THE STRUCTURE OF RECALL IN AMNESIA

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Notice

The guidelines used in the preparation of this thesis were the Publication Manual of the American Psychological Association, Second Edition, (1974), and Sternberg, R.J. (1988). The Psychologist's Companion: A guide to scientific writing for students and researchers. Cambridge University Press: Cambridge.

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DECLARATION

The material contained in this thesis has never been previously submitted for any higher degree and represents the author's own work under the supervision of Dr. Andrew Mayes for experiments 1- 6 carried out in 1986 and 1987 and Professor Gregory Jones for experiment 7 and writing and revising the thesis in 1988-89. The design and analysis of experiment 7 were carried out jointly with Professor Jones.

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Summary

This thesis reports seven experiments on the nature of the functional deficit in amnesia. Experiments 1 to 3 investigate patterns of recall for amnesic subjects and matched controls to investigate a hypothesised specific deficit in recall in amnesia. No significant evidence of a recall deficit in amnesia was found. However, a difference emerged between the two groups in the analyses of the stochastic relationship between recall and recognition. This revealed that in amnesic subjects recall is approximately independent of recognition, whereas in control subjects they are positively related.

The second three experiments investigated a hypothesised selective deficit of spatial memory by comparing amnesic and control memory for the locations of objects or words placed on a grid. The hypothesis that intentional encoding of locations would improve amnesic spatial memory scores resulting in a trade-off of recall and recognition of the item's identities was also examined. No significant evidence of a selective spatial memory deficit in amnesia was found, nor did intentional instructions improve amnesic spatial memory scores. There was no significant evidence of a trade-off of item and location memory in the amnesic group. A further analysis comparing control and amnesic memory for the location of items scored by lenient criteria found no significant difference between the amnesic and control scores for number of items of this type, or for recall and recognition memory of these items .

Fragment and schema models have been applied to normal memory for this type of contextual material. In a final experiment, the predictions of both types of model were contrasted with each other for data on singly and multiply cued recall provided by both normal and amnesic subjects. It was found that amnesics and normal controls formed fragments representing the unrelated triads and schemas representing the related triads. Both the schema and the fragment model parameters displayed uniform patterns of impairment. Thus amnesic memory may be argued to differ from normal memory quantitatively, rather than qualitatively . The implications of these findings for theories of memory and processing in amnesia are disscussed.

CHAPTER ONE

INTRODUCTION

Organic amnesia is memory impairment owing to brain dysfunction which may be caused by lesions or by biochemical abnormality. The impairment usually affects acquisition of new memories post-traumatically, and this is called anterograde amnesia. The disruption of memories which were acquired pre-traumatically is called retrograde amnesia. The resulting impairment may be global, where all processes of memory are affected; or specific, where many memory operations are spared. This thesis reports experimental work on a group of organic amnesics including Korsakoff patients, aneurism patients, a post-encephalitic patient and a victim of a road traffic accident. Concentrating on anterograde deficits, these experiments investigate specific impairments of recall and spatial memory. The empirical work is concerned with a theory of human organic amnesia which is known as the context memory deficit hypothesis.

This chapter begins by describing some medical conditions which result in organic amnesia including Korsakoff syndrome, cerebro-vascular accident, post-encephalitic amnesia, Alzheimer's disease, and Huntington's chorea. The information on aetiology introduces a discussion of the anatomical correlates of organic amnesia.

The Aetiology of Amnesia

Korsakoff Syndrome Trimble (1981) provides a comprehensive description of this syndrome. He notes that the first patients described by Wernicke (1881) who were suffering from this condition were two alcoholics and a case of sulphuric acid poisoning. The symptoms were failure of muscle co-ordination (ataxia), blood clots of the retina (opthalmaplagia), reflex scanning of the eyes when not in motion (nystagmus), pain, loss of sensation and weakness (polyneuropathy), and confusion. This condition became known as Wernicke encephalopathy.

Korsakoff (1889) identified similar symptoms as being associated with excessive vomiting, typhoid fever, or intestinal obstruction. He

believed that any toxic substance affecting the peripheral and central nervous system could be sufficient to cause the syndrome.

Both Wernicke and Korsakoff were unaware that their eponymous conditions often occurred successively in patients. Today alcoholic Korsakoff patients are believed to suffer several acute Wernicke episodes over a period, followed by the relatively stable Korsakoff syndrome. For this reason the condition is often known as the Wernicke-Korsakoff syndrome.

There is debate as to whether the damage which results in this amnesia is caused directly by the toxic effects of alcohol as would probably have been claimed by Korsakoff and Wernicke. Indeed, most recent studies have revealed the role of thiamine deficiency in the atrophy of the brain in this condition. Alcoholics receive so many calories from the alcohol they ingest that they tend to eat little, and become malnourished. Furthermore, alcohol interferes with the intestinal absorption of thiamine. Thiamine is essential for a step in the elimination by carboxylation of pyruvate, the accumulation of which within a brain cell causes irreparable damage. This avitaminosis found in alcoholics would be likely to damage the diencephalon, brain stem and cerebellum. Not all alcoholics become Korsakoff patients, and it is thought that this may be owing to an inherited deficit in Transketolase, which makes some alcoholics abnormally sensitive to thiamine deficiency (Mayes, 1988). The lesions responsible for the amnesia of Korsakoff syndrome are discussed in a later section on neuroanatomy.

Post-encephalitic amnesia This condition results from infection of the brain by the herpes simplex virus which preferentially localises in the medial temporal lobes. Parkin (1987) describes damage to the hippocampus, amygdala, and uncus which is a structure intimately linked to the hippocampus and the limbic system, and also frontal lobe damage. The lesions implicated in the amnesia of this disorder are discussed further below in a later section on neuroanatomy.

Parkin further notes that in more severe cases the Kluver-Bucy syndrome may even result. Patients will then suffer from amnesia, hyperorality, visual agnosia, and altered sexual behaviour. Detailed discussion of this amnesia is found in Rose and Symonds (1960) and in Cermak (1976) and Cermak and O'Connor (1983) who report a patient

known as SS. The patient is not intellectually impaired, and has no short-term memory deficit. He is unable to remember day-to-day events, is disoriented in time and place outside his own home, and has retrograde amnesia for both public and private events. Cermak and O'Connor contrasted his performance to that of Korsakoff subjects in that he possesses "... an above normal ability to analyse information which helped him retain a limited amount of verbal material for short intervals". When his short-term memory capacity was exceeded, his performance was reduced to the poor level of Korsakoff subjects. On the other hand, his performance is similar to that of Korsakoff subjects in a test of false recognition (Underwood, 1965) which is often employed by Cermak and his co-workers (for example, Cermak, Butters and Gerrein, 1973). SS was more liable to recognise words falsely as having been repeats when they were, in fact, homonyms. His recognition was not improved by semantic analysis during learning and this is also a feature of Korsakoff subjects' memory performance. Cermak and O'Connor concluded that although SS can analyse information at input to the extent of temporary retention, this does not facilitate long-term retention. Furthermore, reminders of the analysis he previously carried out do not help him to reconstruct a memory of the material, and he is unable spontaneously to generate possible responses at retrieval.

Cerebro-Vascular Accident This includes emboli, infarctions and haemorrhages. An embolism is a fragment of a blood-clot or an air bubble carried along in the blood stream. An infarction is congestion or blockage of a blood vessel on which part of an organ depends, resulting in death or scarring of the affected starved tissue, the area of lost tissue being called an infarct. The condition arises in places where small arteries do not communicate with each other, and in most cases the work of a blocked vessel may be taken over by others. A haemorrhage may result when an aneurism is ruptured. An aneurism is a bulging of the wall of an artery, and may be balloon-shaped or extend along an artery.

Memory problems result after a sub-arachnoid haemorrhage, where bleeding from a ruptured intercranial vessel leaks into the sub-arachnoid space located between the meninges of the brain. More than half of these patients have ruptured aneurism. The most common site for a brain aneurism is the anterior communicating artery. This is located

roughly behind the bridge of the nose, and is part of an important circuit of arteries known as the Circle of Willis. The region of the brain thought to be implicated in this amnesia is the septum. This has projections to the hippocampus and mediates its activity (Damasio, Graff-Radford, Esliger, Damasio, and Kassell, 1985).

This thesis presents experimental evidence from a group of amnesics including seven Korsakoff patients, two anterior communicating artery aneurism patients, one post-encephalitic, and a head-injured patient. Although the subject group does not include patients who suffer from either Alzheimer's disease or Huntington's Chorea, a brief description is included here to elucidate later discussion of experiments which do include such subjects.

Alzheimer's Disease This condition is the commonest variety of pre-senile dementia. It affects people in the 50's to 60's age group, and it is commoner in females. Dementia is defined as impaired intellectual ability consequent on organic brain disease which is often progressive and in its later stages affects social behaviour and personality. Although there is some evidence of familial inheritance of this condition, in the majority of cases multifactorial inheritance is thought to be more likely. The disease is gradually debilitating, the first signs being amnesia and lack of spontaneity in responses. These symptoms develop over five to ten years to very apparent dementia, often with aphasia, alexia, and apraxia. The dementia once fully developed, the patient may no longer even recognise his or her own image in a mirror, and will claim a stranger is present. Patients show an abnormal EEG, and at autopsy Alzheimer's plaques and tangles are seen. The plaques are found in the frontal lobes, hippocampus and amygdala. The tangles are found in the frontal and temporal cortex.

Recent research characterises the disease as a biochemical disorder involving abnormal neurochemistry, as patients have particularly low levels of the neurotransmitter acetylcholine. This hypothesis was confirmed by Drachman (1977) who induced an Alzheimer-type dementia in young volunteers using the drug scopolamine which impairs cholinergic function and reduces levels of acetylcholine.

Huntington's Chorea This hereditary disease affects males and females equally, becoming apparent between the ages of 35 and 42 years. The most obvious symptoms are disorders of movement and tics leading to ataxia, and loss of balance. Although the movements are lessened in sleep, it is a unique feature of the condition that the movements continue throughout the night. Many patients show personality changes, with various cognitive deficits leading eventually to dementia.

The pattern of dementia is different to that of Alzheimer's disease, and the existence of attendant memory problems is disputed. Thus, Aminoff, Marshall, Smith and Wyke (1975) have studied 11 patients using WAIS and memory tests and concluded that the intellectual deterioration was similar to that naturally occurring with age. No selective deficit in memory was recorded. In contrast, McHugh and Folstein (1975) found with their eight patients increasing difficulty in problem solving, a verbal performance deficit on WAIS, and at this stage, marked memory problems. Their patients were often correctly oriented in time and place, but performed badly on tasks requiring attention and concentration. They were unable to repeat parts of well-known stories or do mental arithmetic in which intermediate results of calculations must be recalled and used later in the procedure, for example, carry-over subtractions. Caine, Hunt Weingartener and Ebert (1978) have noted that patients seem to lose "finely detailed memories for things". This suggests a specific difficulty in retrieving contextual information which is consonant with a recent hypothesis of the cause of amnesia (see Mayes, Pickering and Fairbairn, 1985). Huntington's chorea patients are easily overwhelmed by information, requesting slower presentation, which may also suggest reduced memory capacity.

The Anatomical Correlates of Amnesia

The next section summarises the principal areas of the brain thought to be associated with amnesia.

Lesions of the Neocortex The cortex is the convoluted surface of the cerebral hemispheres and in humans it it is comprised mainly of neocortex. The neocortex is usually divided into sensory, motor, and

association areas. This area receives input from sensory and motor neurons and interprets and integrates it, and there are projections from the neocortex to the limbic system and basal ganglia which are described below. The neocortex is therefore involved to some extent in almost every step of information processing, however, in amnesia, attention is particularly focussed on the role of the frontal and parieto-temporal-occipital (PTO) cortex.

Mayes (1988) explained that the connectivity of the frontal lobes, and their role in planning and ordering motor responses (Luria, 1973) suggests their capacity to store complex, well-established, scripts or plans of action. Like frontal cortex, the PTO cortex receives input which is at a fairly late stage of processing. Thus damage to this area results in impairment of well-established information which involves the integration of a variety of sensory and motor information, such as agnosias. Agnosias disturb the ability to interpret sensory information, even though sensory processing and comprehension of instructions are intact. For example, a patient can still perceive objects normally, but can no longer understand their meaning or purpose (Humphreys and Riddoch, 1987). A specific type of agnosia is autotopagnosia. In this condition, a patient is unable to point to his or her own body parts to command, and although able to name the component parts of a face, is unable to assemble a model face correctly (Ogden, 1985). This is not, therefore, a verbal deficit, but it is described as an impairment of body-image, or an inability to decompose a whole into its component parts (Mayes, 1988).

Warrington and Weiskrantz (1982) further suggest that amnesia results from the disconnection of the frontal cortex from the PTO cortex by lesions of the limbic system and diencephalic structures. This renders the amnesic unable to access the planning capabilities of the frontal lobes, which means that memory requiring anything more than routine operations, such as elaborative encoding, is no longer possible.

DeRenzi (1982) argued that some cortical lesions result in long-term memory deficits. Focal lesions of the association neocortex may cause amnesia for faces, colours or spatial location. This difficulty also obtains both for material acquired both pre- and post-traumatically. Mayes (1984) suggests that assuming these deficits are not due to perceptual difficulties, this may be explained in the following ways. First, it may be

that the specific storage units in the neocortex have been disconnected from limbic structures vital to storage and retrieval of memories. However, this does not account for normal memory of other material. Second, Mayes goes on to suggest that perhaps those cortical regions housed a specific storage system for that type of information, and that this storage system is destroyed.

Mayes (1988) also describes studies of cerebral metabolism, blood flow and blood volume from PET scans. These studies show that the metabolism of the neocortex is reduced in both diencephalic and temporal lobe amnesics. Furthermore, a patient with transient global amnesia demonstrated reduced cerebral metabolism during her attack which returned to normal when her memory recovered (Gazzaniga, 1984).

Lesions of the Diencephalon The diencephalon consists of the hypothalamus, thalamus and mammillary bodies.

Diencephalic amnesias include those involving thalamic lesions. For example, the famous amnesic patient NA suffered memory loss after a stab wound which destroyed the left dorso-medial nucleus of the thalamus, and may also have damaged the mammillary bodies (Markowitsch, 1985). Further lesions of the thalamic region include those around the third ventricle (Rodrigues and Lawson, 1982).

Amnesia has also resulted after tumours have damaged the mammillary bodies (Khan and Crosby, 1972). There has been much controversy as to the role of damage to the mammillary bodies in amnesia. Early theories of amnesia stressed temporal lobe damage as being most important in causing amnesia, particularly lesions of the hippocampus. The mammillary bodies are situated at the posterior end of the hippocampus and claims that damage here is implicated in amnesia were thus consistent with what was known about Papez' (1937) circuit. This circuit was described as a route from the hippocampus to the mammillary bodies, then via the thalamus to the cingulate gyrus and back to the hippocampus. Damage to this circuit was believed to be sufficient to cause amnesia. Thus in this characterisation the role of temporal structures was stressed rather than the role of the mammillary bodies which are diencephalic structures. These early theories implicated the mammillary bodies only to the extent that they were joined to the temporal lobes.

However, the contemporary theories described below call attention to diencephalic structures. For example, Victor, Adams and Collins (1971) report Korsakoff patients who suffer lesions of both the dorso-medial nucleus of the thalamus and the mammillary bodies. These patients are amnesic and yet have no temporal damage. This would argue that the mammillary bodies are implicated in amnesia by virtue of their being diencephalic structures. Indeed, Korsakoff patients are often known as diencephalic amnesics.

The picture is not quite so clear-cut as may be supposed, as Victor, Adams and Collins (1971) in fact concluded that damage to the mammillary bodies was *not* necessary to cause amnesia. This was because they also studied five cases of patients with mammillary body damage only and these patients had no corresponding memory loss. In contrast, 38 of the patients in their study with dorso-medial thalamic lesions did suffer amnesia. Thus the role of lesions to the dorso-medial nucleus of the thalamus was emphasised in their paper.

Furthermore, Squire and Moore (1979) performed a CAT (computerised axial tomography) scan on a patient who sustained a wound through the nostril, penetrating to the base of the scull. This wound missed the fornix, leaving Papez' circuit intact. Because the mammillary bodies are part of this circuit, it was therefore assumed that they too were intact. The wound did penetrate the dorso-medial nucleus of the thalamus resulting in severe anterograde amnesia. This was clear evidence of amnesia with an intact Papez' circuit, and it undermined claims that damage to these structures is both necessary and sufficient to cause amnesia. Furthermore, damage to the mammillary bodies was also dismissed as a causal factor in amnesia.

However, although the evidence cited above suggests that mammillary body lesions are *not* causally related to amnesia, the claim has been challenged by Mair, Warrington and Weiskrantz (1979). In their study they found evidence of mammillary body damage in patient HJ, a Korsakoff patient, and in patient EA, who suffered from diabetes and Korsakoff syndrome. But although they found lesions in the medial thalamus, they could not be certain that this corresponded to the lesions found by Victor et al. (1971). This is because defining the boundaries of the medial dorsal nucleus of the thalamus is fraught with difficulty and also because the Victor et al. (1971) study does not state how many of their

patients suffered damage to the peripheral areas to the medial dorsal nucleus. The Mair et al. (1979) study concluded that conjoint lesions of the mammillary bodies and midline thalamus are likely to result in amnesia, but they were disinclined to implicate the medial dorsal nucleus specifically.

Lesions of the Temporal Lobes The temporal lobes contain the limbic system, which includes the hippocampus, and the basal ganglia, which contains the amygdala. An important tract of axons called the fornix connects the hippocampus with the anterior thalamic nuclei and mammillary bodies in the diencephalon.

Temporal lobe amnesics include post-encephalitic patients who sustain damage to the hippocampus, amygdala and uncus, while leaving the diencephalon intact. Bilateral temporal lobectomy, where the hippocampus and amygdala are removed, also results in amnesia. The extensively studied patient HM underwent this surgery for the relief of severe epilepsy. It was thought that his severe anterograde amnesia was caused by the resulting damage to his hippocampus (Scoville and Milner, 1957).

There have been alternative suggestions, notably that of Horel (1978) who pointed out that in such studies implicating lesions to the hippocampus in memory loss, the lobectomies must have involved damage to the temporal stem. This connects with the amygdala and temporal neocortex and thence with several other structures, including the basal ganglia and medial thalamus. It is known that Korsakoff patients show damage to the medial thalamus resulting in anterograde amnesia (Victor, Adams and Collins, 1971), and thus Horel claimed that his thesis united evidence from temporal lobe and diencephalic amnesias. However, his suggestion has so far received no support from lesion studies with primates (Zola-Morgan, Squire and Mishkin, 1982).

A further alternative has been suggested by Warrington and Weiskrantz (1982). Their Cognitive Mediation Hypothesis suggests that amnesia involves a disconnection of the temporal lobe from the frontal lobe by lesions in the area of the fornix-mammillary body route. They claim that the temporal lobes house the semantic memory system which is accessed and searched with the aid of a mediational system in the frontal lobes. This is an attempt to unify evidence for the importance of the

temporal lobes in memory with data demonstrating the role of the frontal lobes.

In contrast, Rawlins (1985) reiterates the role of the hippocampus in memory functioning. His argument is that there is a small, limited-capacity, limited-duration, short-term store in memory, which normally functions in parallel with an intermediate-term, higher capacity, memory buffer in the hippocampus. This buffer normally associates two or more temporally discontinuous events before they are sent for storage in longer-term memory. It may also hold large numbers of items, if they require only temporary storage. The buffer has a fairly slow decay rate, whereas the other memory store has a faster rate of decay. Therefore when the hippocampus is damaged there still remains a memory processor which although it is less efficient, it can still account for residual learning and memory capacity found after hippocampal damage. Since the hippocampal buffer was responsible for forming associations across time, then there is difficulty in creating new long-term memories.

The above debate as to the relative roles of the diencephalon and temporal lobes has been resolved to some extent by Mishkin's (1982) theory. This theory is influential for two reasons. First, it provides a coherent account of the parts played by the main brain structures thought to be concerned with memory. Second, it has been used as strong anatomical support for an influential hypothesis of the cause of amnesia known as the Context Deficit Hypothesis. This will be described in greater detail in a section devoted to discussion of various theories of amnesia. A brief description of this characterisation of memory function is that an encoded memory consists of a target memory and its context, which may include details of the room in which the memory was formed; the time of day of the memory's formation; or the colour of the encoded memory item. Mishkin argues that contextual information is vital in forming and retrieving memories. Thus what is disrupted in amnesia is the ability to process this information, which disables memory functioning. He claims that there are two independent types of context memory deficit, and argues that contextual memory is still moderately efficient if one of these deficits is present. However, if both obtain, then severe amnesia results.

Mishkin describes two routes through which information is processed. The first focuses mainly on the amygdala and dorso-medial nucleus of the thalamus. The second involves the hippocampus and

anterior nuclei. They are summarised by Mayes (1988) as follows. One route passes from the association neocortex to the hippocampus, hence to the mammillary bodies via the fornix, then to the mammillothalamic tract to the anterior thalamus from where it projects to part of the ventromedial cortex. It is claimed that this system plays an important part in spatial memory. The other route passes from PTO association cortex to the amygdala, then to the magnocellular dorsomedial thalamic nucleus, and on to another part of the orbito-frontal cortex. This system is described as being important for temporal memory as well as in associating target events with reinforcement and making cross-modal associations. Damage to either system results in deficit to the type of context memory which that route mediates. Furthermore, if both routes are damaged, this causes a severe chronic deficit in target memory.

The intention of this chapter has been to review what is known about the role of specific brain areas in memory. This has been achieved first by considering the aetiology of amnesia as a result of illness or injury. Second, some experimental evidence was considered which investigated lesions to some brain areas thought to be associated with memory processing; namely, the neocortex, diencephalon, and temporal lobes. The next chapter provides a critical review of cognitive theories and experiments in the human and animal amnesia literature.

CHAPTER TWO

COGNITIVE THEORIES AND RESEARCH IN AMNESIA

This chapter reviews current influential theories of amnesia. It examines the consolidation hypothesis, the retrieval deficit hypothesis, the encoding deficit hypothesis, the cognitive mediation hypothesis and the context memory deficit hypothesis. The order of the sections is roughly chronological and the length of each section reflects the extent of that theory's influence and the research that it has stimulated. This is particularly the case for the retrieval deficit hypothesis which is given an extended discussion in this chapter because of the body of research it generated and its historical significance in the development of theories of amnesia.

The Consolidation Hypothesis

The consolidation hypothesis is the earliest of the major modern theories of amnesia. The roots of this theory may be discerned in the work of Hebb (1949). He postulated that a stimulus could result in continued reverberation of certain neural circuits, resulting in a structural change in the neural network. This structural change would occur with continued exposure of the stimulus in learning. Milner (1968) suggested that the patient HM was able to form the reverberating circuits, but that this reverberation did not result in any permanent structural change.

Amnesics were shown to be capable of maintaining information for as long as they could rehearse it, but could not do so after any distraction. It was argued that this was because the structural change had not occurred and the reverberating circuits were not effected. Other supporting evidence for Milner's suggestion was claimed in Marslen-Wilson and Teuber (1975). This study found that there was a sparing of remote memories in retrograde amnesia. Retrograde amnesia refers to the loss of pre-traumatic memories. Thus it was argued that those memories consolidated before the brain damage were still retrievable, whereas no consolidation of new memories had occurred since that

damage. To account for this, it was argued that older memories would have received more reverberation over time, and thus become more consolidated.

Further evidence for the consolidation hypothesis was drawn from work on animals who had undergone ECS and humans who had undergone ECT (see, for example, Pearlman, Sharpless and Jarvick, 1961; Flexner, Flexner and Stellar, 1963; Chorover and Schiller, 1965). The arguments concerning the animal evidence are necessarily indirect, as the studies are not so much looking at the performance of amnesics, as extrapolating from a temporary amnesic state in animals which appears to mimic organic amnesia. The claim was that ECT disrupted the consolidation of traces, and indeed the evidence is quite suggestive, especially when short duration stimuli are used. In this case memory is disrupted for the few seconds immediately prior to the electrical stimulus and it was argued that the burst of electrical activity had prevented consolidation of the information input just before the treatment.

The consolidation hypothesis had its limitations, the main problem being that the time-course for consolidation has proved very difficult to establish (see Pearlman, Sharpless and Jarvick, 1961; Flexner, Flexner and Stellar, 1963; Chorover and Schiller, 1965). When longer durations of stimuli up to ten seconds were employed, retrograde amnesia ensued for periods of weeks before the ECT. Since these memories must have been consolidated some time before treatment, it must then be argued that the ECT de-consolidates these traces. Thus not only does ECT prevent the consolidation of traces which are about to be added to long term memory, it must also work retrospectively. Consolidation theorists had difficulty in postulating how and why this retrospective disruption occurs.

Another key problem with consolidation deficit explanations of amnesia is shown in the recovery from retrograde amnesia after ECT, where "shrinking retrograde amnesia" is apparent. Here, recovery of memories occurs in order of time, with older memories returning first and more recent memories perhaps never being recovered (Russell and Nathan, 1946). This phenomenon is difficult to explain using the strict version of the consolidation hypothesis. However, it was suggested that storage was a gradual process which occurred over a long period at the biological level, and that the ECT treatment was preventing the gradually incremental process of consolidation over time.

Another problem for the consolidation theory is that some forms of amnesia are reversible. This has been shown with retrograde amnesia in Korsakoff amnesics by Victor, Adams and Collins (1971), and in anterograde amnesia, or the inability to form new memories after brain damage, by Lewis, Misanin and Miller (1968). Proponents of the consolidation hypothesis did not explain why disrupted consolidation was not always permanent.

The consolidation hypothesis was weakened still further by studies of residual learning in amnesia, and demonstrations of near-normal performance in some types of memory tasks. This evidence is discussed below.

Exploration of the **retrieval deficit hypothesis** of amnesia forms the major part of the work of Warrington and her co-workers during the 1970's. Consequently there is a wealth of important data and experiments to be considered. This research is described in detail because of the contribution it made to the development of modern theories of amnesia. The following summarises the supporting evidence for the retrieval deficit hypothesis.

Demonstrations of Residual Memory Warrington and Weiskrantz (1968a) presented amnesic patients with repeated trials of successive lists of words, each of which they were asked to recall and recognise after varying intervals. Their findings were difficult for the consolidation hypothesis to encompass, because they found that approximately 50% of the false-positive recognition responses were in fact prior list intrusions. Thus the amnesics must have encoded a memory of the earlier list material, and therefore consolidated some traces. An explanation of the phenomenon was given in terms of prior learning interfering with new learning, and amnesics were claimed to suffer from excessive interference. This approach became known as the retrieval deficit hypothesis of amnesia.

Warrington and Weiskrantz (1968b; see also, Weiskrantz and Warrington, 1970a) went on further to investigate amnesic memory for verbal and pictorial material. Their technique of "partial cueing" was to show a series of versions of the stimulus during learning, beginning with a very incomplete representation, and gradually adding more to the picture until the whole stimulus was apparent. Both picture and word

fragments were used. Both amnesics and controls improved their performance as the trials progressed, until all the pictures and words could be recognised in their most incomplete form, and both groups showed significant savings in retention tests on subsequent days. This is important as it shows the amnesics' normal capacity to acquire this ability. It also suggests that they have formed a memory representation which is sufficiently well specified, such that the whole stimulus may be recognised when cued with only part of it. Their results were an embarrassment to consolidation theorists, as it is difficult to explain how if a trace is not consolidated, it may yet be retrieved given appropriate cues.

Demonstrations of the Effects of Different Retrieval tasks: Studies of Cued Recall and Interference Having established that demonstrations of near normal amnesic memory depended on the nature of the retrieval task, Warrington and Weiskrantz (1970) went on to look at the efficacy of different cue types. They discovered their effects did not just hold for cues made from perceptual degradation of the target, but also for initial two or three letter cues to words (Weiskrantz and Warrington, 1970a), and also semantic category prompts (Warrington and Weiskrantz, 1971; Experiment 4: 1974; Experiment 4). Some of these experiments are described in greater detail later in this section.

Warrington and Weiskrantz (1970, 1974) further showed that amnesics are differentially aided by cues in comparison to control subjects. These findings were cited as important support for their claims that amnesics suffered increased PI. As has already been explained, this claim means that previously learnt material is interfering with more recently acquired memories to a pathological extent, this being alleged as the underlying cause of amnesia. Thus, because the cues were less helpful to the controls, it was argued that the amnesics used the cues to help reduce the extreme response competition they suffered, and this allowed discrimination between the target and competing intruding items at retrieval.

Furthermore, Warrington and Weiskrantz (1970, 1974) made even more specific claims, for the following reason. Previous experiments had displayed the cues along with the targets at both retention and testing (see partial cueing experiments above), and the cues were effective. Then by only giving the cues at testing, it was then discovered that the

cues gave their benefits *differentially at retrieval* in other words the cues were more helpful in facilitating recall at testing, rather than providing a richer encoding. So it was argued that this showed that the amnesic deficit lay beyond the stage of initial storage. The cues were claimed to be enhancing an impaired retrieval mechanism, by somehow facilitating selection of the target.

Studies of the Effects of Reduced Response Competition Warrington and Weiskrantz (1974) then went on to discover exactly how the cues were facilitating retrieval. In this experiment, cues of initial letters to target words were given to the amnesics. Some of these initial letters matched four to six simple words including the target, the "narrow set" condition, and some were initial letters which matched ten or more words, including the target, the "wide set" condition.

They found that amnesics were helped relatively more by cues to the narrow set of items than to the wide set of items. This suggests that the probability of an amnesic responding with the correct target was contingent on how many competing alternatives there were at retrieval. In the narrow set condition, there were fewer competing words, so the cues were more likely to elicit the correct target. There was a significant interaction of groups and condition, showing the amnesics were not only poorer than controls at benefiting from cues to the "wide set" but also that they were differentially worse than controls at this measure, in comparison with their relative "narrow set" scores. The demonstration of differential deficits is an important technique in neuropsychology. The argument is that if amnesics show lower levels of memory than controls, very little new information has been provided. However, if they can be shown to have a *selective* differential deficit in a particular function, this suggests that this impaired function is significantly contributing to the amnesic condition. According to such an approach, in the case of the experiment quoted above, the greater response competition experienced by the amnesic group may be a critical functional deficit implicated in the cause of amnesia.

Demonstrations of Increased Proactive Interference in Amnesia Warrington and Weiskrantz (1974) argued that in comparison to controls, amnesics suffered excessive amounts of interference. A specific example of this is proactive interference (hereafter PI, see

Keppel and Underwood, 1962) which occurs when previously learnt information interferes with memory for later information.

The basis for this claim is to be found in the following experiment. The study used two lists of target words, which were constructed from pairs of common words which share the same initial letters. These were specially chosen as they were claimed to be the *only* two words to share these first letters, for example "eno" was used to cue "enormous" and "enough". The initial letters were used as cues to the two lists of target words, and each three-letter cue would obviously cue a word in each list. Subjects were shown the first list three times in succession, and then retrieval was tested after a small filled interval, by cuing with the initial letters. Then the second list was given three times, and retrieval tested again with the same initial letter cues. Subjects were thus required to switch from the first cue-target relation to another response to the same cue.

Warrington and Weiskrantz found no significant difference between amnesic and control performance on the first list. However, thereafter the amnesics persisted in retrieving first list responses throughout the rest of the experiment. For example, if "enormous" were in list one, and "enough" in list two, the amnesic subjects would continue to respond "enormous" to the "eno" cue even after the second list had been presented three times, and was in fact the more recent list.

In an unpublished version of the experiment cited in Weiskrantz and Warrington (1975), Warrington and Weiskrantz also found that when the less common target word of the pair used in the experiment always came first, the same results obtained. That is, it was not the case that amnesics were simply finding the higher frequency word easier to retain. They also showed that when asked to generate both words at free recall, amnesics were more likely to give first list items, whereas controls were more likely to give the more recent, second list items.

Warrington and Weiskrantz argue that this is a clear demonstration of PI, and the earlier material was obviously interfering with retrieval of the later material. However, perhaps interference between the two items would be better shown if at least some of the time second list items were retrieved. As it is, the amnesics never attained in list two their initial level of memory for list one. Also, as error data were not reported, it is not possible to discover if random words were given among the responses, or if all of the errors were errors of commission, or prior list intrusions.

Thus we cannot tell whether both words were encoded, but the first list words dominated at retrieval, or whether the second list words were never encoded. Indeed, it is also the case that many amnesics suffer from "perseveration", or involuntary repetition of certain responses, and it may even be claimed that this is what is occurring, rather than memory failure *per se*.

The claim of excessive proactive interference is a vital component of the retrieval hypothesis of the cause of amnesia. However, it is not clear how Warrington and Weiskrantz would characterise the mechanism whereby a previously encoded trace interferes with a to-be-encoded trace. More specifically, it would have been interesting to have an explanation of the the nature of the interference in the "narrow" versus "wide set" conditions of Warrington and Weiskrantz (1974) described above. In this experiment the initial letter cue either matched the to-be-remembered word plus four to six simple words, this was described as the "narrow set"; or the initial letters matched the to-be-remembered word plus ten or more simple words, and this was known as the "wide set". As interference is a very important concept in the development of Warrington and Weiskrantz' work, it will be useful to examine it in detail.

There are a number of different types of interference, which must be distinguished if we are truly to understand the mechanisms underlying this type of forgetting. In the first instance, in Warrington and Weiskrantz (1974), the interference provoked when initial letters are shared by a number of simple words, only one of which has been recently seen, depends upon phonological similarity, and sometimes semantic similarity. This general principle is illustrated in the following hypothetical example. The initial letters "coo" may denote "cooker" (noun), "cooking" (verb), or "cook" (noun), so it can be seen that these words are confusable by virtue of their phonology and their semantics. The basis for discrimination here is in terms of the word's intrinsic properties, or indeed their organisation in the semantic network.

In contrast, the interference experienced in a typical PI paradigm, for example, Keppel and Underwood (1962), does not share these characteristics. I suggest that here the interference is provoked between two encoded items whose common feature is that both have been recently seen in the context of the experiment. Thus they must be distinguished by reference to their temporal context, and the subject must be able to ascertain which is the more recently encoded item.

Therefore it could be argued that the underlying cause of this type of interference is a deficit in processing contextual information (see Huppert and Piercy, 1976; Mayes, Meudell and Pickering, 1985). This theory claims that contextual information surrounding a target memory is essential in efficient retrieval in normal subjects. As was mentioned earlier, the inability to process contextual information such as temporal or spatial features of a target memory, is claimed in this hypothesis to be the important functional deficit in amnesia. The loss of contextual information renders target memories either totally inaccessible, or prone to interference from other items in memory. The context memory deficit theory is discussed in detail below.

Winocur and Weiskrantz (1976) In this series of experiments, Winocur and Weiskrantz (1976) provided further support for the retrieval deficit hypothesis of amnesia. Using a paired associate learning paradigm, they began with a demonstration of a technique of reducing response competition. Response competition refers to the situation when a number of items from memory are available for output to a particular stimulus, and there is difficulty in establishing which is the appropriate target. Winocur and Weiskrantz alleviated this problem, in experiments one and two of the series, by controlling the number of possible responses at retrieval. This was achieved by combining the paired associates according to rules, which by their nature restrict the number of possible responses to a given stimulus. For example, if the combining rule was that the members of the pair rhymed, as in "peace-niece", then possible competing responses from earlier trials which do *not* rhyme with "peace" can be easily eliminated.

The rules were such that either the items were semantically related, for example, "peace-tranquil"; or phonetically related, for example, "peace-niece". Winocur and Weiskrantz' results showed that even though the rule was not made explicit, amnesics could show excellent initial memory for the first list of these paired associates. It was argued that this was because the possible responses to the first item were constrained by the rules.

However, Winocur and Weiskrantz went on to demonstrate that although first list learning was excellent, learning of the second list was very poor owing to intrusions from the first list. Thus, the benefits of the combining rules of rhyming or semantic relatedness which were able to

reduce response competition, are outweighed by the excessive PI suffered by the amnesics.

This lead to experiment 4 in the series where the technique of reducing response competition was combined with that of increasing the amount of PI. The experiment involved learning paired associate lists under various conditions of training and testing. There were two lists to be learnt under three training conditions. The first list constituted the prior training which would interfere with the second list learning as was hypothesised.

The training conditions were as follows. First, no prior training, as this condition served as a control to demonstrate the effect of proactive interference in later conditions. Here the groups were given one list and then retention was tested 35 minutes later.

The second training condition was prior exposure to unrelated word pairs. This was to show the effect of having previously been presented a list which would *not* be learnt by the amnesics as the pairs were unrelated. The condition shows how much PI is demonstrated in the *absence* of measurable prior learning. The groups were given the unrelated paired associate list four times and then retention was tested 60 seconds later. Then there was a 20-30 minute interval before they were given the rhyming paired associate list four times, and then tested after one minute.

In the third training condition, the first paired associate list was of rhyming pairs, and the second was of semantically related pairs. This condition was to investigate whether the dissociation of the two potentially interfering lists by virtue of their having different linking rules would reduce interference, resulting in relatively improved memory for the second list. In this final training condition, the amnesics and controls were presented with two types of paired associate lists. The phonetic list contained pairs such as "peace-niece", with a rhyming linking rule. Then, in the next list, the link between pairs was semantic. The pairs in this list were constructed using the same first members as those of the phonetic list. Thus, the semantic list contained pairs such as "peace-tranquil", with a semantic linking rule.

In summary, the results of condition one were used as a control for a baseline memory level. In condition two the first list of unrelated pairs showed a poor level of memory in the amnesic group, significantly below that of control subjects, as was expected. Amnesics showed relatively

superior learning of the next, semantically related list, as compared with their previous performance in experiment 1 of the series, when *both* lists were semantically related.

Thus it was shown that when there was very little prior list learning in the first list, amnesic's memory for the second list is significantly better. As well as this, these two to-be-learnt lists were discriminable, as one list contained unrelated pairs, and the other list contained semantically related pairs. This result adds further weight to the claim that the poor list two performance in experiment 1 was in fact due to excessive interference brought about by the similarity of the two to-be-learnt lists.

The most interesting condition in the experiment was condition three. As described above, the method in this case was that although both lists shared the same first member of each pair, the combining rule was different in list one to that of list two. Thus list one may contain "peace-niece", a rhyming linking rule, and list two may contain "peace-tranquil", a semantic linking rule. Here, there were far fewer first list intrusions, even though the rule shift was not made explicit. The amnesics were able to detect the rule shift, and use this information to counteract PI, and to reduce excessive competition at retrieval.

The next section explores in further detail the fundamental premise of the retrieval deficit hypothesis, proactive interference. As it is so important to the development of the theory it will be helpful to investigate the nature and implications of the phenomenon.

The Mechanisms of PI and Release from PI In classic release from PI experiments, such as those of Wickens (1970), PI accumulates during repeated presentations of successive lists of to-be-learnt materials which all share some characteristic. This characteristic may be fairly superficial, such as all material being numbers or letters, or there may be a semantic relationship, for example, all the targets in the lists may be flowers. PI is manifested in subjects' recall scores decreasing with successive lists, and it is argued that this shows how previously learnt material may interfere with the acquisition of new information. This interference may occur because associated retrieval cues to targets are too similar to one another, leading to retrieval failure, or inappropriate intrusions of other items learnt in the experiment (see, for example,

Gardiner, Craik and Birtwhistle, 1972). The importance of retrieval cues in both normal and amnesic memory will be addressed later in this thesis.

Release from PI occurs when a subsequent to-be-learnt list differs markedly with respect to the characteristics shared by its members. This "shift" in the nature of the input may be from letters to numbers, or from garden to wild flowers, and it is associated with a sudden increase in recall for that list. Gardiner, Craik and Birtwhistle (1972) discuss three types of explanation which have been offered to account for release from PI effects.

The first is the attentional hypothesis, proposed by Wickens (1970) in which subjects are perceptually alerted by the change in the nature of the input on the first release trial, producing an orienting response, and the higher arousal which obtains results in the new information being better registered. In terms of the amnesia literature, early claims of attentional sluggishness causing amnesia in Alzheimer subjects have been rejected (see Miller, 1975); however, recent theories have argued that attentional deficits of a more sophisticated type may be implicated in amnesia (Hirst and Volpe, 1984 a, b). These are discussed at length in a later section.

A further explanation of release from PI has been the storage hypothesis, (Posner, 1967), which states that as a result of their similarity, there is increasing amounts of spontaneous interaction between the traces of current items and others from previous trials. It is argued that the release trial items are less vulnerable to this inter-trial interference.

This argument is similar in style to that of the third explanation offered, that of the retrieval hypothesis (Wickens, 1970). Here it is argued that the items during the "PI trials" share retrieval cues, which thus become increasingly ineffective. However, the release trial items initiate fresh cues, which are correspondingly more effective.

These final two explanations would seem to complement the underlying approach of Warrington and Weiskrantz' research on retrieval deficits in amnesia. For example, the explanations are consistent with their emphasis on prior item intrusions (Warrington and Weiskrantz, 1968a). It accords with their emphasis on the fact that demonstrations of near normal performance in amnesic memory may be achieved given the appropriate retrieval method.

The Significance of Release from PI and its Relation to Functional and Neuroanatomical Theories of Amnesia.

Amnesics accrue PI faster than controls for all types of information. Furthermore, it is only under certain conditions that some amnesics may show normal release from PI, whereas controls will demonstrate release from PI under *all* shift conditions, whether the shift is alphanumeric or semantic.

Although it is the case that the protocols for the experiments mentioned above differ so much as to make comparison difficult, there are two important points to be made. One point concerns itself with the question of what functional deficit has been identified by these experiments, and whether this deficit is critical to amnesia. The second point is in connection with anatomical considerations, and the search for the critical lesion in amnesia.

Addressing the functional argument first, Butters and Cermak (1980) gave a possible explanation of why the amnesic phenomenon of release from PI differs from that of controls in important respects. They claimed that failure to show release from PI results from the impoverished encoding capacity of amnesics. Amnesics are claimed habitually to encode items to only a shallow level, and thus will not "notice" a semantic change, as they are only encoding superficial surface characteristics of the targets. However, this explanation does not address why the build-up of PI is much faster in amnesics, and moreover, the encoding deficit thesis itself has been challenged (see, for example, Mayes, Meudell and Neary, 1978, 1980; Meudell, Mayes and Neary, 1980).

If we are to try to argue that failure to show release from PI may be considered as a functional deficit both necessary and sufficient to amnesia, then we must be sure that what we observe in the control population is exactly the same function as that occurring in amnesics. This is because the theoretical rationale would be that this function is intact in non-amnesics, and impaired in amnesics; therefore the disability in this function is contributing to the amnesia in a significant way. Furthermore, if we are to understand this function, we must establish whether release from PI is an "all or nothing" process, which is either intact or impaired, or whether performance on such tasks is on a continuum. Whatever the mechanism for the accumulation and release of PI, it would seem that in amnesics *it is sensitive to intrinsic aspects of*

the target material, whereas in controls this is not the case. Therefore, in contrast to controls, amnesics have been shown at least once not to show release from PI after taxonomic or semantic shifts (Cermak et al., 1974 Kinsbourne and Wood, 1975; Butters and Cermak, 1976; Cermak, 1976; and Moskovitch and Milner {cited in Moskovitch, 1982}).

Neuroanatomical claims have also been made on the basis of this phenomenon. In particular, the issue is important theoretically in neuropsychology because the presence or absence of release effects has been argued to be associated with certain brain lesions. For example, Moskovitch (1982), following Cermak (1976), investigated whether the failure to show release from PI was a result of temporal damage, or whether it was caused by frontal lobe damage. He showed that post-encephalitics with temporal damage, but no frontal damage, show normal release effects. He claimed that Korsakoff patients do not show release effects because of their subsidiary frontal lobe damage. Squire (1982) also reports the same conclusion in a comparison of Korsakoff subjects with ECT patients and NA. In Squire's study, NA and the ECT patients showed normal release from PI, whereas the Korsakoff subjects did not. Moreover, Parkin, Leng and Stanhope (1988) compared an anterior communicating artery aneurism (ACAA) patient with a group of temporal amnesics, Korsakoff amnesics, and controls. They found that the ACAA patient demonstrated very similar performance to that of the Korsakoff subjects in that both failed to show release from PI. They concluded that these two groups both suffered frontal lesions which were responsible for this finding.

If we do not fully understand the functions involved in PI, then the basis for these interesting neuroanatomical arguments is weakened.

Auxiliary Hypotheses of the Retrieval Deficit Hypothesis of Amnesia Although amnesics' increased susceptibility to interference is the main claim of the retrieval deficit hypothesis of amnesia, there are also a number of corollaries concerning other aspects of memory. These are detailed below.

Amnesic short-term memory The retrieval deficit hypothesis claims that amnesics acquire information normally in short-term memory (Weiskrantz and Warrington, 1970b). Baddeley and Warrington (1970) investigated the short-term memory ability of amnesics and controls at

an adaptation of the Peterson short-term memory task (Peterson and Peterson, 1959). In this task a subject is presented with a sequence of three items and required to retain them over a period extending from zero to sixty seconds. Rehearsal is prevented by a distracting task, such as counting backwards. Baddeley and Warrington demonstrated that there was no significant difference between amnesic and control memory performance at any delay. Furthermore, just like controls, the amnesics showed better memory for the more recent items. However, they showed an impaired primacy effect, or memory for the first few items of the list. These items, it is argued, would be in long-term memory. Baddeley and Warrington argued that the results showed intact short-term memory, with impaired long-term memory as shown by the absence of a primacy effect. Moskovitch (1982) argued that amnesics do suffer an impairment of primary or short-term memory. He claims that this is a secondary impairment to their more profound long-term memory deficit, and furthermore, that the short-term memory deficit results from diffuse cortical damage, rather than from the damage which causes the amnesia itself.

Faster forgetting Having argued that amnesics acquire information normally, and have a normal short-term memory, one possible corollary is that they then forget information abnormally quickly. Indeed there is some evidence for this in Baddeley and Warrington (1970). The recency portion of the serial position curve of the amnesics' performance is not equivalent to that of controls. There is very little difference between amnesic and control immediate recall for positions 9 and 10 of a ten-item list, but there is a much bigger difference in recall for positions 7 and 8 of the list. This has been described as "slipping" (Butters and Cermak, 1980), and it suggests a faster rate of information loss in the recency portion of amnesic memory than in controls.

Huppert and Piercy (1982) argue that the retrieval deficit hypothesis entails faster forgetting as an auxiliary hypothesis. They argue that as demonstrations of increased proactive interference have formed an important basis for the retrieval deficit hypothesis, furthermore, proactive interference increases with retention interval, therefore it is argued that amnesics should forget faster than normals. Assuming normal and amnesic performance levels are compared over time, then because there is increasing interference in amnesic memory there will

be less and less target material accessible. In contrast, the rate of loss of target information in the control group will be much less steep. Thus the amnesic group appear to forget faster.

However, this auxiliary hypothesis of the retrieval deficit hypothesis is not supported by the data as Huppert and Piercy (1977) demonstrated that once Korsakoff patients were given training to attain normal levels of performance they did not forget faster than controls. In contrast, Huppert and Piercy (1979) demonstrated faster forgetting in HM, suggesting that temporal lobe damage was associated with faster forgetting. Moreover, Mattis, Kovner and Goldmeyer (1978) compared Korsakoff subjects with post-encephalitic subjects, who are argued to suffer temporal damage. They also found that the group with temporal damage appeared to forget more rapidly. Their recognition performance was at chance, whereas the Korsakoff subjects' recognition was much better. This hypothesis is also supported by Squire (1980) who compared the forgetting rates of Korsakoff subjects, NA, and ECT patients. The Korsakoff subjects and NA were the diencephalic group, and ECT was claimed to disturb temporal lobe function. Squire found that the ECT patients forgot more quickly than NA and the Korsakoff subjects. Furthermore, Parkin and Leng (1987) have also shown that medial temporal lobe amnesics forget more rapidly than Korsakoff subjects. This evidence has been used to argue that medial temporal lobe lesions are associated with faster forgetting, whereas diencephalic lesions are not.

However, there are some conflicting results which are reported in Freed, Corkin and Cohen (1984). They re-tested HM and found he did not forget faster than controls, thus weakening the claim that medial temporal lesions caused faster forgetting. Furthermore, Kopelman (1985) compared the rates of forgetting of control subjects, Korsakoff patients and Alzheimer patients and found no difference between the groups. This weakens both the claim that temporal lesions cause pathologically fast forgetting and the claim that the Alzheimer patients, who suffer both frontal and temporal lobe damage, should forget more quickly than Korsakoff patients.

Retrograde amnesia This is an impairment of memory for information about the amnesic's personal life-history, as well as general knowledge of political or topical affairs from the recent past. The issue is theoretically interesting because the status of an amnesic's retrograde

memory reveals various aspects of memory acquisition in anterograde amnesia generally.

Perhaps as a remnant of early stage models of normal memory (see, for example, Atkinson and Shiffrin, 1968; Waugh and Norman, 1965), remote memory is discussed in the literature as if it were a specialised, discrete, long-term store, and it is implied that information attains this status via processing through short and intermediate-term memory. This characterisation of remote memory lead Warrington and her co-workers to postulate that amnesics would experience a constant level of difficulty in retrieving all old memories, regardless of how long ago they were acquired. This is because the retrieval deficit hypothesis has as an important corollary that information is acquired normally. Warrington and Sanders (1971) argue that subjects' memory difficulties are constant across their entire life history, and they conclude as a result of this that "a unitary functional disorder could account for both retrograde and anterograde effects in the amnesic syndrome" (Warrington and Weiskrantz, 1973: 376). This was a controversial claim and it was challenged by Milner, Corkin and Teuber (1968) and Marslen-Wilson and Teuber (1975) and these studies are discussed in a later section of this chapter.

Memory for motor skills and its theoretical implications Amnesics have been shown to learn motor skills as well as normal subjects. These skills include, mirror drawing (Milner, 1970), pursuit rotor tasks (Corkin, 1968), and learning a new tune on the piano (Starr and Phillips, 1970). Warrington and Weiskrantz (1973: 382) claim that demonstrations of normal memory for such skills provides yet more support for their claims. For example, they quote:

> "One property of motor skills is that retention appears to be remarkably unaffected by interference effects in general (Adams, 1967) and, in particular, proactive interference has not yet been demonstrated (Duncan and Underwood, 1953)."

Thus, Warrington and Weiskrantz' argument is that amnesics suffer excessive PI, and this is the root of their amnesic deficit. Furthermore, any task which is not susceptible to PI, is unimpaired in amnesia.

Summary of Evidence Supporting the Retrieval Deficit Hypothesis

Warrington and Weiskrantz and their co-workers argued that the following phenomena in amnesic memory can be accounted for by the retrieval deficit hypothesis of amnesia.

1. There is evidence of residual memory in amnesics as shown by prior item intrusions during retrieval of a later list of words (Warrington and Weiskrantz, 1968a).
2. Further evidence of residual memory is demonstrated by memory savings at recognition using partial cueing or fragmented versions of the target as a cue to retrieval (Warrington and Weiskrantz, 1968b).
3. Amnesics were differentially aided by cues, and the cues gave their benefits differentially at retrieval (Warrington and Weiskrantz, 1970, 1974).
4. Amnesics showed more proactive interference than controls when the two to-be-learned lists shared the same initial letters (Warrington and Weiskrantz, 1974).
5. Amnesic memory for paired associate word lists could be improved if response competition were reduced by linking the paired associates by rules (Winocur and Weiskrantz, 1976).
6. Proactive interference could be counteracted by making the two to-be-learned lists more discriminable (Winocur and Weiskrantz, 1976).
7. Amnesics showed normal short-term memory in a Peterson paradigm experiment (Baddeley and Warrington, 1970). This suggested that amnesics acquired information normally.
8. In a retrograde amnesia questionnaire, amnesics were claimed to have an equal deficit in memory for all decades in the test, with no sparing of earlier memories (Sanders and Warrington, 1971; Warrington and Sanders, 1971).
9. It was argued that motor skills did not suffer from interference, therefore amnesics should have intact motor skills (Milner, 1970; Corkin, 1968; Starr and Phillips, 1970).

Evidence against the Retrieval Deficit Hypothesis

The Problem of Memory Strength Warrington and Weiskrantz' (1970) claims were challenged by Woods and Piercy (1974) and Squire, Wetzel and Slater (1978) on both methodological and empirical grounds. The methodological arguments are addressed in some detail in Chapter 4 below, as well as forming part of the motivation for Experiments One and Two of this thesis; therefore the points are only briefly described here.

Most experiments which attempt to identify the critical functional deficit in amnesia rely on showing a significant interaction between test and group of the type that demonstrates that amnesics have poorer performance than normal subjects. Huppert and Piercy (1982) point out that it is important to establish that this poor performance is not simply a feature of weak memory in general, and they argue that such effects could be a result of the amnesics' weaker memory. This means that if weak *normal* memory were examined, then any specific functional deficit thought to be responsible for the amnesia may also be demonstrated in normal memory. This would result in an apparent qualitative difference between amnesic and normal memory being in fact a quantitative difference between strong and weak memory. If this were the case, it would erode the fundamental premises of Warrington and Weiskrantz' (1970) arguments, because the functional deficit hypothesised as causing amnesia is observed in normal subjects who are not amnesic.

Woods and Piercy (1974) used Warrington and Weiskrantz' (1970) protocol with normal subjects. They showed a similar group by test interaction in their experiment when performance of normals tested after a one minute delay was compared to normal performance after seven days. They went on to reinterpret Warrington and Weiskrantz' results, suggesting the reason that memory after partial cueing was superior to recognition memory in amnesics was simply that partial cueing was more effective than recognition tests in retrieving weaker memories.

Squire, Wetzel and Slater (1978) also provided support for Woods and Piercy's characterisation of the partial cueing experiments by showing a

similar group by test interaction comparing memory after short delays in post-ECT patients with memory after long delays in normal subjects.

The challenges to the retrieval deficit hypothesis by Huppert and Piercy and Squire and his colleagues were further developed to provide the evidence supporting an alternative theory of amnesia known as the context memory deficit hypothesis. Workers using this theoretical framework have gone on to give evidence of specific deficits in contextual information processing, but they have always made provision in their experiments for control of relative memory strength. This procedure involves ensuring at the outset that control and amnesic memory either for recall or for recognition is equivalent. This technique is known as "matching" and is described in greater detail in Chapter 3 below.

Evidence Against the Increased Proactive Interference Account of Amnesia Huppert and Piercy have been among the main proponents of the context memory deficit theory (see, for example, Huppert and Piercy, 1976), and as such they have emphasised the acquisition stage of memory rather than retrieval. They have favoured an account involving a generalised learning deficit in amnesia. But is this account consistent with demonstrations of intact semantic memory and damaged episodic memory? Surely such a generalised defect would result in equally impaired semantic and episodic memory? Huppert and Piercy (1982) argue that, on the contrary, their approach is not inconsistent with the evidence on which claims of intact semantic memory are based. They explain that semantic memory tests typically probe memories which were encoded many years before the onset of amnesia, whereas tests of episodic memory are measuring retention of fairly recently acquired information. As Huppert and Piercy (1982) point out, what is needed is evidence on pre-onset episodic memory, and demonstrations of amnesic ability to learn new semantic memory entries.

Huppert and Piercy (1982) provide a different interpretation of Winocur and Weiskrantz (1976) results. Winocur and Weiskrantz argue that it is the restraint of the semantic rule which reduces response competition, and improves amnesic memory in their experiment. Huppert and Piercy (1982) suggest that the semantically related pairs represent associations formed many years pre-traumatically, and they are thus very over-learned. This is also the case for amnesics' correct use

of language and motor skills ability. They further claim that this applies to the partial cueing technique. This is because the association between the whole word and a part of the word was formed before the onset of amnesia, presumably when the subject learned to read.

Therefore, for Huppert and Piercy the important distinction is between memories encoded prior to the onset of amnesia, and those acquired after the amnesia. They believe that the cause of amnesia is to be found at the acquisition stage of memory. They describe two experiments in support of their general claim. The first is Rozin (1976), which shows that partial cueing of amnesic memory for two-syllable words is very efficient, but that it is not successful in eliciting two-syllable non-words. The second experiment is an unpublished study by Huppert and Piercy on partial cueing of non-verbal stimuli. These stimuli were not associated previous to the experiment, and partial cueing was found to be unsuccessful in helping amnesic recall.

Evidence Against Sanders and Warrington's (1971) Characterisation of Retrograde Amnesia It was shown by Milner, Corkin and Teuber (1968), that amnesics demonstrated relatively spared remote memory. In other words, memories encoded many years pre-traumatically were still accessible, although information occurring post-traumatically was poorly retained. Thus it was argued that there was a temporal gradient in retrograde amnesia (see, for example, Talland, 1965; Seltzer and Benson, 1974).

This finding posed a problem for a strict version of the retrieval deficit hypothesis of amnesia, because obviously some type of retrieval was very successfully occurring in the case of amnesic remote memory. Also, there was no evidence for increased interference in retrieval from remote memory. It was for this reason that Sanders and Warrington (1971) undertook their study of retrograde amnesia. They found that there was no temporal gradient in retrograde amnesia and their evidence was thus consistent with the retrieval deficit hypothesis. Sanders and Warrington claimed that workers who had found a temporal gradient had done so because they had not controlled for the difficulty of recent versus old remote memories. They claimed that the items testing older memories in experiments such as Milner, Corkin and Teuber (1968) contained events which were more distinct and of longer-lasting fame, than the more recent items.

However, Marslen-Wilson and Teuber (1975) produced further evidence of a temporal gradient in retrograde amnesia. They tested recognition of famous faces from various decades back to the 1920's. Their alcoholic Korsakoff patients had more difficulty with faces from the 1950's and 1960's than with those from the earlier periods.

Squire and Slater (1977) have studied the retrograde amnesia of patient NA, who has suffered a foil wound to the left dorsomedial nucleus of the thalamus, and who is described as a "diencephalic" amnesic. They show that he appears to have virtually normal memory for remote events. Similarly, Scoville and Milner (1957) showed that patient HM, who had an excision of the temporal lobe to relieve epilepsy, also appears to have fairly normal remote memory. In other words both of these patients would show a temporal gradient of performance in a retrograde memory test battery. Thus these studies have shown a temporal gradient in the retrograde amnesia of diencephalic Korsakoff patients, a "pure" diencephalic amnesic, and a temporal lobe amnesic.

Following these studies, Albert, Butters and Levin (1979) attempted to compose a test battery for retrograde amnesia controlling for item difficulty. Their famous faces were divided in to "easy" items, where individuals had been famous for more than a decade, and "hard" items including individuals who had been famous for less than a decade. Subjects were asked the identity of the person in the photograph, and then if unsuccessful they were given a semantic cue, followed by a phonemic cue if necessary. The presentation of hard versus easy items was pseudorandom, and the test proceeded in chronological order, beginning with the 1920's, followed by the 1930's and so on. There was also a recall test in response to questions about old news items, and questions about famous people, and finally a multiple choice test for the answers to questions about people from the 1920's to 1975. The test battery was extensively tested on control subjects in order that all test items were correctly answered by over 80% of the normal subjects. Thus any gradient in the amnesic performance was not due to task difficulty.

The Korsakoff patients showed superior retrieval of more remote facts in comparison with more recent items. This was the case regardless of whether the items were easy or hard, and regardless of method of testing. When a more detailed analysis was made in which memory for pictures of individuals when they were young was compared with memory for pictures when they were old, it was found that Korsakoff

patients were more likely to identify a person from their "young" photograph. Furthermore, Albert et al. (1979) also included a condition where the difficulty of the items is deliberately non-equivalent, and this is the most conservative test of their hypothesis. In this case, when memory for "easy" items from the recent past is compared with memory for "hard" items from the remote past, then it is still found that Korsakoff subjects show superior memory for the harder remote items. Thus the results of Albert et al. (1979) would seem to provide clear evidence which is inconsistent with Sanders and Warrington (1971).

In accounting for their data, Albert et al. (1979) argued that retrograde amnesia in alcoholic Korsakoff patients was at least partly a consequence of chronic anterograde amnesia. Their argument was that a chronic alcoholic patient, prior to developing Korsakoff syndrome, would suffer a mild deficit in acquisition as a result of being almost constantly drunk. This would ensure that personal information, or topical information for the more recent past, may never have been encoded in the first place. In support of this, it has been shown that long-term alcoholics of 10 or more years of alcohol abuse have difficulty in learning complex verbal and non-verbal material (see, for example, Butters, Cermak, Montgomery and Adinolfi, 1977; Ryan and Butters, 1980; Ryan, Butters, Montgomery, Adinolfi and Didario, 1980). Albert et al. (1979) further suggest that when the alcoholic suffers the acute Wernicke-Korsakoff stage of the illness an additional impairment is superimposed upon the already existing deficiency in recent personal and topical memory. This would mean that in comparison with controls, Korsakoff patients would be impaired over even pre-Korsakoff decades, but more recent decades would be more severely impaired.

This view suggests that perhaps with other aetiological groups these results would not obtain. For example, acute-onset amnesics such as head-injured patients would not have suffered a chronic acquisition problem like that of the alcoholic. In this case Sanders and Warrington (1971) may be correct in stating that there is no temporal gradient in retrograde amnesia. Indeed, in consideration of this possibility, Butters and Albert (1982) described an experiment in which patients suffering from Huntington's Chorea were given the Albert et al. (1979) test battery.

Butters and Albert (1982) compared Huntington's Chorea patients' performance with that of Korsakoff patients. They found that unlike the Korsakoff subjects, the Huntington's Chorea group showed a flat gradient

of performance, with equal impairment for all decades in the test. They then went on to consider whether retrograde amnesia performance could be considered as an exaggerated form of normal forgetting. They administered the Albert et al. (1979) test to a group of 50 year olds and a group of 70 year olds. They assumed that forgetting tends to increase with age, therefore the differences between the two groups would reflect normal forgetting rates. They found that the 70 year old group forgot equivalent amounts of information for all decades, showing a flat gradient, just like the Huntington's patients. This suggests that the characteristic temporal gradient found in Korsakoff patients, NA and HM, reflects something more than merely exaggerated normal forgetting. In the case of Korsakoff patients, the attendant problems of chronic alcoholism obviously contribute to their performance. However, in their case, there also appears to be a subsequent additional deficit superimposed upon this chronic acquisition problem. It is this deficit, which they perhaps share with NA, HM, and other amnesics, which may be an important functional deficit implicated in the cause of amnesia. A summary of the sometimes conflicting findings in this area is provided by Cohen and Squire (1981), who compared retrograde amnesia and remote memory impairment of NA, ECT patients and Korsakoff subjects. They found that the impairments in all three groups were temporally graded in at least some of their tests. They concluded that brief retrograde amnesia is present in all amnesia, and that its extent correlates with the extent of anterograde amnesia. Extensive remote memory impairment was argued to be associated with the types of cognitive deficits which impair a subjects ability to reconstruct old memories. Finally, they argue that the site of a lesion (either temporal or diencephalic) determines the nature of the anterograde amnesia suffered, while the extent of the lesion determines the extent of the remote memory impairment.

Evidence from Warrington and Weiskrantz (1978) which is Inconsistent with the Retrieval Deficit Hypothesis. Warrington and Weiskrantz' retrieval deficit hypothesis had been challenged by a number of workers (see, for example, Woods and Piercy, 1974). Warrington and Weiskrantz were themselves concerned about a number of aspects of the original study. In Warrington and Weiskrantz (1978)

they summarize these problems, and describe an attempted replication of the (1974) study. The paradigm remains the same, in that list one is shown to the subject and tested by an initial three letter cue. This cue is also used to cue the second list which the subject learns. There is then a score for total number of words recalled, and also a score for number of first list intrusions recalled in the place of second list items.

First, they were concerned because the greater deficit in amnesics' learning compared to controls did not occur in the earliest retention trials, but developed as the experiment wore on. They explain that the interference phenomena ought to be apparent from the first "reversal" trial; that is the first recall trial after list two has been learned and is in competition with list one. If this is not the case, they argue that what is observed may simply be reflecting "the rate at which incorrect responses are unlearned" (Warrington and Weiskrantz, 1978: 169).

Warrington and Weiskrantz also explain that in the (1978) study they wanted to discover whether both competing responses were equally accessible. Furthermore, in another two experiments they explored whether the cues they provided were in fact constraining choices in response competition. In the (1978) study they found that as with the (1974) experiment, the difference between amnesic and control memory arose much later than the first reversal trial, which weakens their retrieval deficit hypothesis, as interference ought to be strong at this point.

In a second "modified, modified free recall" task amnesics and controls were shown list one and tested for list one recall. Then they were shown list two and asked to generate both of the words they had seen as a response to the three letter cue. Again there was no difference in the memory scores of the two groups, and amnesics did not show more interference than controls.

In a third experiment, Warrington and Weiskrantz (1978) investigated the relative availability of competing responses. This was because they had argued that cues help reduce response competition at retrieval. In the new version of their experiment they argued that if the cue matched only one possible response, "... then "false positive" responses are impossible and therefore retention can be examined in a situation with minimal interference from prior learning or guessing" (Warrington and Weiskrantz, 1978: 170).

They therefore chose stimulus words which were uniquely specified by their first three letters, for example, "aisle", "juice" and "ankle". There were 200 words of this type in the stimulus pool. Subjects were shown the three-letter cues, and asked to generate words from them, success or failure being recorded. The first 20 words which could not be generated by the subject were chosen as the test stimuli for the experiment. Unfortunately, Warrington and Weiskrantz do not explain why they have chosen this method of chosing stimuli; however, it is assumed that the inability to generate the target from the initial letter cue is equated with the uniqueness of that target and this is why these particular words were selected for the targets. The stimulus materials were different for each subject as they depended on which 20 out of the 200 words in the pool a subjects had particular difficulty in generating. However, it is not clear what is the purpose of this manipulation.

The words were then written by the experimenter on a sheet of paper, and the subjects were asked first to copy them, and then to read them aloud. This was the learning phase of the experiment. Subjects were not told retention would be tested and unfortunately Warrington and Weiskrantz do not explain why they chose an incidental paradigm. Retrieval was tested by cues to the 20 "learned" words, and cues to 20 correctly-generated words form the previous phase of the experiment. A group of amnesic and control subjects was tested after 1 hour, and another group of amnesic and control subjects after 24 hours. They found that there was no difference between amnesic and control performance after one hour. This suggests that in conditions of no response competition amnesic memory is improved. Furthermore, the amnesics' scores were superior to the controls at 24 hours delay. However, the range of scores for retention in the control group was very wide, ranging from 4/20 to 15/20, so it is not obvious what this result really means.

After this series of experiments, Warrington and Weiskrantz found it difficult to support the retrieval deficit hypothesis. They then developed their ideas with a new set of experiments, formulating the "Cognitive Mediation", or "Disconnection" hypothesis (Warrington and Weiskrantz, 1982).

The work of Warrington, Weiskrantz and their co-workers reported in this section has been very influential. It has provoked research, raised theoretical controversies, and advanced thinking on the nature and cause

of amnesia. Although the retrieval deficit hypothesis is no longer favoured, it was instrumental in the development of current theories.

The Cognitive Mediation Hypothesis

The retrieval theory of amnesia was refined by Warrington and Weiskrantz (1982). Their claim is that amnesics are unimpaired at methods of retrieval which involve little cognitive mediation. By this they mean retrieval which is rule-based, automatic, and may be described in stimulus-response terms. This may be compared with Wickelgren (1979) which is described later in this chapter. However, Warrington and Weiskrantz (1982) claim that amnesics have an impairment of any retention requiring semantic organisation and elaboration. The term "cognitive mediation" applies to the employment by the subject of organising principles in retention. These may be semantic associations and elaborations; images; or links to personal frames of reference, that is, episodic memory. It is not established whether the impairment operates at encoding or retrieval. Warrington and Weiskrantz (1982) suggest that these cognitive mediation strategies are controlled by the frontal lobes. However, there are challenges to this view which claim that amnesics do benefit from imagery instructions (Jones, 1974; Leng and Parkin, 1988).

The Encoding Deficit Hypothesis

This account has been proposed by Cermak and his co-workers (see, for example, Butters and Cermak, 1980). They claim that amnesics are able to encode only superficial, surface characteristics of information. This approach is influenced by Craik and Lockhart (1972) and their concept of a hierarchy processing stages in memory. The highest level of processing is that of semantic processing, while the lowest level involves only processing of the superficial surface characteristics of the input. Craik and Lockhart (1972) point out that while elaborative processing produced more durable traces and was enhanced by study time, a second mechanism, known as maintenance rehearsal, could retain stimuli by the repetition of the same level of analysis. This keeps an item active in memory and available for recall but the memory is not enhanced by

increased study time. Cermak and Butters speculated that amnesics may habitually employ only maintenance rehearsal and be incapable of elaborative processing. Memory is seen as a by-product of the extent to which an input item is analysed and the greater the degree of analysis the more durable the memory. This reliance on shallow semantic encoding results in a greater susceptibility to proactive interference. This is because deep, semantic encoding is most likely to result in a uniquely specified trace, whereas shallowly processed items will be more similar to one another and more confusable. Thus Butters and Cermak (1980) have argued for a different theory on the basis of the same phenomena as Warrington and Weiskrantz (1974).

With this in mind Cermak and Butters (1972) proceeded to investigate proactive interference effects. In this experiment the degree of similarity of the stimulus material in the two to-be-learned lists was manipulated. In the low PI condition, consonant triads were used in trial one, and word triads in trial two. In the high PI condition, word triads were used in both trials. As PI increased, Korsakoff amnesics presented a greater performance decrement than control subjects. Cermak and Butters (1972) then went on to investigate cued recall performance. This study involved two lists of eight words. After free recall of the first list subjects were told that words from the second list would come from the same four categories as those of the first list. They discovered that giving amnesics category names to cue retrieval did not improve their performance.

This led Cermak and his group to claim that the original encoding of the material could not have been sufficiently precise for the category cueing to be an aid at retrieval. However, this provides only marginal support for the encoding deficit thesis, as Warrington and Weiskrantz (1968) would predict a significant number of intrusions from prior list learning which could also account for the poor performance reported.

More suggestive evidence is found in Cermak, Butters and Gerrein (1973). Subjects read a list of words, noting whenever a word was repeated. The lists contained words which were homophones like "bear" and "bare". There were also words which were semantically associated with others in the list, such as "doctor" and "hospital". Finally, there were some words which were synonymous with others, for example "sunlight" and "sunshine".

Korsakoff patients made significantly more errors in assuming that homophones, and strongly related words were repeated. Furthermore, they performed comparably with controls on synonym and neutral words. Butters and Cermak (1975) explain that the amnesics were encoding the material on superficial acoustic grounds, and because they encoded no deeper semantic element, they were unable to reject acoustically identical or associated words as not being examples of repeated material.

The possibility of amnesics processing material more shallowly than normals was further investigated using a release from PI methodology by Cermak, Butters and Moreines (1974). It was hypothesised that if this change were of a semantic nature amnesics would not show the benefits of release from PI in terms of improved retrieval performance. In the Cermak et al. (1974) study the Wickens (1970) paradigm was used. The to-be-learned material was given in blocks of five lists. In the first instance Korsakoff patients were given four lists of letters and their performance declined across the four lists. Then a shift to numbers was made for the fifth list and the amnesic subjects improved their retrieval scores. In contrast, when the first four lists contained animals, and the fifth list was a shift to vegetables, the amnesics did not improve their retrieval scores and did not show release from PI.

To perform the alphanumeric shift condition the amnesics needed merely to encode the material to a shallow physical level of processing in order to detect the shift in the input information and show release from PI. However, in the semantic shift condition, a release effect could only occur if the amnesics had encoded the input semantically, and their lack of semantic encoding both maximised the amount of accumulating PI and prevented the release from this interference. Failure to show release from PI is not a feature of all amnesics as post-encephalitics show normal release effects (Moskovitch, 1982).

Further support for the encoding deficit hypothesis was provided by Cermak and Reale (1978). Subjects were asked 60 questions. These included 20 orthographic questions of the form "Is this in upper case letters...?"; 20 phonemic questions of the form "Does this rhyme with..?"; and 20 sentence questions of the form "Does this fit into the sentence .....?". These question types thus require increasing degrees of processing from shallow to semantic processing. An unexpected recognition task followed, and the results showed that although the amnesics answered the

questions without error, their recognition was much poorer than that of the controls. This was the case for phonemically and semantically encoded items but the level of recognition for the orthographically encoded items was similar to that of controls. Also, the amnesics did not show better recognition for the semantically encoded items. However, there was a possibility that these results occurred because the Korsakoff subjects were overwhelmed by the recognition task, so a forced-recognition test presented in short blocks was administered in a further study. In addition new questions were asked to try to provoke semantic analysis in the amnesic group of the type "Is this a ...?", the response to which requires the category name, or "Does this .... have a ....?", the response to which requires a defining characteristic of the target. Even in this case the amnesics did not show improved recognition of the semantically encoded words, and their recognition performance remained equivalent to that of orthographically encoded words.

The underlying cause of the encoding deficit was suggested by Cermak, Reale and Baker (1978) to be a deficit in searching semantic memory itself. Collins and Loftus (1975) claimed that there are two types of semantic memory, known as lexical and conceptual semantic memory. The first is organised according to phonemic or orthographic similarity and the second is based upon semantic similarity. In accordance with these suggestions Cermak et al. (1978) prompted amnesic semantic memory using two types of cue which applied to the two types of semantic memory. One set of cues were "category-single letter" cues such as asking for a fruit which begins with the letter A. These cues were designed for searching through lexical semantic memory. The second set of cues were "category-adjective" cues such a asking for a fruit which is red. These cues were designed for searching conceptual semantic memory. Cermak et al. (1978) showed that Korsakoff subjects' search of lexical semantic memory was not significantly slower than that of normal subjects. However, the amnesic subjects were significantly slower than the normal subjects at searching conceptual semantic memory, requiring up to a second longer to search for the target item. It was argued that these results explained why amnesic memory is similar to that of normal subjects when the memory task requires little more than phonemic processing (see, for example, Cermak and Reale, 1978) but that the amnesics were impaired on tasks requiring semantic processing (see, for example, Cermak, Butters and Moreines, 1974).

The Cermak, Reale and Baker (1978) study also showed that if two category searches were performed sequentially there was no facilitation of the second search. In other words, the second search was no faster than the first as would occur in normal subjects (Loftus, 1973). Cermak et al. (1978) concluded that this showed a deficit in priming lexical semantic memory. This is in contrast to Jacoby and Witherspoon (1980) which demonstrated normal priming in amnesic subjects.

The experiments described above provided evidence of an encoding deficit in amnesics resulting in a deficiency of semantic encoding. Furthermore, the underlying cause of this deficit was claimed to be impairment of semantic memory itself. The next section describes evidence which does not support the encoding deficit hypothesis of amnesia.

Evidence Against The Encoding Deficit Hypothesis The encoding deficit theory has been countered by various experiments which investigate the orienting tasks used to demonstrate the depth of processing of which amnesics are capable.

Meudell and Mayes (1980) contrasted a learning condition of word lists, with a learning condition in which subjects were required to repeat each word five times. In the latter condition it was claimed that the repetition prohibits deeper encoding of the words. This experiment showed that the repeat condition (see also Cermak, Naus and Reale, 1976) impaired retention.

However, unlike the Cermak et al. (1976) study, both amnesics and controls were impaired *equally*. This ought not to occur if amnesics habitually encode more shallowly than controls. There was also a clustering effect in amnesic free recall, which suggests that the amnesics had encoded sufficiently deeply to categorise items at retrieval. The experiment also varied the composition of the distractors at recognition. They were either semantically, acoustically or graphemically related to the targets, and it was found that there was no difference in the amount of distraction promoted, in that amnesics made equal numbers of each of the three types of errors in recognition. In order for this to occur, processing must have advanced to a deep level, as otherwise there would only have been graphemic errors.

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The encoding deficit theory claims that orienting tasks which encourage the extraction of meaning or deeper levels of processing will

improve amnesic memory. The claim was investigated by Mayes, Meudell and Neary (1978), using a task which they claimed promoted the extraction of meaning. This was to ask the subjects riddles, the answers to which were the target words. They found that this manipulation impaired amnesic recall, and furthermore "semantic hints" at retrieval helped amnesics as much as controls. Similarly Mayes, Meudell and Neary (1980) and Meudell, Mayes and Neary (1980) also showed no difference in depth of processing in memory or organisation in memory in controls and amnesics.

Rozin (1976) noted that amnesics unimpaired comprehension abilities were not consistent with the encoding deficit hypothesis of amnesia. Moreover, Meudell, Mayes and Neary (1980) showed that amnesic memory for humorous cartoons was better than their memory for those which were not funny and this is difficult to explain if their semantic processing abilities were impaired. Finally, Squire (1982) pointed out that although Korsakoff patients do not benefit from instruction to encode items elaborately, patient NA and ECT patients do benefit from these instructions. He goes on to suggest that the encoding deficit may be a result of the subsidiary frontal lobe damage of Korsakoff subjects rather than a core functional deficit. For these reasons the encoding deficit hypothesis has been largely superseded. However, there are recent reports that the Encoding Deficit Hypothesis may account for the lack of a generation effect in Alzheimer patients. Dick, Kean and Sands (1989a, 1989b) have shown that Alzheimer patients do not show improved memory for self-generated items. In normal subjects, this improvement is due to richer semantic encoding of the item as a result of its generation. Dick et al (1989) suggest that the failure of Alzheimer patients to show this enhanced memory is probably due to an encoding deficit or an impairment of semantic memory.

The Context Memory Deficit Hypothesis

Proponents of the hypothesis argue that memory consists of item and contextual information. Item information roughly corresponds to identity of the target and various other attributes constitute its context. This contextual information includes physical characteristics, such as colour or alphabetical case; temporal aspects, such as recency or frequency of presentation; and spatial aspects such as location.

Contextual information is claimed to play a vital part in the processing of memories in normal subjects and the lack of this contextual information results in amnesia.

This hypothesis of the cause of amnesia was generated in response to the growing body of data which could not be accounted for by previous theories. For example, a whole range of perceptual, motor and cognitive skills are normal in amnesia (Parkin, 1982). Amnesics were shown to have normal priming abilities, suggesting the normal activation of traces at some level of description (Jacoby and Witherspoon, 1982), although explicit retrieval of these primed items is still impaired. Amnesics were shown to have qualitative differences in their memory compared with controls; for example, they confused recency and frequency judgements about memory (Huppert and Piercy, 1976). Finally, amnesics were shown to have deficits in encoding spatial aspects of context, in that when learning and testing took place in distinctive environments, amnesic memory improved more than control memory (Winocur and Kinsbourne, 1978). It was claimed that this showed that the experiment had facilitated amnesics' use of context in retrieval, thus boosting their performance. These data were incompatible with current theory, so the context memory deficit hypothesis was formulated to attempt to integrate these findings in a coherent framework. A summary of the main findings in support of the context memory deficit theory is given below.

Supporting Evidence for the Context Memory Deficit Hypothesis The first experiments cited as support for the context memory deficit hypothesis investigated some aspects of temporal memory. Temporal memory includes the following: memory for the serial order of item presentation; memory for the number of occurrences of items; memory for how recently an item has been encountered; and memory for the dates and times of the encoding for certain episodic information.

Huppert and Piercy (1976) showed that Korsakoff amnesics confuse recency and frequency information and that they make these judgements on the basis of general familiarity or trace strength. Extrapolating from this, Huppert and Piercy suggested that these factors influenced amnesic recognition generally and that amnesic retrieval operated on the basis of familiarity. These experiments are described in more detail below. On the basis of their results Huppert and Piercy (1982) postulated that amnesics suffer an impairment of the type of temporal

information which underlies the recency judgement and an impairment of occurrence information which underlies the frequency judgement.

Squire, Nadel and Slater (1981) investigated the temporal memory deficits in a slightly different experiment by looking at memory for temporal order. They showed subjects two lists of sentences separated by a three minute interval. The amnesic group, who were patient NA and a group of bilateral ECT patients, were tested almost immediately and the controls after a variable delay from 10 seconds up to 90 minutes. Subjects were asked to indicate whether a target sentence was familiar and then to state from which of the two possible lists it came, and also to indicate the order of the sentences within the lists. Squire et al. (1981) found that there was no significant difference between amnesic and normal control performance after a delay. They claim that in this respect amnesic memory is equivalent to that of controls with weaker memory owing to forgetting. They are therefore cautious about suggesting that temporal order information is selectively affected in amnesia.

Supporting evidence has nevertheless been provided more recently by Bowers, Verfaellie, Valenstein and Heilman (1988). Their patient suffered retrosplenial amnesia owing to damage which severed some projections connecting the hippocampus to the thalamus. He was impaired at memory for the temporal order of sentences and faces. Their task involved presentation of two lists with a delay of 30 minutes between them. The amnesic subject was required to recognise the items and to state from which list the target derived. This protocol is very similar to that of Squire, Nadel and Slater (1981) though with different results as the former study failed to find a temporal order deficit. The differing results may have occurred because the Bowers et al. (1988) study used a slightly different scoring method. They only counted temporal memory for correctly recognised items and, furthermore, their recognition score included subjects recognising that non-targets were distractors. Kopelman (1989) has also shown a temporal order deficit with Korsakoff and Alzheimer subjects. His task also involved temporal discrimination of two lists. He pointed out that the temporal order deficit is not associated with Korsakoff subjects' subsidiary frontal lobe damage, although temporal order deficits are reported in frontally lesioned patients by Milner, Petrides and Smith (1985). Because he found the effect with Alzheimer patients, this suggests that the deficit is not just specific to diencephalic amnesics such as Korsakoff patients.

Although amnesics do not demonstrate explicit memory for frequency of occurrence information, they show some *implicit* influence of this factor when tested for this by indirect methods (Johnson, Kim and Risse, 1985). In this study Korean melodies were played to amnesics. Although the amnesics could not distinguish reliably the old, previously heard melodies from new, only recently heard memories, they did come to prefer those old melodies which they had heard often.

Amnesics have been shown to be deficient at detecting the source from which an item of information was relayed (Schacter, Harbluck and McLachlan, 1984). Source memory is defined as the ability to judge from whence an item of information was presented. This usually amounts to a decision of whether information was encoded in an experiment, and if so, some further finer distinction of source within the experiment; or alternatively, the judgement that the item was encountered elsewhere. It can be seen that this type of task is very similar to the tasks of judging from which list an item was derived which are used in temporal order experiments. This type of paradigm does not disclose whether source memory is a different type of memory to temporal memory, since source and serial order are confounded in the design. Thus both of these tasks could be testing different aspects of temporal memory. Experimental evidence is needed which demonstrates source memory deficits and shows that this type of memory involves more than just temporal information, as some have claimed that not all amnesics have a deficit of temporal information (Squire, Nadel and Slater, 1981).

Schacter, Harbluck and McLachlan (1984), used two experimenters who read items of fictional information to amnesic and control subjects such as "Bob Hope's father was a fireman" or some general knowledge fact. The subjects recalled the facts and were required to state the experimenter who had told them the information, or alternatively, to state that the information had been known to them before the experiment. The amnesics reached adequate levels of fact recall, but their performance was at chance for detecting which experimenter was the source of the information. However, they were accurate at detecting information which had an extra-experimental source. It was argued that these data demonstrated a deficiency in amnesic's processing of the context in which they learnt the information, as they could not distinguish the two information sources. However, this does not explain

why the amnesics were accurate at distinguishing extra-experimentally learned information.

Shimamura and Squire (1987) investigated source memory for true facts with Korsakoff amnesics, patient NA, and amnesics with anoxic or ischaemic aetiology. They showed a clear source memory deficit and also that this was unrelated to fact memory impairment. Even those amnesic subjects who did learn some new facts during the experiment were unable to state the source of their new knowledge. Furthermore, there was no significant difference in fact memory between amnesics who had total source memory loss and those who were less impaired for source memory. Shimamura and Squire explained their results in terms of an impaired ability to establish declarative memory. They further suggested that frontal lobe damage may be responsible for the temporal order deficit, since their source memory data correlate with patients' scores on tests of frontal lobe pathology.

Spatial memory has also been shown to be impaired in amnesia. In experiments this usually refers to memory for a display of items in particular locations which are then to be retrieved. Outside the laboratory, spatial memory also includes memory for the current location of personal belongings, such as spectacles, and includes the judgement discriminating previous, no longer applicable, locations from the target location. In this sense temporal information may again be playing a part.

Warrington and Baddeley (1974) demonstrated the deficit in an experiment testing memory for a display of five randomly positioned dots. Amnesics were found to have poorer spatial memory than control subjects. Smith and Milner (1981) have also demonstrated spatial memory deficits with temporal lobectomy patients and Hirst and Volpe (1984) have done likewise with a group of mixed amnesics including stroke, tumour, hypoxic, trauma, and aneurism patients. These experiments are described further in Chapter Four below.

Finally, Winocur and Kinsbourne (1978) provided evidence that amnesics do not process environmental context efficiently. Environmental context includes such things as the contents of a room, the colour of its walls, smells, sounds and perhaps even internal body state and mood. In experiments only simple types of context are investigated, and the subjects' successful retrieval is contingent upon correctly distinguishing competing responses on the basis of their differing contexts. Winocur and Kinsbourne (1978) used an AB-AC

paradigm in which a word was paired with two different partners resulting in two to-be-learned lists. The subject is presented the first list in which word A is paired with word B. After recall of this list, the subject is then presented with the second list in which word A is paired with word C. Thus, in the second trial, the subject must discriminate between the two lists and avoid interference, giving the appropriate partner, C, rather than the prior list item, B. They showed that learning and subsequently testing in a distinctive external context such as a bright red light and the sound of classical music significantly reduced amnesic memory errors and allowed the subjects to distinguish the two lists successfully. The fact that provision of contextual cues at testing improved retrieval is argued to be evidence that under normal circumstances amnesics are unable to encode this information automatically. There was no such radical difference in performance for control subjects who were given distinctive environments in which to learn and be tested. Therefore it was concluded that normal subjects were already utilising contextual cues effectively.

The Nature of Contextual Information in Memory: Extrinsic and Intrinsic Context The context memory deficit hypothesis proposes that amnesia results from an inability to process contextual information. Contextual information may include spatial cues, temporal cues and environmental cues. It has not been established whether this hypothesised deficit occurs at both encoding and retrieval, though most experiments so far have concentrated on encoding tasks (see, for example, Hirst and Volpe, 1984). Workers in the area have defined two types of contextual information, extrinsic and intrinsic context.

Extrinsic Context Baddeley (1982) has characterised extrinsic context as being information which does not alter the interpretation of the target, that is, supplementary details which do not uniquely define the identity of the target.

I will argue, however, that these details serve a purpose in mediating the organisation of episodic memory. In other words extrinsic context does define the identity of the target because it facilitates discrimination between similar targets stored in memory. For example, such extrinsic contextual information will specify where a car is parked today in a car park, and prevent interference from previous parking spaces.

Intrinsic context Baddeley (1982) defined as comprising those details which make a target unique, and which affect its interpretation. I claim that it is useful to specify this still further and argue that intrinsic context is information which specifies an item's meaning and identity.

In the literature, the distinction between intrinsic and extrinsic context is not always made explicit by many workers. Kinsbourne and Wood (1975), however, do define these concepts. Their example of intrinsic context is that of an amnesic not recognising an individual doctor from the scores in attendance because he fails to notice enough about each each person to distinguish him from another.

According to the characterisation proposed above, this is an example of extrinsic context, which underlies episodic memory. This definition is preferred because it allows a useful distinction to be made between the two types of context which may have empirical consequences. If intrinsic context helps to specify the target, in terms of meaning and identity, whereas extrinsic context allows distinctions to be made between similar items in memory in episodic terms, then it may be possible to to show differential impairments in each system. For example, it may be possible to show that amnesics with impaired intrinsic contextual processing, and thus impaired use of semantic memory, may experience difficulty in resolving verbal ambiguities. This would be because the item is not sufficiently constrained in the subject's memory to allow good performance at tasks such as garden path sentences. A suggestion that this may be so is found in Cermak, Butters and Gerrein, 1973, where amnesic subjects assumed that when they read "bare" and "bear" this was an instance of repetition. It could be argued that this shows that the input stimuli did not result in a memory representation for each word which was sufficiently constrained to distinguish between the two.

Warrington and Weiskrantz (1982) would also appear to concur with this characterisation of intrinsic context, by claiming that amnesics have difficulty with intrinsic contextual information because it is elaborately encoded. Warrington and Weiskrantz claim that the unique specification of an item involves the spontaneous organisation of forming associations and categorising memory. Once stored as a collection or encyclopaedia, this system resembles semantic memory. This would also be in accord with the above suggestions that intrinsic context mediates the formulation of semantic memory.

Thus the preferred terminology of the present author is that amnesics may suffer a deficit of extrinsic context underlying episodic memory, or intrinsic context underlying semantic memory, or perhaps damage to both systems. The experiments reported in this thesis pertain only to extrinsic contextual information.

The Role of Contextual Information in Memory; Implications for Processing A requirement of the context memory deficit hypothesis is that contextual information is important to both normal and amnesic memory in retrieving target memories. Unfortunately, the mechanism involved has not been explained. However, limited attempts have been made to specify what operations must be required.

It is argued that it is necessary that contextual information may be dissociated from target memory in terms of either means of encoding or storage. If this were not the case, it would be difficult to account for how one piece of information may still be available in the absence of the other. It is clear that if both context and target were similarly stored then the availability of the target information suggests the integrity of the storage system, and given this, it is difficult to explain why contextual information is not available. For this reason theorists usually argue that the different types of information are handled differently in the cognitive system. The general claim is that the brain damage incurred in amnesia disrupts the processing system which handles contextual material, resulting in memory impairment. So far the discussion in the literature has focussed on a distinction between automatic and effortful encoding (Hasher and Zacks, 1979).

This distinction is primarily descriptive, as the processes involved have not yet been established. Hasher and Zacks (1979) describe automatic processes as not requiring cognitive "effort", not competing for limited processing resources, and as occurring without intention. This is contrasted with effortful processing, which requires capacity, is intentionally initiated, and shows benefits from practice. It is claimed by Hirst and Volpe (1984a, 1984b) that amnesics have a deficit in automatic processing, or more specifically, encoding, and that contextual information is usually automatically encoded.

Some workers believe that if amnesics are encouraged to use their unimpaired effortful processing to encode contextual information, then

there will be an improvement in their contextual memory, (see, for example, Hirst and Volpe, 1984). It should be pointed out here that this argument could only apply to extrinsic context, as intrinsic contextual memory, being processed effortfully in the first place (Warrington and Weiskrantz, 1982), would not benefit from this instruction. Furthermore, Hasher and Zacks (1979) would not predict that intentional instructions would improve contextual memory. In their formulation it is a defining feature of automatic processes that they are not influenced by conscious effort. Hirst and Volpe (1984) suggest that amnesics may opt either to encode target information, or context information, using their unimpaired effortful processing abilities. This will result in a trade-off, where improvement in contextual memory will be associated with poorer target memory. This trade-off was demonstrated by Hirst and Volpe (1984).

I suggest that this characterisation of automatic and effortful processing with respect to amnesic memory is not as helpful as it may initially appear. This is because it is difficult to reconcile the proposal that amnesics are impaired at automatic processing with their virtually normal priming abilities. For example, there is one type of priming which is very fast and automatic which may operate through spreading activation (Neely, 1979). Amnesics are known to perform normally at tasks involving this (Cermak, Butters and Gerrein, 1973). Furthermore, another type of priming, known as attentional priming, may also be normal in amnesia, as amnesics have been shown to demonstrate normal repetition priming effects (Scarborough, Cortese and Scarborough, 1977). These normal priming abilities are presumably using automatic processes, yet the context memory deficit hypothesis argues that automatic processing is impaired in amnesia. Priming is discussed in more detail in chapters 5 and 6 of this thesis.

Does the Context Deficit Result in Selective Impairment of Recall? An important observation in the development of the context deficit hypothesis was that amnesics with poor recall performance could under certain circumstances show almost normal recognition (Brooks and Baddeley, 1976; Cohen and Squire, 1980; Hirst and Volpe, 1982, Hirst, Johnson, Kim, Phelps, Risse and Volpe, 1986; Huppert and Piercy, 1976; Jacoby and Witherspoon, 1982). This would perhaps be expected if context were more important in aiding recall than recognition. In the foregoing, first, the theory of what underlies amnesic recognition will be explored,

and then the implications of these claims for amnesic recall will be described.

The differing performance in amnesic recall and recognition memory provoked Huppert and Piercy (1976) in particular to explore the phenomenon more fully. They concluded that their results reflected Norman and Rumelhart's (1970) model of memory. In this model a crucial distinction is made between *item* and *contextual* information. Huppert and Piercy (1976: 18) argued that:

> "the attributes of an item have attached to them information about the context in which an item occurred. In recall, the contextual information is provided and the subject reconstructs the item from the attributes with the appropriate contextual marker. In recognition, the items are provided and the subject examines the contextual markers to determine whether the item occurred in the appropriate context ".

They go on to hypothesise that since recognition and recall both require contextual information, they would expect equal impairment of both types of retrieval. However, in the event, what they demonstrated was a selective impairment of recall. They point out that although recognition of familiar material is severely impaired, recognition of unfamiliar material is relatively good. This is because the amnesics made positive responses to any familiar item, irrespective of whether it had been part of the experiment. Recognition of familiar information is claimed to require the amnesic to identify its context, that is, its *relative familiarity*. Whereas, they claim:

> "when information is unfamiliar to the subject prior to the experiment, recognition does not require the subject to identify the context he can make a recognition response on the basis of item memory alone. If the item being presented corresponds to an item in memory he makes a positive recognition response, if the item being presented does not correspond to an item in his memory he makes a negative recognition response. According to our hypothesis, item memory is relatively intact, so the hypothesis can explain the good recognition performance of amnesics when tested with unfamiliar material" (Huppert and Piercy, 1976: 19).

In the context of this experiment, "unfamiliar" refers to pictures which were unfamiliar before the experiment. Huppert and Piercy (1976) argue that correct recognition in this case merely requires the subject to ascertain whether they have ever seen the picture before, a "primary familiarity judgement". They claim that if the subject is aware that the picture has been seen, this familiarity judgement alone will result in a correct response. If the subject believes it has not been seen, it may be confidently rejected.

They suggest that this decision by the subject that the item is an unfamiliar item, and only recently presented, is achieved *without using contextual information*. This aspect of their hypothesis is hard to test empirically, but Huppert and Piercy (1976) support their claim with the evidence that Korsakoff patients make many false positive responses to high frequency fillers and stimuli. Alternatively, Jacoby (1984) describes recognition as being a function of the speed of processing an item receives. In his formulation activation of recently perceived items is facilitated and the item is judged to be familiar because its speed of processing is faster than it would be were it unfamiliar. If it is supposed that the facilitation fades with time, then perhaps in Huppert and Piercy (1976) the familiar items which were over-learned do not have such speeded processing as those items which are unfamiliar before the experiment. This may provide an alternative explanation of why unfamiliar items were better recognised than familiar items.

According to Huppert and Piercy (1976), in contrast to unfamiliar items, information which was familiar before the test requires both a primary familiarity judgement and a list membership judgement. In other words, the subject knows a recognition item corresponds to a representation in memory, but must next decide if the picture was part of the experiment. This is a contextual judgement, involving *recency information*, and *source information* respectively, and its operation is therefore impaired.

This type of claim is much more straightforward to test, and much of the work on the context memory deficit hypothesis has focussed on contextual judgements of this sort (see, for example, Huppert and Piercy, 1978; Schacter, Harbluck and McLachlan, 1984). Thus one of the first claims of the context deficit hypothesis was that amnesics are poor at

recall and at types of recognition which require the use of contextual information.

Furthermore, amnesics are claimed to base their recognition judgements on *familiarity*. Parkin, Leng and Montaldi (1989; in press) have defined familiarity as " . . . information stored in memory that allows an individual to be aware that a stimulus has been encountered before without the ability to recall explicitly the circumstances of the encounter."

They further state that in tests of recognition it must be clear exactly what is being tested as there are at least three factors influencing the extent to which recognition is based on familiarity. The first is that familiarity of an item may be "a positive function of its initial exposure duration". The second is that strength of familiarity will decline with time, and the third is that familiarity judgements may depend on there being an established representation of the item already extant. This is activated by the stimulus, and the recognition judgement consists in the decision that this reinstatement means the stimulus is familiar. Thus Parkin et al point out that the nature of the to-be-remembered stimulus will also affect recognition performance according to exposure, latency since last activation, and existence of pre-established representation.

Huppert and Piercy (1978) further developed their theory by explaining the role of trace strength in amnesic memory. In this experiment 80 pictures were shown 24 hours before testing and 80 pictures were shown ten minutes before testing. Half the pictures were presented once and half were presented three times. For recency judgements subjects were required to state whether an item was seen yesterday or today, and the frequency judgement was to state how often the item had been seen. In this experiment Korsakoff subjects based their recency responses as much on number of presentations as on how recently the item had been encountered. Conversely, their frequency judgements were as much based on how recently an item had been seen as they were on how often an item was seen. Huppert and Piercy explain that amnesics suffer damage to two types of contextual information. These are occurrence information which underlies the frequency judgement, and temporal information which underlies the recency judgements. Amnesic recognition decisions are claimed to be based on *trace strength* which is defined as the combined effect of both recency and frequency of item information.

In this way Huppert and Piercy formulated the mechanisms underlying amnesic recognition. However, recall requires more to sustain it than may be provided by estimates of trace strength. Rather, a strong link in memory is required between the target item and its spatio-temporal context. For example, there must be a strong link between a word and the experiment in which it was encountered. Thus Huppert and Piercy (1976; 1978) demonstrated a selective deficit of recall and provided an account of why recall was impaired relative to recognition, in as much as recognition could be sustained by estimates of trace strength, whereas recall required contextual information which was impaired in amnesia.

Hirst, Johnson, Kim, Phelps, Risse and Volpe (1986) also showed a selective deficit in recall, though they did not interpret their results as support for the context deficit hypothesis *per se*. They do, however, say that recognition is less likely to rely on internally generated contextual cues than is free recall.

Further evidence that context affects recall more than recognition resulting in a selective deficit in recall is provided by Craik (1986). He found age-related differences in recall and recognition. Cued recall and recognition do not require self-initiated operations, and are initiated by environmental stimuli. Free recall is a self-initiated operation almost without external environmental supporting cues. Thus Craik demonstrated that cued recall and recognition were not so impaired by aging as free recall. He also found a deficit in older subjects' source memory for facts, and this complements the findings of Schacter, Harbluck and McLachlan (1984) with amnesics. A selective deficit in recall has also been reported by Warrington (1984); Hirst (1985); Rocchetta (1986); and Jetter, Poser, Freeman and Markowitsch (1986) with frontally lesioned patients. Similarly, Brown, Lewis, Brown, Horn and Bowes (1982) showed that recognition and recall are differentially affected by temporary, drug-induced, amnesia.

The demonstrations of a selective recall deficit in amnesia represent good evidence for the context deficit hypothesis. It is for these reasons that the author began an investigation of contextual memory in amnesics with a preliminary series of experiments to discover whether or not there was a selective recall impairment in amnesia. These experiments are reported in chapter three below. The next section of this chapter discusses some evidence from animal research which is of relevance to

investigations of human amnesia, with particular relevance for the context memory deficit hypothesis.

Further Evidence of the Role of the Hippocampus and Allied Areas in Amnesia

As this thesis comprises empirical tests of the context memory deficit hypothesis, discussion of this animal research is included to provide further evidence and a wider perspective on the theoretical claims. In summary, this evidence from animal studies concurs with the claim that contextual information processing impairments are implicated in amnesia. These studies concentrate on investigating the role of the hippocampus in memory.

The Hippocampus and Spatial Memory The experiments most relevant to the context deficit hypothesis are those which investigate the rat's spatial memory abilities. The claim is that after hippocampal damage, a rat has a specific problem with spatial information, and that this difficulty is the critical functional deficit causing amnesia.

Morris, Garrud, Rawlins and O'Keefe (1982) provided evidence of the hippocampus' role in spatial memory. They made rats swim through milky water to a concealed platform in order to test their memory for the route, thus showing their spatial abilities. The experiment was carried out on normal rats, hippocampally-lesioned rats, and finally a sample with neocortical lesions. This last group was included in order to control for the the damage incurred to the cortex in making the hippocampal lesions. They found that although the neocortical lesions slightly disrupted performance, the hippocampal lesions made the rats virtually incapable of the task. These findings are also supported by O'Keefe and Dostrovsky (1971). They recorded the activity of individual neurons in the hippocampus and discovered that certain neurons responded differentially to certain spatial locations. These were termed that neurons' "receptive fields". These findings lead O'Keefe and Nadel (1978) to describe the hippocampus as a "cognitive map".

As this work is so influential, a discussion now follows in more detail the implications of O'Keefe and Nadel's work.

The hippocampus as a "cognitive map" O'Keefe and Nadel (1978) propose that the hippocampus contains an encoded representation of temporal and spatial information in the environment. This representation is distributed throughout the hippocampal tissue, such that groups of neurons will respond only to certain spatial or temporal stimuli. In this sense, there is claimed to be a one-to-one mapping of spatial location to a collection of hippocampal neurons.

More specifically, a distinction is made between a "taxon" and a "locale" memory system in the hippocampus. The taxon system is analogous to semantic memory, in that its elements are claimed to be organised according to feature similarity among memory items. It is claimed that time of encoding is not a factor in the memory representation of this system. Activation of any given memory representation depends on the degree of similarity between the properties of the stimulus, and the properties encoded in the memory representation. Input information activates all those neuronal elements in the hippocampus, which share the properties of the stimulus. There is a trend in this activation such that activation of these elements is more likely with subsequent presentations of the stimulus, although the general tendency of the taxon store is towards decay of the memory instantiation. In this respect, it is of course very different to semantic memory.

The locale memory system is claimed to be analogous to episodic memory. This is because time of encoding is an important organising principle in the encoding of these memory representations. Unlike the taxon system, where repeated presentations were functional in strengthening the encoded trace, the locale system is characterised by single occurrence storage, such that time of occurrence may be used as a basis for retrieval.

This characterisation of the nature of hippocampal structure and function would make a number of predictions relevant to human amnesia research. Stern (1981) provides a summary of possible predictions. For example, damage to the hippocampus should impair the locale system, and as the deep structure of language would be represented here, then one would expect comprehension of hippocampally lesioned humans to be impaired. This prediction is not confirmed by amnesic data, as amnesics have unimpaired comprehension abilities. In any case, it is counter-intuitive that the impaired comprehension predicted is attributed to the

locale, rather than the taxon system. One may have assumed that the system described as subserving semantic memory would play a more important part in comprehension.

Another prediction described by Stern (1981) is that damage to the locale system would result in equal degrees of retrograde amnesia for both remote and more recent memories. This is because the "temporal tagging" of all memories, for which the locale system was responsible, is no longer available.

However, it is argued here that in principle this need not be the case. Stern's argument assumes that information is stored in the hippocampus in a "context-free" state, with all of its characteristic features stripped away, only to be instantiated into the complete form at retrieval. It is as if a memory item is stored in a dormant state, detached from its spatial context, and thus detached from its potential retrieval cues.

It may equally be possible however, that the memories are stored with this information intact. In this case, old memories encoded pre-traumatically will still retain their spatial context, whereas more recent memories will not have this facility. This would result in earlier memories being more easily retrieved than more recent memories, causing a "temporal gradient" in retrieval performance (see, for example, Marslen-Wilson and Teuber, 1975).

Double dissociation of function between the taxon and locale systems would be especially interesting to workers in human amnesia research. This is because the characterisation of information in the taxon and locale systems could reflect the dichotomy described by Huppert and Piercy (1976) between "target" and "context" information. They have in fact shown a dissociation of amnesic memory for target information and memory for temporal and spatial context. This has formed the basis of the context deficit hypothesis of amnesia, and it is discussed in detail earlier in this chapter.

The hippocampus and "erasable memory" One group of workers in this area have disagreed with the claim that the hippocampus is involved primarily with spatial memory. Olton and Feustle (1981) claim that the hippocampally-lesioned rat's amnesia is as a result of other types of memory impairment, rather than simply spatial memory deficits. They cite an experiment which showed that even when spatial memory

considerations were irrelevant, rats were still amnesic after fimbria/fornix lesions in a radial maze task. Thus hippocampal damage must have affected more than just spatial memory. Olton (1983) accounts for this by suggesting that hippocampal damage impairs "working memory" but leaves "reference memory" intact. Working memory refers to "erasable" memory which is useful in the short term, but is soon replaced by other material. In Carlson (1986) this type of working memory has been identified with declarative knowledge. Reference memory is more enduring, and is produced by constant conditions, and Carlson compares this to procedural knowledge.

These terms are ascribed by Carlson (1986) to Squire (1982), and Carlson further explains that declarative knowledge refers to memory for where a car is parked today, whereas procedural knowledge refers to where the car park itself is situated. In fact, the terms procedural and declarative knowledge where brought into current use by Anderson (1976) as aspects of ACT, a network model of cognition. Anderson distinguishes the former as denoting that which we know how to do, and the latter as referring to facts, concepts and beliefs.

In terms of human amnesia research, this dichotomy is mirrored in worker's claims that amnesics have impaired episodic memory, as Kohl (1984) has suggested, and relatively unimpaired semantic memory, as claimed by Kinsbourne and Wood (1975). (However, this account contrasts with Warrington (1975) which showed a specific breakdown of semantic memory, and Cermak, Reale and Baker (1978), which showed that amnesics' search of conceptual semantic memory was impaired).

The hippocampus and "unlearning" Berger and Orr (1983) have suggested that the hippocampus plays a part in "unlearning" responses. They trained normal and hippocampally-lesioned rabbits in a reversal learning, classical conditioning procedure on nictatating membrane responses. This membrane covers the eyeball in response to a puff of air. The rabbit is trained to a conditioned stimulus of a tone signalling the puff of air. The animal's nictatating response is then initiated whenever it hears the tone. Then the meaning of the tone is "reversed", and the tone no longer predicts the puff of air. Therefore the animal must alter its responses accordingly. Berger and Orr found the animals with hippocampal lesions learnt very slowly in the reversal

trials. This suggested that the hippocampus had a role in "unlearning" one response to a stimulus, and learning to make a new response instead.

This finding is of interest if considered in terms of current thinking about human amnesics' episodic memory deficits. It is suggested here, that the efficient working of episodic memory requires the "unlearning" of specific past events, that is, "editing" episodic memory in order to eliminate traces which are superseded by more up-to-date information (cf. Schank, 1982). For example, to use the car-parking example again, there must be some means by which old, no longer relevant locations are not retrieved in favour of today's parking space. Perhaps this processing task is carried out by the hippocampus. If an amnesic is unable to do this, then interference from no longer relevant information will build up, which will give the appearance of deficient episodic memory

The hippocampus and "vertical associations" Wickelgren (1979) makes a distinction in his theory of cognition between "horizontal" and "vertical" associations. A horizontal association is claimed to be the more primitive process, and it involves connections between already formed clusters of concepts in the store, with no subsequent increase in the number of representations in memory, or change in neuronal organisation. In contrast, a vertical association is formed between two or more clusters of neurons, each representing a concept or concepts, with the creation of a superordinate representation instantiated in another neuron. This assigns a label to a cluster of concepts. Stern (1981) suggests that this process also instantiates context into the representation, and as such Wickelgren's thesis could be seen as being complementary to the context memory deficit hypothesis discussed above (see, for example Huppert and Piercy, 1976) in that it stresses the importance of links between a target memory and its context.

Wickelgren's characterisation equates the formation of vertical associations, and thus the incorporation of contextual information, with a physical alteration in the nature and organisation of neurons in the hippocampus. Damage to the hippocampus impairs the ability to form new vertical associations, because of the decreased availability of free neurons. However, it does not impair the ability to strengthen previously formed associations.

Thus, human amnesics have unimpaired use of previously established semantic associations, such as "doctor-nurse", but have

difficulty in learning new paired associates (see, for example, Winocur and Weiskrantz, 1976). The inability to encode context into the memory representation also results in increased susceptibility to interference (see, for example, Warrington and Weiskrantz, 1974). Without the disambiguating context, traces are more confusable, and therefore inappropriate items are retrieved from memory, with many prior item intrusions. Wickelgren explains the ability to demonstrate classical conditioning with reference to amnesics' use of their unimpaired horizontal association system.

This chapter has provided a comprehensive review of the literature in human and animal amnesia to provide the background and justification for the author's experiments which follow in the next three chapters.

CHAPTER THREE

AN INVESTIGATION OF THE HYPOTHESISED SELECTIVE RECALL DEFICIT IN AMNESIA

The context memory deficit hypothesis of amnesia predicts that when control and amnesic recognition memory are approximately equated, the amnesic group will show a deficit in recall relative to that of control subjects. Equalised recognition memory in both groups is achieved by manipulating the study conditions of the normal subjects to depress their normally relatively high memory scores. This technique is known as "matching", and it may be applied to either recall or recognition memory measures. Once recognition scores are rendered equivalent in both groups, the hypothesised lower amnesic recall score is described as a *selective* deficit in recall, since it is argued that amnesia affected recall but has not affected recognition levels. This series of experiments investigates the hypothesised recall deficit in amnesia by studying memory for lists of common words.

Demonstrations of selective deficits in retrieval were first provided to counter suggestions that amnesic memory differed only quantatively from normal memory. The claim was that when normal memory was depressed to amnesic levels then "amnesic" patterns of performance would result (see, for example, Mayes and Meudell, 1981a, b; Mayes, Meudell and Som, 1981; Squire, Nadel and Slater, 1981; Squire, Wetzel and Slater, 1978; Woods and Piercy, 1974).

Proponents of the suggestion that amnesic memory differed *qualitatively* from normal memory drew attention to demonstrations of very good recognition in amnesia, which under certain circumstances even equaled that of controls (see, for example, Hirst and Volpe, 1982; Huppert and Piercy, 1976). This good recognition was allied to the typical poor free recall of amnesics. Evidence of a selective recall deficit is seen as important supporting evidence for the context memory deficit hypothesis of amnesia as contextual information is claimed to be more important in recall than in recognition. In most published experiments recognition is matched in both groups and free recall is compared. Some workers match control and amnesic recognition by manipulating delay to testing (Mayes and Meudell 1981a, 1981b), whereas others manipulate study time (Huppert and Piercy, 1976). Thus amnesics may be trained to

equivalent levels of recognition to those of controls, but their hypothesised contextual processing deficit will be claimed to handicap their recall scores.

Let us consider the effects of matching recognition scores in more detail. In analysis of variance, interpretation is greatly facilitated if either recall or recognition have the same value in both groups. Consider the question of whether recall and recognition are differentially affected in amnesia versus normal memory. ANOVA will only reveal whether there is a differential drop in the score from recognition to recall in the two groups. In other words, it will show whether the absolute level of performance loss from recognition to recall differs between groups. The problem is that such a difference may be hard to interpret. For example, if controls had higher recognition scores than amnesics, then the fact that there is a greater drop in performance for recall in the amnesic group may be due to a scaling problem. For example, suppose recognition performance were 48/50 in the control group and that recall performance were half this, 24/50. There is thus a drop of 24 between control recognition and recall. However, if the amnesics had a recognition score of 20/50 then a similar 50% decrement in the amnesic recall score relative to their recognition score represents an absolute drop of only 10, which is much smaller than that of the controls. In this example, it is simply not possible for the amnesic recall to be 24 less than their recognition. Interpretation is made difficult by a so-called "floor effect".

To recapitulate, if performance were to drop by 50% in this way for recall compared to recognition in both controls and amnesics, ANOVA would lead to the conclusion that there was a difference between the two groups because there was a difference in absolute decrement in score, in spite of the fact that both groups suffered the same proportional decrease in scores. This is an example of the fact that ANOVA is not a neutral theoretical model but rather, assumes that effects are made up of additive components. Thus if the effects are multiplicative (for example, the halving of scores described earlier) then ANOVA may give contentious results. This problem is avoided if performance is matched for one measure of memory and compared on the other measure of memory. In this case, deviations from constant multiplication or any other similar model will show up as deviations in absolute differences also.

There are a number of different methods of assessing retention (see Brown, 1976). In this series of experiments only analyses using the simplest of these, recognition hit rate, will be reported.

It may be noted that it is difficult to obtain controls who are matched to amnesics for age, education and score on WAIS and so matching recognition performance goes some way towards reducing the effect of these extra-experimental variables.

The first two experiments in this series attempted to show a recall deficit in amnesia matching control and amnesic recognition by manipulating the delay before testing. The final experiment of the series compares delay to testing and another method of matching, the manipulation of study times, for their effectiveness at matching control and amnesic memory.

EXPERIMENT 1

Method

Subjects These were seven Korsakoff patients with a mean age of 58 years, ranging from 47 to 66 years, who were resident in long-term psychiatric hospitals in Newcastle-upon-Tyne. Relevant neuropsychological data on these subjects are provided in Appendix A and B. The amnesics in this experiment were ST, JB, HS, JA, HK, RS and SM. The seven control subjects had an average age of 35 years, ranging from 26 to 49 years.

Materials The materials were 80 common unrelated nouns randomly selected from a pool used by the Manchester Amnesia Research Group. The words are listed in Appendix C with their mean frequencies of occurrence. The words were randomly divided into two lists, A and B, of 20 to-be-remembered words each having a set of 20 distractor words for use in the recognition tests. Each target word was separately printed by hand in black ink on a 3x5 inch index card. Recognition was tested by two-choice recognition, with each target word paired side by side with a distractor word on a 3x5 index card. The order of target and distractor on the card was balanced so that the target appeared equally often on the right and the left of the card. The target lists A and B were used equally often as the first list and the second list in the experiment. The first list

was always tested by recall and the second list was always tested by recognition, in order to ensure that all subjects would encode as fully as possible during both conditions.

Design The experiment was a 2 x 2 factorial design. There was one between-subjects factor of group (amnesic and control subjects), and one within-subjects factor of memory test (recall and recognition).

Procedure The aim of this procedure was to match amnesic and control recognition by reducing control recognition using 24 hour delays before testing. Therefore the procedure for amnesics and control subjects differs slightly and is reported separately.

Amnesic subjects were told that they were about to see some words and that they should try to remember them. They were shown the target words from the first list for six seconds per word in random order. The cards were randomised by shuffling. The subjects read each word aloud to the experimenter to ensure that they were focussing their attention on the experiment. They were then tested for their recall of those words virtually immediately afterwards. The subjects were given two minutes for free recall. The following day the amnesic subjects were shown the words from the second list for six seconds per word, again reading each word aloud to the experimenter. They were then tested for their recognition immediately. The amnesic subject looked at each recognition card, again in random order, and indicated which of the two words had been in the target list.

Control subjects were told that they would see some words which they should try to remember. They were shown the first list words for six seconds per word, presented in random order, and read each word aloud as the amnesics had done. There then followed a delay of 24 hours after which the subjects were given two minutes to recall the targets. After a filled interval of about 15 minutes of conversation the control subjects were shown the second list for six seconds per word. After a delay of 24 hours the control subjects were tested for their recognition by looking at the recognition cards in random order and indicating which of the two words was the target.

Results

The raw data are presented in Appendix D. The table of means is reported below. A t-test on the recognition scores of the two groups confirmed that there was no significant difference between the recognition scores of the two groups ($t(12) = 1.56$, $p > 0.10$). When analysed by ANOVA no main effect of group was found, ($F(1,12) = 0.15$). A main effect of memory task was found ($F(1,12) = 100.77$, $p < 0.001$). This was due to recall being a far more difficult task than recognition. The group by memory task interaction did not reach significance ($F(1,12) = 2.10$). Therefore there is no significant evidence of an amnesic recall deficit relative to controls.

Experiment 1

Table of Group and Memory task Means

Maximum score = 20

Amnesic Recall	4.29
Amnesic Recognition	14.86
Control Recall	2.86
Control Recognition	17.00

Discussion

This experiment failed to demonstrate the expected selective deficit in amnesic recall. Therefore it was decided that the experiment should be repeated with a number of modifications based upon the following rationale.

A selective recall deficit in amnesia was demonstrated by Hirst, Johnson, Kim, Risse, Phelps and Volpe (1986) after the present experiment was carried out. Hirst et al. (1986) manipulated study time to match control and amnesic memory and used two groups of amnesics; these comprised Korsakoff amnesics and a group of mixed aetiology amnesics.

The mixed aetiology amnesics had a non-alcoholic control group and the Korsakoff amnesics had an alcoholic control group. The materials of

the Hirst group experiment were two lists of 40 related words and two lists of 40 unrelated words with two alternative forced-choice recognition. Memory measures included recall, recognition and confidence ratings. The words were presented at a rate of one every 0.5 seconds. This was accomplished by dealing the cards as quickly as possible, rather than presenting them by computer (Volpe, personal communication). There was also an eight second rate of presentation condition.

There was a significant group by memory test interaction found for the non-alcoholic controls and the mixed aetiology amnesics. For the Korsakoff subjects and alcoholic controls the picture was not so clear. Recognition was not matched, as at the eight second rate, recognition was 70% for amnesics and 82% for alcoholic controls. A "yoking" procedure was used which meant that three Korsakoff subjects with the lowest recognition for the unrelated words, and three alcoholic controls with the highest scores for unrelated words were dropped from the sample. This procedure resulted in a significant group by memory test interaction, and a three-way interaction of group, memory test and list structure; that is, whether the words were related or unrelated. Further analysis of this showed that the Korsakoff subjects had significantly lower recall than the alcoholic controls for related words, but that in the case of unrelated words the interaction was not significant. A subsequent logarithmic transform of the interaction almost reached significance ($p < 0.07$). Thus good evidence was provided for a recall deficit in mixed aetiology amnesia for both related and unrelated words. The mixed aetiology amnesics are described by Hirst et al. (1986) as "mild" amnesics. They suggested that the recall deficit may possibly only be apparent in these mild amnesics since they failed to get such clear results with the Korsakoff group for unrelated words.

However, it is important to ensure that it is impossible to show recall deficits for unrelated words with Korsakoff patients before we resort to the explanation that "mild" amnesia is simply different to that of Korsakoff amnesia. In the Hirst et al (1986) study, recognition was not matched in the unrelated words condition, and although they used techniques to rectify this, it is not clear whether they would have found a recall deficit had the levels of recognition been equivalent at the outset of the experiment. Thus the following experiment may make an important contribution by again attempting to establish whether it is

possible to show recall deficits for unrelated words with Korsakoff patients.

Experiment 2

The manipulations in this experiment were based on the results of Experiment 1. The delay to testing was reduced to one hour and it was hoped that this would increase the relatively low level of recall while not significantly increasing recognition. Similarly, the presentation time was increased to eight seconds in the hope that this would produce deeper semantic encoding and facilitation of recall.

Lists of 30 words were used in order to decrease the 85% recognition level obtained with lists of 20 words in Experiment 1. Three lists of 30 words with their concomitant lists of 30 distractors were needed as those subjects who failed to achieve a required recognition level were required to repeat that condition with a suitable modification using the spare list. Depending on the subjects' score the modification was either to increase the number of presentations per word in order to enhance recognition, or to increase the delay to testing in order to reduce recognition. This provision gives the experimenter more control over the matching of control and amnesic recognition.

Method

Subjects These were eight Korsakoff patients with a mean age of 57 years ranging from 47 to 64 years, seven of whom were resident in long-term psychiatric hospitals in Newcastle-upon-Tyne, and one of whom was from Prestwich hospital, Manchester. The amnesic subjects were ST, JB, HS, JA, HK, RS, SM and KH. Control subjects had a mean age of 48 years, ranging from 33 to 66 years.

Materials The materials are listed in appendix E. Three target lists were used, each consisting of 30 unrelated words selected from a pool used by the Manchester Amnesia Research Group.

Design This experiment was a 2 x 2 factorial design. There was one between subjects factor of group (amnesics and controls) and one within subjects factor of memory test (recall and recognition).

Procedure The procedure for amnesics and control subjects differs slightly and is thus reported separately.

Throughout the experiment presentation in the learning and recognition phase was in random order which was achieved by shuffling the index cards. The particular lists used for given conditions was balanced as far as possible given the number of subjects in the experiment. The order of the lists was determined by a simple Latin square, where each list was used with recall instructions, recognition instructions, and also featured as a spare list.

Each amnesic patient was told that they would see some cards, and that they should try to remember them. They were asked to read the words aloud, and told that to help them to remember, the list would be practised a number of times. The number of rehearsal trials was set by the experimenter approximately in accordance with the severity of each patient's amnesia. In the first instance, amnesic subjects had two presentations of the words. After each presentation, the cards were reshuffled. More severely amnesic patients read the list four times, but even at this degree of training none performed at ceiling levels. There then followed a delay of 30 seconds, after which each patient was asked to read through the pack of recognition cards, indicating which of the two words on each card had been a target word. This phase of the experiment took approximately four minutes. If the recognition score was less than 23 out of 30 for amnesics, then the procedure was repeated. They would be given the "spare" list the next day, but this time there would be up to a maximum of four rehearsal trials of reading the list. If the recognition score was approximately 23/30 then, after at least 30 minutes delay, the patients were given the learning trial for the second word list. Subjects were given the same number of trials in this phase as they had received in order to achieve the recognition criterion. This time, after a 30 second delay they were asked to recall as many of the words from the list as possible during two minutes of free recall.

For control subjects, each item was given two four-second presentations, as the lists were shown twice. After a delay of one hour subjects were given the recognition test. Control subjects who scored

more than 28/30 were tested the next day with the "spare" list and a delay of 24 hours to testing. In the event this was necessary for only one of the control subjects. Once a satisfactory level of recognition was obtained the delay to testing was used as a guide in the presentation of the second list. There was a day's delay between testing of list one and presentation of list two. List two was tested by free recall for two minutes after a delay of one hour. The control subject who required a 24 hour delay to achieve the appropriate level of recognition was also given a 24 hour delay to testing in the recall condition.

Results

Raw data are presented in appendix F. A table of means is presented below. A t-test was carried out on the recognition scores of the two groups. This confirmed that there was no significant difference between the amnesic and the control recognition scores ($t(14) = 0.15$, $p > 0.20$). There was no significant main effect of group ($F(1,14) = 0.51$). There was a main effect of memory task ($F(1, 14) = 808.19$, $p < 0.001$). The group-by-memory task interaction was not significant ($F(1,14) = 1.16$).

Experiment 2
Table of Group and Memory Task Means
Maximum score = 30

Amnesic Recall	3.25
Amnesic Recognition	23.88
Control Recall	1.62
Control Recognition	23.88

Discussion

It can be seen that in this experiment although the amnesic and control subjects were matched for recognition, there was no evidence of a recall deficit with amnesia. This is consistent with the findings in Hirst,

Johnson, Kim, Phelps, Risse and Volpe (1986) for Korsakoff subjects' memory for unrelated words, although in their study control and amnesic recognition was not matched in this condition.

Thus it would seem that in Korsakoff and non-Korsakoff amnesics memory for related words demonstrates a selective deficit in recall. In contrast, in memory for unrelated words, non-Korsakoff subjects demonstrate a recall deficit and Korsakoff subjects do not. In other words there is no significant difference between the recall scores for unrelated words of Korsakoff and controls when these groups are matched for recognition. This is a counter intuitive result as one might expect the memory of Korsakoff subjects to be *more* likely to differ significantly from the controls for unrelated verbal material which is more difficult to learn. A possible reason for this may that in the previous two experiments recall and recognition were tested using separate lists. There may have been problems associated with the two lists being insufficiently distinctive, thus causing interference. This would particularly affect recall of unrelated word lists as there is no organising principle of semantic relatedness to help distinguish from which list a potential target may have derived. This would result in prior list intrusions and reduced recall scores. The following experiment solves this problem by testing recall and recognition of the same list.

Furthermore, as Hirst et al. (1986) hint that the selective recall deficit may only be demonstrable in the so-called "mild" amnesics of mixed aetiology, some mixed aetiology amnesics are included in the following study along with Korsakoff patients.

EXPERIMENT 3

Experiments 1 and 2 of this series failed to demonstrate a hypothesised recall deficit with Korsakoff amnesic subjects. This recall deficit was demonstrated in a study by Hirst et al. (1986) in mixed aetiology amnesics for both related and unrelated words, and in Korsakoff subjects for related words only. The Hirst group matched amnesic and control recognition by manipulating presentation time, whereas the present study used the technique of manipulating delay to testing. Therefore the following experiment compares these two means of

matching recognition to ensure that the absence of a selective recall deficit in the Korsakoff group is not simply owing to differing experimental protocols.

The experiment was carried out at the same time as the Hirst group had reached the same conclusion and were also investigating whether they could replicate their former results using delay to testing to match recognition (Volpe, personal communication). They succeeded in demonstrating a recall deficit with this method of matching and their results were communicated to me slightly before the present experiment was completed. The Hirst group's study was published as Hirst, Johnson, Phelps and Volpe (1988).

The Hirst group had two aims in their (1988) study; first, to ensure the result was not specific to the means by which recognition was matched; and second, to generalise their results by investigating yes-no recognition. They used six amnesics of mixed aetiology. The materials were two lists of 30 unrelated words of frequency of greater than 20 occurrences per million (Kuçera and Francis, 1967). The words were presented by computer at a rate of one every eight seconds to amnesics and controls, the amnesics being tested after 30 seconds and the controls after 24 hours. Subjects were given one minute for free recall and then two-alternative forced-choice recognition with confidence ratings of one for most confident to three for least confident of correct response.

This experiment differs from their earlier experiment which manipulated presentation time to match recognition as the former study used 40 words and hand dealing index cards for 0.5 seconds, whereas the (1988) study used 30 words and computer presentation for eight seconds. Also, the former experiment on presentation time used a group of mixed and a group of Korsakoff amnesics, whereas the experiment on manipulation of delay uses only mixed amnesics and no Korsakoff amnesics.

The Hirst group found that they had no main effect of subject group but they did find a main effect of type of memory test and a significant group by memory test interaction. Control recall was significantly greater than amnesic recall. They found that confidence ratings for both groups were equated and that amnesics were applying the ratings in a meaningful way. The study also looked at yes-no recognition under circumstances where amnesic recognition was higher than controls. In this case amnesics still showed poorer recall. They found that there was

no significant difference between groups for yes-no recognition showing amnesics and controls shared the same criteria in this judgement.

The present experiment shares some features of the Hirst group's (1988) study. Instead of testing recall and recognition of separate lists as in Experiments One and Two of the series, this study tests recall followed by recognition of the *same* list as did the Hirst group. This also avoids the problem of inter-list distinctiveness. Furthermore, this experiment uses categorised lists with all items taken from Battig and Montague (1969) as the Hirst group reported that their results were more robust for related rather than unrelated items in the (1986) study. Subjects are given one minute for free recall as they were in the Hirst group's experiment. It was decided that the number of words in the to-be-remembered list should be 42, being more similar to the Hirst group (1986) study on manipulating presentation time. This decision was made as it was thought that control recognition would be more easy to match at the level of 80% with a longer word list as this is a more difficult task. The present study includes six Korsakoff amnesics and three amnesics of mixed aetiology, including two aneurism patients and a post-encephalitic patient.

Finally, the present study differs from that of the Hirst group as it tests both means of matching *within the same experiment.* This is a more satisfactory demonstration than has been provided by the Hirst group. They have claimed that the recall deficit is demonstrable using both manipulation of delay to testing and study time on the basis of two separate experiments. These two studies involved different modes of presentation, by computer and by hand dealt cards; differing numbers of to-be-remembered words; and different amnesic populations, thus the present method is to be preferred as an unequivocal test of the hypothesis.

Analyses of conditional probabilities of recognition and recall and recognition given recall are reported and discussed in a concluding section. These are of interest as they reveal whether or not recall and recognition are independent of one another in each group. For example, it may be the case that given correct recognition normal subjects are more likely than amnesics to subsequently correctly recall a target item. This extra information will help to establish whether the structure of recall differs in amnesic and normal subjects.

Method

Subjects These were six Korsakoff amnesic subjects who were HS, JA, HK, RS, SM from hospitals in Newcastle-upon-Tyne and KH who lived in a hospital in Manchester, and three amnesic subjects who live at home with their families in Liverpool. These were two aneurism subjects who were LP and WP, and a post-encephalitic subject, DF. The mean age of amnesics was 47 years, ranging from 22 to 63 years. The mean age of the nine Presentation Control subjects was 41 years, ranging from 29 to 51 years. The mean age of the nine Delay Control subjects was 39 years, ranging from 27 to 49 years.

Materials The pool of 242 words for this experiment was chosen from Battig and Montague (1969). The words are listed in appendix G. Three lists of 84 words were constructed and called lists A, B and C. The three lists were needed because of the means by which amnesic and control recognition was matched. If amnesic recognition was too poor, or control recognition was too good, the third "spare" list could be used with modified learning or testing instructions to rectify this.

Each of the three lists was further subdivided into two lists of 42 words, called list one A and B. The purpose of this was to use one list of 42 words as a target list and the other list of 42 words as distractors in the two-choice recognition test. This means that the materials of the experiment are balanced for each condition as each list of 42 words appears as both target and distractor in the experiment.

Because all the experimental materials were taken from Battig and Montague (1969) the lists are categorised and related. List one included animals, colours, members of the clergy, relatives, parts of the body, furniture and gems. List two included parts of a building, fruit, weather phenomena, natural earth formations, carpenters tools, musical instruments and items of clothing. List three included occupations and professions, fish, kitchen utensils, human dwellings, flowers, insects and vegetables. Frequencies are not reported for these items as it is assumed that their provenance ensures that they are words in common usage.

For the presentation cards each word was hand-written on 3x5 inch index cards in black ink. For the recognition cards the two words, one from each list of 42 words, were written side-by-side on an index card.

The order of the two words on the card was balanced with respect to which word appeared on the left side and which on the right. In summary, for list A there were two sets of 42 presentation cards which were list one A and list one B, as well as a set of 42 cards for recognition on which a list one A word and a list one B word appeared side by side. Lists two and three were assembled a similar way.

Design This experiment was a 2 x 2 factorial design. There was one between-subjects factor of group comprising amnesics; a group of control subjects for whom presentation time was manipulated, hereafter termed Presentation Controls; and a group of control subjects for whom delay to testing was manipulated, hereafter termed Delay Controls. There was one within-subjects factor of memory task (recall and recognition). Contingency analyses were calculated, and for each of the four resultant probability values a one-way ANOVA was carried out with three levels of subject group. Finally, t-tests were carried out to test whether recall and recognition were independent.

Procedure To achieve matched recognition, in this experiment the amnesic subjects and control subjects had separate procedures.

The amnesic group were presented with each of the 42 target words at a rate of one every two seconds approximately. This process of presenting the list was repeated up to four times depending on the severity of the subject's amnesia, the object being to achieve approximately 80% recognition. The amnesics were then given an immediate test of free recall for one minute. This was followed by a test of recognition. The recognition cards were presented at the subjects own pace under the close supervision of the experimenter. This was necessary to ensure that they did not take too long to respond. If the chosen number of list presentations failed to produce 80% recognition the experiment was repeated with another word list and more presentations of the new list. Four amnesic subjects initially failed to achieve the required recognition score and thus repeated the experiment. Their scores from these unsuccessful trials are contained in Appendix J.

Recognition in the control groups was manipulated in two ways in order to ensure the control subjects were matched to the amnesic subjects at a level of approximately 80%. In the first control group, presentation time was manipulated. The subjects were presented with each of the 42

target words at a rate of 0.5 seconds per card. This rate of presentation was previously used by Hirst Johnson, Kim, Phelps, Risse and Volpe (1986). Subjects were given an immediate test of free recall for one minute. Following this, recognition was tested by presenting the cards at the subject's own pace under experimental supervision.

In the second control group, the delay to testing was manipulated in order to match control and amnesic recognition. The subjects were presented with each of the 42 target words at a rate of eight seconds per card. After 24 hours delay, free recall for one minute was tested. This was followed by a recognition test with the same procedure as that of the previous experimental groups.

Throughout the experiment presentation of the individual cards in the learning and recognition phase was in random order, achieved by shuffling the index cards. The particular lists used for given conditions was balanced as far as possible given the number of subjects in the experiment.

Results

Raw data are presented in appendix H. A table of mean scores for recall and recognition is presented below. T-tests were carried out comparing the recognition scores of both control groups with the amnesic group. There was no significant difference between the recognition scores of the amnesics and the Delay Control group, but a trend could be discerned ($t(16) = 2.00$ $p = 0.06$). There was also no significant difference between the recognition scores of the amnesics and the Presentation Control group ($t(16) = 0.77$, $p > 0.20$). Results were analysed by ANOVA. Hereafter the experimental groups are known as Amnesics, Delay Controls (referring to those for whom delay to testing was manipulated), and Presentation Controls (referring to those for whom presentation time of the targets was manipulated).

A main effect of group was obtained ($F(2, 24) = 4.44$, $p < 0.05$). This was because the amnesic group memory scores differed significantly from the those of the Delay Controls ($p < 0.01$ by Newman-Keuls). The amnesic group mean memory score was 22.28, the Delay Controls mean memory score was 26.33, and the Presentation Controls mean memory score was 22.66. This score is the mean of recall and recognition

combined for each group. It can be seen from these means that the Amnesic group was matched to the Presentation Controls in terms of mean memory score. A main effect of memory task was obtained ($F(2, 24) = 1086.39$, $p < 0.01$).

Experiment 3

Mean Scores for Recall and Recognition

Maximum score = 42

Amnesics	Recall	9.33
	Recognition	35.22
Control Delay	Recall	14.33
	Recognition	38.22
Control Presentation	Recall	11.33
	Recognition	34.00

Hirst et al. (1986) suggested that the selective recall deficit may only occur in mixed aetiology, or "mild" amnesics, so this experiment included some non-Korsakoff subjects. A t-test was carried out comparing the performance of the Korsakoff and non-Korsakoff subjects to see if a difference emerged. In fact, there was no significant difference in the two groups' recall scores ($t(7) = 1.58$, $p > 0.10$). Furthermore, there was no significant difference in their recognition scores ($t(7) = -0.61$, $p > 0.20$). Thus although this result should be viewed with caution owing to small sample size, there is a suggestion in this experiment that the memory of Korsakoff subjects does not differ from that of non-Korsakoff subjects. This experiment showed no evidence of a selective deficit in amnesia. Therefore an alternative explanation for these results must be found. This may be achieved by investigating further the nature of the relationship between recall and recognition in amnesic and normal subjects. Tulving and Wiseman (1975) have argued that it is not adequate simply to look at recall and recognition, but that it is important also to look at their stochastic relation. In a 2 x 2 matrix describing the stochastic relation between recall and recognition, there are three separate values to specify, or three degrees of freedom. In the case of recall and recognition, one value is recall and another is recognition. The third value is a quantity such as: the probability

of an item being both recalled and recognised, P(Rn & Rc); or the probability of recognition given recall, P(Rn|Rc). These three values being defined, the fourth is entailed. Commonly, in studies of this type, the probability of recognition given recall is used; that is, P(Rn|Rc). These analyses were done on the data from experiment 3 not only to examine the *overall* probabilities of recall and recognition, but also to investigate the probability of recognition given recall. The values for the probability of recall; the probability of recognition; the probability of recognition and recall and the probability of recognition given recall are shown in appendix I. For each of the four probability values, one-way ANOVAs were carried out with three levels of subject group comprising amnesics, controls for whom recognition was matched by manipulating presentation time, and controls for whom recognition was matched by manipulating delay to testing. Note that the results of the first two analyses are equivalent to those reported earlier in the section when ANOVA was carried out on the raw data rather than probabilities. They are reported here for the sake of completeness.

The results of these analyses are reported under separate sub headings for clarity.

Probability of Recall

No main effect of group was found ($F(2, 24) = 3.29$, $p > 0.05$). The mean probability of recall was 0.22 for amnesics; 0.27 for the Presentation controls and 0.34 for the Delay controls.

Probability of Recognition

A main effect of group was shown ($F(2, 24) = 4.41$, $p < 0.05$). The mean probability of recognition was 0.84 for amnesics; 0.81 for Presentation controls and 0.91 for Delay controls. A Newman-Keuls analysis showed that amnesic recognition differed significantly from that of the Delay controls at the 0.05 level, and furthermore, that the Presentation controls differed significantly from the Delay controls at the 0.05 level.

Probability of Recognition and Recall

A main effect of group was found ($F(2, 24) = 5.64$, $p < 0.01$). The mean probability of recognition and recall was 0.19 for amnesics; 0.26 for Presentation controls and 0.34 for Delay controls. A Newman-Keuls

analysis showed that amnesic probability of recognition and recall differed significantly from that of the Delay controls at the 0.01 level.

Probability of Recognition given Recall

A main effect of group was found ($F(2, 24) = 19.73$, $p < 0.001$). The mean probability of recognition given recall was 0.81 for amnesics; 0.97 for Presentation controls and 1.0 for Delay controls. A Newman-Keuls analysis showed that the amnesic probability of recognition given recall differed significantly from that of both the Presentation controls and the delay controls at the 0.01 level[1].

In summary, the amnesic group are significantly different to the Delay controls in their probability of recall, probability of recognition and probability of recognition and recall. The Delay controls have the highest probability on all of these measures of the three experimental groups. Furthermore, their mean probability of recognition is quite high, and their mean probability of recognition given recall is 1.0 showing that these two measures of memory are functioning at a high level and are not independent in this group.

The amnesic group and the Presentation controls *do not* differ significantly on their probability of recall, recognition, or recognition and recall. This might suggest that the Presentation controls are in effect simulating amnesic responses in the experiment. However, in contrast, the groups differ significantly with respect to recognition given recall, the amnesic group being significantly worse at this measure at the 0.01 level. This very important result shows that although the two groups have the same level of recall and recognition, and moreover, the same probability of an item being both recognised and recalled, the amnesics are significantly *worse* than the Presentation controls at correctly recognising recalled items. This result of course also holds for the Delay controls. It is conceivable that amnesic subjects generate candidate memories when required to retrieve items from memory, but that they are unable to recognise an item as the target memory for output. To test this hypothesis the probability data were analysed using further t-tests to

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[1] Hirst et al (1986: 449) found a probability of recognition given recall of 0.95 for amnesics and 0.97 for controls. Hirst et al (1988: 759) found that amnesics recognised 92% of the items that they recalled and 86% of the items they did not recall. Controls recognised 100% of items that they recalled and and 84% of items they did not recall.

investigate the independence of recall and recognition in the three experimental groups.

Matched-pairs t-tests were carried out comparing the probability of recognition and the probability of recognition given recall. If recognition and recall are independent then these values should be different. If recall and recognition are not independent then these values will be the same. In other words, if recognition is as probable as recognition *and* recall then there are no instances when recognition occurs in the absence of recall thus the two must be mutually dependent. However, if recognition has a higher probability than recognition and recall, then there are instances when recognition occurs in the absence of recall. Thus recognition is independent of recall.

Recall and recognition are not independent in the case of the Presentation controls ($t(16)$ 5.49, $p < 0.001$). This is also true of the Delay controls ($t(16) = 3.95$, $p < 0.01$). In both cases, it can be seen that there was a positive stochastic relation between recognition and recall. In contrast, recall and recognition are independent in the case of the amnesic group, with $t(16) = 0.53$, $p > 0.20$). Thus it has been shown that in amnesic subjects recall of an item does not always entail recognition of that item, and amnesic subjects in this experiment are demonstrating a deficit in the recognition of recalled items.

Discussion

One aim of the experiment was to investigate whether a hypothesised recall deficit in amnesia only obtained when control and amnesic recognition was matched by manipulating presentation time as in Hirst et al (1986). The present experiment failed to show a significant interaction between group and memory task under *either* means of matching recognition. Therefore there is no evidence of a significant amnesic recall deficit. Another aim of the experiment was to investigate whether a selective deficit of recall in amnesia was demonstrated with related words as opposed to unrelated words which were used in Experiment 1 and 2 of the series. Lists of related words were used in this study, yet the selective recall deficit did not obtain in the amnesic group. Thus the results of this study differ from those of Hirst et al. (1986, 1988) in two

ways. First, in contrast to the findings of the Hirst group, no selective recall deficit was found with related words for a group of amnesics including Korsakoff and mixed aetiology amnesics. Second, unlike the Hirst group's study, this experiment found no selective recall deficit using manipulation of study time to match control and amnesic recognition.

What is the reason for this apparent contradiction? One possibility often suggested in these circumstances is that the results differ because of the different patient populations studied. The Hirst et al. (1986) experiment had both a group of mixed amnesics and a group of Korsakoff amnesics. They report in this experiment that their Korsakoff group did not show the effect so clearly. Moreover, the Hirst et al. (1988) study did not include *any* Korsakoff amnesics.

Experiments 1 and 2 above also failed to show a recall deficit using Korsakoff subjects only. The present experiment failed to show a recall deficit using a group of mixed amnesics including Korsakoff subjects. At this point it is perhaps useful to review the evidence for considering Korsakoff subjects as a distinct group of amnesics and to consider whether herein may be the reason why the findings of the studies in this chapter do not agree with those of Hirst and his group.

Squire (1982) proposed that Korsakoff amnesia is qualitatively different to other types. For example, Korsakoff patients fail to show release from PI whereas other types of amnesics, such as post-encephalitics, do show this. The difference is attributed to the subsidiary frontal lobe damage of Korsakoff patients, and thus it does not represent an obligatory symptom of amnesia. Furthermore, the impairment of temporal order judgements reported in Korsakoff subjects (Huppert and Piercy, 1976) has also been argued by Moscovitch (1985) as being caused by frontal damage. Indeed, Squire (1982) has shown Korsakoff subjects' performance on release from PI and temporal tasks to be correlated with their scores on tests sensitive to frontal damage such as the Wisconsin Card Sort and Benton Word Fluency tests.

Thus Korsakoff subjects are argued to represent a separate group of amnesics on the basis of their frontal damage. However, this neuroanatomical distinction is not useful in explaining why the three experiments of this series failed to show a selective recall deficit. The frontal lobes are said to be important in ordering and planning responses, and if anything, Korsakoff patients should be *more* likely to

show recall deficits as a result of such lesions rather than less likely to do so. Ordering and planning processes are surely necessary to support successful free recall, relying as it does on internally generated cues to retrieval.

Experiment 3 of the series included two aneurism subjects and a post-encephalitic subject, thus adding to the group some mixed aetiology amnesics, and still no effect was found. There was no evidence in this experiment that the Korsakoff subjects differed form non-Korsakoff amnesics.

Analysis of the probabilities of recall and recognition demonstrated that although amnesics did not differ from the presentation controls in their absolute level of recognition and recall, the amnesics were significantly worse than the controls at correctly recognising recalled items. Furthermore, t-tests confirmed that in fact, recognition and recall are stochastically independent in amnesia and this is not the case for control subjects.

One influential theory of recall has been that it contains a recognition component. This is the generation-recognition theory of Bahrick (1970) and Anderson and Bower (1974). It was pointed out by Watkins and Gardiner (1979) that this theory is in its simple form an inadequate account of recall mechanisms. However, Jones (1978, 1980) has argued that the generation-recognition process is one of two recall mechanisms. In generation-recognition, recall is entirely dependent upon recognition, but in the second, direct-access mechanism, recall is independent of recognition. It was suggested by Jones (1983) that generation-recognition recall is subserved by memory schemata, while direct-access recall is subserved by memory fragments. Thus, the present phenomenon of a tendency to recall without recognition in amnesia would be attributed according to the dual-mechanism view, to the occurrence of direct-access recall, and thus to relative preservation of memory fragments in amnesia. This possibility is examined further in chapter 5 of this thesis.

In summary, the experiments in this series have shown that perhaps the functional deficit which results in amnesia is not a selective recall deficit relative to that of controls who have approximately equivalent recognition. Rather, the functional deficit of amnesia may have its roots in an inability correctly to recognise recalled items. One possible account for this is found in studies which demonstrate a loss of

familiarity of memories in amnesia, usually evidenced by poor confidence judgements (Mayes and Meudell, 1981a, b; Meudell and Mayes, 1984). Having been output as a response in free recall, the item does not evoke familiarity when encountered by the amnesic at recognition. The implications of these findings for theories of amnesia are discussed in chapter six of this thesis.

The next chapter reports a series of three experiments which investigate a hypothesised deficit in amnesic spatial memory and also explore amnesic incidental and intentional processing of spatial information.

CHAPTER FOUR

AN INVESTIGATION OF THE HYPOTHESISED SPATIAL MEMORY DEFICIT IN AMNESIA

The context memory deficit hypothesis predicts that amnesics will manifest a disproportionate deficit for spatial memory. This spatial recall deficit is hypothesised to result from a deficit in automatic processing of contextual information (Hirst and Volpe, 1984b). Therefore a further prediction is that incidental encoding of location will show a spatial location recall deficit, whereas intentional encoding of the location of target items will improve amnesic spatial memory. A corollary of this is that amnesic recognition and recall may be reduced as a consequence (Hirst and Volpe, 1984a). The empirical justifications of these predictions are explained in more detail below.

It has been widely suggested that memory consists of item and contextual information. Item information roughly corresponds to the identity of the target, and various other attributes constitute its context. This contextual information includes physical characteristics, such as colour or alphabetical case; temporal aspects, such as recency or frequency of presentation; and spatial aspects such as location. It has been claimed that contextual information plays a vital part in the processing of memories in normal subjects and that the lack of this contextual information results in amnesia. Evidence has been provided of a specific amnesic deficit for temporal information (Huppert and Piercy, 1976; Squire, 1982); for frequency information (Huppert and Piercy, 1976); for source information which is memory for the source from which an item of information was imparted (Schacter, Harbluck and McLachlan, 1984) and for spatial information (Smith and Milner, 1981; Hirst and Volpe, 1984). It is on the last of these, the hypothesised spatial memory deficit in amnesia, that the present work focuses.

A spatial context deficit had not been shown for Korsakoff patients, as the Smith and Milner (1981) study was carried out on right and left temporal lobectomy patients, and the Hirst and Volpe (1984) study used material-specific and global temporal amnesics. Therefore it is of importance to establish whether or not these deficits are also shared by Korsakoff amnesics. If Korsakoff patients suffer a spatial memory

deficit, then this contextual deficit may be a general factor in all amnesias. If not, then the amnesia incurred in Korsakoff syndrome must be argued to be of a qualitatively different type to that of other aetiologies.

The context deficit hypothesis argues that the underlying cause of contextual memory deficits is an impairment of automatic processing in amnesia. Hasher and Zacks (1979) argued that in normal subjects contextual attributes are automatically integrated during the effortful encoding of a to-be-remembered item. There have been challenges to this claim, for example, an opposing view is given by Park and Mason (1982) who showed with normal subjects that contextual memory such as spatial and colour memory was *poorer* in an incidental condition than in an intentional condition when subjects were required effortfully to encode these attributes. They concluded that colour and spatial information *were not* automatically encoded. Hirst and Volpe (1984) concur with Hasher and Zacks (1979) and go on to suggest that the automatic component of contextual memory processing is absent in amnesia. Hence, when required to encode objects they will not encode their context. The result will be poor context memory and also lower item memory, as context is argued to be playing a vital part in retrieval. Furthermore, if required to remember the context of objects, amnesic subjects are forced to attempt to encode contextual information effortfully with a subsequent cost to item memory.

There now follows a review of experiments on spatial memory in normal and amnesic subjects.

Studies of Memory for Spatial Location

The next section describes the experimental techniques used in previous studies and also provides a background of findings in cognitive psychology which are of relevance to the context memory deficit hypothesis of amnesia. The discussion begins with a brief definition of terms.

In experiments on contextual memory, memory measures are often studied under intentional and incidental conditions using orienting tasks. In an intentional condition subjects are informed that they should learn specific aspects of the to-be-remembered information, and they are also

informed of the type of retrieval task they will later perform. Incidental conditions refer to cases where subjects are given orienting tasks which concentrate their efforts on aspects of the to-be-remembered information which are peripheral to the subsequent retrieval task, and indeed, the subjects are unaware of the nature of the retrieval task they will later perform. Orienting tasks are designed to allow the experimenter to manipulate the memory processes of the subject. The tasks provoke specific types of memory operation are argued in some cases to prevent other types of memory operation. For example, an orienting task detecting the presence of the letters "e" and "g" can result in poor memory for the targets as semantic processing is argued to be inhibited by this task (Hyde and Jenkins, 1973). However, there have been many criticisms of this characterisation of memory processing (see Eysenck, 1982, for a review).

Previous experiments had suggested that the contextual information of colour and case of words was better remembered under intentional conditions (Light and Berger, 1974; Light, Berger and Bardales, 1975) but with a trade-off that item information was slightly impaired under these conditions. In contrast, Zeichmeister, McKillip, Pasko and Bespalac (1975) and von Wright, Gebhard and Kartunnen (1975) found no difference in item or spatial memory under incidental and intentional instructions, and no trade-off. These studies seemed to be contradictory, in that they found that memory for some types of contextual information, that is spatial location, improved with intentional instructions; however, intentional instructions did not improve memory for other types of context, such as colour and case of words.

Many experiments in the neuropsychology of spatial memory are derived from Mandler, Seegmillar and Day (1977). This experiment looked at item and location memory for 16 toys on a board of 32 squares. The memory measures included number of objects recalled, number of correct locations and number of correct locations containing recalled items. At retrieval of spatial location this study provided the subjects with the locations marked on the board and the subjects decided which item was to be placed on which location. Mandler et al. (1977) hypothesised that the contradictory results of earlier experiments on case, colour, and spatial memory may have occurred because of the circumstances of their incidental conditions. It was for this reason the Mandler et al. (1977) study employed two differing incidental conditions. These comprised one

condition in which subjects were told to remember the objects, and location was *not* mentioned (the standard incidental task); the other incidental condition involved an orienting task of pricing the objects (this was called the the "true" incidental task). Mandler et al. (1977) found that their incidental condition using a pricing task resulted in lower memory scores than the other incidental condition where subjects were required only to learn the objects' names. They argued that this showed the importance of using an appropriate orienting task to ensure a truly incidental condition. They argued that their other incidental condition had allowed some automatic encoding of location to occur and thus was not properly an incidental condition.

They also showed that intentional instructions resulted in significantly better recall of objects, locations and joint object-location scores than the true incidental condition. However, the effect of instructions on contextual memory, that is, location-only, was not significant. This study did not demonstrate a trade-off in item and location information; this supported some previous findings (Zeichmeister et al., 1975; von Wright et al., 1975) but opposed the findings on colour and case memory (Light et al., 1974, 1975). Mandler et al. (1977) argue that spatial memory is therefore qualitatively different to colour memory. Furthermore, in contrast to colour information, subjects actually use spatial information in their encoding of the targets and there is no cost involved in this strategy. Interestingly, they found that the standard incidental condition resulted in a *better* spatial memory score than the intentional condition, with no trade-off of item and location memory. This means that subjects recalled *more* item and location information when they only intended to encode item information, than when they intended to encode both attributes. In summary, this experiment showed improved memory scores with intentional instructions, but with no significant differential effect on location memory and no trade-off of item and location memory.

However, Schulman (1973) in a similar experiment reported that both item location and recognition were impaired in his intentional condition. He argued that spatial location memory was automatic and was actually *disrupted* by intentional instructions. This view opposes that of Hirst and Volpe (1984) who claim that although spatial information is automatic, it is *enhanced* by intentional instructions.

Smith and Milner (1981) applied the general procedure of Mandler et al. (1977) to temporal lobectomy patients. Memory was tested for 16 objects on a blank board which was not marked out as a grid. They studied this under incidental instructions only and pricing was used as the orienting task for this condition. Memory measures included object recall and object location as measured by displacement from original position on the board. Unlike Mandler et al. (1977), subjects were not cued with the original locations, but were required to recall these. They found that left temporal patients recalled fewer objects than right temporal and control subjects. However, the right temporal subjects showed clear deficits in spatial memory in spite of almost normal object recall. Their findings implicate the right hippocampal region in the automatic encoding of spatial location. Smith and Milner (1981) discussed the implications of their findings with reference to the critical lesions implicated in amnesia. They argued that the connectivity of the temporal lobe structures is such that the findings must be extended by testing of patients with different pathologies to determine the extent of contribution of the temporal lobe in spatial memory.

Further evidence of the role of the temporal lobes in spatial memory was reported by Beatty, MacInnes, Porphyris, Troster and Cermak (1989). They demonstrated preserved topographical memory in a patient with right temporal lobectomy. Their task involved memory for geographical information which was encoded pre-traumatically. They concluded that the integrity of the right temporal lobe is not required for retrieval of pre-established spatial memory, although it is important in the acquisition of new spatial memories.

Hirst and Volpe (1984b) extended these findings with a group of mixed aetiology patients including material-specific and global temporal amnesics. They also employed both incidental and intentional instructions in order further to explore the hypothesised deficit in automatic processing in amnesia. In this study the original locations of the objects were marked by pieces of card so all that remained was for the subjects to decide which toy was located on which location. In this respect the Hirst study differs from that of Smith and Milner (1981). It further differs from the Smith and Milner study in that it uses 32 objects rather than 16.

Hirst and Volpe (1984b) found that intentional instructions did not affect the memory scores of control subjects. This was argued to be

because normal subjects were habitually encoding context anyway. In contrast, intentional instructions *improved* amnesic spatial memory. This was as a result of amnesics being unable to encode context automatically and thus they improved their scores in the condition which required them to encode context effortfully. Amnesics had a slightly lower item memory for the intentional condition indicating a trade-off of item and context memory.

To recapitulate, Mandler et al. (1977) found that recall, recognition and spatial memory were better under standard incidental instructions than under intentional instructions, with no trade-off of item and contextual memory. In contrast, Hirst and Volpe (1984a, b) found no difference in incidental and intentional memory for control subjects but amnesics improved their spatial scores under intentional instructions with a consequent trade-off of target memory as the improvement in contextual memory was effected at a cost to item memory.

The following series of experiments aim to extend the findings on context deficits to Korsakoff patients, and further, to seek evidence of a hypothesised deficit in automatic processing in amnesia.

EXPERIMENT 4

Method

Subjects The mean age of the seven amnesic subjects was 58 years ranging from 47 to 66 years. The amnesic subjects were ST, JB, HS, JA, HK, RS, and SM. The five control subjects had a mean age of 42 years, ranging from 27 to 52 years. They were security staff of the University of Manchester.

Materials The materials are listed in appendix K. The target items consisted of two sets of 16 objects which were a range of miniature common household objects, kitchen utensils, animals, clothes and vehicles. They were purchased from toyshops and gift shops and were brightly coloured and of approximately the same size (5cm). These were presented on a 4x4 grid, where each of the 16 squares was of sufficient size to accommodate the to-be-remembered objects. The displays of the objects on the board were randomised between subjects. A cover was used

to restrict the total viewing time of the overall display. Each set of eight objects had a corresponding set of eight recognition cards, with one card for each object. These were hand written in black ink on 3x5 index cards and featured the name of the target object plus four distractor words which were common nouns. These words were written in a column down the middle of the index card. The position of the target on the card was controlled so that it appeared equally often in each of the five positions reading down the card. The order of the cards randomised by shuffling between subjects.

Design This experiment was a 2 x 2 x 2 x 2 factorial design. This comprised two levels of the between-subjects factor of group (amnesics and controls); two levels of the within-subjects factor of treatment (incidental and intentional encoding of spatial location); two levels of the within-subjects factor of memory task (recognition and spatial memory); and finally two levels of the within-subjects factor of trial order (trials one and two).

Procedure Amnesic and control subjects underwent slightly different experimental procedures. In total, each amnesic and control subject learned two displays of eight objects under incidental conditions and two displays of eight objects under intentional conditions.

The amnesic subjects were tested over two days. On the first day, they were seated in front of the board which was covered while the experimenter explained the procedure. When the subject understood the instructions he or she was presented with the first set of objects displayed on the board in the first of two incidental conditions. The experimenter pointed to each object in turn for approximately eight seconds and asked the subject to estimate the cost of the item which was represented by the miniature object. Following this, the board was replaced and the subject was shown the first recognition card of the list and asked to chose the word which identified the target object. Then, whether or not this choice was correct, the subject was then given the correct object to replace on the board. This process was repeated until all eight recognition cards had been seen and all 8 objects were replaced on the board by the subject. After a delay of 30 minutes the second set of objects was presented to the subject according to the same procedure as before. It was hoped that the

short delay would reduce interference and render the two lists more discriminable.

On the second day, the amnesic subjects were presented with the final two sets of objects under intentional encoding conditions. The same procedure was followed except that instead of estimating the cost of each object the amnesic subject was asked to spend the eight seconds presentation time trying to learn the spatial location of the object. To help them to do this they were additionally given a 4x4 grid drawn on paper on which to mark with a cross the position of each object. The paper was withdrawn following presentation.

The control subjects underwent the same procedure as the amnesics except that instead of having retrieval tested immediately after presentation they had a delay of one hour in each trial.

Results

Memory measures consisted of the total of items recognised and the total of items correctly located, both with a maximum score of eight. Raw data are presented in appendix L. Memory scores were analysed by ANOVA.

There was no main effect of group ($F(1,10) = 1.92$). A main effect of treatment was obtained ($F(1, 10) = 6.38$, $p < 0.05$) with the mean incidental memory score being 4.95 and the mean intentional score being 4.15. This shows that intentional instructions to remember both the items and their locations reduced recognition and spatial memory scores.

No group-by-treatment interaction was found, $F(1,10) = 0.90$). This showed that intentional instructions did not affect the amnesic group more than the control group.

A main effect of memory task was obtained ($F(1, 10) = 134.00$, $p < 0.01$). This was because the spatial task was more difficult than recognition. No significant group-by-memory task interaction was obtained ($F(1,10) = 0.72$). Thus there is no evidence that the spatial location task is more impaired in amnesia than are recall and recognition.

Experiment 4

Table of Group, Treatment, Memory Task and Order Means

Group	Treatment	Task	Order	Mean
Amnesics	Incidental	Recognition	Trial one	7.71
Amnesics	Incidental	Recognition	Trial two	7.57
Amnesics	Incidental	Spatial memory	Trial one	3.29
Amnesics	Incidental	Spatial memory	Trial two	1.86
Amnesics	Intentional	Recognition	Trial one	6.43
Amnesics	Intentional	Recognition	Trial two	6.00
Amnesics	Intentional	Spatial memory	Trial one	3.29
Amnesics	Intentional	Spatial memory	Trial two	2.71
Controls	Incidental	Recognition	Trial one	7.60
Controls	Incidental	Recognition	Trial two	7.00
Controls	Incidental	Spatial memory	Trial one	2.00
Controls	Incidental	Spatial memory	Trial two	2.60
Controls	Intentional	Recognition	Trial one	6.20
Controls	Intentional	Recognition	Trial two	5.80
Controls	Intentional	Spatial memory	Trial one	1.20
Controls	Intentional	Spatial memory	Trial two	1.60

No treatment-by-memory interaction was obtained ($F(1,10) = 2.80$) suggesting that in both groups intentional instructions did not differentially affect spatial memory scores. Thus there is no evidence that intentional instructions improve spatial memory. No group-by-treatment by memory task interaction was obtained ($F(1,10) = 1.17$). This means that intentional instructions did not affect spatial memory differentially in the amnesic group. Thus there is no significant evidence of an amnesic spatial memory deficit which is reduced with intentional instructions.

A group-by-memory task by order effect was obtained ($F(1,10) = 4.92$, $p < 0.05$). A table of means is shown below.

Experiment 4

Table of Group, Memory Task and Order Means

Maximum score = 8

Amnesic Recognition Trial 1	7.07
Amnesic Spatial Memory Trial 1	3.29
Amnesic Recognition Trial 2	6.79
Amnesic Spatial Memory Trial 2	2.29
Control Recognition Trial 1	6.90
Control Spatial Memory Trial 1	1.60
Control Recognition Trial 2	6.40
Control Spatial Memory Trial 2	2.00

The interaction is probably due to amnesic spatial memory having decreased in the second trial and control spatial memory having increased in the second trial. This finding shows a disadvantage of using four lists in this type of experiment. As it is apparent that subjects have relatively good memory for eight objects, it would seem that an increase in the number of to-be-remembered objects is preferable to multiple lists.

Discussion

In summary this experiment has shown main effects of treatment and memory task and a significant group by memory task by order interaction. The experiment demonstrated neither an amnesic spatial memory deficit with incidental instructions as shown by Smith and Milner (1981), nor an improvement in amnesic spatial memory with intentional instructions as shown by Hirst and Volpe (1984). One reason for this may be the means by which subjects were asked to encode the spatial positions intentionally by marking them on a grid with a cross. Perhaps this was too abstract a task which did not result in an integrated representation of the target identity and location. It should be further noted that control spatial memory scores were lower than those of

amnesics in the first trial of each condition in the experiment. It is not clear why this occurred. However, in this experiment the control subjects, who were University Security Staff, reported that they initially felt uneasy about doing experiments, although their confidence improved once they had evidence that they were doing the experiment correctly.

In the Hirst and Volpe (1984b) study, the subjects were presented at retrieval with the locations of the display marked by cards. Thus the task was very different in nature to that required of subjects in the present experiment. In effect, in the Hirst and Volpe (1984) study the spatial memory task was of cued recall, which is an easier task than having to recall the locations.

The lack of a spatial memory deficit in the amnesic group in the present study may simply be owing to this difference in technique. However, the Smith and Milner (1981) study did not employ this means of cuing and yet they still showed an amnesic spatial memory deficit. In principle, if temporal amnesics are able to generate both the location of the items and the correct item, then this protocol should also be appropriate for Korsakoff subjects. Second, if subjects' memory is tested both for the locations and for the items themselves, then analyses are possible which can reveal differential effects on item recall and spatial recall. This can not be so easily addressed if, as in the Hirst and Volpe (1984) study, the spatial task is more like cued recall or a discrimination test.

Finally, it is worth noting an intriguing point. As mentioned in some detail above, the spatial memory task in the present experiment is more demanding than that of Hirst and Volpe (1984). Therefore one would have thought it more likely the trade-off of item and contextual memory described by Hirst and Volpe (1984) should be demonstrated, yet this is not the case. As has been noted earlier, there is no evidence that intentional instructions improve amnesic spatial memory at the cost of a decrement in target recall and recognition. This aspect of the present experiment's results may not thus be attributed to differing experimental techniques.

EXPERIMENT 5

The results of the previous study suggested a number of ways in which the experimental protocol may be modified and these were implemented in the following experiment. First, two lists of 16 target objects were used in preference to multiple lists of smaller size. The purpose of this was to eliminate the order effect noted in Experiment 4 and also to reduce recognition which was often at ceiling levels in that experiment.

Having reduced the number of lists, a delay of one day between each list was possible. This should increase the discriminability of the lists in the two conditions and also enhance spatial memory for the targets by reducing interference. (With four lists this delay was not possible, as it was inconvenient to the subjects).

Second, the orienting task of marking crosses on grids in order intentionally to encode spatial position was replaced by a task where subjects were asked simply to look at the targets and their positions and to concentrate on trying to remember them. This was because the previous experiment showed that this task was too demanding and itself reduced memory scores rather than enhancing them.

Third, the orienting task of giving the cost of each target during the incidental condition was replaced by a task in which the subject stated whether the object represented something which was larger or smaller than a chair. This was done because the amnesic subjects were very confused about the prices of objects. They became distracted from the memory task by wondering about the current money system. It was decided that size estimation was more within their scope.

The experimental instructions were read from cards to both groups of subjects. This was because the control group of Experiment 4 seemed less at ease with the experiment than the amnesics and this resulted in a marked difference in performance. This may have occurred because the experimenter was more reassuring in explaining the instructions to the amnesics. This situation was avoided in the following study as the instructions to both groups were tightly controlled by reading from cards.

Finally, both groups were given a free recall task as well as recognition and spatial memory tasks. This enables further differential hypotheses to be investigated. For example, intentional instructions may

affect recall but not recognition, though this comparison was not possible in the previous experiment.

Method

Subjects The mean age of the eight amnesic subjects was 57 years, ranging from 47 to 66 years. The amnesic subjects were ST, JB, HS, JA, HK, RS, SM, and KH. The mean age of the eight control subjects was 54 years, ranging from 46 to 66 years.

Materials The materials are listed in appendix M. The experiment required a white board marked with thirty-two squares, which was eight squares long and four squares wide, and measuring 79 x 39 cm. The long horizontal side was the edge nearest to the subject. This was used to display the objects to the subject. The target items consisted of two sets of 16 objects which were a range of miniature common household objects, kitchen utensils, animals, clothes and vehicles.

There were two sets of 16 recognition cards, one for each set of objects. These showed the target and four distractors printed one beneath the next, in a column down the centre of the card. The target word position was randomised with respect to order in the column on each card.

A cover was used to hide the target objects until it was necessary to reveal them to the subjects, and also to cover the display quickly at the end of the presentation period. This restricted the subject's learning time to the allotted duration. The positions of the objects for each of the two conditions were chosen pseudorandomly. The positions were distributed across the display board such that there were four objects on each horizontal segment of the board. The two resulting displays, one for each condition, remained constant between subjects, although the objects occupying the positions were varied between subjects. The order of presentation of the objects, and the order of presentation of the recognition cards was random between subjects.

Design This experiment was a 2 x 2 x 3 factorial design. There was one between-subjects factor of group, (amnesics and controls); two levels of the within-subjects factor of treatment, (incidental and intentional

instructions); and three levels of the within-subjects factor of memory task (recall, recognition and spatial memory).

Procedure The procedure differed slightly for the two groups, therefore they are described separately.

Amnesic Group

Day one The experimenter arranged the first set of sixteen objects and covered them from view. The appropriate set of recognition cards was placed at hand. The patient was then brought into the room and informal talk accustomed the patient to both the experimenter and the unaccustomed surroundings. The experimenter explained the procedure and checked that the patient understood the instructions. The patient was then asked to cover his or her eyes whilst the experimenter uncovered the objects. At a sign from the experimenter, the patient looked at the first toy as the timer was started. The experimenter pointed at each object in a planned sequence, for exactly five seconds. The amnesic subject judged whether the object represented something which was bigger or smaller than a chair. After this the timer was re-started and the board was removed. After one minute, the patient was asked to recall as many of the objects as possible. Two minutes were allowed for free recall. The experimenter then reminded the subject of the procedure for recognition and replacing the objects in their original position. The patient then read a recognition card, and chose the word which identified the target object. Whether or not this choice was correct, the patient was given the correct object from the display and asked to put it on the board in its place. This process was repeated until all 16 cards had been presented and all 16 objects replaced in their positions. This part of the experiment lasted about eight minutes.

Day two No testing was done with the amnesic group on this day of the study.

Day three The patients were given the learning trial for the other list of sixteen objects in the experiment. The instructions were similar to those in the previous trial, except that instead of judging whether each toy was bigger or smaller than a chair, they were simply told to carefully note that object's position on the board. They were frequently reminded

throughout of the importance of remembering both the object, and its position. After one minute of intervening conversation, the patients were given recall and recognition instructions, as in the previous day's trial.

Control Group

The control subjects had the same procedure as the amnesic sample, except in the following respects. In the control testing there were delays of 24 hours between presentation and testing of list one of the experiment. This was followed by a delay of 24 hours before presentation of list two of the experiment. Testing this list occurred after a day's delay, on day four. Whereas the amnesic version of the study lasted three days with no testing on day two, the control version of the study lasted four days with testing on each day. This procedure was adopted in order to compromise between the need for longer delays in the control sample to avoid ceiling effects, and to preserve the same degree of distinctiveness between lists in both the control and amnesic groups.

Results

Raw data are presented in appendix N. It is important to establish the chance level of performance to eliminate the possibility that guessing accounts for the results in this experiment. For the purposes of this experiment it was calculated that levels of performance in excess of 0.5 for spatial memory scores were above chance. In the event, both amnesics and controls reached this criterion. The data were analysed by ANOVA.

A main effect of group was found ($F(1, 12) = 5.89$, $p < 0.05$). The source of this may be seen in the group means where the amnesic mean memory score was 6.83 and the control mean memory score was 8.88.

A main effect of treatment was also found ($F(1, 12) = 7.02$, $p < 0.05$) this was due to mean performance of recall and recognition being poorer in the intentional condition (the mean incidental score was 8.30 and the mean intentional score was 7.42). This result lends support to the claim that when subjects concentrate on encoding the locations of the objects their memory for the toy's identity is decreased. There is no evidence in this experiment that either group may improve their spatial scores given intentional instructions as no significant interaction between group and treatment was found ($F(1,12) = 1.93$).

Experiment 5

Table of Group Treatment and Memory Task Means

Maximum score = 16

Amnesic Incidental Recall	4.75
Amnesic Incidental Recognition	13.75
Amnesic Incidental Spatial Memory	2.62
Amnesic Intentional Recall	4.63
Amnesic Intentional Recognition	12.50
Amnesic Intentional Spatial Memory	2.75
Control Incidental Recall	7.83
Control Incidental Recognition	15.50
Control Incidental Spatial Memory	5.33
Control Intentional Recall	5.17
Control Intentional Recognition	14.17
Control Intentional Spatial Memory	5.33

A main effect of memory task was obtained ($F(2, 24) = 141.27$, $p < 0.01$). This corresponds to recall and spatial memory being more difficult tasks. Mean recall was 5.59, mean spatial memory was 4.01, and mean recognition memory was 13.97. No group-by-memory task interaction was obtained although there was a main effect of group ($F(1,12) = 0.33$). Thus there is no evidence of a significant spatial memory deficit in the amnesic group, as the spatial location task was not impaired in amnesia to any greater extent than were recall and recognition impaired. No treatment-by-memory task interaction was obtained ($F(1,12) = 2.53$). This means that intentional instructions affect all memory scores in both groups, and no memory measure is differentially influenced by intentional instructions. Thus the reduction in recognition and recall is not accompanied by enhanced spatial memory. No three-way interaction of group-by-treatment-by-memory task interaction was obtained ($F(1,12) = 1.89$). Thus there is no evidence that a trade-off occurred in the amnesic group resulting in poorer recognition and recall but better spatial memory in relation to controls.

Discussion

In summary, these results show main effects of group, treatment and memory task and no interactions achieve significance. Only one claim of the Context Memory Deficit account of amnesia receives limited support. There was a reduction in recall and recognition in the amnesic group when subjects concentrated on encoding spatial location. However, this was also found in the control group. Thus this experiment did not replicate the findings of Smith and Milner (1981) in right temporal amnesics, of a spatial recall deficit. As their study did not include intentional instructions the Smith and Milner study is neutral with respect to the other findings of the present experiment.

The most surprising result in the present study was that although intentional instructions reduce recognition and recall in both groups, there was no effect on spatial memory. Hence the reduction in recall and recognition with intentional instructions was not evidence of the amnesic subjects sacrificing item memory in order to retain contextual memory. In other words, the "trade-off" of Hirst and Volpe (1984b) was not found. The amnesic group had an incidental spatial recall of 2.62 and an intentional spatial recall of 2.75. Similarly, the controls had an incidental spatial recall of 5.33 and an intentional spatial recall of 5.33. In contrast, Hirst and Volpe (1984a, 1984b) found no difference in incidental and intentional memory for control subjects, but did find that amnesics improved their spatial scores under intentional instructions. Furthermore, the amnesics suffered a consequent trade-off of item memory. The present results are more in keeping with those of Mandler et al. (1977) who found with normal subjects that recall, recognition and spatial memory were reduced under intentional instructions, as compared with incidental instructions, and who further found no trade-off of item and contextual memory.

So why do the present results differ from those of Hirst and Volpe (1984a, 1984b)? First, there may simply be a scaling problem. The results may be a function of the increased difficulty of intentionally encoding location. This factor may actively be reducing recognition and recall, but merely preventing improvement of spatial memory. Thus a slightly different protocol may produce results which are more like those of Hirst

and Volpe. Second, the differing results may have occurred because the Hirst and Volpe (1984) study in effect tested spatial memory by cued recall, which means the spatial task of the present experiment is far more demanding. However, again as mentioned earlier, this cannot account for the failure to demonstrate a trade-off of item and contextual memory as one would expect this to be *more* likely to occur if the contextual task were more difficult.

Third, it is important to ensure that the lack of improvement with intentional instructions is not simply a result of initial ceiling level performance in the incidental condition. Although the ideal level of recognition decided upon for the experiment was 80%, the amnesics achieved 86% and the controls 97% and this leaves little room for improvement. This fact may have contributed to the failure to find a group by memory task interaction. A detailed discussion of the impact of ceiling effects and the importance of matching recognition at equivalent levels in analyses of this type is given in chapter three.

Thus a final version of this experiment was carried out in order further to investigate these possibilities, but with important modifications. Notably, Experiment 6 below uses words as the targets rather than objects. Evidence that this modification is appropriate is found in Pezdek, Roman and Sobolik (1986). Pezdek et al (1986) compared relocation accuracy for words and objects in a similar paradigm. They found that delay decreased item memory and relocation accuracy for objects. However, in the case of words, delay impaired item memory but *did not* decrease relocation accuracy. They explain that item and location memory are affected by delay independently in the case of words, whereas item and location memory are more integrated in the case of objects. Pezdek et al. go on to explain that this is probably due to spatial characteristics being more salient in the perception and processing of objects than of words. As the present experiment uses delay to testing in the case of the controls, if it is repeated using words as to-be-remembered items, then this should increase the possibility of showing differential effects on item and contextual memory.

There may be two further reasons why the present results are so different from those of Hirst and Volpe (1984a, 1984b). The first may be that Korsakoff amnesics differ from mixed-aetiology amnesics such that spatial memory deficits are not a feature of their amnesia. This would mean that the claims that spatial memory is damaged in amnesia, and

further, that this is because of an automatic encoding deficit, are sustainable, but simply not applicable to Korsakoff subjects.

Secondly, these results also show that the Korsakoff amnesics, unlike mixed-aetiology amnesics, do not show an improvement in spatial memory with intentional instructions. This inability to improve requires explanation.

It may be the case that the problem lies in the characterisation of what constitutes an automatic process. Hasher and Zacks (1979), Shiffrin and Schneider (1977) and Schneider and Shiffrin (1977) have argued that a truly automatic process cannot be influenced by intentional instructions. In their characterisation this is one of the defining features of automaticity. According to this formulation, the claim that spatial context is mediated by automatic processing is correct, and that automatic processing is damaged in amnesias, but that effortful encoding cannot compensate for this in the case of Korsakoff subjects.

However, this does not explain why the Korsakoff subjects are different to other amnesics. It is preferable to try to solve these seeming contradictions without recourse to simply stating that what is true for a group of mixed-aetiology amnesics is false for Korsakoff patients, particularly if this solution does not explain why. In terms of developing theories of amnesia it is more conservative to continue to investigate the phenomenon more closely before postulating multiple types and causes of amnesia. For this reason, the next experiment explores the nature of spatial memory in more detail, looking particularly at the structure of spatial recall. It was found in chapter three that important differences between amnesics and controls emerge when one considers not only recall and recognition memory, but also their stochastic relationship. It is suggested that this approach will also be fruitful when applied to the data generated by the current series of experiments.

EXPERIMENT 6

Experiment 5 failed to demonstrate either an amnesic spatial recall deficit (Smith and Milner, 1981), or an improvement in amnesic spatial memory with trade-off of item and contextual memory given intentional instructions (Hirst and Volpe, 1984). Several methodological reasons for this were suggested in the preceding section, and modifications designed to deal with these are detailed below. One possibility was that the experiment failed to demonstrate the effects as a result of ceiling effects in control and amnesic recognition and recall in the incidental condition. Therefore, the following modifications were implemented.

The first of these modifications was that common words were used rather than small objects. This has a number of advantages. First, they are less salient and memorable. The aim of this was to reduce recognition in both groups in the incidental condition, and it was hoped that the manipulation would not prove too difficult for the amnesic group. Furthermore, Pezdek et al. (1986) demonstrated the independence of item and location memory in words. This lends further justification to the choice of words as materials in the present experiment, as such a dissociation between item and contextual information can facilitate a demonstration of context deficits, in that it should be possible for experimental manipulations to affect location memory without affecting item memory or vice versa.

Second, presentation times were maintained at five seconds and a delay of one hour to testing was chosen for the control subjects. This reduced the time spent on the experiment and thus it was hoped would increase control subjects' motivation. It was hoped that this would have the effect of enhancing spatial memory, thus maximising the chances of differences between the groups. If the amnesics do in fact suffer a spatial memory deficit then this manipulation would facilitate control spatial memory more than that of the amnesics. It was hoped that the loss in list discriminability would not in itself *reduce* the control spatial scores by too much.

A semantic orienting task was chosen to give the subjects the best conditions for forming a rich encoding of the targets to maintain good recall, particularly in the amnesic group (see Butters and Cermak, 1980,

Responses of Amnesic JA in Experiment 6

	sword	silk	birch	eagle	game	liner	
lion	tractor	shell	silver	novel	swimming	cloves	
			pepper	gas	wood		

Target display for Amnesic JA in Experiment 6

lion			silk		game		eagle
	tractor		liner	sword		birch	
novel		shell		swimming			silver
	cloves	pepper			gas		wood

Figure 1

Aspects of the Display in the Spatial Memory Experiments

but see also Mayes Meudell and Neary, 1980). Subjects were asked to judge whether the words referred to items which were living or non-living.

Finally, this experiment uses a range of techniques of analysis, some of which are applied for the first time in amnesia research. The aim of this was to elucidate aspects of amnesic performance which cannot be explored using conventional analyses. Memory measures of recall, recognition and spatial memory used in experiments one and two were investigated: However, there are other measures of spatial memory which have previously been employed. For example, Warrington and Baddeley (1974) use a different measure for spatial memory, that of measuring distance of displacement from the correct location. Notably, these studies did not use displays on grids as did the present series of experiments. The displacement method can be argued to be inappropriate for use with experiments were such grids are used. The reasons for this are discussed in detail below.

The main criticism of the use of distance of displacement measure in the present experiment is that it violates the following two premises. The first is that the only reason for employing more complex measures of retrieval is if they more adequately describe the responses of the subjects. The second is that in an experiment of this type it is preferable if all items in the analysis may be treated as being equivalent. The use of distance of displacement measures would not conform to the first premise in the present experiment because it would not reveal the extent of a subject's memory for the display any more than the conventional spatial location score. This is shown by the following example. Subjects may either be disposed to see the display as four horizontal lines of objects or as eight columns. These two approaches would profoundly affect the subjects' encoding of the items.

Consider a subject choosing the horizontal encoding (see for example figure 1). He or she may perhaps generate a sentence reading across the board which contained the items. This strategy is seen in the responses of the amnesic subject JA and was also reported by the control subjects. At retrieval, this subject could be expected to place the objects in the correct order across the board but not necessarily in the correct square. Thus any scoring scheme which ignores this aspect will not record what is, in fact, a convincing demonstration of recall, giving this subject a zero score.

Furthermore, suppose we assume subjects are able to have partial encodings of the items in which a proportion of the to-be-recalled information is retained in memory. Suppose further, that a subject could know that an item was "somewhere in the top right" (see figure 1). If the subject in this case does not correctly place the item, he or she may place it in any one of three squares for a displacement score of one; or in one of two squares for a displacement score of two. Any other available squares would not fit the description of "somewhere in the top right".

Next consider a case where a subject knows an item was "somewhere in the middle". Here the subject can incorrectly place the item in a total of eight squares for a displacement score of one; or 11 squares for a displacement score of two. Thus there are a greater number of locations around the target square which may plausibly be chosen in error. The displacement scores quoted can be seen to be inadequately describing the extent of the two subjects' memory for the display. Each of the two subjects may be said to have begun the task with equivalent degrees of memory for the target location. However, probabilistically, the subject trying to place the item in the top right is more likely to choose the correct position.

Moreover, consideration of the example above where a subject encodes items as a horizontal line shows another limitation of the displacement method. Suppose that in the original display there were three items which were positioned along the bottom line of the display, and that there was an empty square on either side of each item (see figure 1). Suppose further that the subject places the items in the correct order, but leaves two empty squares between two of the items instead of one. In this case, the subject may get a displacement score of one for that item. A second subject could place the same item not on the bottom line of the display, but the line above, thus showing he or she did not encode the three items as belonging together on the bottom line. The net effect of this could be that of two subjects with the same displacement scores one of them may have a poor memory for the display as a whole, while the other has attempted a more holistic encoding. In conclusion, these examples also show that items cannot be thought of as equivalent as some have the potential for larger displacement scores than others.

The method of measuring and analysing displacement chosen for this experiment attempts to avoid some of these pitfalls. Spatial memory is re-scored using Lenient criteria. Each subject is given a score of 0.5 for

placing the item anywhere on the correct horizontal line and a score of 0.5 for placing an item on a anywhere on the correct column. This score is summed as the Lenient Score. An item which is in the correct column and the correct horizontal line is in the precisely correct position and therefore scores one. (The maximum Lenient Score for the display as a whole is therefore eight). The Lenient Score provides an index of the extent to which subjects were *almost correct* in their responses rather than placing the items at random.

Finally, the nature of spatial recall was further investigated by looking at conditional probabilities of correct spatial recall given recognition and recall. This allows a number of important questions to be addressed which were not possible in earlier analyses. Examples of such issues are whether amnesics are as likely as controls correctly to place an item if it has been both recalled and recognised; and also whether control subjects are more likely to place an item correctly under conditions of that item's correct recall and recognition than they are when they have failed to recall the item but have gone on subsequently to recognise it. The individual data for these probabilities are reported in Table 1 and raw data in Appendix Q.

Method

Subjects These mean age of the six amnesic subjects was 57 years, ranging from 47 to 62 years. The amnesics were JB, HS, JA, HK, RS, and SM. The six control subjects had a mean age of 39 years ranging from 30 to 50 years.

Materials The targets were two lists of 16 common nouns, one list for the incidental condition and one list for the intentional condition. Details of the materials are reported in Appendix O. Each list appeared equally often in the incidental and intentional condition.

The targets were displayed on the 8x4 board used in Experiment 5. There were two displays in which the positions of the words were chosen in a pseudorandom fashion. One display was assigned to the incidental condition and one to the intentional condition and this was held constant throughout the experiment.

Each of the target words appeared on a 3x5 index card along with four distractor items of similar frequency. The serial position of the

target and distractors was balanced on the cards. The order of the recognition cards was randomised by shuffling between subjects.

Design This was a 2 x 2 x 3 factorial design with two levels of the between-subjects factor of amnesia, two levels of the within-subjects factor of treatment (intentional and incidental instructions), and three levels of the within subjects factor of memory task, (recall, recognition and spatial memory).

Procedure The procedure for amnesics and controls differs slightly so they are described separately.

Amnesic Group

Amnesic subjects were seated in front of the board which remained covered while the experimenter explained the protocol. A modification of the instructions given in Experiment 5 was used. When the details were clear to the subject he or she was presented with the first set of words displayed on the board. The experimenter pointed to each word in turn for five seconds and the amnesic subject was required to state whether the word described an object which was living or not living. The order of pointing at the target words was randomised between subjects. When each word was seen for the correct period the subject was asked to close his or her eyes and the board was removed. The subject was then asked to free recall for one minute. The board was then replaced in front of the subject and there then followed the five-choice recognition test. The subject was shown each card at his or her own pace under the supervision of the experimenter to ensure that each subject did not linger too long over specific targets. Then whether or not the subject had chosen the target correctly the subject was given the correct word and asked to replace it on the board in its previous position.

This procedure was repeated with a modification on the following day for the intentional condition. The modification was that instead of stating whether each target was living or not living, the subjects were told to spend the presentation time trying to learn the locations of the words.

Control Group

The control subject protocol differed from that of the amnesics in that there was a one hour delay between presentation of the targets and

testing for retrieval. In all other respects the procedure was the same for both groups.

Results

Raw data are reported in appendix P. In this experiment the chance level of spatial memory is 0.5. It can be seen that whereas the control subjects meet this criterion, the amnesic subjects do not. This topic is discussed later. The results were analysed by ANOVA.

Experiment 6

Table of Group Treatment and Memory Task Means

Maximum score = 16

Amnesic Incidental Recall	4.33
Amnesic Incidental Recognition	12.50
Amnesic Incidental Spatial Memory	0.50
Amnesic Intentional Recall	3.16
Amnesic Intentional Recognition	11.00
Amnesic Intentional Spatial Memory	0.83
Control Incidental Recall	6.67
Control Incidental Recognition	14.50
Control Incidental Spatial Memory	3.50
Control Intentional Recall	3.67
Control Intentional Recognition	10.83
Control Intentional Spatial Memory	2.83

No main effect of group was obtained ($F(1,10) = 1.94$). There was a main effect of treatment ($F(1, 10) = 8.56$, $p < 0.05$). The mean incidental memory score was 7.08, and the mean intentional memory score was 5.39. The group-by-treatment interaction was not significant ($F(1,10) = 1.68$).

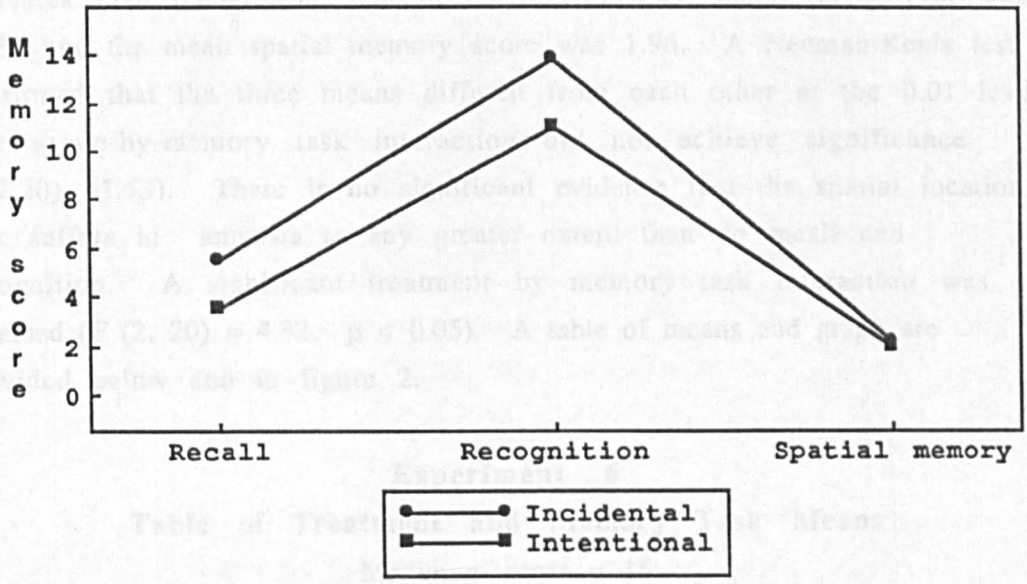

Figure 2

Experiment 6 Significant Treatment by Memory Task Interaction

There is no significant evidence in these data that intentional encoding affects one group more than the other.

A main effect of memory task was shown ($F(2, 20) = 189.52$, $p < 0.01$). This is because recall and spatial memory are more difficult memory measures than recognition. Mean recall was 4.41, mean recognition was 12.33 and the mean spatial memory score was 1.96. A Neuman-Keuls test confirmed that the three means differed from each other at the 0.01 level. The group-by-memory task interaction did not achieve significance ($F(2,20) = 1.53$). There is no significant evidence that the spatial location task suffers in amnesia to any greater extent than do recall and recognition. A significant treatment by memory task interaction was obtained ($F(2, 20) = 4.32$, $p < 0.05$). A table of means and graph are provided below and in figure 2.

Experiment 6

Table of Treatment and Memory Task Means

Maximum score = 16

Incidental Recall	5.50
Incidental Recognition	13.50
Incidental Spatial Memory	2.00
Intentional Recall	5.50
Intentional Recognition	10.92
Intentional Spatial Memory	1.83

Intentional encoding appears to depress recognition and recall in both amnesics and controls but it does not affect spatial memory in this experiment. Therefore there is no significant evidence here that the reduction in recognition and recall results in a concomitant improvement in spatial memory scores.

No group-by-treatment by memory task interaction was found ($F(2,20) = 0.05$). Therefore, not only is there no evidence of an overall spatial memory deficit in the amnesic group but also, there was no significant disproportionate amnesic spatial memory deficit in the incidental condition.

Contingency Analyses

So far, data have been reported which reveal relationships between mean recall, recognition and spatial memory. However, it is also of interest to investigate the extent of spatial memory for items contingent upon those items having been successfully or unsuccessfully recalled or recognised. Therefore, an analysis of conditional probabilities of spatial memory given recall and spatial memory given recognition was made. These techniques and their results are described below.

The analysis of probabilities yields the following probability values which describe spatial memory under circumstances of a subject possessing recognition memory for the target. These probability values describe situations where the subject has a moderately complete encoding of the object and its location. They are described as follows:

1. The probability of spatial recall given correct recognition and recall of the target. This is expressed as $P(Sp \mid {}^{+}Rn \ \& \ {}^{+}Rc)$.
2. The probability of spatial recall given correct recognition in the absence of recall. This is expressed as $P(Sp \mid {}^{+}Rn \ \& \ {}^{-}Rc)$.

The following probability values describe spatial memory under circumstances in which a subject has no recognition memory for the target. They are described thus:

3. The probability of spatial recall when a subject has failed to recognise an item which has been recalled. This is expressed as $P(Sp \mid {}^{-}Rn \ \& \ {}^{+}Rc)$.
4. The probability of spatial recall in the absence of both recognition and recall. This is expressed as $P(Sp \mid {}^{-}Rn \ \& \ {}^{-}Rc)$.

These four possible probability values were calculated for both groups and both treatment conditions. Spatial memory scores were then categorised to disclose whether that item of spatial recall had previously been recalled and recognised.

Table 1
Contingency Analysis
Individual Subject Data

Amnesic Subjects
Incidental Condition

	P(Sp| $^{+}$Rn & $^{+}$Rc)	P(Sp| $^{+}$Rn & $^{-}$Rc)	P(Sp|$^{-}$Rn & $^{-}$Rc)	P(Sp|$^{-}$Rn & $^{+}$Rc)
HS	0	0	0	0
HK	0	0	0	0
RS	0	0	0	0
JB	0	0.08	0	0
JA	0	0.14	0	0
SM	0	0	0.17	0

Intentional Condition

	P(Sp| $^{+}$Rn & $^{+}$Rc)	P(Sp| $^{+}$Rn & $^{-}$Rc)	P(Sp|$^{-}$Rn & $^{-}$Rc)	P(Sp|$^{-}$Rn & $^{+}$Rc)
HS	0	0	0	0
HK	0	0	0	0
RS	0	0	0.12	0
JB	0	0.2	0	0
JA	0.25	0	0	0
SM	0	0	0	0

Table 1 (continued)
Contingency Analysis
Individual Subject Data

Control Subjects
Incidental Condition

	P(Sp| $^{+}$Rn & $^{+}$Rc)	P(Sp| $^{+}$Rn & $^{-}$Rc)	P(Sp|$^{-}$Rn & $^{-}$Rc)	P(Sp|$^{-}$Rn & $^{+}$Rc)
C1	0	0.2	0	0
C2	0.67	0.6	0.5	0
C3	0	0.2	0	0
C4	0.17	0.3	0	0
C5	0.29	0	0	0
C6	0.22	0	0	0

Intentional Condition

	P(Sp| $^{+}$Rn & $^{+}$Rc)	P(Sp| $^{+}$Rn & $^{-}$Rc)	P(Sp|$^{-}$Rn & $^{-}$Rc)	P(Sp|$^{-}$Rn & $^{+}$Rc)
C1	0	0	0	0
C2	0.38	0	0.67	0
C3	0.37	0.2	0.33	0
C4	0	0.1	0	0
C5	0	0.22	0	0
C6	0.2	0.33	0	0

Unfortunately, the low scores and low variance of scores in the amnesic group were such that analysis was not possible, so the interesting questions concerning the extent to which recognition and/or recall predicted correct spatial memory cannot be addressed. However, it may be seen that two amnesic subjects, JA and JB have higher scores for $P(Sp \mid {}^{+}Rn \ \& \ {}^{+}Rc)$ and $P(Sp \mid {}^{+}Rn \ \& \ {}^{-}Rc)$ than for the other two categories. However, neither subject recalled an item and went on to place it correctly in the incidental condition. That is, the single item which they succeeded in placing correctly was recognised but not recalled. Furthermore, JA has a higher probability of spatial memory given recognition and recall in the intentional condition, where he is also the only amnesic who succeeded in placing an item in the correct position and also recognising and recalling that item. Interestingly, this amnesic has a probability of spatial memory given recognition and recall of 0.25 which is of a similar magnitude to the control probabilities for this category.

Control subjects 4 to 6 inclusive have higher scores for $P(Sp \mid {}^{+}Rn \ \& \ {}^{+}Rc)$ and $P(Sp \mid {}^{+}Rn \ \& \ {}^{-}Rc)$ than for the other two category responses. It can be seen that the probability of spatial recall given recognition and recall or recognition-only is higher in the control group than in the amnesic group, suggesting that guessing was probably not an important factor in the control group.

Spatial Memory Scores using Lenient Criteria

One further set of data remain to be analysed from this experiment, these are the spatial memory scores using Lenient criteria, hereafter, "Lenient Scores". These are tabulated in Appendix Q. A comparison between amnesic and control Lenient scores was made for both treatment conditions using t tests. There was no significant difference between amnesic and control Lenient scores in the incidental condition ($t(10) = 0.22$, $p > 0.20$). The was also no significant difference between the amnesic and control Lenient Scores under intentional conditions ($t(10) = 0.45$, $p > 0.20$). Thus the amnesics were performing at the same level as controls in this measure of the ability to place the objects in an approximately correct position. In other words, amnesics are shown to

have as much partial memory for the locations in the display as do control subjects.

This could mean one of two things. Either it shows that amnesics attain a normal level for this type of memory, or it suggests that the control subjects themselves are performing at a very poor level for this type of memory, because they have encoded the locations in an all-or-nothing manner.

This may be discovered by looking in more detail at the extent of subjects' recall and recognition memory for those items which are scored by lenient criteria. If memory for the items is poor, then it suggests that their being placed in an almost correct position was merely chance. If memory for those items is quite good, it supports the hypothesis that the Lenient scores are capturing an ability of amnesics to place items in almost the correct position with the same level of performance as control subjects. Note that throughout the next section the term "memory scores" refers to ***items scored by lenient criteria*** and not to total recall and recognition scores as discussed in previous sections.

The extent of memory for those items which were placed in locations scored by lenient criteria was thus examined. First, for each lenient scored item it was noted whether or not it had been recalled and recognised. Then Amnesic and Control recall and recognition for these items was compared for both treatment conditions using a 2x2x2 ANOVA. This comprised two levels of the between-subjects factor of group, comprising amnesics and controls; two levels of the within-subjects factor of treatment, which were incidental and intentional conditions and two levels of the within-subjects factor of memory task which were recall and recognition.

No main effect of group was found in the analysis ($F(1,10) = 1.02$), which shows no difference in overall performance between amnesics and controls. The mean amnesic score was 2.21 and the mean control score was 2.71. A main effect of treatment was found ($F(1, 10) = 7.64$, $p < 0.05$), which mirrors the findings of earlier analyses that intentional instructions reduce memory scores. The mean incidental score was 3.00 and the mean intentional score was 1.92. The group-by-treatment interaction was not significant ($F(1,10) = 0.11$) which shows that intentional instructions did not improve amnesic memory scores.

A main effect of memory task was shown ($F(1, 10) = 27.67$, $p < 0.005$). This is because recall is so much more difficult than recognition as mean

recall was 1.42 and mean recognition was 3.50. A Newman-Keuls test showed that mean recognition differed from mean recall at the 0.01 level.

There was no significant group-by-memory task interaction which shows that amnesics had the same amount of recall and recognition memory as controls ($F(1,10) = 0.96$). The treatment-by-memory task interaction did not achieve significance thus intentional instructions did not affect memory scores significantly ($F(1,10) = 1.05$). The group-by-treatment by memory task interaction was also not significant ($F(1,10) = 2.60$). This final result also reiterates no difference between the memory scores of the amnesics and the controls. A table of means is provided below.

Experiment 6

Table of Group Treatment and Memory Task Means for Memory for Lenient Scored Items

Amnesic Incidental Recall	1.33
Amnesic Incidental Recognition	5.00
Amnesic Intentional Recall	1.17
Amnesic Intentional Recognition	3.33
Control Incidental Recall	2.66
Control Incidental Recognition	4.50
Control Intentional Recall	1.33
Control Intentional Recognition	3.50

In summary, this analysis has shown, first, that amnesics and controls are equally good at placing items in locations scored by lenient criteria; and second, that the amnesics and the controls have the same amount of memory for those lenient scored items.

Discussion

The analyses carried out on the recall, recognition and spatial memory scores are discussed first. This experiment demonstrates main effects of treatment and memory task and a significant treatment-by-memory task interaction. The experiment shows no significant evidence of an amnesic contextual deficit in automatically encoding spatial memory. Furthermore, it does not provide evidence that this deficit is reduced using intentional instructions.

The failure to demonstrate an amnesic spatial memory deficit may again have been caused by a failure to reduce control recognition to 80%. In the incidental condition, control recognition was at 91%, dropping to 67% in the intentional condition. As has been argued elsewhere, it is advisable to avoid such ceiling effects when attempting to demonstrate results such as this interaction of group and memory task.

One of the aims of this series was to extend the findings of Smith and Milner (1981) and Hirst and Volpe (1984) to include Korsakoff amnesics. This was important as such a replication would add weight to the suggestion that a spatial memory deficit was a fundamental functional deficit in amnesia. So perhaps the failure to demonstrate such a deficit means that Korsakoff subjects represent a separate, discrete group of amnesics and do not share the same functional deficits as mixed-aetiology amnesics? A second possibility is that spatial memory deficits are not a candidate for the fundamental functional deficit in amnesia.

The findings of Kohl (1984), and Kohl and Brandt (1984) militate against these suggestions. These constituted a significant replication of the Hirst and Volpe (1984) findings with a group of Korsakoff subjects. Kohl looked at spatial memory, frequency memory and temporal memory in Korsakoff subjects, frontal subjects, controls and alcoholic controls.

For the spatial test, she investigated memory for four words or abstract line-drawings arranged in the corners of a rectangle. Kohl found that in the case of verbal material a Newman-Keuls analysis showed that Korsakoff subjects were significantly worse than the frontal subjects, control subjects and alcoholic controls under both treatment conditions. Furthermore, the Korsakoff subjects improved their performance under intentional instructions. The same effects were found for non-verbal material. This confirms the Hirst and Volpe (1984) finding that the automatic encoding deficit was compensated by

intentional instructions to encode the locations. She also found that verbal recognition of the groups did not differ significantly under incidental conditions but that Korsakoff subjects were significantly worse than the frontal patients, the controls and the alcoholic controls under intentional conditions. This result was also found with non-verbal material. This confirms the Hirst and Volpe (1984) claim of a trade-off in item memory for contextual memory.

Thus Kohl (1984) shows that Korsakoff subjects perform in the same way as Hirst and Volpe's (1984) mixed-aetiology amnesics and Smith and Milner's (1981) right temporal amnesics in that they show a spatial memory deficit. This opposes the argument that the results of the present series can be attributed to the patient group since Kohl (1984) has shown spatial memory deficit effects with Korsakoff patients. It is intriguing to note that she did not find a spatial memory deficit or frequency memory deficit with her Frontal subjects. Korsakoff subjects differ primarily from other amnesics as a result of their subsidiary frontal damage. Kohl's study confirms that frontally damaged subjects do not show the pattern of results given by Hirst and Volpe's (1984) mixed aetiology amnesics. The fact that the Korsakoff subjects, *with their subsidiary frontal damage*, still showed the spatial memory deficit militates against the claim that the results of the present series are merely owing to the use of Korsakoff subjects rather than mixed aetiology amnesics such as those of Hirst and Volpe (1984), or temporal amnesics such as those of Smith and Milner (1981). In fact, Kohl (1984) states explicitly that Korsakoff subjects...

> "were unable to encode contextual information under incidental learning conditions, but were able to encode this information under intentional conditions, suggesting that the deficit is one of automatic encoding. The failure is due to failure of automatic encoding in the episodic memory system, this is central to their amnesia and not attributable to their purported frontal damage."

It should be noted that although Kohl (1984) states that her Korsakoff subjects had frontal damage as evidenced by Wisconsin Card Sort scores, they did not perseverate during the experiments. Perseveration is a claimed to be key symptom of frontal damage. Thus it

must be assumed that the Korsakoff subjects in Kohl's study have only slight frontal lesions.

It may be seen from the above that Kohl subscribes to the characterisation of automatic and effortful processing outlined by Hirst and Volpe (1984a, 1984b) which has been questioned elsewhere in the present series of experiments. Furthermore, she also claims that the deficient automatic processing is a component of episodic memory. Evaluation of the status of this claim is beyond the scope of the data reported in the present series. The main point of Kohl's statement is that the amnesia of Korsakoff syndrome is not different in character to that of stroke, aneurism or temporal lobe damage, and that the subsidiary frontal damage of Korsakoff subjects is not a factor in the pattern of their memory deficits. Therefore, the fact that the present series failed to replicate the findings of Smith and Milner (1981) and Hirst and Volpe (1984) may not be attributable to the different type of patient group.

Let us now turn to the further analyses carried out in this experiment concerning the Lenient score for spatial memory. These showed that first, amnesics and controls are equally good at placing items in locations which are scored by lenient criteria; and second, that the amnesics and the controls have the same amount of memory for those lenient scored items. This is a novel finding in the literature of amnesic spatial memory. It is an interesting result as it shows that amnesics have a residual capacity of spatial memory. Furthermore, this residual capacity seems to be very similar in nature to the working of normal memory under the same circumstances. It could also be claimed that this level of performance also shows successful processing of context at some level of description by amnesics. Thus the claim of poor spatial context memory in amnesia is weakened, and consequently, the context deficit hypothesis of amnesia is not supported by these data.

General Discussion

The experiments contained in this chapter investigated a hypothesised deficit in amnesic spatial memory. Three experiments were reported, each of which failed to demonstrate such a deficit. Furthermore, they did not provide evidence of improved amnesic spatial memory under intentional instructions. When subjects' responses were

analysed using techniques which looked at partial recall of spatial location there was clear evidence that amnesic and control subjects were equally good at remembering approximate target locations. Both groups showed equally good memory for the general, global aspects of the display. Thus the final experiment in the series demonstrated that although amnesic memory for the correct locations of the items was at chance, they had a residual ability to place the items in locations which were near misses. Finally, those items which were placed in locations scored by lenient criteria were equally well recalled and recognised by both groups.

The context memory deficit hypothesis claims that amnesics have a deficit in automatic encoding of contextual information, and that memory for contextual information is improved when it is intentionally encoded. The hypothesis has difficulty in accounting for the results reported in this chapter. First, no spatial recall deficit was found in the amnesic group and this weakens the claim that amnesics suffer from contextual deficits. Second, no improvement in spatial memory was seen with intentional instructions. This weakens the claim that instructions intentionally to encode context overcome the automatic processing deficit. Furthermore, Smith (1988) has reported a single-case study of the temporal amnesic HM (Scoville and Milner, 1957) in which his spatial memory was at chance and he failed to improve his spatial memory score with intentional instructions.

The lack of a spatial memory deficit in the present series of experiments could arise in several different ways. The first possibility is that spatial memory is not, after all, automatically encoded by normal subjects, and amnesics do not show deficits in spatial memory for this reason. With this characterisation it is thus still possible to claim an automatic encoding deficit in amnesia.

The second possibility is that spatial memory *is* automatically encoded. The argument would then proceed thus; since the amnesics in this study have been shown to have no specific deficit in spatial memory, and since spatial memory is automatically encoded, then it must be argued that there is no automatic encoding deficit in amnesia. It is beyond the scope of this particular series of experiments to resolve the question of whether spatial memory is, in fact, automatically encoded. However, the absence of an effect of instructions on spatial memory is suggestive, as this fulfils one of Schneider and Shiffrin's (1977) criteria for

automaticity. If it is accepted that context is encoded automatically then the second possibility is favoured, that is, amnesics do not have a problem with automatic encoding as they do not have a spatial memory deficit.

Third, there is the possibility that these results obtain because of the patient population studied. All three of these experiments used Korsakoff amnesic subjects, and it is sometimes argued that this amnesia is different in type to that of amnesia which is caused by vascular lesions or temporal lesions. The basis of this claim is that Korsakoff subjects have subsidiary frontal damage and that this factor causes a difference in the functional impairments. If applied to the results reported in this chapter, the argument would have to be that Korsakoff subjects were not impaired at spatial memory because they suffered subsidiary frontal damage. It is improbable that a function is facilitated by damage to a particular area. It is also unlikely that it is the interaction of the diencephalic and frontal damage of Korsakoff syndrome which results in preserved spatial ability in these subjects. In any event this type of argument would be challenged by Kohl (1984) who did find a spatial memory deficit in Korsakoff subjects, and who did not find the spatial deficit in frontal subjects (although it is probable that her Korsakoff subjects suffered only minimal frontal damage). In other words, in her experiments, Korsakoff subjects with diencephalic and frontal lesions have an impairment of spatial memory and frontal patients do not.

A fourth possibility is that spatial deficits in amnesia are only demonstrable if particular experimental procedures are used. For example, the Kohl (1984) study used only four locations, and the Hirst and Volpe study provided subjects with markers for each position at retrieval. These tasks are easier than those reported in this chapter. In contrast, the Smith and Milner (1981) study did not have such simple tests of spatial recall, and instead required subjects to free-recall 16 locations, which is of equivalent difficulty to the task set in the present series of experiments. On the other hand, their display was not on a grid, and their measure of spatial recall was that of displacement from target location. This procedure perhaps increases the likelihood of showing a spatial recall deficit as there is more scope for subjects to make greater errors. (These issues were further discussed in the introduction to the final experiment of this series). Strangely, it would seem from the above that the spatial memory deficit is shown in easier tasks than the present experiment.

Thus it may be that the particular procedure chosen for the present series of experiments was an important factor in the failure to replicate. However, if spatial memory deficits are to be argued to be the functional deficit of amnesia, it is disquieting if they are so labile as one would expect that such a fundamental component of amnesia ought to be relatively robust to demonstrate.

In conclusion, in the previous two chapters some theoretically important patterns of amnesic performance have failed to be displayed in the group of amnesics in this study. However, when more detailed analysis using slightly different techniques was employed, some interesting new findings emerged. For example, in this chapter, an investigation of partial recall of spatial location showed that amnesics had very similar performance to that of control subjects. These analyses also emphasised the importance of investigating not simply the total memory scores in experiments, but also looking at the relationship between spatial memory, recall, and recognition in terms of their patterns of stochastic dependence. For example, in Experiment 3 more detailed analyses of the structure of recall in amnesic memory were carried out, and recall and recognition were found to be independent in the amnesic group but not independent in the control group. This suggests that the links between items in amnesic memory are deficient in comparison with those of normal controls, such that success in accessing an item by recall does not entail recognition. These findings indicated that it would be useful to look more generally at the structure of recall in order to determine how different aspects of an item were linked together to form an integrated memory unit. Perhaps the fundamental difference between amnesics and controls is apparent in the way in which such links are formed in memory? The following chapter investigates this possibility.

CHAPTER FIVE

THE STRUCTURE OF RECALL IN AMNESIA

This experiment aims to show what can be learnt about the nature of the underlying memory representation of triads of common words by comparing the efficacy of single and multiple cues to retrieval. The distributions of correct and incorrect responses to given cues can be used to infer the organisation of the memory structure, allowing a comparison between two models to be made. These comprise the fragment model (Jones 1976, 1978, 1984; 1987; Rubin and Wallace, 1989), and the schema model (Ross & Bower 1981). Data from amnesic subjects were compared to those of control subjects to reveal differences in the memory representations of the two groups.

In terms of theoretical background, the models differ with respect to three main characteristics described below. The description will be made more concrete by reference to the experiment reported here where the target materials are groups of 3 common words, or triads, which are presented together.

The fragment model assumes that the 3 members of the triad, here A, B, and C, are linked to each as shown in figure 3.

It is assumed that the structure is encoded by a subject either in part or in its entirety. When a member of the triad, A, is given as a cue at recall it will allow retrieval only of those other members to which it has intact links. Similarly if two members of a triad, B and C, are given as cues they will only allow retrieval of the third member, A, if it has an intact link to either or both of B and C. A complete encoding of a triad may be termed a "Full" fragment.

To illustrate the case of incomplete encoding of the structure consider the following. There are three possible types of fragment in which two members of a triad are linked and the third is not. These comprise:

A being linked to B but neither being linked to C.
A being linked to C but neither being linked to B.
B being linked to C but neither being linked to A.

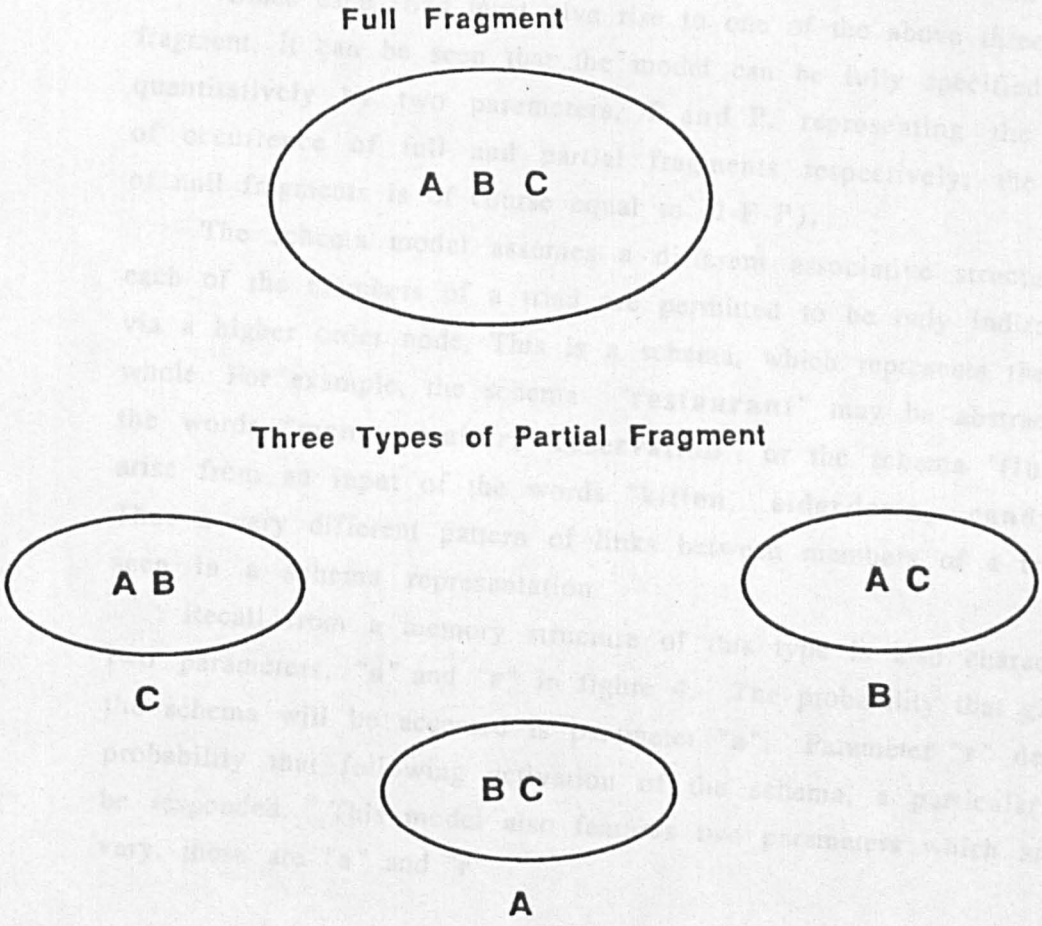

Figure 3

Diagram of Possible Types of Fragments for a Triad of Words, A B & C

All three of these fragments may be described as "partial" fragments and termed "P" fragments. Thus can be seen that if the target materials are triads of common words there are five possible types of memory representation. The third and final possibility is that none of the triad members are linked in memory. This may be termed a "Null" fragment.

Since each triad must give rise to one of the above three types of fragment, it can be seen that the model can be fully specified quantitatively by two parameters, **F** and **P**, representing the probabilities of occurrence of full and partial fragments respectively; the probability of null fragments is of course equal to (1-**F**-**P**).

The schema model assumes a different associative structure. Here each of the members of a triad are permitted to be only indirectly linked via a higher order node. This is a schema, which represents the triad as a whole. For example, the schema "**restaurant**" may be abstracted from the words "**menu, waiter, reservation**", or the schema "**fluffy**" may arise from an input of the words "**kitten, eiderdown, candyfloss**". Thus a very different pattern of links between members of a triad can be seen in a schema representation.

Recall from a memory structure of this type is also characterised by two parameters, "**a**" and "**r**" in figure 4. The probability that given a cue the schema will be accessed is parameter "**a**". Parameter "**r**" denotes the probability that following activation of the schema, a particular word will be responded. This model also features two parameters which are free to vary, these are "**a**" and "**r**".

Fragment Model Predictions The fragment model predictions are apparent if figure 3 is considered. This diagram displays graphically the three categories and five individual configurations of fragment possible when the target material is a triad of three common words. In this experiment the subject is cued first by one word of the triad and then by that word repeated plus a second member of the triad. This is incremental cuing, and is illustrated by the following example. If the target triad were "**menu, waiter, reservation**", then at retrieval the subject would be prompted with one member, for example, "**waiter**", and asked to complete the triad. Then the subject would be prompted with "**waiter, menu**", and would be asked to add the third word, if they had not already done so. Thus there can be four types of subject response for a triad of members **A, B, C,** as follows:

1. The subject recalls both targets given the first cue. This is termed an α response, thus:

Probability of α responses = P(A-B & C) = F

2. The subject recalls one target given the first cue. This is termed a β response. As can be seen in the diagram above, this situation can arise in 2/3 of the P cases. For example, A can cue B if the subject has encoded the fragment "A is linked to B but neither is linked to C". Similarly, A may cue C if the subject has encoded the fragment "A is linked to C but neither is linked to B". Thus:

Probability of β responses = P(A- B or C) = 2/3 P

3. The subject recalls one target given both the first and the second cue. This is termed a γ response. It can be seen from the diagram above that this type of response will arise in the remaining P cases.

Probability of γ responses = P(A-O, B- C or A) = 1/3 P

4. The subject recalls no targets. This is termed a δ response, and arises from all null cases:

Probability of δ responses = P(A- O, B- O) = 1 - F - P

Schema Model Predictions The schema model assumes the associative structure depicted in figure 4. In this diagram "a" represents a link allowing access to the schema, and "r" represents a link allowing a response of a given target to be given. As with the fragment model, there are four types of response a subject may give if it is assumed that a schema has been encoded. These four response types are α, β, γ, and δ, and they are described fully above. For ease of exposition this information will not be repeated here. As has already been noted, a subject is cued first with one member of the triad, and next with that cue repeated plus a further cue of the second member of the triad. Therefore:

1. Number of α responses = P(A-B & C) = ar^2

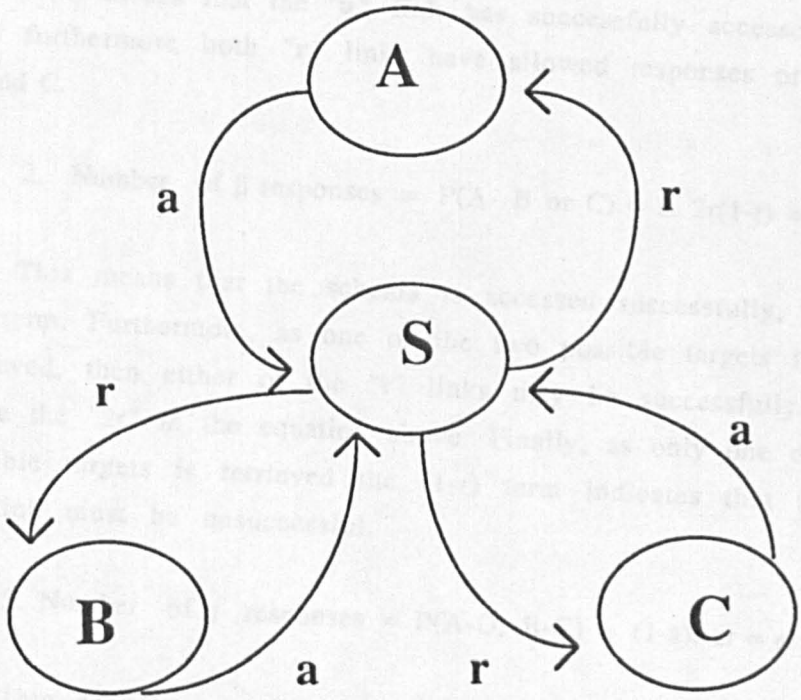

Figure 4

Schema Representation of the Triad ABC.

This means that the "a" link has successfully accessed the schema, and furthermore both "r" links have allowed responses of the two targets B and C.

2. Number of β responses = P(A- B or C) = a. 2r(1-r) = $2ar - 2ar^2$

This means that the schema is accessed successfully, hence the first "a" term. Furthermore, as one of the two possible targets is successfully retrieved, then either of the "r" links may be successfully negotiated, hence the "2r" in the equation above. Finally, as only one of the two possible targets is retrieved the (1-r) term indicates that the remaining "r" link must be unsuccessful.

3. Number of γ responses = P(A-O, B-C) = (1-a). ar = $ar - a^2 r$

This describes the situation when the schema is not accessed by the first cue, (1-a), but the second cue both accesses the schema and outputs a response, hence "ar" in the equation above.

4. Number of δ responses = P(A-O, B-O) = (1-a) [(1-a)+a(1-r)] $+a(1-r)^2$

Taking each part of this equation in turn, this may be explained thus:

(1) Either the first cue does not access the schema, therefore P=(1-a)

(1.i) And either the second cue also does not access the schema, therefore P=(1-a)(1-a)

(1.ii) Or the second cue does access the schema but cannot retrieve the target, therefore P= a(1-r)

(2) Or the second cue accesses the schema, but neither of the two targets can be retrieved, therefore P= $a(1-r)^2$

Experiment 7

The present experiment aims to investigate the extent to which amnesic memory representations resemble normal memory representations with respect to these these two models. It has been claimed by Ross and Bower (1981) that the fragment model fits well to data from experiments in which the target materials are arbitrary and unrelated, rather than data from experiments involving related words. This experiment uses both related and unrelated triads so that the schema model can be applied to the related word responses and the fragment model applied to the unrelated word responses.

It is then possible to compare the parameters of the two models across both groups and to reveal whether the amnesic subjects represent related words and unrelated words in the same way as do normal subjects.

It has already been shown that amnesics have some residual ability to learn related paired-associates and items of highly over-learned information (Cutting, 1978; Hirst, 1982). However there is much controversy as to whether this can be a demonstration of preserved memory ability. Rather, it has been claimed that this is an example of "priming" (Poulos and Wilkinson 1984). This is meant to refer to a subject's ability virtually to free-associate the targets. The present experiment may be able to give a precise description of the nature of the memory structure encoded by an amnesic, and thus address this issue in the following way. The amnesics may not form fragments for unrelated words, thus differing from the normal group, and yet may form schemas for related words, showing an equivalent memory structure to those of normal subjects. Thus the preserved learning of related words in amnesics would therefore be subserved by a preserved ability to form schemas.

Method

Subjects The amnesic subjects had a mean age of 43 years ranging from 23 to 63 years. The amnesic subjects were JA, DF, GG, WP, LP, and RL. The control subjects were 18 members of the University of Warwick of both sexes, aged from 19 to 30.

Materials The stimuli presented were groups of three words or "triads". A total of 42 related triads were generated with common themes of the type described by Ross and Bower (1981). Examples of such triads are "Burglar, Crowbar, Jewellery" and "Rod, Hook, Worm". The 42 unrelated triads were constructed by randomising the related triads, for example, "Thimble, Crowbar, Grandmother" and "Button, Hook, Wolf".

The order of presentation of the triads was held constant across all subjects and conditions. Each subject saw 42 related triads and 42 unrelated triads in a counterbalanced block design. The unrelated triads were prepared by randomising the set of related triads such that it was possible to create two presentation lists. In list one, triads 1 to 42 were related and triads 43 to 84 were unrelated. List two featured 42 unrelated triads first, followed by 42 related triads.

Control subjects 1 to 9 received presentation list one, and control subjects 10 to 18 received the second presentation list. The amnesic subjects were also presented with both lists in equal numbers.

The triads were presented to the control subjects on successive slides using a carousel projector connected to a timer. Each was displayed for four seconds. The triads were shown in blocks of 14 after which there was a retrieval task.

The amnesic subjects were presented with the triads printed in New York 15 point on 3x5 inch index cards. Each card was displayed by the experimenter for 8 seconds. The triads were shown in blocks of seven after which there was a retrieval task. The retrieval task consisted of a response booklet which featured the cues and a space for subjects to write their responses. In the case of the amnesic subjects these were written by the experimenter under their direction.

In this experiment an incremental cueing procedure is used. This means that subjects are cued first with one word from the target triad, to which they may respond with both the remaining members of that triad, with one word only, or perhaps with no word at all. They are then given an additional cue which is another member of the target triad. Subjects then try to recall the third member of the triad. In order that the preparation of the response booklets is more clear, details are given below.

For each triad there are a number of possible cues to retrieval. To illustrate this let the words of the triad equal A, B, and C. The following cueing relationships may then be apparent:

Give A as cue and subject may recall B & C
then Give A & B as cues and subject may recall C
or Give A & C as cues and subject may recall B

Give B as cue and subject may recall C & A
then Give B & C as cues and subject may recall A
or Give B & A as cues and subject may recall C

Give C as cue and subject may recall A & B
then Give C & A as cues and subject may recall B
or Give C & B as cues and subject may recall A

Thus it can be seen that there are six different cue sequences for each triad. All cue sequences were considered equivalent for the purposes of this experiment. To insure that each type of cue appeared equally often in the retrieval task, a Hyper-Graeco-Latin square was used. This was done in the following way. First, two six-figure random numbers containing the digits 1 to 6 were obtained from tables. The letters A to F signified the six different cue types and were written in rows and columns as a Latin square. The rows and columns were then numbered and the first random number used to designate a new order of rows. The second random number was then used to alter the order of columns in the new square. The first row in this final square designates the first six cue types written in the subject's response booklets. As there are 14 trials in each block, the whole process was repeated with two more six-figure random numbers to make a second six-column Hyper-Graeco-Latin square. Written next to the previous square, it can be seen that cues 1 to 12 are then designated by reading the first row of the two squares. Finally, a two-figure random number selected a column from each of the already-created squares, these extra two columns bringing the total number of designated cues per row to 14. The first row therefore instructs the order of cue types for responses 1 to 14, the second row gives the order of cue types for responses 15 to 21, and so on for all 84 trials.

The cues were then hand-written left-justified on A4 paper by the author. The cue consisted of the first item written clearly in block capitals followed by two hand-ruled lines each ending in a question mark. These indicated to the subject that two responses were needed, and that

they should be written on the lines provided. A one centimetre space was allotted between this text and the next line which comprised the same word repeated, plus a second word from the target triad, followed by a ruled line and question mark. A two centimetre space divided this trial from the next. After 14 trials an indication was written to the subject that they must now stop and wait for further instructions from the experimenter.

Two booklets of this type were written. The first, for order one, tested retrieval of related triads 1 to 42, followed by cues for the unrelated triads 43 to 84. The second booklet, for order two, comprised the complementary order. For each booklet the order of the triads varied randomly within the first seven and second seven triads presented in each 14-trial block. Also for all of the response blocks for each subject, the most recently presented seven triads in a 14-trial block were tested second, thus avoiding recency effects.

Each subject was provided with a mask containing a cut-out window of appropriate size allowing subjects to read only one line at a time. This prevented subjects looking forward to the next cue. Retrieval was self-paced and the experimenter allowed up to three minutes per block of 14 trials. The mask was slid down the page to reveal the cues one line at a time. At the end of a page text indicated that the mask should be put under the completed page with the window at the top of the page before the subject could look at the next page of responses. One subject failed to comply with the instructions for reading the response sheets, and for this reason the subject was not included in the analysis.

Amnesic subjects were tested for retrieval in a slightly different way. Their response booklets were the same as those of the control subjects, but they were tested in blocks of seven triads. This speeded the time to read the cues and avoided the situation of amnesics having forgotten the earlier items before they had completed reading the later items. As far as possible the same randomising and counterbalancing measures were implemented for the amnesic retrieval task as were employed for control subjects. The experimenter read the response booklet to the amnesic subjects and wrote their answers as indicated by the subjects. The task was as far as possible self-paced, but the experimenter ensured that the subject did not linger on one question to the detriment of others and that the time taken for responses did not differ greatly from that of control subjects.

Procedure Control subjects were tested in groups. They were told that they were about to participate in a simple memory test. It was explained that they would see 14 slides on which were written groups of three words, and that they were to remember which words appeared together on a slide. The composition of the response booklets was then explained fully, and they were asked not to guess their responses.

The first 14 slides were then shown, after which all subjects attempted to fill the blanks in their response booklets. This was accomplished by sliding a "mask" down the page to reveal the cues. The experiment was self-paced under the supervision of the experimenter. This process was repeated until all 84 slides were seen and all responses attempted.

Amnesic subjects were tested individually. They were given the same instructions as control subjects and reminded of the instructions frequently during the experiment. They were shown seven cards for eight seconds each, and then their memory for those triads was tested. This procedure was repeated until all 84 triads were seen and all responses attempted.

Results

First, we may examine whether there was any overall difference in the effect of amnesia upon schema-based and fragment-based performance by comparing the recall of related and unrelated words respectively. The table below shows the mean frequencies of occurrence of each of the four patterns of recall distinguished earlier (i.e., α, β, γ, and δ). Overall performance was assessed as the percentage of occasions on which either the first or second cue was successful in producing at least some recall. The mean levels of recall for the normal subjects were 90.1% and 61.8% for related and for unrelated words, respectively; the corresponding levels for amnesic subjects were 30.6% and 7.1%. There was significantly higher recall for normal as opposed to amnesic subjects ($F(1,21) = 59.53$, $p < 0.001$), and for related as opposed to unrelated words ($F(1,21) = 26.67$, $p < 0.001$), but no significant interaction between these two factors ($F(1,21) = 0.24$).

Mean Frequencies of Occurrence for Four Patterns of Recall

Words	Group	α	β	γ	δ
Related	Normal	26.06	9.71	2.06	4.18
Related	Amnesic	4.50	7.17	1.17	29.17
Unrelated	Normal	13.41	8.94	3.59	16.06
Unrelated	Amnesic	1.00	1.83	0.33	39.00

Second, we may examine whether an overall consistency in decline with amnesia nevertheless masks significant variation in decline among the mnemonic components identified by the two models. Fragment and schema maximum-likelihood parameter estimates were calculated separately for the data of each subject. They were obtained from the unrelated words data and the related words data, respectively, by writing a FORTRAN program which utilised subroutine NAG EO4JAF (for maximisation subjects to boundary constraints on parameter values).

Fragment and schema parameter values were obtained from the unrelated words data and the related words data, respectively. Separate analyses of variance were carried out on the fragment and the schema parameter values. For the fragment parameters, it was found that the values of **F** and **P** were significantly greater for the normal group than for the amnesic group ($F(1,21) = 27.46$, $p < 0.001$), but did not differ significantly from each other ($F(1,21) = 0.01$). Most importantly, there was no significant interaction ($F(1,21) = 0.17$). For the normal and amnesic groups, the mean values of **F** were 0.319 and 0.020, respectively; the mean values of **P** were 0.298 and 0.052, respectively.

For the schema parameters, the pattern of results was similar to that for the fragment parameters. The values of **a** and **r** were significantly greater for the normal group than for the amnesic group ($F(1,21) = 34.54$, $p < 0.001$), but did not differ significantly from each other

($F(1,21) = 0.07$). Most importantly, there was no significant interaction ($F(1,21) = 0.05$). For the normal and amnesic groups, the mean values of **a** were 0.861 and 0.491, respectively; the mean values of **r** were 0.817 and 0.488, respectively.

Discussion

The results of this experiment provide evidence of a uniform degradation in the structure of recall with amnesia. First, the amnesics exhibited similar decrements in performance for overall recall of related words and unrelated words. Second, for both the schema model and the fragment model the parameters displayed uniform patterns of impairment. In the case of the schema model and related words, the amnesics were poor both at accessing and at retrieving from organising themes as indexed by the **a** and **r** parameters respectively. In the case of the fragment model and unrelated words, the amnesics were impaired at retaining both the full and partial representations of the triads as indexed by the **f** and **p** fragment parameters respectively.

Where does the present pattern of results leave us with respect to the issue of whether any aspect of recall is preserved in amnesia? First, in experiment 3, the analysis of the stochastic relationship between recognition and recall showed that in amnesia the level of recognition given recall was lower than would be expected in the normal population, and it was hypothesised that this may indicate a relative sparing of direct-access recall (Jones, 1978, 1987). This prediction was not confirmed by the data, as there was no relative sparing of fragments in the amnesic group and these are subserved by direct-access recall (Jones, 1978, 1987).

Second, recall of related words was better than that of unrelated words not only for normal participants but also for amnesic subjects. This implies that although the comparatively high absolute levels of performance for schema-based recall may suffer in the general decline of associative memory with amnesia, they still retain their positive differential relative to levels of fragment-based recall. Schema-based recall capitalises on conceptual knowledge which is likely to have been acquired before the onset of the amnesia. This sparing of related material is consistent with a number of studies which showed that amnesics could learn related paired associates, but could not learn unrelated pairs (Cutting, 1978; Warrington and Weiskrantz, 1982; Winocur and

Weiskrantz, 1976). Preserved priming of related word pairs has also been demonstrated with implicit tests of memory or "free-association" instructions by Shimamura and Squire (1984). It was pointed out by Huppert and Piercy (1982) that the relative sparing of memory for related materials in amnesia was a consequence of associations which were acquired pre-traumatically, and thus argued that this type of memory depended on the activation of pre-existing representations. A corollary of this was that there should be no priming of unrelated words in amnesia. There is some evidence that mild amnesics can show priming for unrelated paired associates when tested implicitly (Schacter and Graf, 1986), and no pre-existing representation would be available in this case, but this result was not replicated in more severely impaired amnesics (Schacter and Graf, 1986; Cermak, Bleich and Blackford, 1988). These results suggested that only some aetiologies of amnesia could exhibit priming of new associations. The debate was resolved to some extent by Shimamura and Squire (1989) who demonstrated impaired priming of new associations with Korsakoff patients and patients with anoxia. They concluded that priming of related materials depends on activation of pre-existing representations, and these are unimpaired in amnesia However, priming of unrelated words depends critically on processes of memory which are damaged in amnesia. They argued that the mild amnesics in the Schacter and Graf (1986) study who showed intact priming of new associations are best described as memory-impaired, but not amnesic (Shimamura and Squire, 1989: 725).

These findings provide support for the proposal that amnesics have relatively unimpaired semantic memory but suffer an impairment of episodic memory (Kinsbourne and Wood, 1975, 1982; Cermak, Talbot, Chandler and Woolbarst, 1985). As noted above, the intact semantic memory may include only pre-morbid knowledge and impaired episodic memory may be restricted only to post-morbid knowledge (Cohen and Squire, 1981; Huppert and Piercy, 1982). There is some variation in terminology within the literature on this topic, and others have preferred the dichotomy of intact declarative memory with impaired procedural memory (Cohen, 1984; Cohen and Squire, 1980; Squire, 1982). This distinction is based upon Ryle (1949) who distinguished between "knowing how" and "knowing that". Declarative memory concerns knowledge that may be consciously inspected, such as facts and everyday personal events, whereas procedural memory involves information is not

available for conscious inspection, such as skills and simple classical conditioning (Anderson, 1985; Tulving, 1985). Squire (1986: 22) suggests that the acquisition of new declarative memory depends on the integrity of the medial temporal and diencephalic regions. He also claims that procedural memory is a phylogenetically earlier memory system and does not require the intact operation of these regions.

Finally, and more generally, these results confirm the findings of the earlier experiments in this thesis, namely, that amnesic memory differs quantitatively from normal memory. The implications of these findings are discussed in the concluding chapter which follows.

CHAPTER 6

DISCUSSION: THE STRUCTURE OF RECALL IN AMNESIA

This chapter begins with a brief summary of the findings of this thesis and concludes with an evaluation of the contribution of these experiments to the understanding of amnesia.

The experiments fell into three groups. The first three experiments investigated a hypothesised selective deficit of recall in amnesia. These experiments studied memory for lists of common nouns and compared the memory of amnesics and normal controls who had approximately equivalent recognition scores. No significant evidence of a recall deficit in amnesia was found. The amnesics' recall scores were not significantly different to those of the normal controls of matched recognition scores owing either to longer delays before testing, or shorter presentation times. These findings differ from those of Hirst et al. (1986, 1988). However, a difference emerged between the two groups in the analyses of the stochastic relationship between recall and recognition. This revealed that in amnesic subjects recall is approximately independent of recognition, whereas in control subjects they are positively related.

The second three experiments investigated a hypothesised selective deficit of spatial memory by comparing amnesic and control memory for the locations of objects or words placed on a grid. These experiments also studied the effects of incidental versus intentional encoding of the locations. This was in order to investigate the hypothesis that intentional encoding of locations would improve amnesic spatial memory scores, and the further claim that this would result in a trade-off of recall and recognition of the item's identities. No significant evidence of a selective spatial memory deficit in amnesia was found. Moreover, intentional instructions did not improve amnesic spatial memory scores. There was no significant evidence of a trade-off of item and location memory in the amnesic group. Rather, intentional instructions significantly reduced item memory (that is, recall and recognition) in both amnesics and normal controls, while having no effect on spatial memory. These findings do not replicate those of Hirst and Volpe (1984a, 1984b), Smith and Milner (1981), Kohl (1984) and Kohl and Brandt (1984), although they are consistent with those of Smith (1988). A further analysis was carried out comparing control and amnesic memory for the location of items

scored by lenient criteria. There was no significant difference between the amnesic and control scores for number of items of this type. Furthermore, recall and recognition memory for these items did not differ significantly in the two groups. Thus amnesic memory was again shown to be very similar to that of control memory attenuated by longer delays before testing.

In the final experiment, the structure of the memory representations of normal controls and amnesics was compared. This experiment compared amnesic and control memory for triads of related and unrelated words, which were hypothesised to be represented by memory schemas and by fragments respectively. It was hypothesised that amnesic subjects may be impaired at forming fragments and schemas. This was investigated by comparing the experimental data with hypothetical data generated by the fragment model of memory in the case of the unrelated words, and the schema model of memory in the case of related words, and testing the goodness of fit of these models. Therefore an impairment at forming one or other type of memory representation would be evidenced by a bad fit of that model to the amnesic data. It was found that amnesics, like the normal controls, formed fragments representing the unrelated triads and schemas representing the related triads. Amnesic memory for related words was slightly better than that for unrelated words, though not significantly so. Both the schema and the fragment model parameters displayed uniform patterns of impairment. This would suggest that amnesic and normal subjects form similar memory representations, but that amnesic subjects simply form fewer of these. Thus amnesic memory may be argued to differ from normal memory quantitatively, rather than qualitatively .

Implications of the Present Studies

The main contribution of these experiments is that they re-focus emphasis on some issues in amnesia which have recently received less attention. Experiments 1 to 3 were designed to examine a hypothesised selective deficit of recall in amnesia, but in the event no selective deficits in simple patterns of amnesic and normal memory was demonstrated. One qualitative difference did emerge in a contingency analysis of recall and recognition in experiment 3. This was the finding that amnesic

recall was stochastically independent of recognition (whereas in normal subjects recognition is a positive function of recall). That is, the level of recognition given recall was lower among amnesics than it would be expected to be in the normal population. Mayes and Meudell, (1981) and Weiskrantz, (1978) have also shown that amnesics have a tendency not to recognise recalled words. Let us consider what the implications of such a deficit may be. Jones (1978, 1987) has proposed that there are two mechanisms of recall which are generation-recognition and direct-access. In generation-recognition, recall is entirely dependent upon recognition, but in the second, direct-access mechanism, recall is independent of recognition. It is possible that the observed independence of recognition and recall in amnesia arises because correctly recalled items of amnesic subjects are retrieved using the direct-access mechanism. It was suggested by Jones (1983) that direct-access recall is subserved by memory fragments while generation-recognition recall is subserved by memory schemata. This would suggest that there may be a relative sparing of memory fragments in amnesia. However, this prediction was not confirmed by the data of experiment seven, as a uniform decrement with amnesia in both fragments and schemas was demonstrated.

A second possibility is that the amnesics' failure to recognise recalled items reflects a consequence of the loss of familiarity of memories experienced in amnesia. The loss of reported familiarity of memories is usually examined by asking subjects to give confidence ratings of how certain they are of having correctly recalled an item (Mayes and Meudell, 1981a, b; Meudell and Mayes, 1984). Amnesics have been shown to be very poor at this task. The lack of familiarity of memories makes the judgement of selecting the correct target memory from possible candidate memories very difficult, as all memories seem equally unfamiliar. Therefore, when confronted with the recognition tests in experiments one to three, the amnesics demonstrated an inability to distinguish the previously successfully recalled item from its distractor, as they were both equally unfamiliar.

It can be seen that apart from the findings of the contingency analysis, the results of the first three experiments converge in suggesting that the differences between amnesic and normal patterns of memory behaviour are primarily quantitative rather than qualitative in

nature. These results are consistent with a number of earlier findings which are discussed below.

Demonstrations of Similarity Between Amnesic and Attenuated Normal Memory

Woods and Piercy (1973) attempted to replicate the findings of Warrington and Weiskrantz (1970, 1973) with a group of control subjects. To recapitulate, Warrington and Weiskrantz had found amnesic "yes-no" recognition was significantly worse than that of normal controls, yet they had found no difference in amnesic and control performance on cued recall, and fragment completion. These results were claimed as support for the retrieval deficit hypothesis of amnesia (see earlier section). Woods and Piercy (1973) used a group of normal controls who were tested immediately as a control group and compared their memory with that of a group of normal controls tested after a one week delay, who were the "amnesic" group. They demonstrated all three of the effects found in Warrington and Weiskrantz (1970, 1973) in their study using normal subjects with weakened memory. This showed that the superiority of amnesic cued recall and fragment completion relative to amnesic "yes-no" recognition did not, in fact, represent a specific functional deficit in amnesia, but that these effects were a feature of weak memory in general.

Mayes, Meudell and Neary (1980) showed that amnesics and normal normal controls with memory attenuated by longer retention intervals had similar recognition memory performance for random shapes. This experiment included both unguided learning instructions and "high" and "low" orienting tasks. The "high" orienting task was designed to encourage semantic processing and the "low" orienting task was designed to encourage processing on the basis of physical features of the targets. A second experiment tested memory for faces under both types of learning instructions. Mayes et al. (1980) found that amnesics performed the orienting tasks similarly to the normal controls. The "high" orienting task improved recognition, and the "low" orienting task decreased recognition, relative to the unguided learning condition in both groups. The authors interpreted their results as being inconsistent with those of Butters and Cermak (1975) and the encoding deficit

hypothesis of amnesia. Mayes et al. (1980) argue that both groups benefited equally from the semantic orienting task and thus the amnesics were argued to be employing similar encoding processes to those of the normal controls (see also Meudell and Mayes, 1980). The experiment is reported here because it shows that a deficit which was thought to be associated with amnesia, namely, a deficit in semantic encoding, was found subsequently to be associated with normal attenuated memory.

Meudell and Mayes (1984) investigated further similarities between amnesic and normal memory. They compared the cued recall of amnesic subjects with that of a control group who were tested after a delay and a control group who were subject to brief exposure of to-be-remembered items. It was previously reported that amnesics exhibited a relative superiority of cued recall in comparison with their recognition scores (Warrington and Weiskrantz, 1975). This is termed the amnesic cuing effect. Meudell and Mayes found that the cuing effect could be demonstrated in control subjects with attenuated memory. Furthermore, the loss of familiarity for correctly cued responses demonstrated in amnesic subjects was also present in these normal normal controls. They interpreted their results as opposing those of Warrington and Weiskrantz (1975) and the retrieval deficit hypothesis of amnesia. Meudell and Mayes argued that the cuing effect was not unique to amnesia and could not, therefore, be argued to be indicative of a specific functional deficit of amnesia. This is a further example of a memory deficit postulated to be associated with amnesia being found subsequently to be a feature of normal attenuated memory.

In experiments four to six the hypotheses were: First, that amnesic would show a selective deficit of spatial memory, and second, that intentional instructions would improve amnesic spatial memory for the to-be-remembered items, possibly at the cost of recall and recognition of those items. The experiments showed no difference between the spatial memory of amnesics and normal subjects with attenuated memory owing to longer delays before testing. Furthermore, intentional instructions did not improve amnesic spatial memory, but reduced spatial memory, recognition and recall in both groups. There could be two possible accounts for these results. The first is that a deficit in the processing of contextual information is not, after all, an important factor in amnesia. The second possibility is that all memories lose their contextual attributes as a function of time since encoding, and that in amnesics this is an

accelerated process. Therefore, the present results are demonstrated because the normals have impaired contextual information because they were tested after a 24 hour delay. Thus both groups suffer a deficit in contextual information. At any rate, experiments four to six show that a re-interpretation of automatic memory processing in amnesia is required, and this is discussed in a later section.

Further support for the proposal that normal subjects have deficits in contextual memory was found in Mayes and Meudell (1981a, 1981b). They attempted to integrate their findings of similarity in amnesic and normal attenuated memory. They suggested that normal forgetting may occur as a result of the gradual loss of certain aspects of to-be-remembered information such as contextual features. In consequence, amnesics resemble normal subjects after forgetting because the amnesics did not encode these contextual features in the first place. They suggested that susceptibility to interference in amnesic and normal attenuated memory may be a result of the loss of this contextual information, which would normally render target memories both familiar and distinct from other competing responses.

Mayes, Meudell and Som (1981) also provided evidence pertinent to this claim. They investigated the findings of Winocur and Kinsbourne (1978) who showed that if amnesics learned two word lists in two separate, distinctive environments then a considerable reduction in interference was seen in the amnesic group. The amnesics demonstrated a differential benefit from the distinctive contexts relative to control subjects, and it was argued that this was because, unlike normal controls, amnesics did not process contextual features of to-be-remembered information effectively. When they were encouraged to do so in Winocur and Kinsbourne's experiment their memory was enhanced. Mayes et al. (1981) also demonstrated this sensitivity to a contextual shift in normal subjects tested after a one week delay. They argue that this suggests that inefficient processing of contextual information may not be a specific functional deficit in amnesia, but that it may be a feature of weak memory in general. They mention the possibility that the reasons for this are that amnesics have a problem with retrieving contextual information as claimed by Winocur and Kinsbourne (1978) or that amnesics are impaired at the acquisition of contextual information, which leads secondarily to retrieval problems, as suggested by Winocur and Olds (1978) and Huppert and Piercy (1976).

Meudell and Mayes (1984) proposed that in experiments which show similarities between amnesic and normal attenuated memory, cued recall may be mediated by a type of priming. The following section defines and discusses priming in the context of amnesia research. It begins with a definition of priming and automatic processing and concludes by relating these concepts to the findings of the present experiments.

Priming and Amnesia

Priming is described as "the facilitative effect of an exposure to an item on subsequent processing of that item" (Schacter and Graf, 1987). Schacter and Graf state that priming is shown in implicit tests of memory where the retrieval test makes no explicit reference to any particular experience, as is the case in word completion tasks. They contrast this with explicit tests of memory such as free recall, recognition and cued recall. Meudell and Mayes (1984) argued that cued recall may be mediated by a type of priming. They hypothesised that cued recall involves two processes. The first depends on "recognition-recall" memory or "conscious" memory (Meudell and Mayes 1984: 51) and this is impaired in amnesics. The second aspect of cued recall is like the priming shown by Jacoby and Witherspoon (1982) and this is preserved in amnesic subjects. Meudell and Mayes (1984: 54) claim that when normal subjects are "encouraged to recall quickly and with a minimum of thought" , or "when the cues link poorly with what subjects are trying to retrieve", then the normal subjects will respond on the basis of priming alone. These are the conditions which prevail in experiments where amnesic memory is shown to resemble attenuated normal memory.

One theory of the operation of priming and implicit memory is the activation account (Graf and Mandler, 1984: Mandler, 1980; Morton, 1979; Rozin, 1976). This proposal is described in Schacter (1987; 511) thus:

> ".... priming effects on implicit memory tests are attributable to a temporary activation of pre-existing representations, knowledge structures, or logogens (...) Activation is assumed to occur automatically, independently of the elaborative processing that is necessary to establish new episodic memory traces. An activated representation readily 'pops into mind' on an implicit memory test, but it contains no contextual information about an item's

occurrence as part of a recent episode and therefore does not contribute to explicit memory of the episode."

Thus priming is argued to be subserved by automatic processing, and to occur in the absence of contextual information. It is contrasted with explicit memory performance, which is sometimes also described as "conscious memory" or "recognition-recall" memory. The characterisation of the operation of priming summarised above bore a striking similarity to many features of amnesic memory (see earlier review chapter) and lead researchers to hypothesise that if normal subjects were encouraged to respond in memory tasks on the basis of priming alone, then their performance would resemble that of amnesic subjects.

Supporting evidence for this hypothesis was demonstrated by Mayes, Pickering and Fairbairn (1987). They found that amnesic sensitivity to proactive interference can also by exhibited by control subjects who are given free-association instructions, an implicit memory task. The experiment used the A-B, A-C paradigm, where two lists are presented to the subject. The first list pairs word A with a partner and the second list pairs word A with an alternative partner. Subjects first learn list one, and typically, amnesic subjects find list two learning very difficult and give inappropriate list one responses in the retrieval test. In Mayes et al. (1987) the control subjects were not told that they were performing a memory task, but were asked to rate the strength of associations between the to-be-remembered word pairs. Mayes et al. found that with these implicit memory instructions control subjects suffered list one intrusions during retrieval of list two, and were thus demonstrating proactive interference. This experiment shows that when control subjects are encoding to-be-remembered information implicitly, then their performance closely resembles that of amnesic subjects. Mayes et al. (1987) claim that in this task the control subjects are responding on the basis of priming rather than more explicit recognition-recall, or "conscious" memory.

In summary, it has been argued that amnesic memory resembles that of normal attenuated memory according to the extent to which performance relies on a type of priming; in other words, memory processing which is fast and requires minimal attentional capacity. This proposal finds support in McDowall (1984), who showed amnesic patterns

of recall in normal subjects under conditions of divided attention. McDowall asked subjects to memorize a word list while engaged in an aural letter-monitoring task. He found that the normal controls were not significantly different to the amnesics on measures of total number of recalled words per trial and the number of words recalled over trials. Both groups also benefited equally from the provision of semantic cues. McDowall accounts for these results by arguing that the amnesic pattern of recall performance may be underscored by reduced processing capacity. The next section provides a discussion of automatic processing in amnesia.

Automatic Processing in Amnesia

Demonstrations of similarity between amnesic memory and normal subjects responding on the basis of priming lead researchers to hypothesise that amnesia was caused a deficit in explicit or "conscious" memory while leaving implicit memory or priming intact. According to this approach, amnesics were argued to perform memory tasks on the basis of automatic processing alone.

Support for this suggestion is found in Jacoby (1982). It is known that under certain circumstances amnesics can show almost normal levels of recognition memory (Brooks and Baddeley, 1976; Cohen and Squire, 1980; Hirst and Volpe, 1982, 1986; Huppert and Piercy, 1976; Jacoby and Witherspoon, 1982). Jacoby (1982) proposed that recognition memory could be achieved on the basis of two types of information: perceptual fluency, and re-specification. Jacoby argued that the relative fluency of processing of a previously seen item, in comparison with that of a novel item, allows the subject to attribute this correctly to prior experience of the item. Furthermore, he argued that recognition on the basis of re-specification may only be achieved by the subject recovering a unique specification of the item, and that this demands the retrieval of the context in which that item was last encountered. Jacoby distinguishes two types of processing associated with memory. The first is habitual and automatic and it results in an encoding which is not very distinctive and is not easily discriminable either from prior occurrences of the item or from similar items. This type of processing is fast but inflexible, and may be argued to support implicit memory (Schacter and Graf, 1987). The second type of processing requires attentional resources and results in an

encoding in which context is integrated and thus a more distinctive encoding is produced. This type of processing is more flexible, and is sufficiently well specified to support good retrieval performance, or explicit memory (Schacter and Graf, 1987). Note that according to this characterisation contextual information is *not* mediated by automatic processing, but requires the type of processing which demands attentional resources. Jacoby goes on to report experiments in which normal subjects are encouraged to respond automatically in tests of memory. He trivialised the processing in the task by providing prior experience of the target word. The experiment involved solving a crossword-type puzzle where a word was paired with a related partner whose initial and final letters were shown with a series of blanks for the missing letters, thus: foot - s_ _e. The subject would then insert "shoe". This was followed by a retrieval test in which the first word was given as a cue for the second word. In the condition which is of interest, the solution was provided to the subjects in the first instance, so that in the retrieval test, the subjects could merely respond with the word with which they had been provided. In this condition retention of the second word was significantly poorer than it had been in the previous condition of solving the puzzle. Thus the trivialised processing decreased retention because the subjects had encoded in a relatively automatic fashion which required minimal attentional resources, resulting in a less robust encoding. Jacoby (1982: 105-106) relates his findings to the memory performance of Korsakoff subjects by suggesting that ....

> 'The Korsakoff patient may process information in a more routine, automatic fashion than does the normal subject. This automatic processing does not specify a presented item in terms of its context so the Korsakoff patient is left with a less distinctive encoding than would be produced by a normal subject. This less distinctive encoding does not include sufficient information to distinguish the current presentation of an item from prior presentations of the same item: consequently, the Korsakoff patient has difficulty recalling or recognising items as having occurred in a particular context."

This approach is thus able to account for the findings on contextual memory in amnesia, but interprets these findings differently from the

context memory deficit account of amnesia. For example, in this account, context is not impaired because it is automatically encoded. On the contrary, contextual information is impaired in amnesia because it is mediated by the type of processing which requires attentional resources, and which is impaired in amnesia. Furthermore, in contrast to the context memory deficit account which suggests that amnesics have an automatic processing deficit, automatic processing is described as the habitual type of processing of which amnesics are capable.

This is contrary to the predictions of the context memory deficit hypothesis, which proposes that amnesia results from a deficit in the automatic processing of contextual information. However, experiments reported in this thesis failed to find specific contextual deficits in amnesics. In contrast, it was demonstrated that amnesic memory resembled attenuated normal memory. Meudell and Mayes (1984) have claimed that this situation arises because normal subjects with weakened memory respond on the basis of automatic priming in tests of memory, and it is argued that this is also the case for amnesic subjects. Thus, rather than suffering an automatic processing impairment, amnesics may be argued to have intact automatic processing. This alternative characterisation of amnesic memory processing is more consistent with the findings of the present thesis than is the context memory deficit account.

Priming and Processing in the Present Experiments

Consideration of the above proposals leads to a re-interpretation of the experiments one to six. At the outset, the studies attempted to reveal qualitative differences between amnesic and normal memory. In the event, these were not found, and the experiments showed that amnesic memory resembled attenuated normal memory. This has implications for memory processing in amnesia. First, it is argued that amnesics and normal subjects with attenuated memory respond on the basis of priming in memory tests. Therefore, in experiments 1 to 3 of this thesis similar memory performance was found in both groups because they were performing the memory tasks on the basis of priming rather than explicit, "conscious" memory.

Second, priming is mediated by fast, automatic processing which does not incorporate contextual information into the trace and which

does not result in a robust encoding of to-be-remembered material. This interpretation implies that amnesia results from an impairment of the type of processing which enables elaboration. This is described as "effortful" processing by Hasher and Zacks (1979). This model of memory processing proposed the following general points. Effortful processes are quite slow, require attentional resources and are flexible. Such operations interfere with other cognitive functions which also require capacity. Automatic processes are fast, do not require attentional resources, but are inflexible. They do not interfere with other cognitive functions. Furthermore, they operate without intention and do not benefit from practice. Finally, performance of operations which are carried out by automatic processes should not be improved by the application of conscious effort. In the context of experiments 4 to 6 Hasher and Zacks (1979) would predict that intentional instructions should not improve spatial memory and this was indeed the case. However, their formulation does not provide an account of why recognition, recall, and spatial memory were impaired by intentional instructions. Schulman (1973) also found that intentional instructions reduced recognition and spatial memory. He concluded that forewarning the subjects of the spatial memory test encouraged subjects to use spatial mnemonics. In his experiment there were four possible locations, and they corresponded to compass directions. Thus he suggested that a subjects may encode "shipwreck, north; firecracker, west" and so on, in the experiment and reduce the amount of time spent on each individual word. He also pointed out that the findings are consistent with Eagle and Leiter (1964) who proposed that "intention to learn is crucial to learning only to the extent it generated adequate learning operations". Interestingly, Schulman also describes spatial location information as having been "primed" in his experiment. He uses this word to describe the relatively good memory scores produced under incidental conditions, in the absence of intention to learn. Generally, it would seem that the present results are consistent with those of Schulman, even to the extent of proposing similar processing underlying performance in the task.

Conclusions

The results of the present experiments did not provide support for two major predictions of the context memory deficit hypothesis. Amnesics were not found to suffer from a selective deficit of spatial memory, and there was no evidence of a concomitant automatic encoding deficit. The present results were found to be more consistent with an account of amnesic memory which concentrates on the similarities between amnesic and attenuated normal memory (Mayes and Meudell 1981a, 1981b; 1984). It is proposed that amnesics and normal controls with attenuated memory performed the present experiments on the basis of priming rather than explicit, "conscious" memory. Furthermore, it is argued that memory in amnesic subjects relies on automatic processing alone, and that they suffer an impairment of "effortful" processing.

Appendix A
Aetiologies of Amnesics in Present Study

Details reported here comprise information given by staff on patient's wards or by relatives. Medical notes were not always available, and those which could be consulted did not include details of diagnosis.

JA JA was diagnosed c. 1975, as suffering from Korsakoff Syndrome. He has consumed no alcohol in over 10 years. He lives in St Nicholas' long-term psychiatric hospital, Newcastle-upon-Tyne.

JB JB was diagnosed c. 1980, as suffering from Korsakoff Syndrome. He has consumed no alcohol in over five years. He lived in St Nicholas' long-term psychiatric hospital, Newcastle-upon-Tyne until 1986 when he was moved to a hostel in Newcastle.

HK HK was diagnosed as suffering from Korsakoff Syndrome, c. 1976. He has consumed no alcohol in over 10 years. He lived in St Nicholas' long-term psychiatric hospital, Newcastle-upon-Tyne until 1986 when he was moved to a hostel in Newcastle.

SM SM was diagnosed as suffering from Korsakoff Syndrome, c. 1976, her brother was also a Korsakoff patient. She has consumed no alcohol in over 10 years. She lives in St Mary's long-term psychiatric hospital, Morpeth, Northumberland.

RS RS was diagnosed as suffering from Korsakoff Syndrome, c. 1978. She has consumed no alcohol in over five years. She lives in St Mary's long-term psychiatric hospital, Morpeth, Northumberland.

HS HS was diagnosed as suffering from Korsakoff Syndrome, c. 1976. He has consumed no alcohol in over 10 years. He lived in St Nicholas' long-term psychiatric hospital, Newcastle-upon-Tyne until 1986 when he was moved to a hostel in Newcastle, where he has since recommenced drinking.

ST ST was diagnosed as suffering from Korsakoff Syndrome, c. 1976. He had consumed no alcohol in over 10 years. He lived in St Nicholas' long-term psychiatric hospital, Newcastle-upon-Tyne. He died of cancer in 1986.

KH KH was diagnosed as suffering from Korsakoff Syndrome, c. 1982,. He was given ECT in 1983. He has consumed no alcohol in over five years. He lives in Prestwich long-term psychiatric hospital, Greater Manchester.

DF DF was diagnosed as having suffered an Anterior Communicating Artery Aneurism, c. 1983. This was repaired surgically, and there has been no evidence of associated subsequent strokes. She lives at home with her mother in Manchester.

LP Epileptic since birth, LP contracted encephalitis c. 1976. She lives at home with her family in Liverpool.

WP WP was diagnosed as having suffered an Anterior Communicating Artery Aneurism, c. 1984. This was repaired by surgery, and there has been no evidence of associated further strokes. He lives at home with his family in Liverpool.

RL RL suffered a road traffic accident in December 1978 and sustained near fatal injuries. He lost a section of skull covering the frontal lobes, was incontinent, paralysed, and mute for approximately one year. He was in a coma for approximately 6 weeks. He suffered a fit caused by a "brain leak" in January 1979, information is not available on whether this was of blood or fluid. The leak was repaired by surgery. In April 1979 a metal plate was inserted to protect the entire frontal area of the brain. RL has recovered his speech, movement, reading and writing and his only remaining impairment is in memory. RL has suffered no fits in the last 12 months. He lives at home with his mother in Sutton Coldfield.

GG GG was diagnosed as suffering from Korsakoff's syndrome, c. 1986. He has consumed no alcohol in over two years. He lives with a professional carer in Coventry.

Appendix B

Amnesic subjects' Scores on Neuropsychological Tests

	Date of Birth	WAIS Full Score IQ	Verbal IQ	Performance IQ	Wechsler Memory Quotient
JA	1924	N/a	98	N/a	84
JB	1930	98	98	99	70
KH	1936	N/a	N/a	N/a	84
HK	1940	100	98	103	79
SM	1926	96	98	95	81
WP	1937	98	100	96	70
RS	1936	104	104	109	81
HS	1925	108	110	105	88
ST	1926 d. 1986	98	94	102	77
RL	1945	72	94	82	64
GG	1933	N/a	N/a	N/a	N/a

Amnesic Subjects' Scores for WAIS Subtests
Verbal Scale Items Scaled Scores

	Vocab.	Inform.	Comprehen.	Similarities	Digit Span	Arithmetic
JA	10	8	8	10	5+5	9
JB	9	9	14	7	6+4	5
KH	9	12	N/a	10	5+5	N/a
HK	11	8	9	10	6+6	11
SM	10	9	10	8	8+4	8
WP	10	9	9	6	8+5	7
RS	11	9	12	13	5+4	12
HS	11	11	14	11	6+3	10
ST	10	11	10	12	6+3	11
RL	1	4	5	5	5+2	8
GG	19	N/a	N/a	8	6+5	N/a

Amnesic Subjects' Scores for WAIS Subtests
Performance Scale Items Scaled Scores

	Picture Complet. Symbol	Picture Arrangement	Block Design	Object Assembly	Digit
JA	7	N/a	9	N/a	4
JB	11	6	9	5	5
KH	13	N/a	N/a	N/a	N/a
HK	13	7	9	13	10
SM	10	8	11	8	8
WP	10	14	N/a	7	10
RS	10	11	11	10	6
HS	14	6	9	7	5
ST	12	12	10	10	8
RL	15	6	9	5	7
GG	14	N/a	N/a	N/a	N/a

Notes.

LP (YOB 1966) is not reported here because only some of her scores were available at time of writing. She was diagnosed as amnesic by Dr. Howard Jackson of Park Lane Hospital, Liverpool in 1985, and is currently a member of the Manchester Amnesia Group subject panel. Those scores which are available are the following subtests of the WAIS; arithmetic 11; similarities, 11, digit span, 9; digit symbol, 10; picture completion, 10; picture arrangement, 14; and object assembly, 7.

DF (YOB 1967) also does not have full data; she was referred by her neurologist to Dr. Andrew Mayes and is currently a member of the Manchester Amnesia Group subject panel.

RL has particularly poor verbal scale scores as he has great difficulty in articulating ideas and in conversation. He was of average intelligence pre-traumatically, and he passed Advanced Level Examinations at school. His comprehension abilities are intact as far as can be gathered from his behaviour and capacity in his job. The word-finding difficulties he experiences may be due to the extensive frontal lobe damage he sustained.

GG was diagnosed as a Korsakoff patient by a doctor in Coventry. He was not available to complete all of the assessments, but his history and memory problems were detailed to me by his carer.

Appendix C.

Materials for Experiment 1.

(Target can be seen appearing in both first and second positions on the recognition cards, target items presented here in bold)

List One A (20 cards)

Egg	Pillow	Song	Circle
Cattle	Sun	Whisper	Writer
Affair	Lawn	Evening	Luggage
Cause	Avenue	Teeth	Plane
Field	Reward	Farm	Handle
Port	Sugar	Meadow	Event
Chance	Basket	Amount	Shoulder
Silence	Porridge	Nail	Animal
World	Manner	Plant	Voice
Earth	College	Story	Poet

Mean word frequency = 102.21
Standard Deviation = 100.39
Number of Cases = 40

List One B (20 Cards)

Mercy	Cupboard	Mind	Miner
Passion	Salary	Income	Motion
Key	Butter	Influence	Answer
Shade	Struggle	Husband	Teacher
Factory	Member	Hate	Tower
Nature	Parent	Brother	Heart
Source	Language	Robber	Supper
Speed	Individual	Hall	Pattern
Treacle	Bank	Sale	Minister
Doctor	Timber	Rubber	Page

Mean word frequency = 126.79
Standard Deviation = 140.66
Number of Cases = 40

Appendix D
Raw Data from Experiment 1

Amnesic Subject	Amnesic Recall	Amnesic Recognition	Control Recall	Control Recognition	Control Subject
N = 7					N = 7
ST	3	15	1	18	1
JB	4	18	1	19	2
HS	3	15	3	17	3
JA	5	17	9	16	4
HK	3	16	5	12	5
RS	1	10	1	19	6
SM	11	13	0	18	7

Appendix E
Materials for Experiment 2
Including Word Frequencies[1]

(Target can be seen appearing in both first and second positions on the recognition cards, target items presented here in bold)

List A (30 Cards)

Boat	Minute	Marble	**Arms**
Lion	**Brain**	Aunt	**Celery**
Grandmother	**Banana**	Spinach	**Uncle**
Copper	Room	Velvet	**Car**
Carrot	Play	**Hour**	Scooter
Pear	**Grandfather**	**Mile**	Trombone
Letter	Pea	**Blue**	Mansion
Piano	**Socks**	Shirt	**Chest**
Bus	**Denim**	Purple	**Saw**
Window	Lead	**Wool**	Lime
Guitar	Foot	Hammer	**White**
Fathom	**Drum**	Organ	**Furlong**
Brick	Iron	**Lemon**	Cotton
Trailer	Red	**Brass**	Door
Legs	**Pearl**	Hair	Cow

Mean word frequency = 79.45
Standard Deviation = 110.90
Number of Cases = 56

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[1] Some items were not listed in the word norms, thus their frequencies could not be included in the means. These words were denim, trailer, scooter and trombone from List A; tangerine, screwdriver, oboe and sledge from List B and 'cello, metre, pewter and lettuce from List C.

List B (30 Cards)

Bean	Buffalo	Bronze	Wall
Roof	Mercury	Cottage	Second
Year	Cabin	Strawberry	Father
Brother	Tangerine	Shoes	Picture
Refrigerator	Blouse	Nails	Grey
Pink	Screwdriver	Sledge	Tweed
Nylon	Hall	Stair	Silk
Nephew	Grape	Steel	Taxi
Head	Week	Camel	Potato
Trumpet	League	Chain	Violin
Generation	Eye	Elephant	Ear
Tractor	Aluminium	Flannel	Train
Gloves	Green	Yellow	Trousers
Yard	Oboe	Banjo	Inch
Peach	Niece	Finger	Pig

Mean word frequency = 96.68
Standard Deviation = 238.46
Number of Cases = 56

List C (30 Cards)

Orange	Dress	Toe	**Squirrel**
Plane	**Violet**	**Pewter**	Ceiling
Tomato	**Wolf**	Bull	**Lettuce**
Century	**Van**	Horse	**Day**
Onions	Cousin	**Sister**	Turnip
Ruler	**Coat**	Skirt	**Wrench**
Cherry	**Daughter**	**Heel**	Month
Husband	**Plum**	Linen	**Melon**
Fig	**Satin**	Bicycle	**Canvas**
Lace	**Boat**	Rod	**Triangle**
Cello	**Metre**	Knot	**Flute**
Goat	**Stomach**	Lavender	**Chisel**
Zinc	Cellar	**Decade**	Nose
Corner	Tin	**Clarinet**	**Acre**
Floor	Ore	**Hat**	Black

Mean word frequency = 64.14
Standard Deviation = 152.58
Number of cases = 56

Appendix F

Raw Data from Experiment 2

Amnesic Subject N = 8	Amnesic Recall	Amnesic Recognition	Control Recall	Control Recognition	Control Subject N = 8
ST	3	23	1	24	1
JB	3	28	3	24	2
HS	2	25	1	25	3
JA	6	30	2	19	4
HK	2	20	0	24	5
RS	2	17	1	21	6
SM	3	28	4	29	7
KH	5	25	1	25	8

Appendix G
Materials from Experiment 3

List One A & B.

(For two-choice recognition, one list, A or B, serves as the target items list and the other, A or B, as the distractor items list. Each of the six lists appears equally often as target list and distractor list.)

Lion	Pig
Horse	Donkey
Rat	Cow
Dog	Tiger
Elephant	Cat
Mouse	Deer
White	Green
Brown	Orange
Maroon	Blue
Red	Grey
Black	Pink
Yellow	Purple
Nun	Deacon
Cardinal	Minister
Bishop	Pope
Rabbi	Reverend
Monk	Preacher
Priest	Pastor
Uncle	Brother
Husband	Cousin
Grandmother	Grandfather
Sister	Niece
Nephew	Aunt
Mother	Father
Arms	Mouth
Nose	Head
Eye	Foot
Ear	Legs
Toe	Hand
Stomach	Finger

Bed	Rug
Desk	Stool
Couch	Sofa
Television	Dresser
Bureau	Table
Chair	Lamp
Topaz	Amethyst
Opal	Diamond
Sapphire	Emerald
Ruby	Pearl
Onyx	Jade
Turquoise	Garnet

List Two A & B

Basement	Floor
Chimney	Window
Hall	Roof
Wall	Brick
Room	Door
Ceiling	Stair
Peach	Plum
Pear	Apricot
Grapefruit	Grape
Tangerine	Lemon
Apple	Banana
Cherry	Orange
Cyclone	Wind
Rain	Cloud
Hurricane	Lightning
Sun	Sleet
Storm	Tornado
Hail	Snow
Ocean	Hill
Valley	Volcano
Rock	River
Canyon	Lake

Plain	Sea
Cove	Cliff
Screws	Wrench
Nail	Level
Screwdriver	Hammer
Saw	Chisel
Pliers	Drill
Ruler	Plane
Trumpet	Flute
Trombone	Drum
Tuba	Harp
Guitar	Saxophone
Clarinet	Oboe
Piano	Violin
Sweater	Shirt
Dress	Tie
Skirt	Hat
Shoes	Pants
Slip	Coat
Socks	Blouse

List Three A & B

Professor	Doctor
Dentist	Carpenter
Plumber	Engineer
Businessman	Nurse
Lawyer	Accountant
Salesman	Teacher
Tuna	Shark
Herring	Carp
Cod	Goldfish
Pike	Salmon
Haddock	Whale
Mackerel	Catfish
Bowl	Plate
Fork	Cup

Knife	Sink
Fridge	Spoon
Dish	Pan
Glass	Cooker
Apartment	House
Dormitory	Cottage
Cave	Tent
Mansion	Shack
Igloo	Motel
Tulip	Pansy
Geranium	Iris
Petunia	Daffodil
Daisy	Carnation
Dandelion	Rose
Lily	Orchid
Gnat	Mosquito
Ant	Wasp
Spider	Moth
Butterfly	Beetle
Grasshopper	Fly
Cockroach	Ladybird
Cabbage	Celery
Broccoli	Potato
Spinach	Carrot
Bean	Asparagus
Onion	Lettuce
Turnip	Corn

Appendix H
Raw Data from Experiment 3

Raw data for amnesic performance on immediate testing

N = 9

Amnesic Subject	Amnesic Recall	Amnesic Recognition
DF	5	30
LP	9	41
HS	11	35
JA	13	40
HK	8	33
RS	4	33
SM	15	32
KH	13	35
WP	6	38

Raw data for control performance matched to amnesic performance by manipulating delay to testing

N = 9

Control Subject	Control Recall	Control Recognition
1	18	36
2	20	42
3	13	37
4	5	34
5	13	39
6	16	40
7	19	39
8	18	42
9	7	36

Raw data for control performance matched to amnesic performance by manipulating presentation time
N = 9

Control Subject	Control Recall	Control Recognition
10	10	29
11	9	34
12	11	36
13	14	36
14	7	34
15	16	37
16	13	36
17	9	29
18	13	35

Appendix I

Data from Experiment 3 used in calculations

Subject	N(Rc)	N(Rn)	N(Rc & Rn)
Amnesic Group			
SM	15	32	13
HK	8	33	6
RS	4	33	3
HS	11	35	8
KH	13	35	11
JA	13	40	12
LP	9	41	9
WP	6	38	4
DF	5	30	4
Controls Presentation Time Condition			
C1	14	36	14
C2	7	34	7
C3	11	36	11
C4	16	37	16
C5	10	29	9
C6	9	34	8
C7	13	35	13
C8	9	29	9
C9	13	36	12

Data from Experiment 3 used in calculations (continued)

Subject	N(Rc)	N(Rn)	N(Rc & Rn)
Controls Delay Condition			
C10	13	39	13
C11	16	40	16
C12	5	34	5
C13	13	37	13
C14	20	42	20
C15	19	39	19
C16	18	42	18
C17	18	36	18
C18	7	36	7

Experiment 3 Further Analysis (continued)

Probabilities of Recall and Recognition

	P(Rn)	P(Rc)	P(Rn&Rc)	P(Rn|Rc)
		Amnesics		
SM	0.762	0.357	0.310	0.868
HK	0.786	0.190	0.143	0.753
RS	0.786	0.095	0.071	0.747
HS	0.833	0.262	0.190	0.725
KH	0.833	0.310	0.262	0.845
JA	0.952	0.310	0.286	0.923
LP	0.976	0.214	0.214	1.00
WP	0.905	0.143	0.095	0.664
DF	0.714	0.119	0.095	0.798
		Controls Presentation Condition		
C1	0.857	0.333	0.333	1
C2	0.810	0.167	0.167	1
C3	0.857	0.262	0.262	1
C4	0.881	0.381	0.381	1
C5	0.690	0.238	0.214	0.899
C6	0.810	0.214	0.190	0.888
C7	0.833	0.310	0.310	1
C8	0.690	0.214	0.214	1
C9	0.857	0.310	0.286	0.923

Experiment 3 (continued)

Probabilities of Recall and Recognition

Subject	P(Rn)	P(Rc)	P(Rn&Rc)	P(Rn|Rc)
		Controls Delay Condition		
C10	0.929	0.310	0.310	1
C11	0.952	0.380	0.380	1
C12	0.810	0.119	0.119	1
C13	0.881	0.310	0.310	1
C14	1	0.476	0.476	1
C15	0.929	0.452	0.452	1
C16	1	0.429	0.429	1
C17	0.857	0.428	0.428	1
C18	0.857	0.167	0.167	1

Appendix J

Data from Unsuccessful Trials, Experiment 3

Experiment 3 required that amnesic and control subjects were matched at recognition at a level of about 80%. This meant that several amnesic subjects needed a number of attempts to achieve this performance when they demonstrated less than 80% recognition. After each unsuccessful attempt a subject was given more presentations of the targets to improve their score. The presentations and score in bold are those included in Experiment 3.

Subjects	Number of Presentations	Recall	Recognition
SM	2 presentations	13	30
	2 presentations	3	32
	3 presentations	7	36
	4 presentations	15	32
	4 presentations	**13**	**35**
	5 presentations	21	36
RS	2 presentations	2	25
	4 presentations	2	28
	6 presentations	**4**	**33**
HK	4 presentations	9	23
	6 presentations	**8**	**33**
KH	2 presentations	3	32
	3 presentations	7	36
	4 presentations	**13**	**35**

Appendix K
Materials from Experiment 4

These lists also show distractor items and the target words are underlined.

List 1 A Toys (8 Cards)

Nail	Medal	Fridge	Bottle	Bone
Trousers	Flower	Candle	Cannon	Rabbit
Spoon	Train	Bird cage	Jug	Rubber
Helicopter	Necklace	Crown	Fork	Stamp
Playing card	Saucer	Whistle	Photograph	Bracelet
Trousers				
Iron				
Donkey				

List 1 B Toys (8 Cards)

Cushion	Thread	Toothbrush	Paper clip	Sewing Machine
Windmill	Battery	Cooker	Cup	Arrow
Glass	Cross	Tree	Horse	Coat
Ball of wool	Bed	Plug	Book	Bat
Plane	Biscuit	Glove	Sock	Rake
Zebra	Church	Wardrobe	Doll	Chair
Parcel	Radio	Lion	Motorcycle	Desk
Pig	Duck	Loaf	Torch	Spade

List 2 A Toys (8 Cards)

Slippers	Elephant	Screwdriver	Teapot	Diamond
Sweet	Ball	Screw	Table	Ribbon
Sheep	Kettle	Rocket	Comb	Frying pan
Knife	Banana	Pipe	Letter	Shoes
Toothpaste	Scissors	Safety-pin	Key	Castle
Truck				
Ball				
Hat				

List 2 B Toys (8 Cards)

House	Toy soldier	Hens	Record	Ink pot
Basket	Sword	Cow	Giraffe	Goose
Skipping rope	Vacuum cleaner	Bee	Needle	Soap
Onion	Tie	Lollipop	Lighter	Television
Handkerchief		Kettle	Pencil	Hammer
Matchbox				
Lemon	Funnel	Xmas cracker	Camel	Revolver
Watch	Postbox	Spectacles	Scrubbing Brush	Button
Boat	Penguin	Tiger	Brick	Bucket

* The materials for this experiment and the list of distractors were inadvertently taken from the laboratory by a cleaner.

Appendix L
Raw Data from Experiment 4

Raw data for amnesic spatial memory after immediate testing
N = 6

	Incidental Condition					Intentional Condition			
	Recognition		Spatial Score			Recognition		Spatial Score	
	Trial 1	Trial 2	Trial 1	Trial 2		Trial 1	Trial 2	Trial 1	Trial 2
Name									
JB	7	7	3	1	II	6	6	3	3
HS	8	8	1	2	II	8	6	2	1
JA	8	8	5	1	II	7	8	1	7
HK	7	8	1	1	II	7	6	3	0
RS	8	8	4	0	II	3	1	5	1
SM	8	6	5	3	II	6	8	5	4
ST	8	8	4	5	II	8	7	4	3

Raw data for control spatial memory after 1 hour delay to testing
N = 5

1	7	8	2	2	II	5	3	1	1
2	7	5	0	2	II	8	8	0	1
3	8	8	2	2	II	7	7	0	2
4	8	7	3	4	II	6	8	3	2
5	8	7	3	3	II	5	3	2	2

Appendix M

Materials from Experiment 5

List A Miniature Objects

Sheep	Helicopter
Donkey	Iron
Camera	Hat
Trousers	Fridge
Table	Scissors
Car	Cage
Binoculars	Boots
Whistle	Teapot

List B Miniature Objects

Vacuum Cleaner	Water
Boat	Duck
Gun	Plane
Chair	Motor Bike
Pencil	Sewing Machine
Hens	Cooker
Tree	Television
Bed	Giraffe

* The materials for this experiment and the list of distractor items were inadvertently taken from the laboratory by a cleaner.

Appendix N
Raw Data from Experiment 5

Raw data for amnesic spatial memory after immediate testing
N = 8

	Incidental Condition				Intentional Condition		
	Recall	Recogn	Spatial Mem		Recall	Recogn	Spatial Mem
JB	3	12	4	II	3	11	1
HS	6	14	0	II	6	14	0
JA	4	15	0	II	5	15	1
HK	3	13	1	II	6	10	1
RS	6	13	3	II	3	13	3
SM	5	14	1	II	5	12	2
ST	6	16	8	II	6	14	7
KH	5	13	4	II	3	11	7

Raw data for control spatial memory after 1 hour delay to testing
N = 6

	Incidental Condition				Intentional Condition		
	Recall	Recogn	Spatial Mem		Recall	Recogn	Spatial Mem
1	9	16	3	II	8	16	7
2	11	16	10	II	5	14	7
3	6	16	5	II	3	13	6
4	8	15	6	II	4	16	2
5	2	15	2	II	1	12	1
6	11	15	6	II	10	14	9

Appendix O
Materials from Experiment 6

List One

Pie	Dice
Club	Wool
Inch	Mile
Cruiser	Daisy
Curtain	Tailor
Penny	Shoe
Scooter	Salt
Candle	Gold

List Two

Swimming	Silver
Liner	Eagle
Sword	Gas
Silk	Slipper
Game	Birch
Truck	Coal
Zinc	Carbon
Novel	Cloves

Appendix O
Materials For Experiment 6 (continued)
Pool of Distractor Items*

List One

Violet	Pine	Oak	Submarine	Basketball
Cherry	Tulip	Canary	Postman	Minute
Blacksmith	Aeroplane	Pastry	Chocolate	Soldier
Rose	Boat	Robin	Soup	Book
Fool	Gun	Water	Marbles	Poppy
Crow	Cards	Porridge	Snooker	Destroyer
Knife	Sparrow	Sausage	Tennis	Second
Bus	Hydrogen			

List Two

Soda	Diesel	Denim	Sodium	Bicycle
Football	Spectacles	Car	Yacht	Decade
Steel	Jay	Liner	Petrol	Oxygen
Nitrogen	Garlic	Iron	Blackboard	Hawk
Train	Maple	Slippers	Spruce	Battleship
Day	Magazine	Carnation	Dancer	Socks
Blackbird	Metre	Wood	Arrow	Tin
Copper	Centimetre	Boat	Arrow	Sugar
Year	Century			

* The list of distractors for this experiment was inadvertently taken from the laboratory by a cleaner.

Appendix P

Raw Data from Experiment 6

Raw data for amnesic spatial memory after immediate testing

N = 6

	Incidental Condition				Intentional Condition		
	Recall	Recogn	Spatial Mem		Recall	Recogn	Spatial Mem
JB	2	14	1	II	1	11	2
HS	5	16	0	II	2	11	0
JA	8	15	1	II	4	14	1
HK	5	11	0	II	3	8	0
RS	2	12	0	II	2	8	1
SM	4	10	1	II	7	14	1

Raw data for control spatial memory after 1 hour delay to testing

N = 6

	Incidental Condition				Intentional Condition		
	Recall	Recogn	Spatial Mem		Recall	Recogn	Spatial Mem
1	9	16	2	II	10	16	7
2	8	15	2	II	1	10	2
3	6	16	4	II	2	12	1
4	9	14	10	II	8	13	5
5	4	13	2	II	0	8	0
6	3	13	2	II	1	6	2

Appendix Q
Comparison of Spatial Recall and Lenient Scores Experiment 6

	Amnesic Subjects			
	Incidental Condition		Intentional Condition	
	Spatial Score	Lenient Score	Spatial Score	Lenient Score
HS	0	2.0	0	0
HK	0	2.5	0	2.0
RS	0	1.0	1.0	2.5
JB	1.0	5.5	2.0	3.0
JA	1.0	3.0	1.0	3.5
SM	1.0	3.5	0	2.0
	Control Subjects			
C1	2.0	2.0	0	1.5
C2	10.0	1.5	5.0	2.5
C3	2.0	3.0	2.0	2.0
C4	4.0	3.0	1.0	4.5
C5	2.0	2.5	2.0	1.0
C6	2.0	4.5	7.0	3.5

Recognition and Recall for Items Scored by Lenient Criteria

Amnesic Subjects

	Incidental Condition		Intentional Condition	
	Recall	Recognition	Recall	Recognition
HS	0	4	0	0
HK	1	4	1	2
RS	1	2	1	3
JB	2	9	1	5
JA	3	6	2	6
SM	1	5	2	4

Control Subjects

C1	2	3	0	3
C2	2	2	2	3
C3	1	3	0	0
C4	2	6	1	7
C5	4	4	0	1
C6	5	9	5	7

Appendix R
Raw Data Experiment 7

Amnesic Subjects
Subject Response Types

	A-B & C		A-B or C		A-0, B-C		A-0, B-0	
	α		β		γ		δ	
	Rel.	Unrel.	Rel.	Unrel	Rel.	Unrel	Rel	Unrel.
JA	4	0	8	0	4	0	26	42
DF	0	1	4	0	1	0	27	41
GG	3	2	15	1	0	1	24	39
WP	2	1	3	0	0	0	37	41
LP	17	1	13	10	2	1	10	30
RL	1	0	0	0	0	0	41	42

Control Subjects
Subject Response Types

	A-B & C		A-B or C		A-0, B-C		A-0, B-0	
	α		β		γ		δ	
	Rel.	Unrel.	Rel.	Unrel	Rel.	Unrel	Rel	Unrel.
1	13	7	21	6	1	5	7	24
2	28	23	14	14	0	0	0	5
3	25	27	12	13	1	0	4	2
4	28	28	7	8	2	3	5	18
5	25	22	16	12	0	0	1	8
6	26	14	9	13	2	2	5	13
7	27	22	3	2	2	8	10	10
8	18	5	15	9	0	4	9	24
9	36	30	3	3	11	3	2	6

Summary Table
Subject Response Types (continued)

	A-B & C		A-B or C		A-0, B-C		A-0, B-0	
	α		β		γ		δ	
	Rel.	Unrel.	Rel.	Unrel	Rel.	Unrel	Rel	Unrel.
10	34	32	3	7	5	3	0	0
11	33	7	6	11	2	6	1	18
12	35	4	6	18	0	3	1	17
13	28	2	12	13	0	3	2	24
14	20	0	13	2	3	0	6	40
15	23	6	11	11	1	0	7	25
16	26	12	13	6	0	6	3	17
17	18	1	1	4	15	15	8	22

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