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The amnesic syndrome

THE correlation between hippocampal damage in humans and some form of generalized memory defect has been established for over two decades (cf. Scoville and Milner 1957). Considerable research in the intervening years has failed to characterize this defect with any precision and has consequently failed to determine the exact nature of hippocampal function in humans. We have discussed, in the previous chapter, a model of human hippocampal function derived in part from work with infra-humans and in part from a consideration of the special features of human information storage and memory processing. In this chapter we shall apply this model to the data generated from the study of amnesic patients in the hope of coming to some understanding both of the syndrome of organic amnesia and of the role played in it by hippocampal damage.

The organic amnesia resulting from hippocampal damage, and a variety of other causes to be discussed shortly, is not the only form of memory loss known in the clinic, nor should it be in view of our comments on the non-unitary nature of memory processes. In accordance with the proposed properties of the taxon systems and the nature of its memory stores, damage in a variety of extra-hippocampal areas yields disorders which can be viewed as *limited amnesias* (cf. Whitty and Lishman 1966); examples include *agnosia*, *apraxia*, certain forms of *aphasia*, *prosopagnosia*, which is a specific deficit in face memory, and perhaps the selective deficit in verbal short-term memory associated with conduction aphasia (Warrington and Shallice 1969). Patten (1972b) has analysed some of these modality specific amnesias in detail.

Amnesia consequent upon bilateral damage to the locale system, in contrast, appears to be neither modality nor material specific. Unilateral damage, as we shall see, does lead to some specificity: left-hemisphere damage affecting verbal memory, right-hemisphere damage affecting nonverbal memory. Still, there is no modality specificity in these defects; what is important is the form of the to-be-stored information, not its channel of input. Looking ahead briefly, there are both *retrograde* and *anterograde* components to the organic amnesia syndrome. That is, there is both a loss of previously stored information and a loss in the ability to store new information. The fact of central importance in the syndrome, which has

only recently become clear, is its selectivity. All old memories are not lost; some new information can be stored. The task of any theory of hippocampal function in humans is to provide the basis for understanding the selective memory loss,* which contemporary research is slowly defining. This is parallel to the problem presented by the study of hippocampal damage in infra-humans, where the selectivity of the behavioural effects also constitutes the central problem for theory.

Before turning to an analysis of the syndrome elicited by hippocampal dysfunction there are several methodological problems which must be considered. First and foremost we must consider the evidence relating the organic amnesia syndrome to hippocampal damage. In some cases the damage is known and there is no interpretive problem; in others the damage is unknown and subjects are classified solely on the basis of behavioural observations. Determining the extent to which hippocampal damage is responsible for the observed behaviour is a necessity. Following this we shall briefly describe some of the testing procedures used in the clinic, concentrating on those which assess memory function. Our purpose here is simply to provide the reader with some background concerning the terminology to be used, as well as an idea of the kinds of information which can be derived from the use of particular experimental procedures. Finally, we shall, on the basis of our theory, provide a set of 'predictions' concerning the effects of hippocampal damage. These 'post hoc' statements do not represent attempts at verification of the theory; such verification must come from future research aimed at a finer analysis of the syndrome.

Following these methodological and predictive sections, we shall review the clinical data, touching first upon data pertinent to the functions of the taxon systems which should provide an indication of the minimal learning capabilities of amnesic patients. Then, we turn to data concerned purely with the spatial mapping function of the (right) hippocampus and with its role in exploration and curiosity. Finally, we consider the central problem of the defect in memory storage, which has primarily been studied within the context of verbal learning paradigms. Here, we hope to show that there is a specific deficit in the processing and storage of deep-structure maps, pertinent to both imaginal and language content. This should manifest itself most obviously in the loss of context-specific memory.

15.1. The role of hippocampal damage in organic amnesia

The absence of precision in prior characterizations of human hippocampal function arises not only from the use of inadequate psychological models for memory; frequently the focus of interest was the clinical syndrome of

* Marslen-Wilson and Teuber (1975), in analysing the defect in an amnesic patient, state that his behaviour 'underscores the perplexing quality of severe amnesia: it is not all or none . . . and it does not readily lend itself to those interpretations of amnesia which speak of generalized failure either of storage or retrieval' (p. 362).

organic amnesia, rather than a particular brain structure, and it was impossible to assign functions to a given structure. The syndrome of organic amnesia, which arises after hippocampal damage, has also been associated with several other clinical states, not all of which can be easily characterized as to precise locus of brain damage. To some extent this has confused matters, at least from the perspective of an interest in hippocampal function. It is important to discuss, then, the nature of the patient population under study, both in terms of possible sites of damage and in terms of specific clinical manifestations that obviously have little to do with hippocampal damage.

15.1.1. VARIETIES OF ORGANIC AMNESIA

Our knowledge of the organic amnesia syndrome comes from the study of a variety of patient populations: (1) those with Korsakoff's psychosis, typically as a sequella to prolonged alcoholism or encephalitis (Korsakoff 1889); (2) those with mesial temporal lobe damage, resulting either from an accident, from vascular occlusion or a tumour, from temporal lobe epilepsy, or after an operation to alleviate focal epileptic seizures. These varied populations present somewhat divergent clinical patterns and it thus becomes important to try to establish the extent of hippocampal dysfunction in each of them. The perfect subject for study would have damage limited strictly to the hippocampus in an otherwise normal brain; few, if any, such cases exist.

There are two questions, in practice, which must be answered: (1) to what extent is hippocampal damage responsible for the defects seen in diseases known to involve diffuse damage, such as Korsakoff's psychosis? (2) to what extent is the amnesia seen after temporal lobe surgery for epilepsy a function of the prior existence of an epileptic focus?

15.1.1(a). The role of hippocampal damage in organic amnesia. The operation which left H.M., Scoville's (1954, 1968) classic patient with a profound memory defect involved the removal of much of the mesial temporal lobe, including most of the hippocampus, uncus, and amygdala, but spared the lateral temporal neocortex. Subsequent to the discovery of H.M.'s memory defect, Scoville and Milner (1957) examined some of the patients who had previously undergone similar, though often less drastic, removals. Generalized memory defects were seen in all those patients whose removals included portions of the hippocampus. More pertinent, perhaps, is the fact that no generalized memory defect was apparent when damage was limited to the uncus and amygdala. Sawa *et al.* (1954) and Terzian and Dalle Ore (1955) have also noted that resections limited to the amygdala do not impair general memory function. Thus, it seemed likely, though not proven, that hippocampal damage was crucial for the memory defect. Milner (1968) correlated right temporal lobectomy with

memory defect and found that there was a strong correlation between the extent of hippocampal removal and the gravity of the memory loss. Finally, in what appears to be a definitive answer to this question, Corsi (1972) tested an extensive series of unilateral temporal lobectomy patients with varying degrees of hippocampal damage and found that the amnesic condition was directly related to the loss of hippocampal tissue. As we shall see later these losses were material specific: verbal defects were seen after left hippocampal damage and non-verbal defects after right hippocampal damage. Thus, it now seems established that the generalized amnesia resulting from temporal lobectomy is a direct function of hippocampal damage.*

The question is considerably more complex in the case of patients with Korsakoff's psychosis. This syndrome is defined operationally, so that any number of different pathological states can be associated with it. Most commonly it is seen in the following cases: (1) as the chronic phase of the Wernicke-Korsakoff disease, consequent upon prolonged alcoholism and dietary insufficiency; (2) as the aftermath to an attack of herpes simplex encephalitis; (3) as the aftermath of a stroke, or occlusion of the posterior cerebral arteries (Glees and Griffith (1952) and Victor et al. (1961) report such cases); (4) with certain brain tumours, as in the cases reported by Sproffkin and Sciarra (1952); (5) in association with Alzheimer's disease.** Associated with the last four of these precipitating conditions is clear-cut involvement of hippocampal dysfunction; considerable damage to the hippocampus has been reported in most cases that have been described (cf. Whitty and Lishman 1967). However, in the Wernicke-Korsakoff syndrome, based on chronic alcoholism, pathology has been traced most prominently to the mammillary bodies and the dorsomedial thalamus (Brierley 1966, Victor 1964, Victor, Adams, and Collins 1971). To be sure, these are target areas for outputs from the hippocampus and subiculum, yet one would feel more comfortable if changes were seen in the hippocampus itself. It remains possible that more detailed histochemical investigation at autopsy would show up subtle changes in the hippocampus; only future research can provide an answer to this possibility. We are forced to conclude that the amnesia seen in Korsakoff's syndrome is associated with damage either to the hippocampus itself or to its output systems.

* While the involvement of hippocampal damage in the amnesia produced by temporal lobectomy is well established, it is not always the case that damage to the hippocampus, or more precisely the fornix, always results in amnesia. There are several reports in the literature (e.g. Woolsey and Nelson 1975) of cases involving fornix section without memory loss. We, along with others, have no good explanation for these anomalous reports; it should be kept in mind however, that the subiculum and not the hippocampus is the origin of the fornix.

** This list is by no means exhaustive. Other precipitating factors include: (1) the aftermath of electroconvulsive shock therapy (cf. Squire 1975); (2) operation for anterior artery aneurysm (cf. Lindqvist and Norlen 1966); and (3) concussion (cf. Yarnell and Lynch 1973). We will not be discussing data from such cases in this review, but they do not differ either in the possibilities they offer for study or the problems attendant with such use from the examples offered in the text.

However, we must bear in mind that Korsakoff patients, who comprise the majority of amnesic patients studied in the clinic, typically have extra-hippocampal damage of a diffuse nature. Though most investigators are careful to exclude patients with general dementia from their studies, the presence of diffuse damage could mean that the presenting syndrome for Korsakoff patients would be different than that seen in other patients, and, in fact, several studies have demonstrated deficits in sensory thresholds in these patients (e.g. Oscar-Berman, Goodglass, and Cherlow 1973, Jones *et al.* 1975) which are not apparent in patients with focal hippocampal damage. It is also important to note that there might be different forms of Korsakoff's disease (cf. Lhermitte and Signoret 1972), varying in the extent of damage to different structures or within the two hemispheres. Finally, Korsakoff's psychosis is not a static condition involving a fixed amount of damage. The same patients could vary from study to study, and different patients could vary within a study. Some assessment of the ongoing intellectual deterioration of such patients is important in order to facilitate comparisons between studies. This problem has plagued the study of Korsakoff's psychosis, possibly lying at the root of seemingly contradictory results reported by two groups of investigators as we shall see later (p. 427).

15.1.1(b). The contribution of the epileptic focus. The Montreal series of temporal lobectomy patients all underwent operations for the relief of focal epilepsy. The question of the contribution of epilepsy itself to the amnesic syndrome thus becomes an important one. In the epileptic patient grand mal, and often petit mal, seizures are accompanied by transient amnesias; direct stimulation of the hippocampus can have the same effect (e.g. Bickford *et al.* 1958). In these cases amnesia results from a 'functional lesion' induced by the seizure or stimulation. Consistent with this view is the fact that seizures are associated with automatisms of behaviour which we would relate to the residual functioning of the taxon systems and which bear a striking resemblance to the stereotypies seen in animals with hippocampal lesions.

On the other hand, when areas afferent to the hippocampus are stimulated, 'memories' can often be elicited, as in Penfield's studies (e.g. 1958). This activation can be compared to the syndrome seen in the acute phase of the Wernicke-Korsakoff disease, described by Torda (1969) as a 'state of continuous hallucinosis' during which the patients continuously 'heard and/or saw their thoughts' (p. 279). A similar state has been described as the immediate, though transient, sequella to anterior cingulectomy, and was attributed to a discharging lesion producing downstream effects in the hippocampus (Whitty and Lewin 1960). All these effects can be ascribed to an activation of the hippocampus, during which the normal mechanisms for activating cognitive maps are bypassed.

When the epileptic tissue is removed, generalized amnesia can result if hippocampal tissue is resected. This defect is not apparent prior to operation, except during overt seizure. The fact that amnesia is present post-operatively only and that it is critically linked to hippocampal damage makes it rather unlikely that amnesia depends upon the pre-operative epileptic condition, as was suggested by Isaacson (1972). Perhaps most convincing in this regard is the existence of amnesic patients with known hippocampal damage but without prior history of seizures (e.g. Walker 1957, Rose and Symonds 1960, Victor et al. 1961, Dejong 1973). These cases indicate that amnesia is associated with the disruption of normal hippocampal function and is not dependent upon the establishment of an epileptic focus.

15.1.1(c). Conclusions. Most forms of organic amnesia seem to involve hippocampal dysfunction. Differences between the various patient populations reside in the locus and extent of extra-hippocampal damage. Korsakoff patients suffer from the greatest amount of such damage and the syndrome must be viewed in this light. It would perhaps be simplest to ignore the data generated from the study of these patients, but this is both impractical and unnecessary, the former because a considerable number of studies have utilized Korsakoff patients, and the latter because there are certain constant features in all varieties of organic amnesia and these can be assumed to define the functions of the structure central to the syndrome, the hippocampus.

15.2. Some clinical tests

Most of the testing methods used in the clinic to assess the amnesic syndrome are concerned with the problem of learning and retention of that learning after a variable interval. A major difference between the various retention tests resides in the amount and form of information provided to the subject during retention.

15.2.1. FREE RECALL

A typical free-recall paradigm might go as follows: A subject is shown a set of words in serial order. Following the presentation of this list and some delay interval, retention is assessed by asking the subject to recall as much of the material as possible. With such a test, the probability of correct responses being emitted by chance is usually quite low.

Free-recall tasks can be used to assess the short-term or long-term retention of any form of material. When the delay between item presentation is short, on the order of 5-20s, one can say that short-term memory is being assessed. Delays beyond that assess long-term memory performance, but only if the subject is prevented from rehearsing the test material. The means of preventing rehearsal vary from study to study;

we shall see later that inadequate control over rehearsal could lead to misinterpretation concerning the status of a subject's short-term memory capability.

The importance of optional input strategies can be assessed in free-recall tests by giving the subject explicit instructions, for instance, to use natural-language mediation (see pp. 387-8n), or to form an image between several of the items, and so on. Similarly, the role of organizational factors, such as category inclusion, can be assessed by using test materials which either explicitly convey that organization (as in lists with category-related words blocked together) or contain the possibility of being organized into chunks by the subject. In the latter case the role of instructions could be important in determining the usefulness of having material presented in this chunkable form.

When several free-recall lists are presented in succession one can measure the extent and form of interaction between the lists—this gives some idea of the way in which memories interfere with one another. By varying the form of similarity between the items in successive lists the nature of coding can be assessed. For instance, if acoustically similar words are confused one can conclude that the storage system being tapped depends on an acoustic code and that systems based on other codes are not functional at that time or in that subject. By shifting the form of interaction between lists one can measure the specificity of interference effects; we shall see that such tests have proved quite useful in assessing the nature of the amnesic defect.

The most important feature of free-recall tasks is that the subject is not given any cues at retrieval.* Thus, these tasks measure the way in which the subject spontaneously encodes information and the availability of that information under what might be considered normal retention conditions. In fact, it will be seen that considerable information which has been registered, and stored, during free-recall presentation is not retrieved by most subjects in the absence of some form of retrieval cue at the time of retention. This indicates that the information has been stored in ways that do not allow for or actively interfere with its subsequent retrieval. Given that, the role of instructions during presentation of the test material and of specific cues during the retention test can be assessed in order to determine the nature of coding and the ways in which access to stored material can be improved. Such 'cued' recall tests have proved to be quite important in the assessment of the amnesic defect, as we shall see shortly.

15.2.2. PAIRED ASSOCIATES

A standard psychological test involves the presentation of pairs of stimuli, the first term being the 'stimulus' and the second the required 'response'.

* With the exception of the location in which the task is performed.

During retention the subject is given the 'stimulus' and must provide the 'response'. This task, termed paired-associate learning, provides the possibility of assessing the effects of the relationship between 'stimulus' and 'response' terms, between 'stimulus' and/or 'response' terms in successive lists, and so on. This paradigm has also been used extensively in the study of imagery processes, with subjects being instructed to form images between the 'stimulus' and 'response' items.

Unlike free recall, in the absence of retrieval cues, the paired-associate task incorporates retrieval information, the 'stimulus' items. Though it is a less demanding task than free recall, it allows for the controlled study of the efficacy of different forms of retrieval cues.

15.2.3. RECOGNITION

Recognition tasks involve a judgment on the part of the subject as to whether or not a test item has been experienced before. Usually, test items are familiar objects or words, and recognition consists in judging whether these items have been seen in a particular context, the test situation. Thus, this paradigm can be used as a sensitive measure of context-dependent memory. Less frequently, test items are unique configurations, e.g. a complex picture or a sentence, and recognition can be based solely on whether or not that configuration had ever been experienced, that is, some manner of familiarity judgment. These two situations, as we shall see, produce strikingly different results in amnesic subjects.

There are two basic forms of recognition test: yes-no recognition and forced-choice recognition. The first form involves the presentation of individual items during retention, with the subject responding either yes (the item is recognized as part of the test material) or no. The second form involves the presentation of many items simultaneously at retention, half of these being drawn from the test material, half not. The subject must choose those which were in the presentation list. In both forms the probability of making a correct response by chance is 50 per cent and some correction for guessing is important. By varying the nature of the 'filler' (non-presented) items one can manipulate the 'false' recognition of similar items. This provides a means of determining the nature of coding used during learning. For instance, the consistent false recognition of acoustically similar words tells us something about the form in which items have been stored.

15.2.4. OTHER TESTS

Aside from these standard psychological paradigms which can be used with either verbal or non-verbal materials, a variety of other tests have been utilized in the study of amnesics. Tests for spatial abilities, for instance,

include mazes similar to those used in rats in their complexity, and these can be performed with either visual or tactual information. Tests for motor learning, such as rotary pursuit and mirror-drawing, have been used as well. We shall describe the features and procedures of these tests when analysing the performance of amnesics.

15.3. Predicted effects of hippocampal damage in humans

The present model provides three sets of overlapping predictions concerning the amnesic syndrome. The first is based upon the functions and properties of the hippocampal locale system, and includes those things which the amnesic patient ought not to be able to do under any condition. The second and third sets are based on the functions and properties of the extra-hippocampal taxon systems, the second including abnormalities in behaviour resulting from a forced dependence upon these systems, and the third including those forms of behaviour which the taxon systems can mediate and which should appear relatively intact in the amnesic subject.

15.3.1. PREDICTIONS FROM THE LOCALE SYSTEM

In view of the properties of the locale system, the amnesic patient should be characterized by the following.

- (1) A lack of initiative, curiosity, and incidental learning.
- (2) A deficit in maze learning and other situations requiring the use of a 'spatial' map of the environment.
- (3) A deficit in those learning situations which are dependent upon retention of contextual information.
- (4) An inability to utilize techniques based on spatial imagery.
- (5) The loss of language functions dependent upon the deep-structure properties of the locale system, e.g. the recall of narrative.
- (6) Selective deficits when unilateral hippocampal damage is involved: verbal defects with left hippocampal damage; non-verbal defects with right hippocampal damage.

15.3.2. PREDICTIONS FROM THE TAXON SYSTEMS

The forced use of the taxon systems should lead to two basic abnormalities in behaviour: (1) a tendency to repeatedly attend (and respond) to the same stimuli; (2) considerable proactive and retroactive interference in the learning of similar materials, which follows from the form of information storage in taxon systems. However, the taxon systems can mediate certain forms of memory, as we have seen both in discussing the animal data and in analysing the properties of human memory. Thus, the amnesic patient should (1) learn simple discrimination tasks, (2) learn motor tasks,

(3) be able to utilize such taxon strategies as rehearsal, category inclusion (chunking), natural-language mediation, and so on, and (4) show normal identification and abstraction, within the limits defined by the large interference effects inherent in the use of taxon storage, limits which could be expanded through the provision of cues which act against these interfering effects.

15.4. Taxon tasks

The first formal indication that hippocampal dysfunction did *not* lead to a complete loss of memory storage came from the study of simple motor tasks, examples of behaviours which we would allocate to the taxon systems. Adequate learning has been demonstrated in rotary pursuit, bimanual tracking, mirror drawing, tapping, and other tasks in both Korsakoff and temporal lobectomy patients (Talland 1965, Corkin 1968, Milner 1968, Cermak *et al.* 1973). These motor tasks do not require the participation of the locale system; in addition, they are not particularly subject to interference effects, and this enables amnesics to use taxon learning mechanisms with some efficiency.

Several aspects of the motor learning of H.M. (Scoville's patient, mentioned above) are worth noting. Though capable of showing long-term improvements, H.M. is still deficient at these tasks when compared with control subjects. In both rotary pursuit and bimanual tracking H.M. seems capable of improving only up to a certain point, at which his performance asymptotes; controls continue to improve beyond this point. The early improvement, in both H.M. and controls, is due to a steady reduction in the amount of time required to get back on the target, once off it (in these tasks the subject must maintain contact with a changing target). Later, the controls begin to spend less and less time off the track; this is not evident in H.M.'s error pattern (Corkin 1968). Corkin suggested that H.M.

'might have been less efficient in predicting the irregular shifts in the tracks incumbent in the bimanual task, although the extent to which this strategy was employed by either normal subjects or H.M. was not determined' (p. 264).

Much the same thing was seen in the rotary pursuit task. Thus, improvements dependent upon reducing the duration of errors can be seen in H.M. However, no improvement in staying on the target appears. This inability to benefit from some memory of the sequential changes occurring in a moving target is in contrast with H.M.'s performance on a task involving the learning of an internally generated sequence; Corkin showed that H.M.'s improvement in a tapping task parallels that of control subjects. In a sense, this pattern resembles the difference between maze-

learning deficits and sequential-response facilitation seen in hippocampal rats (e.g. Jackson and Strong 1969).

Another type of learning that appears normal in amnesics is simple discrimination. Korsakoff patients can form what seems equivalent to a passive avoidance response (Talland 1965).^{*} Sidman, Stoddard, and Mohr (1968) tested H.M. on several types of discrimination. Although unable to verbalize the nature of the task he was performing, H.M. showed normal discrimination between a circle and ellipses of varying degrees of similarity to the circle. Talland did not, unfortunately, test his Korsakoff patients on similar tasks. Finally, Gaffan (1972) has shown, in a single amnesic patient, that the learning of a simultaneous colour discrimination is normal, though the patient was quite certain during the retention tests that the task had never been seen before.

It is worth repeating that Korsakoff patients have difficulties in certain sensory tasks which reveal no impairment in temporal lobectomy patients (e.g. Oscar-Berman *et al.* 1973, Jones *et al.* 1975). Such functions clearly have nothing to do with the hippocampus, and the defects seen in their performance by Korsakoff patients are a reminder of the diffuse damage associated with that disease.

In sum, amnesics can learn certain tasks in a normal fashion. These are analogous to those simple tasks which hippocampal animals can learn and are a function of the operation of the taxon systems. As with the animal data, there are indications that, though able to learn certain things, amnesics often learn in a different way. Some further examples of normal learning in amnesic patients will be presented in following sections in the context of our discussion of the verbal memory defect.

15.5. Exploration and spatial mapping

15.5.1. INITIATIVE, CURIOSITY, AND RELATED PHENOMENA

It was early recognized that Korsakoff patients lack initiative, are apathetic about their surroundings, and are generally passive. Meggendorfer (cited by Zangwill 1966) noted that a lack of initiative was an almost invariant aspect of Korsakoff's psychosis, Burger-Prinz and Kaila (cited by Lidz 1942) noted that Korsakoff patients 'let the world come to them and do not go to meet it', and Talland (1965) stated that an 'abnormal measure of inertness and the absence of spontaneity have been regularly associated with the amnesic anomalies' (p. 115).

Talland has documented the essentially respondent nature of the Korsakoff patient; his attempts to engender social intercourse among groups of four or five patients were futile. All questions were answered without elaboration, and elicited at most a single additional comment from another

^{*} Talland cites a comment by Claparede concerning one of his Korsakoff patients. While shaking hands with a female patient he jabbed her with a pin hidden in his hand. A few minutes later, though unable to recall this incident, the patient refused to shake hands with him.

patient before silence was resumed. On the other hand, given a boring task such as drawing circles, these patients can continue at length without interruption.

Much the same thing has been reported following hippocampal damage. H.M. has a placid temperament and has been described as hypoactive (Milner 1966); he also can continue for long stretches of time at boring, repetitive tasks. The patient discussed by Sweet, Talland, and Ervin (1959), who underwent bilateral section of the fornix, was described as apathetic and lacking in spontaneity. Finally, the patient described by Victor et al. (1961), who sustained bilateral hippocampal damage following several strokes, 'tended to remain quiet for long periods of time and rarely would initiate a conversation' (p. 30).

Formal tests of exploratory capacity indicate a profound lack of interest in investigating uncertain or novel situations in Korsakoff patients. Talland has shown that such patients do not resume unsolved problems if disrupted. Another manifestation of this uninterest is a lack of incidental learning. A jigsaw puzzle composed of parts of a newspaper was given to the Korsakoff patients; solution required that they check to see that the assembled words made sense. Contrary to the control subjects, the Korsakoff patients remembered very few of these words.

Overall, the human amnesic patient shows little spontaneous behaviour, little interest in exploring the environment, and few signs of responding to items which are 'out of place'. This picture compares quite well with what is seen in the rat, cat, and monkey after hippocampal damage.

15.5.2. SPATIAL MAPPING

The absence of exploration is assumed to result from the inability of the amnesic patient to develop cognitive maps. Before turning to more specific data on this point it is worth repeating the distinction we drew in the previous chapter; the locale system is involved in non-egocentric, objective spatial representation, not in egocentric spatial representation. The latter function, involving among other things the recognition of objects in different perspectives and orientations, appears intact in amnesic patients (Talland 1965), though it is disturbed by parietal lesions, as we have seen. The correlation between generalized memory defects and deficits in geographic orientation (Benton 1969) is an indication of the form of spatial representation we assign to the hippocampus.

Anecdotally, there are indications of profound spatial disorientation in amnesics. It takes Korsakoff patients considerable time to learn their way about a hospital, while some never manage this. H.M. shows even more profound disorientation; he appears virtually lost when more than two blocks from his home, often going instead to his old house from which he had moved some years before. Another patient (D.C.) described by Milner (1966) could not find his way about the hospital in which he

had been resident for 11 years. Similar descriptions of disorientation for place are included in most reports of amnesic patients (e.g. Dejong 1973) and will not be enumerated here. These reports provide ample reason to suppose that deficits in maze learning and similar tasks would be seen in amnesics.

Talland's patients were profoundly impaired in learning a relatively simple tactual maze consisting of six choice points in a regular pattern. Further, these patients showed no savings when the maze was rotated. He noted that 'those who discovered the pattern of turns formed but a precarious visual-kinesthetic map' (p. 229). Cermak et al. (1973) have also reported a profound defect in maze learning in Korsakoff patients. H.M. and other bilateral temporal lobectomy patients have been tested on a variety of maze problems. Again, a profound impairment was seen in several modalities, indicating that the underlying disability is a general one. On complex mazes the deficit is nearly total; H.M. failed to solve a 28-choice visual stylus maze within 215 trials. In fact, no improvement was seen, in terms of errors, during this training. The same results were reported by Corkin (1965) for the learning of a tactual maze with 10 choice points. H.M. showed no improvement whatever over 80 trials, though he did show a reduction in the time required to move through the maze, replicating a result noted in the 28-choice maze. This decrease in error time, without an improvement in choice performance, would seem to reflect the same factors as H.M.'s performance in bimanual tracking (see p. 420): the possibility for a reduction in the time spent doing the incorrect thing, but no decrease in the number of times the incorrect thing is done.

H.M. has been tested on shortened forms of these two mazes (Milner, Corkin, and Teuber 1968). Initially, he was given 125 trials on an eight-choice visual maze; he showed little sign of learning during these trials. When the maze was further reduced to six choice points H.M. managed to reach the criterion of three errorless runs in succession after 155 trials. Once having learned the maze he showed considerable retention, even after an interval of six days. The pattern of errors made by H.M. on this maze are of considerable interest. The authors noted that there was a 'high build-up of anticipatory errors ... rather than a random distribution of wrong choices' (p. 226). H.M. did not make repetitive errors within a single trial, which argues against any simple response perseveration interpretation of his defect, as the authors pointed out. This pattern of errors bears a striking resemblance to that seen in rats (cf. our discussion of maze errors in rats, pp. 286-90).

H.M. was also tested on a shortened tactual maze containing five choice points. Though he did not reach criterion within 300 trials there was a steady reduction in errors in the course of this training. The results on both the shortened visual and tactual mazes suggest that H.M. can learn simple mazes, but only when there are sufficiently few choice points that

the learning can be mediated by remembering the entire series of choices.

Talland tested his amnesics on a modified form of the Rey-Davis test, which evaluates both incidental and deliberate spatial learning. This task consists in finding the loose peg among a set of pegs on a board. Successive sets are given to the subject, who must then find the loose peg in each set. When coloured pegs are used the subject can find the loose peg by attending to the colours of the pegs (a guidance hypothesis); the loose peg is always coloured differently. When coloured pegs are not used, however, the problem must be solved by noting *where* the loose peg is located. About half of the 16 Korsakoff patients were able to master this task when given colour cues; all but one failed without these cues. When they had been trained with the coloured pegs and then were presented with the same series of peg placements, but using uncoloured pegs, they performed poorly. This transfer test shows that the patients solved the coloured-peg problem through the use of the colours and had not learned about the position of the loose peg. This pattern of results is similar to that seen in hippocampal rats in mazes with and without cues at the choice points (e.g. Leaton 1969, Winocur and Breckenridge 1973).

The maze-learning data provide information relevant to the question of hemispheric specialization in the hippocampal system. For both the visual (Milner 1965) and the tactual (Corkin 1965) mazes, selective deficits were seen after right, but not left, hippocampal damage. These data are consistent with the view that the right hippocampus in humans is concerned with spatial mapping. Milner (1965) pointed out that many of her left-hippocampal patients were markedly dysphasic and certainly could not have learned the visual maze through verbal mediation; they were obviously able to learn it solely through spatial, non-verbal mediation. On the other hand, the right-hippocampal patients resorted to numerous verbal devices, but were nevertheless severely impaired. Further evidence on this point comes from studies by Corsi (1972) and Milner (1971). Subjects were required to tap the members of a spatial array of blocks in a sequence demonstrated by the experimenter immediately beforehand. This non-verbal, spatially cued task is analogous to a digit-sequence test. Corsi embedded a recurrent sequence within the series, making the task logically equivalent to the verbal recurrent-series task described by Hebb (1961). In this situation subjects typically show a learning curve for the recurrent sequence. The right-temporal group with radical hippocampal damage was deficient on this task, showing virtually no learning of the recurrent sequence. H.M. was similarly impaired. Left-temporal patients, with or without hippocampal damage, performed quite well. This picture was reversed when a verbal test was used; right temporals performed well and left temporals were impaired. Korsakoff patients performed poorly on a formally similar task (Meissner 1966). The subjects had to press buttons in a prearranged sequence; amnesics performed poorly at any sequences

beyond one or two buttons. Finally, Samuels et al. (1971) tested Korsakoff patients on the retention of visual 'scenes' involving the presence of a set of figures in a pictorial background. Performance on this task was profoundly impaired, indicating that the amnesics had trouble remembering items in a context.

In sum, Korsakoff patients, temporal lobectomy patients with right hemisphere damage, and those with bilateral damage are deficient in learning even the simplest of mazes. The pattern of responding in the maze is strikingly similar to that seen in the hippocampal rat. These data are consistent with the assumption that the right hippocampus in humans subserves spatial mapping.*

15.6. The memory defect

15.6.1. INTRODUCTION

Until recently it was felt that amnesics could retain, in the long term, nothing beyond the simplest of motor or discrimination tasks. Though some early workers (e.g. Lidz 1942) felt that the problem was more one of faulty retrieval than faulty storage, the traditional view of the amnesia consequent upon mesial temporal lobe excision held that there was a defect in the processes involved in the 'consolidation' of long-term memory (e.g. Milner 1966). In the past 10 years data have been accumulating which suggest that considerable information does enter some long-term memory system, and alternative views of the precise basis for the amnesic defect have arisen.

In principle, there are three types of models: (1) those which hold that there is some general defect in memory storage; (2) those which hold that there is a defect in retrieval processes; (3) those which hold that there is a selective defect in some aspects of storage, or in the storage of certain kinds of information. We hold the last of these positions; there is, according to our view, a selective loss of the ability to establish cognitive maps, and this leads to the loss of that form of long-term memory which utilizes spatio-temporal context for providing efficient retrieval of prior experience. Long-term storage in amnesics, on this view, depends completely upon taxon systems, which fail to provide this context and which work on principles leading to extensive interference between items which are similar in any way. This model predicts both faulty storage and faulty retrieval, characterized by abnormal interference, in much the same way that, in discussing the infra-human data, it predicts faulty place learning and abnormal persistence.

* H.M. has not been tested formally on his mapping ability; his disorientation in space is virtually complete. However, he could draw an accurate floor plan of the house he was living in (Milner et al. 1968, Corkin, personal communication) after eight years, though he could not describe his place of work or the route to and from work. Further, H.M. has recently changed residence, and is unable to draw a floor plan of his new home after 18 months experience (Milner 1976, personal communication).

15.6.2. SHORT-TERM MEMORY

As we noted in the previous chapter, there is good evidence for the existence of short-term memory systems, based on some form of threshold reduction in neural representations, in both verbal and non-verbal modes. The information available in these systems is subject to decay, which can be prevented, as we have seen, by rehearsal. Thus, in order to arrive at a measure of pure short-term memory function and its decay properties rehearsal is often prevented by distracting tasks meant to absorb the subject's attention. Assuming that such distraction does prevent rehearsal, retention observed after a certain delay (c. 20-30 s) would reflect retrieval from a long-term memory system. Thus, in normal subjects rehearsal might not have a particularly dramatic effect upon retention; long-term memory can substitute for the decayed short-term trace. However, in amnesics, who lack long-term memory systems free from strong interference effects, such distraction could have much more serious effects.

Short-term memory, as it is traditionally defined, should be intact in amnesic patients. That is, that part of retention due to retrieval from transiently activated taxon stores in intact subjects should be evident in patients lacking a locale system. Further, given an opportunity to rehearse, amnesics should be capable of maintaining information in these stores for as long as necessary. This being said, the assessment of short-term retention in amnesics, when rehearsal is prevented and interfering material is interpolated between stimulus presentation and retention, could elicit deficits; this follows from what was said above concerning the contribution of long-term memory to short-term retention. This somewhat perplexing situation results from a confusion in the psychological literature between the *processes* being studied and the *means* of studying them. Short-term and long-term memory are terms we have applied to postulated neural processes; they are not necessarily parallel to those processes reflected in retention tests based solely on manipulating retention intervals. It should not be surprising, therefore, that controversy exists in the literature concerning the status of short-term memory in amnesic patients.

It has been suggested that short-term and long-term memory can be differentiated in terms of the nature of coding imposed on information; in the case of verbal information short-term memory is supposed to involve acoustic coding, while long-term memory involves semantic coding (e.g. Baddeley and Dale 1966). The usefulness of this separation seems to break down in the study of amnesics; though their short-term memory capacity seems intact and their long-term storage defective, there are indications that they are not incapable of coding verbal items on a semantic basis. Any discrepancy here could be resolved in either, or both, of two ways: (1) there might be some intact long-term storage in amnesics which utilizes semantic-category coding; (2) there might be semantic

coding within short-term memory itself. We shall see that the former of these is almost certainly true, while the latter remains unclear.

There are two views of the status of short-term memory in amnesic patients, and considerable data which seem to support each position.* Warrington and her colleagues (e.g. Baddeley and Warrington 1970, Warrington and Baddeley 1974, Weiskrantz and Warrington 1975) suggest that amnesic patients have normal short-term memory. On the other side, Butters and his colleagues, in a series of reports (e.g. Samuels *et al.* 1971; Cermak, Butters, and Goodglass 1971, Cermak and Butters 1972, Butters and Cermak 1974, De Luca, Cermak, and Butters 1975) have indicated that there are deficits in short-term memory performance in Korsakoff patients.

For a time it seemed as though these conflicting reports might not be reconciled without assuming important differences in patient populations (cf. Butters and Cermak 1974), a most unfortunate possibility. However, a more parsimonious explanation might be possible, one which extends our understanding of the selectivity of the amnesic deficit and which makes sense of recent reports. A closer look at the results of some of these studies will bring this out. It is important at the outset to try and make some sense of the role played in short-term memory by rehearsal and distraction, as these can easily cloud the issue. With this in mind it is perhaps best to look first at the short-term memory for non-verbal items, which seems less subject to the facilitating effects of rehearsal.

15.6.2(a). Non-verbal short-term memory. Prisko (cited by Milner 1966) tested both unilateral temporal lobectomy patients and H.M. on a modified version of Konorski's delayed paired comparison test, in which two stimuli are presented successively with varying inter-stimulus intervals; the subject must state whether the stimuli were identical or not. Prisko used stimuli which were not easily amenable to verbal rehearsal, such as clicks, tones, and colours (shades), and an interpolated distraction was used on some of the trials. Patients with unilateral lesions performed normally on this task, even with delays of 60 s and interpolated distraction, except for a deficit on nonsense patterns in the right-temporal group. H.M., on the other hand, performed normally only at zero delay, and was severely impaired at the 60 s delay with or without distraction. This failure beyond 30 s is consistent with the results of Sidman *et al.* (1968), who tested H.M. on the delayed matching of ellipses and found that his behaviour was reliably under stimulus control only for 25 s or so. As Milner (1966) pointed out, it seems likely from these results that normal subjects perform well at long intervals through the mediation of some long term memory component; this is in accordance with our previous comments.

* We shall not discuss 'immediate memory' as measured by such things as digit span. This appears intact in amnesics (e.g. Drachman and Arbib 1966, and many others).

The failure of distraction to affect the pattern of results suggests that at these longer intervals rehearsal is not a useful strategy for non-verbal materials.*

In agreement with these results, Wickelgren (1968) demonstrated good short-term memory on a tonal recognition task in H.M. Finally, Warrington and Baddeley (1974) have reported normal performance on a short-term memory task involving retention of the visual location of a dot, after a filled interval, in their mixed group of amnesics. This equivalence in performance between amnesics and controls was evident only at retention intervals up to 30 s, at which time the extent of control errors reached an asymptote, while that of amnesics continued to increase. This is consistent with the notion that, in controls, retention after 30 s was mediated at least partly by a long-term memory system unavailable to the amnesics.

In contrast to the uniform results provided by these studies, work from Butters' laboratory presents a more confusing picture (cf. Samuels *et al.* 1971, Samuels, Butters, and Fedio 1972, Butters *et al.* 1973, De Luca *et al.* 1975). The first two studies, using Korsakoff patients, demonstrated profound deficits in the short-term (9, 18 s) recall of non-verbal material. However, the two subsequent studies found normal short-term retention of non-verbal material, presented visually, auditorally, or tactually, in both unilateral temporal lobectomy patients and Korsakoff patients. The final study reported deficits in retention of nonsense shapes at 20 s in Korsakoff patients. This confusion has not been clarified to date, though Butters and Cermak (1975) now seem to feel that the short-term retention of non-verbal material, in the absence of potentially interfering distraction, is relatively normal in amnesic patients, at least up to 15 s or so.

All of the reports on non-verbal short-term memory are in agreement in finding that the results are not influenced by distraction during the retention interval, though it is important to note that this distraction has usually involved an interpolated verbal task, even if sometimes presented visually. This is pertinent because De Luca *et al.* (1975) have shown that distraction only prevents good short-term retention in verbal tasks in Korsakoff patients if it is verbal, and presumably thereby interferes directly with the test material. Butters and Cermak (1975) cite a study in progress which indicates that auditory distraction might be capable of interfering with the short-term retention of an auditory non-verbal task in amnesics; this is a possibility which only future research can clarify.

The following conclusions can be drawn from this survey: (1) non-verbal short-term memory seems to decay in about 15-30 s, depending on the nature of the material; (2) verbal distraction does not interfere with nonverbal short-term memory, in agreement with recent work in normal human subjects; (3) beyond 15-30 s performance is mediated by long-term

memory systems; (4) amnesics perform normally during the time when short-term memory mediates performance, but are deficient beyond that.*

15.6.2(b). *Verbal short-term memory.* When we move to a consideration of short-term memory for verbal material, matters become even more confusing. H.M.'s verbal short-term memory is normal so long as he is not distracted, and he utilizes rehearsal to maintain his short-term memory for as long as is necessary. Once distracted, however, he loses the material completely, as well as the entire chain of rehearsal. Wickelgren (1968) tested H.M. in a short-term recognition paradigm and showed that a strength model of short-term memory (similar to the one we proposed above) fitted H.M.'s performance adequately; further, his decay functions were within the normal range. Though other temporal lobectomy patients have not been tested as exhaustively as H.M., it seems clear that much the same effects hold for them as well; when undistracted, short-term memory is normal. The fact that recall after distraction is so poor in amnesics, compared with normals, indicates again that retention in the latter can be mediated by a long-term memory component.

Baddeley and Warrington (1970) demonstrated normal short-term memory capability in a group of amnesics in two ways: (1) the amnesics showed normal recency effects in serial list learning, that is, they preferentially recalled the terminal items in the list; (2) the amnesics showed normal decay functions up to delays of 60 s in the Peterson forgetting paradigm, which involves presentation of three items and recall after varying delays. The normal forgetting curve seen on the Peterson task is surprising, as this task involves the interpolation of a distracting problem (typically backwards counting) between presentation and recall. As such, these data seem to be in disagreement with those obtained from temporal-lobe patients; as we have just seen, verbal short-term memory is seriously disrupted in these patients by distraction.** They are also in disagreement with a series of reports from Butters' laboratory (e.g. Samuels *et al.* 1971, Cermak *et al.* 1971, Goodglass and Peck 1972, Cermak and Butters 1972, Butters *et al.* 1973, De Luca *et al.* (1975) indicating that distraction during the retention interval can yield short-term memory defects in verbal tests.

The final study in this series of reports provides the most detailed information on the nature of the effect of distraction upon short-term verbal retention in amnesics. Both verbal and non-verbal distractors were used in conjunction with the verbal material, and it was seen that only, verbal distractors elicited a defect. In the absence of any distraction, amnesics retained verbal materials quite well. These data suggest that

* This was confirmed, as we shall see, in a study by De Luca *et al.* (1975).

* The demonstration that H.M. estimates time accurately up to, but not beyond, about 20 s (Richards 1973) is probably a manifestation of this accurate short-term memory system.

** Corsi (1969) has extended this result to patients with unilateral left hippocampal damage.

distractors influence short-term memory performance by pre-empting the limited processing capacity of the verbal short-term memory system, thereby preventing rehearsal. The authors argue that the distraction task used by Baddeley and Warrington, backwards counting by 2's, was not sufficiently difficult to prevent rehearsal, a point also raised by Cermak et al. (1971) and Butters and Cermak (1974).

The weight of the available data support the conclusion that verbal short-term retention, when assessed in a paradigm employing verbal distraction, is defective in amnesics. Thus, we would accept that there is probably something about the methods used by Baddeley and Warrington which could account for the discrepant results; the possibility of rehearsal in the face of the distraction they used is a good candidate. This does not mean that amnesic patients lack a verbal short-term memory system. The results of the serial-list learning experiment (Baddeley and Warrington 1970) are quite clear on this point. It does mean, however, that information in this system decays within a short period of time unless rehearsed, and that in normal subjects a long-term system can mediate retention when the short-term system has been disrupted.

15.6.3. LONG-TERM RETENTION

Notwithstanding the considerable interest shown in short-term memory function in amnesics, it is clear that the primary focus of interest in this syndrome is in long-term memory. Most theoretical treatments have stressed this function, and we shall discuss the problem at some length. Three major theoretical positions have emerged in recent years, as we noted above. More specifically, these suggest that (1) there is a failure in the transfer of information from a short-term memory system to a long-term system (e.g. Milner 1966), (2) there is a failure in the selective forgetting of unwanted information, leading to abnormal interference during retrieval (e.g. Warrington and Weiskrantz 1971, Weiskrantz and Warrington 1975), and (3) there is a deficit in the spontaneous encoding of inputs along semantic, or other complex, lines, leading to poorer recall and increased interference from more simply coded memories (e.g. Butters and Cermak 1975).

At the present time there is sufficient evidence to disqualify all of these models, though the phenomena they purport to explain remain to be accounted for. In what follows we hope to show that a selective deficit in the storage of cognitive maps and the concomitant dependence upon interference-prone taxon memory systems can account for the known facts of the amnesic's long-term memory defect. We shall be concentrating here upon the long-term memory for verbal information, as the bulk of the data pertain to this. Our discussion of maze learning and spatial orientation has already demonstrated that the long-term storage of spatial maps is probably defective in amnesics; an analysis of verbal learning will raise the possibility

of an analogous defect in the formation and storage of semantic maps.

In addition to reviewing data from studies of new learning, we shall look briefly at the way in which previously stored information is affected by the onset of amnesia, that is, the retrograde aspect of the disorder. Here, we shall show that old memories incorporated in semantic maps are also disrupted in amnesics, regardless of the time of storage. We shall be stressing the selectivity of the defect in terms of the form of memory affected, while at the same time attempting to show that this selective defect holds for both retrograde and anterograde deficits, and regardless of the form of retention test employed. Finally, we shall discuss the way in which abnormal interference effects result from the absence of a memory storage system designed to limit interference, in accordance with our previous discussion of the properties of imagery and cognitive mapping.

15.6.3(a). Basic effects. The long-term retention (beyond 60 s) of verbal material has been shown to be impaired in amnesics in a host of studies. In a way, the really interesting studies are those which define circumstances under which some long-term memory can be displayed by amnesics. The first indication that information was indeed getting into some kind of verbal long-term memory store came from several studies showing that the provision of information at the time of retrieval could improve the performance of amnesics (Warrington and Weiskrantz 1968). Such findings invalidated any theory of a general defect in the consolidation of information from short-term to long-term memory. It has now been shown, in a number of ways, that the provision of cues at the time of retention markedly improves the performance of amnesic patients.

The importance of these retrieval cues in the performance of subjects at retention is intuitively sensible; efficient recall of any specific bit of information from the welter of stored experiences requires a highly selective means of retrieving that information. Tulving and Pearlstone (1968) have emphasized this fact by drawing a distinction between the *availability* and *accessibility* of stored information, that which is available is not necessarily accessible. One of the functions of the cognitive-mapping system is to provide the basis for effective access to memory by imposing upon the latter a spatio-temporal framework which makes the identification of information simpler. Of course, certain forms of information 'emerge' from the cognitive map itself and are totally dependent upon it. Thus, knowledge of places requires the map, as does knowledge for the specific context within which events occurred. No manner of retrieval cue should enable the amnesic to improve the recall of such information; the facts are simply missing.

However, retrieval cues can aid the amnesic in attempts to get at information stored in the taxon systems, information which might be interfered with in the absence of such cues. Faulty retrieval derives from,

in part, the category-based storage properties of the taxon systems. Retrieval cues act to circumvent these properties by specifying the information required. Thus, we are suggesting that the amnesic deficit includes both faulty storage (no maps) and faulty retrieval (no context).

15.6.3(b). *Faulty retrieval.* The poor retrieval of amnesic patients is generally characterized by abnormally large interference effects from information stored at other times.* This has been shown in a variety of situations in highly specific ways. Thus, repeated testing on word lists yields considerable intrusions from one list to another (cf. Weiskrantz and Warrington 1975 for a review of these interference effects), and interference itself can be shown to depend upon the similarity between items in different lists (cf. Butters and Cermak 1975 for a review of relevant studies). To put it in simple terms, the amnesic patient fails to provide outputs which are appropriate to the context required.

Retrieval cues, be they in the nature of partial information (Warrington and Weiskrantz 1968, 1970) or reminders of the categories to which words belonged, serve to reduce markedly the number of potential response alternatives. Thus, they reduce the possibility of responding with items which are contextually inappropriate. This effect holds for both recall and recognition testing. Recognition tests, as we noted earlier, are typically stronger tests of contextual memory than are recall tests. Proper recognition, in most experiments, is based not on some simple familiarity process, but rather on the knowledge that a certain (already familiar) item, or pattern, occurred in a particular experimental context. It has been known for some time that amnesic patients are particularly defective at recognition tests, and we would assert that this reflects the dependence of recognition on contextual memory. At the same time, however, it has been noted anecdotally that amnesic patients can often recognize faces as familiar or not, even though they cannot specify where or when the person in question was seen. This suggests that if recognition memory was assessed with materials which were truly unfamiliar, thereby eliminating any dependence upon contextual information, it might prove relatively normal in amnesic patients. Recent work confirms this prediction (Piercy and Huppert 1972, Huppert and Piercy 1976). When the recognition of familiar, high-frequency words was tested the standard deficit appeared. However, when the recognition of unfamiliar, low-frequency words was tested recognition memory was substantially improved, though still slightly inferior to normal performance.**

In free-recall tests a wide variety of retrieval cues have been shown to

* These interference effects show up in a variety of situations, including those involving hypothesis behaviour (e.g. Oscar-Berman 1973).

** These data indicate that, contrary to Gaffan's (1972, 1974) claim, familiarity detection is intact in amnesics, at least when spatio-temporal context is unimportant to the familiarity or unfamiliarity of the test material.

facilitate the performance of amnesics. That this effect works through improving retrieval of otherwise inaccessible material, rather than through improving storage, was shown by Cermak (1975). He found that cues available only at the time of stimulus presentation did not facilitate retrieval. Such cues must be present during the retention test in order to be useful. This result has been confirmed by Kinsbourne and Wood (1975). Much of the work on the use of retrieval cues has been aimed at uncovering a presumed defect in semantic encoding by amnesics, a proposal forwarded by Butters, Cermak, and their co-workers (cf. Butters and Cermak 1975).* While this work has suggested that amnesic patients encode less information about the semantic properties of words, it has not gone beyond that to an explanation of why this faulty semantic encoding should lead to the variety of defects seen in amnesic patients. This defect is limited to the *spontaneous* use of semantic encoding (Cermak, Butters, and Gerrein 1973); amnesics *can* encode semantic properties of words when directed to do so. Thus, the defect might be related to the short-term memory deficit reported by this group of investigators; in the above study, semantic information seemed to be spontaneously available to amnesics for at least a short period of time. Further, the demonstration that cues given at stimulus presentation do not, by themselves, facilitate recall indicates that the improvement seen with cues given at retrieval lies in their helping the subject gain access to stored material. Semantic retrieval cues could not be effective unless information was stored on the basis of semantic properties in the first instance.

The facilitating effects of cued recall can be mimicked by the use of memory tests which benefit subjects able to use any form of similarity between test items. Thus, lists in which words from the same category are clustered together elicit improvements in amnesics (Baddeley and Warrington 1973), while paired associates invoking phonetic or semantic relations between the stimulus and response items yield normal performance in amnesics (Winocur and Weiskrantz 1976).

The performance of amnesics during cued recall is consistent with the notion that excessive interference is at least partially responsible for the amnesic deficit, as Warrington and her co-workers have suggested. However, there is little evidence to support their contention that this interference owes to the mechanism they propose, a loss of selective forgetting, whatever that might actually mean. This position cannot predict the selective loss of any storage facility and, as we shall see, there is accumulating evidence that such a selective defect is central to the amnesic syndrome.

One form of retrieval aid which does not seem to facilitate amnesic

* These authors are talking about the semantic properties of single words, which is something quite distinct from what psycholinguists mean when they use the term or what Tulving (1972) meant by the same term.

performance concerns the use of imagery techniques (e.g. Baddeley and Warrington 1973). This suggests that there are forms of storage, facilitated in intact subjects by imagery instructions, which are unavailable to amnesic patients; we shall pursue this point shortly.

To sum up, the use of cued recall, or recognition tests with unfamiliar materials, indicates that certain forms of long-term verbal memory can be vastly improved in amnesic patients. This improvement seems to reside in the provision of a way to use available category memory without invoking the strong interference effects which typically plague memory retrieval from these systems. There is no indication in this work that amnesic patients can use anything other than material-specific, or category, cues, depending for their efficacy upon identity with, association with, or similarity to the to-be-recalled information. None the less, such cues can be quite effective in helping the amnesic in many clinical tests; that these tests have generally concentrated upon the retention of individual items, or clusters of categorially related items, is surely of some significance.

15.6.3(c). Faulty storage. As we have just seen, the use of cued recall tests indicates that amnesics can store information about the categorial properties of much verbal information. However, these studies do not directly test the ability of the subject to store contextual memory.

Some evidence pertinent to this question comes from studies exploring the effects of the use of imagery upon verbal learning in amnesics. With the exception of the study by Cermak (1975), the use of imagery totally fails to improve the performance of amnesic patients with bilateral damage (Jones 1974, Baddeley and Warrington 1973).* As expected, patients with unilateral damage in the left hemisphere, who show profound verbal memory defects (cf. Corsi 1972), are benefited by the use of the imagery techniques (Patten 1972a, Jones 1974). These data indicate that the hippocampus is crucial to the facilitating effects of imagery.**

Other evidence pertinent to the question of faulty storage comes from the study of the retrograde effects of hippocampal damage. Initial thinking on the nature of these effects suggested that retrograde loss was most marked in the period just prior to the operation, with a relative sparing of older memories (e.g. Milner 1966). The development of a more precise method for the assessment of 'old' memories, the long-term questionnaire, provided evidence against this assumption by showing that there was considerable memory loss at all times tested, including periods 30 and 40 years prior to the onset of hippocampal dysfunction (Sanders and Warrington 1971).

* Cermak showed that amnesics were facilitated by instructions to tie two words together; Baddeley and Warrington showed that amnesics could not benefit from imaging four words together.

** Amnesic patients had no trouble forming an image, e.g. a picture, but they could not store information which would enable them to retrieve that image. This is further evidence that imagery works, not through the storage of a picture, but rather through the storage of some set of instructions (or deep structure) that enables the subject to recreate the image when needed.

More recent work (Marslen-Wilson and Teuber 1975, Sanders and Warrington 1975), which combined the long-term questionnaire technique with cued retention, showed that much of the old material thought to have been lost was actually available, though inaccessible without cueing. Though amnesics required more cues than did controls, they still showed a vast improvement in performance. These data, then, in common with the data from studies of cued recall in recent learning, indicate that amnesics have trouble getting at stored information. However, Kinsbourne and Wood (1975) have provided evidence that the deficit in retention of old memories goes beyond a loss of access. Amnesics were asked to produce a personal memory in response to high-imagery, high frequency words. Whereas control subjects produce highly specific memories in this situation, amnesics

'do not retrieve individual events at all. They characteristically, instead, and contrary to instruction, retrieve categorial information about the appropriate use or general availability of the object represented by the stimulus word' (p. 278).

The authors concluded that their amnesics could retrieve considerable general information from their early life, but could not retrieve specific event memories at all. They suggested that there is a selective failure in what Tulving (1972) referred to as episodic memory and what we have referred to as context-dependent locale memory. A similar suggestion was made by Gardner *et al.* (1973) and by Huppert and Piercy (1976).

Unfortunately, there is little direct evidence at present which would address the question of the role of the hippocampus in semantic maps, as we defined them above.* Lackner (1974) has shown that H.M. can detect certain linguistic ambiguities, but at a markedly reduced rate. We know from the work of Talland (1965) and others (e.g. Murray and Hitchcock 1973) that Korsakoff patients are quite poor at the recall of narrative, but this could be attributable to many things. Future research will have to elucidate the precise nature of any hippocampal role in context-specific memory. Present evidence suffices to assert that there is, in amnesics, a selective deficit in the establishment and long-term storage of event memory.

15.6.3(d). Conclusions. The study of the long-term aspects of memory loss in amnesic patients reveals several basic features to the syndrome.

- (1) There appears to be a selective loss in the storage of event memory, or memory which is specified as to spatio-temporal context.
- (2) There is relatively normal storage of category memory, up to and

* Glowinski (1973) has shown that temporal-lobe epilepsy patients are markedly impaired at the recall of narrative texts, even when tested immediately. One can think of many tests that would get at the question of the role of the hippocampus in deep structure; the recent work of Bransford and McCarrell (1974) provides many examples of tests that should help to pinpoint this function.

including categories based on the semantic features of individual items.

- (3) Retrieval from these category stores is considerably retarded in amnesics because of the powerful interference effects acting between items of a similar nature. Normal subjects can circumvent this difficulty through the use of context-specific memories, which are largely free of interference problems.
- (4) The provision of retrieval aids, at the time of retention, helps amnesics to gain access to the information available in their taxon category stores. It cannot, however, substitute for the lost event-specific memory system. Such memories are neither accessible nor available to the amnesic.
- (5) This loss of event-specific memory seems to include those memories formed before the onset of the disease or the surgical intervention.

15.6.4. OVERALL CONCLUSIONS

This review of the amnesic literature confirms that a theory postulating cognitive-mapping deficits after human hippocampal damage provides a decent fit to the available data. Clear-cut evidence for defects in exploration and spatial mapping is available, and there seems little doubt that the right hippocampus is crucial to these functions. Evidence for defects in semantic mapping and the use of imagery techniques is also available, while the assumption of a selective storage defect makes sense of the concomitant retrieval defect, expressed as abnormal interference, that is seen in amnesics. In virtually every way the effects of damage to the hippocampus in humans seem analogous to those seen in infra-humans.

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