted that age interacted with performance at all three stages. They concluded that a general ageing effects factor was operating.

Table 5.20 shows that in sample A the correlations of age with median RT and SD change with time since head injury. In the early months median RTs and age correlated well, with some significant coefficients involving SD, too. At 6 months post-trauma only occasional significant correlations were observed, though strongly significant values were noted at 12 months before the return to non-significant findings at the 24- and 36-month points. These findings appear difficult to explain. It might be argued that the negligible correlations observed at the final follow-up points merely reflected the greatly reduced sample size at

those follow-ups, although sample B's results showed large, significant values at 24 and 36 months with small numbers. provided no convincing evidence of strong associations between age and RT indices, although with its more restricted age range (18-34 years) this is perhaps not good test of the putative relationship. There are a number of differences between the current research and most of the existing literature. Most important amongst these is that head-injured subjects are the focus of the current study. Given the age-related risk of suffering a head injury (see section 2.1); most of the subjects studied were in the age range 15-25 years. This 'restriction' upon age narrow, young band may have produced increased instability in terms of the coefficients obtained when correlating RT indices with age, and a lower probability of detecting any age relationship. It could be, too, that age effects are much more likely to be observed when brain functioning is significantly compromised. This would be consistent with sample A's results (table 5.20): if the age variable interacts with cerebral integrity, then the improvement in brain function efficiency which occurs with increasing time post-trauma would be expected be associated with a reducing correlation coefficient between median RT/SD (as indices of cerebral efficiency) and age.

In his review of the field, Welford (1980b) concluded that there is good evidence to indicate slower RT in females compared with males (with the possible exception of the early teenage years), the probable basis of this difference being biological. The findings in the currrent research are opposed to this conclusion. Although female and male patients in sample A showed no differences in terms of severity of injury or age, sufficient evidence accrued across the various follow-ups to suggest a marginal female superiority in RT performance. It may be that this is just a chance finding, although another finding from the study helped to validate it as meaningful - females took a significantly shorter time (p<.10) to return to school/work after injury. The explanation as to why females should better/faster recovery is not clear, though occasional findings in the literature relating to recovery from aphasic deficits have suggested a faster improvement in females (eg. Basso et al. 1982).

In the current study estimated premorbid IQ was also used as a reference variable to aid consideration of the RT findings. Estimated premorbid IQ rather than observed IQ was used for this purpose given the extensive literature indicating IQ deficits associated with head injury (section 2.5.3). Validity of the estimates was suggested by the negligible correlations noted between Performance IQ and

verbal IQ with indicators of head injury severity. Over the sequence of follow-ups, the IQ variables showed varying correlations with RT indices. In the early months no significant associations between the IO and RT variables were indicated, occasional significant values were noted at the 12 month point, and more consistent sigificant findings were obtained at 6 and 24 months. There are a number of studies indicating a negative relationship between IQ and Rabbitt & Goward, 1986). However, the review by Nettlebeck (1980) concluded that 'The degree of correlation may be reduced, or even disappear, among samples with average and above-average intelligence..' (page 357). present study the mean estimated premorbid IQ sample A (approximately 108) lay towards the top end of the average range, at about the 70th percentile compared with the general population. This finding, coupled with Nettlebeck's position probably offers the most parsimonious explanation for the lack of clear relationships between IQ and RT in the current research.

b. Other Memory Task Results

In terms of accounting for the findings obtained in this thesis, the main source of information against which to discuss the results is undoubtedly the available literature associated with Sternberg's paradigm specifically, and RT

more generally. However, particularly given the clinical nature of the research, a contribution to discussion and theorising is also offered by the findings from other memory tasks in the study, including their relation to RT At each follow-up subjects in sample A were administered the Rey AVLT and WAIS digit span (WDS), and at the 6- and 24-month points WMSs were completed. Subjective data on memory functioning were obtained (SMQ) after 2 years post-trauma. Inclusion of these memory measures allowed investigation of the recovery process in areas other than memory scanning, and also made it possible to coordinate these findings with those from the The Rey AVLT offers measures scanning RT data. of new learning, the effects of proactive and retroactive interference, and both recall and recognition scores. The WDS assesses immediate memory/attentional span, and the WMS factors reflect short-term memory/learning, attention and concentration, and orientation; see Lezak (1983) for a detailed description.

The Rey AVLT. WDS, and WMS all showed some sensitivity to severity, in that ES subjects' performance was often significantly poorer than those in other groups. The best indicator in this respect was the Rey, which showed poorer ES scores from the 3-month follow-up onwards. Correlations computed to compare Rey variables with the severity indices

of U/C and PTA also reflected this sensitivity; for sample A, only the 12-month data failed to yield significant coefficients. Data for sample B indicated significant correlations at the 36-month point. Thus, the deficit in new learning resulting from head injury appeared to be proportional to severity.

Compared with the Rey. the WDS yielded a smaller number of significant t-test comparisons for the ES group against the others, and fewer significant correlations with severity indices. However, at 24 months the WDS was able to detect significantly poorer performance in the ES group. One interpretation for the WDS findings is that immediate memory, or attentional span is generally less vulnerable to impairment by head injury.

WMS factor scores showed a good relationship with severity, both in terms of correlation analyses and with regard to ES subjects' performance compared with those in other groups at the 6 month point (factor 1 being most sensitive). Much weaker associations were observed at the 24 month follow-up, though factor 2 (attention/concentration) performance still discriminated between ES subjects and those in the S and VS groups.

Overall, the additional clinical memory tasks were less sensitive than the RT indices, at every follow-up point, to severity of initial head injury. The Rey performed closest to the RT findings. The lower sensitivity compared with RT measures was also apparent from the point of view of detecting improvement between follow-ups. A small number of Rey variables showed improvement for sample A between 6 and 12 months post-injury, though no between-follow-up comparisons for the ES group achieved significance. The WMS factor scores offered no evidence of significant recovery between 6-24 months, and no between-follow-up comparisons proved significant for the WDS variables.

The findings from these other memory tests are consistent with the existing literature (eg Russell & Smith, 1961; Schacter & Crovitz, 1977; Brooks & Aughton, 1979b) in reflecting significant associations between head injury severity and level of memory impairment. Many relevant studies have employed the WMS (section 2.5.1), with poor scores being obtained long after the trauma (Brooks, 1976). Brooks (1976) also concluded that WDS often shows a good recovery following head injury, suggesting that immediate memory capacity is perhaps less adversely affected by head injury. Such an argument receives some support from the current finding of a relatively weaker connection between WDS and severity indices compared with other memory

variables, although it should be remembered the immediate memory capacity of ES subjects remained poorer than other subjects even 24 months after injury. learning is said to show a slow recovery curve (2.5.1), although Schacter & Crovitz (1977) pointed out that studies needed to include more follow-up points to allow sufficient test data to be gathered for an adequate description of The present study included a large number of recovery. and tended to support (via Rey findings) the follow-ups that verbal learning recovers slowly: some prediction significant changes were noted in Rey variables beyond 6 months.

As discussed in section 2.5.1, there is a debate concerning the relationship (or expectation of a relationship) between subjective and objective memory measures. Sunderland et al (1984), however, reported significant associations between the two types of measure, and the current research supports their findings: although the associations were much lower at 24 months, RT data obtained at 6 months after injury correlated significantly with SMQ scores. This finding is encouraging, suggesting that early pessimism concerning the connection between 'real-life' memory impairment and memory test deficit may have been premature, or overstated. Discovery of meaningful correlations between these two aspects of memory performance opens up the possibility of

predicting the level of subsequent subjective memory impairment experience from objective testing soon after head injury. Such predictions could lead to improved counselling with regard to future educational and occupational difficulties arising out of the trauma.

It was clear from the results presented in chapter 5 that significant correlations existed between the clinical memory tests of Rey, WDS and WMS, and the RT measures obtained from the memory scanning task. The most obvious findings were provided by the Rey variables. At 3, 6, 24. and 36 months a large number of significant correlations of these variables and the RT measures (particularly median RT) were observed. The Rey is a learning task which measures the rate at which new information is aguired and allows the effects of interference to be assessed. Given that the Sternberg memory scanning paradigm was designed to offer an information processing task it is perhaps not surprising that its principal index, RT, correlated well with the Rey attentional/procesing memory variables of interference and rate of new learning. Ιt is consistent that the size of these correlations rises with positive set size, as the latter is a major determinant of information processing speed. The Rey variable 'A1', which is a measure of span (and initial learning), rather than speed of processing, generally showed lower correlations with RT measures (as did the WDS).

The associations between RT memory scanning measures and discussed clinical, memory test results will be further below in considering elements of a model describing memory scanning in head-injured people. For the can be concluded that clinical memory tests moment it showed sensitivity to severity of head injury and to the cognitive recovery. However, this sensitivity process of lower than that demonstrated by the RTindices. although the pattern of findings was consistent with that expected from the existing literature. The poorer sensitivity findings noted for the clinical memory tests, in terms of both relationship with severity of head injury and detection/description of cognitive recovery, once again point up the value of the findings observed for the memory scanning procedure. There is, therefore, a case to be made developing Sternberg's paradigm to for provide an additional clinical neuropsychological tool for routine use in the assessment of cognitive dysfunction following head injury and its subsequent recovery. In future research it will be important to examine the usefulness of the paradigm in terms of its relationships with outcome measures such as academic or occupational performance.

6.5 A MODEL OF MEMORY SCANNING IN HEAD-INJURED PEOPLE

6.5.1 Introduction

The data gathered in the pilot study and main investigation for this thesis point to the value of using an information processing approach to the examination of cognitive deficit following head injury. The results obtained indicated slower processing in head-injured subjects, and suggested they are more vulnerable to distraction or the presence of irrelevant information. The findings in the main study also strongly support Sternberg's hypothesis: serial, exhaustive memory scanning fits the observed data. and a linear relationship between number of items to be scanned and RT was noted.

Although the RT differences between positive and negative set trials was initially very variable, from the 6-months point onwards 75% of them lay in the 30-70msec range. This overlaps with the 40msec quoted by Sternberg (1975) for normal being typical subjects. Sternberg also indicated that 400msec a representative intercept was In the present research patient subjects showed higher values than this in the early post-trauma months. 12-month point the 'normal' value though by the obtained (the ES group remained markedly higher).

Similarly, the 40msec per item slope weight typically seen in normals was not approximated for sample A until the 36-month follow-up, with ES subjects generally showing larger values.

It can be concluded from the present study that Sternberg's paradigm has yielded findings which indicate both its sensitivity to initial severity of head injury, and its ability to reflect the process of recovery. Over time patient subjects' RT performances changed towards that expected from normals. The paradigm offered insights into the nature of the disturbance in cognitive functioning produced by head injury, and helped to describe the return towards normality. Sternberg's procedure potentially offers a valuable method for investigating the cognitive disturbance arising from head injury. If it can be developed to provide data to predict recovery then it will assist the process of counselling patients and their relatives on the longer-term implications of the cognitive damage sustained. It may also be possible to gain insights from the paradigm into the processes underlying cognitive thereby assisting any rehabilitative disruption, interventions which may be offered.

Discussion continues in the literature with regard to the most appropriate model to account for memory scanning data,

although Sternberg's remains the most acceptable. In their information processing models Meyer, Osman, and Kounis (1988) considered various theories and concluded that the most popular model, and the one with the greatest support, is Sternberg's. These authors felt that recent parallel processing models, such as the model, may eventually offer closer parallels with current concepts of brain structure and neural mechanisms. The Cascade model is similar to that proposed by Sternberg in construing discrete stages and in assuming that responses stimuli are mediated by a set of processes ordered according to encoding, retrieval, decision, and response preparation, through which information passes direction. Because the Cascade model includes parallel operations it would be impossible to estimate the absolute duration of a stage using the method of subtraction.

However, the primary purpose of the current thesis was not to critically examine Sternberg's model against others, but was to test out some of its predictions with head-injured patients and to assess its sensitivity in relation to severity of trauma and recovery. In this regard, a number of theoretical questions remain. For example, how are the findings of this thesis on brain-damaged subjects to be incorporated into Sternberg's model, and which concepts of brain functioning, attentional mechanisms, and information

processing are most useful in assimilating these findings into the model?

Clinical observational description has long reference to a deficit in attention following head injury (section 2.5.2). The concept of 'attention' in clinical studies is often an uncertain one, and the literature reflects the confusion (see Van Zomeren, 1981, for brief review). Posner and Bois (1971) specifically addressed the problem in an excellent discussion paper. After considering various concepts, and some of the available studies. these authors suggested that there components of attention:

- 1. Alertness (sensitivity to external stimuli)
- Selectivity (ability to filter out irrelevant stimuli)
- Central processing (limitations on the ability to simultaneously process a number of stimuli)

These are key components in the understanding and description of the memory scanning deficits noted in patients in this thesis. These components will be considered individually, and will then be included in a model.

6.5.2 Alertness/Arousal

Some psychophysiologists use concepts of 'alertness' or CNS in discussing attention. It has been arousal argued 1979) that this state of readiness to receive (Ommaya, stimuli, and to respond on a specific task, is partly cortical-subcortical maintained by connections: particularly implicated are the frontal cortex and the brainstem Reticular Activating System (RAS). It seems very pertinent that the primary damage acquired in head injury (section 2.2.1) is of diffuse contusional lesions to the under surfaces of the frontal lobes and to the poles of the temporal lobes, resulting in loss of brain cells, coupled with the shearing of axons in the white matter of the brain (particularly brainstem).

evidence in relation to physiological indicators of arousal/alertness and RT performance is beginning to accumulate. Fo example, it has been shown (see Van Zomeren et al, 1984, for brief review) that EEG changes accompany a in RT studies. These cerebral changes forewarning Negative Variation termed Contingent (CNV) or the Expectancy Wave, and reflect the person's preparation to respond following the warning stimulus. The early stages of these preparations particularly involve frontal cortical activity, and the very occasional studies using headinjured subjects which have been undertaken to date point to reduced CNV effects in this group. Stuss et al (1985) also speculated on the pathophysiology of the attentional deficit they observed with head-injured patients (using Brown-Peterson and Stroop tests) suggesting that this could be related to brainstem dysfunction and/or a lesion affecting fronto-RAS connections.

Welford (1980b) considered arousal (equivalent to general alertness) in terms of the 'inverted -U' hypothesis when seeking to explain the finding that prolonged on-task performance leads to RT slowing and a marked positive association between RT and SD of RT in normal subjects. Welford viewed the RT slowing as being produced changes ('CNS fatigue'), rather than by the marginal alterations in sense organ processing, nerve conduction speed, or motor activation. Findings from the current research might be seen as being consistent with Welford's view in that, for subjects whose CNS information processing ability was reduced through acquired brain damage, SD was proportional to median RT (Table 5.19). Of course, the brain damaged subjects were not experiencing prolonged on-In addition, however, in the current task testing. research a significant correlation between median RT and SD was also observed for normal control subjects. The research reviewed by Nettlebeck (1980) was interpreted as

indicating a clear relationship between RT and cortical arousal in various groups of subjects with brain dysfunction, including brain-damaged war veterans and schizophrenic patients.

In the past, Arousal Theory in relation to RT performance has received support from the findings of diurnal variation and anxiety effects (reviewed by Frewer & Hindmarch, 1988). In their own work, Frewer and Hindmarch observed diurnal variation, though only in their anxious and elderly subgroups, with slower choice RT being noted generally in these subjects. Broadbent (1988) reviewed the finding that added noise can aid auditory RT and the idea that this reflects maintenance of arousal (or readiness to respond). Yozawitz, Berenhaus, and Sutton (1985) Bruder, observed pair of auditory 'clicks' facilitated affective patients on an auditory RT task. The authors concluded that the clicks tended to overcome patients' originally-low level of arousal, favouring the explanation that the two not processed independently, but rather clicks were together, so producing an enhanced stimulus intensity.

In an important study, Holloway and Parsons (1971) found that in brain-damaged patients evoked heart rate (EHR) failed to show the predicted drop in anticipation of an expected (forwarned) stimulus to which an RT was required.

Also, unlike the findings for non brain-damaged subjects. no positive correlation between EHR and RT was noted. Emmerich, Fantini, and Ellermeier (1989) also investigated the suggestion that an auditory tone could facilitate a subsequent RT. Their experiment confirmed the effect, using simple auditory RT and a tonal background The findings indicated significant facilitation masker). with low levels of background tone (but not with a randomly-varying narrow-band noise). Emmerich et al offered little discussion on the meaning of their finding. though they did comment that "results are consistent with notion that the facilitation of RT....is due to the modulation of ongoing neural activity (initiated by the tonal background) which occurs as a result of signal presentation".

6.5.3 Selective Attention

Posner and Bois' (1971) use of the term 'selectivity' referred to the ability of a subject to filter out, or ignore, irrelevant information so that only selected elements are processed fully. This mechanism assists the rate of processing information as the system has a limited capacity. More recent consideration of selective attention has included the concept of automatic processing (preattentive) and conscious, controlled processing; Schiffrin

Schneider (1977) hypothesised that information is processed as far as is possible in the automatic mode (drawing upon overlearning in long-term memory) to minimise demands upon the limited capacity processor. The processing information which requires conscious control attention) draws upon this limited capacity. Baddeley's idea that a Central Executive (CE) component of working memory is necessary for the strategic handling of incoming information is also relevant here. His concept, Norman & Shallice (1980) and that of involving a Supervisory Attentional System (SAS), can be envisaged as assisting in the selection of information for processing (eq. in situations where automatic processes are unable to handle the incoming information).

Focussed Attentional Deficits (FADs) can arise i f the ongoing automatic processing confounds the response processing of a simultaneous consciously-controlled task: the FAD results from receipt of a stimulus for which there is a strong, conflicting response tendency. The Stroop test (Stroop, 1935) offers an exemplar task in the condition where the printed name of a colour (eg 'RED') is displayed in ink of a different colour, and the subject is asked to ink. The distraction of the word name the colour of the meaning is difficult to overcome and so tends to interfere with the controlled processing of the ink colour name.

Research has not offered support for the existence of FADs relation to response competition: using head-injured subjects. neither Chadwick (1976) nor Thomas in (reported by Van Zomeren et al. 1984) noted Stroop interference effects, beyond a general slowing brain-damaged subjects. When Van Zomeren et al (1984)noted these Stroop effects with head-injured subjects, they occurred on a choice RT task for which the competing responses had not been learned. Van Zomeren and his colleagues concluded that they had observed a DAD (Divided Attention Deficit), rather than a FAD (see below).

their review of the concept of attention Beringer. Ιn Wandmacher, and Gortelmeyer (1988) noted that theories often make reference to serial versus parallel processing, selective attention either being introduced at an early stage of the model (parallel processing being restricted to simple sensory aspect), or a later stage (selection for serial processing at semantic encoding stage). In a mixed group of brain-damaged subjects Callan, Holloway and Bruhn (1972) observed failure to filter, or select out, auditory distractor stimulus (tone) introduced immediately prior to the target visual stimulus presentation. other studies (eg Holloway & Parsons, 1971; Van Zomeren, 1981), these authors noted that the expected autonomic habituation to the distractor stimulus occured in the other groups but was much delayed in brain-damaged subjects. The latter can be regarded as poor selectivity (failure to inhibit response to distractor).

Although Miller and Cruzat (1981) in their card-sorting note an interaction did not between number irrelevant stimuli and type of subject (severe head injury, mild head injury, normal control), thereby implying a lack of support for a selective deficit hypothesis, the pilot study in the current research observed such an interaction (table 4.1). Not only was an interaction seen, but the significance of the effect was greater (p<.001) than for interaction of severity and target information load The experimental work in this thesis, therefore, provides evidence in favour of the selective attentional hypothesis in the explanation of information processing characteristics in head-injured subjects.

The pattern of earlier findings led Nettlebeck (1980) to suggest that in brain-damaged people two components of the central attentional process have become disengaged, so that although the reflex awareness of a stimulus is recorded this orienting response neither habituates with repetition, nor does it coordinate with the normal autonomic activity of EHR reduction. Reduction in EHR may be regarded as an index of readiness to respond on a specific task, and the

positive relationship between this reduction and subsequent RT reflects the attentional process. Brain-damaged people might be characterised as being overly sensitive to incoming stimuli if they are unable to habituate sufficiently their orienting responses, thereby compromising their ability to selectively attend to taskrelated stimuli. Such a mechanism failure might be evidenced by a proneness to distraction by irrelevant stimuli which interferes with subsequent performance. reduction in level of task attention and the lack of a correlation between EHR and RT (poor readiness to respond) contributes to the deficit in the attentional process.

A number of studies have investigated the performance of head-injured people under interference conditions on RT and learning tasks (eg Van Zomeren, 1984; Stuss et al. 1985) and have observed that head-injured subjects show significantly greater interference/distractibility effects than normals, so supporting an attentional model of cognitive dysfunction.

6.5.4 Central Processing

The concept of central processing is useful to consider in conjunction with attention, and there is evidence (Van Zomeren, et al. 1984) to suggest that head-injured subjects

process information more slowly than non brain-damaged Schiffrin and Schneider's (1977) concept of subjects. 'divided attention' does not imply division between two assigned tasks, but rather recognises that in coping with life it is necessary to process information from more than one source at a time, and so a limited capacity has to be shared. Evidence for DADs (Divided Attention Deficits), in the form of slower rates of information processing in brain-damaged subjects is strong (eq. Miller, 1970; Gronwall & Sampson, 1974; Van Zomeren, 1981). The absence of differences in the errors of normal controls and headinjured subjects suggests that the poorer performance of the latter does not arise from some general processing, but rather from a difference in rate of processing.

Findings from the present main study confirm this slower processing, and also provide some evidence (via slope weights) that extremely—severely damaged subjects manifest a differential level of deficit, (ie, they do not just suffer a uniform slowing, independent of the processing load, but rather a slowing which is proportional to the amount of information to be processed). Van Zomeren (1981) pointed out that slower central processing will result, of itself, in poorer attention.

In considering age-related RT slowing, Welford (1980c) used the concept of signal-to-noise ratio. He postulated that an older brain receives weaker signals from its sense organs and, due to loss of brain cells, signals between diferent CNS areas will also be weaker. He concluded that a poorer signal-to-noise ratio results, with consequently less efficient processing and, therefore, RT slowing. might also be predicted, according to Welford's argument that this less efficient processing would also produce greater variablity in response time and more errors. The large SDs noted for patients in the current research represent irregularity of performance and tend to support Welford's position. This irregularity did not, however, produce high error rates, and long RT trials were not associated with error responses. In fact. the data presented in section 5.4.2b tends to suggest that errors were more likely to occur following an attentional 'high' or faster central processing of information, the subsequent presumably resulting from a fluctuation error trial downwards of the attentional level (similar to Welford's 'CNS fatigue?). If a reduced level of attention was the factor, then inadequate stimulus coding (insufficient to allow a strong match with the target) might be the processing stage implicated. Certainly the data offered in 5.4.2b does not suggest that errors usually occurred as a result of attempting to process information too quickly.

Van Zomeren et al (1984) specifically pointed put that no research has directly addressed the question of relationships between information processing speed and the formation of memory traces. The question is important, because the slowing of information processing after head injury carries with it the prediction that patients will be unable to store information in memory as efficiently as they did pre-trauma. In the present research data on this issue was provided by the inclusion of the Rey AVLT and WMS memory tests, and adverse effects upon these measures from the head injury were observed. The results in chapter 5 (eg, figure 5.11; tables 5.22 & 5.25) both confirm the prediction, and highlight the significant relationship between degree of memory trace disruption and rate of processing (as measured by median RT).

`Combination of elements of the above discussion with findings from the current research lead to the thesis that slowing of RT and its increased variablity seen injured subjects needs to include reference to general arousal, task-related attention, and information processing capacity. The latter two concepts are not mutuallyexclusive, as Van Zomeren (1981) has indicated.

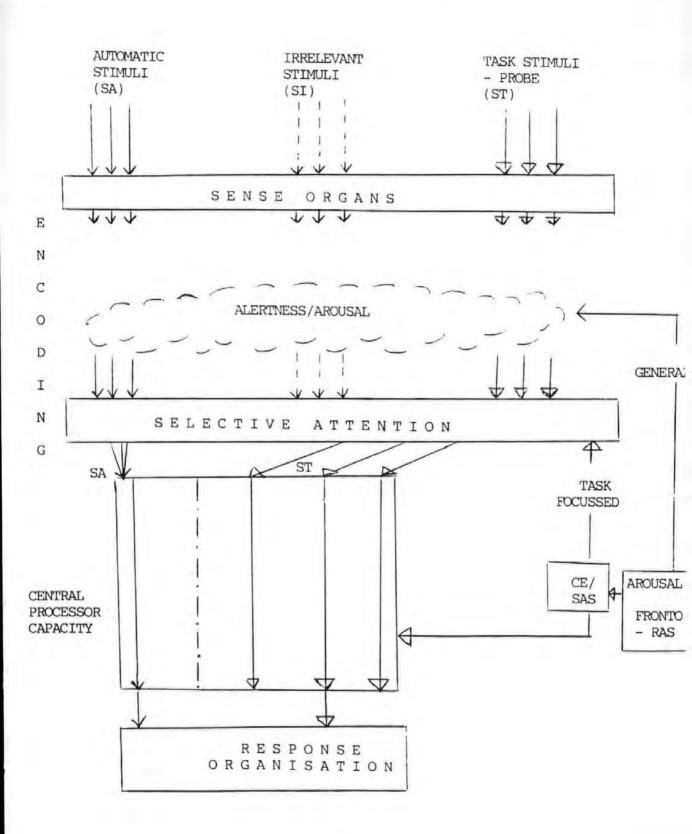
6.5.5 Elements of a Model

Rather than seeking to introduce additional concepts, it seems more profitable that theorising upon memory scanning performance should seek to synthesise ideas already available. This synthesis should, if possible, link to our current understanding of brain functioning. Figure 6.1 provides a diagrammatic representation of some of the key elements in an attentional model of memory scanning, based upon the preceding discussion. The situation depicted relates to an undamaged system. The model hypothesises 3 types of incoming stimuli: those (ST) directly relating to the specific task receiving attention (ie, probe stimuli), referring to automatic, overlearned behaviours those (SA) which do not require direct continuous direct attentional control (eg, very regular car driving), and those (SI) from other sources which are irrelevant to any current automatic or focussed information processing.

Figure 6.1 shows the reception of these 3 types of stimuli at the person's sense organs being influenced by the person's general alertness. or arousal level. alertness is presumed to involve modulation/monitoring by a fronto-RAS system, which may be reflected in EEG and EHR activity. This is seen as the beginning of the encoding of stimuli. The SA stimuli, as they are required for on-going non-conscious activities pass through the selective attention stage into the central processor. The selective attention process filters out irrelevant information (SI) and sustains the task information (ST). Activity in this stage may be reflected in CNV and EHR changes which accompany task preparedness. The central processor has only a limited capacity, and SA stimuli are presumed to require only a very small component of this capacity. This leaves maximal processor capacity available for the information, and the focussed task of serial memory scanning of items against the incoming probe stimulus information.

The model could also include Baddeley's concept of a Central Executive (CE)/Norman and Shallice's suggestion of a Supervisory Attentional System (SAS), discussed by Baddeley (1986). The CE/SAS supervises the Central Processor activity, directing it towards the memory scanning task and the comparison of the probe with the

FIGURE 6.1: NORMAL MEMORY SCANNING



positive set items held in memory. This process may be assisted by the CE/SAS influencing the Selective Attention stage, so that incoming probe information is favoured.

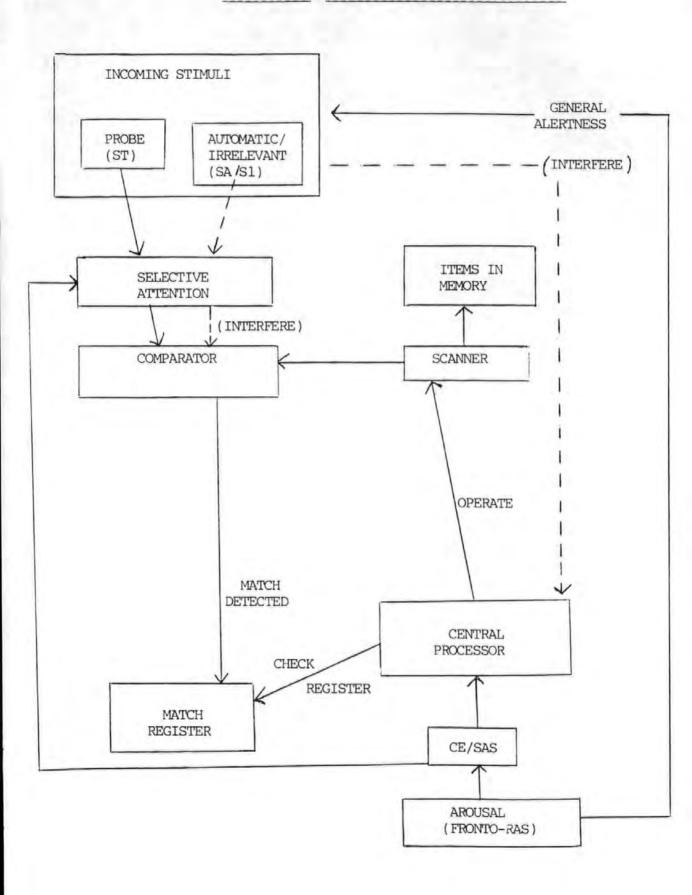
admittedly simplistic description of the processing situation may be compared with the author's 'worst case' detailed illustration of memory scanning by head-injured people (Figure 6.2). In this situation, it is hypothesised that the maintenance of general alertness by the fronto-RAS system is rendered faulty by the differential brain damage acquired in the head This reduced level of general alertness results degraded/attentuated stimuli entering the attention process, making it more difficult to rapidly discriminate the ST stimuli from the SI information.

In addition, altered arousal stemming from the traumatic fronto-RAS damage results in faulty CE/SAS functioning. As pointed out in section 6.5.2, the SAS concept is linked to the initiation of voluntary behaviour, particularly in those situations where routine selection of operations is unable to cope (for example, environmental dangers, or novel stimulus input. Faulty SAS processing produces less effective selective attention processing of probe stimuli rather than other incoming stimuli. Some of the latter, therefore, 'leak through' into the memory scanning stage

(Comparator) where the probe is compared with the positive set item(s) in memory using the Scanner. The inclusion of non-probe stimuli in this process interferes with normal, efficient scanning so that this stage is prolonged (thereby yielding the abnormally-long median RTs in ES subjects noted in the main study of this thesis).

Beyond the slowing down of the comparison process, the inclusion of non-probe stimuli may also produce more errors: in the main study the frequency of errors for the normal control sample was .02, for most head-injured subjects was approximately .03, and for the ES sample was shown in figure 6.2, the Central Processor's limited capacity should be dedicated to operating scanner and checking the scan register for a match. The Central Processor's required arousal, mediated via the CE/SAS, is changed as a result of damage to the fronto-RAS The Central Processor's functioning is, therefore, system. impaired and it operates the Scanner less efficiently than normal: the Scanner checking the positive set items memory with probe information proceeds more slowly. Central Processor's operating effect upon the Scanner is possibly also compromised by some non-probe stimuli taking up some of its limited capacity. Presumably, the reduced central processing efficiency will also slow down its checking of the match register. The overall outcome of

FIGURE 6.2: MEMORY SCANNING IN HEAD INJURY



this memory scanning system damaged by traumatic brain injury is commensurate with the findings noted in this thesis for patients soon after injury.

The resultant effects are also consistent with everday life observations of severely head-injured patients soon after trauma, whose behaviours show increased distractibility and intrusion of irrelevant stimuli into conscious processing (sometimes labelled 'frontal lobe' behaviour). General arousal mechanisms often seem disturbed in these patients, and frequently reports are obtained from the patient and their relatives of very long sleep periods and the difficulty of going through a day without feeling mentally exhausted and/or having to take a 'nap'.

It can be hypothesised that the cumultive effect upon ES subjects' memory scanning performance of the above attentional and processing deficits is slower than normal information processing and a higher slope weight than normal with increasing amounts of information to process. With the recovery over time of brain arousal mechanisms, and the resultant improvement in the functioning of selective attention, Central Processor, and the Central Executive it is hypothesised that close to 'full strength' (probe stimuli) enter the more efficient selective ST attention process, which filters out more of the SI.

leading to less interference with ST and the availablity of more of the central processor's capacity.

importance of the selective attention process reflected in the present pilot study finding that the addition of different levels of irrelevant information interacts significantly with head injury severity to determine RT. It is clear, too, from the significant interaction observed between irrelevant information and months post-injury that the selective attention process recovers over time. The increased variability of RT (ie, noted in both the pilot and main studies for more severely head-injured subjects may have arisen from selective attention failure (an inability to sustain the selective function consistently over time), or from fluctuations in general arousal level (varying 'ready to respond' ability).

The suggested model includes a number of attentional components, and the introduction of a controlling process seems necessary: a strategic level is required, to offer a supervisory or conscious control function. Baddeley (1986) proposed a Central Executive, and Norman and Shallice suggested a Supervisory Attention System' (SAS). For the latter (see Baddeley, 1986) it has been hypothesised that the frontal lobes are its organic substrate, a suggestion

which is highly relevant to the current thesis, given that frontal areas usually sustain the maximal damage in head Relevant, too, is the observation in this thesis of significant recovery in memory scanning performance over time - not just in the first few months post-injury, but beyond 1-2 years. This extended recovery period difficult to account for on the basis of specific neuronal recovery: postulating a 'plasticity' mechanism appears highly dubious (given that most subjects were in their late teenage years, or older), and a 'diaschisis' explanation is unsatifactory as this refers to the temporary disturbance in functioning of areas associated with the site of primary damage (eg. oedema, intracranial pressure changes, vascular changes). Tissue affected by diaschisis has not sustained significant direct damage and recovers function after the 'shock' effects of the cerebral insult have dissipated: the time course following head injury described in this thesis is too long to be attributable to this cause.

It may be that with the inbuilt redundancy in brain tissue increased sensitivity (in terms of neurotransmitter sensitivity and increase in neuronal receptors) may develop in the spared tissue. Whilst this may be one possible explanation for the observation of cognitive recovery beyond 12 months after head injury, it is also valid to view this extended recovery period in neuropsychological

process terms: for example, if the CE/SAS exists specifically dependant upon the integrity of frontal lobe then severe head injury will compromise its functioning, operation. Ιt can bе argued that the strategic. supervisory role which the CE/SAS offers will also be slow is this role a 'higher order' one it to cope with a rich input of perceptual requires information and a large variety of ongoing cognitive operations (in fact, all of those in which there is a component of conscious processing).

Ιn extremely severe head-injured patient soon trauma there will be severe damage to any integrating or controlling cognitive processes such as the CE/SAS. observation following head injury supports this coordinated cognitive activity: patients disorientated, they lack the ability to maintain a coherent and sequential memory system (during PTA), and sociallyunacceptable behaviours such as swearing and overt sexual activity are not inhibited. Extremely severely injured patients at this stage find it impossible to focus and sustain concentration upon one cognitive task for any length of time, and their attention is often distracted by irrelevant stimuli. As recovery proceeds, the brains of patients gradually re-establish continuous become orientated, and an overall supervisory, conscious

control over cognitive activity and general behaviour begins to be re-asserted. In the current research the main study finding of initially a very disorganised memory scanning performance, followed by ES subjects showing higher weights and intercepts as recovery proceeds, is consistent with the slow re-establishment of the CE/SAS function.

The recovery process in relation to memory scanning performance may be viewed as a gradual improvement general arousal level after head injury, as frontal and fronto-RAS connections are re-established, allowing better 'ready to respond' status. Some reduction in RT and Associated with this recovery in SD should occur. arousal condition is the brain's regaining of conscious control over the processing of information: the CE/SAS can direct the selective attention stage so that the incoming probe stimulus is favoured, totally irrelevant stimuli are excluded from further processing, and other stimulation which can be processed automatically is not allowed to take more than a minimal amount of the available limited capacity in the central processor. This recovery stage should be associated with reduced interference effects from irrelevant information and a consequent improvement in RT and its variability.

The more severely head-injured a subject, the longer will this phase of recovery take, and in the present research even at 6- and 12-months post-trauma ES subjects were showing steeper information load slopes. This suggested that difficulties were still being experienced by these subjects in terms of impaired selective attention, thereby interfering with the item scanning process in memory. By 24 months these differential difficulties for ES subjects had resolved to the point where no significant differences were noted when comparing their slope weights with those produced by other patients. However, as figure 5.4d suggests, the ES subjects were still processing information more slowly at that follow-up.

The above depicted model is undoubtedly too simplistic and inadequate in its present form, but it does allow some integration of the available literature with findings from the current study. Any further development of the model, testing of its usefulness, would require additional research. Particularly appropriate would be concurrent and memory scanning measurement physiological in headinjured subjects, to investigate arousal-performance relationships.

As discussed earlier in this thesis (section 6.5.2), it may be helpful to include examination of the Contingency Wave (or CNV) via EEG measurement, to assess 'readiness to respond'. Also, possible physiological facilitation effects upon arousal could be investigated using preparatory auditory or visual stimulation, and measurement of evoked heart rate may help to explore arousal hypotheses in understanding the cognitive functioning of head-injured patients in the Sternberg paradigm.

It would be interesting to manipulate probe stimulus discriminability and the addition of irrelevant information to the probe. Selective attention components might be profitably examined by employing, for example, distractor stimuli and then checking for habituation of response.

Although the field is relatively new, the efects medication aimed at cognitive enhancement could be explored using the memory scanning model. Rabbitt (1988) review of cognitive models predicted a close relationship between information processing rate and other aspects of memory, including capacity. He argued for the development of wider models which could include span, recognition memory, free recall, and information processing speed. Quite rightly, Rabbitt pointed out that the latter is not a 'master variable' determining all other cognitive

functions. The present thesis offers a start to this development by exploring the associations between RT indices of information processing and a number of other memory variables including span, learning rate, interference, and both recall and recognition measures.

6.6 SUMMARY

This chapter offered discussion of the results of the main study, including the findings that the Sternberg memory scanning paradigm was sensitive both to the severity of head injury and to the process of recovery. The pattern of findings indicated clear support Sternberg for the of serial, exhaustive memory scanning. operation Additional clinical findings included the observation that having to undergo neurosurgery was not associated with a poorer RT outcome, though there were some suggestions that additional right hemisphere damage was a sign of a poor prognosis for information processing recovery. No evidence adverse effects from anticonvulsant medication were noted, although there was only a limited opportunity to in the current research, and explore this aspect evidence to support the idea that female subjects would produce slower RTs.

The main study also demonstrated good associations between RT indices and other memory test variables, and the significance of these was discussed.

Finally, the main findings were discussed in relation to the existing literature. Possible attentional mechanism disturbances to account for the poorer information processing noted following head injury were considered, and elements of a model to describe memory scanning in headinjured subjects were put forward. Some suggestions for future research were offered.

CHAPTER 7

SUMMARY & CONCLUSIONS

SUMMARY & CONCLUSIONS

Chapter 1 of this thesis argued that although experimental psychology approaches have much to offer to the development of Clinical Neuropsychology, their full contribution has not yet been realised due to the origins of Clinical Neuropsychology. Research has often been driven by Medical and Surgical Neurology, where the interest has been centred on the quantification and profiling of cognitive deficits associated with specific lesions and diagnoses. Its development, too, has been much influenced the by psychometric tradition and its attendant test approach, rather than the stronger and richer theory-based literature. Where Clinical Neuropsychology experimental studies have drawn upon this literature, significant advances in our understanding of cognitive dysfunction have emerged.

The primary purpose of this thesis was to examine one aspect of cognitive dysfunction following head injury, and its recovery, by investigating memory scanning performance using Sternberg's paradigm.

Chapter 2 offered a review of head injury variables relevant to the thesis, including demographic factors, the mechanisms by which primary and secondary brain damage are

acquired in head injury, and the methods by which severity of head injury may be judged. With regard to the latter, length of PTA is a useful index. Chapter 2 also considered outcome following head injury, both physical and psychological. Whilst psychosocial aspects were more briefly outlined, cognitive abilities primarily affected by head injury and the focus of this thesis — namely memory and attention, were reviewed in some detail.

The literature in relation to memory scanning chapter 3. The chapter included considered in introductory section on the use of RT studies in the examination of information processing. A number variables were reviewed in terms of their relationships with RT, including age, CNS fatigue, and general arousal. The effects of brain damage upon RT performance were also discussed in chapter 3, including slower responses and higher RT variation. The available literature suggests that severity of brain damage correlates with RT disturbance. The most imprtant study in relation to head injury, RT, and attention was that carried out by Van Zomeren (1981). The study is rare in that it used repeated RT testing with a head-injured sample, extending up to 2 years post-injury.

remainder of chapter 3 was concerned with consideration of Sternberg's (1969) paradigm, which formed the basis of the thesis' main study. The memory scanning procedure was decribed in detail, as was his contention and exhaustive high-speed scanning of contents of memory occurs. Sternberg has concluded that in item recognition memory scanning there is a linear relationship between RT and positive set size, the positive zero intercept approximates 400 msec, and the positive and negative trial plots are parallel. Chapter 3 devoted considerable space to an outline of the evidence supporting of the conflicting views that memory scanning is exhaustive, or is self-terminating. Sternberg's model to describe exhaustive scanning was presented, and a brief review of the general literature undertaken. The latter included considerable support for Sternber's view, although the 'special' circumstances under which self-terminating scanning might occur were also mentioned.

The review of clinically-relevant studies provided in chapter 3 suggested that significant age effects operate on memory scanning speed. Chronic schizophrenic patients have been shown to scan more slowly than acute patients or normals, and aphasic patients may also show slow memory scanning, higher intercepts, and steeper RT slopes. People with a mental handicap perform similarly, as do patients

with Parkinson's disease or multiple sclerosis. The chapter concluded that Sternberg's paradigm offered a potentially-sensitive method for detecting changes in cognitive functioning following acquired brain damage.

The pilot study for the thesis, designed to check that head—injured subjects could cope with tasks employing a high information load, was described in chapter 4. The experiment involved small samples of mild and severe head—injured subjects who were tested at 1, 3, and 6 months—post—trauma. Also, the information processing task employed included a load variable and a level of irrelevant information variable, yielding a 3-factor design. The dependant measure was RT. Chapter 4 described the experimental procedure, and results were examined in terms of median RT and standard deviation of RT.

The pilot study confirmed the feasibility of using an information processing approach to study cognitive functioning after head injury, the results also indicating that severe head injury subjects showed slowed processing ability. The addition of irrelevant information was found to differentially-penalise the RT performance of severe subjects, and these subjects also provided evidence of greater RT variability. The pilot study results also suggested that recovery in information processing ability

can be observed during the first 6 months post-injury, this recovery being predictable.

Chapter 5 presented the main study, centred upon the use of Sternberg's memory scanning paradigm. The study's general the description of an aspect of cognitive aim was dysfunction stemming from head injury, and the charting of its progress over the subsequent 2-3 year period. Another aim was to consider the findings from examining scanning with those obtained from a number of other memory tests already used in clinical practise. A subjective measure of memory performance was also included in Memory scanning performance was examined in terms study. of its relationships to clinical variables, such as PTA, to estimated premorbid IQ. and to a limited number demographic variables. The experiment included groups of mild/moderate, severe, very severe, and extremely severe head-injured patients in the main sample, a normal control sample, and a 'back-up' patient sample (for the 2-3 years post-trauma interval). The hypotheses generated included prediction that memory scanning performance would be sensitive to cognitive recovery at least 12 months after injury, and that the level of impairment of performance would be related to initial severity of trauma. also predicted that ES subjects would not show a complete recovery in memory scanning ability.

Median RT and SD of RT were used as indices of memory scanning performance, and a number of hypotheses relating to information processing slope weights and linearity, according to head injury severity, were also advanced. Parallel positive and negative RT slopes were expected, and it was predicted that practice effects would not be observed.

Chapter 5 described the experimental procedure in detail, and also provided the results. Major analyses pointed to significant differences in memory scanning performance according to trauma severity, and positive set size, and also to the interaction of time post-injury with other variables, including severity of head injury. Time postinjury also yielded a significant main effect, thereby confirming the predicted recovery in memory scanning ability. Subsequent group t-test analyses indicated that the relatively limited evidence of recovery in the main patient sample beyond 6 months post-trauma was based upon deficits in ES (principally) and S subjects' memory scanning. Single Case analyses, however, suggested that recovery continued longer than was suggested by group ttests (extending beyond 2 years in a number of cases), and binomial test results also provided some support for recovery between 12-24 months post-injury.

Comparisons of different severity groups at each follow-up generally pointed to the significantly poorer memory scanning of ES subjects, and correlations of PTA with RT performance at each follow-up showed strong relationships at 3 and 6 months after injury with a gradual weakening at and 24 months. As hypothesised, the memory scanning performances of the control group were significantly better than those of the patient samples.

The prediction of exhaustive memory scanning was confirmed, that plots of positive and negative trial RTs were approximately parallel, with negative RTs being generally Production of regression equations confirmed slower. parallel plots, and linearity was generally very good. ES subjects showed the highest intercepts at each follow-up Although it was predicted that and steeper RT slopes. error trials would yield faster RTs, this hypothesis was not supported. There was evidence that errors were higher in patient groups (particularly ES) than the control group, though these did not show a tendency to occur on fast RT trials, but rather on trials subsequent to fast trials. This finding was discussed in relation to an explanation that, after attentional 'highs', errors were more likely to occur subsequently with the waning of attention.

Analyses of RT variability in chapter 5 produced similar findings to those noted for median RT, though some results were less striking. There was a general lack of evidence for recovery over time, except in the S and ES groups. High correlations of SD with median RT were noted for all 3 Also considered in chapter 5 samples. were the relationships of other variables to memory scanning. Overall, having to undergo neurosurgery did not adversely affect memory scanning RT, though some evidence was noted that additional/partial lateralisation of brain damage to hemisphere associated with the right was poorer performance. Median RT results soon after injury were not found to have any predictive value for time to return to work, and the taking of anti-convulsant medication was not associated with poorer memory scanning (although this aspect was difficult to examine, given small numbers).

Although head injury in adults tends to be restricted to a fairly narrow age band, significant correlations were noted with median RT and SD. Two unexpected observations were the occasional (and striking at the 12 months follow-up) superiority of RT performance in female subjects, and the lack of consistent correlations between estimated premorbid IQ and RT indices.

Other, clinical memory, measures of cognitive functioning injury generally showed somewhat head lower sensitivity than memory scanning to severity of head injury and to recovery over time, although ES subjects produced significantly poorer results. The clinical memory tests (particularly the Rey AVLT and Wechsler Memory scale) also often showed significant correlations with PTA, and with median RT. Although subjective memory (SMQ) scores at 24 months after injury generally showed only nonsignificant correlations with memory scanning results at the same follow-up, significant correlations of the 24month SMQ data with the RT results at 6 months post-trauma were observed.

Chapter 6 of this thesis provided detailed discussion and interpretation of all of the findings described in chapter 5. Chapter 6 also offered a model for the impaired memory scanning performance found following head injury, drawing upon concepts of general arousal, selective attention, central processing, a Central Executive/SAS, and ideas put forward by Sternberg. Finally, chapter 6 advanced some for future suggestions research, including conjoint measurement of memory scanning, neurophysiological physiological variables, possible beneficial effects of medication upon memory scanning, and additional research on the effects of introducing irrelevant information. The

utility of the Sternberg memory scanning paradigm will need to be tested out in future research using 'real world' outcome variables such as job functioning.

APPENDIX A: PILOT STUDY DATA

APPENDIX A1: BACKGROUND AND CLINICAL DATA

TABLE A1.1: BACKGROUND INFORMATION, PILOT STUDY

<u>Subj.</u>	<u>Age</u>	<u>Sex</u>	<u>Occupation</u>	<u>Cause</u>	<u>Severity</u>
1	17	М	Plumber	RTA	Mild
2	19	F	Hairdresser	RTA	Mild
3	19	F	Clerk	RTA	Mild
4	23	M	Teacher	RTA	Mild
5	19	F	Clerk	Fall	Mild
6	19	M	Student	Fall	Mild
7	18	M	Apprentice	RTA	Mild
8	19	F	Shop Assist.	RTA	Severe
9	50	M	Driver	RTA	Severe
10	28	M	Brick Layer	RTA	Severe
11	21	M	Draughtsman	RTA	Severe
12	54	M	Machine Op.	Industrial	Severe

TABLE A1.2 CLINICAL DATA, PILOT STUDY

Subj.	Time U/C	PTA	Skull#	<u>Haematoma</u>	WAIS
1	<=24hrs	? 0	?Ant.	No	12
2	Minutes	1hr	No	No	8
3	Minutes	0	No	No	11
4	10'-15'	12hrs	No	Sub: RT	16
5	0	0	No	No	9
6	Minutes	Minutes	No	No	12
7	0	1.5hrs	No	No	9
8	3 Weeks	3+ Wks	No	No	5
9	4 Days	5 Days	No	Sub: R	11
10	Hours	4 Days	RP	SAH: RP	_
11	4 Days	14 days	No	?SAH	12
12	6 Days	3+ Wks	FDep	Sub: F	-
-n·			_		

Time U/C = Time Unconscious Sub = Subdural P = Parietal SAH = Sub-arachnoid Haemorrhage

APPENDIX A2: SUBJECTS' MEAN, SD, MEDIAN RTs

SUBJECT 1: REACTION TIMES (msec)

ONE	MONTH	FOI I	OW-IIP

		ONE BIT			-	rwo B	ITS	TH	THREE BITS		
		<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	4	_8	
Mean	:	741	1094	1094	790	1235	1413		$13\overline{1}9$		
S.D.	:	75	189	220	128	354	288	44	435	1574	
Media	n:	742	1118	1100	781	1110	1427	754	1303	1525	

THREE MONTH FOLLOW-UP

		(ONE B	IT	-	IWO B	ITS	TI	THREE BITS			
		<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>		
Mean	:	768	1026	1172	833	1422	1444	871	1336	1920		
S.D.	:	43	179	297	74	443	683	70	396	1277		
Median	n :	763	940	1184	841	1355	1334	873	1274	1406		

SIX MONTH FOLLOW-UP

		ONE BIT			\mathbf{T}	TWO BITS			THREE BITS			
		<u>0</u>	<u>4</u>	<u>8</u>	<u>o</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>		
Mean	:	765	1007	1036	831	957	1359	811	1356	1590		
S.D.	:	79	160	227	129	163	194	59	451	738		
Mediar	า:	749	956	984	783	930	1339	759	1222	1382		

SUBJECT 2: REACTION TIMES (msec)

ONE MONTH FOLLOW-UP

			_	110		,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,	01		
	• (ONE BIT			rwo bi	ITS	THREE BITS		
	<u>o</u>	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	4	8
Mean :	763	1006	1156	795	993	1295	865	1399	2154
S.D. :	104	155	230	47	139	371	96	459	586
Median:	744	993	1212	801	1021	1143	836	1304	2031

THREE MONTH FOLLOW-UP

		ONE BIT			-	rwo Bi	TS	Ti	THREE BITS		
		<u>0</u>	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	
Mean	:	722	892	1024	764	1063	1050	802	1336	2668	
S.D.	:	48	86	158	39	237	168	31	735	2565	
Media	n:	733	890	1049	755	962	1062	804	1161	1664	

SIX MONTH FOLLOW-UP

					T	WO B	ITS	THREE BITS			
		<u>0</u>	<u>4</u>	<u>8</u>	<u>o</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>	
Mean	:	704	871	861	732	859	1202	$7\overline{7}4$	1258	1954	
S.D.	:	38	218	120	60	109	343	56	714	1374	
Median	า :	702	758	842	713	829	1057	767	1003	1232	

SUBJECT 3: REACTION TIMES (msec)

ONE	MONTH	FOLLOW-UP	

		ONE BIT			1	rwo B	TS	Ti	THREE BITS		
		<u>0</u>	4	<u>8</u>	<u>O</u> .	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>	
Mean	:	979	916	1095	888	1615	3317	815	1092	1735	
S.D.	:	92	92	168	133	393	1418	38	265	1154	
Median	า:	805	932	1063	853	1572	2746	800	972	1429	

THREE MONTH FOLLOW-UP

		ONE BIT			•	TWO BITS			THREE BITS		
		<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>	
Mean	:	806	955	1023	863	1010	1193	838	1450	2223	
S.D.	:	51	92	190	68	142	290	51	362	1123	
Median	1 :	810	939	1004	867	950	1114	857	1415	1773	

SIX MONTH FOLLOW-UP

		0	NE BI	T	TWO BITS			THREE BITS			
		<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	
Mean	:	739	857	932	793	921	1178	875	1169	1646	
S.D.	;	39	84	146	81	107	413	291	270	706	
Media	n:	733	870	877	785	937	1018	780	1111	1385	

SUBJECT 4: REACTION TIMES (msec)

ONE MONTH FOLLOW-UP

		0	NE B	IT	-	TWO BITS			THREE BITS		
		<u>0</u>	<u>4</u>	<u>8</u>	<u>o</u>	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>	
Mean	:	711	892	1011	725	1041	1717	804	1297	2300	
S.D.	:	48	212	156	70	216	831	35	327	1405	
Media	n:	719	802	967	759	999	1700	805	1346	1898	

THREE MONTH FOLLOW-UP

		0.	NE B	ſΤ	7	TWO B	(TS	Ti	REE I	BITS
		<u>0</u>	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>
Mean	:	769	926	1131	837	1104	1499	766	1466	2429
S.D.	:	48	102	164	91	185	787	60	510	1676
Media	: ח	765	902	1104	852	1097	1194	769	1361	1642

SIX MONTH FOLLOW-UP ONE BIT TWO BITS THREE BITS

					DIV LI	NATIT I	. OLLLON	OI.			
		0	NE BI	T	TWO BITS			Ti	THREE BITS		
		<u>0</u>	<u>4</u>					<u>0</u>			
Mean	:	677	823	929	749	1074	1236	813	1338	2365	
S.D.	:	62	110	137	51	233	351	49	363	1252	
Median	n:	675	802	930	745	1032	1118	794	1314	2184	

SUBJECT 5: REACTION TIMES (msec)

				•	•				
			0	NE MON	TH F	OLLOW-	-UP		
	0	NE BIT	r	7	WO B	ITS	T	HREE I	BITS
	<u>0</u>	4	<u>8</u>	Λ	Λ	Q	0	1	۵
Mean :	658	776	1003	680	1020	1473	812	$13\overline{9}4$	$17\overline{0}4$
S.D. :	60	94	275	50	213	572	38	728	374
Median:	646	775	919	669	975	1391	820	1106	1674
				EE MON					
		NE BI			TWO B			HREE 1	
	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>
	657		958	659	944	1155	692 38 695	1381	1621
S.D. : Median:	38	216	204	40	160	283	38	855	810
Median:	654	811	905	650	885	1148	695	1122	1236
				OTV 166					
	•	11E D.T.		SIX MO					
		NE BI			WO B	rrs	TT	HREE I	BITS
M	0	4	8	0	<u>4</u>	445	<u>U</u>	4	1520
Mean :	981	919	1101	686	890	1147	746	1111	1570
S.D. : Median:	42	337	3/3	44 680	1.00	20/	78	293	239
median:	0/8	709 .	1009	080	833	1118	TI <u>0</u> 746 78 766	1041	1494
		SUB.	TECT	6: RE	encert	אוד עכ	MES		
		БОБС	, 10.	O. 112	incli	JIV 111	-1110		
			0	NE MON	TH F	OLLOW-	-UP		
	0	NE BIT			WO B			HREE I	BITS
	<u>0</u>	4	<u>8</u>	<u>0</u>	4	8		4	8
Mean :	686	8 8 3	983	6 9 3	$10\overline{5}5$	$11\overline{6}7$	844	$13\overline{9}2$	2008
S.D. :	51	133		61	241		97	248	806
Median:		856	944	682	977	1104		1407	1889
			THR	EE MON	ITH I	FOLLO	V -UP		
	0	NE BIT	Γ	7	TH E			HREE I	BITS
	0 <u>0</u>	NE BIT	Γ <u>8</u>	<u>0</u>	TWO B:	ITS <u>8</u>	T1 <u>0</u>	4	3ITS <u>8</u>
Mean :	<u>0</u> 625	<u>4</u> 838	Γ <u>8</u> 935	0 0 080	TWO B:	ITS <u>8</u>	TI <u>0</u> 679	4	
S.D. :	0 625 31	<u>4</u> 838 182	Γ <u>8</u> 935 145	0 680 64	TWO B 4 943 162	ITS <u>8</u> 1204 206	T1 <u>0</u> 679 37	<u>4</u> 941 208	<u>8</u> 1778 517
	0 625 31	<u>4</u> 838 182	Γ <u>8</u> 935 145	0 680 64	TWO B 4 943 162	ITS <u>8</u> 1204 206	T1 <u>0</u> 679 37	<u>4</u> 941 208	<u>8</u> 1778 517
S.D. :	0 625 31	<u>4</u> 838 182	935 145 911	0 680 64 676	WO B: 943 162 914	175 <u>8</u> 1204 206 1204	TI <u>0</u> 679 37 683	<u>4</u> 941 208	<u>8</u> 1778 517
S.D. :	0 625 31 618	4 838 182 756	0 8 935 145 911	0 680 64 676 SIX MC	WO B: 4 943 162 914 ONTH I	TS 1204 206 1204 FOLLOW	™ <u>0</u> 679 37 683 √ –UP	4 941 208 901	8 1778 517 1731
S.D. :	0 625 31 618	4 838 182 756	7 <u>8</u> 935 145 911	0 680 64 676 SIX MC	WO B: 943 162 914 ONTH I	ITS	TI <u>0</u> 679 37 683 V -UP	4 941 208 901 HREE I	8 1778 517 1731
S.D. : Median:	0 625 31 618	4 838 182 756 NE BIT	935 145 911	0 680 64 676 SIX MO	WO B: 943 162 914 ONTH I TWO B: 4	TTS	TI <u>0</u> 679 37 683 ∛ -UP TI <u>0</u>	4 941 208 901 HREE I	8 1778 517 1731 3ITS 8
S.D. : Median: Mean :	0 625 31 618	4 838 182 756 NE BIT 4 755	935 145 911 911	0 680 64 676 SIX MC 0 631	WO B: 943 162 914 ONTH I WO B: 4 886	$\begin{array}{c} 1TS \\ & \underline{8} \\ 1204 \\ 206 \\ 1204 \\ \hline FOLLOWITS \\ & \underline{8} \\ 1034 \\ \end{array}$	TI <u>0</u> 679 37 683 N-UP TI <u>0</u> 649	4 941 208 901 HREE I 4 927	8 1778 517 1731 3ITS 8 1860
S.D. : Median:	0 625 31 618 0 0 594 24	4 838 182 756 NE BIT 4 755 170	935 145 911 911	0 680 64 676 SIX MO 0 631 38	WO B: 4 943 162 914 ONTH I WO B: 4 886 260	TTS	TI 0 679 37 683 V-UP TI 0 649 51	4 941 208 901 HREE I 4 927 257	8 1778 517 1731 3ITS 8

SUBJECT 7: REACTION TIMES (msec)

		0	NE MON	TH FO	DLLOW-	UP		
0	NE BI	T	7	WO B	[TS	T	HREE I	BITS
)	4	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>
3	1240	1374	844	1171	1765	896	1398	2294

 Mean
 :
 843
 1240
 1374
 844
 1171
 1765
 896
 1398
 2294

 S.D.
 :
 125
 201
 440
 70
 272
 690
 132
 318
 919

 Median:
 847
 1220
 1360
 840
 1180
 1542
 854
 1299
 2118

0

THREE MONTH FOLLOW-UP

		(ONE BI	T	TWO BITS			THREE BITS		
		<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u> "	<u>8</u>
Mean	:	762	$10\overline{10}$	998	823	1282	1285	909	1792	1786
S.D.	:	85	204	210	116	311	316	122	444	617
Media	n:	786	987	955	805	1322	1175	870	1262	1614

SIX MONTH FOLLOW-UP

		(ONE BI	T	7	WO B	ľTS	T	IREE I	BITS
		0	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>
Mean	:	824	1073	$11\overline{5}5$	835	1174	$13\overline{27}$	860	1689	2325
S.D.	:	125	99	268	103	307	235	106	1473	1360
Mediar	1 :	804	1064	1055	797	1125	1271	820	1310	1704

SUBJECT 8: REACTION TIMES (msec)

ONE MONTH FOLLOW-UP

			Oi	ATT TATOL	4 1 1 1 7 7		O1		
	(ONE B	ΙT	7	TWO BITS		THREE BITS		
	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>
Mean :	878	1224	1327	905	1417	1872	1559	2638	3112
S.D. :	56	313	361	65	359	590	470	1338	915
Median:	891	1084	1237	902	1360	1841	1331	2071	2900

THREE MONTH FOLLOW-UP

		0	NE BI	T	7	rwo Bi	[TS	TF	REE I	3ITS
		<u>0</u>	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>
Mean	:	755	926	$11\overline{0}0$	790	1139	1421	900	1291	2434
S.D.	:	52	155	174	47	246	530	121	336	1948
Median	:	758	878	1031	783	1147	1294	879	1228	1765

SIX MONTH FOLLOW-UP

		0				TWO BITS			THREE BITS			
		<u>0</u>	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>o</u>	<u>4</u>	<u>8</u>		
Mean	:	671	765	904	794	996	1420	805	1095	1894		
S.D.	:	44	105	166	48	179	538	66	396	1364		
Media	n:	687	744	880	806	961	1232	807	953	1525		

SUBJECT 9: REACTION TIMES (msec)

	ONE	HTNOM	FOI	J.OW-	JIP.
--	-----	-------	-----	-------	------

		0	NE BI	T	T	WO BI	TH	THREE		
		<u>0</u>	4	8	<u>0</u>	4	8	0	4	8
Mean	:						_	_	_	
S.D.	:		*			*			*	
Media	n:									

THREE MONTH FOLLOW-UP

		C	NE B1	[T]	7	TWO B	[TS	THREE BITS			
		<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	
Mean	:	787	1653	1702	948	2080	3395	988	2304	3378	
S.D.	:	92	700	622	63	746	1439	91	1027	1697	
Median	٦.	768	1492	1550	954	1992	3225	992	1921	2885	

SIX MONTH FOLLOW-UP

		(ONE BI	T		rwo bi	ITS	T	THREE BITS			
		<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>		
Mean	:	748	1230	1933	824	1722	2226	792	1615	2703		
S.D.	:	100	355	1054	99	632	1083	76	339	1243		
Median	n:	700	1127	1739	785	1699	2015	773	1678	2640		

^{* -} subject still in PTA

SUBJECT 10: REACTION TIMES (msec)

ONE MONTH FOLLOW-UP

		(ONE B	ΙΤ	7	rwo bi	ITS	TI	THREE BITS			
		<u>0</u>	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>		
Mean	:	1006	1306	1561	1018	$14\overline{2}1$	2298	1065	3264	3315		
S.D.	:	66	214	408	136	200	904	132	1702	2458		
Media	n:	987	1355	1410	1015	1447	2375	1019	3368	2561		

THREE MONTH FOLLOW-UP

			ONE B	T	7	rwo Bi	[TS	TH	THREE BITS			
		0	4	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	4	<u>8</u>		
Mean	:	819	$10\overline{1}2$	$11\overline{9}9$	894	$12\overline{7}4$	$13\overline{8}4$	914	2516	1622		
S.D.	:	22	134	224	73	203	402	80	2005	742		
Mediar	1 :	821	. 929	1220	881	1218	1282	891	1880	1323		

SIX MONTH FOLLOW-UP

		(ONE B	ΙT	7	WO B	ITS	THREE BITS			
		0	4	<u>8</u>	<u>0</u>	4	<u>8</u>	<u>0</u>	4	<u>8</u>	
Mean	:	800	1005	1089	870	$10\overline{3}1$	$19\overline{2}0$	854	1459	1530	
S.D.	:	36	142	173	72	108	893	35	429	489	
Median	า :	796	970	1048	867	1042	1555	855	1507	1484	

	<u>SUB.</u>	JECT 11:	REACT	CION T	<u> </u>	(msec)	<u>)</u>	
	· ·	0	NE MOI	יים עידינו	าt t:ดพ_	_I:ID		
	ONI	E BIT 4 8 333 1265 344 248 237 1197	NE MOI	TWO B	ITS	Tł	IREE F	BITS
	<u>0</u>	<u>4</u> 8	<u>0</u>	<u>4</u>	<u>8</u>	<u>0</u>	<u>4</u>	<u>8</u>
Mean :	874 13	333 1265	876	1289	2073	896	1807	2595
Median:	869 1	237 1197	853	1258	1671	893	1442	2170
	OM	THR	EE MOI	TWO B	FOLLOW	7−UP Ti	JDEE 1	פודכ
	0	4 8	0	1 WO D.	·8	0 11	1KEE 1	8
Mean :	7 <u>8</u> 0 10	$0\overline{1}0 \ 10\overline{9}2$	7 8 5	1393	1660	8 4 3	1393	$33\overline{3}7$
S.D. :	21	315 179	91	669	825	37	469	1151
Median:	779	E BIT 4 8 010 1092 315 179 906 1084	774	1103	1446	842	1211	3862
			SIX MO	ONTH I	FOLLOV	I-UP		
	ON	E BIT 4 8 119 1163 227 252 151 1179	7	rwo B	ITS	Tŀ	IREE F	BITS
	0	4 8	<u>0</u>	4_	. <u>8</u>	0	4	8
Mean :	739 13	119 1163	798	1307	1881	742	1431	2132
Median:	764 1	227 232 151 1179	763	1175	1728	730	1310	1808
			, 00	22,0	2.20	, 00	1010	2000
	a	7000 40	222.00			, ,		
	SUB	JECT 12:	REAC	TON	ITMES	(msec)	<u>1</u>	
		0	NE MOI	VTH FO	DLLOW-	-UP		
	ON	E BIT		rwo bi	ITS_	TH	HREE I	BITS
Mann	<u>0</u>	$\frac{4}{303}$	1760	2502	<u>8</u>	1626	$\frac{4}{2020}$	<u>8</u>
Mean :	205	203 3011 551 1 <i>2</i> 75	303	2303 558	1803	233	2151	1988
Median:	1318 2	E BIT <u>4</u> <u>8</u> 283 3011 551 1275 236 2776	1856	2364	3337	1535	3113	4119
	ONI	THR E BIT	EE MON	HTV ROWN	FOLLOW FTS	V−UP Ti	agge i	RTTS
	0	4 8	0	4	8	0	4	8
Mean :	1008 1	E BIT 4 8 445 1966	1078	1997	2963	$10\overline{7}5$	$24\overline{2}4$	2606
S.D. :	66 3	368 648 273 1787	82	1532	1951	91	1/32	TOID
Median:	1008 1	2/3 1/8/	1096	1493	2387	1088	1807	1968

80 392 810 984 1223 1651

TWO BITS THREE BITS 0 4 8 0 4 8

SIX MONTH FOLLOW-UP

<u>0 4 8</u>

984 1400 1939

ONE BIT

58 466 436 871 1187 1387

S.D. : Median:

APPENDIX A3:

PILOT STUDY: CORRELATION COEFFICIENTS

TABLE A3.1: CORRELATIONS OF AGE WITH MEDIAN RT AT 6-MONTH FOLLOW-UP

ONE BIT TWO BITS THREE BITS $\frac{0}{100} = \frac{4}{100} = \frac{8}{100} = \frac{4}{100} = \frac{8}{100} = \frac{1}{100} =$

TABLE A3.2 CORRELATIONS OF PTA WITH MEDIAN RT & SD (a) Median RT

6/12 FU: ONE BIT TWO BITS THREE BITS

R = : .40 .37 .33 .62 .31 .46 .57 .43 .47

(b) SD

6/12 FU: ONE BIT TWO BITS THREE BITS

R = :-0.06 0.37 0.13 0.03 0.33 0.53 0.18 0.20 0.51

* = p < .05 R = Correlation Coefficient ** = p < .01

APPENDIX B1: PARALLEL FORMS OF REY AVLT

${\tt Name:}$.						Date:		
Assessmen	it;							
					FORM	1		
LIST A	1	2	2	4	5	TIOT D	D D	D
PIDI W	_1	_2	_3	_4	<u>5</u>	LIST B	Rec.B	Rec.A
DRUM	_	_	_	_	_	DESK	_	_
CURTAIN	_	_	_		_	RANGER	 -	_
BELL	_	_	_	_	_	BIRD	_	
COFFEE	_	_	_	_	_	SHOE	_	_
SCHOOL	_				_	STOVE	_	
PARENT	_	_	_	_	_	MOUNTAIN		_
MOON	_	_	_	_	_	GLASSES		
GARDEN		_	_	_	_	TOWEL	_	_
HAT	_	_	_	_	_	CLOUD	_	_
FARMER		_	_	_	_	BOAT		_
NOSE	_	_	_	_	_	LAMB	_	_
TURKEY	_	_	_	_	_	GUN		-
COLOUR	_	_	_	_	_	PENCIL	-	_
HOUSE	_	-	. —	_	_	CHURCH	_	_
RIVER	_	-	_	_	_	FISH	-	_
moma r								
TOTAL						UTTO		.
RECOGNITI CREATURE	ON A	•			זת	HITS =		False + =
TEMPLE						VER (15)		PRISONER
						TY		COFFEE (4)
BELL (3) SUGAR						RENT (6)		SUMMER
COLOUR (1	2)					OCTOR		GARDEN (8)
NOSE (11)						RUM (1) HIP		HAT (9)
MILE						RMER (10)		PARTY
SCHOOL (5	: 1							FAMILY
HORSE	<i>)</i>)					OUSE (14) INE		CURTAIN(2)
WINE								CHIEF
MINE					MC	OON (7)		TURKEY(12)

Name:						Date:			
Assessmen	t: .								
					70 D*f				
					FORM	2			
LIST A	_1	_2	_3	_4	<u>5</u>	LIST B	Rec.B	Rec.A	
CONTRACT		_	-	_	_	TABLE	_	-	
VOICE	_	_	_	-	_	QUEEN	_	_	
WINTER	_	_	_	_	_	DOLLAR	_	_	
GRASS	_	_	-	_	_	FIRE	_	_	
DIAMOND	_		_	_	_	RAILWAY	-	_	
CAMP	_	_	_	_	_	TOWER	_	_	
BUTTER	_	-	_	_	_	LETTER	-	_	
CHARM	-	-	_	_	_	STREET	-	_	
VESSEL	-	-	-	_	_	STREAM	-	-	
POTATO	_	_	_	_	_	CATTLE	_	_	
MARKET	_	-	_	_	-	MOTHER	_	_	
BEAST	_	_	_	_	_	COAST	_		
CLOTHING	_	_	_	_	-	RECORD	_	_	
VILLAGE	-	-	-	-	_	SOIL	-	-	
HOME	_	-	_	-	_	PICTURE		_	
TOTAL	:								
RECOGNITION	ON A					HITS =		False + =	
CHURCH	on n	•				(15)	MET		
GENTLEMAN					SKIN	(10)		SS (4)	
WINTER (3)					CAMP	(6)	HAL		
AUTHOR					DEGRI			RM (8)	
CLOTHING (13)					CONTI			SSEL (9)	
MARKET (11)					MONT			TER	
	COUNTRY					TO (10)	SHO		
	DIAMOND (5)				VILL		VOICE (2)		
SHOES	- *				BATTI		BOTTLE		
CABIN						ER (7)	BEAST (12)		

				, .	,	Date:		
Assessmen	t: .							
						0		
					FORM	3		
LIST A	_1	_2	_3	_4	<u>_5</u>	LIST B	<u>Rec.B</u>	Rec.A
BOOK	_		_	-	_	BABY	_	_
FLOWER	_	_	_	_	_	MEAT	_	
TRAIN	-	_	_	_	_	ARTIST	_	
R⊍G	_	-	_	_	_	DOOR	_	-
MEADOW	-	-		_	_	LIBRARY	-	_
HARP	_	_	_	_	-	PRINCE	_	~
SALT	_	-	-	_	_	BROTHER	_	_
FINGER	_	_	_	-	_	STREET		_
APPLE	_	_	_	-	-	HOUSE	_	_
CHIMNEY	_		_	-	_	SOLDIER	_	_
BUTTON	-	_	_	-	· -	GOLD	_	_
KEY	_	-	-	-	_	GARDEN	_	_
DOG	-	-	-	-	_	JACKET	_	_
GLASS		_	_	_	_	CHAPEL	-	_
BATTLE	-	-	-	_	_	PERFUME	-	_
TOTAL	:							
	· · · ·							
RECOGNITION OF THE RECOGNITION O	ON A	•				HITS =	I	Talse + =
MURDER				•	BATTL	E (15)	ARM	
FOREST				(COIN		R⊍G	(4)
TRAIN (3)				1	HARP	(6)	CHRIS	STMAS
BRAIN				:	SWEET		FING	ER (8)
DOG (13)				1	BOOK	(1)	APPLI	E (9)
BUTTON (11	L)			(CHAIR		PALAC	ČE .
CHILD				(CHIMN	EY (10)	ANIMA	\L
MEADOW (5)			(GLASS	(14)	FLOWER (2)		
HOUR				1	HEAVE	N	NURSI	Ξ
LEMON				5	SALT	(7)	KEY	(12)

Name: Assessmer	 nt :					Date:		
					FOR	14		
LIST A	_1	_2	_3	_4	_5	LIST B	Rec.B	Rec.A
SHEPHERD		_	_	_	_	SADDLE	_	_
NEEDLE	•••	_	_	_	_	BODY	_	-
COLOUR		_	_	_	-	SPARROW		_
ARMY	_	_	-	_	_	ANCHOR	_	_
ORCHARD	-	_	-	-	_	WOODS	_	_
RABBIT	_	_	~	_	_	WITNESS	_	_
APPLE	-	_	_	_	_	PUPIL	_	_
WHISTLE	_	_	_	_	-	VALLEY	_	_
TUNNEL	-	_	-	_	_	CASTLE	_	_
CANOE		-	-	-	_	COLLAR	-	
FELLOW	-	_	-	_	_	FARM		_
DREAM		_	-	-		STAR	_	_
CURRANT	_	_	-	-	-	PRESIDENT	_	_
STORM	_	-	_		_	HOSPITAL	-	_
BOTTLE	_	-	_	_	-	FORM	_	_
TOTAL	.:							
RECOGNITI	ON A	 1 .			· · · · ·	HITS =		
KING				В	OTTLE	(15)	SUPPER	
CELL					ACKAC		ARMY (-
COLOUR (3	3)				ABBIT		SANDW	
SALARY	7				EATHE		WHISTI	
CURRANT (13)					RD (1)	TUNNEI	
FELLOW (1	1)			M	DMENT		MEMORY	
CHANNEL				S	FORM	(14)	DISEAS	=
ORCHARD (5)				ANOE		NEEDLE	
PUZZLE				TATIO		LAW		
COWARD				AF	PPLE	(7)	DREAM	(12)

APPENDIX B2: EXAMPLE STERNBERG SOFTWARE

EXAMPLE OF STERNBERG DATAFILE PRINTOUT

DATA FROM PATIENT ON LIST ALL FROM 1 TO 20 AND QUESTIONS & DATA FILES AS BELOW:

ITEM	1	IS	POSITIVE	TIME	FROM	\$N.MILLER/3Y/2
ITEM	2	IS	CORRECT?		FROM	\$N.MILLER/3Y/2
ITEM	3	IS	STIMULUS		FROM	\$N.MILLER/3Y/2
ITEM	4	IS	NEGATIVE	TIME	FROM	\$N.MILLER/3Y/2
ITEM	5	IS	CORRECT-?).		\$N.MILLER/3Y/2
ITEM	6	IS	STIMULUS		FROM	\$N.MILLER/3Y/2

PAT.	& IT 1	EMS 2	 З	 4	5	6
1	410	1	 6	609	1	3
2	521	1	6	746	1	2
3	475	1	6	1088	1	7
4	636	1	1	956	1	8
5	741	1	1	679	1	4
		1	1	726	1	3
6	780					
7	798	1	1	1163	1	4
8	665	1	6	1122	1	8
9	576	1	1	950	1	5
10	679	1	1	798	1	5
11	543	1	6	849	1	2
12	550	1	6	870	1	4
13	542	1	1	663	1	8
14	433	1	6	785	1	9
15	491	1	1	595	1	2
16	440	1	6	609	1	8
17	599	1	6	614	1	5
18	364	1	1	739	1	2
19	368	1	1	886	1	0
20	555	1	6	606	1	2

STERNBERG COMPUTER PROGRAM: SET SIZE 2 - COMMENTS

Lines	<u>Operation</u>			
120-140	check for disk error in setting up datafile (see lines 790-860)			
150-300	introduce the Sternberg program			
310-460	seek input of subject's filename for disk storage, and check that filename does not already exist (to prevent overwriting)			
480-660	define the 'space' (ie, number of digits) required for each variable			
790-860	create datafile on disk			
910-950	seek choice of data set, from sets stored in program			
960-1040	dimension space into which data will be read			
1050-1120	collect chosen data set for presentation in			
230-1330	instruct subject on responding, and start testing			

1340-1510 present a positive or negative set stimulus, and time subject's response. If no response occurs within 10 seconds (line 1440) remind subject on how to respond (lines 1460-1510) 1520-1540 remind subject to release the response button if this has not occurred following a response 1550-1600 code each data item (stimulus) as a positive, or as a negative set member, according to chosen data set 1610-1680 record subject's response as correct or as an error 1690-1890 record if subject responded in advance of stimulus presentation 1900-2350 provide hard-copy of ID information, response times, and accuracy of response 2360-2450 store data on response times and accuracy of response on disk in subject's named datafile provide 5 parallel data sets, for repeat 2470-2720 testing

```
100 M$=CHR$(13)
110 GOTO150
120 IFDS(20THEN RETURN
130 IFDS=50THENRETURN
140 PRINTDS$:DCLOSE#3:PRINT"STOP!~ERROR"
150 PRINT"X"
160 FORI=1 TO 8:PRINT:NEXTI
170 PRINT"THIS IS A REACTION TIME PROGRAMME"
180 PRINT
190 PRINT"- (STERNBERG). IT STORES RESPONSE"
200 PRINT
210 PRINT"TIMES, ETC., ON DISC."
220 T=TI
230 IFTI-T(180 GOTO230
240 FOR I=1 TO 8:PRINT:NEXTI
250 PRINT"FIRST YOU NEED TO NAME A FILE"
260 PRINT
270 PRINT"WHERE THE PATIENT'S RESPONSES"
280 PRINT
290 PRINT"WILL BE STORED.
300 PRINT:PRINT
310 INPUT"WHAT IS THE FILE NAME ?":NF$
320 IFLEN(NF$)>14THENPRINT-"TOOLONG":GOTO310
330 PRINT"""
340 FORI=1TO8:PRINT:NEXTI
350 PRINT"CHECKING THAT FILE DOES NOT "
360 PRINT
370 PRINT"ALREADY EXIST....."
380 DOPEN#3.(NF$).D1
390 PRINT:PRINT:PRINT
400 IFDS()62THENPRINT"STOP!-THERE IS AN ERROR"
410 IFDS<>62THENPRINT"FILE EXIST":PRINT"ERROR".DS:DIRECTORYD1
420 IF DS()62 THEN DCLOSE#3:STOP
430 IFDS=62THENPRINT"OK-FILE NOT EXIST": DCLOSE#3
440 PRINT"PAXIENX'S FILENAME=",NF$
450 T=TI
460 IFTI-T(180 GOTO460
```

```
480 \text{ QS}\%(1) = 4
500 QS%(2)=1
520 QS%(3)=1
540 QS%(4)=4
560 QS%(5)=1
580 QS%(6)=1
620 QP%(1)=1
630 FOR I=2TO6
640 QP%(I) = QP%(I-1)+QS%(I-1)+1
650 NEXTI
660 RL=QP%(6)+QS%(6)+1
770 PRINT"X"
780 FORI=1TO8:PRINT:NEXTI
790 PRINT"CREATING DATA FILE"
800 DOPEN#3,(NF$),D1,L(RL)
810 GOSUB120
820 RECORD#3.(20)
830 GOSUB120
840 PRINT#3.CHR$(255)
850 GOSUB120
860 DCLOSE#3
870 REM:STERNBERG 1966 (SCIENCE)
880 GOSUB120
890 REM:+VE SET SIZE=2 (1,4/2,4/3,7/2,7/1,6)
900 REM: - 5 EXAMPLES
910 PRINT"SET1= 1.4/SET2= 2.4/SET3=3.7/SET4=2.7/SET5=1.6"
340 PRINT"WHICH DATA SET?"
950 INPUT N
960 DIM ER(250,2)
970 FOR I=1 TO 250
980 FOR J=1 TO 2
990 ER(I,J)=0
```

```
1000 MEXT J
1010 NEXT I:DIM TA$(250)
1020 DIM AN(100.2)
1030 FOR J=1 TO N
10:40: 0W=0
1050 FOR I=1 TO 250
1060 READ TA$(I)
1070 AF TA$(1)="-99" GOTO1100
1080 QW=QW+1
1090 NEXT I
1100 NEXT J
1140 READ Q$:IF Q$<>"END" THEN1110
1120 GOSUB2730
1130 PRINT"ENTER DATE:"
1140 INPUT ZOS
1150 PRINT"ENTER PT NAME:"
1160 INPUT ZP$
1170 PRINT"ENTER RUN NAME:"
1180 INPUT ZR$
1190 PRINT"HOW MANY TARGETS"
1200 INPUT X
1210 FOR I=1 TO X:PRINT"INPUT TARGET"
1220 INPUT AS(I) NEXT I
1230 PRINT"""
1240 PRINT"PRESS THE . RED . BUTTON":PRINT""
1250 PRINT"AS FAST AS YOU CAN WHEN YOU SEE-"
1260 PRINT"":FOR Z=1 TO X:PRINTA$(Z):NEXT Z
1270 PRINT"":PRINT"":PRINT"
1280 PRINT"":PRINT"FOR OTHER NUMBERS"
1290 PRINT" PRINT"PRESS THE XBLACKX BUTTON AS FAST AS YOU CAN"
1310 PRINT"TYPE Y WHEN READY"
1320 INPUT X$
1330 IF X$()"Y" THEN1300
1340 FOR I=1 TO 250
1350 IF TA$(I)="-99" THEN1900
1360 PRINT"": PRINT"X"
1370 FOR J=1 TO 10:PRINT"":NEXT J
1380 PRINT"
                                 "TA$(I)
1390 /CO=CO+1
1400 POKE 59459.255
1410 POKE 59471,255
1420 SYS(826)
1430 Q=(PEEK(1000)+256*PEEK(1001))/1000
1435 Q=Q*1.307
1440 IF QC10 THEN1520
1450 AN(CO,1)=-1:AN(CO,2)=-1
1460 PRINT"X":PRINT"PRESS THE RED BUTTON":PRINT""
1470 PRINT"WHEN YOU SEE ":FOR P=1TOX:PRINTA$(P)
1480 NEXT P:PRINT"":PRINT"FOR OTHER NUMBERS- PRESS BLACK"
```

```
1500 IF TI-T(600 THEN1500
1510 GOTO1710
1520 T=TI
1530 IF TI-T>600THENPRINT"PLEASE LET GO OF THE BUTTON"
1540 IF PEEK(59471)()255 THEN1530
1550 T=TI
1560 IF TI-T(60 THEN1560
1570 U=0
1580 FOR K=1 TO X
1590 IF TA$(I)=A$(K) THEN U=1
1600 NEXT K
1610 AN(CO.1)=Q
1620 IF U=1 THEN IF PEEK(1002)=254 THEN AN(CO.2)=1
1630 IF U=1 THEN IF PEEK(1002)=253 THEN AN(CO,2)=0
1640 IF U=0 THEN IF PEEK(1002)=253 THEN AN(CO.2)=1
1650 IF U=0 THEN IF PEEK(1002)=254 THEN AN(CO.2)=0
1668 PRINT"X":T=TI
1670 FOR Z=1 TO 10:PRINT"":NEXT Z
1680 IF TI-T(30 GOTO1680
1690 PRINT"
                         GET READY"
1700 IF TI-T(120 GOTO1700
1710 PRINT"X":T=[1
1720 IF TI-T<60 GOTO1720
1730 NEXT I
1740 T=TI
1750 IF PEEK(59471)(>254 THEN1780
1760 ER(I,1)=1
1770 ER(I+2)=INT(((TI-T)/60)*100)/100
1780 IF TI-TOR THEN1750
1790 IF ER(I.1)=0 THEN1880
1800 IF PP=
              1810 PRINT"YOU RESPONDED TO "TA$(I)
1820 PRINT"TARGETS ARE-"
1830 FOR Z=1 TO X
1840 PRINTA$(Z)
1850 NEXT Z
1360 T=TI
1870 IF TI-TC600 THEN:1870
1880 NEXT [
1890 GOTO1940
1900 PRINT"TYPE Y FOR RESULTS"
1910 INPUT X$
1920 IF X$()"Y" THEN1900
1930 DIMDA$(200.6)
1940 OPEN 3,4
1950 CMD 3
1960 PRINT"TARGET RESULTS FOR "ZP$
1970 PRINT" ££££££££££££££££
1980 PRINT"":PRINT"DATE:"ZO$
1990 PRINT"RUN NAME IS "ZR$
```

```
2000 PRINT""
2010 PRINT"TARGETS ARE"
2020 FOR W=1 TO X:PRINTA$(W):NEXT W:PRINT:PRINT
2025 FORI=1TOQW:AN(I,1)=AN(I,1)*1000:NEXTI
2030 FORI=1T05
2040 PRINT"NUMBER"I"="AN(I.1),AN(I.2),"SHOWN="TA$(I)
2050 NEXTI
2060 FOR I=6 TO QW
2070 A$=STR$(AN(I,1)):B$=STR$(AN(I,2))
2080 A$=MID$(A$,2,4)
2090 B$=MID$(B$,2,1)
2100 FOR N=1TOX
2110 IF TA$(I)=A$(N) GOTO2140
2120 NEXTN
2130 GOTO2180
2140 NP%=NP%+1
2150 DA$(NP%,1)=A$:DA$(NP%,2)=B$
2160-DA$(NP%,3)=TA$(I)
2170 GOTO2210
2180 NN%=NN%+1
2190 DA$(NN%,4)=A$:DA$(NN%,5)=B$
2200 DA$(NN%,5)=TA$(I)
2210 NEXTI
2220 PRINT"POSITIVE TIMES:"
2230 PRINT" ÉÉÉÉÉÉÉÉÉÉÉÉÉ
2240 PRINT
2250 FORI=1TO20
2260 PRINTI,DA$(I,1),DA$(I,2),DA$(I,3)
2270 NEXTI
2280 PRINT: PRINT
2290 PRINT"NEGATIVE TIMES:"
2300 PRINT" £z£££££££££££
2310 PRINT
2320 FORI=1TO20
2330 PRINTI, DA$(I,4), DA$(I,5), DA$(I,6)
2340 NEXTI
2350 PRINT#3:CLOSE 3
2360 DOPEN#3.(NF$),D1
2370 FOR RN=1TO20
2380 FOR I=1TO6
2390 RECORD#3,(RN),(QP%(I))
2400 GOSUB120
2410 PRINT#3.DA$(RN.I)
2420 GOSUB120
2430 NEXTI
2440 NEXTRN
2450 DCLOSE#3
2460 GOT03010
2470 REM:SET1 = 1,4
2480 DATA 6.0.1.5.4.6.7.4.9.5.4.3.1.2.4
2490 DATA 4,9,1,7,1,4,4,2,4,5,0,1,1,7,5
```

```
2500 DATA 4.6,2,9,4,1,2,6,9,4,1,1,3,1,1
2510 DATA -99
2520 REM:SET2= 2.4
2530 DATA 8,2,2,9,6,4,5,9,6,2,4,4,3,4,2
2540 DATA 8,1,9,1,4,4,3,0,2,4,7,0,9,4,2
2550 DATA 5,8,4,2,4,1,2,2,8,5,2,4,0,9,2
2560 DATA -99
2570 REM:SET3= 3,7
2580 DATA 2,0,3,7,9,2,9,7,7,3,0,6,7,5,3
2590 DATA 4,7,6,1,3,1,8,7,6,7,3,4,3,3,8
2600 DATA 3,1,5,7,1,3,3,9,7,7,3,6,7,4,1
2610 DATA -99
2620 REM:SET4= 2.7
2630 DATA 2,9,6,7,0,5,2,3,2,7,2,3,2,4,7
2640 DATA 0,5,2,2,5,7,5,7,3,6,7,7,8,9,2
2650 DATA 8,1,7,7,5,3,2,3,0,2,9,7,7,1,2
2660 DATA -99
2670 REM:SET5= 1,6
2680 DATA 2,0,6,7,1,3,6,6,6,2,7,8,1,4,1
2690 DATA 3,1,4,1,8,5,6,5,2,1,4,1,8,6,9
2700 DATA 6,2,1,8,6,1,5,2,6,0,6,1,1,2,6
2710 DATA -99
2720 DATA END
2730 DATA 169.1
2740 DATA 141,232,3
2750 DATA 169,0
2760 DATA 141,233,3
2770 DATA 120
2780 DATA 169,197
 2790 DATA 170
 2800 DATA 202
 2810 DATA 208,253
 2820 DATA 24
 2830 DATA 173,79,232
2840 DATA 201,254
 2850 DATA 240,21
 2860 DATA 201,253
 2870 DATA 240,17
 2880 DATA 238,232,3
 2890 DATA 208,10
 2900 DATA 238,233,3
 2910 DATA 173,233,3
 2920 DATA 233,40
 2930 DATA 240,2
 2940 DATA 208,224
 2950 DATA 88
 2960 DATA 141,234,3
 2970 DATA 96,999
 2980 L=826
 2990 READ X: IF X<256 THEN POKEL, X:L=L+1:GOTO2990
```

3000 RETURN 3010 END 3020 END

APPENDIX C1:

MAIN STUDY: MEDIAN, SD, & MEAN RT DATA, SAMPLE A

TABLE C1.1: SAMPLE A MEDIAN CORRECT RT (msec)

				month	Follow-up				
	POSI	TIVE S	ET		NEGATIVE SET				
Subj.	1	2	3	4	1	2	3	4	
1	744	649	650	702	646	638	674	691	
1 2 3	507	648	866	897	627	640	849	810	
3	452	454	539	507	476	432	570 ⁻	521	
4	452	571	501	626	456	681	548	671	
5	784	824	1038	1218	763	752	1133	1205	
6	457	624	624	655	513	732	751	657	
7	356	376	460	509	396	519	541	538	
8	573	523	733	744	572	554	736	792	
9	938	911	1147	1535	998	992	1169	2222	
10	610	726	730	770	668	917	751	930	
11	641	617	731	687	656	740	737	791	
12	361	409	569	687	378 510	421	576	707	
13	537	568	513	545	518	623	529	634	
14	717	804	1089	1058	692	979	991	972	
15 16	PTA	PTA 4436	PT A	PTA 3169	PT A 3067	PTA	PTA	PTA	
17	5396 DNA	DNA	3074 DNA	DNA	DNA	3121 DNA	2454 DNA	31.95 DNA	
18	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
19	504	809	750	692	667	799	895	1138	
20	NT	NT	NT	NT	NT	NT	NT	NT	
21	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
22	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
23	295	341	344	379	361	339	409	408	
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
25	445	566	688	757	539	646	669	860	
26	806	864	1129	1069	958	934	1104	1184	
27	NT	NT	NT	NT	NT	NT	ΝT	NT	
28	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
29	NT	NT	NT	NT	NT	NT	NT	NT	
30	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
31	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
32	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
33	NT	NT	NT	NT	NT	NT	NT	NT	
34	391	521	481	542	388	573	608	605	
35	NT	NT	NT	NT	NT	NT	NT	NT	
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
37	865	958	1018	1354	1019	1197	1216	1262	
38	DNA	DNA	DNA	DNA	DNA DNA	DNA	DNA DNA	DNA	
39 40	DNA PTA	DNA PTA	DNA PTA	DNA PTA	PTA	DNA PTA	PTA	DNA PTA	
41	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
42	275	298	348	347	315	292	418	414	
. ~	4/5	200	J-70	J-1/	010	476	* 1.0	414	

PTA= subject untestable, still in PTA NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up

M/E= data not available, micro, or experimenter error

TABLE C1.1: SAMPLE A MEDIAN CORRECT RT (msec), (cont)

			Three	-month	Follow-u				
	POSIT	IVE SE	T		NEGATIVE SET				
Subj.	-1	2	3	4	1	2	3	4	
1	482	517	629	575	561	648	632	662	
2 3	579	530	603	666	614	624	665	760	
3	362	390	462	495	398	408	482	510	
4	404	515	512	543	493	552	612	650	
5	976	1840	1533	1761	.936	1722	1418	1675	
6	534	510	623	586	608	612	662	722	
7	324	373	444	426	389	430	538	577	
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
9	474	572	685	786	521	636	741	794	
10	533	798	828	813 DNA	614	807	804	734	
11 12	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA	DNA	
13	DNA	DNA	DNA	DNA	DNA	DNA	DNA DNA	DNA DNA	
14	5.77	644	639	721	644	675	692	764	
15	1364	1347	1610	1535	1085	1631	1630	1526	
16	356	457	551	632	432	540	625	688	
17	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
18	460	788	620	682	445	698	699	734	
19	369	559	883	689	540	549	855	917	
20	543	534	505	589	578	513	600	643	
21	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
22	3159	1740	1829	2122	1676	1548	1541	1858	
23	272	316	293	313	305	344	370	371	
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
25	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
26	DNA	DNA	DNA	DNA	D NA	DNA	DNA	DNA	
27	801	895	1252	980	811	805	1161	908	
28	956	1084	2126	2083	980	1186	1660	1928	
29	398	467	551	1009	660	850	759	1420	
30	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
31	1261	M/E	M/E	M/E	1365	M/E	M/E	M/E	
32	NT	NT	NT	NT	NT	NT	NT	NT	
33	404	547	512	542	410	531	551	585	
34	302 508	332	374	379 576	332	473	508	431	
35 36	330	453	535 534	576	541 404	545	648	599 501	
30 37	DNA	368 DNA	524 DNA	690 DNA	404 DNA	421 DNA	542	581	
37 38	DNA	DNA	DNA	DNA	DNA DNA	DNA	DNA DNA	DNA	
39	357	382	431	469	386	531	472	DNA 502	
40	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA	
41	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
42	230	241	318	306	283	307	337	319	

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up

M/E= data not available, micro. or experimenter error

TABLE C1.1: SAMPLE A MEDIAN CORRECT RT (msec), (cont)

	POSIT	IVE SE		month	nth Follow-up NEGATIVE SET				
Subj.	1	2	3	4	1	2	3	4	
1.	490	546	593	633	546	547	607	617	
2	533	515	579	631	633	615	543	860	
3	352	458	479	472	421	464	482	448	
4	402	477	516	518	400	491	552	539	
5 6	946 396	993 479	1168 509	954 576	973 469	953 400	894 594	1100	
7	316	395	429	466	349	499 427	584 468	634 483	
8	494	503	597	618	499	570	690	670	
9	443	552	626	666	513	630	711	630	
10	667	798	917	825	711	935	993	973	
11	581	607	602	577	592	603	688	596	
12	347	423	421	453	397	440	489	482	
13	506	619	482	590	555	679	590	739	
14	535	628	660	638	604	703	740	725	
15	804	898	977	1032	834	1001	932	1063	
16	317	363	416	488	372	512	563	573	
17	327	348	408	426	351	407	484	486	
18 10	517	657	61·8	595	563	680	671	744	
19 20	490 526	669 475	M/E 450	701 479	525 501	754 537	M/E 508	870 559	
21	581	648	991	969	695	549	1009	1318	
22	820	1099	1418	1176	962	999	1185	1398	
23	302	285	297	452	358	267	332	433	
24	337	383	413	401	327	368	475	516	
25	314	442	499	531	535	547	588	566	
26	504	520	589	758	627	600	677	949	
27	750	767	1058	930	709	723	931	906	
28	1524	1249	1533	1995	1398	1494	1369	1612	
29	405	452	M/E	641	460	554	M/E	629	
30	712	863	1473	1061	684	873	1460	1012	
31	621	961	1529	1017	748	1208	1284	1292	
32 33	411 DNA	396 DNA	477 DNA	499 DNA	469 DNA	544 DNA	654 DNA	581 DNA	
33 34	311	333		374	312	353			
35				528		490	550		
36	304	307	420	426	393	440	497		
37		949	914	1122	886	1122	1020	1271	
38	449		421	500	391	449	422	542	
39	314	366		446		423			
40		760	1595	955		818	1074	981	
41	838			1792	905	1423			
42	231	235	294	308	258	282	346	386	

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C1.1: SAMPLE A MEDIAN CORRECT RT (msec) (cont)

Twelve-month Follow-up

	POSIT	IVE SE		-month	<u>roi row-u</u> NEGAT	D IVE SE	ጥ	
	, 0011				1,120,111	1.2 55	•	
Subj.	1	2	3	4	1	2	3	4
_	450	485				===		=
1 2	470	475	614	503	528	528	658	583
2	443	559	660	760	564	666	715	712
3	381	452	474	508	431	430	454	480
4	413	415	483	526	438	477	479	531
5	448	435	526	627	492	505	518	610
6	404	410	408	477	407	430	500	506
7	327	363	405	447	355	416	471	470
8	366	523	520	741	481	561	637	677
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
10	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
11	414	479	532	572	495	529	591	589
12	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
13	411	394	518	697	440	503	636	818
14	529	539	524	635	640	589	594	732
15	426	449	714	704	881	562	627	792
1:6	311	423	750	456	389	625	866	533
17	333	321	469	387	377	433	511	466
18	399	459	495	504	517	545	598	602
19	758	640	687	589	697	663	879	702
20	530	468	532	643	602	574	697	739
21	899	667	1160	1175	787		1484	919
22	668	768	947	1004	455	722	910	1045
23	307	387	386	391	377	473	423	462
24	291	322	409	397	346	370	418	432
25	307	450	380	511	393	373	482	479
26	493	564	658	605	485	594	726	740
27	446	620	567	653	591	613	777	828
28	752	M/E	M/E	777	690	M/E	M/E	835
29	401	446	449	505	441	586	552	528
30	678	603	1097	968	734	720	898	1193
31	814	M/E	1882	1476	987	M/E	1335	1425
32	375	406	517	430	460	529	567	566
33	229	377		435	357		427	454
34	289	325	361	333	321	351	391	357
35	379	420	399	438	464	429	440	468
36	247	278	314	297	327	353	361	420
37	524	661	856	895	687	718	855	1099
38	365	448	472	503	450	454	461	520
39	297	358	412	448	362	420	498	475
40	470	568	502	752	500	656	453	747
41	1002	1467	1750	1551	1006	1558	1456	1882
42	216	235	216	262	283	256	253	317

subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C1.1: SAMPLE A MEDIAN CORRECT RT (msec) (cont)

Twenty-four-month Follow-up

	POSIT	IVE SE	<u>епсу-т</u> Т	our-illo	NEGAT	<u>w-up</u> IVE SE	т	
	1 0011	- 11 Di	•		пдетт	14.10 01	•	
Subj.	1	2	3	4	.1	2	· з	4
1	478	471	508	646	476	524	564	615
2 3	452	478	473	633	496	539	562	649
3	319	344	408	531	393	397	475	560
4	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
5 6	382	414	491	456	408	435	485	512
6	324	362	402	404	386	415	420	511
7	296	328	434	456	330	380	459	468
8	DNA	DNA	DNA	DNA.	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
10	709	994	1144	1021	831	1022	1172	1083
11	369	391	474	473	428	474	580	583
12	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
13	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
14	568	603	589	614	558	682	605	750
15	381	483	858	454	389	461	544	527
16	325	340	300	424	420	451	463	581
17	287	326	343	400	320	377	415	428
18	438	544	502	533	566	629	650	673
19	619	628	982	646	846	738	1040	690
20	496	513	393	507	465	559	517	550
21	838	749	1395	1183	879	742	1343	1415
22	406	547	586	646	413	597	574	741
23	333	311	400	378	351	436	444	511
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
25	369	386	413	463	401	380	434	568
26	480	492	654	780	639	599	778	1011
27	506	528	577	575	561	596	719	792
28	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
31	605	607	734	738	642	764	949	770
32	321	413	446	388	423	504	533	483
33	352	360	394	463	392	390	512	531
34	370	425	459	515	400	462	483	625
35	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA.	DNA
38	325	375	380	508	379	392	419	447
39	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
41	558	691	774	1026	688	776	825	1079
42	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA

PTA= subject untestable, still in PTA

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C1.1: SAMPLE A MEDIAN CORRECT RT (msec) (cont)

Thirty-six-month Follow-up

	POSIT	IVE SE		1X-mon	NEGATI		Т	
Subj.	1	2	3	4	1,	2	3	4
1	479	431	509	538	478	460	549	584
2	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
2 3	308	407	430	386	372	437	491	516
4	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
5	328	384	423	424	386	397	426	461
6	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
7	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
10	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
11	418	479	462	457	441	507	490	522
12	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
13	459	467	435	473	407	433	430	487
14	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
15	353	519	469	594	479	500	590	666
16	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
17	276	306	305	355	287	306	440	425
18	372	547	608	556	501	766	674	687
19	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
20	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
21	795	753	1082	1153	748	761	1047	1435
22	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
23	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
25	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
26	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
27	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
28	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
31	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
32	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
33	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
34	336	363	419	480	361	377	500	488
35	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
39	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
41	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
42 ·	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C1.2: SAMPLE A SD OF CORRECT RT (msec)

	POSIT	IVE SE		nonth 1	Follow-up NEGAT	IVE SE	Γ	
Subj.	1	2	3	4	1	2	3	4
1 2 3 4 5 6 7	193 152 101 67 172 52 64	175 541 112 69 318 148 61	105 585 102 108 363 89 68	284 438 147 131 350 240 107	259 198 94 96 226 121 53	108 793 70 131 344 274 73	114 1368 95 79 351 168 108	159 375 142 203 407 136 103
8 9 10 11 12 13	95 143 127 143 42 124	88 306 235 148 52	193 355 348 120 151 81	178 778 329 252 82 175	91 193 161 109 54 98	49 194 413 128 59 147	187 473 193 135 116	136 503 284 112 85 181
14 15 16 17 18	112 PTA 2323 DNA PTA 241	254 PTA 1804 DNA PTA 243	233 PTA 1291 DNA PTA 167	404 PTA 998 DNA PTA 279	190 PTA 1306 DNA PTA 174	350 PTA 1665 DNA PTA 386	546 PTA 998 DNA PTA 420	502 PTA 973 DNA PTA 338
20 21 22 23 24 25	NT PTA PTA 55 DNA 118	NT PTA PTA 81 DNA 158	NT PTA PTA 139 DNA 314	NT PTA PTA 106 DNA 273	NT PTA PTA 38 DNA 111	NT PTA PTA 53 DNA 92	NT PTA PTA 68 DNA 154	NT PTA PTA 95 DNA 173
26 27 28 29 30 31 32	194 NT PTA NT PTA PTA PTA	305 NT PTA NT PTA PTA PTA	383 NT PTA NT PTA PTA PTA	302 NT PTA NT PTA PTA PTA	430 NT PTA NT PTA PTA PTA	314 NT PTA NT PTA PTA PTA	232 NT PTA NT PTA PTA PTA	265 NT PTA NT PTA PTA
33 34 35 36 37 38	NT 87 NT DNA 304 DNA	NT 94 NT DNA 363 DNA	NT 109 NT DNA 366 DNA	NT 224 NT DNA 362 DNA	NT 117 NT DNA 576 DNA	NT 142 NT DNA 325 DNA	NT 124 NT DNA 453 DNA	NT 163 NT DNA 286 DNA
39 40 41 42	DNA PTA DNA 62	DNA PTA DNA 68	DNA PTA DNA 54	DNA PTA DNA 93	DNA PTA DNA 57	DNA PTA DNA 83	DNA PTA DNA 124	DNA PTA DNA 69

PTA= subject untestable, still in PTA

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up

M/E= data not available, micro. or experimenter error

TABLE C1.2: SAMPLE A SD OF CORRECT RT (msec) (cont)

	POSIT	IVE SET	<u>Three</u>	-month	Follow-up NEGATI	OVE SE	Γ	
Subj.	1	2	3	4	1	2	3	4
1 2 3	75 320	106 119	117 201	163 223	141 231	156 89	180 145	146 371
4	48	73	75	73	85	87	74	104
	39	73	128	95	103	114	214	116
5	189	647	367	456	142	582	385	426
6	72	116	100	124	101	108	92	105
7	47	64	90	113	49	55	88	108
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9	120	99	229	226	75	169	81	117
10	187	286	179	306	345	280	217	209
11	DNA	DNA	DNA	D NA	DNA	DNA	DNA	DNA
12	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
13	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
14	11 ²	139	98	111	148	197	117	155
15	440	556	451	594	235	998	157	626
16	107	190	254	239	162	101	113	152
17	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
18	118	235	199	181	80	144	157	149
19	128	168	299	284	235	125	370	186
20	119	168	116	300	139	159	100	169
21	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
22	899	585	447	1079	751	414	442	449
23	48	70	65	81	36	64	85	67
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	D NA
25	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
26	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
27	420	224	442	239	425	278	594	152
28	343	559	880	1031	293	807	780	687
29	75	205	188	342	160	169	215	1717
30	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA
31	289	M/E	M/E	M/E	413	M/E	M/E	M/E
32	NT	NT	NT	NT	NT	NT	NT	NT
33	86	69	187	119	92	53	131	121
34	51	73	99	97	56	95	169	142
35	97	102	113	75	101	84	103	
36	84	109	170	201	86	89	103	180
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
39 40	101 PTA	43 PTA	67 PTA	97 PTA	44 PTA	70	54	74
41 42	DNA 48	DNA 56	DNA 40		DNA 48			DNA B1

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up

M/E= data not available, micro. or experimenter error

TABLE C1.2: SAMPLE A SD OF CORRECT RT (msec) (cont)

2 81 162 159 189 210 104 117 234 3 52 117 82 158 72 58 98 104 4 58 93 144 116 83 89 106 92 5 203 176 272 289 263 161 252 318 6 78 106 190 95 111 110 112 128 7 58 55 99 116 58 67 74 108 8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148					month	Follow-up		_	
1 79 269 79 127 74 99 117 123 2 81 162 159 189 210 104 117 234 3 52 117 82 158 72 58 98 104 4 58 93 144 116 83 89 106 92 5 203 176 272 289 263 161 252 318 6 78 106 190 95 111 110 112 128 7 58 55 99 116 58 67 74 108 8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148		POSIT	IVE SET			NEGAT	IVE SE	Г	
2 81 162 159 189 210 104 117 234 3 52 117 82 158 72 58 98 104 4 58 93 144 116 83 89 106 92 5 203 176 272 289 263 161 252 318 6 78 106 190 95 111 110 112 128 7 58 55 99 116 58 67 74 108 8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148	Subj.	1 .	2	3	4	1	2	3	4
3 52 117 82 158 72 58 98 104 4 58 93 144 116 83 89 106 92 5 203 176 272 289 263 161 252 318 6 78 106 190 95 111 110 112 128 7 58 55 99 116 58 67 74 108 8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148							9.9		123
4 58 93 144 116 83 89 106 92 5 203 176 272 289 263 161 252 318 6 78 106 190 95 111 110 112 128 7 58 55 99 116 58 67 74 108 8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148									234
5 203 176 272 289 263 161 252 318 6 78 106 190 95 111 110 112 128 7 58 55 99 116 58 67 74 108 8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148									
6 78 106 190 95 111 110 112 128 7 58 55 99 116 58 67 74 108 8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148									
7 58 55 99 116 58 67 74 108 8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148									
8 71 89 121 138 121 75 97 157 9 79 133 90 85 80 101 123 148									108
						121	75	97	157
									148
									379
·									89 75
									240
									177
15 229 94 280 998 74 306 211 731	15		94						731
									180
									100
									283 388
									50
									340
									485
									109
									132
									116
									181 353
									807
									318
									254
									459
									206 DNA
									138
									72
									62
									611
									104
									97
									362 293
									86

NT= subject not tested, poor physical/cognitive condition

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TABLE C1.2: SAMPLE A SD OF CORRECT RT (msec) (cont)

	POSIT	EVE SE	Twelve- T	-month	Follow-up NEGATI		,	
Subj.	1	2	3	4	1	2	3	4
1234567890112141567890122222222223333333333442	82 121 149 63 64 63 101 101 101 101 101 101 101 101 101 10	105 112 109 138 121 100 101 101 102 103 104 105 107 107 107 107 107 107 107 107 107 107	104 138 149 118 177 22NAA 199 106 130 130 130 130 130 130 130 130 130 130	205 148 178 178 178 178 178 178 178 178 178 17	132 126 67 56 63 57 62 40 DNA 105 DNA 105 174 132 49 63 468 132 41 52 116 60 128 177 48 63 48 185 48 184 35	88 125 142 555 150 127 150 151 151 151 151 151 151 151 151 151	162 162 163 163 164 165 165 165 165 165 165 165 165	92 92 77 68 96 58 88 DNA 302 121 520 111 215 138 438 237 526 102 138 238 130 473 916 133 916 133 184 104 104 104 104 104 104 105 106 106 107 107 108 108 108 108 108 108 108 108 108 108

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up

M/E= data not available, micro. or experimenter error

TABLE C1.2: SAMPLE A SD OF CORRECT RT (msec) (cont)

		Tw	entv-f	our-mo	nth Follow	√-up		
	POSIT	IVE SE	T					
Subj.	1	2	3	4	1	2	3	4.
1	107	69	99	115	133	62	79	114
2	84	88	77	116	87	97	110	338
3	55	58	86	144	56	66	97	145
4	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
5	61	112	100	84	54	98	112	109
6	65	45	95	88	39	87	106	81
7	41	24	5.7	84	63	66	79	75
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
10	325	244	292 156	403	279	424	311 99	310
11 12	73 DNA	74 DNA	156 D NA	107 DNA	94 DNA	103 DNA	DNA	105 DNA
13	DNA	DNA	DNA	DNA	DNA DNA	DNA	DNA	DNA
14	143	211	126	206	144	162	127	224
15	87	187	358	397	88	151	347	94
16	78	82	83	140	105	73	144	162
17	55 55	85	120	82	59	69	96	71
18	100	113	114	79	92	68	119	82
19	299	271	730	1133	263	212	830	197
20	159	152	83	86	226	141	67	125
21	301	284	1505	263	239	261	366	371
22	74	187	145	171	96	152	191	315
23	962	102	101	51	57	54	72	86
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
25	83	51	150	122	154	91	115	112
26	90	111	282	249	158	112	246	296
27	96	81	132	219	187	74	129	396
28	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
31	94	98	261	187	116	229	426	115
32	106	98	114	102	51	109	138	119
33	46 165	65	60	92	35	77	106	104
34 35	165	110	149	129	67 DNA	174	63 DN3	114 DNA
35 36	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA	DNA DNA	DNA DNA	DNA
36 37	DNA	DNA	DNA	DNA	DNA DNA	DNA	DNA	DNA
3 <i>7</i> 38	48	64	78	87	123	57	53	87
39	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
41	107	693	206	241	187	227	289	318
42	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
			;					

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TABLE C1.2: SAMPLE A SD OF CORRECT RT (msec) (cont)

		Thi	irty-s:	ix-mont	h Follow-	up		
	POSIT	IVE SET	Γ		NEGATI	VE SE	Γ	
Subj.	1	2	3	4	1	2	3	4
1	9,9	82	92	119	183	119	93	81
2	DNA	DNA	DNA	DNA	D NA	DNA	DNA	DNA
3	50	84	80	105	67	55	74	88
4	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
5	41	99	85	98	64	79	70	106
6	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
7	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9 10	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA	DNA DNA	DNA
11	51	86	102	76	72	DNA 86	100	DNA 61
12	DNA	DNA	DNA	DNA	D NA	DNA	DNA	DNA
13	69	99	95	97	78	49	158	71
14	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
15	83	84	167	284	113	140	169	282
16	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
17	61	54	61	87	59	69	119	75
18	191	125	134	208	192	175	162	139
19	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
20	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
21	166	323	352	282	403	239	318	383
22	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
23	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
25	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
26	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
27 28	DNA DNA	DNA D NA	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA
28 29	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA DNA
30	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
31	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
32	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
33	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
34	70	56	56	100	63	70	99	146
35	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
39	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
41	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
42	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C1.3: SAMPLE A MEAN CORRECT RT (msec)

One-month Follow-up NEGATIVE SET POSITIVE SET 3 4 2 3 4 2 1 Subj. 1 756 745 663 709 728 771 684 686 1 2 549 683 890 889 688 671 945 880 3 451 462 526 527 485 446 568 547 4 463 686 721 475 547 514 637 555. 5 1256 813 864 1217 1257 779 827 1158 6 460 637 553 805 769 696 664 619 391 7 384 468 512 421 509 562 546 570 803 805 8 592 544 789 776 582 1300 9 953 985 1274 1765 1056 1072 2093 1055 824 954 10 600 758 808 910 708 11 648 656 733 767 657 756 760 809 12 362 398 562 674 398 421 598 704 543 577 517 561 656 589 697 13 527 1062 739 1120 1156 705 901 1105 1191 14 PTA PTA PTA PTA 15 PTA PTA PTA PTA 16 5536 4879 3844 3465 3255 3054 2955 3238 17 DNA DNA DNA DNA DNA DNA DNA DNA PTA PTA PTA PTA PTA PTA PTA 18 PTA 595 771 765 706 834 1020 1130 19 818 20 NT NT NT ТИ NT NT NT NT21 PTA 22 PTA 443 23 390 421 359 344 403 313 354 DNA DNA DNA DNA 24 DNA DNA DNA DNA 877 25 482 594 821 830 562 656 689 1102 1211 26 766 988 1199 1144 1045 1026 27 NT NT NT NT NT NT NT NT PTA PTA PTA PTA PTA PTA 28 PTA PTA 29 NT NT NT NT NT NT NT NT PTA PTA PTA PTA PTA PTA PTA 30 PTA 31 PTA 32 PTA PTA PTA PTA PTA PTA NT 33 NT NT NT NT NT NT NT 529 34 406 524 599 437 595 653 648 NT 35 NT NT NT NT NTNT NT DNA 36 DNA DNA DNA DNA DNA DNA DNA 1342 37 981 1047 1179 1283 1214 1287 1369 38 DNA DNA DNA DNA DNA DNA DNA DNA 39 DNA DNA DNA DNA DNA DNA DNA DNA PTA PTA PTA PTA 40 PTA PTA PTA PTA 41 DNA DNA DNA DNA DNA DNA DNA DNA 328 42 287 308 343 374 320 435 412

PTA= subject untestable, still in PTA

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up

M/E= data not available, micro. or experimenter error

TABLE C1.3: SAMPLE A MEAN CORRECT RT (msec) (cont)

	DOGT	TUD OD		-month	Follow-u		ım.	
	POSTI	IVE SE	T		NEGAT	IVE SE	iT	
Subj.	1	2	3	4	1	2	3	4
1	483	528	656	618	597	665	702	678
2	644	528	664	689	666	606	674	912
3	358	387	438	499	415	440	493	533
4	417	519	545	528	503	572	651	671
5	1005	1718	1575	1673	954	1742	1425	1662
6	530	545	618	609	619	637	680	726
7	332	381	464	438	400	429	542	575
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9	491	604	737	843	533	694	754	807
10	564	821	796	898	694	900	872	785
11 12	DNA DNA	DNA DNA	DNA DNA	DNA DNA	DNA	DNA	DNA	DNA
13	DNA	DNA	DNA	DNA	DNA DNA	DNA DNA	DNA DNA	DNA DNA
14	566	676	647	707	628	715	679	802
15	1619	1577	1827	1687	1209	1884	1621	1744
16	382	493	674	675	486	550	646	679
17	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
18	459	798	656	689	457	694	781	750
19	378	588	865	780	540	588	928	903
20	512	566	546	696	600	569	582	711
21	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
22	3151	1980	1756	2452	1863	1552	1683	1877
23	280	326	301	339	315	348	365	373
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
25	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
26	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
27	820	939	1347	996	1003	875	1346	963
28	1031	1214	1893	2146	1056	1309	1671	2064
29	416	567	583	1019	653	857	824	1874
30	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA
31	1231	M/E	M/E	M/E	1413	M/E	M/E	M/E
32	NT	NT	NT	NT	NT	NT	NT	NT
33		548			427		571	
34	305	349			342			
35	531	478	563	588	561	537		644
36	355	399	536	663	423	450	559	
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
39	377	383	442	479			461	511
40	PTA	PTA			PTA			PTA
41	DNA				DNA			
42	248	259	320	318	292	304	333	331

PTA= subject untestable, still in PTA NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C1.3: SAMPLE A MEAN CORRECT RT (msec) (cont)

	POSIT	IVE SE		month	Follow-up NEGAT	IVE SE	T.	
Subj.	1	2	3	4	1	2	3	4
1	502	615	586	669	540	571	659	663
2	526	570	618	711	699	629	592	869
3	373	493	482	499	429	457	512	476
4	408	494	531	536	432	464	569	540
5	973	959	1153	1039	978	942	976	1159
6	408	512	541	589	473	539	604	674
7	336	401	443	481	374	429	486	497
8	480	532	617	655	527	575	683	689
9	472	584	653	658	502	646	728	682
10	728	821	1029	932	786	851	1069	1111
11	580	602	622	620	595	625	730	621
12	340	426	430	478	394	447	504	497
13	553	855	592	614	540	719	709	781
14	540	641	682	677	601	741	732	793
15	854	899	1078	1539	825	1144	983	1479
16	325	398	442	464	379	491	553	637
17	339	359	411	426	378	419	511	496
18	531	653	634	621	589	735	708	868
19	661	722	M/E	771	512	963	M/E	988
20	515	482	469	484	545	554	522	568
21	590	651	1072	1008	767	609	1058	1248
22	888	1073	1455	1292	1097	994	1229	1484
23	333	313	320	466	369	290	331	444
24	347	394	441	417	357	377	496	526
25	335	442	479	530	469	621	603	543
26	505	540	583	873	694	662	695	944
27	756	737	1006	1179	735	753	993	1027
28	1525	1190	1618	2038	1384	1654	1459	1865
29	446	510	M/E	785	480	602	M/E	782
30	768	923	1593	1178	699	927	1628	1103
31	631	958	1758	1223	786	1280	1393	1424
32	460	436	506	523	478	551	625	597
33	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
34	315	338	353	394	365	359	406	461
35	341	434	507	536	388	529	545	554
36	302	342	437	449	400	449	492	511
37	650	1053	940	1107	934	1246	991	1462
38	436	384	437	535	421	450	433	556
39	336	385	478	473	417	429	486	549
40	768	761	1772	1011	762	840	1308	1058
41	847	1399	1868	1767	905	1448	1824	1806
42	248	258	307	316	256	300	361	362

PTA= subject untestable, still in PTA NT= subject not tested, poor physical/cognitive condition

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M/E= data not available, micro. or experimenter error

TABLE C1.3: SAMPLE A MEAN CORRECT RT (msec) (cont)

			Twelve	-month	Follow-up	<u>]</u>		
	POSI	TIVE	SET			V	IEGATIVI	E SET
Subj.	1	2	3	4	1	2	3	4
1	456	490	624	587	561	544	706	605
2	479	559	668	722	545	685	737	702
3	416	469	461	498	447	431	470	492
4	404	418	506	511	446	460	501	539
5	445	474	568	636	506	551	548	637
6	411	405	428	496	433	441	501	515
7	328	355	403	455	377	409	484	475
8	397	548	571	735	483	582	649	702
9	DNA	DNA						
10	DNA	DNA						
11	449	474	540	553	518	578	595	599
12	DNA	DNA						
13	420	435	571	808	496	512	645	850
14	505	552	564	669	670	610	618	730
15	457	459	749	741	937	609	685	994
16	327 336	479	796 479	483	488	604	926	558 476
17 18	391	317	512	392 525	387 533	444	520	476 504
19	879	466 768	782	626	522 899	549 792	617 1039	584 737
20	519	477	565	636	624	589	695	737 777
21	964	676	1181	1218	915	852	1497	1063
22	697	788	1019	1028	572	750	945	1003
23	329	415	394	409	381	491	441	471
24	309	334	424	400	363	387	434	432
25	342	461	384	506	409	466	456	476
26	472	575	695	658	497	664	725	746
27	481	608	623	654	610	632	811	886
28	787	M/E	M/E	874	748	M/E	M/E	872
29	449	464	510	563	456	603	584	558
30	651	711	1219	1068	751	770	1021	1357
31	839	M/E	2340	1512	1014	M/E	1382	2023
32	397	428	528	456	509	523	678	614
33	312	372	373	449	361	379	437	460
34	303	345	364	351	337	347	408	386
35	397	442	392	476	378	425	446	496
36	265	288	354	312	334	3,73	379	431
37	502	696	943	923	880	746	930	1064
38	386	426	475	518	483	506	469	529
39	318	371	487	511	362	420	495	572
40	507	575	502	776	547	666	492	764
41	1010	1474	1810	1649	1050	1591	1744	2024
42	236	237	235	291	271	256	265	334

PTA= subject untestable, still in PTA

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DNA= subject did not attend for follow-up

M/E= data not available, micro, or experimenter error

TABLE C1.3: SAMPLE A MEAN CORRECT RT (msec) (cont)

		Tw	enty-f	our-mor	th Follo	w-up		
	POSI		SET				EGATIV	E SET
Subj.	1	2	3	4	1	2	3	4
1	485	485	520	637	510	534	575	620
2	469	484	477	609	501	553	608	720
3	332	363	425	557	396	404	503	602
4	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
5	394	447	509	480	408	435	535	546
6	343	362	416	413	400	414	455	506
7	304	328	435	480	347	389	451	464
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
10 11	836 389	1037 422	1166 527	1048 486	890 439	1169 495	1180 587	1095 575
12	DNA	DNA	DNA	DNA	DNA	DNA	DNA	D NA
13	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
14	564	651	525	704	593	696	615	795
15	417	507	854	651	397	484	609	545
16	338	371	337	478	449	474	490	620
17	302	344	380	407	338	379	436	451
18	450	557	527	553	588	634	655	
19	703	697	1124	1022	799	745	1304	735
20	541	533	433	512	529	528	515	555°
21	910	817	1826	1158	965	831	1363	1445
22	426	611	633	658	434	600	605	833
23	549	352	426	378	366	441	448	497
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
25	382	401	449	488	448	389	474	555
26	479	501	712	841	640	610	804	1016
27	516	543	613	672	655	578	732	923
28	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
30 31	DNA 633	DNA 614	DNA 811	DNA 755	DNA 676	DNA 846	DNA 1046	DNA 800
32	349	415	473	408	428	478	550	513
33	361	374	417	491	401	412	550 550	542
34	407	472	488	517	399	511	484	650
35	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	336	399	394	486	402	391	416	472
39	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
41	554	905	792	1074	695	802	946	1033
42	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA

PTA= subject untestable, still in PTA

NT= subject not tested, poor physical/cognitive condition

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C1.3: SAMPLE A MEAN CORRECT RT (msec) (cont)

		Th	irty-s	ix-month	Follow-	up		
	POSI	TIVE	SET			N	EGATIVE	SET
Subj.	1	2	3	4	1	2	3	4
1	485	444	529	554	551	483	564	576
2	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
3	305	427	418	430	385	442	504	550
4	DNA	DNA	DNA	DNA	DNA	DNA	DŅĀ	DNA
5	329	412	426	435	378	417	432	483
6	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
7	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
10	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
11	422	438	464	480	451	513	501	414
12	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
13	448	465	463	490	419	431	482	508
14	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
15	380	514	521	700	503	527	610	751 DV3
16	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
17	282	315	319	350 605	294	342	440	437
18	326	558	639	605	484	803	737	692
19	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
20	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
21 22	830 DNA	858	1049 DNA	1212 DNA	911	836	1051 DNA	1528
23	DNA	DNA DNA	DNA	DNA	DNA DNA	DNA DNA	DNA	DNA DNA
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA DNA
25	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
26	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
27	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
28	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
31	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
32	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
33	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
34	362	378	422	481	372	395	513	522
35	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
39	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
41	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
42	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA

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TABLE C1.4: 'ON'/'OFF' ANTICONVULSANT MEDICATION

	POSIT	IVE SI	ET			NE	475 583 5 608 736 6 589 594 7 457 484 4	
Subj.	1	2	3	4	1	2	3 .	4
6-0N	396	479	509	576	469	499		634
6-0FF	373	439	491	530	431	475		501
14-ON	530	577	642	694	584	608		654
14-OFF	529	539	524	635	640	589		732
33-ON	333	418	445	449	397	457	484	459
33-OFF	299	369	360	438	359	356	433	444

APPENDIX C2:

MAIN STUDY: MEDIAN, SD, & MEAN RT DATA, SAMPLE B

TABLE C2.1: SAMPLE B MEDIAN CORRECT RT (msec)

Twenty-four month Follow-up									
	POSI		SET				EGATIV	E SET	
Subj.	1	2	3	4	1	2	3	4	
1 2 3 4 5 6 7 8 9	427 469 372 562 374 419 529 4366 436 575	616 545 388 690 383 478 598 1512 440 986	596 573 348 904 464 601 640 M/E 434 810	576 534 328 1389 512 630 627 5430 617 1241	479 415 367 700 398 514 533 2146 348 653	593 514 407 935 428 457 528 1735 425 1034	562 714 404 933 501 628 661 M/E 640 857	601 628 446 1394 512 640 701 2321 547 1381	
				ix mon	th Follow				
	POSI	TIVE	SET			N	EGATIV	E SET	
Subj.	1	2	3	4	1	2	3	4	
1 2 3 4 5 6 7 8 9 10	504 502 337 442 328 444 807 1198 351 415	572 615 343 508 393 441 734 1343 419 749	653 615 346 783 402 430 914 1571 433 716	517 616 394 856 446 554 1055 1483 514 978	506 450 347 557 374 461 774 1437 389 477	522 525 356 639 424 514 734 1250 438 782	624 591 392 729 478 599 833 1879 560 939	559 610 474 902 472 643 1084 1440 540 802	

TABLE C2.2: SAMPLE B SD OF CORRECT RT (msec)

Twenty-four month Follow-up									
	POSI	TIVE S	SET			N	EGATIV	E SET	
Subj.	1	2	3	4	İ	2	3	4	
1	60	129	124	218	66	160	102	130	
2	105	121	90	182	166	148	212	99	
3	136	1113	91	86	60	94	70	323	
4	219	1235	245	2732	189	772	463	980	
5	70	85	85	117	85	103	59	90	
6	115	140	242	148	96	180	97	237	
7	148	184	112	143	148	153	120	192	
8	3195	2319	M/E	3335	1789	2866	M/E	2390	
9	98	91	86	211	249	96	217	153	
10	187	248	281	224	424	142	269	395	
Thirty-six month Follow-up									
	POSI		SET	2310710			EGATIV	E SET	
Subj.	1	2	3	4	1	2	3	4	
_									
1	84	114	149	65	120	75	111	133	
2	100	90	79	147	102	104	151	132	
3	40	28	97	515	74	66	83	80	
4	118	222	152	271	139	172	142	218	
5	64	57	102	146	50	60	71	82	
6	117	. 110	155	393	156	113	114	102	
7	120	170	213	200	123	203	193	236	
8	488	560	659	924	587	804	1080	573	
9 10	94	113	81	97	106	71	276	181	
	95	124	349	198	173	130	234	259	

TABLE C2.3: SAMPLE B MEAN CORRECT RT (msec)

Twenty-four month Follow-up									
	POSI	TIVE	SET				IEGATIV	E SET	
Subj.	1	2	3	4	. 1	2	3	4	
1 2 3 4 5 6 7 8 9	428 472 391 557 392 455 560 5320 447 619	654 539 647 1103 408 472 598 2512 434 1004	619 585 397 931 460 695 640 M/E 443 819	666 608 439 2490 501 627 651 5926 646 1218	480 472 383 693 429 527 570 2569 407 733	647 571 410 1218 426 450 593 3461 447 1036	598 736 421 1090 506 639 665 M/E 676 894	616 655 516 1590 511 670 717 3202 602 1415	
	POST	<u>Th</u> TIVE	irty-s SET	ix mon	th Follow		na artu	E GER	
	1051	1145	761			N	EGATIV	E SET	
Subj.	1	2	3	4	1	2	3	4	
1 2 3 4 5 6 7 8 9	505 498 336 470 346 424 808 1323 362 422	595 619 349 578 387 428 717 1430 414 774	650 617 377 809 438 474 852 1716 436 786	530 639 544 858 481 639 1080 1833 489 892	523 469 344 597 377 459 784 1610 412 554	553 555 365 697 416 511 744 1547 450 801	638 634 407 772 468 595 840 2071 662 945	603 637 474 895 478 636 1103 1628 604 901	

APPENDIX C3:

MAIN STUDY: MEDIAN, SD, & MEAN RT DATA SAMPLE C

TABLE C3.1: SAMPLE C MEDIAN CORRECT RT (msec)

	POSIT		rst As SET	sessme	nt Sessio		EGATIVE	SET
Subj.	1	2	3	4	1	2	3	4
1 2 3 4 5 6 7 8 9	358 442 412 520 324 300 305 M/E 346 464	M/E 391 411 578 323 327 330 446 391 402	416 392 483 671 345 356 350 510 409 436	386 450 576 671 342 369 407 440 482 647	347 446 394 575 360 344 334 M/E 343 431	M/E 397 437 559 343 385 389 421 362 477	466 418 506 624 399 409 448 527 428 586	461 491 543 754 428 412 472 459 468 648
	POSIT		cond 1 ET	\ssessm	ent <u>Sessi</u>		EGATIVE	SET
Subj.	1	2	3	4	1	2	3	4
1 2 3 4 5 6 7 8 9	331 311 339 475 304 262 324 315 347 619	332 355 435 601 320 305 335 420 372 401	454 407 437 612 357 351 362 491 401 429	378 411 443 641 354 401 331 474 335 540	379 347 421 509 298 316 364 447 389 710	424 394 467 578 351 359 416 474 360 532	445 491 499 672 410 420 426 554 441 636	436 470 467 631 416 393 435 552 400 663
	POSIT		ird As SET	sessme	nt Sessio		EGATIVE	SET
Subj.	1	2	3	4	1	2	3	4
1 2 3 4 5 6 7 8 9	324 343 342 470 308 300 283 325 324 323	336 322 437 551 333 320 347 416 325 348	376 324 458 484 360 332 363 459 384 M/E	382 393 474 742 377 368 342 481 373 482	391 352 400 437 289 336 344 428 336 440	378 398 452 565 340 351 437 465 366 479	436 432 535 565 365 366 464 498 385 M/E	426 472 498 658 430 388 441 512 386 609

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C3.1: SAMPLE C MEDIAN CORRECT RT (msec) (cont)

			ourth	Assessment	Sess	<u>ion</u>		
	POSIT	TIVE	SET				NEGATIVE	SET
Subj.	1	2	3	4	1	2	3	4
1	311	307	354	368	359	355	420	411
2	294	335	329	334	337	376	444	422
3	345	423	448	514	410	465	509	529
4	399	517	546	534	502	487	560	609
5	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
6	271	316	359	417	359	357	423	441
7	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
10	313	M/E	427	401	442	M/E	490	515

TABLE C3.2: SAMPLE C SD OF CORRECT RT (msec)

		<u>F</u>	irst .	Assessment	Session	Ĺ		
	POSIT	CIVE	SET			NE	GATIVE	SET
Subj.	1	2	3	4	1	2	3	4
1	54	M/E	191	131	47	M/E	86	86
1 2 3	56	68	76	86	105	60	68	47
	152	71	90	107	89	99	103	116
4	103	75	92	121	97	89	100	179
5	106	38	66	100	91	71	91	86
6	62	42	55	55	48	84	85	48
6 7	50	84	65	142	76	93	156	86
8	M/E	90	127	69	M/E	78	72	158
9	48	84	123	132	43	69	87	103
10	76	73	96	196	142	99	213	202
			<u>econd</u>	Assessment	Sessio			
	POSIT	TIVE	SET			NE	GATIVE	SET
Subj.	1	2	3	4	1	2	3	4
1	40	40	177	77	67	106	111	67
	25	45	116		45	54	76	108
2 3	38	53	85	86	55	71	59	79
4	52	72	164		64	80	87	136
5	55	66	69	110	53	53	60	80
6	42	40	65		45	54	109	42
7	53	53	49	77	145	70	61	83
8	73	66	128		67	54	79	76
9	49	76	87	91	88	68	59	74
7	77	70	07	91	00	00	Ja	7 7

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

10 149 83 150 142 85 128 105 100

TABLE C3.2: SAMPLE C SD OF CORRECT RT (cont)

	POSIT		nird A	ssessmen	t Session		EGATIVE	SET
Subj.	1	2	3	4	1	2	3	4
1 2 3 4 5 6 7 8 9	42 24 48 111 48 34 36 61 56	39 58 86 62 52 41 47 50 83 75	86 78 112 350 68 77 74 113 97 M/E	206 131 80 185 95 73 63 74 84 191	105 72 88 151 42 83 44 106 74 254	63 66 96 50 47 45 66 96	75 95 97 118 62 94 65 71 58 M/E	108 126 103 271 72 45 109 107 69 160
	DOCT			Assessme	nt Sessio		2027145	C TOTAL
Subj.	POSIT	2	3 3	4	1	N1 2	EGATIVE 3	SET 4
1 2 3 4 5 6 7 8 9	38 30 31 129 DNA 29 DNA DNA DNA 30	44 94 54 72 DNA 51 DNA DNA DNA M/E	114 77 100 170 DNA 53 DNA DNA DNA	69 67 109 196 DNA 135 DNA DNA DNA	81 36 71 74 DNA 46 DNA DNA DNA	79 72 53 79 DNA 78 DNA DNA DNA M/E	89 92 60 76 DNA 83 DNA DNA DMA	60 180 67 64 DNA 58 DNA DNA DNA 104

TABLE C3.3: SAMPLE C MEAN CORRECT RT (msec)

		Fi	i <u>rst</u> As	sessme	nt Session			
	POSI	rive 3	SET			1	VEGATIVE	SET
		•						
Subj.	1	2	3	4	1	2	3	4
1	368	M/E	468	432	344	M/E	481	469
2	445	409	409	481	480	418	440	500
3	459	422	503	574	409	478	527	564
4	529	580	672	685	541	572	656	811
5	351	324	345	367	366	347	406	437
6	316	329	365	371	354	379	413	415
7	311	381	381	431	353	414	502	492
8	M/E	455	551	462	M/E	451	523	518
9	346	383	454	507	339	370	438	497
10	445	407	448	651	465	502	665	685

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

TABLE C3.3: SAMPLE C MEAN CORRECT RT (msec) (cont)

				Assessment	Sess				
	POSIT	IVE	SET		•		NEGATIVE	SET	
Subj.	1	2	3	4	1	2	3	4	
1	335	343	488	392	389	445	489	442	
2	311	364	442	427	343	404	499	515	
3	349	436	470	475	408	485	505	492	
4	480	490	669	667	511	587	673	671	
5	318	342	369	390	308	363	426	423	
6	271	311	373	396	321	360	431	385	
7	325	347	370	355	400	435	435	448	
8	341	432	505	517	466	473	566	566	
9	460	386	406	361	399	363	454	420	
10	652	422	482	561	708	559	627	654	
Third Assessment Session									
	POSIT	IVE _	SET			- 	NEGATIVE	SET	
Subj.	1	2	3	4	1	2	3	4	
1	325	340	403	437	408	392	435	459	
2	339	329	356	442	380	404	443	512	
3	350	335	478	474	419	456	548	523	
4	468	542	584	789	465	584	566	748	
5	316	338	375	416	303	345	372	431	
6	300	317	359	366	358	358	403	390	
7	292	362	377	3 58	344	432	452	458	
8	333	412	479	477	449	487	498	533	
9	334	355	398	364	336	405	400	413	
10	326	359	M/E	537	493	473	M/E	629	
Fourth Assessment Session									
	POSIT	IVE	SET				NEGATIVE	SET	
Subj.	1	2	3	4	1	2	3	4	
1	312	314	391	383	366	385	442	422	
2	304	367	358	352	339	388	446	513	
3	348	422	484	539	403	461	530	530	
4	431	547	610	584	509	493	583	590	
5	DNA	DNA	DNA	DNA	DNA	DNA		DNA	
6	274	328	356	437	350	385	424	451	
7	DNA	DNA	DNA	DNA	DNA.	DNA	DNA	DNA	
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
10	306	M/E	419	408	485	M/E	570	539	

DNA= subject did not attend for follow-up M/E= data not available, micro. or experimenter error

APPENDIX C4:

MAIN STUDY: CLINICAL VARIABLES RAW DATA

Table C4.1
Main Study: Clinical Data, Sample A

<u>Sub</u>	<u>GCS</u>	<u>U/C</u>	PTA	<u>sev</u>	<u>AC</u>	<u>sku</u>	<u>CAT</u>	<u>LAT</u>	SUR	FIT	<u>AC</u>
1	11	.,5	1	M/M	4	RP	RPH	R	RPH	No	No
2	12	1	12	VS	2	LPO	No	L	No	HO	12
3	14	0	0	M/M	3	NAD	No	No	No	No	No
4	7	. 3	5	S	4	LT		L	No	No	6
5	7	16	7+	S	1	No	R	?R	No	No	16
6	13	0	7	S	2	RF	R	R	RF	No	6
7	4	48	28	VS	1	No	RFT	R	No	No	No
8	10	50	7+	VS	6	#	No	No	OT	No	No
9	6	12	6	S	4	AF	R	?R	No	No	No
10	13	?0	4	S	1	RO	No	No	No	No	No
11	8	4	1+	S	8	No	No	No	No	No	3
12	6	13	1	M/M	4	No	No	?L	No	No	No
13	8	72	. 5	M/M	2	No	No.	No	OT	No	2
14	4	103	30	ES	1	NAD	RT	R	No	НО	10
15	5	384	42	ES	4	No	L	No	No	No	No
16	12	0	20	VS	1	NAD	NAD	No	No	No	No
17	14	0	. 01	M/M	1	No	No	No	No	No	No
18	3	408	45	ES	1	No	NAD	L	No	НО	12
19	14	. 3	. 01	M/M	8	RP	NAD	R	R	НО	No
20	8	12	8	VS	1	RF	No	R	No	No	No
21	4	336	35	ES	2	RP	RPH	В	No	No	9
22	4	1080	42+	ES	1	NAD	R	R	No	No	No
23	11	48	10	VS	7	#	No	No	No	No	No
24	11	?0	?.01	M/M	1	No	No	No	No	No	No
25	8	12	. 6	M/M	1	LP	NAD	L	No	No	No
26	4	39	5	S	3	NAD	ABN	No	No	No	No
27	7	313	17	S	2	NAD	NAD	R	No	No	No
28	4	744	56+	ES	1	No	R	?R	No	No	9 N-
29	6	350	15+	VS	3	RT	RT	H	RC	No	No
30	3	800	120	ES	4	No	L	L	No	No	No
31 32	4	976	50	ES	5	R NAD	R	R	RC	No	No
33	11 3	1032 192	42+ 15	ES VS	1 2	NAD	OED LPH	L L	No	Yes	30 9
34	13	?0	1	M/M	1	NAD	ABN	No	No No	HO HO	Yes
3 4 35	4	.5+	25	W/M VS	1	RFP	R	R		No	No
36		.1	23 7	S	_	RT	R		RC		No
37	11 5	96	38	ES	7 4	?#	LT	R L	RC No	No HO	15
38	7	113	5	S	6	No	No	No	No	No	No
39	3	50	14	VS	1	NAD	RFH	В	No	No	7
40	4	750	98	ES	4	No	No	No.	No	No	No
41	7	12	1	M/M	4	#	F	No	OT	Yes	Yes
42	?	?O	.3	M/M	2	# No	r No	R	No	No	No
74	;	: 0		1.1/ 1.1	4	140	140	1.7	140	140	140

(see Table C4.2 for key)

Table C4.2 Main Study: Clinical Data, Sample B

<u>Sub</u>	<u>GCS</u>	<u>U/C</u>	<u>PTA</u>	<u>SEV</u>	<u>AC</u>	<u>SKU</u>	CAT	LAT	SUR	<u>FIT</u>	<u>AC</u>
1 2 3 4 5 6 7 8	12 11 3 3 14 4 14 3 12	100 5 240 12 .25 72 .25 72		V/S M/M E/S M/M V/S V/S S E/S M/M	3 1 3 3 6 1 6 2	LTP No # LF No No LT	RFH NAD RED No L RSD No RF LF	R R R No L R No L	No No RED No LC RC CSF OT LC	HO HO No HO No No Yes No Yes	.5 24 No No 18 No 34 No 36
10	5	513	42	E/S	2	NAD	ABN	Ĺ	No	но	12

GCS= Glasgow Coma Scale; U/C= Hours unconscious:

PTA= Days of post-traumatic amnesia; SEV= Head Injury severity:

M/M= Mild/moderate; S= Severe;

ES= Extremely severe; VS= Very severe;

AC= Cause of Head Injury:

1= RTA, Car 5= Occupational

2= RTA, Motor Cycle 6= Sport 3= RTA, Cycle 7= Home 4= RTA. Pedestrian 8= Other

SKU= Skull fracture? - LTP= Left temperoparietal;

LF= Left frontal; LT= Left temporal;

#= Yes, unspecified; NAD= No abnormality demonstrated;

CAT= CT Scan?; RFH= Right frontal haemorrhage;

L= Left abnormality; ABN= Abnormal, unspecified

LAT= Evidence of additional lateralised cerebral damage;

SUR= Neurosurgical intervention; OT= other operation;

RC= Right craniotomy;

CSF= CSF leak;

FIT= epileptic fits:

HO= Yes, in hospital only

APPENDIX C5:

MAIN STUDY: DEMOGRAPHIC DATA

Table C5.1: Main Study, Demographic Data, Sample A

<u>Sub</u>	<u>Age</u>	<u>Sex</u>	Return to Work(mth)	Education	Social Class	<u>Handed</u>
1	32	F	4	Degree	2.	L/R*
2	39	M	3	O/Ã	3	R
3	17	F	1	Α	2	R
4	21	M	2	Α	1	R
5	19	F	8	CSE	5	,R
6	17	M	3	0	2	R
7	20	M	4	UNIV	5	R
8	36	M	11	15	5 2 5 3	L
9	31	F	No	15	4	R
10	20	F	2	Ō	4	R
11	14	M	?1	Ō	3	R
12	16	M	U/E	15	7	L
13	16	F	2	CSE	3	R
14	18	F	8	15	4	R
15	29	F	No	0	3	R
16	18	F	5	A	3 6	R
17	15	F	. 25	A	6	R
18	18	M	5	CSE	3	R
19	17	M	5	CSE	4	L
20	48	M	4	U/K	3	R
21	17	M	22	0	4	R
22	18	M	No	16	4	R
23	18	F	4	0	3	R
24	20	M	U/K	UNIV	6	R
25	13	M	N/A	N/A	6	R
26	13	M	2	N/A	6	R
27	18	M	6	CSE	2	R
28	32	M	No	Degree	2	R
29	21	M	No	U/K	3	L*
30	18	F	12	A	6	Ŕ
31	50	M	No	15	3	R
32	17	M	No	0	4	R
33	17	M	4	0	2	Ŕ
34	35	F	U/E	0	0	R
35	17	M	9	UNIV	6	R
36	19	F	5	A	2	R
37	50	M	No -	UNIV	2	R
38	1.9	F	No	0	2	R
39	23	M	No	U/K	4	R
40	1.3	F	9	N/A	6	R
41	21	M	18	15	4	R
42	18	F	U/K	0	2	R

Return to work/school:

UNIV= Currently University student; Degree= gained degree;

U/E= Unemployed at time of head injury;

U/K= Unknown; N/A= Not applicable, residential school;

* = Non-dominant hand responses, dominant hemiplegia
Education: 15 = left school at 15, no exam certificates;
CSE= gained 1(+) CSEs; O/A= gained 1(+)'O' or 'A' levels;

APPENDIX C6:

MAIN STUDY: ADDITIONAL RT DATA, t-TESTS & CORRELATIONS

TABLE	C6.1:	SAMPLE	A	&	WITH	IIN	SEVERITY	GROUP
	•	T-TESTS	B	EΤV	VEEN	FOI	LOW-UPS	<u> </u>

	<u>-</u>	Positive	Set 10220	<u> </u>	
M/M (5) M/M (10)	FU 1 v 3: 3 v 6: 6 v 12: 12 v 24:	<1 <1	2 1.478 <1 <1 <1	3 <1 <1 <1 <1	<1 <1 <1 <1
S (6) S (8)	1 v 3: 3 v 6: 6 v 12: 12 v 24:	<1 1.853*	<1 <1 1.356 1.661	<1 <1 1.627 <1	<1 1.097 1.599 <1
VS (8) VS (10)	1 v 3: 3 v 6: 6 v 12: 12 v 24:	<1 <1	1.298 <1 <1 <1	1.422 <1 <1 2.231*	1.402 <1 <1 <1
ES (11) ES (8) A (15) A (25) A (38)	3 V 6: 6 V 12: 12 V 24: 1 V 3: 3 V 6: 6 V 12: 12 V 24:	1.242 <1 1.669 <1 1.183	1.116 3.171*** <1 1.102 <1 1.778* 1.197	<1 1.382 <1 1.113 <1 1.241 1.382	1.107 1.099 1.386 1.290 <1 1.156 1.367
					-
M/M (5) M/M (10)	FU 1 v 3: 3 v 6: 6 v 12: 12 v 24:	< 1	Set 2 <1 <1 <1 <1	3 <1 1.153 <1 <1	4 <1 <1 <1 <1
M/M(n=5) M/M (5) M/M (10) M/M (8)	1 v 3: 3 v 6: 6 v 12: 12 v 24: 1 v 3: 3 v 6: 6 v 12:	1 <1 <1 <1 <1	2 <1 <1 <1	<1 1.153 <1	<1 <1 <1
M/M(n=5) M/M (5) M/M (10) M/M (8) S (5) S (6) S (8) S (6) VS (5) VS (8) VS (10)	1 v 3: 3 v 6: 6 v 12: 12 v 24: 1 v 3: 3 v 6: 6 v 12: 12 v 24: 1 v 3: 3 v 6: 6 v 12: 12 v 24:	1 <1 <1 <1 <1 <1 <1 <1 1.503	2 <1 <1 <1 <1 <1 <1 1.581	<1 1.153 <1 <1 <1 <1 1.371	<1 <1 <1 <1 <1 <1 1.480
M/M(n=5) M/M (5) M/M (10) M/M (8) S (5) S (6) S (8) S (6) VS (5) VS (8) VS (10) VS (5) ES (6) ES (11)	1 v 3: 3 v 6: 6 v 12: 12 v 24: 1 v 3: 3 v 6: 6 v 12: 12 v 24: 1 v 3: 3 v 6: 6 v 12: 12 v 24: 3 v 6: 6 v 12: 12 v 24:	1 <1 <1 <1 <1 <1 <1 1.503 <1 1.804 <1 <1	2 <1 <1 <1 <1 <1 1.581 <1 1.300 1.193 <1	<1 1.153 <1 <1 <1 <1 1.371 <1 1.363 1.956*	<1 <1 <1 <1 <1 <1 1.480 <1 1.229 1.056 <1
M/M(n=5) M/M (5) M/M (10) M/M (8) S (5) S (6) S (6) S (8) S (6) VS (5) VS (8) VS (10) VS (5) ES (6) ES (11) ES (8) A (15) A (25) A (38)	1 v 3: 3 v 6: 6 v 12: 12 v 24: 1 v 3: 3 v 6: 6 v 12: 12 v 24: 1 v 3: 3 v 6: 6 v 12: 12 v 24: 3 v 6: 6 v 12: 12 v 24:	1 <1 <1 <1 <1 <1 <1 1.503 <1 1.804 <1 <1 <1 <1 <1 <1 <1 <1 <1 <1	2 <1 <1 <1 <1 <1 1.581 <1 1.300 1.193 <1 1.144 <1 2.722***	<1 1.153 <1 <1 <1 <1 1.371 <1 1.363 1.956* <1 1.812 <1 1.621	<1 <1 <1 <1 <1 <1 <1 <1 <1 <1 <1 <1 <1 <

TABLE 6.2: RT's ON TRIALS PRECEDING ERROR TRIALS

SAMPLI Subj.	E <u>A</u> : <u>FU</u>	<u>Set</u> Size	<u>Mediar</u> <u>+VE</u>	N RT	Preceding 1	Trial RT's
1	1m	1	744	646*	-435	+437
	Зm	2	51 <i>7</i>	648*	-461	+427
		4	575*	662	+563	-486
		4	575	662*	+424	+414
	6m	4	633*	617	-541	+586
	12m	1	470*	528	4 05	+328
		1	470	528*	+423	+365
	24m	1	478*	476	+662	-418
		4	646	615*	-666	+711
	36m	2	431 *	460	-624	+431
		3	509*	549	-727	+484
		4	538*	584	-503	+397
2	1m	1	507	627*	-530	+501
		3	866*	849	-867	+754
		4	897*	810	-628	N/A
	Зm	1	579*	614	+707	-153
		4	666*	760	+669	-666
	6m	2	515	615*	-656	+599
		3	579*	543	-660	+418
	12m	2	559*	666	+393	-697
		3	660*	715	-405	+454
	24m	1	452*	496	+347	-371
		3	562	473*	+646	+531
		4	633*	649	+667	-206
3	1m	2	454*	432	-463	+398
		2	432	454*	-441	+545
	Зm	3	462*	482	+333	+318
	6m	2	458*	464	+499	-393
	12m	3	474*	454	-487	+365
	24m	1	319	393*	+279	-389
		3	408	475*	-373	+342
		4	531 *	560	-551	+381
		4	531	560*	-466	-616
	36m	1	308*	372	-4 58	-450
		3	430*	491	+358	+435
		4.	386*	516	-503	+397
		4	386	516*	-648	+367

^{*} denotes whether error trial occurred on a postive or negative run

^{-/+} denotes whether preceding trial was positive or negative

N/A denotes either that preceding trial was an error, or that there was no preceding trial

TABLE 6.2: RT's ON TRIALS PRECEDING ERROR TRIAL (cont)

<u>Subj</u>	<u>FU</u>	<u>Set</u> Size	Median +VE	RT -VE	Preceding	Trial RT's
5	1m 3m 12m	3 2 1 2 2	1038 1840 448 435*	1133* 1722* 492* 505	+950 +2304 +376 +701	+1142 +1297 +458 -375
	24m	2 4	414 512	435* 456*	-316 +640	-373 -260 -428
	36m	1 2 4	328 384* 424*	386* 397 461	-423 +343 +368	+307 +441 -316
6	1m 6m	2 1 2 3	624* 396 479	732 469* 499*	-780 +492 -550	-602 +399 +478
	12m	1 4	509* 404 477*	584 407* 506	-641 -358 +440	-430 +452 -453
	24m	1 2 3	324* 362 402*	386 415* 420	-402 -511 -444	-396 +356 +564
7	1m	1 2	356 376*	396* 519	-377 -418	+351 +364
	Эm	3 4	444 426*	538* 577	+665 -375	−601 +695
	12m	3	405* 405	471 471*	-572 -714	-471 +413
4.0	24m	4	456*	468	-363	-399
10	1m Зm	2 4 2	726* 770* 798*	917 930 807	-662 -722 -1454	+678 +1662
	6m	1 2 4	796* 667* 798* 976*	711 807 825	-1454 -705 -1164 -1257	+592 -805 -958 +873
	24m	2	994*	1022	+1672	−95 0
11	1m 6m	3 2 4	731* 607 577*	737 603* 596	-602 -533 +580	+597 +658 -533
	12m	1 1 2 2	414* 414 479* 479	495 495* 592 529*	+605 +331 -483 +362	-478 +322 +363 -500
	36m	3 4	462* 462 457*	490 490* 522	+406 -612 +618	-548 +504 -606

^{*} denotes whether error trial occurred on +ve/-ve run

^{-/+} denotes whether preceding trial was +ve/-ve

TABLE 6.2: RT's ON TRIALS PRECEDING ERROR TRIAL (cont)

Sub j	<u>i. FU</u>	<u>Set</u> Size	<u>Media</u> +VE	n RT -VE	Preceding	Trial RT's
14	3m	1 4	577 721*	644* 764	-348 +729	+663 -737
	6m 12m	3 1	660* 529	740 640*	-524 -495	+300
	1 2111	2	539	589*	-493 -483	+322 +437
		3	524*	594	-709	+300
		4	635*	732	+583	-641
	24m	1	568	558*	-903	+924
		3	589*	605	+577	-437
		4	614*	750	-866	-854
15	6m	4	1032*	1063	-1023	+984
	1 7	4	1032 714	1063*	-1023	N/A
	12m 24m	3 2	483*	627* 461	-684 -363	+627 -469
16	1m	1	5396	3067*	+5106	+5608
	2	4	3169* 356*	3195	+3169	+4280
	Зm	1 1	356°	432 432*	+346 -328	-408 -348
	6m	2	363	512*	-326 -291	+203
	O,III	3	416	563*	+372	-612
	12m	2	423*	625	-376	N/A
	24m	3	300	463*	+281	+264
17	6m	2	348	407*	~302 °	+266
		3	408	484*	+430	-452
	12m	4 1	426* 333*	486 377	+342	+258
	1 2111	1	333"	377*	+223 +306	-297 -272
		2	321 *	433	-280	+211
		2	321	433*	-434	+294
		3	469*	511	+404	+332
		3	469	511*	+381	+359
		4	466*	387	-404	+387
	24m	1	287	320*	-238	-228
		2	326*	376 376*	-276	+208
		2 3	326 343*	376* 415	−188 −261	+208
		3	343	415*	+321	+209 -403
18	Зm	1	460*	445	+290	N/A
		4	682*	734	+683	+530
		4	682	734*	-521	-618
	6m	2	657	680.*	+602	+388

TABLE 6.2: RT's ON TRIALS PRECEDING ERROR TRIAL (cont)

<u>Subj</u>	<u>. F⊍</u>	<u>Set</u> Size	Media +VE	n RT <u>-VE</u>	Precedia	ng Trial RT's
18	12m	1	399*	517	-639	N/A
		1	399	517*	-554	-517
	24m	2	544	629*	+718	+781
		3	502	650*	-684	+548
19	1m	3 .	750*	895	-629	+567
	Зm	1	369*	540	-537	-283
		3	883*	855	+883	-1073
	6m	2	669*	754	-656	-53 5
	24m	2	628*	738	-869	+1521
20	Зm	2	534	513*	-730	+529
	6m	1	526*	501	-482	+614
		1	526	501*	-501	+360
		3	450*	508	-437	-582
	14m	4	507*	550	-423	+462
21	12m	2	667	878*	+1039	-931
		2	1160	1484*	+975	-1548
	36m	1	795	748*	-1970	-748
22	Зm	3	1829*	1541	-1163	-1521
	6m	2	1099*	999	+643	-1367
		4	1176*	1398	-882	+708
	24m	2	547*	597	-449	+531
		3	586*	574	+497	-422
		4	646	741*	-835	+656
23	1m	4	379	408*	+378	-441
	Зm	1	272	305*	+279	-262
		2	316*	344	-456	-280
		3	293	370*	+415	-450
	6m	1	302	358*	+301	-413
		4	452*	433	-432	+413
	12m	1	. 307*	377	-375·	-373
25	1m	2	566	646*	-646	+477
	_	3	688*	669	+318	-626
	6m	1	314	435*	+290	+615
	12m	1	307*	393	+247	-177
		4	511	479*	+465	N/A
	24m	1	369	401*	+367	-862
		2	386	380*	+371	+337

TABLE 6.2: RT's ON TRIALS PRECEDING ERROR TRIAL (cont)

Subj.	<u>FU</u>	<u>Set</u> Size	<u>Media</u> +VE	n RT -VE	Preced	ling Trial RT's
27	3m	1 2	801* 895*	811 805	-1470 +579	-1935 -589
	12m	2 3	620*	613	+620	+661
	12m	3	567* 719	777 577*	+487 -537	−905: −741
		4	575	792*	+543	+691
31	3m	1	1261	1365*	-1512	-1222
	6m	1 3	621 1529*	746* 1284	+538 -1025	-684
	12m	3	1882	1335*	-1025 -1351	-998 -1335
32	6m	2	396*	544	-611	+346
		3	477	654*	-311	+360
		4. 4	499* 499	581 581 *	+399	-328 - 424
	12m	1	375*	581* 460	+358 -647	+424 -355
	T 2 111	1	375	460*	+287	-356
		2	406	529*	+272	N/A
	24m	1 2	321	423*	-368	-420
			413	504*	-399	N/A
		4	388	483*	+379	N/A
33	Зm	1	404	410*	-390	+396
	12m	3 3	512 364*	551* 427	+449	-593
	1 2111	3	364	427*	+279 -300	-278 +2 4 3
		4	435*	454	-3 5 6	+243
		4	435	454*	+329	-351
34	3m	3	374	508*	-931	+447
	6m	4	374*	419	-319	+450
	1-2m	4 1	374	419*	+405	-344
	1 2111	4	289* 333*	321 357	+249 +333	-339 -342
	24m	2	425	462*	-331	+347
37	1m	2	958*	1197	-1014	-1197
	1.0	3	1018*	1216	-1058	-1218
	12m	1	524* 524	687	+326	-1270 +524
		1 2	524 661*	687* 71¦8	+533 -883	+524
		2	661	718*	-863 -711	-8 4 9 +888
		-		: - 0	,	, 500

denotes whether error trial was on +ve/-ve run
+/- denotes whether preceding trial was +ve/-ve
N/A denotes either that preceding trial was an error,
or that there was no preceding trial

TABLE 6.2: RT's ON TRIALS PRECEDING ERROR TRIAL (cont)

Subj	<u>. FU</u>	<u>Set</u> Size	Media +VE	n RT <u>-VE</u>	Preceding	ng Trial RT's
38	6m	4	500	542*	-673	+551
	12m	2	448*	454	-347	-913
		4	503*	520	+358	N/A
		4	503	520*	+388	-430
	24m	2	375*	392	+390	+335
		4	508*	447	-460	+261
40	6m	1	722*	697	+501	-582
		2	760*	818	+490	-819
		2	760	818*	-786	+858
		4	955*	981	+1212	+1235
	12m	1	470*	500	-452	+371
		2	656	568*	+521	-580
		4	747*	752	+602	+620
41	12m	3	1750*	1456	-1487	-1381
	24m	1	558*	688	-490	+379
		1	558	688*	+432	-592
		2	691*	776	-534	-815
		2	691	776*	+811	-592
42	1m	1	275*	315	-329	-411
		4	347*	414	-356	-321
	Зm	3	318*	337	+316	-327
	6m	1	231	258*	-194	-227
		3	294*	346	+290	+421
	12m	1	216*	283	-224	+200
		1	216	283*	+202	+216
		2	235*	256	-274	N/A
		2	235	256	-274	+234

denotes whether error trial was on +ve/-ve run +/- denotes whether preceding trial was +ve/-ve N/A denotes either that preceding trial was an error,

or that there was no preceding trial

TABLE 6.2: RT's ON TRIALS PRECEDING ERROR TRIAL (cont)

SAMPLE Subj.	<u>C</u> : <u>FU</u>	<u>Set</u> Size	Median +VE	RT -VE	Preceding 1	Trial RT's
1	1	4	386*	461	-375	+927
2	2 3 4	1 2 3 3 4 3	311* 355* 407* 324 393* 335	347 394 491 432* 472 376*	-296 +338 -535 +295 +347 -580	+264 -364 -416 -639 -411 N/A
3	1 3	1 4	412 474*	394* 498	-411 -507	-452 -437
4	1 2 3 4	1 2 3 3	520* 601* 484 546*	575 578 565* 560	+330 +670 -466 +854	-436 -550 +484 -499
5	1 2 3 3	2 3 4 2 2 3 4	323 345 342 320 333* 360* 377*	343* 399* 428* 351* 340 365 430	+342 +295 -236 -294 +295 +452 N/A	-343 +283 -335 +462 -318 +281 -384
6	1 2 3	2 3 4 2 4 2 4 4 2 4	327 356* 369* 305* 401* 320* 368* 368 316* 417*	385* 409 412 359 393 351 388 388* 357 441	-313 +424 -337 +269 +329 +320 -295 -456 +352 -517	+296 -407 -401 -360 +399 +339 +287 +376 -351 -422

TABLE 6.2: RT's ON TRIALS PRECEDING ERROR TRIAL (cont)

SAMPLE Subj.	<u>C</u> : <u>FU</u>	<u>Set</u> Size	<u>Median</u> +VE	RT -VE	Preceding 1	Trial	RT's
7	1	1 2 3 4	305* 330 350 407*	334 389* 448* 472	-443 -373 +352 -460	-379 +324 -433 -602	
	2 3	3 4 3	362* 331* 363*	426 435 464	-393 -667 -437	-483 +288 +333	
	J	4	342	441*	+274	-458	
8	1 2 3	4 4 2	443 474* 416*	459* 452 465	+359 -666 +377	+444 +484 +418	
9	3	2 3 1 3 4 4	372 401* 324* 384 373* 373	360 441 336 385* 386 386*	-249 +435 -330 +369 +392 +245	+556 +313 +290 -356 -406 -346	
10	1 3 4	1 3 4 3 4	464 436* 482 427 401*	431* 586 609* 490* 515	+275 +328 +427 +501 +275	-415 -542 -477 +372 +314	

		TABL	E 6.3	: AVERAG	E SD OF	RT		
		itive				ive Se		
1/12 FU: A (n=23)	<u>1</u>	<u>2</u>	<u>3</u>	4	<u>1</u>	<u>2</u>	<u>3</u>	. <u>4</u>
	200	205	230	286	237	222	252	251
— - -	254	196	200	214	264	206	212	200
<u>M/M(8)</u>								
	121	127	135	195	121	136	168	162
sd :	62	59	75	77	63	99	99	78
<u>Sev(7)</u>	4.00	040	050	0.40	404	057	200	050
	128	218	252	340	191 107	257	233	273
sd : VS(6)	48	91	128	191	107	101	126	131
	385	272	323	316	398	262	315	307
	434	364	341	343	428	369	346	336
ES(2)	151	00-1	J-1.	040	120	303	0.10	000
	235	309	300	383	361	338	500	394
	87	55	67	21	161	13	47	108
3/12 FU:								
A (27)								
•	173	197	215	263	177	213	201	242
	182	177	179	245	155	233	173	223
M/M(5)	~ 0	0.5	100	1.40	110	400	170	4.00
	7.0	95 40	126	142	113	102	170	139
sd : Sev(7)	31	40	90	77	69	37	111	41
	159	231	231	235	182	231	241	186
	119	188	118	112	131	161	175	104
VS(9)	117	100	+10		101	101	1,0	-0.
Av. SD	111	114	142	177	112	94	115	245
	78	55	63	95	62	40	43	280
ES(6)								
	367		415	576	320	512	331	420
	265	189	270	382	220	337	253	235
6/12 FU:								
A (41)	1 2 0	160	300	222	1.40	170	104	226
	128	163	208	222	140	178	194	236
sd : <u>M/M(11)</u>	85	137	156	185	89	145	167	178
	124	190	156	148	106	190	169	163
sd :	122	221	127	101	55	200	150	96
Sev(10)		201		101		200	200	,,,
	112	133	180	207	143	134	154	185
•	60	61	101	138	71	50	84	113
<u>VS(9)</u>		•						
	97	98	113	147	97	103	86	147
	53	36	22	95	55	40	21	81
ES(11)	450	045	252	270	200	265	200	400
	: 172	215	350	370 346	206	265	333	429
sd :	: 57	95	179	246	111	144	203	202

	TAB	LE 6.	3: AV	ERAGE	SD OF	RT (con	<u>it)</u>	
	Posi	tive				gative		
12/12 FU: A (39)	<u>1</u>	2	<u>3</u>	<u>4</u>	1	<u>.</u> <u>2</u>	<u>3</u>	<u>4</u>
Av. SD :		110 60	186 180	167 98		24 124 9 81		198 212
M/M(10)								
ed :	133 151	123 93	173 151	165 113	13 12			206 275
<u>Sev(8)</u> Av. SD : sd :		88 27	112 36	108 31		76 122 24 3 8		119 84
<u>VS(10)</u> Av. SD :		97	142	130		56 92		112
sd : ES(11)	39	34	74	42	2	27 29	64	48
Av. SD :		127 44	301 273	246 101	20	00 146 06 60		325 238
24/12 FU: A (26)								
	149 179	144 129	200 209	191 185		23 132 59 83		177 103
M/M(7) Av. SD : sd :	12 4 79	191 217	220 211	262 304	13	31 129 72 68		153 76
<u>Sev(5)</u>	266	97	81	97)2 88		164
sd : VS(8)		217	13	30		7 30		91
Av. SD :	197 291	105 30	1·07 54	121 47		95 111 54 42		160 82
<u>ES(6)</u> Av. SD :	129	168	302	201	1.1	18 162	245	189
sd : B (10)		62	320	106		50 59		112
Av. SD :		399 395	151 76	333 335	24 27	18 285 71 306		360 328
36/12 FU: A (10)	200	030	, 0		Δ,	2 000	122	020
Av. SD :		109 74	122 83	143 75		31 108 02 58		144 101
B (10) Av. SD :	_	159	204	296		53 180		200
sd : C (10)		143	170	247		16 213		138
Av. SD :		59 16	117 84	111 45)2 70 59 22		117 60
su :	20	10	O~ <u>1</u>	40	_	13 44	19	00

TABLE C6.4		SAMPLE A & S		ROUPS
	r-1ESIS E Positiv	ETWEEN FOLLOW Se Set	<u>r-0P5</u>	
GROUP: FU M/M(n=5) 1 v M/M (5) 3 v M/M (10) 6 v M/M (8) 12 v	3: 2.005* 6: 1.026 12: <1	2 1.288 1.160 <1 <1	3 <1 1.264 <1 <1	1.311 <1 <1 <1
S (5) 1 v S (6) 3 v S (8) 6 v S (6) 12 v	12: 1.593	<1 <1 1.606 <1	<1 <1 1.673 <1	<1 1.301 2.071* <1
VS (5) 1 v VS (8) 3 v VS (10) 6 v VS (5) 12 v	6: <1 12: <1	1.112 <1 <1 <1	1.230 1.026 1.699 1.588	<1 <1 <1 <1
ES (6) 3 V ES (11) 6 V ES (8) 12 V A (15) 1 V A (25) 3 V A (38) 6 V A (27) 12 V	12: 1.266 24: <1 3: 1.537 6: <1 12: <1	2.338** 2.788*** 1.273 <1 1.397 1.891* <1	<1 <1 <1 1.114 <1 <1	<1 1.547 <1 1.566 <1 1.803*
GROUP: FU M/M(n=5) 1 v M/M (5) 3 v M/M (10) 6 v M/M (8) 12 v	6: <1 12: <1	re Set 2 1.021 <1 <1 1.142	3 1.533 <1 <1 <1	<1 <1 <1 <1 <1
S (5) 1 v S (6) 3 v S (8) 6 v S (6) 12 v	6: <1 12: 2.546**	<1 1.304 <1 3.023***		1.388 <1 <1 <1
VS (5) 1 v VS (8) 3 v VS (10) 6 v VS (5) 12 v	6: <1 12: 1.038	<1 <1	1.609 2.291**	1.039 1.057 <1 1.967*
ES (6) 3 v ES (11) 6 v ES (8) 12 v	12: <1	2.530**	<1 1.547 <1	<1 1.105 1.438
A (25) 3 V A (38) 6 V	6: <1	1.900*	<1 <1	< 1

TABLE C6.6: BINOMIAL TEST VALUES FOR RT SD, SAMPLE A

Positive Set

<u>FU</u> :	<u>1</u> .	<u>2</u>	<u>3</u>	<u>4</u>
1- 3m: 3- 6m: 6-12m: 12-24m: 24-36m:	.00 1.60 .00	1.55 .40 1.97** .00 .00	.52 .83 1.50 .00 2.21**	1.03 .80 1.28 .00
		Negative Se	<u>:t</u>	
FII.	1	2	3	4

<u>FU</u>: <u>1</u> <u>2</u> <u>3</u> 4 1.55 1.20 2.96 2.55*** 1- 3m: 2.25** . 52 1.03 1.67* 3- 6m: 1.20 1.20 6-12m: .96 12-24m: .39 24-36m: 1.58 . 50 3.84*** . 39 1.54 . 59 . 00 . 31

TABLE C6.6: T-TEST VALUES, MEDIAN RT & SD FOR NEUROSURGERY & NO-GENERAL ANAESTHETIC SUB-GROUPS, SAMPLE A

		•	POSITIVE		
FU (n1,	<u>n2)</u> :	<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
3/12	RT:	<1	2.319**	1.409	1.119
(7,20)	SD:	1.407	1.747*	1.269	1.174
6/12	RT:	1.692*	<1	<1	<1
(7,32)	SD:	<1	<1	<1	<1
12/12	RT:	<1	<1	<1	<1
(7,30)	SD:	<1	<1	<1	<1
24/12	RT:	1.159	<1	<1	<1
(4,21)	SD:	<1	<1	<1	<1
			<u>NEGATIVE</u>		
		<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
3/12	RT:	<1	1.338	1.205	< 1
(7,20)	SD:	1.161	1.996*	<1	< 1
6/12	RT:	1.149	<1°	<1	<1
(7,32)	SD:	1.529	<1	<1	<1
12/12	RT:	<1	<1	<1	<1
(7,30)	SD:	<1	<1	<1	<1
24/12	RT:	1.139	1.006	<1	<1
(4,21)	SD:	<1	<1	1.389	1.670

NB: Significant values favour better performance in the sub-group undergoing neurosurgery

TABLE C6.7: T-TEST VALUES, MEDIAN RT & SD FOR RIGHT HEMISPHERE & LEFT HEMISPHERE SUB-GROUPS, SAMPLE A

	POSITIVE						
FU (n1,	<u>n2)</u> :	<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>		
1/12	RT:	1.298	<1	<1	<1		
(8,5)	SD:	1.091	1.013	<1	1.639		
.3/12	RT:	1.867*	1.269	2.515**	2.381**		
(15,4)	SD:	<1	1.796*	1.308	2.157**		
6/12	RT:	<1	<1	<1	<1		
(15,8)	SD:	<1	<1	<1	1.406		
12/12	RT:	1.022	<1	<1	< 1		
(14,8)	SD:	1.244	1.102	<1	< 1		
24/12		2.377**	2.012*	2.786**	1.478		
(10.5)		1.494	2.446**	1.769*	1.830		
			NEGATIVE				
		<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>		
1/12	RT:	1.191	<1	<1	<1		
(8,5)	SD:	<1	1.631	1.248	1.664		
3/12	RT:	2.661**	1.862*	2.489**	2.439**		
(15,4)	SD:	1.565	2.286**	1.208	1.099		
6/12	RT:	<1	<1	<1	<1		
(15,8)	SD:	<1	<1	<1	<1		
12/12		<1	<1	<1	<1		
(14,8)		<1	<1	<1	<1		
24/12		1.120	1.589	1.512	1.385		
(10,5)		1.783*	2.666**	1.726	<1		

NB: Significant values favour better performance in the sub-group who did not sustain additional right hemisphere damage

TABLE C6.8: CORRELATIONS OF TIME TO RETURN TO WORK/SCHOOL WITH MEDIAN RT, SD, U/C, PTA & AGE, SAMPLE A

MEDIAN RT

<u>FU(n):</u>	1m(17)	3m(18)	6m(26)	12m(26)	24m(19)
Set 1+ve:	.09	.45	.44*	.73**	.62**
-ve:	.07	.42	.49*	.70**	.53**
2+ve:	. 04	. 42	.54**	.65**	.45*
-ve	. 02	. 42	.46*	.70**	.40
3+ve	. 05	. 41	.10	.73**	.60**
-ve	. 04	. 46	.08	.74**	.55**
4+ve	. 06	. 46	.69**	.79**	.67**
-ve	. 05	. 45	.71**	.66**	.69**
		SD O	F RT		
FU(n):	1m(17)	3m(18)	6m(26)	12m(26)	24m(19)
Set 1+ve:	.07	.19	.18	.31	.09
-ve:	.06	.02	.39*	.43*	.38
2+ve:	.11	. 40	. 04	.59**	.71**
-ve:	.09	. 38	. 20	.41*	.38
3+ve:	.14	. 34	. 36	. 55**	.59**
-ve:	.28	. 26	. 38	. 58**	.29
4+ve:	.15	. 21	.42*	.70**	.12
-ve:	.21	. 26	.40*	.71**	.49
U/C:	. 41	.12	.42*	.40*	.44*
PTA:	. 39	.41	.39*	.38	.39
AGE:	. 28	21	01	01	06

^{* =} P<.05; ** = P<.01

TABLE C6.9: T-TEST VALUES, MEDIAN RT & SD FOR ANTICONVULSANT & NON-ANTICONVULSANT SUB-GROUPS, SAMPLE A

POSITIVE

FU (n1,	<u>n2)</u> :	1	2	3	4
3/12:					
ES (3,3) S (3,4)	SD : RT :	2.441* 2.169* <1 1.366	4.037** 2.489* <1 <1	1.426 <1 <1 <1	1.552 1.341 <1 <1
6/12					
ES (6,5) S (3,7)	SD:	<1 3.349*** <1 <1	1.322 <1 <1 <1	3.102** 2.576** <1 <1	<1 1.805 <1 <1
12/12					•
<u>ES</u> (3,5)	RT: SD:	1.061 1.362	<1 <1	<1 1.404	<1 <1
			NEGATIVE		
<u>FU (n1,</u>	<u>n2)</u> :	<u> </u>	2	3	4
3/12:					
ES (3,3) S (3,4)	RT: SD: RT: SD:	3.630** 2.188* <1 1.501	5.292** 1.198 <1 <1	2.150* <1 <1 <1	2.126 1.176 <1 <1
6/12					
ES (6,5) S (3,7)	SD: RT:	<1 <1 <1 <1	<1 <1 <1 <1	2.112* 2.670** <1	<1 <1 <1 <1
12/12					
<u>ES</u> (3,5)	RT:	<1	<1 <1	<1	1.746
(3,3)	SD:	< 1	\1	< 1	1.386

NB: Significant values favour better performance in the sub-group prescribed anticonvulsant medication

TABLE C6.10: T-TEST VALUES, MEDIAN RT & SD FOR FEMALE & MALE SUB-GROUPS, SAMPLE A

		POSITIVE		
<u>FU (n1,n2)</u> :	1	2	3	4
1/12 RT:	<1	<1	<1	<1
(12,11) SD:	<1	<1	<1	1.268
3/12 RT:	1.255	<1	<1	<1
(13,14) SD:	1.559	<1	1.284	1.081
6/12 RT:	<1	<1	<1	1.279
(17,24) SD:	<1	<1	<1	<1
12/12 RT:	2.370**	2.060**	1.635	2.132**
(16,23) SD:	2.235**	2.138**	1.177	<1
24/12 RT:	1.353	<1	<1	1.175
(11,15) SD:	<1	<1	1,337	<1
		NEGATIVE		
	<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
1/12 RT:	<1	<1	<1	<1
(12,11) SD:	<1	<1	<1	1.318
3/12 RT:	1.641	<1	<1	1.135
(13.14) SD:	1.874*	<1	1.899*	1.080
6/12 RT:	1.105	1.064	1.012	1.694*
(17,24) SD:	1.435	2.072**	<1	<1
12/12 RT:	1.431	1.847*	2.454**	1.755*
(16,23) SD:	1.818*	1.687*	1.845*	1.182
24/12 RT:	1.754*	1.062	1.631	1.622
(11,15) SD:	1.096	<1	1.513	1.850*

NB: Significant values favour better performance in the female sub-group

TABLE C6.11: PEARSON CORRELATIONS, MEDIAN RT & SD WITH VERBAL IQ & PERFORMANCE IQ SAMPLE A

			POSITIVE		
<u>FU</u>		1_	2	3	4
1/12((n=23)				
VIQ	RT: SD:	.10 .09	.12 .02	.10 .06	.11 11
PIQ	RT: SD:	.18	.31	. 32	.33
3/12	(27)				
VIQ	RT: SD:	24 20	24 28	22 15	23 21
PIQ	RT: SD:	18 08	24 22	22 14	23 15
<u>6/12</u> VIQ	(41) RT:	10	14	33*	04
PΙQ	SD: RT: SD:	35* .01 30	08 06 .00	39* 34* 38**	33* .02 .01
12/12	(39)	24	0.2	1.2	1.0
VIQ	RT: SD:	24 44** 15	03 12	.12 02	18 17
PIQ	RT: SD:	15 36*	02 10	. 12 03	11 09
<u>24/12</u> VIQ	(26) RT:	4 5*	33	41*	24
PIQ	SD: RT:	.01	16 28	46* 40*	45* 18
LIX	SD:	01	18	47*	47*

TABLE C6.11: PEARSON CORRELATIONS, MEDIAN RT & SD WITH VERBAL IQ & PERFORMANCE IQ SAMPLE A cont

NE	G	Α	Т	T	V	F.

<u>FU</u>		<u> </u>	2	3	4
1/12((n=23)				
VIQ PIQ	RT: SD: RT: SD:	.09 .17 .24 .33	.14 01 .33 .14	.11 14 .34 .05	.08 10 .30 .09
3/12	(27)				
VIQ PIQ	RT: SD: RT: SD:	26 22 19 17	23 13 23 11	21 14 22 17	24 16 24 13
6/12 VIQ PIQ	(41) RT: SD: RT: SD:	14 22 .00 07	12 20 04 17	31 33* 32* 33*	16 11 07 01
12/12 VIQ PIQ	(39) RT: SD: RT: SD:	.17 23 07 23	.03 11 02 12	.11 .00 .12 .00	01 01 .00 01
24/12 VIQ PIQ	(26) RT: SD: RT: SD:	45* 27 41* 20	36 25 33 26	41* 53** 41* 56**	25 19 24 01

APPENDIX C7:

MAIN STUDY: REGRESSSION RAW DATA

TABLE C7.1: MEDIAN RT REGRESSION DATA, SAMPLE A
POSITIVE

3/12 Follow-up

1 -13 718 36 39 453 .77 2 139 383 .96 33 511 .76 3 25 426 .75 47 310 .98 4 45 425 .76 41 319 .87 5 152 587 .97 205 1016 .67 6 58 451 .80 27 496 .68 7 54 290 .97 38 298 .89 8 72 463 .83 DNA DNA DNA 9 203 626 .90 105 367 .99	<u>Subj</u>	Weight	Interc.	Corr.	<u>Weight</u>	<u>Interc</u> .	Corr.
2 139 383 .96 33 511 .76 3 25 426 .75 47 310 .98 4 45 425 .76 41 319 .87 5 152 587 .97 205 1016 .67 6 58 451 .80 27 496 .68 7 54 290 .97 38 298 .89 8 72 463 .83 DNA DNA DNA	1	-13	718	36	39	453	.77
4 45 425 .76 41 319 .87 5 152 587 .97 205 1016 .67 6 58 451 .80 27 496 .68 7 54 290 .97 38 298 .89 8 72 463 .83 DNA DNA DNA		139					
5 152 587 .97 205 1016 .67 6 58 451 .80 27 496 .68 7 54 290 .97 38 298 .89 8 72 463 .83 DNA DNA DNA	3	25	426	. 7.5	47		
6 58 451 .80 27 496 .68 7 54 290 .97 38 298 .89 8 72 463 .83 DNA DNA DNA		45	425	.76	41	319	. 87
7 54 290 .97 38 298 .89 8 72 463 .83 DNA DNA DNA	5		587	. 97	205	1016	. 67
8 72 463 .83 DNA DNA DNA							
9 203 626 .90 105 367 99							
				. 90			. 99
10 48 588 .90 87 526 .79							
11 25 606 .64 DNA DNA DNA							
12 114 222 .98 DNA DNA DNA							
13 -3 549 .18 DNA DNA DNA							
14 131 590 .91 43 539 .93							
15 PTA PTA PTA 78 1270 .77							
16 -804 -603094 92 269 .99							
17 DNA DNA DNA DNA DNA							
18 PTA PTA PTA 50 513 .46							
19 51 563 .49 128 304 .76							
20 NT NT NT 28 513 .67							
21 PTA PTA DNA DNA DNA							
22 PTA PTA PTA -302 296860							
23 26 276 .95 10 274 .63 24 DNA DNA DNA DNA DNA							
28 PTA PTA PTA 442 457 .90 29 NT NT NT 192 127 .89							
30 PTA PTA PTA PTA PTA							
31 PTA PTA PTA M/E M/E M/E							
32 PTA PTA PTA NT NT NT						-	
33 NT NT NT 38 407 .73							
34 41 381 .79 27 279 .96							
35 NT NT NT 29 447 .71							
36 DNA DNA DNA 124 169 .97							
37 153 667 .92 DNA DNA DNA							
38 DNA DNA DNA DNA DNA							
39 DNA DNA DNA 39 314 99							
40 PTA PTA PTA PTA PTA							
41 DNA DNA DNA DNA DNA	41						
42 27 251 .94 31 198 .88	42	27	251	. 94	31	198	

TABLE C7.1: MEDIAN RT REGRESSION DATA, SAMPLE A (cont)
POSITIVE

12/12 Follow-up

<u>Subj</u>	Weight	Interc.	Corr.	Weight	Interc.	Corr.
1	48	447	. 99	24	456	. 45
2	36	475	. 89	1.05	343	. 99
3	38	345	.82	40	353	.,96
4	39	382	. 92	41	358	. 95
5	20	966	. 24	62	356	.92
6	57	348	. 98	22	370	.80
7	48	281	. 97	40	285	.99
8	47	436	. 94	112	257	. 93
9	74	386	. 97	DNA	DNA	DNA
10	59	654	. 74	DNA	DNA	DNA
11	-2	596	15	53	368	.99
12	32	332	. 9	DNA	DNA	DNA
13	12	521	. 22	98	260	. 91
14	34	530	. 79	30	481	.74
15	76	737	.99	110	299	. 90
16	56	255	. 99	76	295	. 52
17	36	288	. 97	31	300	.59
18	20	548	. 42	35	377	. 95
19	63	474	. 84	46	784	83
20	-17	524	68	86	395	. 84
21	151	421	. 91	132	645	. 70
22	139	782	. 72	119	550	. 98
23	46	219	. 75	25	305	. 79
24	22	328	. 85	41	254	. 91
25	71	270	. 95	54	277	. 79
26	83	395	. 92	43	473	. 79
27	72	677	. 79	83	669	. 73
28	170	1151	. 70	M/E	M/E	M/E
29	83	303	. 99	32	372	. 95
30	127	470	. 64	126	546	. 79
31	176	393	. 60	265	683	. 75
32	35	360	.89	27	364	.58
33	DNA	DNA	DNA	40	270	. 91
34	19	291	. 91	17	285	. 73
35	66	277	. 97	16	370	. 78
36	48	245	. 91	19	238	. 83
37	142	551	. 91	131	407	. 97
38	20	386	. 48	34	338	. 95
39	54	274	. 8	51	252	. 99
40	153	625	. 48	78	378	. 79
41	318	687	. 90	193	960	. 78
42	29	194	. 94	12	203	. 70

TABLE C7.1: MEDIAN RT REGRESSION DATA, SAMPLE A cont POSITIVE

36/12 Follow-up

<u>Subj</u>	Weight	Interc.	Corr.	Weight	Interc.	Corr.
1	54	391	. 85	26	426	.72
2	54	375	.83	DNA	DNA	DNA
3	70	226	. 95	26	319	. 62
4	DNA	DNA	DNA	DNA	DNA	DNA
5	30	361	.80	32	308	. 93
6	28	303	. 95	DNA	DNA	DNA
7	59	232	. 96	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA
10	109	696	.76	DNA	DNA	DNA
11	40	328	. 93	10	429	. 50
12	DNA	DNA	DNA	DNA	DNA	DNA
13	DNA	DNA	DNA	1	456	. 07
14	12	563	. 80	DNA	DNA	DNA
15	59	396	. 35	67	316	. 85
16	26	283	. 61	DNA	DNA	DNA
17	36	250	. 97	24	252	. 92
18	24	244	. 65	61	368	. 77
19	44	611	. 31	DNA	DNA	DNA
20	-9	499	2	DNA	DNA	DNA
21	168	621	.72	140	59 5	. 90
22	76	357	. 96	DNA	DNA	DNA
23	22	300	.71	DNA	DNA	DNA
24	DNA	DNA	DNA	DNA	DNA	DNA
25	31	331	. 97	DNA	DNA	DNA
26	106	336	. 96	DNA	DNA	DNA
27	26	483	. 94	DNA	DNA	DNA
28	DNA	DNA	DNA	DNA	DNA	DNA
2 9	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA
31	53	540	. 90	DNA	DNA	DNA
32	23	334	. 57	DNA	DNA	DNA
33	37	301	. 93	DNA	DNA	DNA
34	47	325	. 99	49	273	. 98
35	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA
38	55	259	. 91	DNA	DNA	DNA
39	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA
41	149	391	. 97	DNA	DNA	DNA
42	DNA	DNA	DNA	DNA	DNA	DNA

TABLE C7.1: MEDIAN RT REGRESSION DATA, SAMPLE A (cont)

NEGATIVE

3/12 Follow-up

<u>Subj</u>	Weight	<u>Interc</u> .	Corr.	Weight	<u>Interc</u> .	Corr.
1	17	620	. 89	29	5 5 4	. 82
2	76	542	. 85	48	546	. 92
3	27	431	. 59	41	347	. 96
4	51	461	.61	53	444	. 99
5	171	537	. 91	191	960	. 68
6	45	551	. 53	39	553	. 94
7	45	387	. 83	67	316	. 98
8	84	453	. 91	DNA	DNA	DNA
9	385	383	. 84	92	442	. 98
10	62	662	. 62	36	651	.51
11	40	631	. 93	DNA	DNA	DNA
12	114	235	. 97	DNA	DNA	DNA
13	25	513	. 53	DNA	DNA	DNA
14	85	696	. 76	38	600	. 95
15	PTA	PTA	PTA	131	1138	. 65
16	- 28	3030	11	85	358	. 99
17	DNA	DNA	DNA	DNA	DNA	DNA
18	PTA	PTA	PTA	87	427	. 83
19	151	498	. 97	144	356	. 93
20	NT	NT	NT	25	526	.74
21	PTA	PTA	PTA	DNA	DNA	DNA
22	PTA	PTA	PTA	54	1521	. 46
23	21	327	. 77	22	292	. 9 3
24	DNA	DNA	DNA	DNA	DNA	DNA
25	99	432	. 95	DNA	DNA	DNA
26	85	833	. 91	DNA	DNA	DNA
27	NT	NT	NT	65	760	. 50
28	PTA	PTA	PTA	332	609	. 98
29	NT	NT	NT	219	375	. 82
30	PTA	PTA	PTA	PTA	PTA	PTA
31	PTA	PTA	PTA	M/E	M/E	M/E
32	PTA	PTA	PTA	PTA	PTA	PTA
33	N/T	N/T	N/T	55	383	. 92
34	69	372	.84	33	353	. 57
35	N/T	N/T	N/T	28	515	. 70
36	DNA	DNA	DNA	65	324	. 95
37	75 DV3	987	.90	DNA	DNA	DNA
38	DNA	DNA	DNA	DNA	DNA	DNA
39	DNA	DNA	DNA	29	401	. 59
40	PTA	PTA	PTA	PTA	PTA	PTA
41	DNA	DNA	DNA	DNA	DNA	DNA
42	42	254	. 83	14	277	. 79

TABLE C7.1: MEDIAN RT REGRESSION DATA, SAMPLE A (cont)

NEGATIVE

12/12 Follow-up

<u>Subj</u>	Weight	<u>Interc</u> .	Corr.	Weight	<u>Interc</u> .	Corr.
1	27	511	.92	30	501	.61
2	61	510	. 57	49	541	. 90
3	10	429	. 49	17	406	. 93
4 5	48 32	376 900	. 89 . 4 7	28 37	411 440	. 95 . 88
6	58	402	. 98	37 37	369	. 95
7	44	321	. 95	40	328	.90
8	63	449	. 91	66	423	. 98
9	43	51'3	.68	DNA	DNA	DNA
10	85	691	. 84	DNA	DNA	DNA
11	10	596	. 27	34	465	. 94
12 13	30 46	376 526	. 92 . 71	DNA 126	DNA 283	DNA . 97
14	40	520 593	.84	28	569	.54
15	62	803	.81	-20	766	18
16	65	342	. 91	67	435	. 43
17	48	312	. 95	35	361	. 78
18	53	531	. 91	31	489	. 95
19	107	467 490	. 92	23	673	. 30
20 21	15 233	311	. 69 . 87	53 100	520 767	. 88 . 40
22	150	762	.96	196	294	. 40
23	29	275	. 54	21	383	.60
24	67	253	. 98	31	315	. 97
25	44	426	.82	27	390	. 80
26	104	453	. 83	90	412	. 96
27	80	618	. 87	88 W (F	484 M (F)	. 95
28 29	52 54	1339 419	. 60 . 9 7	M/E 23	M/E 470	M/E .47
30	157	615	.61	119	381	. 91
31	171	704	.84	150	849	.99
32	45	451	. 74	36	442	. 91
33	DNA	DNA	DNA	36	308	. 93
34	37	279	. 99	1.5	318	. 66
35	48	378	. 91	32	345	. 94
36 37	42 105	357 812	. 97 . 83	29 137	294 497	. 94 . 94
38	43	343	.84	22	317	. 94 . 85
39	37	365	.96	42	335	. 88
40	111	616	. 85	54	455	.50
41	293	75	. 94	253	844	. 90
42	45	206	. 98	10	253	.42

TABLE C7.1: MEDIAN RT REGRESSION DATA, SAMPLE A (cont)

NEGATIVE

24/12 Follow-up

36/12 Follow-up

<u>Subj</u>	Weight	Interc.	Corr.	Weight	Interc.	Corr.
1	46	431	. 99	41	416	. 89
2	48	441	. 96	DNA	DNA	DNA
3	58	312	. 94	49	333	. 98
4	DNA	DNA	DNA	DNA	DNA	DNA
5	36	370	. 99	25	354	. 97
6	38	338	. 90	DNA	DNA	DNA
7	49	286	. 96	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA
10	91	801	. 80	DNA	DNA	DNA
11	57	374	. 94	23	434	.82
12	DNA	DNA	DNA	DNA	DNA	DNA
13	DNA	DNA	DNA	24	380	. 90
14	50	524	.76	DNA	DNA	DNA
15	50	356	. 90	65	396	. 97
16	50	355	.90	DNA	DNA	DNA
17	36	294	. 96	55	228	.89
18	34	544	. 96	47	541	. 53
19	-17	870	14	DNA	DNA	DNA
20	21	470	. 64	DNA	DNA	DNA
21	221	543	. 85	234	413	. 94
22	96	341	.92	DNA	DNA	DNA
23	49	313	. 96	DNA	DNA	DNA
24	DNA	DNA	DNA	DNA	DNA	DNA
25	56	307	. 85	DNA	DNA	DNA
26	129	433	. 89	DNA	DNA	DNA
27	82	463	. 98	DNA	DNA	DNA
28	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA
31	57	639	. 58	DNA	DNA	DNA
32	21	433	.58	DNA	DNA	DNA
33	54	322	. 91	DNA	DNA	DNA
34	70	319	. 94	50	306	. 89
35	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA
38	23	352	. 98	DNA	DNA	DNA
39	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA
41	122	537	.93	DNA	DNA	DNA
42	DNA	DNA	DNA	DNA	DNA	DNA

TABLE C7.2: MEDIAN RT REGRESSION DATA, SAMPLE B POSITIVE

24/12	Follow-up	
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<u>Subj</u>	Weight	Interc.	Corr.	<u>Weight</u>	Interc.	Corr.
1	43	447	. 64	12	532	. 22
2 3	22 13	475 352	. 65 . 49	35 17	501 312	. 78 . 85
4	270	213	. 95	152	268	. 96
5 6	50 76	309 343	. 96 . 97	36 32	302 388	. 96 . 70
7	34	515	.87	92	647	. 85
8	M/E	M/E	M/E	108	1128	. 85
9 10	54 182	348 448	. 76 . 83	50 166	304 301	. 97 . 92

NEGATIVE

24/12 Follow-up 36/12 Follow-up

Sub_	<u>Weight</u>	<u>Interc</u> .	Corr.	<u>Weight</u>	Interc.	Corr.
1	34	475	. 77	26	488	. 64
2	84	358	. 82	55	408	. 97
3	23	348	.93	42	288	. 93
4	208	471	. 92	113	426	. 98
5	42	356	. 96	35	350	.92
6	55	423	. 79	63	397	. 99
7	66	444	. 93	103	599	. 84
8	M/E	M/E	M/E	64	1342	. 30
9	81	287	.81	58	338	. 90
10	201	480	.83	113	467	. 74

Interc= intercept; Corr= correlation coefficient: M/E= micro./experimenter error;

TABLE C7.3: MEDIAN RT REGRESSION DATA, SAMPLE C
POSITIVE

	1st Fol	low-up		2nd Fol	low-up	
<u>Subj</u>	Weight	Interc.	Corr.	Weight	Interc.	Corr.
1 2 3 4 5 6 7 8 9	12 3 56 55 7 24 33 M/E 43 58	354 413 330 474 315 279 267 M/E 301 342	.63 .10 .93 .94 .84 .98 .96 M/E .97	26 35 31 51 19 46 5 55 0	308 283 335 455 287 214 326 288 366 550	.58 .95 .81 .89 .92 .99 .37 .89 04 27
	3rd Fol	low-up		4th Fol	low-up	
<u>Subj</u>	Weight	Interc.	Corr.	Weight	Interc.	Corr.
1 2 3 4 5 6 7 8 9	21 15 42 64 23 21 19 51 21	301 308 324 391 286 276 286 293 300 256	.95 .59 .91 .91 .99 .92 .71 .95 .84	22 11 53 43 DNA 48 DNA DNA DNA 33	281 295 300 391 DNA 221 DNA DNA DNA 292	. 91 . 75 . 98 . 82 DNA . 99 DNA DNA DNA

Interc= intercept; Corr= correlation coefficient;
M/E= micro./experimenter error; DNA= did not attend;

TABLE C7.3: MEDIAN RT REGRESSION DATA, SAMPLE C cont

	1st Fol	low-up		2nd Fol	low-up	
<u>Subj</u>	Weight	<u>Interc</u> .	Corr.	<u>Weight</u>	<u>Interc</u> .	Corr.
1 2 3 4 5 6 7 8 9	41 16 52 60 26 23 47 M/E 44 76	315 399 341 478 318 331 293 M/E 291 346	.93 .49 .99 .87 .87 .93 .98 M/E .98	19 47 17 46 41 29 22 40 11	373 309 421 483 266 299 355 408 370 645	.84 .89 .68 .84 .95 .83 .90 .93 .43
	3rd Fol	low-up		4th Fol	low-up	
<u>Subj</u>	Weight	Interc.	Corr.	<u>Weight</u>	<u>Interc</u> .	Corr.
1 2 3 4 5 6 7 8	16 39 38 66 45 8 32 29	367 315 377 391 244 348 342 405	. 76 . 99 . 83 . 94 . 98 . 68 . 77 . 98	22 32 40 39 DNA 31 DNA DNA	331 314 378 441 DNA 317 DNA DNA	.83 .87 .98 .90 DNA .92 DNA DNA

Interc= intercept; Corr= correlation coefficient;
M/E= micro./experimenter error; DNA= did not attend;

APPENDIX C8:

MAIN STUDY: MEMORY TEST RAW SCORES

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B

<u>Sample</u>	<u>A</u>	1/	12 FOLI	LOW-UP				
<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u> 14</u>	<u>A5</u>	<u>TotA</u>	<u>B</u>	A Del
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17	2 5 8 5 6 11 5 4 5 7 7 PTA 6 DNA	7 10 10 9 7 7 15 7 4 7 9 5 10 6 PTA 8 DNA	9 6 14 11 9 11 15 9 6 10 9 8 12 6 PTA 10 DNA	11 7 14 10 11 12 15 9 5 10 11 8 12 7 PTA 12 DNA	11 10 15 13 11 11 15 10 6 12 14 10 13 7 PTA 11 DNA	40 38 61 48 43 47 71 40 25 44 48 36 54 33 PTA 47 DNA	5 5 8 7 4 6 5 2 2 4 5 3 4 6 PTA DNA	9 6 14 11 10 6 10 8 3 9 10 10 8 1 PTA DNA
18	PTA	PTA	PTA	PTA	PTA	PTA	PT A	PTA
19	7	10	10	11	11	49	5	9
20	2	2	3	4	4	15	2	0
21 22	PTA PTA 10	PTA PTA	PTA PTA	PTA PTA 15	PTA PTA 14	PTA PTA 60	PTA PTA 7	PTA PTA 15
23 24 25	DNA 5	10 DNA 8	11 D NA 9	DNA 11	DNA 11	DNA 44	DNA 7	DNA 13
26	7	7	8	4	5	44	6	9
27	NT	NT	NT	NT	NT	NT	NT	NT
28	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA
29	NT	NT	NT	NT	NT	NT	NT	NT
30	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA
31	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA
32	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA
33	NT	NT	NT	NT	NT	NT	NT	NT
34	7	9	11	12	12	51	6	10
35	NT	NT	NT	NT	NT	NT	NT	NT
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
37	7	9	11	10	10	47	5	8
38	DNA	D NA	DNA	Dna	DNA	DNA	DNA	DNA
39	DNA	DNA	DNA	Dna	DNA	DNA	DNA	DNA
40	PTA	PTA	PTA	PTA	PTA	PTA	PTA	PTA
41	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
42	8	9	9	10	13	49	6	10
					trials			40

A1-A5= A trials: TotA= total of trials A1-A5; B= list B score; A Del= recall after interference;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Recall on Lists A & B

Sample	A	3	3/12 FC)LLOW-U	<u>JP</u>			
<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	<u>A5</u>	<u>TotA</u>	<u>B</u>	A Del
1	6	9	11	12	13	51 50	9	9
2 3	8	9	11	10 15	12	58		8
4	8 7	11	15 13	15 15	15 15			14
5	5	13 8	8	10	10	41		15 6
6	6	8	10	10	12.			10
7	9	12	15	15	15	63		12
			DNA	DNA	DNA			DNA
9	4	8	Q	10	11	42		9
10	5	9	9 11	11	12	40	9 8	8
11		DNA	DNA	DNA	DNA			DNA
12	DNA	DNA	DNA	DNA		DNA		DNA
13					DNA			DNA
14	6	7	8	9	9	39		4
15	6	6	6	6	9 7	31	Ō	Ô
16	10	14	14	14	15	67	14	14
			DNA		DNA		DNA	DNA
18	9	11	13	13	14	60		13
19	7	8	11	10	12	48	10	10
20	5	6			10	34	4	
					DNA		DNA	
	3	3			4		2	
23	8	11	8			48	9	9
24	DNA		DNĀ					
25			DNA				DNA	
26			DNA				DNA	
27	7				8		3	
28	_	_	_			_		_
29	7	10	11	14	15	57	15	15
30	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
31	_	_	-	_	-			_
32	NT	NT	NT	NT	NT	NT	NT	NT
33	7	8	10	10	13		5	5
34	5	8	12	11	14	50		11
35	6	8	8	9	12	43	11	11
3.6	7	13	15	15	15	65	15	15
3 <i>7</i>	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	DNA	DNA						DNA
39	7	11	10					9
40	PTA		PTA		PTA		PTA	-
41	DNA				DNA		DNA	
42					13		11	11
A1-A5=	A tr	ials:	TotA=	total	of tri	als A1-A	45 :	

A1-A5= A trials; TotA= total of trials A1-A5; B= list B score; A6= recall after interference;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Recall on Lists A & B

<u>Sample</u>	<u>A</u>	<u>.</u>	6/12 FC	OLLOW-	<u>JP</u>				
<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	<u>A5</u>	T	ot A	<u>B</u>	<u>A Del</u>
1 2	8 7	9 8	12 9	13 9	14 10		56 43	7 4	12 10
3	10	12	11	13	13		59	10	12
4 5	6 4	10 6	10 10	15 11	15 12		56 43	10 5	15 13
6	6	11	10	10	14		51	7	9
7	10	13	15	15	15		68	10	13
8	4	5	7	6	9		31	5	5
9 10	5 5	11 9	9 12	10 15	13 1 4		48 55	7 4	7 10
11	4	7	9	8	13		41	5	12
12	6	9	11	11	12		49	5	11
13	5	7	9	9	10		40	6	9
14 15	4 6	8 8	11 9	11 9	11 8		45 40	7 4	4 0
16	7	13	14	15	14		63	11	14
17	5	10	12	12	12		51	5	10
18	7	11	13	13	13		57	8	14
19	6	8	12	10	10		46	6	6
20 21	4 5	5 8	6 8	9 9	8 10		32 40	4 5	3 8
22	5 3	5	7	7	7		29	3	6
23	7	8	9	13	13		50	5	14
24	9	11	14	13	14		61	11	15
25 26	7 6	7 9	9 10	9 13	10		42	6	8
20 27	6	7	5	7	12 8		50 33	6 7	10 4
28	-	_	_	, -	_		_	_	-
29	5	8	13	15	15		56	7	14
30	6	4	4	6	5		25	0	5
31 32	4 6	5 9	4 9	5 8	6 11		24 43	3 7	0 3
33	DNA	DNA	DNA	DNA	DNA		DNA	DNA	DNA
34	5	8	11	14	14		52	7	14
35	4	7	9	11	11		42	6	10
36 37	7 6	11 9	15 8	14	14		61	8 5	13
37 38	8	13	15	8 14	8 15		39 65	5 5	6 14
39	6	13	13	15	15		62	6	11
40	5	6	6	9	8		34	4	3
41	5	11	12	15	14		57	5	14
42 11-15=	7 1 tr	ials:	12 Tot A=	14	13 of tria	ale	55 ג <u>ו</u> 1 ג	.5.	12

A1-A5= A trials: TotA= total of trials A1-A5; B= list B score; A6= recall after interference;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

<u>Sample</u>	<u>A</u>	12	2/12 FC	DLLOW-U	LLOW-UP					
<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	<u>A5</u>	<u>TotA</u>	<u>B</u>	A Del		
1 2 3 4 5 6 7 8 9 10 11 12 13 14	6 9 7 8 7 14 6 DNA DNA 7 DNA 7	11 10 13 9 12 10 14 11 DNA DNA 10 DNA 8 6	13 11 14 11 14 12 15 9 DNA DNA 9	14 10 15 11 13 14 15 12 DNA DNA 12 DNA 10 8	15 13 15 13 14 13 15 10 DNA DNA 11 DNA 12	59 53 66 51 61 56 73 48 DNA DNA 49 DNA 48 37	4 5 11 6 5 8 14 6 DNA DNA 8 DNA 4 7	13 9 15 12 12 11 11 6 DNA DNA 9 DNA 9		
15 16 17 18 19 20 21 22 23 24 25 26 27 28	5 9 7 8 6 5 5 8 7 0 7 6 6	8 12 10 11 10 9 8 10 15 8	9 15 11 13 12 9 10 10 12 15 11	10 14 14 14 11 9 8 11 14 15 11	11 15 14 14 13 8 12 14 13 15 14 14	43 65 56 60 52 40 44 51 56 70 51 53 38	2 10 9 8 3 5 3 4 8 10 4 5 7	6 14 13 12 9 3 9 12 10 14 12 10 6		
29 30 31 32 33 34 35 36 37 38 39 40 41 42	11 4 5 8 7 8 7 9 - 7 5 6 9	12 3 7 10 10 11 10 13 - 10 9 7 8 12	13 5 8 7 13 12 11 15 - 13 14 7 10	15 6 9 6 10 12 13 15 - 15 14 9 12	15 7 8 10 12 12 14 15 - 15 13 11 12	66 25 37 41 52 55 67 60 57 39 48 56	8 2 5 6 3 7 6 - 5 11 7 4 6	14 2 5 3 10 12 12 15 - 15 11 4 7 11		

A1-A5= A trials; TotA= total of trials A1-A5;

B= list B socre; A Del= recall after interference;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample	A	24	4/12 F	OLLOW-	<u>UP</u>			
<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	<u> </u>	<u>TotA</u>	<u>B</u>	A Del
1	7	12	13	14	14	60.	6	14
2 3	6 10	10 15	8 15	11 15	11	46	7	7
4	DNA	15 D NA	DNA	DNA	15 DNA	70 D NA	13 DNA	14 DNA
5	8	12	11	15	15	61	5 5	15
6	6	13	14	14	15	62	6	13
7	6	15	15	15	15	66	13	14
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
10	5	9	12	11	13	15	4	9
11	5	11	14	14	15	59	6	12
12	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
13 1 4	DN A 7	DNA	DNA	DNA	DNA	DNA	DNA	DNA
14 15	9	6 9	7 8	7 10	6 10	33 46	6 4	0 5
16	9	13	15	15	15	67	9	15
17	6	12	12	14	14	58	9	12
18	5	8	10	10	14	47	4	12
19	4	9	10	11	14	48	6	11
20	8	11	12	12	11	54	7	8
21	4	7	8	11	13	43	2	10
22	7	9	7	9	9	41	4	8
23	10	10	11	13	15	59	13	13
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
25	5	8	10	10	12	45	5	9
26	6	10	10	12	12	50 50	7	11
27 28	7 DNA	11 DNA	10	12 DNA	12	52	6 DN3	6 DNA
26 29	DNA DNA	DNA DNA	DNA DNA	DNA	DNA DNA	DNA DNA	DNA DNA	DNA DNA
30	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
31	2	5	7	8	7	29	5	5
32	8	10	, 9	8	12	47	7	4
33	6	6	10	7	9	38	6	7
34	7	12	13	15	15	62	8	15
35	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	7	11	14	15	14	61	7	13
39	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
41	6 DNA	7 DNA	9 DN3	10	13	45 DNA	6 DN3	12
42	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA

A1-A5= A trials; TotA= total of trials A1-A5; B= list B score; A Del= recall after interference;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample	ole A 36/12 FOLLOW-UP							
<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	<u>A5</u>	<u>TotA</u>	B	A Del
1	8	12	15	15	15	65	_ 9	12
2	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
3	13	14	13	15	15		12	15
4	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
5	5		13	15	15		7	14
6	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
7	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
9	DNA	DNA	DNA	DNA	DNA	DNA	DNA	
10	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
11	6 DN3			12	14		6	12.
12	DNA	DNA	DNA		DNA	DNA	DNA	DNA
13	7 DNA	9 DMA			13		6 DNA	12
14 15	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
16	6 DNA	8 DNA	7 DNA	11 DNA	9 DNA	41 DNA	7 DNA	1 DNA
10 17			DIVA -		DNA -	DINA -	DNA –	DNA -
18	– 9	- 9	11		13		- 7	6
19	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
20	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
21	3	7	10	8	11		4	9
22	DNA	DNÁ	DNA	DNA		DNA	DNA	DNA
23	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
24	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
25	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
26	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
27	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
28	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA		DNA
31	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
32	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
33	-	-	_	_		-	_	_
34	6	10	14	14	14	57	6	12
35	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
38	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
39	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA
41	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA:
42	DNA	DNA	DNA	DNA	DNA	DNA	DNA	DNA

A1-A5= A trials: TotA= total of trials A1-A5; B= list B score; A Del= recall after interference;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Interference & Recognition Scores

Sample	<u>A</u> <u>1/</u>	12 FOL	LOW-UF	<u>.</u>	<u>3/1</u>	2 FOLL	OW-UP
<u>Subj.</u>	Pro%	Ret%	Reco	<u>F+</u>	Pro%	Ret%	Reco F+
1	0	18	15	0	33	31	15 0
2	0	40	14	0	0	33	14 0
3	0	6	15	0	0	6	15 0
4	0	15	15	0	0	0	15 0
5	20	9	11	4	20	40	11 1
6	0	46	15	0	0	17	15 0
7	55	33	15	0	0	20	15 0
8	40	20	15	0	0	44	13 0
9	50	50	15	0	DNA	DNA	DNA DNA
10	20	25	15	0	0	39	12 0
11	0	29	15	0	DNA	DNA	DNA DNA
12	40	0	11	0	DNA	DNA	DNA DNA
13	43	39	15	0	DNA	DNA	DNA DNA
14	14	86 DT3	12	1	0	56	12 1
15	PTA	PTA	PTA		33	98	8 6
16	16	36	11	0	30	7 Data	15 0
17	DNA	DNA	DNA PTA		D NA 11	DNA	DNA DNA
18	PTA 29	PTA 18	12	P1A 0	0	7 17	15 0 15 0
19 20	NT	NT	NT	NT	0	60	15 0 9 0
20	PTA	PTA	PTA		DNA	DNA	DNA DNA
22	PTA	PTA	PTA		66	50	12 5
23	30	7	15	0	0	18	14 2
24	DNA	D NÄ	DNA		DNA	DNA	DNA DNA
25	0	18	15	0	DNA	DNA	DNA DNA
26	14	18	15	ŏ	DNA	DNA	DNA DNA
27	NT	NT	NT	NT	57	63	13 0
28	PTA	PTA	PTA			_	
29	NT	NT	NT	NT	0	0	15 1
30	PTA	PTA	PTA		PTA		PTA PTA
31	PTA	PTA	PTA			•••	
32	PTA	PTA	PTA	PTA	NT	NT	NT NT
33	NT	NT	NT		43	62	15 0
34	14	17	15	0	0	21	15 1
35	NT	NT	NT	NT	0	8	15 0
36	DNA	DNA	DNA	DNA	29	0	15 0
37	29	20	15	0	DNA	DNA	DNA DNA
38	DNA	DNA	DNA	DNA	DNA	DNA	DNA DNA
39	DNA	DNA		DNA	28	68	13 0
40	PTA	PTA	PTA		PTA	PTA	PTA PTA
41	DNA	DNA		DNA	DNA	DNA	DNA DNA
42	25	23	14	0	29	15	15 0

Pro%= Proactive Interference; Reco= Recognition; Ret%= Retroactive Interference; F+= False positives;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Interference & Recognition Scores

Sample 1	<u>A 6/1</u>	2 FOLL	OW-UP		12/	12 FOL	LOW-U	-
<u>Subj.</u>	Pro%	Ret%	Reco	<u>F+</u>	Pro%	Ret%	Reco	<u>F+</u>
1	13	14	15 15	0	33	13	15	0
2 3	43	0 8	15 15	0 0	44 0	31	14 15	0
4	0 0	0	15	0	14	0 8	15	0 0
5	0	0	15	Ö	38	14	15	0
6	ő	36	15	Ö	0	15	15	Ö
7	Ŏ	13	15	Ö	Õ	27	15	ŏ
8	Ö	40	14	Ŏ	_	_	_	_
9	Ō	46	15	Ō	DNA	DNA	DNA	DNA
10	20	29	13	0	DNA	DNA	DNA	DNA
11	0	8	14	0	0	18	15	0
12	16	8	15	0	DNA	DNA	DNA	DNA
13	0	10	15	1	43	25	15	0
14	0	64	15	0	0	71	14	1
15	33	98	15	2	60	46	12	1
16	0	0	15	0	0	7	15	0
17	0	17	15	0	0	7	15	0
18	0	0	15	0	0	14	15	0
19	0	40	15	0	50	31	10	0
20	0	63	11	0	0	63	10	1
21	0	20	13	3	40	25	15	0
22 23	0 29	14 0	14 15	. 0	50 0	14	15	0
23 24	0	0	15	0	0	23 7	14 15	0 0
25	14	20	15	0	43	14	15	0
26	0	17	15		17	29	15	2
27	ŏ	50	15	1 2	0	25	15	Õ
28	_	_	_	_		_	_	_
29	0	7	15	0	27	7	15	0
30	98	0	13	2	50	29	14	2
31	25	98	9	3	0	38	14	1
32	0	73	13	0	25	70	13	0
33	DNA	DNA		DNA	57	17	15	0
34	0	0	15	0	13	Ó	, 15	0
35	0	9	15	0	0	14	15	0
36	0	7	15	0	33	0	15	0
37	17	25	15	0	_	_	_	_
38	38	7	15	0	29	0	15	0
39 40	0	26	15	0	0	15	15	0
40 41	20 0	63 0	15 15	0	0	64	14	1
42	14	8	15 15	0 0	33 33	42 15	13	1 0
74	Τ.44	0	ΤЭ	U	55	10	15	U

Pro%= Proactive Interference; Reco= Recognition:
Ret%= Retroactive Interference; F+= False positives;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Interference & Recognition Scores

Sample	<u>A</u> 24/	12 FOL	<u>LOW</u> -UI	<u> </u>	<u>36</u>	/12 FO	LLOW-UP
<u>Subj.</u>	Pro%	Ret%	Reco	<u>F+</u>	Pro%	Ret%	Reco F+
1	14	0	15	0	0	20	1 5 0
2	Ó	36	14	0	DNA	DNA	DNA DNA
3	0	7	15	0	8	0	1 5 0
4	DNA	DNA		DNA	DNA	DNA	DNA DNA
5	38	0	15	0	0	7	15 0
6	0	13	15	0	D NA	DNA	DNA DNA
7	0	7	15	0	DNA	DNA	DNA DNA
8	DNA	DNA		DNA	DNA	DNA	DNA DNA
9	DNA	DNA		DNA	DNA	DNA	DNA DNA
10	20	31	13	0	DNA	DNA	DNA DNA
11	0	0	15	0	0	14	15 0
12	DNA	DNA		DNA	DNA	DNA	DNA DNA
13	DNA	DNA		DNA	14	8	15 0
14	14	98 50	11	1	DNA	DNA	DNA DNA
15	56	50	15	0	0	89	14 1
16	0	0	15	0	DNA	DNA	DNA DNA
17	0	14	15	0	30	13	15 0
18	20	14	15	1	22	54	15 0
19 20	0 13	21 27	14	2 0	DNA	DNA DNA	DNA DNA
20 21	50	27	14 14	1	DNA O	DNA	DNA DNA 14 0
22	43	11	15	0	DNA	18 DNA	14 0 DNA DNA
23	0	13	15	0	DNA	DNA	DNA DNA
24	DNA	DNA		DNA	DNA	DNA	DNA DNA
25	0	25	15	0	DNA	DNA	DNA DNA
26	ő	8	15		DNA	DNA	DNA DNA
27	14	50	15	0	DNA	DNA	DNA DNA
28	DNA	DNA		DNA	DNA	DNA	DNA DNA
29	DNA	DNA		DNA	DNA	DNA	DNA DNA
30	DNA	DNA		DNA	DNA	DNA	
31	0	29	12	3	DNA	DNA	DNA DNA
32	30	67	14		DNA	DNA	DNA DNA
33	0	22	15	0	DNA	DNA	
34	0	0	15	0	DNA	DNA	DNA DNA
35	DNA	DNA	DNA	DNA	DNA	DNA	DNA DNA
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA DNA
37	DNA	DNA	DNA	DNA	DNA	DNA	DNA DNA
38	0	7	15	0	DNA	DNA	DNA DNA
39	DNA	DNA		DNA	DNA	DNA	DNA DNA
40	DNA	DNA		DNA	DNA	DNA	DNA DNA
41	0	8	14	0	DNA	DNA	DNA DNA
42	DNA	DNA	DNA	DNA	DNA	DNA	DNA DNA

Pro%= Proactive Interference; Reco= Recognition; Ret%= Retroactive Interference; F+= False positives;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample	<u>B</u>	<u>24</u>	/12 FO	LLOW-U	<u>P</u>			
<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u> 14</u>	<u> A5</u>	<u>TotA</u>	В	A Del
1	8	12	13	14	14	61	5	14
2	6	7	9	11	11	44	5	12
3	5	8	10	11	14	48	6	12
4	7	9	8	9	12	43	8	10
5	7	11	11	13	15	57	6	12
6	5	4	3	5	7	24	3	4
7	7	10	12	12	13	54	5	13
8	3	5	6	4	6	24	1	2
9	_	_	-	_	_	_	_	_
10	5	7	9	6	7	34	3	5

Recall on Lists A & B

36/12 FOLLOW-UP

<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	<u>A5</u>	<u>TotA</u>	<u>B</u>	A Del
1	11	14	15	15	15	70	7	15
2	7	12	10	13	13	55	5	13
3	8	8	10	11	13	50	12	15
4	6	9	11	9	10	45	8	9
5	9	11	13	12	13	58	6	13
6	3	6	5	6	7	27	3	5
7	6	10	12	13	13	54	8	13
8	3	5	4	5	5	22	3	0
9	7	13	15	15	15	65	10	14
10	4	6	7	5	7	29	2	4

A1-A5= A trials: TotA= total of trials A1-A5: B= list B score: A Del= recall after interference:

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Interference & Recognition Scores

Sample	B 24/	12 FOL	LOW-UP		36/	12 FOL	LOW-UP	
<u>Subj.</u>	Pro%	Ret%	Reco	<u>F+</u>	Pro%	Ret%	<u>Reco</u>	<u>F+</u>
1	38	0	15	0	36	0	15	0
2	17	9	15	1	29	0	15	0
3	0	14	15	0	0	8	15	0
4	0	16	12	0	0	10	14	0
5	14	20	15	0	33	0	15	0
6	40	43	11	1	0	28	14	0
7	29	0	15	0	0	0	15	0
8	66	33	6	3	0	98	10	6
9	_	_	_	_	0	7	15	0
10	40	29	13	2	50	43	12	3

Pro% = Proactive Interference; Reco = Recognition
Ret% = Retroactive Interference; F+ = False positives

TABLE C8.2: RAW DATA FOR DIGIT SPAN AT EACH FOLLOW-UP SAMPLES A & B

Sample A	1/12 FOLLOW-UP			_3/1	3/12 FOLLOW-UP		
Subj.	DIG F	DIG B	TOT	DIG F	DIG B	TOT	
1	7	. 3	10	8	5	13	
2	8	7	15	9	8	17	
3	7	5	12	9	4	13	
4	8	4 2	12 6	9 5	7	16 9	
5 6	4 6	6	12	7	4 5	12	
7	9	8	17	, -	- -	12	
8	5	4	9	DNA	DNA	DNA	
9	4	2	6	6	4	10	
10	7	5	12	7	5	12	
11	6	3	9	DNA	DNA	DNA	
12	6	4	10	DNA	DNA	DNA	
13	9	· 6	15	DNA	DNA	DNA	
14	7	4	11	8	5	13	
15	PTA	PTA	PTA	7	3	10	
16	5	4	9	6	7	13	
17 18	DNA PTA	DNA	DNA	DNA	DNA	DNA	
19	6 FIA	PTA 4	PTA 10	· 8	5 4	13 10	
20	NT	NT	TN	7	5	12	
21	DNA	DNA	DNA	DNA	DNA	DNA	
22	DNA	DNA	DNA	3	1	4	
23	6	7	13	7	6	13	
24	DNA	DNA	DNA	DNA	DNA	DNA	
25	4	. 3	7	DNA	DNA	DNA	
26	5	4	9	DNA	DNA	DNA	
27	NT	NT	NT	8	4	12	
28	PTA	PTA	PTA	_	_	-	
29	NT	NT	NT	6	5	11	
30	PTA PTA	PTA	PTA	PTA	PTA	PTA	
31 32	PTA	PTA PTA	PTA PTA	NT.	TN	NT	
33	NT	NT	NT	8 14.1	5	13	
34	6	5	11	7	4	11	
35	NT	NT.	NT	8	6	14	
36	DNA	DNA	DNA	8	7	15	
37	8	5	13	DNA	DN A	DNA	
38	DNA	DNA	DNA	DNA	DNA	DNA	
39 .	DNA	DNA	DNA	-	_	_	
40	PTA	PTA	PTA	PTA	PTA	PTA	
41	DNA	DNA	DNA	DNA	DNA	DNA	
42	7	5	12	7	7	14	

DIG F= digit forward;
TOT= total digit span;
NT= subject not tested, poor physical/cognitive state;

TABLE C8.2: RAW DATA FOR DIGIT SPAN AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample A	6/12	FOLLOW-	<u>-UP</u>	12/1	12/12 FOLLOW-UP		
<u>Subj.</u>	DIG F	DIG B	TOT	DIG F	DIG B	TOT	
1	8	5	13	8	, 5	13	
2	9	7	16	_	_	_	
3	8	4	12	6	6	12	
4	9	4	13	9	8	17	
5	5	5	10	6	4	10	
6	8	6	14	7	6	13	
7	8	7	15	9	8	17	
8 9	5 5	5 4	10 9	באנא.	ראים.	ר ואנד	
10	7	4	11	DNA DNA	DNA DNA	DNA DNA	
11	7	3	10	6	4	10	
12	7	6	13	DNA	DNA	DNA	
13	7	6	13	8	5	13	
14	7	5	12	8	6	14	
15	7	4	11	8	4	12	
16	7	6	13	7	7	14	
17	8	7	15	6	7	13	
18	8	5	13	7	6	13	
19	5	4	9	6	4	10	
20	7	3	10	6	4	10	
21	4	4	8	5	4	9	
22	5	3	8	5	5	10	
23	8	7	15	6	7	13	
24	8	6	14	8	8	16	
25 26	4	3	7	5	4	9	
26 27	6 6	4 6	10 12	7 7	5 5	12 12	
28	-	-	12	/ -	- -	12	
29	7	6	13	7	4	11	
30	8	5	13	6	6	12	
31	7	3	10	7	3	10	
32	8	4	12	7	6	13	
33	DNA	DNA	DNA	8	7	15	
34		5	10	7	4	11	
35	5 8	6	14	8	6	14	
36	7	6	13	8	7	15	
37	8	5	13	_	_		
38	9	8	17	9	7	16	
39	_	_	-	_	-	-	
40	5	3	8	7	3	10	
41	7	6	13	6	6	12	
42	7	5	12	6	4	10	

DIG F= digit forward; DIG B= digit backward; TOT= total digit span; PTA= subject still in PTA: NT= subject not tested, poor physical/cognitive state;

TABLE C8.2: RAW DATA FOR DIGIT SPAN AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample A	24/12 FOLLOW-UP			36/12 FOLLOW-UP		
<u>Subj.</u>	DIG F	DIG B	<u>TOT</u>	DIG F	DIG B	TOT
1	7	5	12	7	5:	12
2	8	6	14	_	-	_
3	8	4	12	7	6.	13
4	D NA	DNA	DNA	DNA	DNA	DNA
5	7	6	13	6	4	1'0
6	7	5	12	DNA	DNA	DNA
7	7	7	14	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA
10	6	5	11	DNA	DNA	DNA
11	6	4	10	7	5	12
12	DNA	DNA	DNA	DNA	DNA	DNA
13	DNA	DNA	DNA	8	6	14
14	8	6	14	DNA	DNA	DNA
15	8	4	12	7	4	11
16	8	7	15	DNA	DNA	DNA
17	9	8	17	8	フ	15
18	6	5	11	8	4	12
19	6	4	10	DNA	DNA	DNA
20	7	5	12	DNA	DNA	DNA
21	5	5	10	6	3	9
22	6	3	9	DNA	DNA	DNA
23	8	7	15	DNA	DNA	DNA
24	DNA	DNA	DNA	DNA	DNA	DNA
25	5	5	10	DNA	DNA	DNA
26	7	4	11	DNA	DNA	DNA
27	7	5	12	DNA	DNA	DNA
28	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA
31	6	2	8	DNA	DNA	DNA
32	7	4	11	DNA	DNA	DNA
33	9	7	16	DNA	DNA	DNA
34	7	6	13	7	5	12
35	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA
38	9	7	16	DNA	DNA	DNA
39	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA
41	6	6	12	DNA	DNA	DNA
42	DNA	DNA	DNA	DNA	DNA	DNA

DIG F= digit forward; DIG B= digit backward; TOT= total digit span; PTA= subject still in PTA; NT= subject not tested, poor physical/cognitive state;

TABLE C8.2: RAW DATA FOR DIGIT SPAN AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample B	24/12 FOLLOW-UP			<u>36/12</u>	36/12 FOLLOW-UP			
Subj.	DIG F	DIG B	TOT	DIG F	DIG B	TOT		
1 2	6 7	4 5	10 12	6 7	5 5.	11 12		
3 4 5	4 6 7	4 5 5	8 11 12	4 5 7	5 5 6	9 10 13		
6 7	5 8	5 4	10 12	5 5	4 4	9		
8 9	4 -	3	7 -	5 7	5 7	10 14		
10	4	2	6	4	3	7		

DIG F= digit forward; DIG B= digit backward; TOT= total digit span

TABLE C8.3: RAW STEN DATA FOR WECHSLER MEMORY SCALE FACTORS, SAMPLES A & B

Sample A	6/1	2 FOLLOW	<u>-UP</u>	24/12 FOLLOW-UP		
Subj.	<u>F_1</u>	<u>F 2</u>	<u>F_3</u>	<u>F 1</u>	<u>F 2</u>	<u>F_3</u>
Subj. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37	F 1 9.0289600080 - 6499987675910 86 63.46DN 99.8	7.3 8.4 7.2 7.9 3.1 8.1 8.8	4.9 6.5 5.7 6.5 4.2 5.7 6.5 5.7 6.5	F 1 10.0 6.6 10.0 DNA 9.6 10.0 DNA DNA 6.5 7.6 DNA DNA DNA - 4.1 10.0 - 5.5 8.8 6.4 DNA 10.0 - DNA 10.0 - DNA	F 2 7.5 8.4 7.0 8.4 7.4 DNA 5.7 DNA 6.4 - 4.1 5.4 DNA DNA 5.7 DNA 6.4 - 4.1 5.4 DNA DNA DNA DNA DNA DNA DNA DNA DNA DNA	6.5 6.5 5.7 DNA 5.7 6.5 DNA DNA 5.7 6.5 DNA 1.1 4.9 4.9 4.9 1.1
38 39	9.5 8.6	9.7 7.2	6.5 5.7	9.3 DNA	9.3 DNA	6.5 DNA
40 41 42	5.0 7.0 3.8	3.1 7.5 7.0	4.2 6.5 6.5	DNA 9 . 6 DNA	DNA 7.5 DNA	DNA 6.5 DNA

F 1= Factor 1: F 2= Factor 2: F 3= Factor 3:

TABLE C8.3: RAW STEN DATA FOR WECHSLER MEMORY SCALE FACTORS, SAMPLES A & B

Sample B	24/	12 FOLLO	V-UP	<u>36/1</u>	2 FOLLO	W -UP
<u>Subj.</u>	<u>F 1</u>	<u>F 2</u>	<u>F 3</u>	<u>F 1</u>	<u>F_2</u>	<u>F 3</u>
1 2	9.1	6.3	5.7 -	-	_	_
3 4	8.7 9.3	4.2 6.7	4.9 5.7	9.7 10.0	4.5 6.3	5.7 5.7
5 6	_ _	-	_	9.0 6.5	7.6 4.5	5.7 5.9
7 8	9.4 3.6	7.5 2.1	6.5 1.9	9.0 4.5	6.1 5.4	6.5 5.7
9 10	2.8	3.2	_ 2.6	10.0 2.6	8.8 3.4	6.5 4.9

F 1= Factor 1: F 2= Factor 2: F 3= Factor 3:

TABLE C8.4: RAW DATA FOR NATIONAL ADULT READING TEST & SUBJECTIVE MEMORY SCALE, SAMPLES A & B

	Sample A					Sample B		
	<u>NA</u>]	<u>RT</u>	<u>SMQ</u>	<u>NA</u>	<u>RT</u>	SMO		
<u>Subj</u> .	VIQ	PIQ	<u>24m FU</u>	VIQ	PIQ	24m FU		
Subj. 123456789011231451678901232222222233333333333441	VIQ	PIQ	136 136 136 136 137 162 DNA 103 140 DNA 103 140 DNA 117 74 128 137 114 125 142 - 135 DNA 121 168 - DNA DNA DNA 121 168 - DNA	VIQ 107 108 102 103 85 109 - 113 90	PIQ 108 109 105 105 113 93 109 - 113 96	24m FU 142 146 139		
42	_	_	_					

NART= National Adult Reading Test; PIQ= performance IQ; SMQ= Subjective Memory Questionnaire; VIQ= verbal IQ: DNA= did not attend:

APPENDIX C9: GROUP MEMORY TEST SCORES

TABLE C9.1a: MEAN & SD, REY VARIABLES, SAMPLE A Recall Scores on List A Trials

1/12 FU	1	2	3	4	5	<u>Total</u>
A Mean: (n=23)SD: M/M Mean: (8) SD: S Mean: (7) SD: VS Mean: (6) SD: ES Mean: (2) SD:	6.0 2.1 6.1 1.9 5.3 0.9 6.5 3.1 7.5	8.0 2.5 8.5 1.7 7.1 1.6 8.7 3.9 7.5	9.4 2.6 10.3 1.9 9.1 1.6 9.0 3.8 8.5 2.5	10,0 3.0 11.1 1.6 9.0 2.9 10.3 4.1 8.5 1.5	10.8 2.9 12.0 1.5 10.3 3.2 10.7 3.5 8.5 1.5	45 11.4 48 7.4 42.7 7.5 45.2 17.7 40 7.0
<u>3/12 FU</u>	•					
A Mean: (25) SD: M/M Mean: (5) SD: S Mean: (7) SD: VS Mean: (9) SD: ES Mean: (4) SD:	6.6 1.6 6.6 1.0 5.9 1.1 7.4 1.4 6.0 2.1	9.0 2.6 8.8 1.2 9.1 2.7 9.9 2.3 6.8 2.9	10.2 3.0 12.0 1.6 10.1 3.0 10.4 2.5 7.8 3.4	10.8 3.1 12.2 1.7 11.1 2.7 11.0 2.7 7.8 3.7	12.0 2.8 13.4 1.0 11.9 2.4 12.9 1.7 8.5 3.6	48.9 12.0 53.0 5.7 49.7 11.3 51.3 9.5 36.8 15.6
6/12 FU						
A Mean: (40) SD: M/M Mean: (11) SD: S Mean: (10) SD: VS Mean: (9) SD: ES Mean: (10) SD:	5.9 1.6 6.6 1.7 5.7 1.2 6.0 1.9 5.2	8.7 2.4 9.2 1.6 9.4 2.1 8.9 3.1 7.3 2.1	10.1 2.9 11.4 1.4 10.5 2.8 10.6 3.1 7.9 2.7	11.1 3.0 12.1 2.0 11.7 2.8 12.0 3.2 8.5 2.2	11.6 2.7 12.4 1.6 13.0 2.0 12.2 2.6 8.7 2.4	47.4 11.1 51.6 6.5 50.3 9.1 49.7 12.8 37.6 9.5

A1-A5= A trials:

Total = total of trials A1-A5:

TABLE C9.1a: MEAN & SD, REY VARIABLES, SAMPLE A (cont)

Recall Scores on List A Trials

12/12 FU	1	2	3	4	5	Total
A Mean: (n=37)SD: M/M Mean: (10) SD: S Mean: (8) SD: VS Mean: (10) SD: ES Mean: (9) SD:	7.3 1.9 7.5 1.4 7.1 0.9 8.2 2.5 6.1 1.5	9.8 2.3 10.6 2.2 10.1 1.7 10.7 1.5 7.7 2.2	11.2 2.5 12.0 1.5 11.6 2.2 12.2 2.1 8.7 2.2	11.7 2.6 12.5 1.8 12.6 1.9 12.6 2.1 9.0 2.4	12.4 2.7 13.5 1.2 12.9 2.2 12.8 2.2 10.4 2.5	52.4 10.3 56.1 6.9 54.4 8.3 56.5 9.0 41.9 9.2
24/12 FU						
A Mean: (26) SD: M/M Mean: (7) SD: S Mean: (5) SD: VS Mean: (8) SD: ES Mean: (7) SD:	6.5 1.9 6.4 1.8 7.8 1.6 7.4 1.6 6.0 2.3	9.9 2.4 10.7 2.6 10.0 2.3 9.6 2.9 7.7	10.7 2.5 11.7 2.0 11.2 2.3 10.4 3.4 8.0 1.1	11.7 2.6 12.7 2.1 11.6 2.7 11.3 3.3 9.0 1.3	12.5 2.5 13.9 1.0 12.2 2.4 12.1 2.9 10.1 2.8	51.3 10.1 55.4 8.9 52.8 10.1 50.8 13.2 40.9 6.6
36/12 FU						
A Mean: (10) SD:	7.3 2.7	10.7 2.3	11.9 2.3	12.9 2.3	13.4 1.9	

TABLE C9.1b: MEAN & SD, REY VARIABLES, SAMPLE B

Recall Scores on List A Trials

	1	2	3	4	5	<u>Total</u>
24m Mean: (10) SD:		8.1 2.5	9.0 2.9		11.0 3.3	
36m Mean: (10) SD:		9. 4 3.0	10.2 3.7	10.4 3.7	11.1 3.4	

A1-A5= A trials:

Total = total of trials A1-A5;

TABLE C9.2a: MEAN & SD, MORE REY VARIABLES, SAMPLE A
Recall of List B. & Interference & Recognition Scores

<u>1/12 FU</u>	<u>B</u>	A Del	Pro%	Ret%	Reco	<u>F+</u>
A Mean: (23) SD: M/M Mean: (8) SD: S Mean: (7) SD: VS Mean: (6) SD: ES Mean: (2) SD:	5.0 16 5.5 1.5 4.9 1.6 4.3 1.8 5.5	8.5 3.5 10.4 1.9 8.3 2.6 7.7 4.5 4.5 3.5	19.1 17.5 18.9 16.8 14.9 16.7 23.5 20.3 21.5	29.2 23.1 17.4 10.8 27.4 14.4 39.0 28.6 53.0 33.0	13.5 3.1 14.0 1.5 14.4 1.4 11.8 5.1 13.5 1.5	0.2 0.8 0.0 0.0 0.6 1.4 0.0 0.0 0.5
3/12 FU						
(4) SD:	1.5 7.2 2.7	9.1 4.2 11.0 1.7 9.4 4.1 9.7 3.5 4.8 5.0	16.2 19.6 12.4 15.2 18.7 19.5 11.2 16.3 27.5 25.2	30.2 25.5 18.0 8.2 25.3 21.5 30.7 24.8 52.8 32.2	13.7 2.0 15.0 0.0 13.5 1.6 13.9 1.9 11.8 2.5	0.7 1.5 0.2 0.4 0.1 0.4 0.3 0.7 3.0 2.6
6/12 FU A Mean:	6.1	9.3	9.5	23.5	14.5	0.4
(40) SD: M/M Mean: (11) SD: S Mean: (10) SD: VS Mean: (9) SD: ES Mean: (10) SD:	2.2 6.7 1.9 6.4 1.7 6.4 2.4 4.6	4.3 11.2 2.6 10.7 3.2 10.4 3.8 4.9 3.9	18.5 5.2 6.9 5.8 12.3 11.4 15.3 19.3 28.8	26.6 11.4 11.1 20.0 17.9 17.6	1.2 15.0 0.0 14.7 0.6 14.4 1.3 13.7	1.8 0.1 0.3 0.3 0.6 0.0 1.0

B= List B score; A Del= Recall after Interference: Pro%= Proactive Interference: Reco= Recognition: Ret%= Retroactive Interference: F+= False Positives:

TABLE C9.2a: MEAN & SD, MORE REY DATA, SAMPLE A (cont)

Recall of B, & Interference & Recognition Scores

				_		
12/12 FU	<u>B</u>	A Del	Pro%	Ret%	Reco	<u>F+</u>
(n=37)SD: M/M Mean: (10) SD: S Mean: (8) SD: VS Mean: (10) SD:	2.7 6.2 2.8 6.3 1.2 7.7 3.1 4.9	11.5 2.4 11.3 2.8 10.0 3.2 6.1			14.4 1.3 14.3 1.6 15.0 0.0 14.2 1.6 14.0 0.9	0.3 0.6 0.1 0.3 0.7 0.1 0.3 0.7
24/12 FU						
(26) SD: M/M Mean: (7) SD: S Mean: (5) SD: VS Mean: (8) SD: ES Mean:	7.6 2.6 8.4 2.5 7.0 2.8 4.6	3.8 12.4 1.9 10.0 3.4	16.8 2.0 4.9 2.6 5.2 13.1 15.9 28.0	22.1 22.7 10.7 9.0 19.6 12.3 20.1 14.5 41.7 29.5	14.4 1.0 14.7 0.5 14.6 0.5 14.3 1.3	0.3 0.7 0.3 0.7 0.0 0.0 0.1 0.3 0.9
36/12 FU						
	7.1 2.0		8.2 10.7			

TABLE C9.2b: MEAN & SD, MORE REY VARIABLES, SAMPLE B

Recall B, & Interference & Recognition Scores

24/12 FU:	В	A Del	Pro%	Ret%	Reco	<u>F+</u>
B Mean: (10) SD:	4.7 1.9	9.3 4.2	27.1 20.3	18.2 13.8	13.0 2.9	0.8 1.0
36/12 FU: B Mean: (10) SD:	6.4 3.1	10.1 5.1	14.8 18.8	19.4 29.5	14.0 1.6	0.9 1.9

B= List B score; A Del= Recall after Interference; Pro%= Proactive Interference; Reco= Recognition;

Ret%= Retroactive Interference; F+= False Positives:

TABLE C9.3: T-TESTS, REY AVLT, SAMPLE A

Recall Scores on List A trials

1/12 FU	<u>J</u> :		<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
M/M(8) M/M S	V	S(7) VS(6) VS	1.120 <1 <1	1.637 <1 <1	1.227 <1 <1	1.707 <1 <1
M/M	~	S(7) VS(9) ES(4) VS ES ES		<1 1.181 1.346 <1 1.362 1.937*	1.383 1.421 2.348* <1 1.179 1.437	<1 1.012 2.221* <1 1.607 1.579
M/M) v v v v	S(10) VS(9) ES(10) VS ES ES	<1	1.493 <1 2.300** <1 2.232** 1.291	<1 <1 2.653*** <1 2.113** 1.997*	<1 <1 3.919**** <1 2.867*** 2.749***
	V V V V V		<1 <1 2.087* 1.247 1.678 2.209**	<1 <1 2.894*** <1 2.590** 3.470****	<1 <1 3.879**** <1 2.769*** 3.617***	<1 3.48****
	v v v v		1.403 1.090 <1 <1 1.612 1.345	<1 <1 2.568** <1 1.907* 1.600	<1 <1 4.368**** <1 2.879*** 1.878*	<1 1.041 3.95**** <1 2.022* 1.790*
	_					

TABLE C9.3: T-TESTS, REY AVLT, SAMPLE A (cont)

Recall Scores on Lists A & B

1/12 FU	<u>[</u> :	<u>A5</u>	Total A	<u>B</u>	A Delay
M/M	v S(7) v VS(6) v VS		< 1	<1 1.290 <1	1.744 1.383 <1
M/M M/M S	: v S(7) v VS(9) v ES(4) v VS v ES v ES	<1 2.612** <1 1.657	<1 1.460	1.720 <1 1.365 2.218** <1 1.562	<1 <1 <1 2.409** <1 1.598 1.789
M/M M/M S S	: V S(10) V VS(9) V ES(10) V VS V ES V ES	<1 4.104**** <1 4.433****	<1 <1 3.996**** <1 3.049*** 2.314**	<1 2.028*	<1 <1 4.300**** <1 3.632**** 3.141***
M/M M/M S S	U: v S(8) v VS(10) v ES(11) v VS v ES	<1 3.288**** <1 2.111**	3.782**** <1 2.949****		<1 3.21****
M/M M/M S S	U: v S(10) v VS(8) v ES(7) v VS v ES	1.591	<1 3 460****	<1 <1 2.679** <1 3.057**** 2.139*	1.461 1.610 3.88**** <1 1.806 1.921*
*=p< .05	**:	=p<.025;	***=p<	.01; **	**=p<.005;

TABLE C9.3: T-TESTS, REY AVLT, SAMPLE A (cont)

Interference & Recognition Scores

1/12 FU	<u>!</u> :	Pro%	Ret%	Reco	<u>F+</u>
M/M	v S(7) v VS(6) v VS	<1 <1 <1	1.515 1.760 <1	<1 1.018 1.220	1.219 <1 1.219
M/M M/M S S	L: V S(7) V VS(9) V ES(4) V VS V ES V ES	<1 <1 1.054 <1 <1 1.186	1.403 1.403 2.102* <1 1.522 1.219	2.067* 1.271 2.915** <1 1.424 1.540	<1 <1 2.415** <1 2.229* 2.061*
M/M M/M S S	Y S(10) V VS(9) V ES(10) V VS V ES V ES	< 1	1.313 <1 2.861**** <1 1.996* 2.096*	1.663 1.540 2.402** <1 1.662 1.056	<1 <1 2.221** 1.496 1.561
M/M M/M S S	U: V S(8) V VS(10) V ES(11) V VS V ES V ES	1.145	<1 1.072 3.131**** 1.401 3.457**** 2.066*	1.230 <1 <1 1.334 3.131****	<1 <1 2.345** <1 <1 2.261**
M/M M/M S	<u>U</u> : v S(10) v VS(8) v ES(7) v VS v ES v ES	<1 1.876* 3.362**** 1.727 3.232**** 1.585	< 1	<1 <1 1.705 <1 1.470	<1 <1 1.247 <1 1.984* 1.868*

TABLE C9.4: CORRELATIONS OF REY VARIABLES AT EACH FOLLOW-UP WITH U/C & PTA, SAMPLE A

Recall Scores on List A trials

		<u>A1</u>	<u>A2</u>	<u>A3</u>	<u> 14</u>
1/12 FU:	U/C:	0.37	0.10	0.49	06
(n=23)	PTA:	0.33	0.19	0.06	0.01
3/12 FU:	U/C:	0.48*	0.44*	0.42*	0.41*
(27)	PTA:	0.36	0.32	0.30	0.28
6/12 FU:	U/C:	37*	- 49**	- 44**	56**
(41)	PTA:	26	42**	52 * *	48**
12/12 FU:	U/C:	10	1 ¹ 5	18	18
(39)	PTA:	07	01	0.00	0.00
24/12 FU:	U/C:	18	42*	57**	58**
(26)	PTA:	13	52**	62**	61**
36/12 FU:	U/C:	29	78**	76**	82**
(10)	PTA:	30	74**	73*	78**

Recall Scores on Lists A & B

		<u> </u>	Total A	<u>B</u>	<u>A Del</u>
4 (40 171)	11./0	1.0	0.5	0.6	0.0
1/12 FU:		18	07	06	22
(23)	PTA:	14	. 06	08	35
3/12 FU:	U/C:	0.40*	16	0.46*	0.41*
(27)	PTA:	0.27	17	0.35	0.28
6/12 FU:	U/C:	-0.60**	58**	49**	58**
(41)	PTA:	-0.60**	52**	47**	54**
12/12 FU:	U/C:	15	49**	12	18
(39)	PTA:	0.02	41 *	0.01	0.00
24/12 FU:	U/C:	51 * *	~.55**	33	47**
(26)	PTA:	56**	59**	41*	53**
36/12 FU:	U/C:	83**	78**	36	89**
(10)	PTA:	- .79**	−.75 *	33	89**

Interference & Recognition Scores

			Pro%	Ret%	<u>Reco</u>	<u>F+</u>
1/12	FU:	U/C:	. 42*	. 25	. 08	. 09
(23)		PTA:	. 23	. 35	01	.01
3/12	FU:	U/C:	.69**	. 45*	.61**	.68**
(27)		PTA:	.46*	.41*	.49**	.58**
6/12	FU:	U/C:	. 37*	. 43**	. 14	.32*
(41)		PTA:	.55**	. 35*	. 15	. 29
12/12	FU:	U/C:	.12	. 26	. 00	.00
(39)		PTA:	.09	. 38*	. 08	. 11
24/12	FU:	U/C:	. 38	. 28	23	. 37
(26)		PTA:	.53**	. 42*	35	. 45*
36/12	FU:	U/C:	. 05	.80**	71*	.52
(10)		PTA:	01	.81**	~.69*	. 53

TABLE C9.5: CORRELATIONS OF REY VARIABLES AT EACH FOLLOW-UP WITH U/C & PTA, SAMPLE B

Recall Scores on List A trials

		<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
24/12 FU: (n=10) 36/12 FU: (10)	PTA:	24 35 23 45	24 36 47 75**	22 35 36 68*	26 39 51 74**
			cores on Li		N Del

		Recall A5	Scores on Lists Total A	A & B B	A Del
24/12 FU:	PTA:	26	32	24	27
(26)		39	57	36	41
36/12 FU:		38	41	28	33
(10)		66*	69*	45	61

Interference & Recognition Scores

		Pro%	Ret%	Reco	<u>F+</u>
24/12 FU:	U/C:	06	06	22	21
(26)	PTA:	. 05	. 00	36	31
36/12 FU:	U/C:	. 50	. 30	39	. 36
(10)	PTA:	. 24	. 63	63	. 59

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A

Recall Scores on List A trials - RT

1/12	FU:	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	33 40 42* 44* 45* 45* 46* 47*	39 48* 52* 53* 55** 55** 57**	36 46* 51* 54* 57** 56** 57**	24 37 40 43* 46* 46* 47* 49*
3/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.16 .39* .96** .96** .94** .96**	.11 .33 .94** .94** .92** .94** .92**	.10 .31 .93** .93** .91** .93**	.08 .30 .93** .93** .90** .93**
6/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	64**63**53**131156**53**	65**61**48**50**141251**47**	65**63**51**54**071058**52**	63**62**48**53**010353**49**
12/12	<u>FU</u> :				,
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	02 .07 10 10 09 09 .09	05 .05 13 13 12 12 .06 .12	05 .05 14 14 14 14 .05 .12	05 .05 14 14 14 15 .06

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A (cont)

Recall Scores on List A trials - RT

24/12	<u>FU</u> :	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	58**61**50**53**47*62**50**46*	55**48*51**52**48*50**45*46*	46* 37 42* 43* 45* 40* 36 40*	36 32 40* 42* 31 33 25 27
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	60 57 52 30 52 45 62 52	61 73* 78** 66* 66* 73* 65*	29 51 65* 52 38 42 45 43	72* 86** 94** 85** 82** 45** 84** 83**
		Recall Sco	res on List	s A & B -	RT
1/12	<u>FU</u> :	<u>A5</u>	Total A	<u>B</u>	A Del
Set	1 +:: 2 +:: 3 +:: 4 +:	3952*52*54**57**57**58**60**	40 51* 55** 58** 60** 60** 62** 63**	31 38 39 41 42* 43* 44*	- 47* - 55** - 56** - 58** - 59** - 58** - 60** - 61**
3/12	<u>FU</u> :				
Set					

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD. SAMPLE A (cont)

Recall Scores on Lists A & B - RT

6/12	<u>FU</u> :	<u>A1</u>	Total A	<u>B</u>	<u>F+</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	70** 70** 57** 63** 04 01 63** 60**	71** 70** 56** 60** 04 01 61** 57**	64**64**55**58**060460**58**	52** 53**- 43** 53** 05 03 46** 45**
12/12	<u>FU</u> :				
Set ·	1 +: -: 2 +: -: 3 +: -: 4 +: -:	04 .04 16 16 15 15 07 .12	44** 40* 54** 54** 60** 60** 33*	04 .06 09 09 08 08 .07	08 01 12 14 12 12 .02 .08
24/12	FU:				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	29 13 29 29 17 16 17	51**43*4951**43*45*39*40*	56**49*60**46*58**52**47*47*	30 18 32 31 23 20 10 13
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	41 60 69* 51 49 57 61	62 75** 81** 64* 66* 67* 75** 69*	53 46 48 34 47 44 55	18 47 54 55 31 43 39

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD. SAMPLE A (cont)

Interference & Recognition Scores - RT

1/12	<u>FU</u> :	Pro%	Ret%	Reco	<u>F+</u>	
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	18 19 24 24 24 23 22 .81	.49* .57** .64** .67** .67** .66** .67**	70**78**89**90**91**91**91**	04 03 05 05 02 02 01 03	
3/12	<u>FU</u> :					
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.58** .66** .69** .68** .72** .69** .71**	.47* .59** .55** .55** .56** .55** .54**	.19 .44 .92** .92** .94** .94** .93**	.30 .55** .92** .93** .95** .94** .95**	
6/12	FU:					
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.35* .29 .24 .30 .06 .04 .33* .25	.29 .29 .24 .36* .04 .03 .23	.60** .51** .31 .37* .03 .01 .52**	.70** .62** .41** .50** .09 .05 .62** .44**	
12/12 FU:						
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.20 .26 07 09 .14 .15 .39*	.30 .45** .15 .15 .15 .15 .16 .42**	.02 .09 .12 .12 .20 .21 .19	.07 .15 .17 .17 .27 .27 .27	

**=p<.01;

*=p<.05:

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A (cont)

Interference & Recognition Scores - RT

24/12	FU:	<u>Pro%</u>	Ret%	Reco	<u>F+</u>	
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.32 .17 .36 .21 .51** .29 .26	.25 .15 .27 .26 .19 .13 .01	60**50**58**65**39*46*41*36	53** .54** .37 .48* .42* .54** .29	
36/12	<u>FU</u> :					
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	35 38 28 07 28 20 33 27	03 .29 .36 .41 .12 .26 .20	57 71* 72* 47 65* 72* 77** 76**	14 .09 .15 .01 08 .05 .08	
		Recall Sco	ores on Lis	ts A & B -	SD	
1/12	FU:	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	35 35 37 32 39 32 46* 38	38 42 47* 44* 51* 49* 58** 52*	37 40 44 40 49 51* 53* 52*	26 37 35 32 41 41 44* 42	
<u>3/12 FU</u> :						
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.07 .25 .61** .49** .59** .61** .43*	.01 .20 .57** .45* .54** .67** .39*	.00 .19 .55** .42* .52** .55** .37	.02 .17 .54** .42* .52** .55** .35	

^{*=}p<.05; **=p<.01;

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD. SAMPLE A (cont)

Recall Scores on List A - SD

6/12	<u>FU</u> :	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	29 51** 29 42** 29 27 35* 46**	41*53**3039*36*36*42**50**	27 48** 26 31* 23 23 46** 52**	29 47** 27 41** 22 21 42** 56**	
12/12	<u>FU</u> :					
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	06 .38* 09 12 03 06 01	07 .36* 10 15 06 10 03 10	07 .36* 12 16 07 11 .02 .09	09 .34* 13 16 01 13 .03	
24/12	<u>FU</u> :					
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.19 41* 18 47* 46* 50** 32 21	10 31 42* 45* 35 41* 22 24	05 18 36 32 34 39* 23 43*	03 16 29 33 17 32 19 24	
<u>36/12 FU</u> :						
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	19 45 53 47 56 52 42 57	64* 63 61 68* 71* 78** 77**	30 29 37 46 56 55 74**	71*76**79**84**89**90**80**	

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD. SAMPLE A (cont)

Recall on Lists A & B - SD

1/12	<u>FU</u> :	<u>A5</u>	Total A	<u>B</u>	A Del		
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	42 54** 53** 49* 57** 60** 61** 59**	41 45 49* 45* 53* 55** 61**	31 28 34 30 37 41 44* 38	46**51*54**57**49*69**66**		
3/12	<u>FU</u> :						
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	03 .17 .53** .40* .50** .54** .34 .46*	50** 32 06 19 13** 08 29 01	04 .22 .58** .47* .56** .59** .41*	04 .16 .52** .38* .51** .54** .35		
6/12	<u>6/12 FU</u> :						
Set	1 +: -: 2 +: -: 3 +: -: 4 +:	41**54**32*52**31*31*56**68**	36*55**31*45**303049**60**	45**53**2740*34*36*48**51**	40*34*1842**292545**59**		
12/12 FU:							
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	06 .38* 14 17 08 11 07	26 05 53** 54** 56** 56** 42** 36*	10 .34* 08 13 03 08 02 09	08 .34* 13 16 09 12 05 12		

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH
FU WITH MEDIAN RT & SD, SAMPLE A (cont)

	Recall	on	Lists	A &	В	_	SD
--	--------	----	-------	-----	---	---	----

24/12	<u>FU</u> :	<u>A5</u>	Total A	<u>B</u>	A Del
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.20 10 10 23 01 16 05 24	.05 26 32 41* 29 40* 22 32	.35 41* 28 48* 44* 35 25	.10 16 07 24 07 17 13 28
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	43 46 45 61 68* 71* 89** 81**	51 59 63* 70* 78** 79** 81** 82**	34 42 49 44 51 63 33	54 36 24 62 48 52 83**
		Interfer	ence & Recog	nition -	SD
1/12	<u>FU</u> :	Pro%	<u>Ret%</u>	Reco	<u>F+</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	19 22 18 20 16 05 09	.48* .47* .54** .56** .48* .65** .62**	74**67**74**71**74**75**	04 02 .07 .09 .08 .11 .05
3/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.58** .60** .68** .61** .75** .70** .69**	.50** .45* .59** .68** .54** .47* .52**	.13 .29 .67** .59** .77** .60**	.24 .37 .76** .71** .84** .70**

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A (cont)

Interference & Recognition - SD

6/12	<u>FU</u> :	%Pro	%Ret	Recog	<u>False+</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.32* .14 .08 .11 .18 .25 .34* .31*	.28 .06 06 .23 .22 .12 .47**	.08 .27 .18 .42** .02 .05 .29 .45**	.15 .35* .20 .45** .11 .13 40**
12/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.29 .45** .04 .07 .20 .29 .37*	.11 .37* .19 .14 .25 .27 .31	06 .19 .13 .05 .18 .11 .09	01 .22 .18 .11 .24 .18 .12 04
24/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	02 .15 .18 .28 .40* .16 .12	.00 .15 .06 .19 .06 .11 .15	.07 38 32 58** 20 33 24	.07 .28 .12 .35 .48* .67** .46*
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.17 .18 21 .16 27 03 23	.42 .19 .04 .49 .30 .31 .72*	38 63 64* 71* 83** 78** 93**	04 06 11 .18 .18 .16 .63

TABLE C9.7: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD. SAMPLE B

Recall Scores on List A - R'	Recall	Scores	on List.	A -	RT
------------------------------	--------	--------	----------	-----	----

24/12	<u>FU</u> :	<u> </u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	15 23 26 26 17 15 17	16 24 26 27 17 15 17	16 24 26 27 17 15 17	1928303120182130
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	45 50 52 61 47 59 64*	39 47 47 57 42 56 56	47 52 56 61 47 60 55	34 44 49 59 43 59 56 52
	Re	call Score	s on Lists	A & B - RT	
24/12	<u>FU</u>	<u>A5</u> <u>T</u>	otal A	<u>B</u>	<u>ADel</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	18 27 31 31 20 18 20 29	41 51 52 52 42 41 45	16 24 27 27 17 16 18 25*	21 30 32 33 22 20 23 32
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +:	48 57 61 70* 56 70* 63*	44 52 55 64* 49* 63 62	35 38 54 58 42 53 48 39	55 65* 70* 78** 64* 78** 73* 69*

TABLE C9.7: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE B (cont)

Interference & Recognition - RT

24/12	<u>: FU</u> :	Pro%	<u>Ret%</u>	<u>Reco</u>	<u>F+</u>
Set	1 +:: 2 +:: 3 +:: 4 +::	.36 .30 .31 .23 .35 .37 .33	.07 .01 03 03 .07 .08 .08	21 .29 31 32 22 20 23 31	09 .18 20 21 11 09 11 19
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	30 31 03 06 17 14 16 32	.71* .81** .84** .87** .77** .77** .77**	67* .78** .85**89**79**09**82**76**	.74** .82** .90** .92** .82** .94** .82**
		Recall o	n List A -	SD	
24/12	<u>FU</u> :	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	18 04 28 23 23 09 14	19 04 28 23 24 09 14	19 04 28 25 24 10 16 25	22 08 30 27 27 13 18
36/12	FU:				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	55 59 54 55 61 33 58	50 58 48 54 66* 47 75**	59 66* 52 60 67* 53 75**	51 60 50 53 68* 47 65* 52

TABLE C9.7: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE B (cont)

Recall on Lists A & B - SD

24/12	<u>FU</u> :	<u>A5</u>	Total A	<u>B</u>	A Del
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	21 08 27 25 27 12 16 26	46 37 38 45 55 39 34 47	19 05 26 22 23 09 13 22	24 11 30 28 30 15 18 29
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	65* 71* 63 65* 62** 59 69* 63	59 66* 55 60 71* 54 72*	44 48 39 40 57 37 21	73* 79** 72* 71* 83** 69* 66* 73*

Interference & Recognition - SD

24/12	<u>FU</u> :	Pro%	Ret%	Reco	<u>F+</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.31 .47 29 .06 .33 .31 .05 04	.06 .21 16 04 .08 .12 .00 02	24 09 31 29 29 15 20	12 .03 26 19 16 03 11
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	30 24 32 31 05 26 48 20	.89** .94** .85** .88** .94** .90** .83**	84**90**83**85**95**85**74**88**	.87** .92** .84** .88** .97** .91** .73**

TABLE C9.8: DIGIT SPAN MEAN & SD SCORE AT EACH FU

		1/12	Follow	v-up	3/12	Follow	7-up		
Gro	<u>up</u>	<u>. F</u>	<u>B</u>	<u>Total</u>	<u>F</u>	<u>B</u>	<u>Total</u>		
A M/M S VS ES	Mean: SD: Mean: SD: Mean: SD: Mean: SD: Mean: SD: Mean: SD:	1.1	4.5 1.5 4.4 1.0 3.7 1.4 5.5 1.9 4.5 0.5	10.8 2.7 10.9 2.2 9.4 2.5 12.0 3.2 12.0	6.9 1.2 7.0 0.7 6.8 1.1 7.0 0.8 6.5 2.1	5.0 1.5 4.8 1.2 5.1 1.3 6.0 1.1 3.5	12.2 2.6 12.2 1.5 12.3 2.3 13.3 1.8 10.0 3.7		
		6/12	6/12 Follow-up			Follow	7−up		
		<u>F</u>	<u>B</u>	<u>Total</u>	<u>F</u>	<u>B</u>	<u>Total</u>		
A M/M S VS ES	Mean: SD: Mean: SD: Mean: SD: Mean: SD: Mean: SD: Mean:	1.3 6.5 1.4 6.4 1.0	5.0 1.3 5.2 1.1 5.0 1.4 5.9 1.3 4.1 0.8	11.9 2.3 11.9 2.2 11.9 2.3 13.2 2.1 10.8 2.0	6.7 1.0 6.6 1.0 6.8 0.7 7.0 0.8 6.7 1.1	5.4 1.4 5.3 1.4 5.8 1.4 6.1 1.5 4.8 1.2	12.4 2.2 11.9 1.9 13.1 2.5 13.4 2.2 11.4 1.6		
		24/12	Follow	-up	36/12	36/12 Follow-up			
		<u>F</u>	<u>B</u>	<u>Total</u>	<u>F</u>	<u>B</u>	<u>Total</u>		
M/M S VS ES	SD: Mean: SD: Mean: SD: Mean: SD: Mean:	0.9 6.5	5.4 1.3 6.4 0.8 5.8 1.1	1.4 12.9 2.4	7.1 0.7	4.9 1.1	12.0 1.7		
<u>Sam</u> j	ole B	24/1	2 Follo	ow-up	36/1	2 Follo	w-up		
	Mean: SD:				5.5 1.1	4.9 1.0	10.4 2.0		

F= digits forward: B= digits backward: 135

TABLE C9.9: t-TESTS, DIGIT SPAN, SAMPLE A

<u>1/12 FU</u> :	Forward	<u>Backward</u>	<u>Total</u>
M/M(8) v S(7) M/M v VS(6) S v VS	<1 <1 <1	1.049 1.326 1.913*	1.194 <1 1.590
3/12 FU: M/M(5) v S(7) M/M v VS(9) M/M v ES(4) S v VS S v ES VS v ES	<1 <1 <1 <1 <1 <1	<1 1.819* 1.327 1.382 1.723 2.710**	<1 1.165 1.128 <1 1.124 1.683
6/12 FU: M/M(11) v S(10) M/M v VS(9) M/M v ES(10) S v VS S v ES VS v ES	<1 <1 <1 1.497 <1	<1 1.237 2.538** 1.382 1.735* 3.415***	<1 1.335 1.189 1.297 1.132 2.487**
12/12 FU: M/M(10) V S(8) M/M V VS(9) M/M V ES(9) S V VS S V ES VS V ES		<1 1.211 <1 <1 1.587 1.990*	1.151 1.487 <1 <1 1.691 1.997*
24/12 FU: M/M(7) v S(10) M/M v VS(8) M/M v ES(7) S v VS S v ES VS v ES	2.798*** <1 <1 1.636 2.607*** <1	1.603 <1 1.894* 1.236 3.818*** 2.642**	2.063* <1 1.459 1.475 4.007**** 1.990*
*=p<.05; **=	p< .025:	***=p<.01;	****=p<.005;

TABLE C9.10: t-TESTS, WECHSLER MEMORY SCALE, SAMPLE A

M/M M/M S S	v v v	S(10) VS(9) ES(10) VS ES	Factor <1 <1 3.464** <1 3.314** 2.909**	**	Factor : <1 1.071 1.939 1.574 1.570 3.895***		Factor 3 <1 1.162 1.155 1.095 2.486** 3.596****
24/12	FU	:					
M/M M/M S	V V V V		<1 <1 1.702 <1 1.026 1.026		3.918**; 3.918**; 1.087 <1 5.353**; 5.353**;	* *	<1 <1 <1 <1 <1
*=p<.0	5;	**=p<	.025;	* * * =p	< .01;	***	=p<.005;
*=p< .1	0;	* * = p	(.05;	***=p	< .01		

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample	<u>A</u> <u>1/</u>	12 FOL	LOW-UF) -	<u>3/1</u>	2 FOLL	OW-UP	
Subj.	Pro%	Ret%	Reco	<u>F+</u>	Pro%	Ret%	Reco	<u>F+</u>
1	0	18	15	0	33	31	15	0
2	0	40	14	0	0	33	14	0
3	0	6	15	0	0	6	1'5	0
4	0	15	15	0	0	0	15	0
5	20	9	11	4	20	40	11	1
6	0	46	15	0	0	17	15	0
7	55	33	15	0	0	20	15	0
8	40 50	20	15	0	0	44	13	0
9	50	50	15	0	DNA	DNA	DNA 12	DNA
10	20 0	25 20	15 15	0	0.	39 DATA	DNA	O DNA
11 12	40	29 0	11	0 0	DNA DNA	DNA DNA	DNA	DNA
13	43	39	15	0	DNA	DNA	DNA	DNA
13	14	86	12	1	0	56	12	1
15	PTA	PTA		PTA	33	98	8	6
16	16	36	11	0	30	7	15	0
17	DNA	DNA	DNA		DNA	DNA	DNA	DNA
18	PTA	PTA	PTA		11	7	15	0
19	29	18	12	0	0	17	15	ŏ
20	NT	NT	NT	NT	Ö	60	9	Ö
21	PTA	PTA	PTA	PTA	DNA	DNA		ONĀ
22	PTA	PTA	PTA	PTA	66	50	12	5
23	30	7	15	0	0	18	14	2
24	DNA	DNA	DNA	DNA	DNA	DNA		DNA
25	0	18	15	0	DNA	DNA	DNA I	ONA
26	14	18	15	0	DNA	DNA	DNA 1	DNA
27	NT	NT	NT	NT	57	63	13	0
28	PTA	PTA	PTA	PTA	_	_	_	
29	NT	NT	NT	NT	0	0	15	1
30	PTA	PTA	PTA	PTA	PTA	PTA	PTA I	PTA
31	PTA	PTA	PTA	PTA	_	_	_	_
32	PTA	PTA	PTA	PTA	NT	NT	NT	NT
33	NT	NT	NT	NT	43	62	15	0
34	14	17	15	0	0	21	15	1
35	NT	NT	NT	NT	0	8	15	0
36	DNA	DNA		DNA	29	0	15	0
37	29	20	15	0	DNA	DNA		DNA
38	DNA	DNA	DNA		DNA	DNA		DNA
39	DNA	DNA	DNA		28	68 DTA	13	0
40	PTA	PTA	PTA		PTA	PTA		PTA
41	DNA	DNA	DNA		DNA	DNA		DNA
42	25	23	14	0	29	15	15	0

Pro%= Proactive Interference; Reco= Recognition; Ret%= Retroactive Interference; F+= False positives;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Subj. Pro% Ret% Reco F+ Pro% Ret% Reco	<u>F+</u>
1 13 14 15 0 33 13 15 2 43 0 15 0 44 31 14	0 0
3 0 8 15 0 0 0 15	ő
4 0 0 15 0 14 8 15	0
5 0 0 15 0 38 14 15	0
6 0 36 15 0 0 15 15	ō
7 0 13 15 0 0 27 15	0
8 0 40 14 0	_
9 0 46 15 0 DNA DNA DNA D	NA
	NA
11 0 8 14 0 0 18 15	0
	NA
13 0 10 15 1 43 25 15	0
14 0 64 15 0 0 71 14	1
15 33 98 15 2 60 46 12	1
16 0 0 15 0 0 7 15	0
17 0 17 15 0 0 7 15	0
18 0 0 15 0 0 14 15 10 15 0 50 21 10	0
19· 0 40 15 0 50 31 10 20 0 63 11 0 0 63 10	0
20 0 63 11 0 0 63 10 21 0 20 13 3 40 25 15	1 0
22 0 14 14 0 50 14 15	0
23 29 0 15 0 0 23 14	0
24 0 0 15 0 0 7 15	ő
25	Ö
26 0 17 15 1 17 29 15	2
27 0 50 15 2 0 25 15	Ō
28	_
29 0 7 15 0 27 7 15	0
30 98 0 13 2 50 29 14	2
31 25 98 9 3 0 38 14	1
32 0 73 13 0 25 70 13	0
33 DNA DNA DNA DNA 57 17 15	0
34 0 0 15 0 13 0 15	0
35 0 9 15 0 0 14 15	0
36 0 7 15 0 33 0 15	0
37 17 25 15 0	_
38	0
39 0 26 15 0 0 15 15 40 20 63 15 0 0 64 14	0
40 20 63 15 0 0 64 14 41 0 0 15 0 33 42 13	1 1
42 14 8 15 0 33 15 15	0

Pro%= Proactive Interference; Reco= Recognition: Ret%= Retroactive Interference; F+= False positives;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample	Sample A 24/12 FOLLOW-UP				<u>36</u>	36/12 FOLLOW-UP			
<u>Subj.</u>	Pro%	<u>Ret%</u>	Reco	<u>F+</u>	Pro%	Ret%	Reco	<u>F+</u>	
1	14	0	15	0	0	2.0	1/5	0	
2	0	36	14	. 0	DNA	DNA	DNA	DNA	
3	0	7	15	0	8	0	15	0	
4	DNA	DNA		DNA	DNA	DNA	DNA	DNA	
5	38	0	15	0	0	7	15	0	
6	0	13	15	0	DNA	DNA	DNA		
7	0	7	15	0	DNA	DNA		DNA	
8	DNA	DNA		DNA	DNA	DNA		DNA	
9	DNA	DNA		DNA	DNA	DNA		DNA	
10	20	31	13	0	DNA	DNA	DNA	DNA	
11	0	0	15	0	0	14	15	0	
12	DNA	DNA		DNA	DNA	DNA	DNA		
13	DNA	DNA	DNA	DNA	14	8	15	0	
14	14	98	11	1	DNA	DNA	DNA		
15	56	50	15	0	0	89	14	1	
16	0	0	15	0	DNA	DNA	DNA		
17	0	14	15	0	30	13	15	0	
18	20	14	15	1	22	54	15	0	
19	0	21	14	2	DNA	DNA	DNA		
20	13	27	14	0	DNA	DNA	DNA		
21	50	23	14	1	0	18	14	0	
22	43	11	15	0	DNA	DNA	DNA		
23	0	13	15	0	DNA	DNA	DNA		
24	DNA	DNA		DNA	DNA	DNA	DNA		
25 26	0	25	15	0	DNA	DNA	DNA		
20 27	0 14	8 50	15 15	0	DNA	DNA	DNA		
28	DNA	DNA		O DNA	DNA	DNA	DNA		
29 29	DNA	DNA	DNA	DNA	DNA DNA	DNA DNA	DNA		
30	DNA	DNA		DNA	DNA	DNA	DNA DNA		
31	0	29	12	3	DNA	DNA	DNA		
32	30	67	14	0	DNA	DNA	DNA		
33	0	22	15	Ö	DNA	DNA	DNA		
34	ő	0		0	DNA	DNA	DNA		
35	DNA	DNA		DNA	DNA	DNA	DNA		
36	DNA	DNA	DNA	DNA	DNA	DNA	DNA		
37	DNA	DNA		DNA	DNA	DNA	DNA		
38	0	7	15	0	DNA	DNA	DNA		
39	DNA	DNA		DNA	DNA	DNA	DNA		
40	DNA	DNA		DNA	DNA	DNA	DNA		
41	0	8	14	0	DNA	DNA	DNA		
42	DNA	DNA		DNA	DNA	DNA	DNA		
							~1111		

Pro%= Proactive Interference; Reco= Recognition; Ret%= Retroactive Interference; F+= False positives;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Recall on Lists A & B

<u>Sample</u>	<u>B</u>	<u>24</u>	/12 FO	<u>LLOW-U</u>	P			
<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	<u>A5</u>	<u>TotA</u>	<u>B</u>	A Del
1	8	12	13	14	14	61	5	14
2	6	7	9	11	11	44	5	12
3	5	8	10	11	14	48	. 6	12
4	7	9	8	9	12	43	8	10
5	7	11	11	13	15	57	6	12
6	5	4	3	5	7	24	3	4
7	7	10	12	12	13	54	5	13
8	3	5	6	4	6	24	1	2
9	_	_	_		-	_	_	_
10	5	7	9	6	7	34	3	5

Recall on Lists A & B

36/12 FOLLOW-UP

<u>Subj.</u>	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>	<u>A5</u>	TotA	<u>B</u>	A Del
1	11	14	15	15	15	70	7	15
2	7	12	10	13	13	55	5	13
3	8	8	10	11	13	50	12	15
4	6	9	11	9	10	45	8	9
5	9	11	13	12	13	58	6	13
6	3	6	5	6	7	27	3	5
7	6	10	12	13	13	54	8	13
8	3	5	4	5	5	22	3	0
9	7	13	15	15	15	65	10	14
10	4	6	7	5	7	29	2	4

A1-A5= A trials; TotA= total of trials A1-A5; B= list B score; A Del= recall after interference;

TABLE C8.1: RAW DATA FOR REY AVLT AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample B 24/12 FOLLOW-UP				36/	12 FOL	LOW-UP		
<u>Subj.</u>	Pro%	Ret%	<u>Reco</u>	<u>F+</u>	Pro%	<u>Ret%</u>	<u>Reco</u>	<u>F+</u>
1	38	0	15	0	36	0	15	0
2	17	9	15	1	29	0	15	0
3	0	14	15	0	0	8	15	0
4	0	16	12	0	0	10	14	0
5	14	20	15	0	33	0	15	0
6	40	43	11	1	0	28	14	0
7	29	0	15	0	0	0	15	0
8	66	33	6	3	0	98	10	6
9	_	_	-	_	0	7	15	0
10	40	29	13	2	50	43	12	3

Pro% = Proactive Interference; Reco = Recognition Ret% = Retroactive Interference; F+ = False positives

TABLE C8.2: RAW DATA FOR DIGIT SPAN AT EACH FOLLOW-UP SAMPLES A & B

Sample A	1/12	FOLLOW-	<u>-UP</u>	3/1	3/12 FOLLOW-UP			
Subj.	DIG F	DIG B	TOT	DIG F	DIG B	TOT		
1	7	3	10	8	5	13		
2	8	7	15	9	. 8	17		
3	.7	5	12	9	4	13		
4	8	4	12	9	7	16		
5	4	2	6	5	4	9		
6	6	6	12	7	5	12		
7	9.	8	17	DATA	_ DN3	 T) N I R		
8	5	4	9	DNA	DNA	DNA		
9	4 7	2 5	6 12	6 7	4 5	10 12		
10 11	6	3	9	DNÁ	DNA	DNA		
12	6	4	10	DNA	DNA	DNA		
13	9	6	15 15	DNA	DNA	DNA		
14	7	4	11	8	5	13		
15	PTA	PTA	PTA	7	3	10		
16	5	4	9	6	7	13		
17	DNA	DNA	DNA	DNA	DNA	DNA		
18	PTA	PTA	PTA	8	5	13		
19	6	4	10	6	4	10		
20	NT	NT	NT	7	5	12		
21	DNA	DNA	DNA	DNA	DNA	DNA		
22	DNA	DNA	DNA	3	1	4		
23	6	7	13	7	6	13		
24	DNA	DNA	DNA	DNA	DNA	DNA		
25	4	3	7	DNA	DNA	DNA		
26	5	4	9	DNA	DNA	DNA		
27	NT	NT	NT	8	4	12		
28	PTA	PTA	PTA			_		
29	NT	NT	NT	6	5	11		
30	PTA	PTA	PTA	PTA	PŢA	PTA		
31	PTA	PTA	PTA	_	_	_		
32	PTA	PTA	PTA	NT	NT	NT		
33	NT	NT	NT	8	5	13		
34	6	5	11	7	4	11		
35	NT	NT	NT	8	6	14		
36	DNA	DNA	DNA	8	7	15		
37	8	5	13	DNA	DNA	DNA		
38	DNA	DNA	DNA	DNA	DNA	DNA		
39	DNA	DNA	DNA					
40	PTA	PTA	PTA	PTA	PTA	PTA		
41	DNA	DNA	DNA	DNA	DNA	DNA		
42	7	5	12	7	7	14		

DIG F= digit forward:
TOT= total digit span:
NT= subject not tested, poor physical/cognitive state:

TABLE C8.2: RAW DATA FOR DIGIT SPAN AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample A	6/12 FOLLOW-UP			12/12 FOLLOW-UP		
<u>Subj.</u>	DIG F	DIG B	<u>TOT</u>	DIG F	DIG B	TOT
1	8	5	13	8	5	13
2	9	7	16	-		_
3	8	4	12	6	6	12
4	9	4	13	9	8	17
5 6	5 8	5 6	10 14	6 7	4 6	10 13
7	8	7	15	9	8	13 17
, 8	5	, 5	10	_	_	* /
9	5	4	9	DNA	DNA	DNA
10	7	4	11	DNA	DNA	DNA
11	7	3	10	6	4	10
1,2	7	6	13	DNA	DNA	DNA
13	7	6	13	8	5	13
14	7	5	12	8	6	14
15	7	4	11	8	4	12
16	7	6	13	7	7	14
17	8	7	15	6	7	13
18 19	8 5	5 4	13 9	7 6	6	13 10
20	7	3	10	6	4 4	10
21	4	4	8	5	4	9
22	5	3	8	5	5	10
23	8	7	15	6	7	13
24	. 8	6	14	8	8	16
25	4	3	7	5	4	9
26	6	4	10	7	5	12
27	6	6	12	7	5	12
28	_	_	-	-	-	_
29	7	6	13	7	4	11
30 31	8 [.] 7	5 3	13 10	6 7	6 3	12 10
31 32	8	4	12	7	6	13
33	DNA	DNA	DNA	8	7	15
34		5	10	7	4	11
35	5 8	6	14	8	6	14
36	7	6	13	8	7	15
37	7 8	5	13	-	_	_
38	9	8	17	9	7	16
39	-	_ 3	_	_	-	-
40	5	3	8	7	3	10
41	5 7 7	6	13	6	6	12
42	7	5	12	6	4	10

DIG F= digit forward; DIG B= digit backward; TOT= total digit span; PTA= subject still in PTA; NT= subject not tested, poor physical/cognitive state;

TABLE C8.2: RAW DATA FOR DIGIT SPAN AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample A	24/12 FOLLOW-UP			36/12 FOLLOW-UP		
Subj.	DIG F	DIG B	TOT	DIG F	DIG B	TOT
1	7	5	12	7	5	12
2	8	6	14	_	_	_
3	8	4	12	7	6	13
4	DNA	DNA	DNA	DNA	DNA	DNA
5	7	6	13	6	4	10
6	7	5	12	DNA	DNA	DNA
7	7	7	14	DNA	DNA	DNA
8	DNA	DNA	DNA	DNA	DNA	DNA
9	DNA	DNA	DNA	DNA	DNA	DNA
10	6	5	11	DNA	DNA	DNA.
11	6	4	10	7	5	12
12	DNA	DNA	DNA	DNA	DNA	DNA
13	DNA	DNA	DNA	8	6	14
14	8	6	14	DNA	DNA	DNA
15	8	4	12	7	4	11
16	8	7	15	DNA	DNA	DNA
17	9	8	17	8	7	15
18	6	5	11	8	4	12
19	6	4	10	DNA	DNA	DNA
20	7	5	12	DNA	DNA	DNA
21	5	5	10	6	3	9
22	6	3	9	DNA	DNA	DNA
23	8	7	15	DNA	DNA	DNA
24	DNA	DNA	DNA	DNA	DNA	DNA
25	5	5	10	DNA	DNA	DNA
26	7	4	11	DNA	DNA	DNA
27	7	5	12	DNA	DNA	DNA
28	DNA	DNA	DNA	DNA	DNA	DNA
29	DNA	DNA	DNA	DNA	DNA	DNA
30	DNA	DNA	DNA	DNA	DNA	DNA
31	6	2	8	DNA	DNA	DNA
32	7	4	11	DNA	DNA	DNA
33	9	7	16	DNA	DNA	DNA
34	7	6	13	7	5 DVA	12
35	DNA	DNA	DNA	DNA	DNA	DNA
36	DNA	DNA	DNA	DNA	DNA	DNA
37	DNA	DNA	DNA	DNA	DNA	DNA
38	9	7	16	DNA	DNA	DNA
39	DNA	DNA	DNA	DNA	DNA	DNA
40	DNA	DNA	DNA	DNA	DNA	DNA
41	6 DNA	6 DNIA	12	DNA	DNA	DNA
42	DNA	DNA	DNA	DNA	DNA	DNA

DIG F= digit forward; DIG B= digit backward; TOT= total digit span; PTA= subject still in PTA: NT= subject not tested, poor physical/cognitive state;

TABLE C8.2: RAW DATA FOR DIGIT SPAN AT EACH FOLLOW-UP SAMPLES A & B (cont)

Sample B	24/12	FOLLOW-	<u>-UP</u>	<u>36/1</u>	2 FOLLOW	-UP
Subj.	DIG F	DIG B	TOT	DIG F	DIG B	TOT
1 2	6 7	4 5	10 12	6 7	5 5	11 12
3	4	4	8	. 4	5	9
4 5	6 7	5 5	11 12	5 7	5 6	10 13
6	5	5	10	5	4	9
7	8	4	12	5	4	9
8	4	3	7	5	5	10
9	-	_	_	7	7	14
10	4	2	6	4	3	7

DIG F= digit forward; DIG B= digit backward: TOT= total digit span

TABLE C8.3: RAW STEN DATA FOR WECHSLER MEMORY SCALE FACTORS, SAMPLES A & B

Sample A	6/12 FOLLOW-UP			24/12 FOLLOW-UP			
<u>Subj.</u>	<u>F_1</u>	<u>F 2</u>	<u>F 3</u>	<u>F 1</u>	<u>F 2</u>	<u>F_3</u>	
1 2 3 4 5	9.1 7.0 9.2 9.8 6.9	7.3 8.4 7.2 7.9 3.1	4.9 6.5 5.7 6.5 4.2	10.0 6.6 10.0 DNA	7.5 8.4 7.2 DNA	6.5 6.5 5.7 DNA	
6 7 8 9 10	9.6 10.0 7.0 7.0 7.8	8.1 8.8 6.6 4.1 6.6	5.7 6.5 6.5 5.7 6.5	9.6 10.0 DNA DNA 6.5	7.2 8.4 DNA DNA 5.9	5.7 6.5 DNA DNA 5.7	
11 12 13 14	8.0 - 6.6	5.4 - - 5.2	5.7 - - 4.9	-	5.7 DNA DNA	6.5 DNA DNA	
15 16 17 18	4.4 9.9 9.9 8.9	5.2 7.5 8.5 7.9 3.7	4.9 6.5 5.7 6.5	4.1 10.0 - -	6.5 8.4 - -	4.9 3.4 - -	
19 20 21 22 23	7.3 6.8 7.5 5.9 9.1	6.3 4.0 2.7 8.4	3.4 5.7 4.2 .4.2 6.5	5.5 - 8.8 6.4	4.1 - 5.1 4.4	1.1 - 4.9 4.2	
25 24 25 26 27	10.0 - 8.6 6.6	8.4 - 6.3 6.6	6.5 - 5.7 6.5	DNA 10.0 -	DNA 5.3 -	DNA 4.2 -	
28 29 30 31	6.2 3.7 4.2	7.6 5.9 5.3	5.7 5.7 5.4	DNA DNA DNA	DNA DNA DNA	DNA DNA DNA	
32 33 34 35	6.6 DNA - 9.5	6.9 DNA - 7.2	4.9 DNA - 5.7	8.1 8.1 7.9 DNA	4.6 8.8 7.0 DNA	5.7 6.5 6.5 DNA	
36 37 38 39	9.6 8.6 9.5 8.6	7.6 7.0 9.7 7.2	5.7 6.5 6.5 5.7	DNA DNA 9.3 DNA	DNA DNA 9.3 DNA	DNA DNA 6.5 DNA	
40 41 42	5.0 7.0 3.8	3.1 7.5 7.0	4.2 6.5 6.5	DNA 9.6 DNA	DNA 7.5 DNA	DNA 6.5 DNA	

F 1= Factor 1: F 2= Factor 2: F 3= Factor 3:

TABLE C8.3: RAW STEN DATA FOR WECHSLER MEMORY SCALE FACTORS, SAMPLES A & B

Sample B	24/12 FOLLOW-UP			36/12 FOLLOW-UP			
<u>Subj.</u>	<u>F 1</u>	<u>F 2</u>	<u>F 3</u>	<u>F 1</u>	<u>F_2</u>	<u>F 3</u>	
1	9.1	6.3	5.7	_	_	_	
2	_	_	-	_	<u> </u>	_	
3	8.7	4.2	4.9	9.7	4,.5	5.7	
4	9.3	6.7	5.7	10.0	6.,3	5.7	
5	_	_	_	9.0	7.6	5.7	
6	_	_	_	6.5	4.5	5.9	
7	9.4	7.5	6.5	9.0	6.1	6.5	
8	3.6	2.1	1.9	4.5	5.4	5.7	
9			_	10.0	8.8	6.5	
10	2.8	3.2	2.6	2.6	3:.4	4.9	

F 1= Factor 1: F 2= Factor 2: F 3= Factor 3:

TABLE C8.4: RAW DATA FOR NATIONAL ADULT READING TEST & SUBJECTIVE MEMORY SCALE. SAMPLES A & B

		Samp		Sample B			
	NAI	RT	SMQ	<u>NA</u>	.RT	<u>SMQ</u>	
<u>Subj</u> .	VIQ	PIQ	<u>24m FU</u>	VIΩ	PIQ	24m FU	
1234567890112345678901232222222223333333333442	- 113 - 112 121 - 111 99 - 105 94 110 - 101 84 - 98 913 - 111 113 117 - 119 99 - 111 117 114 111 117 114 111 117 114 111 117 114 111 117 118 119 111 111 111 111 111 111 111 111	113 - 112 118 - 111 102 - 107 99 110 - 104 94 - 102 103 113 115 - 116 102 - 111 115 116 107 96 111	136 136 136 136 103 162 	107 108 102 102 113 85 109 - 113 90	108 109 105 105 113 93 109 - 113 96	142 146 — 139 — — — —	

NART= National Adult Reading Test; PIQ= performance IQ; SMQ= Subjective Memory Questionnaire; VIQ= verbal IQ; DNA= did not attend;

APPENDIX C9: GROUP MEMORY TEST SCORES

TABLE C9.1a: MEAN & SD, REY VARIABLES, SAMPLE A Recall Scores on List A Trials

1/12 FU	1	2 .	3	4	5	<u>Total</u>
A Mean: (n=23)SD: M/M Mean: (8) SD: S Mean: (7) SD: VS Mean: (6) SD: ES Mean: (2) SD:	6.0 2.1 6.1 1.9 5.3 0.9 6.5 3.1 7.5	8.0 2.5 8.5 1.7 7.1 1.6 8.7 3.9 7.5 1.5	9.4 2.6 10.3 1.9 9.1 1.6 9.0 3.8 8.5 2.5	10.0 3.0 11.1 1.6 9.0 2.9 10.3 4.1 8.5 1.5	10.8 2.9 12.0 1.5 10.3 3.2 10.7 3.5 8.5	45 11.4 48 7.4 42.7 7.5 45.2 17.7 40 7.0
3/12 FU						
A Mean: (25) SD: M/M Mean: (5) SD: S Mean: (7) SD: VS Mean: (9) SD: ES Mean: (4) SD:	6.6 1.6 6.6 1.0 5.9 1.1 7.4 1.4 6.0 2.1	9.0 2.6 8.8 1.2 9.1 2.7 9.9 2.3 6.8 2.9	10.2 3.0 12.0 1.6 10.1 3.0 10.4 2.5 7.8 3.4	10.8 3.1 12.2 1.7 11.1 2.7 11.0 2.7 7.8 3.7	12.0 2.8 13.4 1.0 11.9 2.4 12.9 1.7 8.5 3.6	48.9 12.0 53.0 5.7 49.7 11.3 51.3 9.5 36.8 15.6
<u>6/12 FU</u>						
A Mean: (40) SD: M/M Mean: (11) SD: S Mean: (10) SD: VS Mean: (9) SD: ES Mean: (10) SD:	5.9 1.6 6.6 1.7 5.7 1.2 6.0 1.9 5.2 1.2	8.7 2.4 9.2 1.6 9.4 2.1 8.9 3.1 7.3 2.1	10.1 2.9 11.4 1.4 10.5 2.8 10.6 3.1 7.9 2.7	11.1 3.0 12.1 2.0 11.7 2.8 12.0 3.2 8.5 2.2	11.6 2.7 12.4 1.6 13.0 2.0 12.2 2.6 8.7 2.4	47.4 11.1 51.6 6.5 50.3 9.1 49.7 12.8 37.6 9.5

A1-A5= A trials; Total= total of trials A1-A5;

TABLE C9.1a: MEAN & SD, REY VARIABLES, SAMPLE A (cont)

Recall Scores on List A Trials

12/12 FU	1	2	3	4	5	<u>Total</u>
A Mean: (n=37)SD: M/M Mean: (10) SD: S Mean: (8) SD: VS Mean: (10) SD: ES Mean: (9) SD:	7.3	9.8	11.2	11.7	12.4	52.4
	1.9	2.3	2.5	2.6	2.7	10.3
	7.5	10.6	12.0	12.5	13.5	56.1
	1.4	2.2	1.5	1.8	1.2	6.9
	7.1	10.1	11.6	12.6	12.9	54.4
	0.9	1.7	2.2	1.9	2.2	8.3
	8.2	10.7	12.2	12.6	12.8	56.5
	2.5	1.5	2.1	2.1	2.2	9.0
	6.1	7.7	8.7	9.0	10.4	41.9
	1.5	2.2	2.2	2.4	2.5	9.2
24/12 FU						
A Mean: (26) SD: M/M Mean: (7) SD: S Mean: (5) SD: VS Mean: (8) SD: ES Mean: (7) SD:	6.5	9.9	10.7	11.7	12.5	51.3
	1.9	2.4	2.5	2.6	2.5	10.1
	6.4	10.7	11.7	12.7	13.9	55.4
	1.8	2.6	2.0	2.1	1.0	8.9
	7.8	10.0	11.2	11.6	12.2	52.8
	1.6	2.3	2.3	2.7	2.4	10.1
	7.4	9.6	10.4	11.3	12.1	50.8
	1.6	2.9	3.4	3.3	2.9	13.2
	6.0	7.7	8.0	9.0	10.1	40.9
	2.3	1.7	1.1	1.3	2.8	6.6
36/12 FU						
A Mean:	7.3	10.7	11.9	12.9	13.4	56.2
(10) SD:	2.7	2.3	2.3	2.3	1.9	10.0

TABLE C9.1b: MEAN & SD, REY VARIABLES, SAMPLE B Recall Scores on List A Trials

	1	2	3	4	5	<u>Total</u>
24m Mean: (10) SD:		8.1 2.5	9.0 2.9	9.5 3.4	11.0 3.3	43.2 12.8
36m Mean: (10) SD:		9.4 3.0	10.2 3.7	10.4 3.7		

A1-A5= A trials:

Total= total of trials A1-A5;

TABLE C9.2a: MEAN & SD. MORE REY VARIABLES, SAMPLE A
Recall of List B. & Interference & Recognition Scores

<u>1/12 FU</u>	<u>B</u>	A Del	Pro%	<u>Ret%</u>	Reco	<u>F+</u>
A Mean: (23) SD: M/M Mean: (8) SD: S Mean: (7) SD: VS Mean: (6) SD: ES Mean: (2) SD:	5.0 16 5.5 1.5 4.9 1.6 4.3 1.8 5.5	8.5 3.5 10.4 1.9 8.3 2.6 7.7 4.5 4.5 3.5	19.1 17.5 18.9 16.8 14.9 16.7 23.5 20.3 21.5	29.2 23.1 17.4 10.8 27.4 14.4 39.0 28.6 53.0 33.0	13.5 3.1 14.0 1.5 14.4 1.4 11.8 5.1 13.5 1.5	0.2 0.8 0.0 0.0 0.6 1.4 0.0 0.5 0.5
3/12 FU A Mean: (25) SD: M/M Mean: (5) SD: S Mean: (7) SD: VS Mean: (9) SD: ES Mean: (4) SD:	6.2 2.8 7.2 2.8 4.9 1.5 7.2 2.7 4.8 2.6	9.1 4.2 11.0 1.7 9.4 4.1 9.7 3.5 4.8 5.0	16.2 19.6 12.4 15.2 18.7 19.5 11.2 16.3 27.5 25.2	30.2 25.5 18.0 8.2 25.3 21.5 30.7 24.8 52.8 32.2	13.7 2.0 15.0 0.0 13.5 1.6 13.9 1.9	0.7 1.5 0.2 0.4 0.1 0.3 0.7 3.0 2.6
A Mean: (40) SD: M/M Mean: (11) SD: S Mean: (10) SD: VS Mean: (9) SD: ES Mean: (10) SD:	6.1 2.2 6.7 1.9 6.4 1.7 6.4 2.4 4.6 2.3	9.3 4.3 11.2 2.6 10.7 3.2 10.4 3.8 4.9 3.9	9.5 18.5 5.2 6.9 5.8 12.3 11.4 15.3 19.3 28.8	23.5 26.6 11.4 11.1 20.0 17.9 17.6 20.5 45.5 36.2	14.5 1.2 15.0 0.0 14.7 0.6 14.4 1.3 13.7 1.8	0.4 1.8 0.1 0.3 0.6 0.0 1.0

B= List B score; A Del= Recall after Interference: Pro%= Proactive Interference; Reco= Recognition; Ret%= Retroactive Interference; F+= False Positives;

TABLE C9.2a: MEAN & SD, MORE REY DATA, SAMPLE A (cont)

Recall of B. & Interference & Recognition Scores

12/12 FU	<u>B</u>	A Del	Pro%	Ret%	Reco	<u>F+</u>
A Mean: (n=37)SD: M/M Mean: (10) SD: S Mean: (8) SD: VS Mean: (10) SD: ES Mean: (9) SD:	6.3 2.7 6.2 2.8 6.3 1.2 7.7 3.1 4.9 2.1	9.7 3.8 11.5 2.4 11.3 2.8 10.0 3.2 6.1 3.8	20.3 20.7 24.8 18.7 16.4 14.6 14.2 21.3 25.0 24.0	23.3 19.2 15.4 12.9 13.6 9.9 22.7 16.2 41.2 21.5	14.4 1.3 14.3 1.6 15.0 0.0 14.2 1.6 14.0 0.9	0.3 0.6 0.1 0.3 0.7 0.1 0.3 0.7
24/12 FU						
A Mean: (26) SD: M/M Mean: (7) SD: S Mean: (5) SD: VS Mean: (8) SD: ES Mean: (7) SD:	6.5 2.4 7.6 2.6 8.4 2.5 7.0 2.8 4.6 1.5	10.0 3.8 12.4 1.9 10.0 3.4 10.0 3.7 6.3 3.7	11.4 16.8 2.0 4.9 2.6 5.2 13.1 15.9 28.0 19.9	22.1 22.7 10.7 9.0 19.6 12.3 20.1 14.5 41.7 29.5	14.4 1.0 14.7 0.5 14.6 0.5 14.3 1.3 13.7	0.3 0.7 0.3 0.7 0.0 0.0 0.1 0.3 0.9
36/12 FU						
A Mean: (10) SD:	7.1 2.0	10.8 4.1	8.2 10.7	24.8 26.9	14.8 0.4	0.1 0.3

TABLE C9.2b: MEAN & SD, MORE REY VARIABLES, SAMPLE B

Recall B. & Interference & Recognition Scores

24/12 FU:	<u>B</u>	A Del	Pro%	Ret%	Reco	<u>F+</u>
B Mean: (10) SD:	4.7 1.9	9.3 4.2	27.1 20.3	18.2 13.8	13.0 2.9	0.8 1.0
36/12 FU: B Mean: (10) SD:	6.4 3.1	10.1 5.1	14.8 18.8	19.4 29.5	14.0 1.6	0.9 1.9

B= List B score: A Del= Recall after Interference: Pro%= Proactive Interference: Reco= Recognition: Ret%= Retroactive Interference: F+= False Positives:

TABLE C9.3: T-TESTS, REY AVLT, SAMPLE A

Recall Scores on List A trials

1/12 F	<u>U</u> :		<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
M/M(8) M/M S	v	S(7) VS(6) VS		1.637 <1 <1	1.227 <1 <1	1.707 <1 <1
M/M	_	S(7) VS(9) ES(4) VS ES	1.191 1.283 <1 2.492** <1 1.243	<1 1.181 1.346 <1 1.362 1.937*	1.383 1.421 2.348* <1 1.179 1.437	<1 1.012 2.221* <1 1.607 1.579
M/M) v v v v	S(10) VS(9)		1.493 <1 2.300** <1 2.232** 1.291	<1 <1 2.653*** <1 2.113** 1.997*	<1 <1 3.919**** <1 2.867*** 2.749***
12/12 M/M(10 M/M M/M S S VS) V V V V		<1 <1 2.087* 1.247 1.678 2.209**	<1 <1 2.894*** <1 2.590** 3.470****	<1 <1 3.879**** <1 2.769**** 3.617***	< 1
	V V V V		< 1 < 1	<1 <1 2.568** <1 1.907* 1.600	<1 <1 4.368**** <1 2.879*** 1.878*	<1 1.041 3.95**** <1 2.022* 1.790*
* OI	_	سان سان			04	

TABLE C9.3: T-TESTS, REY AVLT, SAMPLE A (cont)

Recall Scores on Lists A & B

<u>1/12 FU</u>	<u>J</u> :	<u>A5</u>	Total A	<u>B</u>	A Delay
M/M	v S(7) v VS(6) v VS	1.300 <1 <1	1.371 <1 <1	<1 1.290 <1	1.744 1.383 <1
M/M M/M S S	J: V S(7) V VS(9) V ES(4) V VS V ES V ES	1.542 <1 2.612** <1 1.657 2.299**	<1 <1 1.986* <1 1.460 1.737	1.720 <1 1.365 2.218** <1 1.562	<1 <1 2.409** <1 1.598 1.789
M/M M/M S S	J: V S(10) V VS(9) V ES(10) V VS V ES V ES	<1 4.104**** <1	<1 <1 3.996**** <1 3.049*** 2.314**	<1 <1 2.327** <1 2.028* 1.740*	<1 <1 4.300**** <1 3.632**** 3.141***
M/M M/M S S	FU: V S(8) V VS(10) V ES(11) V VS V ES V ES	<1 3.288**** <1	<1 <1 3.782**** <1 2.949**** 3.491****	1.358 1.645	<1 1.184 3.69**** <1 3.21**** 2.409**
M/M M/M S S	TU: V S(10) V VS(8) V ES(7) V VS V ES V ES	1.591	<1 <1 3.469**** <1 2.317** 1.870*	<1 <1 2.679** <1 3.057**** 2.139*	1.461 1.610 3.88**** <1 1.806 1.921*

TABLE C9.3: T-TESTS, REY AVLT, SAMPLE A (cont)

1/12 F	<u>U</u> :	Pro%	Ret%	Reco	<u>F+</u>
M/M(8) M/M S	v S(7) v VS(6) v VS		1.515 1.760 <1	<1 1.018 1.220	1.219 <1 1.219
M/M	U: v S(7) v VS(9) v ES(4) v VS v ES v ES	<1 <1 1.054 <1 <1 1.186	1.403 1.403 2.102* <1 1.522 1.219	2.067* 1.271 2.915** <1 1.424 1.540	<1 <1 2.415** <1 2.229* 2.061*
M/M	U) v S(10) v VS(9) v ES(10) v VS v ES v ES	< 1	1.313 <1 2.861**** <1 1.996* 2.096*	1.663 1.540 2.402** <1 1.662 1.056	<1 <1 2.221** 1.496 1.561
M/M	FU:	1.145	<1 1.072 3.131**** 1.401 3.457*** 2.066*	1.334	<1 <1 2.345** <1 <1 2.261**
	FU:		< 1	<1 <1 1.705 <1 1.470 <1	<1 <1 1.247 <1 1.984* 1.868*

TABLE C9.4: CORRELATIONS OF REY VARIABLES AT EACH FOLLOW-UP WITH U/C & PTA, SAMPLE A

Recall Scores on List A trials

		<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
1/12 FU: (n=23) 3/12 FU: (27) 6/12 FU: (41) 12/12 FU: (39) 24/12 FU: (26)	PTA: U/C: PTA: U/C: PTA: U/C: PTA: U/C: PTA:	0.37 0.33 0.48* 0.36 37* 26 10 07 18 13	0.10 0.19 0.44* 0.32 49** 42** 15 01 42* 52**	0.49 0.06 0.42* 0.30 44** 52** 18 0.00 57**	06 0.01 0.41* 0.28 56** 48** 18 0.00 58** 61**
36/12 FU: (10)		29 30	78** 74**	76** 73*	82** 78**

Recall Scores on Lists A & B

		<u>A5</u>	Total A	<u>B</u>	A Del
1/12 FU: (23) 3/12 FU: (27)	PTA: U/C:	18 14 0.40* 0.27	07 .06 16 17	06 08 0.46* 0.35	22 35: 0.41* 0.28
6/12 FU:	U/C:	-0.60**	58**	49**	58**
(41)		-0.60**	52**	47**	54**
12/12 FU:		15	49**	12	18
(39)		0.02	41*	0.01	0.00
24/12 FU:	U/C:	51**	55**	33	47**
(26)	PT A :	56**	59**	41*	53**
36/12 FU:	U/C:	83**	78**	36	89**
(10)	-,	79 * *	75*	33	89**

Interference & Recognition Scores

		Pro%	<u>Ret%</u>	Reco	<u>F+</u>
1/12 FU	: U/C:	.42*	. 25	. 08	. 09
(23)	PTA:	. 23	. 35	01	. 01
3/12 FU	: U/C:	.69**	. 45*	.61**	.68**
(27)	PTA:	. 46*	.41*	.49**	.58**
6/12 FU	: U/C:	. 37*	.43**	. 14	.32*
(41)	PTA:	. 55**	. 35*	. 15	. 29
12/12 FU	: U/C:	.12	. 26	. 00	.00
(39)	PTA:	. 09	. 38*	. 08	. 11
24/12 FU	: U/C:	. 38	. 28	23	. 37
(26)	PTA:	.53**	.42*	35	. 45*
36/12 FU	: U/C:	. 05	.80**	71*	. 52
(10)	PTA:	01	.81**	- .69*	. 53

TABLE C9.5: CORRELATIONS OF REY VARIABLES AT EACH FOLLOW-UP WITH U/C & PTA. SAMPLE B

Recall Scores on List A trials

		<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
24/12 FU: (n=10) 36/12 FU: (10)	PTA: U/C:	24 35 23 45	24 36 47 75**	22 35 36 68*	26 39 51 74**
		Recall S <u>A5</u>	Scores on Li Total A	sts A & B <u>B</u>	A Del
24/12 FU: (26) 36/12 FU: (10)	PTA: U/C:	26 39 38 66*	32 57 41 69*	24 36 28 45	27 41 33 61

Interference & Recognition Scores

		Pro%	<u>Ret%</u>	<u>Reco</u>	<u>F+</u>
24/12 FU:		06	06	22	21
(26)	PTA:	. 05	. 00	36	31
36/12 FU:	U/C:	. 50	. 30	39	. 36
(10)	PTA:	. 24	. 63	63	. 59

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A

D11	Cassas		7 3 - 4	3	4	DT
Recall	ocores.	OH	LIST	м	trials	– KI

1/12	FU:	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +:	33 40 42* 44* 45* 45* 46* 47*	39 48* 52* 53* 55** 55** 57**	36 46* 51* 54* 57** 56** 57**	24 37 40 43* 46* 46* 47* 49*
3/12	FU:				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.16 .39* .96** .96** .94** .96**	.11 .33 .94** .94** .92** .94**	.10 .31 .93** .93** .91** .93** .91**	.08 .30 .93** .93** .90** .93**
6/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	64** 63** 53** 56** 13 11 56** 53**	65**61**48**50**141251**47**	65**63**51**54**071058**52**	63**62**48**53**010353**49**
12/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	02 .07 10 10 09 09 .09	05 .05 13 13 12 12 .06 .12	05 .05 14 14 14 14 .05	05 .05 14 14 14 15 .06

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A (cont)

Recall	Scores	on	List	Δ	trials	_	RT

24/12	<u>FU</u> :	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	58**61**50**53**47*62**50**46*	55**48*51**52**48*50**45*46*	46* 37 42* 43* 45* 40* 36 40*	36 32 40* 42* 31 33 25 27
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	60 57 52 30 52 45 62 52	61 73* 78** 66* 66* 73* 65*	29 51 65* 52 38 42 45	72* 86** 94** 85** 82** 45** 84** 83**
	1	Recall Sco	res on List	s A & B -	RT
1/12	FU:	<u>A5</u>	Total A	<u>B</u>	A Del
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	39 52* 52* 54** 57** 57** 58** 60**	40 51* 55** 58** 60** 60** 62** 63**	31 38 39 41 42* 43* 44*	47*55**56**58**59**58**60**
3/12	FU:			-	
Set	1 +: -: 2 +: -: 3 +: -: 4 +:	.07 .30 .29** .93** .90** .92**	40*29 .43* .44* .36 .41* .37	.13 .36 .49** .95** .93** .95** .92**	.06 .28 .91** .92** .89** .91** .89*

**=p<.01;

*p<.05;

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A (cont)

Recall Scores on Lists A & B - RT

6/12	<u>FU</u> :	<u>A1</u>	Total A	<u>B</u>	<u>F+</u>
Set	1 +:: 2 +:: 3 +:: 4 +::	70** 70** 57** 63** 04 01 63** 60**	71**70**56**60**040161**57**	64**64**55**58**060460**58**	52**53**43**53**050346**45**
12/12	<u>FU</u> :				•
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	04 .04 16 15 15 07	44**40*54**54**60**60**33*30	04 .06 09 09 08 08 .07	08 01 12 14 12 12 .02 .08
24/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	29 13 29 29 17 16 17 16	51**43*4951**43*45*39*40*	56** 49* 60** 46* 58** 52** 47* 47*	30 18 32 31 23 20 10
36/12	F⊍:				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	41 60 69* 51 49 57 61	62 75** 81** 64* 66* 67* 75** 69*	53 46 48 34 47 44 55	18 47 54 55 31 43 39

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A (cont)

Interference & Recognition Scores - RT

1/12	FU:	Pro%	<u>Ret%</u>	Reco	<u>F+</u>			
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	18 19 24 24 24 23 22	.49* .57** .64** .67** .67** .66** .67**	70**78**89**90**91**91**91**	04 03 05 05 02 02 01 03			
3/12	<u>FU</u> :							
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.58** .66** .69** .68** .72** .69** .69**	.47* .59** .55** .55** .56** .55** .54**	.19 .44 .92** .92** .94** .94** .93**	.30 .55** .92** .93** .95** .94**			
6/12	<u>FU</u> :							
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.35* .29 .24 .30 .06 .04 .33* .25	.29 .29 .24 .36* .04 .03 .23	.60** .51** .31 .37* .03 .01 .52**	.70** .62** .41** .50** .09 .05 .62** .44**			
12/12	12/12 FU:							
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.20 .26 07 09 .14 .15 .39*	.30 .45** .15 .15 .15 .15 .16 .42**	.02 .09 .12 .12 .20 .21 .19	.07 .15 .17 .17 .27 .27 .27			

^{*=}p<.05; **=p<.01;

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A (cont)

24/12	<u>FU</u> :	Pro%	Ret%	Reco	<u>F+</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.32 .17 .36 .21 .51** .29 .26	. 25 . 15 . 27 . 26 . 19 . 13 . 01	60** 50** 58** 65** 39* 46* 41* 36	53** .54** .37 .48* .42* .54** .29
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	35 38 28 07 28 20 33 27	03 .29 .36 .41 .12 .26 .20	57 71* 72* 47 65* 72* 77** 76**	14 .09 .15 .01 08 .05 .08
		Recall Sc	ores on Lis	ts A & B -	SD
1/12	<u>FU</u> :	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +:	35 35 37 32 39 32 46* 38	38 42 47* 44* 51* 49* 58**	37 40 44 40 49 51* 53* 52*	26 37 35 32 41 41 44*
3/12	<u>FU</u> :				
Set	1 +: -: 2 +: -:	.07 .25 .61** .49**	.01 .20 .57** .45*	.00 .19 .55** .42*	.02 .17 .54** .42*

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH
FU WITH MEDIAN RT & SD, SAMPLE A (cont)

Recall Scores on List A - SD

6/12	<u>FU</u> :	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u> 44</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	29 51** 29 42** 29 27 35* 46**	41* 53** 30 39* 36* 36* 42** 50**	27 48** 26 31* 23 23 46** 52**	29 47** 27 41** 22 21 42** 56**
12/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	06 .38* 09 12 03 06 01	07 .36* 10 15 06 10 03	07 .36* 12 16 07 11 .02 .09	09 .34* 13 16 01 13 .03
24/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.19 41* 18 47* 46* 50** 32 21	10 31 42* 45* 35 41* 22	05 18 36 32 34 39* 23 43*	03 16 29 33 17 32 19 24
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	19 45 53 47 56 52 42 57	64*636168*71*78**77**	30 29 37 46 56 55 74**	71*76**79**84**89**80**81**

*=p<.05;

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE A (cont)

Recall on Lists A & B - SD

1/12	<u>FU</u> :	<u>A5</u>	Total A	<u>B</u>	A Del
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	42 54** 53** 49* 57** 60** 61** 59**	41 45 49* 45* 53* 55** 61**	31 28 34 30 37 41 44*	46**51*54**57**49*69**66**
3/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	03 .17 .53** .40* .50** .54** .34	50**32061913**082901	04 .22 .58** .47* .56** .59** .41*	04 .16 .52** .38* .51** .54** .35
6/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	41**54**32*52**31*31*56**68**	36*55**31*45**303049**60**	45**53**2740*34*36*48**51**	40*34*1842**292545**59**
12/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	06 .38* 14 17 08 11 07 09	26 05 53** 54** 56** 56** 42** 36*	10 .34* 08 13 03 08 02 09	08 .34* 13 16 09 12 05 12

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH
FU WITH MEDIAN RT & SD. SAMPLE A (cont)

Recall	on	Lists	A	8	В	_	SD
--------	----	-------	---	---	---	---	----

24/12	<u>FU</u> :	<u>A5</u>	Total A	<u>B</u>	A Del
Set	1 +:: 2 +:: 3 +:: 4 +::	.20 10 10 23 01 16 05 24	.05 26 32 41* 29 40* 22 32	.35 41* 28 48* 44* 35 25 31	.10 16 07 24 07 17 13 28
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	43 46 45 61 68* 71* 89** 81**	51 59 63* 70* 78** 79** 81** 82**	34 42 49 44 51 63 33 48	54 36 24 62 48 52 83** 61
		Interfer	ence & Recog	nition -	SD
1/12	<u>FU</u> :	Pro%	Ret%	Reco	<u>F+</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	19 22 18 20 16 05 09	.48* .47* .54** .56** .48* .65** .62**	74**67**74**71**74**75**	04 02 .07 .09 .08 .11 .05
3/12	FU:				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.58** .60** .68** .61** .75** .70** .69**	.50** .45* .59** .68** .54** .47* .52**	.13 .29 .67** .59** .77** .77**	.24 .37 .76** .71** .84** .82** .70**

^{*=}p<.05;

^{**=}p<.01:

TABLE C9.6: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD. SAMPLE A (cont)

Interference & Recognition - SD

6/12	<u>FU</u> :	%Pro	%Ret	Recog	<u>False+</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.32* .14 .08 .11 .18 .25 .34* .31*	.28 .06 06 .23 .22 .12 .47**	.08 .27 .18 .42** .02 .05 .29	.15 .35* .20 .45** .11 .13 40**
12/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.29 .45** .04 .07 .20 .29 .37*	.11 .37* .19 .14 .25 .27 .31	06 .19 .13 .05 .18 .11 .09	01 .22 .18 .11 .24 .18 .12 04
24/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	02 .15 .18 .28 .40* .16 .12	.00 .15 .06 .19 .06 .11 .15	.07 38 32 58** 20 33 24 25	.07 .28 .12 .35 .48* .67** .46*
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	.17 .18 21 .16 27 03 23	.42 .19 .04 .49 .30 .31 .72*	38 63 64* 71* 83** 78** 93**	04 06 11 .18 .18 .16 .63

TABLE C9.7: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD. SAMPLE B

Recall Scores on List A - RT

24/12	<u>FU</u> :	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +:	15 23 26 26 17 15 17	16 24 26 27 17 15 17	16 24 26 27 17 15 17	1928303120182130
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	45 50 52 61 47 59 64* 63	39 47 47 57 42 56 56	47 52 56 61 47 60 55	34 44 49 59 43 59 56 52
	Re	call Score	s on Lists	A & B - RT	•
24/12	<u>FU</u>	<u>A5</u> <u>T</u>	Total A	<u>B</u>	<u>ADel</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	18 27 31 31 20 18 20 29	41 51 52 52 42 41 45	16 24 27 27 17 16 18 25*	21 30 32 33 22 20 23 32
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	48 57 61 70* 56 70* 67*	44 52 55 64* 49* 63 62 59	35 38 54 58 42 53 48 39	55 65* 70* 78** 64* 78** 73* 69*

TABLE C9.7: CORRELATIONS OF REY VARIABLES AT EACH FU WITH MEDIAN RT & SD, SAMPLE B (cont)

Interference & Recognition - RT

24/12	FU:	Pro%	Ret%	Reco	F+
Set	1 +: -: 2 +: -: 3 +: -: 4 +:	. 36 . 30 . 31 . 23 . 35 . 37 . 33 . 21	.07 .01 03 03 .07 .08 .08	21 .29 31 32 22 20 23 31	09 .18 20 21 11 09 11
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	30 31 03 06 17 14 16	.71* .81** .84** .87** .77** .91** .77**	67* .78** .85**89**79**09**82**76**	.74** .82** .90** .92** .82** .94** .82**
		Recall o	n List A -	SD	
24/12	<u>FU</u> :	<u>A1</u>	<u>A2</u>	<u>A3</u>	<u>A4</u>
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	18 04 28 23 23 09 14 23	19 04 28 23 24 09 14 24	19 04 28 25 24 10 16 25	22 08 30 27 27 13 18
36/12	FU:				
Set	1 +: -: 2 +: -:	55 59 54 55	50 58 48 54 66*	59 66* 52 60 67*	51 60 50 53 68*
	3 +: -: 4 +: -:	61 33 58 58	47 75** 50	53 75** 52	47 65* 52

TABLE C9.7: CORRELATIONS OF REY VARIABLES AT EACH
FU WITH MEDIAN RT & SD, SAMPLE B (cont)

Recall on	Lists	A &	В —	SD
-----------	-------	-----	-----	----

24/12	<u>FU</u> :	<u>A5</u>	Total A	<u>B</u>	A Del
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	21 08 27 25 27 12 16 26	46 37 38 45 55 39 34	19 05 26 22 23 09 13	24 11 30 28 30 15 18 29
36/12	<u>FU</u> :				
Set	1 +: -: 2 +: -: 3 +: -: 4 +: -:	65* 71* 63 65* 62** 59 69* 63	59 66* 55 60 71* 54 72* 57	44 48 39 40 57 37 21	73* 79** 72* 71* 83** 69* 66*

Interference & Recognition - SD

<u>24/12 FU</u> :	Pro%	Ret%	Reco	<u>F+</u>
Set 1 +: -: 2 +: -: 3 +: -: 4 +: -:	.31 .47 29 .06 .33 .31 .05 04	.06 .21 16 04 .08 .12 .00	24 09 31 29 29 15 20 29	12 .03 26 19 16 03 11 20

36/12 FU:

Set	1 +:	30	.89**	84**	. 87**
	 :	24	.94**	90 * *	.92**
	2 +:	32	.85**	83**	.84**
	-:	31	. 88**	85**	.88**
	3 +:	05	.94**	 .95**	. 97**
	-:	26	.90**	85**	.91**
	4 +:	48	.83**	74**	.73**
		- 20	87**	- AB**	Q1 * *

TABLE C9.8: DIGIT SPAN MEAN & SD SCORE AT EACH FU

		1/12	Follow	<u>-up</u>	3/12	Follow	-up
Gro	up	<u>F</u>	<u>B</u>	<u>Total</u>	<u>F</u>	<u>B</u>	<u>Total</u>
A M/M S VS ES	Mean: SD: Mean: SD: Mean: SD: Mean: SD: Mean: SD: Mean: SD:	6.1 1.2 6.1 1.0 5.7 1.4 6.0 1.1 7.5 0.5	4.5 1.5 4.4 1.0 3.7 1.4 5.5 1.9 4.5 0.5	10.8 2.7 10.9 2.2 9.4 2.5 12.0 3.2 12.0	6.9 1.2 7.0 0.7 6.8 1.1 7.0 0.8 6.5 2.1	5.0 1.5 4.8 1.2 5.1 1.3 6.0 1.1 3.5	12.2 2.6 12.2 1.5 12.3 2.3 13.3 1.8 10.0 3.7
		6/12	Follow	-up	12/12	Follow	<u>r-up</u>
		<u>F</u>	<u>B</u>	<u>Total</u>	<u>F</u>	<u>B</u>	<u>Total</u>
A M/M S VS ES	Mean: SD: Mean: SD: Mean: SD: Mean: SD: Mean: SD: Mean: SD:	6.7 1.3 6.5 1.4 6.4 1.0 7.1 1.0 6.7	5.0 1.3 5.2 1.1 5.0 1.4 5.9 1.3 4.1 0.8	11.9 2.3 11.9 2.2 11.9 2.3 13.2 2.1 10.8 2.0	6.7 1.0 6.6 1.0 6.8 0.7 7.0 0.8 6.7 1.1	5.4 1.4 5.3 1.4 5.8 1.4 6.1 1.5 4.8	12.4 2.2 11.9 1.9 13.1 2.5 13.4 2.2 11.4 1.6
		24/12	Follow	<u>up</u>	36/12	Follow	-up
		<u>F</u>	<u>B</u>	<u>Total</u>	<u>F</u>	<u>B</u>	<u>Total</u>
M/M S VS ES	SD: Mean: SD: Mean: SD: Mean: SD:	0.9 6.5 1.0 7.8 0.4	5.4 1.3 6.4 0.8 5.8 1.1	2.2 12.3 2.2 14.4 1.4 12.9 2.4	7.1 0.7	4.9 1.1	12.0 1.7
Sam	ple B	24/1	2 Follo	w-up	36/1	2 Follo	ди-шр
	Mean: SD:	5.7 1.4	4.1 1.0	9.7 2.2	5.5 1.1	4.9 1.0	10.4 2.0

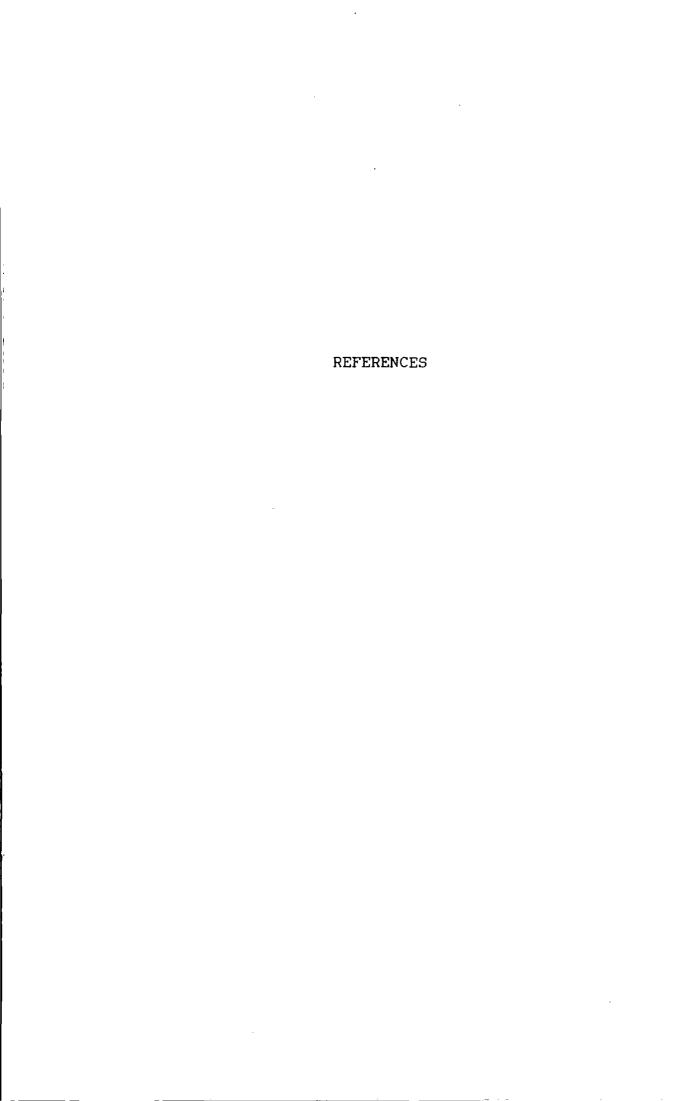
F= digits forward: B= digits backward:

TABLE C9.9: t-TESTS. DIGIT SPAN. SAMPLE A

<u>1/12 FU</u> :	Forward	Backward	<u>Total</u>
M/M(8) v S(7) M/M v VS(6) S v VS	< 1	1.049 1.326 1.913*	1.194 <1 1.590
3/12 FU: M/M(5) v S(7) M/M v VS(9) M/M v ES(4) S v VS S v ES VS v ES	<1	<1 1.819* 1.327 1.382 1.723 2.710**	<1 1.165 1.128 <1 1.124 1.683
6/12 FU: M/M(11) v S(10) M/M v VS(9) M/M v ES(10) S v VS S v ES VS v ES	<1 <1 1.497	<1 1.237 2.538** 1.382 1.735* 3.415***	<1 1.335 1.189 1.297 1.132 2.487**
12/12 FU: M/M(10) V S(8) M/M V VS(9) M/M V ES(9) S V VS S V ES VS V ES	< 1 < 1	<1 1.211 <1 <1 1.587 1.990*	1.151 1.487 <1 <1 1.691 1.997*
24/12 FU: M/M(7) v S(10) M/M v VS(8) M/M v ES(7) S v VS S v ES VS v ES	<1	1.603 <1 1.894* 1.236 3.818*** 2.642**	2.063* <1 1.459 1.475 4.007**** 1.990*
*=p<.05; **=	=p<.025;	***=p< .01;	****=p<.005;

TABLE C9.10: t-TESTS, WECHSLER MEMORY SCALE, SAMPLE A

6/12 FU: M/M(6) v M/M v M/M v S v S v VS v	S(10) VS(9) ES(10) VS	Factor 1 <1 <1 3.464**** <1 3.314*** 2.909****	Factor 2 <1 1.071 1.939 1.574 1.570 3.895****	Factor 3 <1 1.162 1.155 1.095 2.486** 3.596****
<u>24/12 FU</u> :				
s v	VS(3)		3.918**** 3.918**** 1.087 <1 5.353**** 5.353****	<1 <1 <1 <1 <1 <1 <1 <1
*=p<.05;	**=p<	.025; ***=p	<.01; ****	=p<.005;



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