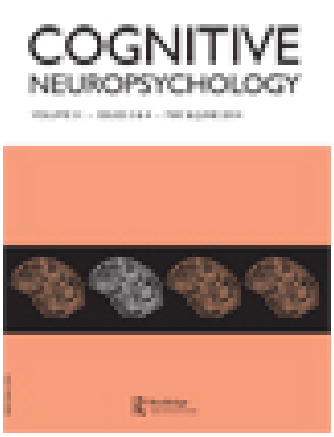

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The problem with amnesia: The problem with human memory

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A commentary on *Human Memory and Amnesia*, edited by L. A. Cermak. Hillsdale: Lawrence Erlbaum Associates Inc. 1982, ISBN 0-89859-095-7, £41.65, $50.00.

This book is notionally the record of a meeting, held in 1979, which was intended to bridge the gap between researchers working on normal memory processing and those studying the amnesic syndrome. Most of the Western world's work on amnesia is represented here and I was grateful to have good summaries of the individual research efforts in an easily digestible form. A spirit of reconciliation was clearly in the New England air, and a number of the authors were at pains to try and explain why different laboratories have, in the past, come to different conclusions. This effort included a good deal of methodological criticism of others but, although the chapters in the book were meant to have been written after the discussion, it is clear that a number of them were not revised; some quite severe criticisms remain unanswered. Correspondingly, on the editorial side, it is odd to find authors giving detailed accounts of another's research when this research, sometimes with a number of graphs, is described on another page by the original author, but without any cross-referencing.

KINDS OF AMNESIA

The two main problems with the book concern what is it that has to be explained and what means are to be used to explain it. With regard to the first problem it is clear that "amnesia" does, at least, have a clinical meaning. As Wood, Ebert and Kinsbourne remark, "any neuropsychologist who is accustomed to seeing patients with the amnesic syndrome feels confident in being able to diagnose that syndrome after

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perhaps only an informal conversation" (p. 191). The syndrome thus discovered involves the patient's inability to remember current events (anterograde amnesia) and/or events for some years predating the onset of the condition (retrograde amnesia). Unfortunately, the group of amnesics that has been studied most, the Boston Korsakoffs, have additional problems which appear to be due to frontal lobe involvement. This is especially well documented by Moscovitch. Now, there is nothing wrong with studying the alcoholic Korsakoff syndrome (clinically defined) rather than amnesia (psychologically defined). However, there is no reason, apart from semantic slippage, to imagine that they constitute the same problem, either in terms of appropriate method or likely solution. The tensions arising from this conflict permeate the whole book. The initial, almost casual demonstration that amnesics do not have a deficit in short-term memory tasks (reiterated and replicated here by Warrington with a number of traditional short-term tasks), has transmuted into an industry that has taken on board all the pre-theoretical baggage of the 70s memory research with all the same controversies and neobehaviourist confusions between STM as an experimental paradigm and STM as a theoretical construct. This effort has not been without its methodological interest but it does not seem to have advanced our understanding of the *primary* amnesic syndrome. Indeed, it only seems to have advanced our understanding of the Korsakoff syndrome in as much as it forced the acknowledgement that British and American Korsakoffs are significantly different; the latter, unlike the former, are very passive with attentional deficits and deficits in short-term memory tasks (Baddeley) in spite of being selected as having IQ scores of 100+. Why this difference should occur (social reasons? different kinds of alcohol? diet?) is a question no one poses, but the effect has been to turn the consequences of the equation of Korsakoff's syndrome and amnesia into even more fruitless controversy and irrelevant theoretical conjecture.

Korsakoff's disease is not the sole cause of amnesia. Other relevant etiologies include Huntington's Disease, encephalitis, ECT and trauma, either accidental or surgical. While there is a little documentation on the differences between these groups (including three very informative little case reports by Wood, Ebert & Kinsbourne) the overall impression is still that of hope that these differences will prove unimportant. However, within the paradigmatic amnesia itself (i.e., anterograde and retrograde) there are differences enough, documented by Butters and Albert and by Squire and Cohen, particularly in the temporal slope of the retrograde amnesia. I am left with the suspicion that not only are the different patient groups characterised by distinct mixes of additional deficit but that the amnesia itself is not subject to a single or singular account. There are hints of such conclusions in the book. Winocur postulates "a reduction in those skills that are fundamental to the memory process" (p. 142), and Cermak concludes "we are left . . . with the knowledge that the amnesic patient's entire information-processing-for-memory system is impaired to some

extent" (p. 56). Alas, no one seems to be able even to sketch what such skills or such a system might be or even indicate how processes related to memory can be distinguished, even in principle, from those underlying the intellectual abilities that remain intact in many of these patients.

THE USE OF THEORY

The second problem lies in the available theoretical apparatus. For some reason there is a belief around that anyone who uses a term like "encoding" is an information-processing theorist. In relation to laboratory studies of amnesia (delayed recall, release from PI, recognition memory, etc.) there are still supporters of "encoding deficits", "storage deficits" and "retrieval deficits" as exclusive or primary causes, although a number of authors spend some time demolishing such positions (Winocer, Baddeley, Moscovitch) or, at least, the subset they don't personally subscribe to (Huppert & Piercy). The argument Moscovitch puts forward against "encoding theories" hinges on the claim that "Encoding theory rests on the assumption that memory cannot be dissociated from cognition" (p. 355). Forcing the Korsakoff into complex processing of stimulus materials doesn't particularly help him to recall these materials and "none of the cognitive disorders (e.g., attentional deficits—case JM) that have been investigated thus far lead to global amnesia" (p. 355). Rejection of the encoding hypothesis, in the absence of any explicit discussion of the relationship between "encoding", "cognition" and "memory" seems totally unjustifiable. If there is an implicit hypothesis discussable it is that all and only products of cognitive operations are stored in "long-term" memory. I see no reason why this should be so. The term *encoding* as a cognitive operation (particularly equated with experimental procedures designed to manipulate "encoding strategies") has been merged with *encoding* as an operation involved in putting something into memory. Split these two apart and the arguments against encoding deficits in amnesia, inasmuch as these refer to storage, seem to dissolve. Suppose, that is, that the only cognitive deficit suffered by the "pure" amnesic involves the mechanisms responsible for placing the products of other cognitive operations into storage. The deficit could take a number of forms. For example, we could examine the hypothesis that only the products of certain kinds of cognitive operation could be placed in storage by amnesics or that only certain *kinds* of product, however obtained, were preserved.

At the moment I am not *advocating* either one of these positions. My objective is simply to point out that the model space available is a good deal richer than Moscovitch, and many others, seem to realise. Asking "Is it encoding?" is just another example of the "Twenty Questions" approach.

The same points apply to the arguments presented for or against "retrieval" theories. The chief argument against is that the amnesic's retrieval deficit should extend uniformly across the entire life span. The

dissociation between anterograde and retrograde amnesia, especially in non-Korsakoff amnesics, refutes this prediction. Moscovitch takes this line of reasoning a little further — but relegates his analysis to a footnote:

> The theory may be modified to accommodate these findings — for example, amnesics may have lost the ability to lay down and independently generate appropriate retrieval cues only for those events that occurred after their traumata. This retrieval theory is sufficiently different from the former type and has so much in common with both encoding and consolidation theory that it is best to forgo a discussion of it until someone chooses to adopt it as his or her own (p. 355, footnote 3).

At least here we have an implied distinction between "retrieval cues" and the content of the memory and a hint of the interaction between storage and retrieval phases that Tulving has stressed repeatedly. (I am surprised, though, to hear that theories cannot be discussed unless someone has explicitly espoused them!)

Kinsbourne and Wood indicate how the underlying three-stage model of encoding, storage and retrieval is in principle inappropriate, and the general swing is towards considering the *interaction* of encoding and retrieval. Schacter and Tulving comment that "the dichotomy between 'storage phenomena' and 'retrieval phenomena' is no longer theoretically defensible" (p. 24). This position will help people to avoid some of the primitive errors of theorising already referred to, but the way forward is not clear. Empirically, it may be difficult to assign the origin of a performance deficit but that does not require we blur the obvious—storage and retrieval operations *are* different. The swing to considering their interaction leads to some focus on cue utilisation (where the frontal lobe pathology of the Boston Korsakoffs confuses the issue again) and a little gesturing towards encoding specificity and state dependent learning. Alas, these two get confused in some authors' minds. The difference is that state dependent effects are not found in *recognition* memory, a fact which only Baddeley seems to see the relevance of: retrograde amnesia extends to recognition as well as recall.

A MYSTERIOUS DICHOTOMY

There is some effort to make a theoretical link between the amnesic deficit and the episodic/semantic distinction. One thread is the argument that because episodes include (by definition) information about time and space their recall must be state dependent. But this is clearly a *non sequitur*. More seriously, there are attempts by Schacter and Tulving, by Wood, Ebert and Kinsbourne and by Kinsbourne and Wood to locate amnesia within Tulving's delineation of episodic memory. This runs into some difficulties when one considers the kinds of things that amnesics can learn (see the next section).

Yet the episodic-semantic distinction continues to be invoked in the literature. For example, Parkin (1982), gives an excellent review of the residual learning capacity of amnesics. On the basis of this capacity alone, and ignoring the data on retrograde effects, he claims: "It therefore seems reasonable to describe organic amnesia as a severe impairment of episodic storage with the relative sparing of semantic memory" (p. 431). He points out that the term "semantic" is to be understood more generally than a store of linguistic knowledge, as "a general memory system whose contents are continually modified by any new experience" (p. 430) and distinct from "the storage of specific information about the actual occurrence of the experience" (p. 431, footnote 1). The circularity of the distinction as Parkin uses it resembles that of Tulving (1983). As Baddeley (1984) points out, Tulving "simply labels tasks which amnesics can perform as semantic and then concludes that their semantic memory performance is intact" (p. 239).

The problem, yet again, is the absence of proper theory. Parkin mentions neither storage nor retrieval but sticks with an attempt merely to define two memory systems (semantic and episodic): yet nobody has been able to make that distinction, or any other based solely on two *types* of information, stick. Baddeley (1984) in his latest comments tries new vs old (which cannot be complete because the amnesic can learn some things) and procedural vs declarative, which Parkin criticises (following Johnson-Laird, 1982) as devoid of empirical content.

It really is infuriating. The pattern of amnesic loss seems to make intuitive sense but every attempt to grasp it formally has failed.

In a different attempt to characterise the amnesic loss a distinction has been made between two kinds of facts in memory — salient and detailed. It has been suggested that details are lost more rapidly from memory in the normal case and that ECT can further disrupt these details. Since remote memories would not have these details in normals, ECT patients would thus show more apparent loss for recent memories, thereby giving rise to the graded retrograde amnesia that has been reported. Squire and Cohen (1979), however, claimed that ECT affected memory for both kinds of fact in recent memories but did not affect recall of detailed factual material for more remote memories. Absolute comparisons of recent and distant memory revealed that the patients' recall for recent memories was better than for distant memories before the ECT, but was worse for recent than distant memories after ECT. Squire and Cohen argued that since salient information should be at least as abundant in recent memories as in distant ones, this pattern should not be possible on the above theory. They repeat this claim in the volume under review. These arguments are put forward in order to establish that the steeply graded retrograde amnesia is not an "artefact". The nature of the threatening artefact is that a general retrograde loss, which would support a "general" storage disruption, is disguised by the nature of the materials used. Thus, the slope in the amnesic recall functions might reflect disruption of detail, rather than disruption

being a function of the "age" of a memory. The "age" hypothesis would, of course, be a pure biological account of the effects of ECT, related perhaps, to some as yet undetermined long-term biochemical "fixing" of the memory engrams which would not respect such psychological categories as "salient" and "detailed". To find a difference in susceptibility between *psychologically* defined types of memory of the same age would weaken the biological case. Some such biological account, implicit in Moscovitch's espousal of the "consolidation" hypothesis, may, indeed, be true, but the relevant methodological issues are far from settled. To rescue the idea of general loss the psychological theorist could argue that since prominent past events get frequently rehearsed after the initial experience, the *nature* of the code might change as a function of rehearsal. Further, it could be claimed that the two codes require significant variations in the way the retrieval processes operate. The problem is to find the appropriate characterisation of the codes. The dichotomy between salient and distinct may be misconceived, but that would not bear on the general principle. Rehearsal must have some effect on old memories and we have simply failed to identify what the effect is.

Pushed this far, the argument seems to be in principle undecidable. It must be the responsibility of proponents of "general loss" to put forward a proper psychological theory in order to account for the temporal slope in the retrograde function. It seems unlikely that this could be done without a relatively detailed processing account together with a detailed examination of exactly what is and what is not recalled of distant and recent memories.

In the main, the contributors have a rational approach to the relationship between biological and psychological accounts. The occasional exception, then, comes as a slight shock. Goggin, discussing storage differences between pictures and words, is interested in their biological substrate. Because it seems unlikely in the forseeable future that "we will be able to determine directly whether or not the 'engrams' or stored representations for words and pictures differ" (p. 220) we will have to rely on the "indirect indicants" of psychology. But, of course, psychological methods can contribute nothing at all to the answer to the biological question unless we have a theory of the mapping between the two levels. This should be clear as soon as one considers the nature of the biological question. Suppose one claimed that the engrams for words and pictures were identical in type. If such a claim has any meaning it could be that, say, both individual words and individual pictures are stored in single neurons. But there is no piece of psychological information that could indicate such a fact even indirectly. Words and pictures could be processed nonetheless in very different ways (by using different criteria in decision-making processes, for example) and therefore give rise to differences in the results of experiments using the two kinds of materials. If the engrams for pictures and words differ, by virtue of the nature of the two kinds of stimuli, there need be no necessary difference in the way we overtly behave towards them: processing differences could compensate for or cancel out the differences in the engrams.

WHAT AMNESICS CAN LEARN

Baddeley, as well as Wood, Ebert and Kinsbourne, describes the kinds of task that amnesics can apparently learn at normal rates. These include jigsaw puzzles, eye-blink conditioning, pursuit rotor tracking, detection of anomalies in pictures (for example, a life belt lying on the bottom in an underwater scene), maze and tune learning, and unscrambling sentences. Remember that while the amnesics' performance improves with number of trials, they may have no awareness that they have ever done the task before. At one point it was believed that learning was restricted to perceptual and motor skills, because these skills purportedly involved different neural structures from other memory tasks. Baddeley points out that such a belief is untenable given that a wide range of amnesic patients "show apparently normal learning both on verbal tasks and on complex and apparently semantically based perceptual tasks" (p. 327). What the tasks have in common is that they allow the patient to reflect learning without having to consider the provenance of the information that was used.

In an attempt to give this characterisation of amnesic learning some theoretical force Baddeley invokes a distinction between relatively automatic retrieval processes and the active problem-solving aspect of recall that he terms "recollection". In his exposition of the latter, Baddeley covers much the same ground as Norman and Bobrow (1979) and Williams and Hollan (1981)—an interesting and encouraging convergence. Suppose, then, that amnesic patients lack the ability to recollect, although they can still build up and run off "procedures". The consequences of the disability would include:

An inability to use incidental detail as confirmation of the correctness of something retrieved.

An inability to reject incorrect associations produced by automatic procedures.

An inability to iterate a retrieval cycle to follow clues and check them through memories with episodic characteristics (cf Williams & Hollan, 1981).

The concept of recollection, as Baddeley quickly points out, only goes a little way towards accounting for amnesia. Nonetheless, it has the outstanding advantage that it is dynamic and makes contact with real memory phenomena. He could have pushed the idea much further than he did in accounting for a number of standard experimental results but hung back in face of "our inability to conceptualise adequately the complex processes of normal memory" (p. 333).

WHY ARE WE WHERE WE ARE?

Schacter and Tulving, in the opening chapter, believe "we may be on the verge of a golden age" of interaction between memory theorists and

neuropsychology. However, their overview of this interaction gives no clearer view of the future than does the latest round of disarmament talks. They point to some methodological traps, as do a number of other authors. Indeed, if the contributors to this volume have one thing in common it is the excellence and persistence of the arguments with which they hope to correct the errors of others. On some topics there is almost unanimity (Gaffan's, 1974, "familiarity" hypothesis is attacked from five sides with only Schacter & Tulving approving); on others the battle rages on.

What seems to be wrong? There are two dominating problems, one to do with the use of theory and one to do with the view of amnesia. With respect to the first problem, there is no reason to believe that the processes of memory are less complex than those of reading. There is ample suspicion of the same degree of variation in amnesics as there is, for instance, in patients with acquired dyslexia. But there is no model for memory displayed in this volume that approaches the complexity of the models *routinely* used in the study of reading. (Coltheart, Patterson, & Marshall 1980; Newcombe & Marshall, 1981; Shallice, 1981) It is notable, for example, that no neuropsychologist uses any current version of schema theory (e.g., Rumelhart & Ortony, 1977). "Schemata" only get mentioned once, by Squire and Cohen, and then only in arguing that old memories become "schematised" and so resistent to disruption. Nothing of the dynamics of schema models is discussed. In general the theoretical discussions are very thin. We get no feel for the processes of memory, no embracing of the underlying complexities. All this reflects the neobehaviourism still endemic in memory research. Task stages and experimental variables are still given theoretical status without the benefit of those hypothetical constructs that might give purpose to any postulated mechanisms. The mere acknowledgement that subjects can adopt different strategies does not in and of itself constitute a cognitive theory.

Goggin raises a number of related issues. She examines the "behavioural psychologists' methodology" in trying to answer the question of whether words and pictures are stored differently. She discusses selectively, pointedly and lucidly the variables, dependent and independent, that have been used in such studies and concludes "it is unreasonable that a coherent theory of word-picture storage differences will be developed" (p. 230). The multiple differences between pictures and words will not allow the question to be answered in a theoretically interesting way. Rather than concluding that it was a *silly* question to begin with, she decides the next best thing: that it was the *wrong* question. Following the arguments in Newell's (1973) "Twenty Questions" article, Goggin first concludes we should study the processing and storage of words and pictures separately and worry less about their similarities and differences. The importance of this for amnesia research is to eliminate attempts to squeeze too much theoretical meaning out of picture-word comparisons in memory. Next she makes the suggestion that we look more at the central processes used in cognitive tasks and

examine more complex tasks. Finally, one might look at individual differences. In the end then, we have the cognitive prescription with the seeming paradox that more complex tasks have at least the potential for resolution, whereas the simpler behaviourist methodology leads to a total impasse.

It was a surprise to find this chapter ending up where it did, considering where it started from. This kind of discussion, written from the inside and ending on the outside, is an essential component of paradigm shift. "I have been there and it doesn't work" is convincing testimony.

The second issue is related to the first. If models of function are simple, their breakdown is going to be simple. Almost without exception there is the assumption that the central deficit is the same for all amnesics (at least with respect to the amnesia) and, usually, that there is only one deficit. There is precious little evidence directly against these suppositions, but, then, there has not been much attempt to find such evidence. I feel uneasy about this state of affairs, again by analogy with dyslexia. Most, if not all, acquired dyslexics who are studied from the viewpoint of information processing are described as having multiple functional lesions (e.g., Morton & Patterson, 1980). The differences between patients are at least as illuminating as the similarities. Marshall (1982) has made similar points with respect to the aphasias. Now, it may be the case that the processes of memory are simpler than the processes of reading, and that there are consequently fewer ways in which memory can be disrupted. Maybe individual differences in the retrograde amnesia, for instance, are absolutely unimportant. Unfortunately we don't know because, in general, groups are studied rather than individuals, and the contributors to Cermak's book give no hint of possible changes in methodology.

Meanwhile we are left with an excellent reference volume for experimental work on amnesia together with ample demonstration of what Cermak himself calls "the skimpy theoretical base on which we are now operating" (p. 374).

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