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Review Article

Amnesia:
Organic and Psychogenic

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This paper describes the clinical features of selected examples of organic and psychogenic amnesia, and it discusses the nature of the dysfunction that these amnesias entail. The anterograde component of organic amnesia involves a severe impairment in acquiring (or learning) new information, rather than accelerated forgetting, and this may reflect an underlying limbic or neurochemical dysfunction. Retrograde amnesia has a basis which is (at least partially) independent of anterograde amnesia — in some patients, it appears to involve a failure to reconstruct past experience from contextual cues, and this may reflect a superimposed frontal dysfunction. Two types of confabulation are discussed, one of which ('provoked') is a normal response to poor memory, and the other ('spontaneous') appears to reflect incoherent, context-free retrieval, associated with more severe frontal pathology. It is argued that many cases of psychogenic amnesia may resemble organic amnesia, in that they result from an impaired acquisition of information at the time of initial input, perhaps thereby predisposing the subject to subsequent retrieval difficulties.

> The stream of thought flows on; but most of its segments fall into the bottomless abyss of oblivion. . . . Selection is the very keel on which our mental ship is built. And . . . its utility is obvious. If we remembered everything, we should on most occasions be as ill off as if we remembered nothing.
>
> (William James, *The Principles of Psychology*, 1890)

> We oftentimes find a disease quite strips the mind of all its ideas, and the flames of a fever in a few days calcine all those images to dust and confusion, which seemed to be as lasting as if graven in marble.
>
> (John Locke, *Essay concerning Human Understanding*, 1690)

In recent years, the truth of these two maxims has been demonstrated in Luria's brilliant case histories, which have illustrated the crippling consequences of an extraordinarily absorptive and retentive memory (Luria, 1969), as well as the devastating effects of a 'shattered' one (Luria, 1976). Memory plays a central organising and selective role in our mental life. Moreover, its anomalies of function were the starting point for psychoanalysis; its impairment is an early and common sign of organic dysfunction; and its disruption in dementia is one of the most prevalent and distressing of psychological disabilities. For these various reasons, the study of memory and amnesia should be of immense interest to the general psychiatrist.

Regrettably, progress in this topic has sometimes been impeded by the narrow perspectives of particular disciplines, and by their conflicting uses of terminology. Because of this, the present review will begin with a brief outline of the terminology to be employed here; and it will then consider the topic in four sections:

(1) clinical aspects of organic amnesia
(2) the nature of the deficits in organic amnesia
(3) the neuropathology of amnesia
(4) psychogenic amnesia.

The paper will summarise the principal features of selected examples of organic and psychogenic amnesia, and it will discuss the main theories which purport to account for these disorders in terms of the registration, retention, or retrieval of information. In addition, it will draw attention to resemblances between organic and psychogenic amnesia, in the hope that the extensive research on the nature of the former may also shed some light on the latter.

Terminology

Experimental psychologists distinguish between a *primary* (or *short-term*) memory of very limited capacity, which holds information for a few seconds only, and a *secondary* (or *long-term*) memory of much greater capacity and durability. *Secondary memory*, therefore, encompasses all material recalled beyond a period of a few seconds, and items within it may have occurred within the remote or recent past. *Remote* and *recent memory* tend to be very

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loosely defined, but generally refer to the relative *age* of particular memories; whereas *retrograde* and *post-traumatic amnesia* are precisely defined in terms of the *onset* of an injury or illness. *Retrograde amnesia* (RA) refers to memory loss for events occurring before the onset of a lesion, whilst *post-traumatic amnesia* (PTA) refers to memory loss for events following this, and, with respect to head trauma, it ends with the return of the continuous registration of 'personal' memories. *Anterograde amnesia* (AA) refers to an impairment in learning new material; it is commonly used synonymously with PTA, although, in traumatic cases, such an impairment may persist long after the termination of PTA.

There have been various attempts to investigate the subcomponents of primary and secondary memory. *Working memory* refers to a particular model of primary memory, distinguishing its sub-components and emphasising its information-processing activity. Secondary memory has been considered in terms of *episodic* (personal) memory and *semantic* (conceptual) memory, or, alternatively, in terms of *conscious recollection* and memory for skills (*procedural* memory): these will be discussed below. Traditionally, primary and secondary memory were thought of as separate *stores* operating in series: nowadays, greater emphasis is placed upon the *processes* they entail, and some authorities argue that they operate in parallel rather than in series.

The present paper will use the term *psychogenic amnesia* to refer to all instances in which memory loss is presumed to have a psychological, rather than an organic, basis. This term allows for the facts that (i) *both* organic and psychogenic amnesia involve disordered 'function', and that (ii) some *combination* of conscious and unconscious ('hysterical') factors may precipitate a psychologically based amnesia.

The following two cases illustrate the way that some of these concepts will be used in the present paper.

A.B. is a 62-year-old Korsakoff patient whose Wernicke encephalopathy occurred in 1970, after many years of heavy drinking. He performs at a normal or near-normal level at tests of primary memory, such as digit span. He can find his way about the hospital (procedural memory), although he is unable to describe ('recollect') his route and he soon gets lost if he leaves the immediate vicinity. He shows severe impairment at tests of secondary memory which require him to learn new material (AA). He recalls virtually nothing about recent events, either in his personal experience (episodic memory) or requiring conceptual knowledge, such as the name of the present Prime Minister (semantic memory). He also performs very poorly on a remote memory test, which asks about news events from the late 1930s to the early 1980s – although there is relative sparing of memories preceding the mid-1950s. Obviously, both RA (pre-1970) and AA (post-1970) have contributed to this man's remote memory impairment.

By contrast, C.D. is a 24-year-old man who suffered a moderately severe concussion injury in a road traffic accident. He developed an RA of 10–15 minutes and a PTA of 24 hours. During the period of his PTA, his performance at tests of primary memory was impaired, but this recovered soon afterward. When reassessed some weeks after his accident, his recall of recent and remote events appeared completely intact except that his RA and PTA remained unchanged, i.e. there was a discrete gap in his episodic memory. He also showed a mild impairment on formal learning tests (AA).

Clinical aspects of organic amnesia

The amnesic states seen in clinical practice can be classified along two dimensions – organic *vs* psychogenic, and discrete (time-limited) episodes *vs* persistent memory impairment. Table I provides examples of amnesic disorders falling within the four quadrants of this classification. Obviously, there is some overspill between the quadrants: for example, head injury and hypoglycaemia often give rise to discrete amnesic 'gaps', but sometimes they may produce more persistent memory impairment as well. The clinical characteristics of selected examples of organic amnesia will be briefly discussed in the present section.

Lishman (1987) has described in detail the clinical assessment and investigation of organic amnesia. Some clinical tests of memory, e.g. the use of a

TABLE I
Amnesic states

Discrete episode	*Persistent impairment*
Organic	
Toxic confusional state	Drug toxicity
Head injury	Amnesic syndrome, specific
Epilepsy	
Alcoholic 'black-out'	Dementia, global
Hypoglycaemia	
Transient global amnesia	
Post-ECT	
Psychogenic	
Fugue states	Pseudodementia
Situation-specific e.g. for an offence	

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memory and orientation scale and recall of a passage of prose, appear to be more discriminating than others (Kopelman, 1986*a*); and formal neuropsychological assessment is usually essential.

Head injury

Head injury is the classical instance of a discrete, organic amnesia. The familiar pattern of memory loss consists of a brief period of retrograde amnesia (RA), a longer period of post-traumatic amnesia (PTA), and islets of preserved memory within the amnesic gap (Russell & Nathan, 1946). Occasionally, PTA may occur without any RA, although this is more common in cases of penetrating lesions. Sometimes, there is a particularly vivid memory for images or sounds occurring immediately before the injury, on regaining consciousness, or during a 'lucid' interval between the injury and the onset of PTA.

As is well known, the duration of PTA is assumed to reflect the degree of underlying diffuse brain pathology. It is predictive of eventual cognitive outcome (Brooks, 1984), psychiatric outcome (Lishman, 1968), and social outcome (Russell & Smith, 1961), although these relationships are weaker than is often assumed. There appears to be a relationship with age such that older subjects tend to have a longer PTA and more serious deficits at a given PTA (Russell & Smith, 1961), whereas in subjects under 30 PTA is sometimes found to be less effective as a predictor of subsequent memory impairment (Brooks, 1972).

Following a mild head injury, a neurotic ('post-traumatic') syndrome sometimes ensues in which 'forgetfulness' is a prominent complaint. The aetiology of this syndrome remains controversial, but it is clear that the symptoms often persist long after the settlement of any compensation issues. In more severe head injury, the ability to learn new material is the slowest cognitive deficit to recover, and the pattern of residual memory deficit resembles, in many respects, that seen in the Korsakoff or 'amnesic' syndrome (Brooks, 1984; Baddeley *et al*, 1987; Merskey & Woodforde, 1972).

Alcoholic black-out

Goodwin *et al* (1969) described discrete episodes of 'memory loss for significant events' which occurred in 64% of a sample of 100 hospitalised alcoholics. These 'black-outs' were always associated with prolonged alcohol abuse and severe intoxication, and were of two types. In the 'fragmentary' type, the subject became aware of his memory loss only after being told about an event later: there were 'islets' of preserved memory, and the amnesia tended to shrink through time in a manner analogous to the RA of a head injury. In the 'en bloc' variety, the subject usually became aware of the memory loss on awakening from sleep with a sense of 'lost time': the amnesic gap had a definite starting point, islets of preserved memory were rare, and the memories were very seldom recovered. In a minority of cases, the subjects 'came round' while alert and awake, realising that they had no recollection of what they had been doing: sometimes they had travelled, paid cheques, stayed in hotels, in a similar manner to a 'fugue state'. Three subjects reported 'en bloc' black-outs lasting between 2 and 5 days. In addition, 'state-dependent' phenomena were reported by 61% of the patients who had black-outs: commonly, the subjects would hide money when drunk, be unable to find it when sober, and would retrieve it again when intoxicated. The authors cited a 47-year-old woman who wrote letters when drinking, was unable to decipher them when sober, and would resume writing as soon as she had had a few drinks: "It was like picking up the pencil where I had left off."

The diagnosis of an alcoholic black-out can be important in medico-legal cases in which a defendent may claim amnesia for his offence, as will be discussed below.

ECT

This is an iatrogenic form of 'discrete' amnesia, and the adverse effects of ECT on the recall of personal memories were noted from an early stage (e.g. Janis & Astrachan, 1951). In recent years, the pattern of the memory deficit has been carefully mapped out. Verbal memory appears to be particularly sensitive to disruption, and unilateral ECT to the non-dominant hemisphere produces considerably less memory impairment than bilateral ECT (Squire, 1977), making it important to identify the non-dominant hemisphere by a valid procedure (Kopelman, 1982). Squire and his colleagues have found that subjects tested within a few hours of ECT show a retrograde impairment for information from the preceding 1–3 years, a pronounced anterograde deficit on recall and recognition tests, and accelerated forgetting of newly learned information (Squire, 1977, 1981; Squire *et al*, 1984). Although these deficits have been attributed by some to cholinergic depletion, they are more widespread than would be expected on such a basis, and would seem more likely to be attributable to gross metabolic disruption.

Follow-up studies, 6–9 months after ECT, reveal that memory performance on objective tests returns

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to normal, apart from a persistent loss of material acquired within a few hours of the convulsions (Squire, 1977; Freeman *et al*, 1980; Squire & Slater, 1983; Frith *et al* 1983). However, complaints of memory impairment persist (Squire, 1977; Freeman *et al*, 1980; Frith *et al*, 1983), and are still evident after 3 years (Squire & Slater, 1983). Squire & Slater (1983) found that the complaints focus upon the period for which there has been an initial, authentic (retrograde and anterograde) amnesia. Freeman *et al* (1980) reported that the subjects who complain most do, in fact, perform poorer on objective tests than those who do not complain, but Squire & Slater (1983), using similar tests, did not find this. Squire & Slater (1983) and Frith *et al* (1983) both indicate that the 'complainers' are the patients who have recovered least well from their depression.

Korsakoff syndrome

The Korsakoff syndrome is the classical example of a 'persistent' organic amnesia. It is best defined as "an abnormal mental state in which memory and learning are affected out of all proportion to other cognitive functions in an otherwise alert and responsive patient" (Victor *et al*, 1971).

Lawson (1879) and Korsakoff (1889) both described profound amnesia occurring as a consequence of extensive alcohol abuse, but they also recognised that other factors could produce an identical syndrome. Although Korsakoff indicated that recent memories tend to be more severely affected than remote, he emphasised that the retrograde component to the amnesia is very variable and sometimes extends back many years or decades. Subsequent clinical and experimental studies have confirmed the extensiveness of the remote memory impairment, but tend to find that there is indeed a relative sparing of the most distant memories, whether tested in terms of the recognition or recall of famous faces and news events, or the recall of personal memories (Butters & Albert, 1982; Squire *et al*, 1984; Baddeley & Wilson, 1986).

Korsakoff (1889) also drew attention to the patients' disorientation in time and their inability to recall the temporal sequence of events. The disorientation in time can result in an under-estimation of the time spent in hospital (Moll, 1916) and of the patient's own age (Zangwill, 1953), rather similar to that described in some schizophrenic patients. The failure to recall the temporal context of events may contribute to confabulation, which often involves the inappropriate and jumbled recall of genuine events, rather than the fabrication of fictions (Korsakoff, 1889; Victor *et al*, 1971). "Telling of a trip she had made to Finland before her illness . . . [the patient] mixed into her story her recollections of the Crimea, and so it turned out that in Finland people always eat lamb and the inhabitants are Tartars" (Korsakoff, 1889).

It is commonly stated that both Wernicke and Korsakoff failed to recognise the relationship of their two syndromes (e.g. Victor *et al*, 1971). However, Korsakoff (1889) did in fact notice confusion, ataxia, nystagmus, and opthalmoplegia in some of his patients. Describing patients seen in an acute neurological service, Victor *et al* (1971) reported that Wernicke signs preceded the amnesia in 96% of Korsakoff cases; whereas the recorded prevalence in psychiatric series tends to be rather lower. Moll (1916) suggested that the disorder can have either an acute or an insidious onset; and recent studies have confirmed that the initial clinical manifestations of the disorder may range from acute coma (Torvik *et al*, 1982), through the classical acute Wernicke syndrome, to an insidious onset (Cutting, 1978*a*). Moreover, the characteristic neuropathology is identified in many cases at autopsy who have survived in the community without being diagnosed in life (Harper, 1983; Torvik *et al*, 1982). This latter finding provides some support for those who believe that the clinical Korsakoff syndrome may represent an extreme form of the memory impairment commonly found in non-Korsakoff alcoholics (e.g. Butters & Albert, 1982).

Alzheimer-type dementia

Memory impairment is a prominent early symptom in dementing patients, but, unlike the above disorders, it takes place in the context of widespread cortical atrophy and multiple cognitive deficits. Morris and Kopelman (1986) have suggested that the pattern of memory impairment in Alzheimer-type dementia can be viewed as the outcome of 'information-processing' deficits superimposed upon an amnesic syndrome; and there is evidence that this pattern may differ from that seen in normal aging (Huppert & Kopelman, 1987).

There is a profound impairment in learning new information but, surprisingly, the forgetting rate of 'acquired' information is normal in early cases, once adequate learning has been accomplished, a pattern which is consistent with that expected on the basis of cholinergic depletion (Kopelman, 1985*a*). There is also an extensive impairment of remote memory, although, to date, objective tests have failed to establish evidence of any relative sparing of the most distant memories (Wilson *et al*, 1981). Superimposed on this pattern is a substantial impairment at tests of

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primary memory, e.g. digit span (Miller, 1973; Kopelman, 1985*a*); and there may also be impairment in certain aspects of semantic memory, requiring knowledge of facts or concepts (Weingartner *et al*, 1983). Furthermore, the impact of these various memory deficits upon the patients' daily lives is likely to be potentiated by an interaction with the effect of their other cognitive defects. For example, the impairment of primary memory is most severe in younger patients, who have the most advanced neuropathology, and this deficit may exacerbate the effect of any subtle dysphasic difficulty (Kopelman, 1986*b*). Similarly, as discussed below, the combination of amnesia and severe frontal dysfunction may precipitate very florid confabulation in some cases.

Clinical trials of cholinergic 'replacement' therapy to alleviate the memory disorder in dementia have tended to be a little more successful than trials of other pharmacological agents, although all are disappointing. These studies have been reviewed in detail elsewhere (Hollander *et al*, 1986; Kopelman & Lishman, 1986; Kopelman, 1986*c*).

Confabulation

Berlyne (1972) distinguished between 'momentary' confabulation, which is fleeting and has to be provoked, and 'fantastic' confabulation which is sustained, wide-ranging, grandiose, and spontaneous. These might better be termed 'provoked' and 'spontaneous' confabulation as the former is seen only in response to questions probing the subject's memory, whereas the latter is readily evident in his or her everyday conversation.

Provoked confabulation is common in amnesic patients when given memory tests and may represent a normal response to a faulty memory; it may be related to the distortions and intrusions commonly produced by healthy subjects when they remember something poorly (Bartlett, 1932). In a series of 16 Alzheimer and 16 Korsakoff patients described elsewhere (Kopelman, 1985*a*,*b*), the present author obtained instances of provoked confabulation in seven Alzheimer and eight Korsakoff cases, usually in the immediate or delayed (45 minutes) recall of a prose passage. Moreover, when 17 healthy subjects were asked to recall this passage after a 1-week delay, eight subjects showed similar distortions or confabulations, e.g. in recalling a woman whose purse had been stolen in a high street, it was said that she was a thief who was stopped by the police, or that the episode took place near a railway station. (A paper reporting these data in more detail is in preparation.)

Spontaneous confabulation, on the other hand, is a pathological phenomenon, which may result from the superimposition of frontal lobe pathology on an organic amnesia. This suggestion was first made by Luria in 1976, and, subsequently, a number of other researchers have identified an association between confabulation and the presence of either frontal lobe pathology or evidence of frontal dysfunction (Stuss *et al*, 1978; Kapur & Coughlan, 1980; Baddeley & Wilson, 1986). According to Luria (1976), this kind of confabulation may sometimes involve the inappropriate recall of real memories, jumbled in temporal sequence (cf. Korsakoff, 1889; Victor *et al*, 1971), whereas Berlyne (1972) emphasised the fabrication of fictions: it seems likely that the latter can be grafted on to the former. Several authors have noted that the confabulations can be preoccupying, bizarre, and held with firm conviction, in which case they may be distinguishable from delusional memories only in that they occur in the context of an organic amnesia rather than a psychosis. Possibly because of the association with fairly severe frontal lobe pathology, spontaneous confabulation appears to be much commoner in the more advanced stages of Alzheimer's disease than in the chronic phase of Korsakoff's syndrome. In Berlyne's (1972) series, it occurred in eight out of 62 dementing patients, but in only one Korsakoff patient who was described as having had a frontal lobe syndrome following a head injury. In the present author's series, it was seen in only two cases, both of whom were Alzheimer patients.

Kapur and Coughlan (1980) described a 48-year-old building contractor who had an (anterior communicating) aneurysm clipped following a subarachnoid haemorrhage. CT scan showed a well-defined area of left frontal hypodensity. An initial right-sided weakness recovered rapidly, but he was left with a pronounced personality change including irritability, apathy, and sexual disinhibition. Performance at delayed recall and 'frontal' tests was poor, but he did surprisingly well at recognition tests. Confabulation was prominent and spontaneous: "He would claim . . . in the morning to have fictitious business appointments when in fact he was attending a day centre, would frequently dress for dinner in the evening in the mistaken belief that guests were coming . . . When questioned about holidays or outings over the past few days, he would again report events that bore no relation to actual happenings. When the correct version of events was brought to his attention, he would either seem puzzled . . . or place the confabulated event in another temporal context".

Nature of the deficits in organic amnesia

Neuropsychologists have devoted considerable attention to trying to identify the nature of the

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memory deficits in organic amnesia. With regard to anterograde amnesia, many theories have been propounded but they can be broadly classified under four main headings, corresponding to the stage or aspect of memory processes considered impaired. Some theories postulate deficits in the 'acquisition' processes occurring during and shortly after the initial 'registration' of information, whereas others propose impairments in subsequent 'retention' or 'retrieval'.

Faulty encoding

These theories propose that there is a deficit in the psychological processes involved in the initial 'registration' of information. In particular, it has been suggested that, whilst amnesic patients are able to 'encode' the direct, sensory properties of information, they have difficulty in 'processing' its more meaningful (semantic) qualities. For example, Korsakoff patients perform particularly badly at learning word pairs (e.g. hungry–thin) whose recall is normally facilitated by thinking of semantic links between the words (Cutting, 1978*b*). On the other hand, this does not seem to be true of all cases (Squire, 1982), and giving instructions or orienting tasks which encourage the extraction of meaning from a stimulus produces, at most, a relatively small enhancement in the amnesic patients' subsequent recall of that stimulus (Meudell *et al*, 1979; McDowall, 1981). It seems unlikely that a failure to encode semantic information is the fundamental deficit in amnesia, although it remains plausible that there exists some impairment in initial 'encoding' which is at present poorly defined and measured.

Faulty consolidation

A second type of hypothesis proposes that there is impairment in the physiological processes which are assumed to occur shortly after initial registration and to establish ('consolidate') information in memory into some relatively permanent form (e.g. Meudell *et al*, 1979; Moscovitch, 1982). These processes were traditionally thought to involve the 'transfer' of information from primary to secondary memory, operating during a time-period of something less than a minute. The hypothesis was suggested by the finding that primary memory (holding information for a few seconds) is relatively intact, in the presence of profound amnesia, in patients with hippocampal lesions, head injury, and in some studies of Korsakoff patients (e.g. Milner, 1966; Brooks, 1984; Kopelman, 1985*b*). In one particularly intriguing study, Lynch and Yarnell (1973) interviewed six concussed American footballers within 30 seconds of their injury and at intervals thereafter. Although they were initially able to give a lucid account of events occurring just before the blow, they subsequently developed a 'relatively complete' retrograde amnesia.

An attraction of consolidation theory is that the properties and time-course of action of the cholinergic neurotransmitter system might appear to make it an excellent 'candidate' as the physiological substrate of the process (see Kopelman, 1985*a*, 1986*c*). A problem is that such a rapid consolidation process cannot explain why a retrograde amnesia of more than a few seconds can occur; and Squire *et al* (1984) have recently postulated 'consolidation' lasting as long as two years to try to account for this difficulty (see below).

Accelerated forgetting

A third possibility focuses attention upon 'storage' (retention) rather than learning processes. In recent years, it has been suggested that patients who have hippocampal lesions show faster forgetting, even after material has been adequately learned (Huppert & Piercy, 1979; Squire, 1981). In one version of this theory, the accelerated forgetting appears to be viewed as being superimposed upon (and arising independently of) any 'acquisition' deficit (Huppert & Piercy, 1979); whereas, in another version, it is seen as a consequence of a faulty (and protracted) consolidation process (Squire *et al*, 1984).

There is some evidence that normal aging causes a slight increase in the forgetting rate of visuospatial information (Huppert & Kopelman, 1987), and that the gross metabolic disruption following ECT produces accelerated forgetting (Squire, 1981). However, there is surprisingly little evidence that structural lesions produce accelerated forgetting. Patients who have the Korsakoff syndrome, penetrating and non-penetrating head injury, and Alzheimer-type dementia have all been shown to exhibit a normal forgetting rate (relative to age-matched controls), once efforts have been taken to ensure that adequate initial learning has taken place (Huppert & Piercy, 1978; Squire, 1981; Kopelman, 1985*b*; Baddeley *et al*, 1987). It seems that the clinical impression of faster forgetting in such patients arises because information has never been adequately absorbed at initial input.

Faulty retrieval

Two types of retrieval hypothesis have been postulated. 'Pure' retrieval hypotheses postulate a retrieval deficit arising independently of any failure

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in 'acquisition' processes. For example, Warrington & Weiskrantz (1973) postulated that amnesic patients are unable to suppress inappropriate responses during recall or recognition tasks. They noted that amnesic patients sometimes respond erroneously to memory tests with what had been the correct replies to previous tests (even if these had not been recalled at the appropriate time); and, secondly, that the provision of retrieval cues (e.g. initial letters of words to be recalled) can improve their performance. On the other hand, it has been shown that healthy subjects also exhibit these phenomena when given recall tests at relatively long retention intervals (e.g. a week) – suggesting that they may be a consequence of poor memory, rather than its cause (e.g. Woods & Piercy, 1974). Moreover, restricting the number of choices in a recognition or cued recall test does not necessarily improve amnesics' performance, relative to controls, in the way that this hypothesis predicts that it should (Huppert & Piercy, 1976; Warrington & Weiskrantz, 1978).

A modified retrieval hypothesis stresses evidence that retrieval processes are dependent upon the nature of initial encoding, in terms of a network of cognitive and affective associations, and it suggests that retrieval deficits arise in consequence of initial encoding deficits. However, when couched in these terms, the distinction between an encoding and retrieval deficit becomes essentially linguistic (Squire, 1980).

Retrograde amnesia

In summary, the evidence reviewed above would seem to suggest that the deficit in anterograde amnesia occurs at the stage of memory 'acquisition,' although the precise nature of this fault, in terms of (psychological) encoding or (physiological) consolidation, remains to be specified. Accelerated forgetting does not seem to occur in cases of structural lesions, and any retrieval deficits appear to take place as a consequence of the initial 'acquisition' deficit.

Such an explanation fails to account for retrograde amnesia and for its temporal gradient, with relative sparing of more remote memories in, for example, the Korsakoff syndrome. However, various clinical studies have suggested that retrograde and anterograde amnesia may occur, and show improvement or deterioration, independently of each other (e.g. Goldberg *et al*, 1982; Kapur *et al*, 1986). Differing anatomical substrates for the two aspects of amnesia have been postulated, but there is very little agreement on which site produces retrograde amnesia.

At a psychological level, several factors have been implicated. Squire *et al* (1984) cited the occurrence of a relatively short (1–2 years) retrograde impairment in some head injury cases, post-ECT confusional states, and a bitemporal lobectomy patient, as evidence for disruption of a prolonged 'consolidation' process. Much more commonly, the length of retrograde amnesia in head injury is a few seconds or minutes only (Russell & Smith, 1961), and it certainly seems very plausible that such a short RA might result from a disrupted physiological process, such as 'consolidation'. However, the more extensive remote memory impairment in Korsakoff and Alzheimer cases requires a different explanation.

Butters & Albert (1982) demonstrated a loss of recent memories in chronic alcoholics, which they attributed to a progressive anterograde impairment during the period of heavy drinking. Korsakoff patients perform substantially worse than other alcoholics at remote memory tests, and Butters & Albert suggested that, superimposed on the alcoholics' loss of recent memories, Korsakoff patients exhibit a 'generalised' retrograde impairment, which has an abrupt onset at the time of the Wernicke encephalopathy. Alzheimer patients may manifest a similarly generalised retrograde loss, in which a relative sparing of the most remote memories may be more apparent than real (Wilson *et al*, 1981). It has been suggested that this generalised, retrograde loss consists of a 'general impairment in reconstructing past memories' (Squire *et al*, 1984); and, in this connection, it may be relevant that various authors have emphasised the role of contextual cues (e.g. time, order, place) in the 'reconstruction' (conjuring up) of past memories (e.g. Baddeley, 1982).

Memory for context

There is now evidence that Korsakoff patients and some (but not all) other amnesic patients have particular difficulty in recalling the context of information (Huppert & Piercy, 1976; Mayes *et al*, 1985). In studies of anterograde amnesia, they show a disproportionate difficulty in recalling the temporal sequence of events, the place where something was learned (spatial context), and the source of information even when the information itself has been recalled. In studies of retrograde amnesia, they exhibit problems in dating or ordering events.

There are problems of method in most of these studies, but evidence is accumulating that it is the presence of concomitant frontal lobe pathology in amnesia which may underlie the failure to recall the context of information (Squire, 1982; Mayes *et al*, 1985). Since some degree of frontal dysfunction is common in Korsakoff and Alzheimer patients, it seems reasonable to suppose that this failure to recall

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contextual information may contribute to their difficulty in retrieving from remote memory – a hypothesis currently being tested by the author. As mentioned above, frontal dysfunction and impaired recall of temporal context have also been implicated as factors producing 'spontaneous' confabulation: it seems plausible that spontaneous confabulation may reflect more extensive frontal lobe pathology, resulting in the extremely incoherent (context-free) retrieval of past memories and associations.

Memory without awareness

Whatever the fundamental deficit in organic amnesia, certain aspects of memory *do* appear to be relatively preserved in amnesia, and these appear to be those components which do not require conscious recollection of personal experiences. In the nineteenth century, James Mill, Henry Maudsley and William James had all emphasised that an essential component of memory is the "additional consciousness that we have thought or experienced (something) before" (James, 1890). However, at the turn of the century, Claparede (1911) demonstrated that "the feeling that (a memory) belongs to the person's experience can be absent in situations in which retention is evident". He cited a Korsakoff patient whose finger he pricked with a pin, and who avoided shaking Claparede's extended hand the next day without knowing why. This lady was able to learn her way around the hospital, e.g. to the bathroom, but was completely unable to describe her route.

Modern experiments have confirmed that severely amnesic subjects can show normal conditioning (Weiskrantz & Warrington, 1979), and can acquire and retain affective reactions despite their profound impairment of 'conscious recollection' (Johnson *et al*, 1985). They can also acquire and show retention in certain skills, e.g. performing jigsaw puzzles or reading back-to-front writing, in the absence of any recall or recognition of having performed the tasks before. In a particularly striking example, Starr & Phillips (1970) described a pianist who was taught a new piece of music but the next day had no recall of it: on being hummed the first few bars, he was able to continue playing the piece, although still unable to recall the name of the piece or that he had been taught it before. Such skill (or 'procedural') learning does not necessarily appear to depend upon well-established premorbid learning, and most researchers postulate that it depends upon a subsystem of memory independent of that which subserves 'conscious recollection' (Moscovitch, 1982; Squire *et al*, 1984).

Probably related is the 'priming' phenomenon, in which a learning episode has a transient, facilitative effect on the performance of a subsequent task. For example, viewing a series of words during a study session makes it especially likely that amnesic subjects will give those words in response to some subsequent test, even though their performance at a recognition test of those words may be severely impaired. Shimamura & Squire (1984) propose that priming involves the activation of 'pre-existing elements or processing structures', which, in turn, may be necessary for skill learning to develop.

A further suggestion, put forward in different ways by Gillespie (1937) and Tulving (1972), is that 'semantic memory' is selectively spared in organic amnesia, i.e. memory for facts, concepts, and language (as opposed to memory for personal experiences or events). Unfortunately, the status of semantic memory as an independent memory subsystem remains equivocal, because the tests used to examine it have usually required the recall of material which has been 'over-learned' premorbidly. Moreover, the concept of 'semantic memory' is itself something of a conglomerate, in that the memory system which subserves our use of language (commonly spared in amnesia) may well differ from that which enables us to name the present or past Prime Minister (characteristically affected in amnesia).

The neuropathology of amnesia

Neuroanatomy and neurophysiology

Table II lists the principal types of pathology which give rise to an amnesic syndrome. Consideration of these pathologies has led many authorities to postulate that two types of lesion are particularly likely to disrupt the formation of new memories: bilateral hippocampal lesions and dieneephalic lesions.

TABLE II
Aetiology of the amnesic syndrome

Thiamine deficiency	—alcohol
	—malnutrition
	—malabsorption
Head injury	
Post-encephalitis	—e.g. herpes simplex
Anoxia	—e.g. CO poisoning
Vascular lesions	—e.g. thalamic infarction
	—subarachnoid haemorrhage
Tumour (third Ventricle)	
TB meningitis	
(Bilateral) temporal lobectomy	

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Scoville & Milner (1957) reported moderate or severe amnesia in eight patients who had undergone bilateral temporal lobectomy for management of psychosis or epilepsy, the amnesia being most severe in three cases in whom the removal of the hippocampi was most extensive. No amnesia was seen in a case who had a unilateral operation only, nor in a case in whom the hippocampi were spared. One of the most severe cases (H.M.) has been described in detail (e.g. Milner, 1966). He had a retrograde component to his amnesia extending back 2 or 3 years before his operation, and a profound anterograde amnesia. More than a decade after his operation, he was unable to find his way to the home in which he had lived for several years; he performed the same crossword puzzle repetitively without showing practice effects; and he mourned afresh every time he learned that his uncle had died. However, his immediate memory span, his IQ, and his ability to hold conversation were all normal (Milner, 1966). Patients who have suffered a herpes simplex encephalitis sometimes manifest a similarly severe amnesia, and the neuropathology typically involves a profound necrosis of bilateral, medial temporal structures as well as some involvement of the orbitofrontal regions (Hierons *et al*, 1978). The hippocampi and the temporal lobes are, of course, particularly implicated in the neuropathology of Alzheimer's disease (Tomlinson & Corsellis, 1984); and the temporal poles are especially vulnerable to bilateral contusions in head injury (Teasdale & Mendelow, 1984).

Diencephalic involvement was first implicated from autopsy studies of Korsakoff cases. Malamud & Skillicorn (1956) and Victor *et al* (1971) noted that the pathology occurred in the paraventricular and peri-aqueductal grey matter, particularly in the mammillary bodies and medial-dorsal nucleus of the thalamus. Victor *et al* (1971) pointed out that all 24 of their cases, in whom the medial-dorsal nucleus of the thalamus was implicated, had a history of memory impairment (Korsakoff syndrome), whereas five cases in whom it was not affected had a history of Wernicke features only. By contrast, the mammillary bodies were implicated in all the Wernicke and Korsakoff cases examined. Amnesia in association with unilateral or bilateral involvement of the thalamic nuclei, with apparent *sparing* of the mamillary bodies, has been described in cases of tumour, trauma, and infarction (McEntee *et al*, 1976; Squire & Moore, 1979; Speedie & Heilman, 1982). However, Mair *et al* (1979) have described two amnesic cases whose autopsies showed lesions in the mammillary bodies and the midline of the thalamus, but *not* in the medial dorsal nuclei. Mair *et al* suggested that the lesions they described might 'disconnect' a critical circuit running between the temporal lobes and the frontal cortex.

A potential difficulty for the view that the diencephalon and hippocampi are critical in memory formation is that lesions of the fornix bundle, which transmits connecting fibres between them, do not always seem to produce amnesia in animals and man. However, the more recent animal studies suggest deficits consistent with those seen in human amnesia: namely, a relative sparing of primary memory and the learning of affective associations, together with a profound impairment of secondary memory as measured on recognition tests (Owen & Butler, 1981; Carr, 1982). A more serious problem, documented by Markowitsch (1984), is that lesions within the hippocampi and thalamic nuclei themselves do not always give rise to amnesia and, when they do, there is usually concomitant involvement of other nuclei. Whilst acknowledging that there may be 'nodal points' in the neural substrate of memory, Markowitsch argues that the thalamic nuclei and hippocampi do not represent discrete 'functional entities', but are embedded in complex neural circuits and 'neuronal assemblies'. Similarly, Mishkin (1978) has argued that *combined* lesions within the hippocampal (medial limbic) and amygdaloid (basolateral limbic) circuits are required to produce severe amnesia; and it seems plausible to suppose that neurotransmitter depletion within these circuits, which contain cholinergic pathways, may be critical in producing amnesia. The dorsomedial thalamus and medial temporal lobes are the anatomical locations ('nodal points') where these two circuits converge, and are, therefore, particularly vulnerable to the effect of discrete, structural lesions (Speedie & Heilman, 1982).

Neurochemistry

Recent attention has focused upon the neurochemistry of amnesia. In the Korsakoff syndrome, the role of thiamine deficiency was confirmed by the De Wardener & Lennox (1947) study of prisoners of war in South East Asia; and, more recently, Blass & Gibson (1977) have postulated a hereditary abnormality of transketolase metabolism which predisposes some alcoholics to this disorder. However, this latter study was based on only four cases, and other studies indicate wide variability in transketolase activity amongst Korsakoff and other alcoholics (Leigh *et al*, 1981).

In Alzheimer's disease, three sources of evidence suggest that cholinergic depletion contributes to the memory disorder:

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(a) neuropathological studies demonstrating reduction in cholinergic enzymes and synthesis
(b) pharmacological studies examining the effect of cholinergic 'blockade' in healthy subjects
(c) clinical trials administering cholinergic agents to Alzheimer patients.

These studies have been reviewed in detail elsewhere (Kopelman, 1986c), and it seems likely that cholinergic depletion accounts for some, but not all, of the memory deficits seen in Alzheimer-type dementia. A small study has suggested that there may also be depletion of cholinergic neurons in Korsakoff's syndrome (Arendt *et al*, 1983), and the pattern of the anterograde memory deficit in this disorder would indeed be consistent with such an observation: however, there is insufficient neuropathological evidence to place any great weight on this hypothesis as yet.

The role of other neurotransmitter systems in memory and dementia remains to be elucidated. An impairment of the ascending noradrenergic pathways in Korsakoff's syndrome has been implicated by McEntee *et al* (1984), but their results have not been replicated (Martin *et al*, 1984). Moreover, animal studies implicate the adrenergic system in mechanisms mediating attention and arousal, and trials of vasopressin, which modulates adrenergic activity, indicate an effect on arousal and mood rather than memory (Kopelman & Lishman, 1986).

Psychogenic amnesia

Diagnosis and phenomenology

Table I includes psychologically based amnesias, giving fugue states and situation-specific memory loss as examples of 'discrete' amnesias, and depressive pseudodementia as a more persistent impairment. In both discrete and persistent amnesia, the differentiation of psychogenic from organic causation can be surprisingly difficult. In the discrete type, the diagnosis usually relies on such features as the rate and circumstances of the onset, loss of personal identity (rare in organic amnesia except in advanced dementia), and whether new learning is affected (often spared in psychogenic amnesia, although not necessarily so). In the persistent type, the past and family history, the results of physical investigations, the current pattern of deficit, and the patient's response to it, all provide important clues, but the diagnostic problem can remain perplexing.

The phenomenology of psychogenic amnesia often bears interesting resemblances to organic amnesia. For example, there may be islets or fragments of preserved memory within the amnesic gap. A woman, who was due to meet her husband to discuss divorce, recalled that she was 'supposed to meet someone' (Kanzer, 1939). A young man, who slipped into a fugue following his grandfather's funeral, recalled a cluster of details from the year (1979) which he described (after recovery) as having been the happiest of his life (Schachter *et al*, 1982). The subject may adopt a detached attitude to these memory fragments, describing them as 'strange and unfamiliar' (Coriat, 1907). In many cases, semantic knowledge remains intact, e.g. foreign languages and the names of streets, towns, and famous people (Kanzer, 1939; Schachter *et al*, 1982), whereas in others it is also implicated (Coriat, 1907; Abeles & Schilder, 1935; Kanzer, 1939). Similarly, performance at verbal learning tests has been reported as unaffected (Abeles & Schilder, 1935), mildly impaired (Schachter *et al*, 1982), or more severely impaired (Gudjonsson & Taylor, 1985). Memory for skills is often preserved (e.g. Coriat, 1907), but in the Padola case (Bradford & Smith, 1979) retention of a rudimentary knowledge of aerodynamics and of other skills (e.g. solving jigsaw puzzles) was taken as evidence against an organic amnesia – an interpretation in conflict with the recent findings regarding the relative preservation of 'procedural' memory, reviewed above. Sometimes, memory retrieval may be facilitated by chance cues in the environment (e.g. Abeles & Schilder, 1935; Schachter *et al*, 1982) but deliberate cueing is often unsuccessful (Coriat, 1907; Kanzer, 1939), and the results of amytal abreaction are very variable.

Fugue states

'Fugue states' refer to a syndrome characterised by the abrupt onset of a loss of personal ('episodic') memory and of the sense of personal identity, associated with a period of wandering. The episode usually resolves within a few hours or days, after which the subject is left with a residual amnesic gap for the period of the fugue. Fugue states are rare, but their study has helped to elucidate the factors predisposing to psychogenic amnesia. These are listed in Table III.

There is always a severe precipitating stress, and fugues are reported much more commonly in wartime (Sargant & Slater, 1941). However, they also occur following marital or emotional discord or at times of financial difficulty. A history of depressed mood just before the onset of the fugue is virtually always present, including those cases in which there is some other, formal psychiatric diagnosis (Berrington *et al*, 1956). Occasionally, the fugue may even represent a 'flight from suicide' (Stengel, 1941). For example, Abeles & Schilder (1935) described a

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TABLE III
Psychogenic amnesia: factors predisposing to fugue states

1. Precipitating stress e.g.	—marital discord	(Kanzer, 1939)
	—financial problems	(Kanzer, 1939)
	—offending	(Wilson *et al*, 1950)
	—war stress	(Sargant & Slater, 1941; Parfitt & Gall, 1944)
2. Depressed mood		(Kanzer, 1939; Stengel, 1941; Berrington *et al*, 1956)
3. Suicide attempts		(Stengel, 1941; Berrington *et al*, 1956)
4. Past history of head injury		(Abeles and Schilder, 1939; Berrington *et al*, 1956)
5. Past history of alcohol abuse		(Kanzer, 1939; Berrington *et al*, 1956)
6. Epilepsy		(Stengel, 1941)
7. Other neurosis		(Sargant & Slater, 1941)
8. Other organic disorder		(Kennedy & Neville, 1957)
9. Tendency to lie		(Stengel, 1941; Wilson *et al*, 1950; Berrington *et al*, 1956)

woman who deserted her husband for another man: after a week she determined to return to her family but, as she descended the Underground stairway, she was contemplating suicide. "Instead amnesia developed", the authors tersely reported. Stengel (1941) claimed that suicide never occurs during a fugue, but may take place as the subject emerges from the fugue. A past history of an organic amnesia is also common, arising, for example, from a head injury, an alcholic black-out, or from epilepsy. Berrington *et al* (1956) reported that 16 of their 37 cases had previously experienced a severe head injury, and a further three cases head injury of uncertain severity. On the other hand, epilepsy is reported less commonly than is generally supposed, Stengel giving the highest figure (10%). In summary, it appears that some subjects, who have experienced an earlier organic amnesia, are predisposed to developing a psychogenic memory loss, in the presence of depressed mood and a severe precipitating stress. On the other hand, several authors have remarked that these patients tend to be rather unreliable personalities with either a tendency to lie or a legal motive for wishing to have a fugue state substantiated.

E.F. was a 46-year-old man who described 12 to 15 episodes of 'going blank' during the previous 5 years. He said that these episodes lasted 2 to 36 hours, and that, on 'coming round', his feet were often sore, he was a long way from home, and he had no idea of the time or what had been happening during the previous hours. For example, he found himself on one occasion near the Thames, 10 miles from his home, with his clothes sopping wet. There was a history of epilepsy since he was 19, ECT and bilateral leucotomy at 33, major cardiac surgery, recurrent depression, and two serious suicide attempts. He had recently married for the third time: his wife was many years younger than himself and was pregnant. Marital difficulties were suspected, but were vehemently denied by both partners. Mr E.F. asked for a psychiatric report after being charged with driving whilst disqualified, without any insurance, and whilst under the influence of alcohol: his defence was that he had been in a fugue state.

Amnesia for offending

A common form of psychogenic amnesia with important implications is that claimed for a criminal offence. Most commonly, this has been described in association with homicide, in which it is claimed in 30–40% of cases (Table IV). A study in Broadmoor Hospital (Hopwood & Snell, 1933) and another in Brixton remand prison (Taylor & Kopelman, 1984) showed that amnesia also occurs in other violent crimes, although less commonly. In these studies, amnesia was found rarely or not at all in non-violent crime: this may have reflected the locations of the studies, but there is evidence that in violent crime recall by victims and eyewitnesses is also impaired. Taylor & Kopelman (1984) found that amnesia for an offence occurs in three types of circumstance:

(a) homicide cases in which the victim is closely related to the offender (a lover, wife, or family member), and the offence is unpremeditated and takes place in a state of high emotional arousal

TABLE IV
Homicide studies

	n	*% Amnesic*
Leitch (1948)	51	31
Guttmacher (1955)	36	33
O'Connell (1960)	50	40
Bradford & Smith (1979)	30	47
Taylor & Kopelman (1984)	34	26
Parwatikar *et al* (1985)[1]	105	23

1. Pre-trial evaluations only; rate for convicted homicides not given.

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(b) chronic alcohol abusers who commit a crime when severely intoxicated and whose victims are much less commonly related to them
(c) a small number of schizophrenic patients who commit criminal damage when floridly psychotic.

G.H. was a 40-year-old man, who had been born in Egypt, and was Jewish by religion. He had lived in England for 20 years, and was married to a 33-year-old Englishwoman. There was a son aged 9 and a daughter aged 3. The wife was having an affair with a musician, and G.H. was being treated for depression as an out-patient at a teaching hospital. He was taking an antidepressant and a benzodiazepine. On the day of the offence, the couple had a furious row during which G.H. threatened to kill the musician. His wife shared a bedroom with her daughter, and the last thing G.H. remembered was going to kiss the child goodnight. He reported that he could not remember what happened after that until the police arrived, but that he had been told that he phoned the police himself. He was charged with the murder of his wife by stabbing.

Depression and pseudodementia

A more persistent, psychologically based memory impairment is that occurring in a depressive pseudodementia, the clinical features of which have been described elsewhere. Memory impairment appears to be common in depression, although in the majority of cases it is relatively mild and poorly correlated with the severity of the depression (e.g. Kopelman, 1986*a*). The nature of the impairment in those cases which masquerade as 'dementia' is unclear, but one suggestion is that the disorder involves the more 'effortful' aspects of memory processes, and is closely associated with deficits in attention, motivation, and drive (Cohen *et al*, 1982). Affective disorder also gives rise to some particularly interesting state-dependent phenomena: depressives show a relative facility in recalling unpleasant events (Lishman, 1972; Lloyd & Lishman, 1975); this increased accessibility of unpleasant memories has a diurnal variation (Clark & Teasdale, 1982); and patients with mood swings show superior performance at memory tests if mood is consistent between learning and recall than if the mood changes (Weingartner *et al*, 1977).

Mechanisms in psychogenic amnesia

What mechanisms underlie psychogenic amnesia? One possibility is that many instances of psychogenic amnesia may result from faulty encoding of information at initial input (Kopelman, 1985*a*) and that deficits in retrieval may arise in consequence of this. In support of this view, it should be noted that psychogenic amnesia commonly occurs in states of abnormal mood or extreme arousal, in which normal cognitive processing might well be compromised. Fugues arise in the context of depressed mood and severe stress; amnesia for offences in situations of extreme emotional arousal, severe intoxication, or florid psychosis; and memory disorder in depression in association with impaired attention and motivation. This view, emphasising a disturbance in memory 'acquisition', suggests that the impairment in psychogenic amnesia mimics that in organic amnesia—of which there is so often an earlier history.

A second possibility is, of course, that psychogenic amnesia is an example of 'motivated forgetting' or repression. There is a considerable literature purporting to test the 'repression hypothesis' by showing that subjects tend to recall 'pleasant' events more readily than 'unpleasant' (e.g. Meltzer, 1930) and that this phenomenon cannot be accounted for by the relative affective intensity of the memories (Master *et al*, 1983). Although Freud (1915/17) wrote that "it is an undoubted fact that disagreeable impressions are easily forgotten", this kind of experimental study has often been criticised on the grounds that it fails to take account of the role of 'antagonistic impulses' in engendering repression—"an attempt at flight by the ego from libido which is felt as danger" (Freud, 1915/17). Experimental tests of repression in this sense tend to rely on very crude laboratory analogues, but Erdelyi & Goldberg (1979) have reviewed a number of concepts in contemporary cognitive psychology which appear to resemble repression. In this connection, the recent observations in organic amnesia of memory without awareness, and especially of preserved affective reactions in the presence of profound amnesia, are particularly intriguing.

A third possibility, strongly advocated by Bower (1981), is a (primary) retrieval deficit. Bower suggested that the 'discrete' psychogenic amnesias may reflect mood-state dependent phenomena, similar to those described in depression. According to this view, the experiences of a 'dissociative state' would be retrieved if the subject could be restored to the same subjective state in which they originally occurred. Fugue states and amnesia for offences are both associated with depressed mood and extreme states of arousal (Berrington *et al*, 1956; Taylor & Kopelman, 1984); and the latter are also associated with heavy abuse of alcohol in which state-dependent phenomena have commonly been described (Goodwin *et al*, 1969). Against this view, the state-dependent phenomena seen in laboratory experiments tend to

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be small and undramatic; mood disorder and alcohol abuse are common, whereas psychogenic amnesia is rare; and the only direct test of the hypothesis has failed to support it (Wolf, 1980).

Conclusion

This review has attempted to summarise the clinical features and the pattern of the memory dysfunction in selected examples of organic and psychogenic amnesia. It has also surveyed various theories which purport to explain the nature of the dysfunction.

It appears that the anterograde amnesia produced by structural lesions, including the widespread neuropathology of Alzheimer's disease, involves a severe impairment in acquiring (or learning) new information, and any retrieval deficits arise as a consequence of this. Contrary to clinical impression, the forgetting rate of new information is not accelerated, once adequate learning has been accomplished – although elderly people do show somewhat poorer retention of visuospatial material than younger subjects, and the metabolic disruption which follows ECT may produce accelerated forgetting. The precise deficit in psychological 'encoding' or physiological 'consolidation', which underlies this acquisition (or learning) deficit, remains to be elucidated. However, the simultaneous implication of two limbic circuits has been postulated as critical for the occurrence of anterograde amnesia, and it is known that these pathways contain cholinergic fibres. Future research is likely to clarify further the role of neurotransmitter depletion in producing amnesia.

Both psychological and pathophysiological evidence suggest the partial independence of the retrograde and anterograde components of amnesia. Retrograde amnesia may result from a number of factors, including a failure to make use of contextual cues (with regard to time, order, place, and source) in 'reconstructing' past experience. There is controversy as to where the critical lesions lie which produce retrograde amnesia, but there is evidence that frontal lesions disrupt the recall of contextual information. Moreover, two types of confabulation have been postulated, one of which ('provoked') may be a normal response to poor memory, but the other ('spontaneous') is characterised by the extremely incoherent (context-free) retrieval of memories and associations, and appears to reflect more extensive frontal pathology.

Finally, it has been postulated that many instances of psychogenic amnesia may resemble organic in that they result from the impaired acquisition of information at initial input, perhaps thereby predisposing the subject to subsequent retrieval difficulties in consequence of this. Such a view does not exclude the role of other possible mechanisms: organic amnesia provides a model for 'memory without awareness', and state-dependent phenomena may occur as occasional instances of retrieval difficulty.

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