PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

# Implications of Neuropsychological Evidence for Theories of Normal Memory

A. D. Baddeley

*Phil. Trans. R. Soc. Lond. B* 1982 **298**, 59-72
doi: 10.1098/rstb.1982.0072

*Phil. Trans. R. Soc. Lond.* B **298**, 59–72 (1982) 
*Printed in Great Britain*

# Implications of neuropsychological evidence for theories of normal memory

By A. D. Baddeley
*M.R.C. Applied Psychology Unit, 15 Chaucer Road, Cambridge CB2 2EF, U.K.*

The relation between neuropsychology and the study of normal cognitive function is discussed in the context of recent research on human memory. It is suggested that neuropsychological evidence has clear implications for the fractionation of human memory into subsystems. The distinction between long-term and short-term memory, between semantic and episodic memory, and the further fractionation of short-term or working memory all offer examples of concepts that have been successfully applied within the neuropsychological domain, and where the neuropsychological evidence has led to a modification and development of the original concept. Attempts to offer a cognitive interpretation of the amnesic syndrome are discussed. While none of these is entirely satisfactory, such work has led to a potentially important distinction between autobiographical memory or recollection, which is defective in amnesic patients, and a more perceptual or procedural learning process, which appears to be intact in such patients. Recent research on normal subjects is beginning to reveal a similar distinction. It is concluded that the relation between neuropsychology and the study of normal cognitive function continues to be an extremely fruitful one.

## Introduction

The last decade has seen a steady growth in the influence on neuropsychology of the techniques and concepts of cognitive psychology, and conversely of the influence of neuropsychological evidence on cognitive theory. How fruitful is this relation? In a recent review of research on the human amnesic syndrome, Meudell & Mayes (1981) suggest that 'contemporary neuropsychologists, including those who study amnesia, are parasitic upon cognitive psychologists who study intact humans'. They go on to refer to 'the suspicion that cognitive psychology is not a good source of powerful functional explanatory notions' and suggest that research workers should be 'more prepared to look for theoretical ideas in other directions'.

While this is not, I believe, a very widespread view, coming as it does from two investigators who have carried out some of the more careful and systematic neuropsychological work on amnesia in recent years, I think it deserves to be taken seriously. I shall argue that all three assumptions are erroneous, that the relation between the study of normal cognitive function and neuropsychology is symbiotic rather than parasitic, that this approach is making substantial progress, and finally that while ideas from any source are to be welcomed, a neuropsychology that does not relate to normal cognitive psychology will be at best incomplete.

Consider first the charge that neuropsychology is parasitic on normal cognitive psychology. The term 'parasitic' implies that the parasite derives its nourishment at the expense of the host, who receives no benefit in return and indeed may ultimately be harmed or killed by the parasite. I shall argue that, far from being a drain on the resources of cognitive psychology, neuropsychology is an invaluable source of stimulation, that it represents a chance of testing the ideas of cognitive psychology that has both revealed limitations and suggested new developments. To argue that the breakdown of cognitive function should not be studied in the context

of normal functioning seems to me perverse in the extreme. Similarly, as a cognitive psychologist I regard the way in which normal function breaks down in the brain-damaged patient as providing an invaluable source of evidence regarding the organization and functioning of normal cognition.

It is, however, possible to argue that given the present state of development of neuropsychology and cognitive psychology, any attempt to relate them is likely to be misleading. I shall argue against this view, using examples from research on the fractionation of memory systems, and on the amnesic syndrome. I shall argue that in each of these cases, the concepts and techniques of cognitive psychology have thrown light on neuropsychological problems, which in turn have enriched our understanding of normal cognition. Both issues are complex and a detailed analysis is beyond the scope of this paper. However, Shallice (1979) provides an excellent review of the fractionation of memory systems, while work on the amnesic syndrome is reviewed by Baddeley (1982) and Meudell & Mayes (1982).

## The fractionation of memory systems

Perhaps the strongest influence of neuropsychological evidence on theories of normal memory occurs in connection with the question of whether human memory is best conceptualized as a single entity, or should be fractionated into a number of separate subsystems. This has been a controversial topic within experimental psychology for at least 20 years. The area continues to be controversial, but I would argue that there is general agreement about most of the functional distinctions between separate aspects of memory. Controversy remains as to whether such functional differences should be regarded as reflecting a modular structure with different aspects of memory based on different storage systems, or whether they are best conceptualized in terms of different types of coding within a single complex general memory system.

Neuropsychological evidence bears on this central controversy in two ways. First, it has played an important role in establishing the functional separability of different aspects of memory. Regardless of the particular theoretical language chosen to express such diversity, this does represent a major development in our conception of human memory. Secondly, neuropsychological evidence bears heavily on the question of whether systems should be regarded as modular or general. Indeed, one might argue that neuropsychological data provide virtually the only truly convincing evidence on this point. The existence of a functional distinction between two aspects of memory such as short-term memory and long-term memory can in principle be equally well handled by a model assuming separate modular systems, or separable aspects of a single general system. However, if a specific lesion clearly disrupts one type of memory, leaving the other intact, this strongly suggests a degree of modularity. It need not imply anatomically separable memory *stores*, but it certainly suggests separable modular processes.

Work on the fractionation of memory systems has been concerned with four major distinctions. The first of these, the distinction between visual and verbal memory, is discussed by De Renzi (this symposium) and will therefore not be further considered here. The other three concern first the distinction between long-term and short-term memory, secondly the question of whether short-term memory itself should be split into subcomponents, and thirdly the question of whether long-term memory can be usefully separated into two aspects, semantic and episodic. I shall consider these in turn.


PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

*Long-term and short-term memory*

The suggestion that human memory might comprise two separate systems, a temporary short-term storage system and a more permanent long-term memory, goes back at least to the 1940s (Hebb 1949). During the 1950s the issue remained relatively uncontroversial, simply because there was virtually no interaction between research on long-term memory (l.t.m.) and short-term memory (s.t.m.). Work on l.t.m. at that time was largely atheoretical, concerned with the learning of lists of unrelated words or nonsense syllables under highly constrained conditions and primarily restricted to North America (see, for example, Underwood & Schulz 1960). Work on s.t.m. was heavily influenced by Information Theory, had strong links with applied research and with much of the work being carried out in Britain (Broadbent 1958).

The question of whether it is in fact necessary to assume separate s.t.m. and l.t.m. systems was raised by Melton (1963), who proposed an interpretation of the phenomena of short-term memory in terms of the Associationist Interference Theory that was dominant at that time in the study of long-term memory. Melton's paper stimulated a good deal of work on the question of whether or not a distinction was necessary. The evidence supporting such a distinction included: (1) the observation that certain tasks appeared to have two separate components that behaved very differently, one being a durable long-term component and the other a transient short-term or primary memory component (Waugh & Norman 1965; Glanzer & Cunitz 1966; Peterson 1966), (2) the claim that short-term memory tended to rely heavily on speech coding whereas long-term memory depended primarily on meaning (Conrad 1964; Baddeley 1966*a*, *b*), and (3) evidence from neuropsychological studies, and in particular from Milner's research on the amnesic patient H.M., who suffered from a bilateral lesion of the temporal lobes and hippocampus.

H.M. showed relatively normal short-term memory as measured by digit span, coupled with disastrously bad long-term learning ability (Milner 1966). A similar pattern of results is shown by a range of amnesic patients who typically are unable to learn new material ranging from words to faces and from stories to geographical routes, whether performance is tested by recall or recognition (Baddeley 1982). The amnesic syndrome is typically associated with damage to the temporal lobes, hippocampus or mammillary bodies. Common causes include the after-effects of alcoholism (Korsakoff's Syndrome) and encephalitis.

A study by Baddeley & Warrington (1970) attempted to relate the neuropsychological evidence to developments in the study of normal memory by testing amnesic patients on a series of tasks that were assumed to have separable long-term and short-term components. A typical example of such tasks is free recall, in which the subject is presented with a sequence of unrelated words, and asked to recall them in any order he or she might choose. The performance of normal subjects on this task shows a very clear tendency for the last few items presented to be very well recalled, the recency effect. However, if recall is delayed for 20 or 30 s during which the subject is occupied with another task, the recency effect disappears, leaving performance on the last few items no better than performance on items from the middle of the list, which are relatively unaffected by the delay. The recency effect was therefore assumed to represent the function of a short-term memory system, while performance on earlier items was assumed to rely on long-term memory. If amnesic patients are assumed to have normal short-term memory but defective long-term memory, their free recall performance should show normal recency coupled with impaired performance on earlier items in the list. This is exactly what we observed.

We studied s.t.m. by using a number of tasks, including that devised by Peterson & Peterson

(1959) in which the subject is presented with a short sequence of items (e.g. three unrelated words) and required to retain them over an interval of 0–60 s. During this delay, rehearsal is prevented, usually by a counting task. Our amnesic patients showed normal performance on this test, implying normal s.t.m. It is perhaps worth noting that some amnesics do show defective performance on this task (see, for example, Butters & Cermak 1975), but this is almost certainly associated with a more general cognitive impairment (see Baddeley 1982; Warrington 1982). We examined a total of six experimental tasks: in all cases we found evidence for unimpaired s.t.m. in our amnesic patients that showed the expected dramatic impairment in l.t.m. performance.

The case for a distinction between long-term and short-term memory was further strengthened by evidence from a second type of patient, one who appeared to have normal l.t.m. but defective s.t.m. Shallice & Warrington (1970) studied the performance of a patient, K.F., whose ability to repeat back strings of digits was limited to one or two items. His s.t.m. as measured by performance on the previously described Peterson task or in terms of the recency effect in free recall was grossly impaired, but despite this his long-term learning ability was quite normal (Warrington & Shallice 1969). Similar patients were subsequently reported by Warrington *et al.* (1971) and by Saffran & Marin (1975). The occurrence of a double dissociation between l.t.m. and s.t.m. performance, with some patients being intact on l.t.m. but impaired on s.t.m. while others showed the reverse, argues strongly for a separation of these two functions (see Shallice (1979) for a more detailed analysis).

### *The fractionation of short-term memory*

While the work of Shallice & Warrington argued strongly for a separation between l.t.m. and s.t.m., it also posed some problems for the model of s.t.m. that was most popular at the time, that of Atkinson & Shiffrin (1968). This assumed that memory involves a linear sequence of memory stores, with a set of brief sensory memory systems feeding on to s.t.m., which in turn feeds information into l.t.m. On such a model, one might expect that a grossly defective s.t.m. would be incapable of supporting normal long-term learning. And yet, neither K.F. nor subsequent patients proved to have any substantial learning difficulty. Indeed, apart from problems in comprehending certain syntactically complex sentences, their general cognitive abilities appeared to be remarkably unaffected by their gross defect in short-term memory performance. This suggests either that s.t.m. is not necessary for long-term learning, or else that their impaired memory span reflects a defect in only one component of a more complex s.t.m. system. The observation that such patients are much less impaired in their immediate memory for visually presented material than they are for spoken material (Warrington & Shallice 1969, 1972) argued for a further fractionation of short-term memory into separate auditory and visual components.

Atkinson & Shiffrin (1968) assumed that s.t.m. acts as a working memory, which plays a crucial role not only in learning, but also in other tasks such as reasoning and language comprehension. The comparatively unimpaired cognitive performance of K.F. clearly cast doubt on this view. In order to explore this further, Baddeley & Hitch (1974) required normal subjects to carry out a series of tasks while at the same time rehearsing aloud a sequence of up to six random digits. This could be regarded as producing in normal subjects a situation analogous to that experienced by K.F. Whereas K.F. has a grossly impaired s.t.m., the normal subject is given a task that will use up most of the capacity of his s.t.m., hence presumably impairing its use for other tasks such as comprehension and learning.


PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

The results indicated clear but by no means massive impairments in performance, and induced Baddeley & Hitch (1974) to propose a model of working memory that comprised a controlling central executive system aided by a number of subsidiary slave systems. One of these, termed the Articulatory Loop, was assumed to be based on subvocal speech and to be responsible for the speech-based characteristics of short-term memory. In general, this model was able to handle virtually all the data from studies of normal s.t.m. without recourse to a visual store capable of retaining sequences of digits, such as that postulated by Shallice & Warrington. It accounted for data from patients such as K.F. by assuming an impairment in the operation of the Articulatory Loop. However, while this was reasonably plausible for patients who had some evidence of aphasia, as occurs with most subjects with impaired short-term memory, problems were raised by a study in which Shallice & Butterworth (1977) observed in detail the speech of one patient, J.B., who had grossly impaired digit span. She showed apparently normal fluent speech, a result that does not support the view that her s.t.m. deficit is based on an impaired articulatory system.

A second problem for the initial working memory view was raised by the fact that visually presented items were recalled significantly better by patients with impaired s.t.m. than those presented auditorily. The absence of the normal auditory advantage is not problematic for an articulatory interpretation, but the positive enhancement of visual presentation does raise problems. Such problems could in principle be handled in terms of a second slave system that had been postulated to account for the use of spatial imagery in memory, termed the Visuo-spatial Scratch Pad (Baddeley & Lieberman 1980). However, such a system is primarily concerned with recalling a single complex spatial array rather than a sequence of separate items such as are involved in the serial recall of visually presented digits. The position was therefore one wherein the neuropsychological data suggested that the current working memory model might be oversimplified, but were not in themselves sufficiently strong to force a modification of the model.

At this point I began a series of experiments with a colleague from Strasbourg, Pierre Salamé, who was visiting on a Royal Society C.N.R.S. exchange. We were investigating the effects of unattended noise on memory, and carried out a study in which, instead of the normal noise stressor used in this area, namely white noise, we studied the effects of unattended speech. Our subjects were presented with a sequence of digits that appeared on a cathode ray tube at a rate of one every 0.75 s. Under control conditions, the subject simply read the digits and then tried to recall them in the appropriate order. There were two experimental conditions, one in which each digit was accompanied by a spoken word which the subject was instructed to ignore, and a second in which spoken nonsense syllables accompanied each visual digit, again with instructions to ignore. There was a very clear impairment in performance, regardless of whether the unattended spoken material was nonsense syllables or words.

Later experiments showed that the crucial factor in causing disruption was the similarity in sound between the digits to be remembered and the auditory disrupting material. We further observed that preventing the subject from verbally rehearsing the visually presented digits, by requiring him during presentation to repeat an irrelevant word such as 'the', abolished the effect of unattended speech on performance (Salamé & Baddeley 1982).

A simple way of accounting for our results is to assume two separate s.t.m. systems, both of which are capable of holding digit sequences. One system is phonologically based; material gains access to this system via speech. The subject may use this system to help him remember sequences of visually presented digits if he speaks the digits, even subvocally. However, he


PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

cannot shut out irrelevant auditory material, and if this is at all phonologically similar to the material he is trying to remember, performance will be impaired. If the subject is required to utter a continuous stream of irrelevant speech, he will not be able to encode the visually presented digits phonologically. The digits will therefore not gain access to the phonological store, and hence performance will not be influenced by the irrelevant spoken material, which gains obligatory access to this store (Salamé & Baddeley 1982).

Our data have therefore led us to assume two separate short-term stores, a position very similar to that suggested by the data from patients with defects of s.t.m. We do, however, remain to be convinced that the memory system accessed by visual presentation does in fact store information in a visual form, rather than using some more abstract code. The evidence from Warrington & Shallice (1972) is suggestive but not convincing. We are at present undertaking a series of experiments on normal subjects to explore this point further. Whatever the outcome of these experiments, there is no doubt that neuropsychological work on patients with defective s.t.m. has in recent years been leading, rather than merely following current developments in the understanding of short-term and working memory.

### *Semantic and episodic memory*

Tulving (1972) suggested that we should distinguish two aspects of l.t.m. namely semantic and episodic memory. Semantic memory is concerned with our store of knowledge about the world; it enables us to answer questions about the meaning of words, or facts about the world, such as 'What is the capital of France?' or 'What is the chemical formula for salt?' It may be distinguished from episodic memory, which is concerned with memory for specific personally experienced incidents. Remembering what you had for breakfast or an encounter with someone on holiday are examples of episodic memory. Remembering which words have just been presented to you in an experiment also relies primarily on episodic memory, although semantic factors involved in understanding words would also clearly play a role in performing the task.

While this distinction has generated a good deal of interest within cognitive psychology, it has so far led to relatively little research. In my own view, the strongest evidence in support of such a distinction comes from neuropsychology, where a number of authors have suggested that the classic amnesic syndrome represents a defect of episodic memory coupled with unimpaired semantic memory (e.g. Kinsbourne & Wood 1975).

There is of course abundant evidence for defective episodic memory in amnesic patients, involving as it does grossly impaired memory for words, pictures, faces or incidents (Baddeley 1982). Evidence for normal semantic memory is slightly less plentiful. However, Talland (1965) reports normal vocabulary in amnesic patients who may be unimpaired in generating instances from semantic categories such as animals or fruit (Baddeley & Warrington 1973). They may also show normal speed and accuracy in performing the classic semantic memory task of verifying statements about the world, such as 'Canaries have wings' (Baddeley 1982). In conclusion, it appears that a patient may be grossly amnesic and yet may show normal semantic memory.

Further neuropsychological evidence in favour of the semantic–episodic distinction comes from Warrington (1975), who describes three patients who appear to show selective impairment of semantic memory. Such patients show a defective knowledge of objects or pictures of objects, which is not due to general perceptual or intellectual deterioration. As a number of models of semantic memory would suggest, general information about a concept (whether, for example, a


PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

picture represents an animal or not) is more resistant to impairment than more detailed information (whether the animal is dangerous or not). Such patients do show more general learning impairments, as indeed might be expected in view of the importance of semantic coding in long-term learning. Their memory impairment, however, is by no means as disruptive as that found in the classic amnesic syndrome. They may be well oriented in place and time, and able in conversation to refer back and forth to important events in their lives in a way that simply does not occur in the classic amnesic syndrome (Warrington 1975).

Although the distinction between semantic and episodic memory has evoked a good deal of discussion, it has produced relatively little research in normal cognitive psychology. That, together with a comparative rareness of patients with specific semantic deficits has, I believe, limited the development of the concept. However, there is evidence for a renewed interest in this distinction, stimulated at least in part by neuropsychological data from studies of the amnesic syndrome.

## The amnesic syndrome

Over the past decade the analysis of the amnesic syndrome has represented one of the most active areas of application of cognitive psychology to brain-damaged patients. As discussed earlier, the amnesic syndrome is reflected primarily in defective long-term episodic memory. Since it can in certain carefully selected patients occur as a very dense but pure amnesia it represents an important challenge to our theories of normal memory. A detailed account of theoretical research on amnesia is beyond the scope of the present paper; more extensive accounts are given both by Baddeley (1982) and by Meudell & Mayes (1982). What follows is a summary of the current position.

Explanations of the amnesic deficit can be divided into three broad categories, those implying the inadequate input or encoding of information, those implying a defect of storage, and those assuming that the amnesia reflects a retrieval deficit. Research on normal memory over the last decade had been heavily influenced by the concept of Levels of Processing (Craik & Lockhart 1972). This argues that the durability of a memory trace will depend on the way in which the material to be remembered was processed during learning. I could for instance present the word *dog*, require the subject to process it in a specified way, and subsequently ask him to recall it. If processing was concerned only with superficial characteristics of the material, such as the type fount in which the word was printed, subsequent recall would be poor. Somewhat deeper processing, for example by asking 'Does the word rhyme with *log*?' will lead to better recall, while a question requiring semantic coding, for example 'Does the word refer to an animal?' will lead to the best long-term retention. This line of research suggested the hypothesis that amnesic patients may show poor learning because of inadequate initial processing of material to be remembered. Such a view has been advocated by Cermak & Butters in a range of papers (e.g. Butters & Cermak 1975; Cermak *et al.* 1973).

Convincing evidence occurs that some alcoholic (Korsakoff Syndrome) patients are both grossly amnesic and also exhibit impoverished encoding strategies, tending not to encode material as deeply as their non-amnesic controls. Subsequent evidence, however, suggests that this population of patients does in fact show two separable deficits, a general intellectual blunting that is revealed in their impaired encoding of new material, together with a separate global amnesia. Such a view is supported both by the occurrence of patients who show encoding deficits without global amnesia (Moscovitch 1982) and, most importantly, by patients who are




PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

densely amnesic but show no evidence of either general intellectual impairment or impoverished encoding (Baddeley & Warrington 1970; Cermak 1976; Mayes *et al.* 1980).

In an effort to test the encoding hypothesis directly, a number of investigators have induced their amnesic patients to encode material deeply and richly. An encoding deficit hypothesis would predict that such a manipulation would affect the amnesic patients differentially. The exact nature of the differential effect depends on the particular encoding hypothesis proposed. If it is suggested that the memory of amnesic patients is defective because they do not *spontaneously* use appropriate strategies, then inducing them to encode in an appropriate manner should enhance their performance substantially more than that of control patients, who are assumed to be already using deeper encoding strategies. A second version of the encoding hypothesis, however, might suggest that the problem lies not in selecting a strategy, but on the effect of that strategy on long-term learning. This would assume that amnesic patients are poor learners, not because they fail to use deep encoding strategies, but rather because such encoding does not help them in the same way as it helps normal subjects. Such a hypothesis would predict that manipulation of encoding strategy would have *less* effect on amnesics than it has on normals.

Comparisons between amnesic and normal patients are of course complicated by substantial differences in overall level of performance. However, there are ways of coping with this problem, and when this is done the evidence suggests that amnesic patients and normals respond to the manipulation of encoding strategies in exactly comparable ways. Although some amnesic patients may show little enterprise in employing learning strategies, this is by no means always so, while the effect of such strategies appears to be qualitatively the same in amnesics and normal subjects (Baddeley 1982; Cermak & Reale 1978; Meudell & Mayes 1982).

A second interpretation of the amnesia deficit is in terms of memory storage or consolidation (see, for example, Milner 1966). The consolidation hypothesis is usually interpreted as suggesting that the memory trace is established but fails to stabilize, leading to rapid forgetting. Such a view makes the simple prediction that amnesic patients should forget faster than control patients. The evidence on this point appears to be clear: patients may be grossly amnesic and yet show normal forgetting both in short-term memory (see, for example, Baddeley & Warrington 1970; Warrington 1982) and in long-term memory (Huppert & Piercy 1978). Comparing rates of long-term forgetting is complicated by the problem of equating levels of initial learning in amnesic and control patients. This can be done either by giving the amnesic patients a greater amount of learning time, by giving them fewer items to learn, or by testing initial performance after a shorter delay. All three methods of equating learning produce the same result, that there is no substantial difference in rate of forgetting between amnesic and control patients.

In an otherwise thoughtful and carefully argued review, Meudell & Mayes (1982) attempt to preserve a consolidation hypothesis of this type by arguing that attempts to equate initial level of learning may be masking genuine forgetting differences. Such a view seems implausible for two reasons, first because it is extremely difficult to imagine a forgetting model in which differential rates of forgetting could be masked in this way, and secondly because even when initial level is not equated, amnesic patients do not appear to show faster forgetting (Brooks & Baddeley 1976).

While a consolidation interpretation of the amnesic syndrome is normally assumed to imply a less stable trace and faster forgetting, other possibilities exist. However, until such hypotheses


PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

have been developed and their behavioural implications spelled out, the concept of consolidation must remain a physiological assumption that could be associated with any of a wide range of psychological accounts of the amnesic syndrome.

If amnesic patients encode information normally and show normal forgetting rates, then the most plausible source of deficit might seem to be in the process of memory retrieval. A number of retrieval hypotheses have been proposed, and it is probably true to say that none of them can confidently be ruled out. Warrington & Weiskrantz (1970) have suggested that amnesia may result from an excessive sensitivity to interference from earlier learning. On this view, the amnesic patient has difficulty in accessing the appropriate memory trace because of his inability to deal with other irrelevant traces. An alternative retrieval theory by Gaffan (1974) suggests that amnesic patients may be defective in their ability to evaluate the familiarity of a previously experienced item. A third hypothesis, advocated by Winocur & Kinsbourne (1978) and Huppert & Piercy (1978), suggests that amnesics are particularly bad at associating an item with its context. They argue that contextual associations play a crucial role in retrieving and evaluating old memories.

In the case of each of these interpretations, an attempt has been made to show that amnesics are differentially sensitive to some component of retrieval. Warrington & Weiskrantz (1970) argued that certain modes of testing memory are particularly resistant to interference, and hence tend to abolish the memory impairment normally shown by amnesics. An example of this is the use of partial word cueing. The patient is presented with a series of words that he is required to remember. He is then tested by either recognition memory or the presentation of the first few letters of the word, which he is required to complete (e.g. present *statue*, test with *sta* . . .). Amnesics do appear to be very poor at recognition memory, but relatively unimpaired when cued in this way. However, before interpreting this as a characteristic feature of amnesia, it is necessary to rule out the possibility that partial word cueing is simply an effective way of accessing *any* weak memory trace, and is not peculiar to the performance of amnesic patients. Such an interpretation was suggested by Woods & Piercy (1974) and is supported by data from Squire *et al.* (1978), who observe an enhanced sensitivity of partial cueing after a 7 day delay, and Mayes & Meudell (1981), who report that partial cueing is differentially sensitive after a 6 week interval, though not a 7 day interval. However, Weiskrantz & Warrington (1975) failed to replicate Woods & Piercy's result, while the data of Mayes and Meudell are hard to interpret because of a floor effect and an atypical sampling of learning material. The issue therefore remains an open one.

With Gaffan's familiarity theory, although supporting evidence from animal studies has been obtained (Gaffan 1974), attempts to obtain similar effects in humans have so far been unsuccessful (Baddeley 1982), although evidence does exist that amnesics are less confident about even their accurate memories than controls. However, once again normal subjects also show this characteristic when judging equivalently weak memories (Mayes *et al.* 1980).

Evidence in favour of a contextual interpretation of amnesia was presented by Winocur & Kinsbourne (1978), who had their subjects study two related lists of highly associated word pairs, for example the word *army* would be associated with *soldier* in one list, and with the word *battle* in the other. Their amnesic patients showed much less interference when the two lists were learned in separate highly dissimilar rooms, while their control subjects showed little effect of learning environment. However, Mayes *et al.* (1981) have shown quite analogous results for normal subjects. Subjects who had learnt two related lists in two highly dissimilar




PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

rooms showed little advantage over subjects tested in only one environment when recall was immediate. However when a week's delay was interpolated between learning and test, to allow a weakening of the memory trace, a clear advantage occurred for those subjects who had learnt the two lists in dissimilar rooms. Once again, a phenomenon that had been assumed to be characteristic of amnesia proved to be characteristic of weaker memory in general.

There is at present, then, no unequivocal evidence for a qualitiative difference between the memory of amnesics and normal memory, provided that normal memory is measured at a point when it is as weak as amnesic memory. In so far as the various retrieval interpretations demand a qualitative difference between amnesic and normal memory, they must be regarded as being so far without strong support.

### *What can amnesics learn?*

Despite the massive overall impairment in the long-term memory of amnesic patients, it has become increasingly apparent over recent years that certain aspects of memory may be comparatively unimpaired. The classic observation here is that of Claparède, who on one occasion concealed a pin in his hand when he shook hands with an amnesic patient. The following day the patient showed no evidence of being able to recall the incident but was unwilling to shake hands, suggesting some form of learning. Subsequent research has shown that amnesics are capable of showing a wide range of such learning, including classical conditioning, the acquisition of motor skills, learning jigsaw puzzles, or detecting the anomalies in cartoons or pictures (Baddeley 1982; Meudell & Mayes 1982). In all these cases, the subject tends to show unmistakable evidence of learning, but no evidence that he consciously remembers performing the task on which he shows learning.

One of the neatest demonstrations of this phenomenon is that of Jacoby & Witherspoon (1982), who selected a number of homophones (e.g. *read* and *reed*). The less common of the two forms was then incorporated in a definition, and the subject was asked to produce the word (e.g. Q. *What is the part of a clarinet that vibrates*? A. *The reed*). Subjects were then asked to recognize the words they had produced when presented together with other words. The amnesic patients performed very badly on this part of the task. Subjects were then asked to spell a series of words that included those that had been previously presented. There was a clear tendency for spelling to reflect the form of the homophone that had previously been defined (e.g. *reed* rather than *read*). This tendency was just as strong in amnesics as in controls. indicating clear evidence of learning despite their inability to recognize the word in question.

Once again, one must ask the question of whether the occurrence of clear evidence of learning in the absence of conscious remembering is peculiar to amnesia, or is characteristic also of weak normal memory. Once again, the answer appears to be that it can also be found in normal subjects. Meudell & Mayes (1981) examined this effect in a study in which both normal and amnesic subjects were required to search cartoons for particular objects. Both groups showed clear evidence of learning, taking progressively less time to locate the objects over successive trials. When retested after 7 weeks, the amnesics showed evidence of speeded performance on items that they were unable to recognize. However, when tested some 17 months later, controls showed an equivalent result, with clear enhancement of speed in searching cartoons that they were quite unable to recognize.

As Jacoby & Witherspoon (1982) point out, there is a growing body of evidence for two types of normal memory, one of which appears to depend on awareness, and is sensitive to such

variables as depth of processing during encoding. The other aspect of learning appears to depend simply on frequency of presentation; it is reflected in increased speed of processing, and may be unaccompanied by any awareness of the repetition. For example, Kolers (1976) studied the ability of his subjects to read sentences presented in transformed text (for example inverted or reversed). Reading a particular transformed sentence enhanced the subsequent reading of that sentence even after a year's delay, but there was a very low correlation between memory measured in terms of speed, and memory measured by recognition. Jacoby & Dallas (1981) have explored the distinction between these two types of memory, one of which they term *autobiographical memory* and measure by recognition, while the other is termed *perceptual memory* and measured in this case by testing the subject's ability to report the previously presented word when it is shown briefly under near threshold conditions. In one study Jacoby & Dallas varied the depth of processing of the words presented, requiring subjects either to

TABLE 1. EFFECTS OF LEVEL OF PROCESSING ON RECOGNITION MEMORY AND PERCEPTUAL RECOGNITION (PROBABILITIES OF HITS)

(Data of Jacoby & Dallas (1981).)

| question type . . . | physical | | rhyme | | semantic | | new |
|---|---|---|---|---|---|---|---|
| form of memory | yes | no | yes | no | yes | no | words |
| recognition memory | 0.51 | 0.49 | 0.72 | 0.54 | 0.95 | 0.78 | — |
| perceptual recognition | 0.78 | 0.81 | 0.82 | 0.80 | 0.80 | 0.83 | 0.65 |

judge whether a given letter occurred in the relevant word (e.g. *brain* – letter R?), make a rhyme judgement about the word (e.g. *brain* – *rhymes with train*?) or make a semantic judgement (e.g. *brain* – is the centre of the nervous system?). Table 1 shows the results obtained for words processed in each of these ways, tested either by recognition memory or by visual detection. When tested by recognition memory, the standard levels of processing effects occur; deeper processing leads to better memory, while words associated with 'yes' responses are remembered better than 'no' items. With visual detection, however, although there is a clear effect of prior presentation, neither depth of processing nor the nature of the responses influence performance. A similar distinction between two types of normal memory has been suggested by a number of authors. Mandler (1980) postulates two processes in recognition, one based on retrieving relevant context that allows the learning episode to be recalled, the other based on a judgement of familiarity, which in turn is based on the facility with which a previously experienced item is processed. Such items appear to 'jump out' at the subject.

On the basis of the amnesia literature Baddeley (1982) has suggested that two types of retrieval process should be distinguished, a relatively automatic process, together with a slower and less direct process termed *recollection*, which depends critically on the subject's evaluation of material retrieved. Such a concept focuses attention on to the question of how memory is evaluated, and what processes are used to separate genuine from erroneous recall. At present we can do little more than speculate on his point.

The distinction between the two types of memory suggested obviously bears closely on the proposed dichtomy between semantic and episodic memory. The unconscious procedural aspect of memory, which appears to be intact in amnesics, would seem to have many of the characteristics that one would associate with semantic memory. However, as Jacoby & Dallas (1981) point out, there are problems with such a view. I would expect this issue to be one of the growing points in cognitive psychology in the next few years.


PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

## Discussion

I would argue that the interaction between cognitive psychology and neuropsychology has proved extremely fruitful over the last decade. This is particularly so in the analysis of memory into component systems. The initial l.t.m.–s.t.m. distinction was subsequently refined by splitting s.t.m. into its subcomponents. Neuropsychology has provided further evidence for the semantic–episodic distinction and for the possibly related distinction between recollection or autobiographical memory and the automatic or perceptual component of learning. Such distinctions are useful at a conceptual level, while the neuropsychological evidence bears on the difficult question of whether particular components should be regarded as modular subsystems or as separable aspects of a unitary system.

It is less clear that we have made progress in understanding the amnesic syndrome, and it is clearly this feeling that caused Meudell & Mayes (1982) to be critical of the influence of cognitive psychology on neuropsychology. While I would argue that we do know considerably more about the syndrome than 10 years ago (Baddeley 1982), I do not think that we yet have a convincing theoretical interpretation. I would agree with Mayes & Meudell that this stems from the inadequacy of current theories of memory. Having made the important conceptual distinction between the input, the storage and the retrieval functions of a memory system, cognitive psychologists have, I think, been perhaps too simplistic in assuming that it is possible to separate out these three functions experimentally. This rarely proves to be so, because the way in which material is encoded is likely to influence both its durability in memory and its subsequent retrievability. That is not of course to say that we can go no way towards this, but cases in which a phenomenon can confidently be ascribed to one stage rather than another are the exception rather than the rule. Most variables that influence memory probably have effects at more than one stage.

A consequence of this is that one probably needs to test specific theories rather than stage theories of a given phenomenon. With amnesia, we have been able to test some input and storage theories, but at present we simply do not have adequately developed theories of normal retrieval. I think that one of the great contributions of neuropsychology to cognitive psychology at present is in making this point particularly clearly and in stimulating further development.

What of Mayes & Meudell's suggestion that neuropsychology should look elsewhere for its ideas? They themselves clearly favour an interpretation of the amnesic syndrome in terms of consolidation. They are attempting to explore this possibility by using the electroencephalogram, and report some evidence for lower power in the e.e.g. of amnesic patients immediately after the presentation of the material that they are required to remember. It is tempting for a cognitive psychologist to point to the rather unimpressive record of consolidation theories in explaining learning over the last decade, together with a similar lack of convincing results from electrophysiological studies of memory. While this might be justified, I think it would be a mistake to suggest that such approaches should not be explored. It is, however, important to note that, even if such efforts are successful, observing electrophysiological or biochemical correlates of amnesia would not constitute a satisfactory theory. To quote Donchin's presidential address to the Psychophysiological Society, 'Success in the psychophysiological enterprise requires that the psychophysiological data provide insight concerning the underlying processes, rather than a list of correlations between products' (Donchin 1981, p. 497).

Neuropsychology is concerned with both the brain and the mind. As such it is obviously an


PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

important part of its remit to be concerned with the anatomy, electrophysiology and neurochemistry of the brain. However, neuropsychology is a separate discipline from neurology or neurophysiology: it is concerned with the *psychological* consequences of *physical* damage to the brain. While a cognitive psychology without neuropsychology would be impoverished, a neuropsychology devoid of cognitive psychology would inevitably be grossly incomplete.

## References (Baddeley)


Atkinson, R. C. & Shiffrin, R. M. 1968 Human memory: a proposed system and its control processes. In *The psychology of learning and motivation: Advances in research and theory*, vol. 2 (ed. K. W. Spence), pp. 89–195. New York: Academic Press.

Baddeley, A. D. 1966*a* The influence of acoustic and semantic similarity on long-term memory for word sequences. *Q. Jl exp. Psychol.* **18**, 302–309.

Baddeley, A. D. 1966*b* Short-term memory for word sequences as a function of acoustic, semantic and formal similarity. *Q. Jl exp. Psychol.* **18**, 362–365.

Baddeley, A. D. 1982 Amnesia: a minimal model. In *Human memory and amnesia* (ed. L. S. Cermak), pp. 305–336. Hillsdale, N.J.: Erlbaum.

Baddeley, A. D. & Hitch, G. J. 1974 Working memory. In *The psychology of learning and motivation*, vol. 8, pp. 47–90. New York: Academic Press.

Baddeley, A. D. & Lieberman, K. 1980 Spatial working memory. In *Attention and performance VIII* (ed. R. Nickerson), pp. 521–539. Hillsdale, N.J.: Erlbaum.

Baddeley, A. D. & Warrington, E. K. 1970 Amnesia and the distinction between long- and short-term memory. *J. verb. Learn. verb. Behav.* **9**, 176–189.

Baddeley, A. D. & Warrington, E. K. 1973 Memory coding and amnesia. *Neuropsychologia* **11**, 159–165.

Broadbent, D. E. 1958 *Perception and communication.* London and New York: Pergamon Press.

Brooks, D. N. & Baddeley, A. D. 1976 What can amnesic patients learn? *Neuropsychologia* **14**, 111–122.

Butters, N. & Cermak, L. S. 1975 Some analyses of amnesic syndromes in brain-damaged patients. In *The hippocampus*, vol. 2 (ed. R. Isaacson & K. H. Pribram), pp. 377–409. New York. Plenum Press.

Cermak, L. S. 1976 The encoding capacity of a patient with amnesia due to encephalitis. *Neuropsychologia* **14**, 311–326.

Cermak, L. S., Butters, N. & Gerrein, J. 1973 The extent of verbal encoding ability of Korsakoff patients. *Neuropsychologia* **11**, 85–94.

Cermak, L. S. & Reale, L. 1978 Depth of processing and retention of words by alcoholic Korsakoff patients. *J. exp. Psychol.: hum. Learn. Memory* **4**, 165–174.

Conrad, R. 1964 Acoustic confusion in immediate memory. *Br. J. Psychol.* **55**, 75–84.

Craik, F. I. M. & Lockhart, R. S. 1972 Levels of processing: a framework for memory research. *J. verb. Learn. verb. Behav.* **11**, 671–684.

Donchin, E. 1981 Surprise! . . . Surprise? *Psychophysiology* **18**, 493–513.

Gaffan, D. 1974 Recognition impaired and association intact in the memory of monkeys after transection of the fornix. *J. comp. Physiol. Psychol.* **86**, 1100–1109.

Glanzer, M. & Cunitz, A. R. 1966 Two storage mechanisms in free recall. *J. verb. Learn. verb. Behav.* **5**, 351–360.

Hebb, D. O. 1949 *Organization of behavior.* New York: Wiley.

Huppert, F. A. & Piercy, M. 1978 The role of trace strength in recency and frequency judgments by amnesics and control subjects. *Quart. Jl exp. Psychol.* **30**, 346–354.

Jacoby, L. L. & Dallas, M. 1981 On the relationship between autobiographical memory and perceptual learning. *J. exp. Psychol.: gen.* **3**, 306–340.

Jacoby, L. L. & Witherspoon, D. 1982 Remembering without awareness. *Can. J. Psychol.* (In the press.)

Kinsbourne, M. & Wood, F. 1975 Short-term memory processes and the amnesic syndrome. In *Short-term memory* (ed. D. Deutsch & J. A. Deutsch), pp. 258–291. New York: Academic Press.

Kolers, P. A. 1976 Reading a year later. *J. exp. Psychol.: hum. Learn. Memory* **2**, 554–565.

Mandler, G. 1980 Recognizing: the judgment of previous occurrence. *Psychol. Rev.* **87**, 252–271.

Mayes, A. & Meudell, P. 1981 How similar is the effect of cueing in amnesics and in normal subjects following forgetting? *Cortex* **17**, 113–124.

Mayes, A., Meudell, P. & Neary, D. 1980 Do amnesics adopt inefficient encoding strategies with faces and random shapes? *Neuropsychologia* **18**, 527–540.

Mayes, A., Meudell, P. & Som, S. 1981 Further similarities between amnesia and normal attenuated memory: effects with paired-associate learning and contextual shifts. *Neuropsychologia* **19**, 655–664.

Melton, A. W. 1963 Implications of short-term memory for a general theory of memory. *J. verb. Learn. verb. Behav.* **2**, 1–21.




PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Meudell, P. & Mayes, A. 1982 Normal and abnormal forgetting: some comments on the human amnesic syndrome. In *Normality and pathology in cognitive function* (ed. A. Ellis). London: Academic Press. (In the press.)

Milner, B. 1966 Amnesia following operation on the temporal lobes. In *Amnesia* (ed. C. W. M. Whitty & O. L. Zangwill), pp. 109–133. London: Butterworth.

Moscovitch, M. 1982 Multiple dissociations of function in the amnesic syndrome. In *Human memory and amnesia* (ed. L. S. Cermak), pp. 337–370. Hillsdale, N.J.: Erlbaum.

Peterson, L. R. 1966 Short-term verbal memory and learning. *Psychol. Rev.* **73**, 193–207.

Peterson, L. R. & Peterson, M. J. 1959 Short-term retention of individual verbal items. *J. exp. Psychol.*, **58**, 193–198.

Saffran, E. M. & Marin, O. S. M. 1975 Immediate memory for word lists and sentences in a patient with deficient auditory short-term memory. *Brain Lang* **2**, 420–433.

Salamé, P. & Baddeley, A. D. 1982 Disruption of short-term memory by unattended speech: implications for the structure of working memory. *J. verb Learn verb. Behav.* (In the press.)

Shallice, T. 1979 Neuropsychological research and the fractionation of memory systems. In *Perspectives on memory research* (ed. L. G. Nillson), pp. 257–277. Hillsdale, N.J.: Erlbaum.

Shallice, T. & Butterworth, B. 1977 Short-term memory impairment and spontaneous speech. *Neuropsychologia* **15**, 729–735.

Shallice, T. & Warrington, E. K. 1970 Independent functioning of verbal memory stores: a neuropsychological study. *Q. Jl exp. Psychol.* **22**, 261–273.

Squire, L. R., Wetzel, C. D. & Slater, P. C. 1978 Anterograde amnesia following ECT: an analysis of the beneficial effects of partial information. *Neuropsychologia* **16**, 339–348.

Talland, G. A. 1965 *Deranged memory: a psychonomic study of the amnesic syndrome*. New York: Academic Press.

Tulving, E. 1972 Episodic and semantic memory. In *Organization of memory* (ed. E. Tulving & W. Donaldson), pp. 381–403. New York: Academic Press.

Underwood, B. J. & Schulz, R. 1960 *Meaningfulness and verbal learning*. Chicago: Lippincott.

Warrington, E. K. 1975 Selective impairment of semantic memory. *Q. Jl exp. Psychol.* **27**, 635–658.

Warrington, E. K. 1982 The double dissociation of short- and long-term memory deficits. In *Human memory and amnesia* (ed. L. S. Cermak), pp. 61–76. Hillsdale, N.J.: Erlbaum.

Warrington, E. K., Logue, V. & Pratt, R. T. C. 1971 The anatomical localization of selective impairment of auditory verbal short-term memory. *Neuropsychologia* **9**, 377–387.

Warrington, E. K. & Shallice, T. 1969 The selective impairment of auditory verbal short-term memory. *Brain* **92**, 885–896.

Warrington, E. K. & Shallice, T. 1972 Neuropsychological evidence of visual storage in short-term memory tasks. *Q. Jl exp. Psychol.* **24**, 30–40.

Warrington, E. K. & Weiskrantz, L. 1970 Amnesic syndrome: consolidation or retrieval? *Nature, Lond.* **228**, 628–630.

Waugh, N. C. & Norman, D. A. 1965 Primary memory. *Psychol. Rev.* **72**, 89–104.

Weiskrantz, L. & Warrington, E. K. 1975 Some comments on Woods' and Piercy's claim of a similarity between amnesic memory and normal forgetting. *Neuropsychologia* **13**, 365–368.

Woods, R. T. & Piercy, M. 1974 A similarity between amnesic memory and normal forgetting. *Neuropsychologia* **12**, 437–445.

Winocur, G. & Kinsbourne, M. 1978 Contextual cueing as an aid to Korsakoff amnesics. *Neuropsychologia* **16**, 671–682.



PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES