16

The Structure and Mechanisms of Memory

Alan J. Parkin

Memory, of all the powers of the mind, is the most delicate and the most frail.

Ben Johnson

The study of human memory disorder has a long history, making it, perhaps, the oldest topic within neuropsychology. As with most areas of psychology, longevity is no guarantee of theoretical development and, in many ways, amnesia remains as much a challenge now as it did 50 years ago. In this chapter I will discuss the contribution that research into human amnesia has made to our understanding of normal memory and will conclude by considering the direction of future research into human memory disorder.

WHAT IS AMNESIA?

The term amnesia is so general as to be largely meaningless except as an indication that someone has a faulty memory. As we shall see, memory can go wrong in many different ways and this has major implications at both theoretical and clinical levels. The first distinction we must make is between psychogenic disorders (also known as functional disorders) and those whose origin is organic.

Psychogenic disorders are losses of memory which arise following psychological trauma. They can be classified into a number of subtypes. Most common is *dissociative* or *hysterical amnesia* in which there is a partial loss of memory for events surrounding a traumatic event. Other types include *post-traumatic stress disorder* (PTSD), *fugue,* and *multiple personality disorder*. Their existence has proved valuable as a device in fiction but their frequency of occurence in this context betrays a scant knowledge of how memory impairment can arise as a consequence of psychological trauma.

Organic disorders are by far the most important from the point of view of cognitive neuropsychology. They exist in two essential subtypes: (a) *transient,* where memory function is impaired for a limited period, following which normal functioning returns; and (b) *chronic,* where the impairment is permanent.

The most well-known transient organic disorder is *transient global amnesia* (TGA) in which the person experiences a severe loss of memory which can range from a few hours to several days. There is a degree of systematicity in the pattern of recovery (Kapur, Millar, Abbott, & Carter, 1998) and usually only events during the TGA period itself are permanently lost

(Hodges, 1998). TGA has been associated with epilepsy and electroconvulsive therapy (ECT). Because of its duration and systematic availability, post-ECT amnesia is the only transient state which has received extensive investigation at a cognitive level, but there is continuing controversy about the possible long-term effects of ECT (Parkin, 1997c).

The cognitive neuropsychology of memory has focused mostly on patients with chronic disorders of memory. These include the *dementias,* in which there is a gradual loss of memory, *focal retrograde amnesia,* where the primary deficit is a loss of memory for the past, and the *amnesic syndrome,* which is dominated by a severe and permanent anterograde amnesia.[1]

Most of the research discussed in this chapter involves the amnesic syndrome and so it would be useful to review the syndrome's characteristic features at this point (in Parkin, 1997c. pp. 87–88). They are as follows:

1. Unimpaired short-term storage as measured by tasks such as digit span.
2. A severe and permanent anterograde amnesia, with exceptionally poor performance on tests of recall such WMS-R hard-paired associate learning. WMS-R logical memory and visual reproduction scores are very low. Recognition is also poor, with chance performance often observed on tests such as RMT.
3. Semantic memory, and other intellectual functions, as measured by tests such as WAIS-R, are generally intact. Thus, to be classified as amnesic a patient would have an average IQ but scores on the WMS-R indices (especially the delayed memory index) would be well below normal levels.
4. Skill learning, conditioning, perceptual learning, and priming are relatively intact.
5. Retrograde amnesia will inevitably be present but its extent can be extremely variable with some patients having extensive deficits and others lacking memory only for very recent parts of the premorbid period.

THE NEURAL BASES OF MEMORY AND AMNESIA

Figure 16.1 illustrates the various brain structures which, when damaged, can give rise to the amnesic syndrome. These structures occur in two distinct areas of the brain, the *midline diencephalon* and the *medial temporal lobes.*

Much of what we know about the amnesic syndrome has stemmed from investigations of patients with Korsakoff's Syndrome. These patients have suffered damage to a variety of diencephalic structures, most notably the dorso-medial thalamic nucleus, the mamillary bodies, the mamillo-thalamic tract, and certain areas adjacent to the third ventricle. However, there is also a degree of cortical involvement, especially in the frontal cortex, and more recently it has been pointed out that Korsakoff patients may have additional damage in the medial temporal lobe.

Although Korsakoff's Syndrome is the primary cause of amnesia arising through damage to the diencephalon, there are other causes. The thalamic region is prone to vascular disorders which can give rise to amnesia (Parkin, Rees, Hunkin, & Rose, 1994; see Figure 16.2). Diencephalic amnesia can also arise from the presence of tumours (Parkin & Hunkin, 1993b). The floor of the third ventricle is adjacent to diencephalic structures and tumors here can exert local pressure on those structures, causing memory loss. Tumors can also damage the mamillary bodies, as can paranasal penetrating head injuries (Dusoir, Kapur, Byrnes, McKinstry, & Hoare, 1990). A case of paranasal penetrating head injury involving more extensive damage is NA. When he was first scanned it was claimed that he had a lesion restricted to the dorso-medial nucleus of the thalamus. More detailed neuroimaging has since shown much more extensive damage, including complete destruction of the mamillary bodies. Significantly, however, there is no damage to the hippocampal formation (Parkin, 2000b).

[1]Retrograde amnesia refers to an inability to remember things known prior to the precipitating trauma or illness. Anterograde amnesia refers to difficulty in acquiring new information following the trauma or illness.

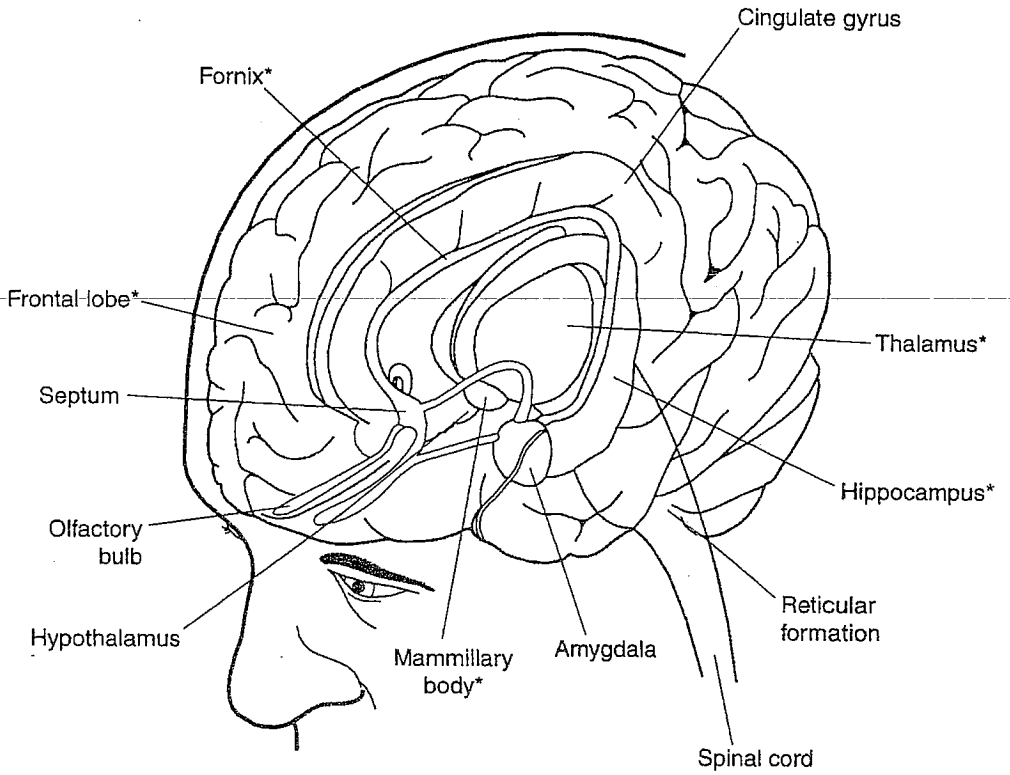

Figure 16.1: Diagram of the limbic system and related structures. Areas indicated with an asterisk are known to be associated with memory function.

Involvement of the temporal lobes in memory was only firmly established in the 1950s, although it had long been suspected from isolated clinical reports. The evidence came from temporal lobectomy in which areas of the temporal lobe responsible for epileptic seizures were selectively removed. The most famous of these patients is HM, whose dense amnesia I have discussed in detail elsewhere (Parkin, 1996b). Notes taken during his operation and others led to the view that the critical structure involved in amnesia was the hippocampus. However, conclusions about HM's underlying pathology must now be qualified by the discovery that he also has significant atrophy of the mamillary bodies, at least in the present day (Corkin, Amaral, Gonzalez, Johnson, & Hyman, 1997).

A patient with selective damage to the CA1 field of the hippocampus resulting from a small ischaemic lesion has been described by Zola-Morgan, Squire, and Amaral (1986). RB exhibits anterograde amnesia with little evidence of retrograde amnesia. In a follow-up study it has been argued that lesions restricted to CA1 essentially produce anterograde amnesia alone, whereas more extensive damage, although still restricted to the hippocampal formation, gives rise to anterograde amnesia and an extensive retrograde amnesia (Rempel-Clower, Zola, Squire, & Amaral, 1996).

Selective ischaemic lesions of the hippocampal formation are rare and attempts to examine amnesia following hippocampal pathology have largely relied on survivors of herpes simplex encephalitis. This is a viral infection of the brain that rapidly causes extensive brain damage centred on the temporal lobes. MRI scans of patients who survive this illness invariably show hippocampal lesions but the damage is always more extensive than this, extending most commonly into the temporal cortex and the prefrontal cortex (especially orbito-frontal). There is

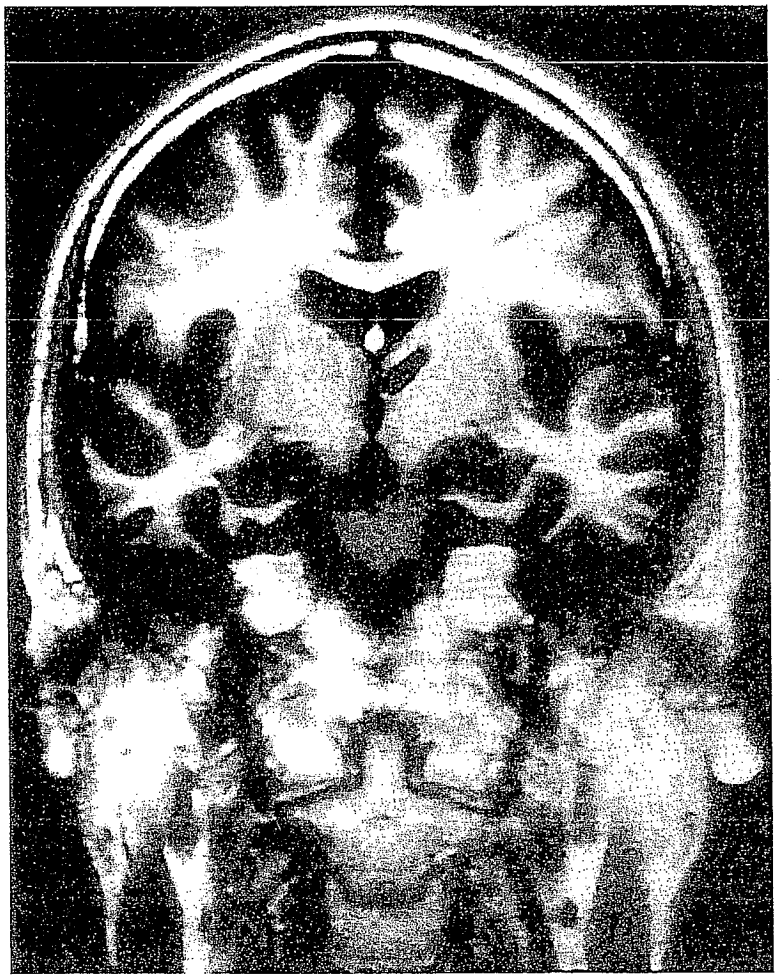

Figure 16.2: MRI scan of patient JR. Note the dark region in the centre. A small lesion in the region of the dorsomedial nucleus of the thalamus.

little indication that herpes simplex encephalitis leads to any significant damage to the diencephalic structures identified with amnesia (Parkin & Leng, 1993).

The question of whether there are two forms of amnesia arising from damage to two different areas of the brain (the midline diencephalon and the medial temporal lobes) is not easily answered because the data are not as "clean" as one would like. Firstly, as we have seen, the major sources of "diencephalic" and "temporal lobe" amnesia stem from aetiologies which produce large lesions extending beyond those structures assumed to be crucial to the memory impairment. Secondly, we may be restricted by the accuracy of the neuropathological information available as, for example, in the case of HM who has now been shown to have additional diencephalic damage. There are, therefore, relatively few cases of the amnesic syndrome in which we can be confident there is no overlapping lesion.

Another important fact to bear in mind when considering the various lesion sites which contribute to amnesia is that they are all part of the *limbic system*. The hippocampus projects to the anterior thalamus both directly, via the fornix, and indirectly, via the mamillary bodies and mamillothalamic tract. There are further projections from the anterior thalamic nucleus to

cingulate and frontal cortex as well as projections back to the hippocampus and adjacent cortical areas via the cingulum. If memory relied on a "circuit" then damage anywhere along that circuit could disrupt memory functioning.

THE STRUCTURE OF MEMORY

The Dichotomy between Short and Long Term Storage (STS and LTS)

William James' seminal distinction between primary and secondary memory (James, 1890) had, by the end of the 1960s, transformed into the dichotomy between *short-* and *long-term store* (STS and LTS) (Atkinson & Shiffrin, 1968). Various lines of evidence from normal subjects were put forward in support of this dichotomy. Further evidence came from patients who had suffered lesions to the hippocampus as a result of temporal lobectomy, the most well-known of these patients being HM. Atkinson & Shiffrin considered this line of evidence provided "perhaps the single most convincing demonstration of a dichotomy in the memory system" (p. 97).

Milner (1966) reported that patients with bilateral surgical lesions in the hippocampal region shows a severe and persstent disorder of memory. Although material known prior to the operation remained intact, patients were unable to acquire further information. Immediate registration of new information seemed to take place normally and material which could be rehearsed verbally was retained for many minutes without loss. However, material which could not be verbalized decayed in a matter of seconds, and interruption of rehearsal led to immediate loss.

The pattern of impairment is the most consistent feature of all patients presenting the amnesic syndrome, regardless of etiology. They invariably show normal range performance on tests of immediate apprehension such as digit span alongside marked impairments of longer-term retention (Parkin & Leng, 1993). Indeed, without the evidence from amnesia the STS/LTS distinction would be rather shaky. Functional double dissociation of the serial position curve in unimpaired subjects, so long considered as incontrovertible evidence for the STS/LTS distinction, is now thought to reflect differences in strategic influences on memory. Otherwise, only a few drug studies provide support for the STS/LTS distinction (Parkin, 2000a).

The Nature of LTS: Distinctions between Episodic, Semantic, and Procedural Memory

By the early 1980s the pattern of deficits in amnesic patients was also forming an important foundation for theories concerning the organization of LTS. Anecdotal observations had, across many years, documented residual learning by amnesic patients in a variety of situations. In a detailed summary of these findings (Parkin, 1982) it was noted that amnesic patients retained skills acquired in the premorbid period and, at a more limited level, acquired new skills in the postmorbid period. There was also evidence of perceptual learning, classical and operant conditioning, alteration of verbal preferences, and even concept formation. Despite their wide variation, one factor links all these instances of residual learning: in no case did patients have any significant recollection of the events that gave rise to this learning. Thus descriptions of residual learning were consistently accompanied by comments such as:

> When shown the [closure] pictures again, one hour later, H.M.'s performance improved by 48%, although he did not remember having taken the test before." (Milner, Corkin, & Teuber, 1968, p. 230)

Observations of this kind quickly became an essential component for theories proposing that LTS was best regarded as a tripartite structure comprising *episodic*, *semantic*, and *procedural*

memory (Tulving, 1985). This proposal drew distinctions between memory which is consciously accessible and that which is not (episodic/semantic versus procedural), and between memory which involves recollecting a specific past event and that which does not (episodic versus semantic). The nature of LTS in amnesic patients had a direct bearing on both distinctions.

Little controversy surrounds the view that data from amnesics supports the distinction between episodic/semantic memory on the one hand and procedural memory on the other. As well as the evidence documented in Parkin (1982) there has been a wealth of new evidence supporting preserved procedural memory in amnesic patients (Parkin & Leng, 1993). Nonetheless it should be pointed out that this evidence does not support the existence of a "procedural memory system"; it supports the idea that there are a variety of learning systems within the cognitive system that operate outside the sphere of cognitive awareness/penetrability. Furthermore, it can be speculated that these procedural abilities show the closest affinity with the learning capabilities of other animals.

In contrast, the idea that amnesia supports the distinction between episodic and semantic memory is a matter of some considerable argument. At a clinical level it is quite easy to be convinced that amnesia results in a selective failure of episodic memory. Thus an amnesic patient will typically converse normally and show relatively unimpaired performance on tests of intelligence and language. In contrast, memory for ongoing events, such as a previously presented word list or the lunch time menu, will be severely disrupted. This relative imbalance in performance was held to support the relative sparing of semantic memory (the system underlying language and general knowledge) from episodic memory (the system needed for recollecting specific events; Tulving, 1985).

Unfortunately this seemingly straightforward conclusion is marred by one major problem—the tests used in the original formulations of this argument were not equally sensitive. Put simply, the tasks used to test preserved semantic memory were easier than those used to test episodic memory. A key line of evidence for the claim that amnesic patients have normal semantic memory is their normal performance on intelligence tests like the WAIS-R. However, it has been pointed out that these tests deal primarily with information already acquired by early adult life. This creates a major problem because retrograde amnesia exhibits a temporal gradient—the earlier that information is acquired in life, the less vulnerable it is to the effects of a brain lesion (see later section). As a result, normal performance on WAIS-R is not a sensitive test of spared semantic memory (Squire, 1987).

The point is illustrated by the case of PZ, a university professor who became amnesic shortly after writing his autobiography (Butters, 1984). As one would expect, his ability to recall episodes portrayed in his autobiography showed a marked temporal gradient, with only those from the 1920s intact. However, when his semantic memory for scientific terms was assessed he showed a similar retrograde amnesia and was only able to define those terms acquired earlier in his career. This parallel loss of event memory and general knowledge has also been demonstrated elsewhere. For example, Verfaellie, Reiss, and Roth (1995) have shown that Korsakoff patients are poor at defining words that came into use during the decades for which they have a dense amnesia for events.

Doubts about the episodic/semantic distinction have led the majority of researchers to use the umbrella term *declarative memory*—referring to any memory that is consciously accessible. This solves some of the problems raised above for the episodic/semantic distinction but there is the odd case which it does not easily handle. RFR became densely amnesic as a result of herpes simplex encephalitis (Warrington & McCarthy, 1988). He could retain little information on standard memory tests and could not even identify close friends and family. It was thus surprising to discover that he had nonetheless learned the meaning of new words and abbreviations (e.g., AIDS) that had come into use during the period for which he was now amnesic. In addition he retained a remarkable ability to describe his friends in a general sense, even though he could not remember any event involving a particular person. There are now other

instances of remote memory breaking up in selective ways. For example, Kapur, Young, Bateman, and Kennedy (1989) described a dense amnesia for public events with intact personal event memory, and the converse was described by O'Connor, Butters, Miliotis, Eslinger, and Cermak (1992).

The episodic/semantic debate has received new impetus from the study of children who have suffered brain lesions that disrupt memory. Vargha-Khadem et al. (1997) described three patients (aged 14–22 yrs) with brain injuries that occurred at birth, age 4, or age 9. MRI revealed bilateral hippocampal pathology in all three cases. Despite their pronounced amnesia for everyday events, all three patients attended normal schools and attained levels of language competence, literacy, and factual knowledge within the low average to average range. On the basis of these findings it was suggested that the episodic and semantic components of memory are partly dissociable, with only the episodic component being fully reliant on the hippocampus. (See also Mishkin, Vargha-Khadem, & Gadian, 1998.)

Ahern, Wood, and McBrien (1998) described a 9-year-old male whose amnesia resulted from congenital brain damage. Memory, attention, vocabulary, and reading skills were measured prior to, and following, a reading intervention. Despite severe impairment for day-to-day events, reading and vocabulary acquisition were shown to be within normal limits at age 9. Along with observational data, these results are interpreted as evidence of a dissociation between semantic and episodic memory systems. These types of finding, which echo a much earlier study (Wood, Brown, & Felton, 1989), have been highlighted by Tulving and Markowitsch (1998). They propose that episodic memory is distinguishable from other forms of conscious memory because only episodic memory relies exclusively on the hippocampus.

Whereas, with a few exceptions, the data from adult studies argue for a single declarative memory system, the child data appear to support the episodic/semantic distinction. My own view is that the child data must be treated carefully. First, it is notable in the study by Vargha-Khadem et al. (1997) that learning achievement was "low average to average," suggesting that it was not entirely normal. In addition, educational achievement may not be a measure of semantic memory alone—grammar learning is widely conceived to be procedural and the development of reading depends a lot on processes which become automatized. These are forms of learning known not to be impaired in amnesia. Moreover, normal development of arithmetic skills must be considered in light of the fact that arithmetical priming occurs in amnesic patients (Delazer, Ewen, & Benke, 1997). Finally there is the simple problem of degree of learning. By definition an episode happens only once, whereas the types of knowledge tapped by attainment tests are the result of many hours of tuition (and in the case of a child known to have a brain injury, probably an above average number of hours).

The Nature of LTS: The Distinction between Implicit and Explicit Memory

During the 1980s human memory research became dominated by a new Zeitgeist, the distinction between implicit and explicit memory (Schacter, 1992b). Unlike the procedural versus declarative (episodic/semantic) approach in which the emphasis is on defining the nature of different stores, this approach attempts to understand LTS by examining how it responds to different forms of memory test, known as explicit and implicit tests. *Implicit memory* tests (also known as indirect tests) can be defined as those in which memory for past experience can be demonstrated without requiring conscious access to the past. In contrast *explicit memory* tests (or direct tests) tests do require conscious recollection of a previous experience. Once again research on human amnesia has figured heavily, and some would say crucially, in the ensuing debate.

Modern experimental evidence for implicit memory has been drawn from a variety of different tasks but here we will concentrate on the fragment completion task (Tulving, Schacter, & Stark, 1982). In this task subjects first study a list of unusual multisyllabic words such as

VENDETTA. After a retention interval, which may be anything from a few minutes to a week, the subjects are presented with what is ostensibly a word puzzle test comprising a series of word fragments from which some letters are missing (e.g., _EN_ _TT_). It is not explained to the subjects that half of the solutions to the fragments are words presented previously, and they are simply asked to complete as many of the fragments as possible. The usual result of this type of experiment is that people produce more completed fragments for words in the previously exposed list than for words not in the list–a phenomenon known as repetition priming.

The interpretation of repetition priming effects has been extremely controversial. Proponents of an implicit memory interpretation have laid great emphasis on the supposed stochastic independence between priming and explicit recollection of solution words. However, others have viewed this statistical argument with more than a little scepticism (Parkin, 1999). Nor has the alternative solution of the test awareness criterion been received with universal enthusiasm. In this procedure priming effects are assessed separately in people who realize, or do not realize, that there is a link between the fragment test and the previous list. Interpretative difficulties arise because the direction of awareness cannot be accurately specified–is the link noted when the subject solves a fragment and recognizes the solution word or does the fragment itself cue explicit recollection of the solution? It is impossible to tell.

The alternative approach is to look for implicit memory effects when we know that explicit memory is impaired or absent. If priming effects are observed under these conditions it cannot be because explicit recollection has helped out. By far the best situation for observing this is to carry out implicit memory experiments on amnesic subjects, where one can assume, a priori, that explicit recollection is essentially inoperative in task performance. Implicit memory has been extensively investigated in amnesic patients and there have been several studies concerned specifically with fragment completion. These have shown intact performance on the fragment completion task despite impairment on parallel explicit tests. Similar results have been obtained with the closely related stem completion task and also the picture completion task. These findings support the argument that implicit test performance can be normal in the absence of explicit memory, and undermine the view that implicit performance may always be contaminated by explicit influences (Jenkins, Russo, & Parkin, 1998; Vaidya, Gabrieli, Verfaellie, Fleischman, & Askari, 1998).

Beyond the basic definition of implicit memory as performance under a set of task demands in which memory is accessed without recollection of specific previous events, there have been recent attempts to specify the *type* of memory underlying priming effects. Schacter (1992a) proposed that certain implicit memory effects are caused by a perceptual representation system which comprises a series of subsystems each dealing with a particular domain of information. Each of these subsystems contains information about the form and structure of a particular stimulus category (e.g., a word or object) but does not store information about meaning; it is therefore termed presemantic.

For reasons that are too detailed to go into here, it can be shown that the mechanisms involved in fragment completion can be considered as presemantic, as in many of the other tasks which demonstrate implicit memory in amnesic subjects. An additional issue is whether implicit memory effects can also be obtained at a semantic level. McAndrews, Glisky, and Schacter (1987) presented amnesic subjects with puzzle sentences such as THE HAYSTACK WAS IMPORTANT BECAUSE THE CLOTH RIPPED and asked them to think of the concept that made sense of the sentence (e.g., "parachute"). This task was then presented again at intervals of up to a week and the solution time was compared with that for similar novel sentences. At all intervals a semantic or conceptual priming effect was obtained, with previously exposed sentences being understood quicker than novel sentences. The study compared severely amnesic subjects with moderately amnesic subjects. Despite far better recognition memory for the sentences in the moderately impaired group, the extent of conceptual priming

for the two patient groups was indistinguishable, demonstrating that the priming effect could not be attributed to some of the amnesics having explicit memory available.

Evidence for more remarkable semantically-based implicit memory comes from the study of KC, a man who became densely amnesic following a closed head injury. He was shown pictures accompanied by a vaguely related sentence (e.g., a picture of a man in a hospital with the sentence "MEDICINE cured HICCUP"). Even after an interval of a year, KC could reliably produce the final word of many of these sentences despite having no explicit recollection of having seen them before (Tulving, Hayman, & MacDonald, 1991).

A particular issue of interest has been whether amnesic patients can show priming for novel associations, as again this would indicate that implicit memory was operating at a semantic level. Graf and Schacter (1985) exposed amnesics to pairs of unrelated words (e.g., WINDOW - REASON) and then gave a stem completion test for the second member of each pair. During the stem completion test the second word was either paired with its original partner (e.g., WINDOW - REA___) or a different one (e.g., OFFICER - REA___). In this way priming could be compared as a function of same or different semantic context. The logic of this experiment is that a higher level of priming in the same context versus different context indicates that new semantic associations have been established between the previously unrelated words in the pairs, and are contributing to the priming effect. Graf and Schacter's finding was ambiguous in that only the less memory-impaired patients showed better priming in the same context condition, suggesting the effect may have been explicitly mediated. Shimamura and Squire (1989) followed up the Graf and Schacter study by presenting amnesic subjects with sentences in which two words were highlighted (e.g., A BELL WAS HANGING OVER THE BABY'S CRADLE). The same manipulation of context was then undertaken but no evidence of enhanced priming in the same context was found. Musen and Squire (1993) obtained inconclusive results with regard to novel association priming in amnesia. Using reading speed as a measure, they compared pre-exposed pairs of unrelated words with re-pairings of the same words. Implicit memory would be demonstrated by faster reading of the unchanged word pairs. Two experiments which used a single exposure trial did not show any evidence of novel association learning but, when multiple exposures were used, evidence for novel association learning was obtained.

Failure to demonstrate implicit memory for associations in amnesia is consistent with recent work reported by Cohen, Poldrack, and Eichenbaum (1997). Control and amnesic subjects viewed a series of 40 scenes twice. A third presentation followed in which half the scenes were presented in their "old" form (i.e., unchanged) and half were manipulated (each scene contained a set of objects, such as a chair and an orange, in which the spatial arrangement was arbitrary so allowing changes in position). Memory was measured in two ways: reaction time to questions about the pictures, and amount of time subjects spent viewing the region of the scenes that had been manipulated. The results are shown in Figure 16.3. Both groups of subjects took longest to answer questions about scenes that were only presented at test. Control subjects also took longer to answer questions about manipulated "old" scenes than unchanged scenes but this did not occur for amnesics. This suggests that the amnesics were not sensitive to the change of relational information in the "old" stimuli. This was confirmed by eye movement patterns (again see Figure 16.3) which showed that controls spent more time viewing the critical region of manipulated scenes. These data show that the implicit memory underlying faster processing of old stimuli is based on individual item representation.

Deficits in amnesics' encoding of relational information is also shown by recent data from Reinitz, Verfaellie, and Milberg (1996). Twelve amnesics and age-matched controls were presented with compound words (e.g., cowboy) and performed either a deep or a shallow encoding task. Later they received an incidental old /new recognition test or perceptual identification test that contained old, recombined, partially new, and completely new words. Controls were better than amnesics at discriminating old from recombined stimuli, but there was no differ-

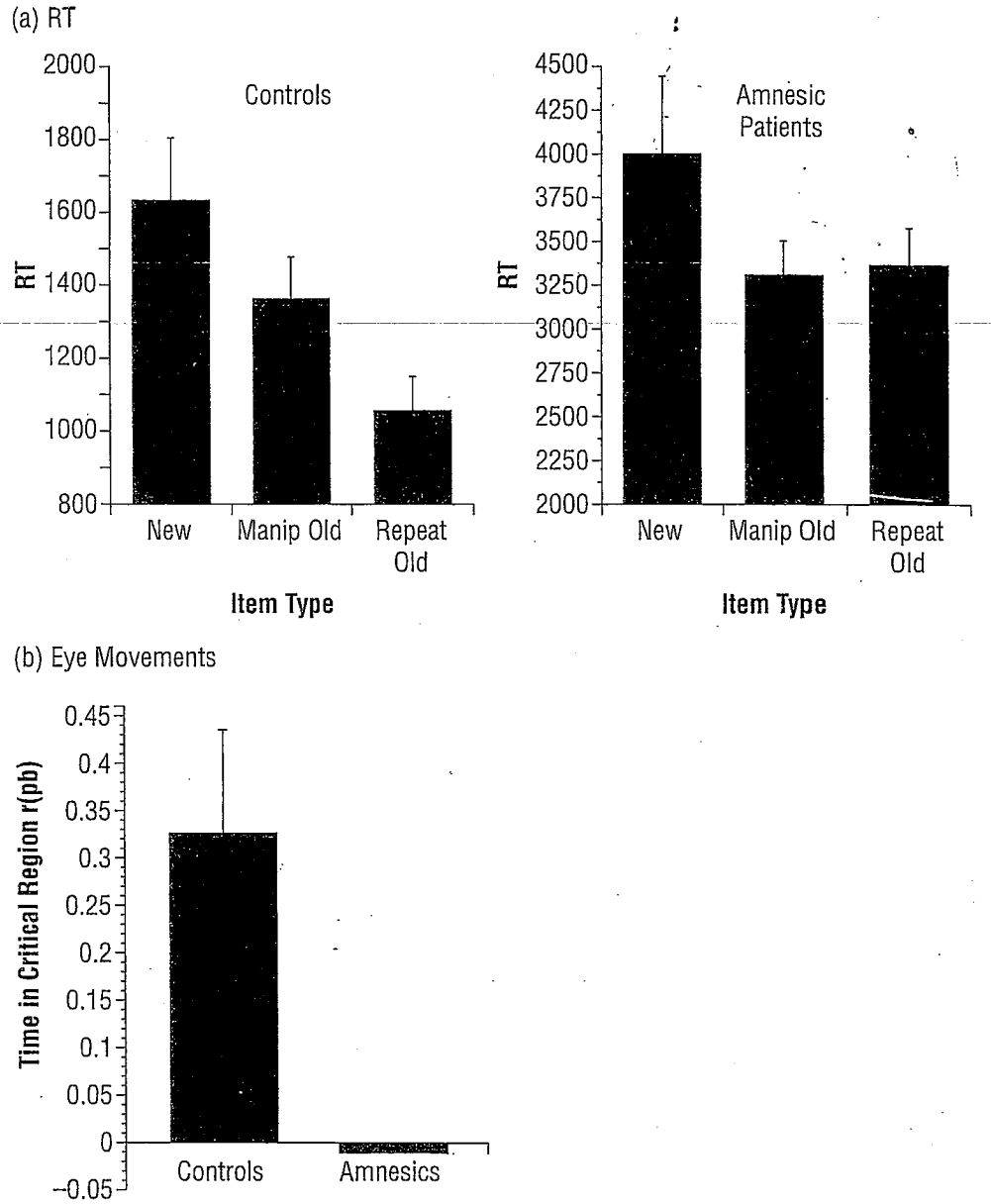

Figure 16.3: (a) Reaction times to answer questions about new scenes, manipulated scenes, and repeated old scenes in the relational manipulation test for controls (left) and amnesic patients (right). Both control subjects and amnesic patients showed a repetition effect (repeated old vs. new scenes), but only the control subjects showed a relational manipulation effect (repeated old vs. manipulated scenes). (b) Point biserial correlations indicating the amount of viewing time during which eye movements were directed to the critical regions of the scenes in which relational manipulations occurred, comparing the manipulated items versus the repeated old items. The positive value on this measure for control subjects indicates that they spent more time in the critical regions for the manipulated items than for the repeated old times; the near-zero value for amnesic patients indicates that they failed to show this relational manipulation effect.

ence between the patients and controls in discriminating old from new stimuli. This indicates that the amnesics experienced specific difficulties learning the inter-relation between the elements of the compound words.

MECHANISMS OF MEMORY

Now we turn to the issue of what has gone wrong in amnesia and what this reveals about the psychological mechanisms of memory.

Within memory research it is still the accepted view that memory involves *encoding, storage,* and *retrieval* stages. Encoding refers to those aspects of the stimulus that are extracted to form the basis of a memory trace for that stimulus. With word stimuli, for example, three encoding dimensions—orthography, phonology, and semantics—are identified. Storage refers to those processes that may lead to the alteration or loss of information while it is held in long-term memory. At a physiological level storage can be linked to the process of *consolidation*—those biological processes that ultimately underlie trace formation. Retrieval refers to those processes that are responsible for bringing information out of storage.

Research into the stages of memory has largely focused on encoding and retrieval rather than storage. The reason for this is principally a pragmatic one. An encoding deficit can be identified if it is shown that a memory disordered patient benefits disproportionately from a manipulation designed to enhance encoding. Similarly retrieval deficits can be inferred if situations are devised which make the same information differentially available. In contrast, storage impairments tend to be identified by default—they are only assumed if neither encoding or retrieval impairments can be demonstrated.

Encoding

The first theoretical account of amnesia in the postcognitive period was put forward by Milner (1966). Based essentially on case HM, it was proposed that amnesia arose from a deficit in consolidation. This theory was motivated solely by observational data—HM had normal STS function but, even with a few seconds distraction, information was lost. It seemed a straightforward and completely intuitive idea that he simply did not form permanent memories. There were complications to this interpretation however. HM was known to be capable of new learning in a number of situations, and this necessitated an explanation which separated motor skill and perceptual skill memory from whatever other form of memory was impaired in HM. The theoretical plot was further complicated by two seminal studies.

Warrington and Weiskrantz (1970) carried out an investigation in which single words were pre-exposed followed, at a later stage, by a fragmented word test. In this test words were presented through a filter which enabled successively larger amounts of information about the target word to be revealed (see Figure 16.4). It was found that amnesic subjects could identify previously exposed words in a more fragmented form than new words.

In retrospect we can see this experiment as a striking demonstration of implicit memory, but this was not the authors' interpretation at the time. They considered their finding to provide evidence against the consolidation theory and argued instead that it showed a retrieval deficit. Essentially they postulated that amnesic patients suffer from massive proactive interference at the time of retrieval—many targets come to mind but none seem correct. Ingeniously they suggested that the word fragments served as surrogate retrieval cues in that only the target word "fitted." Evidence quickly accrued to rule out the retrieval deficit theory however, one argument being that if retrieval was affected one would not see variable retrograde amnesia in the presence of comparable levels of anterograde amnesia. A retrieval deficit should affect anterograde and retrograde memories equally.

In a second seminal experiment, Craik and Lockhart (1972) brought the notion of "encoding

Figure 16.4: Degraded words of the type used by Warrington and Weiskrantz (1970). Adapted from: "Disorders of Memory" in *Clinical Neuropsychology: Behavior and Brain Science* by J. Bradshaw and J. B. Mattingly, copyright © 1995 by Academic Press, reproduced by permission of the publisher.

levels" to the fore with a central finding that "shallow" levels of encoding gave rise to poorer memory than "deeper" levels, so launching the levels of processing movement. They demonstrated this with verbal stimuli, showing that orthographic processing produced far inferior memory to semantic processing. The very poor levels of retention associated with shallow processing quickly promoted the view that amnesia could arise from an encoding deficit. A central piece of evidence concerned the performance of amnesics on the "release from proactive interference" paradigm. Here triads of words from the same category are presented with distracting activity interpolated before recall. As learning progresses, the use of successive triads from the same category leads to a diminution of recall and an increase in intrusions from previous triads. At some point a "release" trial occurs in which a triad from a different category is presented. In normal subjects recall is enhanced at this point and this occurs for both semantic (e.g., taxonomic) and nonsemantic (e.g., alpha-numeric) switches. Butters and Cermak (1980) found that amnesics only showed "release" when nonsemantic categories were used and this supported the view that amnesic patients did not differentiate words on the basis of their semantic features and, thus, suffered from an encoding deficit. However, this effect has been questioned by failures to replicate, suggestions that it is really an effect of super-imposed frontal lesions, and the fact that the interpretation of "release" as an encoding phenomenon may itself be suspect (Gardiner, Craik, & Birtwhistle, 1972; Mayes & Downes, 1997). An alternative attempt to demonstrate an encoding deficit in amnesia involved the introduction of a semantic orienting task at acquisition, with the prediction that amnesics would gain a disproportionate benefit from this. Results from this manipulation also produced equivocal results (Mayes & Downes, 1997).

Recent research into frontal lobe memory disorders has again focused attention on encoding, and the role it plays in judgements of veridicality. Interest in this area has been revived by the discovery of frontal patients whose memory disorder is characterized by a tendency to make high levels of false alarms in the presence of a normal hit rate. Delbecq-Derousne, Beauvois, and Shallice (1990) reported RW who made large numbers of false alarms on forced-choice recognition. This was mirrored by an abnormally high intrusion rate on recall. (For discussion see Dodson & Schacter, this volume.)

Another frontal patient who produces large numbers of false alarms on most tests of recognition memory is JB (Parkin, 1997b; Parkin, Ward, Bindschaedler, Squires, & Powell, 1999). His hit rate is within the normal range (around 80%) but his false alarm rate is extremely high (around 40%). In one experiment he was asked to judge whether he was "sure" or "not sure" about whether he had correctly recognized a stimulus. For both hits and false alarms he gave

predominantly "sure" responses. Like RW he produces large numbers of recall intrusions in amongst correct information (see also Schacter, Curran, Galluccio, Milberg, & Bates, 1996).

Retrieval based and encoding based explanations for JB's deficit have recently been explored (Parkin et al., 1999). Attempts to enhance retrieval by the use of monetary incentive, accuracy instructions, or response restrictions (only allowing JB to make a total response equal to the original number of targets) were found to have no effect. In contrast, the imposition of semantic encoding tasks, including imagery during acquisition, greatly reduced false alarms without affecting hit rates. Other evidence favouring an encoding deficit was that JB exhibited no deficit on yes–no recognition tests tapping premorbid general knowledge and, on a test of autobiographical cueing, the quality of his premorbid memories was normal. A recent study of another frontal patient, MR, has shown similar findings with, if anything, the patient showing greater responsiveness to the encoding manipulation than JB (Ward & Parkin, in press).

Another recent study involving JB explored the nature of his memory traces when he is exhibiting high levels of false alarms. JB has a normal hit rate so he must be storing away sufficient information for target identification; the question is what is this information? A clue is provided by the only experiment in which JB has shown a normal pattern of recognition. This involved a recognition test for abstract designs in which one set of distracters were devised from a completely different set of features (nonoverlapping) and another where, for each target, there was a distracter that differed by only one feature (overlapping) (See Figure 16.5).

JB's performance was massively impaired on the overlapping condition but he made no false alarms on the nonoverlapping condition. This result suggested that JB's memory deficit might arise because he learned items at a "subtarget" level, meaning as collections of features rather than an integrated whole. To test this idea further we constructed an experiment in which the nonoverlapping condition involved a target list constructed using one half of the alphabet, and a recognition test constructed from the other half. Thus, although all items were words, they differed entirely in their letter constituents. This condition was contrasted with an overlapping condition in which targets and distracters were constructed from the whole alphabet. The results showed that both controls and JB performed better on the nonoverlapping condition, even though neither JB nor the controls had conscious knowledge of the manipulation used (indicating that the manipulation is tapping into data-driven aspects of recognition). This effect and its unconscious characteristics have recently been replicated and it has also been shown that older subjects are more susceptible to the effects of overlap, suggesting greater data driven recognition with age (Parkin, Ward, Squires, & Townshend; accepted subject to revision).

Our interpretation of JB's deficit, therefore, is that his high false alarm rates reflect an

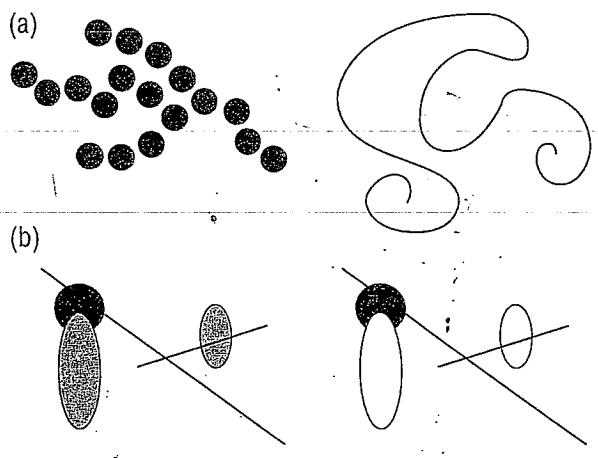

Figure 16.5: Stimuli from (a) non-overlapping and (b) overlapping figures test.

impaired encoding process that operates at the level of features and does not effectively encode the whole stimulus. For words, this "sublexical" representation means that on a standard overlapping test distracters resemble targets because they share common letter elements. Discrimination is eased for JB when there is no overlap. This also explains the interesting finding that JB is prepared to make high levels of recognition responses when there are in fact no words from the target list in the supposed test. Since the lists nonetheless overlap at the sub-lexical level, this effect is entirely predictable (Parkin et al., 1999)

Storage, Consolidation, and Temporal Gradients

Since the early writings of Ribot (1882) it has been known that the extent of retrograde amnesia shows a lawful pattern: the vulnerability of a given memory is inversely related to its time of initial formation—the so-called *Ribot's Law* or *temporal gradient* (see Figure 16.6). Psychologists are not that accustomed to "laws" and when one is found it usually generates a lot of theoretical and empirical interest. This is not the case with Ribot's law, however, and its explanation still largely remains a mystery.

One idea, the continuity hypothesis, came from the frequent association of temporal gradients with Korsakoff's Syndrome. Because of this syndrome's frequent alcoholic aetiology, it was proposed that the gradient actually arose from increasingly poor encoding stemming from the cumulative effects of constant intoxication. However, this theory can be discounted on the grounds that temporally graded retrograde amnesia can be found in amnesic patients without a history of alcoholism, including rare examples of Korsakoff's Syndrome of non-alcoholic aetiology (Parkin, Blunden, Rees, & Hunkin, 1991).

An alternative approach is that temporal gradients stem from *redundancy* inherent in the long-term storage of information. Briefly, it can be argued that every time a memory is retrieved, the record of that retrieval constitutes another memory. As each retrieval will be carried out in a different context, the information associated with a given memory will become richer. If one assumes that, on average, older memories will have been retrieved more, then they will be associated with a higher degree of redundancy (over-representation) and will be able to withstand partial degradation of storage sites (Parkin, Montaldi, Leng, & Hunkin, 1990).

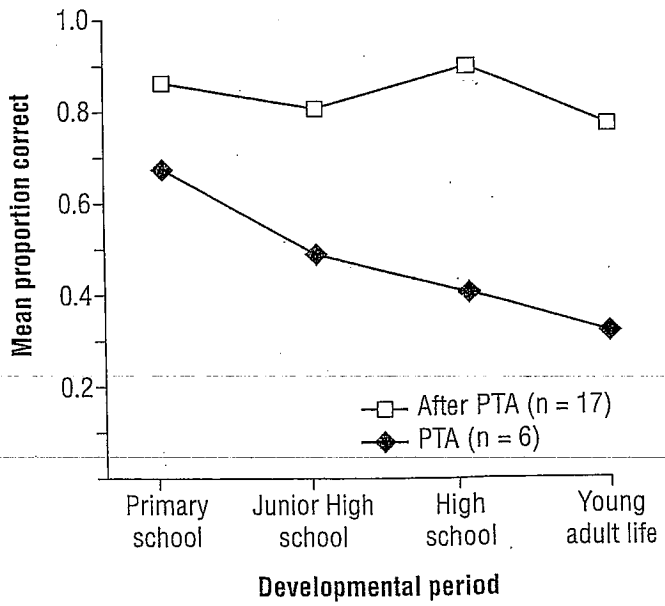

Figure 16.6: Mean proportion of correct recall of autobiographical events plotted across developmental periods for head-injured patients in PTA (n = 6) and after PTA (n = 17). From: Levin, H. S., et al. *Journal of Neurology, Neurosurgery, and Psychiatry*, 48, p. 561, with permission from the BMJ Publishing Group.

A different theory, which one could term the *long-term consolidation hypothesis*, suggests that temporal gradients arise because the process of consolidation is extremely prolonged. In a study referred to earlier, Rempel-Clower et al. (1996) described three patients with lesions restricted to the hippocampal formation. One had a lesion only within CA1 field whereas the other two had CA1 damage plus more extensive lesions into the dentate gyrus and entorhinal cortex. Only the latter two showed extensive retrograde amnesia (in one case 25 years). On the grounds that it was implausible to suggest that the hippocampal formation was a storage site, these authors argued that the hippocampal formation may serve to modulate an extremely long-term consolidation process disruption of which can cause a long-term temporally-graded retrograde amnesia. Although this seems implausible from an adaptive point of view, the theory does help to account for certain puzzling case histories (e.g., Kapur et al., 1997; O'Connor, Sieggreen, Ahern, Schomer, & Mesulam, 1997). In both these instances epilepsy was associated with normal initial retention but with subsequent forgetting over longer time periods—something that might be expected if a long-term consolidation process was interupted.

Retrieval

Kapur (1992) has identified what he has termed *focal retrograde amnesia* (FRA). This refers to a memory impairment in which the primary deficit is a loss of remote memory, with performance on anterograde tests only mildly impaired. These patients are interesting because the selective impairment of retrieval may shed light on the way the process operates normally. A problem with this type of disorder, however, is that some of the patients may be suffering from psychogenic disorders resulting from psychological trauma, or they may be malingering—in some way faking or dissimulating memory impairment. However, once these possibilities have been discounted, there remains a range of cases where focal retrograde amnesia occurs in the context of known brain injury (Kapur, 1992; Kapur, Ellison, Parkin, Hunkin, & Burrows, 1994).

There are essentially two types of patient showing this unusual deficit: those with identifiable brain lesions and those who appear to have developed the disorder from extremely minor injuries (Parkin, 1996a). Examining the former group, two important points emerge: they almost all have lesions to the temporal lobes but with sparing of the hippocampus (see Figure 16.7); and performance on tests of anterograde memory is not normal although it is far better than that found in amnesics.

With these two points in mind it is relatively easy to explain what is going on. As we have seen, the hippocampus is crucial for memory function and the general view is that it in some way moderates the process of consolidation in some way. However, because the hippocampus is small it cannot be the actual storage site of memory. For storage to occur the hippocampus must in some way recruit sites in the temporal cortex. It would appear that FRA patients have an intact consolidation mechanism but lack the normal availability of storage sites because of temporal lobe damage. As a result of damage to the storage areas, remote memory is disrupted and patients do not learn new information normally. It should also be borne in mind that the assessment of anterograde amnesia made in these cases usually involves relatively short retention intervals which maximizes the possibility that patients will perform normally.[2]

A rather different case of FRA is DH (Hunkin, 1997; Hunkin, Parkin, Bradley, Jansari, &

[2] Cases where FRA has been reported following minor brain injury (often not even involving unconsciousness) are more problematic. For example De Renzi, Luchelli, Muggia, and Spinnler (1995) report a man who presented with a dense retrograde memory loss following a road accident, even though there was no evidence of head trauma. In addition he reported loss of procedural memory such as how to shave. This case is rather similar to others that have been reported (see Parkin, 1996a) and there is some disagreement as to how these cases should be interpreted. Some, such as Luchelli, Muggia, and Spinnler (1995), have suggested that these impairments have an organic basis (see Kapur, 1996, for a critique of this study). However, it also the case that these disorders might be of psychogenic origin. Loss of procedural memory, for example, is frequently considered as a sign of psychogenic or malingered amnesia. In connection with this it is notable that most of these cases recover their lost memories.

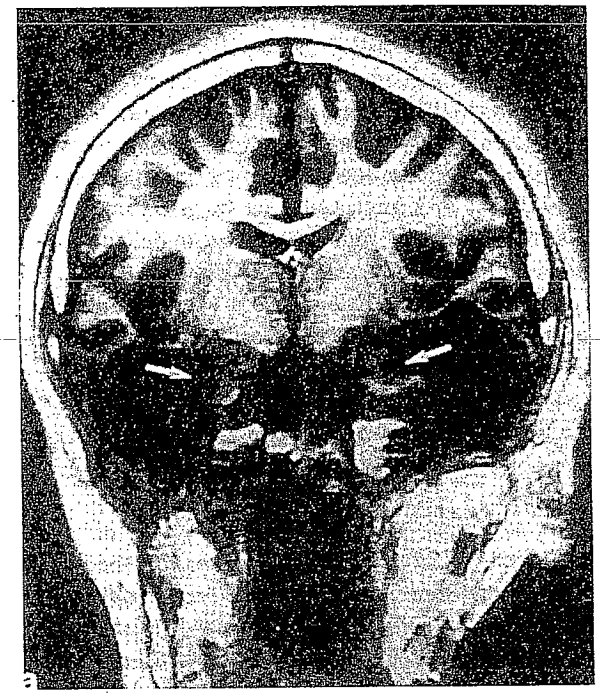

Figure 16.7: Cross-section of patient TJ's brain. Arrows indicate intact hippocampal regions. Large dark areas adjacent to arrows correspond to temporal lobe lesions. Reprinted from *Neuropsychologia*, 32, Kapur, N., Ellison, D., Parkin, A. J., Hunkin, N. M., et al. (1994), Bilateral temporal lobe pathology with sparing of medial lobe temporal lobe structures: lesion profile and pattern of a memory disorder, with permission of Elsevier Science.

Aldrich, 1995), who suffered a closed head injury at the age of 21. Assessment of anterograde memory function indicated near-normal performance. However, autobiographical memory was severely impaired in that he could not recollect a single personal event from the premorbid period. Interestingly he had good memory for public events occurring in the premorbid period. DH's focal retrograde amnesia also had a surprising neuropathology, with two lesions in the occipital lobes plus a small lesion in the right parietal lobe. Notably there were no lesions in the temporal cortex or hippocampal formation. Recent functional neuroimaging has consistently identified a parietal lobe structure known as the *precuneus* as being active in retrieval situations (Fletcher, Frith, Grasby, & Shallice, 1995). Although detailed comparison is not possible, the location of DH's lesion appears to be in the region of the precuneus.

If DH's memory impairment stems from a lesion to the right precuneus, what are the implications of this for a psychological explanation of his deficit? Damasio and Damasio (1993) have suggested that memory retrieval involves a reconstructive process in which various features of entity (e.g., colors, shapes, emotions) are retrieved from different locations in the cortex and combined to give a unified representation. Areas of the brain responsible for this combinatorial function are known as *convergence zones* (CZ's) and these will vary in their scope depending on the type of information being represented. Thus, for an object, a CZ might only be visual. At the highest level of this hierarchy are CZ's which deal with events. Here an event will be characterised as the time-locked co-occurrence of various entities which together constitute a particular event. It is conceivable the precuneus may be involved in this hypothetical combinatorial process regarding events. Accordingly premorbid event retrieval cannot occur because the various entities that need to be retrieved concurrently to reproduce the event are no longer bound together by a CZ. For a related idea concerning the retrosplenial cortex see Gainotti, Almonti, Di Betta, and Silveri (1998).

More recently Incisa Della Rocchetta and Milner (1993) have demonstrated that frontal lesions too can cause problems in retrieval. A well known phenomenon in normal memory is the part list cueing effect. If subjects are asked to learn a word list and, at test, presented with part of the list as a cue, their recall of the other items will be poorer than if no cues are given

at all. This deficit presumably arises because the cue words come to dominate the retrieval process at the expense of the uncued items. The authors found that, compared with controls and other brain damaged subjects, patients with left frontal lesions showed a much greater part-list cueing effect. This suggested that the left frontal lobe was specifically involved in strategic aspects of retrieval.

The exaggerated part list cueing effect shown by patients with left frontal lesions is also relevant to a much more recent debate relating to the Hemispheric Encoding Retrieval Asymmetry (HERA) theory. On the basis of functional neuroimaging of normal adults, this theory proposed that the left frontal cortex was dominant for all encoding operations and the right frontal cortex was involved with retrieval (Tulving, Kapur, Craik, Moscovitch, & Houle, 1994). Subsequent neuroimaging has cast serious doubt on the HERA hypothesis and suggested instead that the nature of materials, rather than the type of memory operation, may determine frontal hemispheric asymmetry (Kelley et. al., 1998; Nolde, Johnson, & D'Esposito, 1998; Nolde, Johnson, & Raye, in press).

Finally, it is worth mentioning that at least some of the memory difficulties associated with what is considered to be a psychogenic disorder—dissociative states—may be related to retrieval difficulties. Dissociative states are commonly reported after witnessing or committing violent crimes, or experiencing other negative life events such as bereavement. Parkin and Stampfer (1995) described the case of "Elizabeth," who had a long history of adverse life events including alcoholic parents, a violent husband, and the death of her baby. She presented with "atypical psychosis" which was characterized by a "memory block" in that she could not recall any of these adverse events. Neuropsychological testing at the time of this impairment produced a marked "frontal profile" (e.g., inability to perform the Wisconsin Card Sort; highly abnormal copying of the Rey Figure). However, over the weeks of the investigation the memory block "lifted" and, in parallel, the frontal signs disappeared. These concomitant changes suggest that the memory block was associated with frontal lobe dysfunction in this case.

The Role of Context in Memory

Context can be defined as information associated with a specific memory that allows differentiation of that memory from other memories. In modern research it is usual to distinguish between two forms of context—intrinsic and extrinsic. *Intrinsic context* refers to features that are an integral part of the stimulus itself. In a face, for example, intrinsic contextual features would be eye color, hair length, size of nose and so on. In the case of words, intrinsic context would relate to the particular meaning extracted from the word at the time of learning. *Extrinsic context* corresponds to those features that are merely incidentally associated with the stimulus itself. These extrinsic features include time of encounter and surroundings (often referred to as temporo-spatial attributes). Studies of amnesia have concentrated on memory for extrinsic context.

The most widely supported account of amnesia is the context deficit theory (CDT) which was first suggested 20 years ago. (The fact that it survives today is witness both to the lack of theoretical development in the field and the growing preoccupation with functional neuroimaging as a means of addressing the cognitive neuroscience of memory.) Before we can examine the theory, however, a more detailed consideration of the term "recognition" is required. Mandler (1980) emphasized that recognition memory comprises two separable components, *familiarity* and *recollection*. Familiarity is the awareness that a stimulus has been encountered before, without requiring recall of when or where. Recollection refers to the additional ability to retrieve the specific context within which a familiar stimulus has been encountered.

CDT has its origins in two studies reported in the late 1970s. Winocur and Kinsbourne (1978) looked at the performance of Korsakoff amnesics on the A $\rightarrow$ B; A $\rightarrow$ C learning paradigm. (This is a procedure where subjects learn two sets of word pairs, but the sets share

the same initial word for each pair, so learning of the first set impedes learning of the second.) They found that the degree of cross-list interference shown by Korsakoff amnesics was disproportionately reduced if learning of the two lists occurred in physically distinct environments. More widely cited, however, is the study of Huppert and Piercy (1978) who investigated the performance of Korsakoff amnesics and normal people on a task involving temporal discrimination. Subjects saw 80 pictures on one day followed by a different set of 80 pictures on the following day. Within each set half the pictures were shown once and the remainder were shown three times. Ten minutes after the presentation on day 2 ended subjects were shown a sample of pictures from days 1 and 2. Subjects were asked to decide whether or not each picture had been seen "today" (i.e., on day 2 as opposed to day 1). As one would expect, both groups placed pictures seen three times on day 2 in the "today" category most often, and pictures shown once on day 1 least often. However, the groups differed in that amnesics were just as likely to categorize as "today" pictures seen three times on day 1 as those seen once on day 2. In contrast, control subjects placed very few of the repeated day 1 pictures in the "today" category and correctly categorized over two thirds of the pictures seen only once on day 2. The performance of amnesics indicates that their decisions about context were based on the overall familiarity of each picture. Thus, pictures presented three times on day 1 seemed as recent to amnesics as those presented once on day 2, both being associated with similar amounts of familiarity. In contrast, the performance of normal subjects seems determined both by familiarity and recollection. The more accurate identification of pictures presented three times on day 2 compared with those presented once suggests that familiarity does play a role in normal subjects' judgements. However, the fact that normal subjects could accurately distinguish pictures presented three times on day 1 from those presented once on day 2 indicates that they were not just responding on the basis of familiarity. Their responses involved an additional search stage in which each picture was linked to its specific temporal context (i.e., "day 1" or "day 2"). The fact that the amnesics could not do this implies they have no record of temporal context.

Recently Jacoby and colleagues (Jacoby, 1991; Jacoby, Toth, & Yonelinas, 1993) have proposed the *process dissociation framework* as an experimental method of measuring the extent to which a subject's recognition response is determined by familiarity or contextual retrieval. In a typical experiment using the process dissociation framework, subjects are presented with two successive lists of words, one spoken and the other written. Two types of recognition test follow. In the exclusion condition, subjects are required to demonstrate contextual recollection by identifying only words from list 2. In the inclusion condition, no contextual demands are imposed and subjects are just told to indicate which words were presented earlier. The assumption of this methodology is that recognition of a list 1 word in the exclusion condition indicates that the subject has forgotten the context of that word's presentation and that the recognition response is based on familiarity. The extent to which familiarity based responding is occurring is based on comparing incorrect recognition of list 1 words in the exclusion condition with the recognition rate for those words in the inclusion condition.

Using the process dissociation framework, Verfaellie and Treadwell (1993) demonstrated that amnesic recognition memory was largely based on estimates of familiarity, suggesting that the amnesic deficit lies primarily in failure to remember context. However, a critique of this study has argued that the Jacoby procedure is difficult to interpret with amnesics because their recognition memory is so defective (Roediger & McDermott, 1994). A subsequent reply (Verfaellie, 1994) addresses these concerns and upholds the original conclusion. However, more recent studies have produced further objections to the logic of the process dissociation procedure (e.g., Russo, Cullis, & Parkin, 1998).

In conclusion, there is good evidence to suppose that the amnesic syndrome can be associated with a failure to make use of context in memory but the issue of whether this is an encoding or storage failure remains to be determined.

It is worth noting here that the role of context in memory may contribute to understanding *post-traumatic stress disorder* (PTSD). The effects of PTSD on memory are somewhat complex. Typically the sufferer is amnesic for the traumatic experiences but, from time to time, flashbacks occur in which the traumatic experience is vividly recreated in the individual's mind. There is now abundant evidence that PTSD victims also have impaired ability to remember new information. Importantly, flashbacks are known to be sensitive to *state dependency* in that they are more likely to occur if the individual's mood state matches that experienced at the time of the traumatic experience. Also, consistent with the state dependency view of PTSD, victims of PTSD show greater recall of trauma related words but poorer recall of neutral and positive affect words (Bremner & Marmar, 1998).

The role played by context in memory and amnesia has also been noted in one of the few well documented case studies of fugue. Fugue is a rare dissociative disorder in which patients lose their identity and may assume a new one. In this case a young man was deeply disturbed by the death of his grandfather and remained in a fugue state for some time, exhibiting only fragments of memory about his own past (Schacter, Wang, Tulving, & Freedman, 1982). However, while watching the funeral scene in the film *Shogun* his memory of himself came back, demonstrating the important role that state dependency may play in the mediation of psychogenic memory dysfunction.

Executive Aspects of Memory

The predominant view of memory impairments which originate from frontal damage is that they reflect an *executive disorder*. This view arises from early ideas about frontal function such as those proposed by Luria (1966) and, more recently, the framework put forward by Norman and Shallice (1986). Norman and Shallice proposed that an individual's responses can be controlled in two fundamentally different ways. The majority of responses are under fairly automatic control and are triggered by environmental cues which engage specific schema, each of which has many subcomponents. To deal with clashes between routine activities the authors proposed a *contention scheduling* process in which the relative importance of different actions is assessed and routine behavior adjusted accordingly. As we all know, however, our behavior is not simply a set of routine automatic operations and there are many occasions when we deliberately and consciously imposed a specific strategy to achieve our goal. To explain these "willed" actions the authors propose an additional *Supervisory Activating System* (SAS) which is activated whenever the routine selection of operations is inappropriate (for a review see Humphreys & Riddoch, this volume).

The "executive theory" of frontal memory impairments views retrieval as a problem-solving act (in the words of Neisser [1967, p. 285], " . . . out of a few stored bone chips, we remember a dinosaur"). The success of retrieval depends on the use of appropriate strategies. This idea has been formalized by suggesting that retrieval has two components—a description stage and a verification stage (Norman & Bobrow, 1979)—an idea which has much in common with the older idea of generation-recognition. According to the theory, "description" involves forming a hypothesis about the contents of memory and "verification" involves establishing "the truth" of memory. (It is notable that this theory gets no further in establishing the information source used to establish truth.)

Evidence to support this distinction comes from ROB, a woman who sustained damage to the caudate nucleus, a basal ganglia structure interrelated with the frontal lobes (Hanley, Davies, Downes, & Mayes, 1994; see also Hanley & Davies, 1997). On a recall and recognition test matched for difficulty (Calev, 1984), she showed very poor performance on recall but normal performance on recognition. Hanley, et al. suggest that this deficit would arise if there had been disruption to the system specifying descriptions. Thus ROB's recall is impaired because targets cannot be specified accurately but recognition is normal because a recognition

test bypasses the need for specifying any description. Hanley et al. go on to suggest that high false alarm/intrusion rates arise from defective verification—a deficit that, more generally, could be used to account for exaggerated part list cueing effects and confabulation.

It is interesting to note that frontal lobe theories of memory have placed a heavy emphasis on retrieval. Milner (1964) proposed the deficits observed were essentially retrieval based, and more recently Schacter et al. (1996) proposed a "focused retrieval deficit" to explain high false alarm rates in their patient BG. Their view was that BG's retrieval description amounted to little more than "a bunch of words" (p. 806), with the result that recognition only operated effectively when there was a strong taxonomic distinction between targets and distracters. One could equally argue, however, that BG's deficit is one of encoding—perhaps he has efficient retrieval processes but lays down very superficial traces.

In the case of JB referred to earlier (Parkin et al., 1999), we have argued that the explanation of his high false alarm rate rests squarely in the domain of encoding efficiency. This is still consistent with an executive explanation because, presumably, the choice of an appropriate strategy depends on intact decision making. In JB's case it is plausible to argue that this deficit in encoding stems from inappropriate allocation of attention. Informal observations of JB indicate that his recollection of events is certainly unusual (Parkin, 1997b). At a more fundamental level it has been shown that JB lacks fundamental inhibitory processes—he does not, for example, exhibit negative priming (Metzler & Parkin, 2000).

There is, however, evidence that deficits in frontally-mediated retrieval strategies do underlie pathological false recognition. Recently it was shown that high false alarm rates by frontal patients in a test involving the identification of famous people could be markedly reduced if a stringent response criterion was introduced—only say "yes" if you can recollect the occupation of the person (Rapcsak, Reminger, Glisky, Kaszniak, & Comer, 1999). Another study showed that MR, a patient with a left frontal demyelinating lesion, produced high levels of false alarms in a task involving recognizing famous faces and names amongst nonfamous distractors (Ward et al., 1999). In a number of instances these false alarms were associated with the false recollection of additional biographical knowledge as well. An interesting feature of these findings was that MR exhibited high false-alarm rates only for people. For instance, in one experiment he was asked to recognize famous names from fiction (e.g., Oliver Twist) from amongst distractors (e.g., Agnes Blyth). Subsequently, using the same list of names, he was asked to recognize which were book titles. When recognizing the names of people he produced false alarms but when recognizing the names of books he did not, even though the stimuli were identical. MR's deficit was attributed to a lax retrieval criterion which allowed the triggering of partial biographical knowledge by unfamiliar faces. (This can be likened to the first-day-on-holiday effect, where one mistakenly thinks that a number of the fellow guests are colleagues from work. This presumably arises because recognition thresholds for frequently encountered people are lower.) In addition Ward argues that MR's propensity to produce false alarms in a person-identification task is also detrimentally influenced by distorted meta-memorial knowledge concerning the potential rate of encountering famous people.

THE FUTURE OF AMNESIA RESEARCH

What should be clear from this chapter is that the study of human amnesia has branched off from the original question: Why do amnesics forget? We have seen the development of many different but interconnected research themes. Most of these themes still have some degree of currency—even the issue of STS/LTS and amnesia has been revived recently (Carlesimo, Marfia, Loasses, & Caltagirone, 1996).

An important development in the future is likely to be the increasing interest in neuropsychiatric disorders of memory from a clinico-legal, as well as a cognitive, point of view. Accounts of "recovered memories" of sexual abuse have provoked wide interest and legal proceedings.

Cognitive neuropsychology has entered the fray with various lines of research connected with false memory and the veridicality aspect of memory function (Schacter, 1999). However, the broader relevance of this research depends on developing paradigms which more closely reflect the circumstances in which false recollections infiltrate real life.

The enterprise of explaining amnesia itself has been rather in the doldrums since the late 1980s. Partly this stems from the gradual tide of support for single case methodology which brings with it the ever increasing fractionation of issues. Theories of amnesia emerged mainly from group studies, particularly of Korsakoff patients, and the single case design seems to have done little for general theories. Thus we have increasingly seen the study of amnesia as a kaleidoscope of disorders with no obvious link to one another (Parkin, 1997a). In addition, with interest in the implicit/explicit distinction on the wane, another major motivation for amnesia studies is decreasing.

Another factor working against amnesia as a front line topic of theoretical research is that so much research effort is being invested in functional neuroimaging. It is curious to think that, at the birth of cognitive neuropsychology, neuroanatomical correlates of cognitive dysfunction were regarded as largely irrelevant—model building was seen as the essential occupation. Such high-minded concerns now seem to have deserted us and functional neuroimaging has offered us a neo-localizationist framework which has been embraced in a largely uncritical fashion. Yet one step back shows us that this large (and extremely expensive) science has done very little to enhance theorizing. It has been concerned principally with identifying neuroanatomical correlates of basic memory functions (many of which we already knew about). Amnesia research has always depended on a healthy theoretical sector and functional neuroimaging has not driven theory to any significant extent. When the Memory Disorders Research Society was formed in the late 1980s, Laird Cermak proposed, in only semi-joking fashion, that there should be a moratorium on MRI scanning. I would only partly agree with him, because good scanning has enabled better differentiation of patients, but I agree with the underlying tone. A little less emphasis on neuroanatomical correlation and more thought about theory would be no bad thing, both for amnesia research and cognitive neuropsychology more generally. The tangibility of neuroimaging data should not lead us to overestimate its explanatory value.

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