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# Problems of learning and memory: one or multiple memory systems?

L. WEISKRANTZ

*Department of Experimental Psychology, University of Oxford, Oxford OX1 3PS, U.K.*

## SUMMARY

Learning, and hence memory, is ubiquitous not only throughout the animal kingdom, but apparently throughout many regions of the brain. Is all learning reducible to a single common form? Neuropsychological dissociations suggest that the mammalian brain possesses a number of different and potentially independent memory systems, with different mechanisms and anatomical dispositions, some of which are neurally widely dispersed and others of which are narrowly organized. Among the types considered are: (i) short-term memory; (ii) knowledge and skills; (iii) stable associative memory; (iv) event memory; and (v) priming. As double or multiple dissociations do not lead to logically inevitable conclusions, it has been argued that an alternative to multiple memory systems is variable modes of processing. But these, too, would be dissociable on the same lines of evidence. Dissociations, if strong and absolute, have strong pragmatic power when they are combined with evolutionary and neuroscientific evidence. Multiple memory systems may possibly share some common cellular mechanisms, but such mechanisms do not define the separate properties at the systems level.

## PROBLEMS OF LEARNING AND MEMORY: ONE OR MULTIPLE SYSTEMS?

It is an ironic fact that in these days of interest in what is called parallel distributed processing, such processes carried out in parallel are not independent but tied together in distributed networks. Yet organisms, at one level, are obviously collections of parallel systems that are potentially independent, although normally interactive. In the field of perception, this is obvious. No one would think of challenging the assumption that the visual system is, in a deep sense, independent of an auditory system, an olfactory system, a somatosensory system, etc. although these potentially independent systems become interactive at later stages. But in the area of memory, there continues to be a reluctance in some quarters to consider a similar possibility. Yet a capacity to learn and remember is so important for survival, is challenged in such a great variety of situations over a wide range of time gaps and for such widely differing adaptive skills, that apparently there is just as much reason, perhaps even more, to consider separate parallel systems here as in perception.

Establishing independence for perceptual systems is relatively easy because we can isolate the stimulus that impinges on receptors. If the eyes are closed, hearing is not affected, or not much. And, importantly, vice versa. Implicitly, these dissociations that we establish in our everyday life take the form of double or even n-dimensional dissociations, and it is these dissociations that suggest independent systems.

We know from neuropsychology, neurophysiology and neuroanatomy that the independence of the perceptual systems is maintained well beyond the

receptor levels. That is, vision can be affected uniquely by damage to one region of the brain, and audition to another. Moreover, activity in some regions of the brain is primarily controlled by events that strike the retina and elsewhere by events that strike the ear. So in principle, even if it were the case that – as in a fantasy world – we could not close our eyes or shield our ears, it could still be inferred from neuropsychology that we possessed parallel perceptual systems, at least up to certain stages of processing.

I want to consider whether similar evidence in the memory domain leads to similar inferences, i.e. evidence from dissociations linked to neuropsychology and neurophysiology, leading to inferences of independent systems – but of course, normally interacting systems – just as perceptual systems normally interact.

Before reviewing illustrative evidence for multiple memory systems, using neuropsychological and other neurological evidence, there are some distinctions and disclaimers to make. First, it is useful to distinguish between what Crowder (1989) has called ‘coding’ modules and ‘process’ modules. Some memories are modality-specific and even within a modality it is now clear that the nervous system goes to some trouble to separate attributes such as colour, motion, spatial location, etc. Moreover these can be dissociated neuropsychologically. All these would be called ‘coding’ modules. Such evidence collectively is itself very substantial evidence for independent systems, perhaps each with special mechanisms required for translating inputs into durable changes, but I am not concerned here with the storage of modular attributes as such. Secondly, neurons are neurons and nothing

more than neurons. At the level of cellular mechanism there are limited degrees of freedom. It may be, even if there are independent memory systems, that the cellular events used in all are similar or even identical. I think this is, in fact, unlikely and we still may not have exhausted all degrees of freedom. There are drugs (nootropics), however, which have clear positive effects on learning in animals but which have no direct or readily comprehensible effect on synaptic mechanisms as these are commonly understood (Vicent *et al.* 1985; Mondadori *et al.* 1986, 1987). Nevertheless, the question of what happens at the cellular level when we say that an organism learns or remembers or what cellular preconditions must exist for these to occur is a question at a different level of analysis from that of whether there are multiple memory systems. Finally, I certainly am not claiming to advance an exhaustive or universal taxonomy of different memory systems, nor will I address many important particular issues that relate to acquisition, storage and retrieval. To do so independently of specific species adaptations, in any event, would be counter-productive. For example, birds have a special requirement and system for birdsong, which may be particular to them. Creatures that navigate have different memory requirements from those that do not and so forth. Humans, but probably no other species, have a capacity for syntactic language, with its own huge mnemonic requirements. The evidence cited here is illustrative rather than exhaustive (it says nothing, for example, about habituation) and is drawn largely but not exclusively from human neuropsychology. It follows, in part, classifications put forward by Warrington (1979), Tulving (1983) and Weiskrantz (1987).

First, short-term memory (STM). I still prefer this in the present context to the more recent term, working memory, for one reason that will become clear and also because working memory is used in somewhat different senses in animal and human memory research. Strong evidence for a dissociation of STM from long-term memory (LTM) comes from the original investigations of Warrington *et al.* (Warrington & Shallice 1972; Warrington 1981; Warrington *et al.* 1971) of patients with lesions of the posterior temporal lobe of the left hemisphere who are severely impaired on Brown-Peterson tasks (i.e. in recalling random sequences of digits or consonants after short intervals of time during which rehearsal is prevented), and have very abbreviated digit- or letter-spans of only one or two items in length. In serial position effects, the recency component is more transient than the primacy component (the serial position effect refers to the advantage in retaining the early and the late items of a list. The 'primacy effect' refers to the advantage for the items presented early, the 'recency effect' to the items presented last) (see figure 1). This transience is even more marked in these patients. Importantly, these patients have no difficulty in long-term memory (the primacy effect is intact) they can memorize supra-span lists at a normal rate and retain them over days. On the other hand, just the opposite pattern is seen with amnesic patients, who have severe problems with long-term retention. In these patients, as shown by Baddeley

& Warrington (1970), the primacy component, but not the recency component, is depressed, and there is ample evidence that amnesic patients can be perfectly normal on just the tasks in which the Warrington & Shallice cases are so poor (figure 2). Relatively pure short-term memory cases are uncommon but a number of have been studied (see, for example, Saffran & Marin (1975); Caramazza *et al.* (1981); Basso *et al.* (1982); Vallar & Baddeley (1984); Friedrich *et al.* (1984); Shallice (1988)).

These studies concentrated on verbal material. Indeed, typically such patients have a much smaller deficit, or none at all, when visual material is used. It is natural to consider that normal subjects exploit a kind of silent rehearsal using a verbal acoustic code (Conrad 1962) (what Baddeley calls the 'articulatory loop') when input to a long-term store is over-loaded. The patients in question, it might be argued, lack this capacity to silently rehearse. Hence it has been doubted whether there is any clear homologue of a STM process at the animal level (Weiskrantz 1970). It is hard to imagine animals using articulatory loops in order to silently rehearse, though perhaps parrots and some other birds are an exception; it would make an amusing study.

Nevertheless, and perhaps surprisingly, good evidence for short-term memory in animals does exist in the visual modality, though I would hesitate to call it the 'visual scratch pad'. A highly instructive study has been reported by Wright *et al.* (1985). They gave the same form of test to human subjects, monkeys and pigeons (except that with the human subjects kaleidoscopic 'meaningless' patterns were shown rather than picture postcards). Subjects were shown a series of four coloured slides, each displayed for 1 s. The slides were different on every trial. After a variable and predetermined delay, following the last item in any series, the subjects were shown a probe item. If the probe was a repeat of one of the items in the list of four they had to indicate by making one response; if not, they had to make another response. The results are shown in figure 3. Note that all three species show both primacy and recency effects. The difference between them is quantitative rather than qualitative. The authors conclude that no single-process explanation can adequately explain the range of findings with serial position effects; two or more underlying memory processes are implicated in all three species. It is worth mentioning, in passing, that the Wright *et al.* study is unusual in isolating a primacy effect in animals.

The above study used normal subjects. Gaffan & Weiskrantz (1980) found clear evidence for a recency effect in monkeys independent of order of visual list items, using a delayed non-matching paradigm. In keeping with a large volume of literature on delayed non-matching paradigms by Mishkin *et al.* (Mishkin 1978, 1982; Mishkin *et al.* 1982), we found that a particular lesion in the limbic system caused more rapid decay of retention of lists over this short recency domain. We also showed that lesions of neocortex that do severely affect another category of memory operations was without significant effect on our task, once

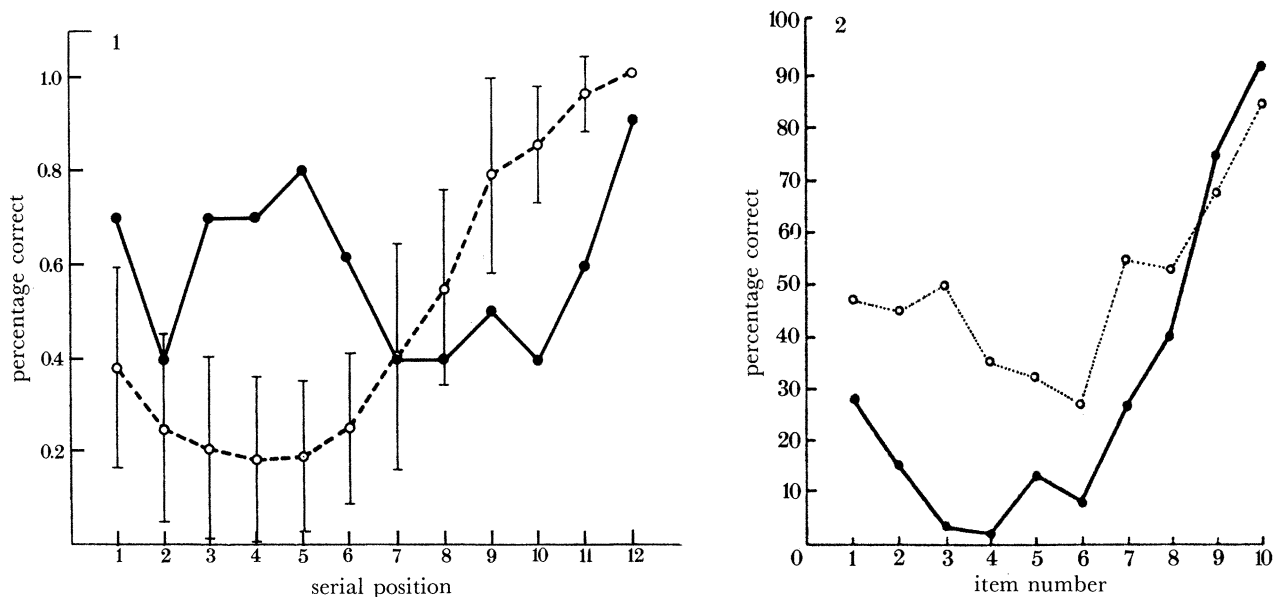


Figure 1. Free-recall performance of control subjects and a patient (PV) with short-term memory difficulty. In this task, to emphasize the recency effect, subjects were instructed to recall the final items first. PV's recency effect is limited to the last item; (●), PV; (○), control. (From Vallar & Papagno (1986) with permission.)

Figure 2. Serial position curve for immediate free recall for amnesic and control subjects; (●), amnesics; (○), control. (From Baddeley & Warrington (1970) with permission.)

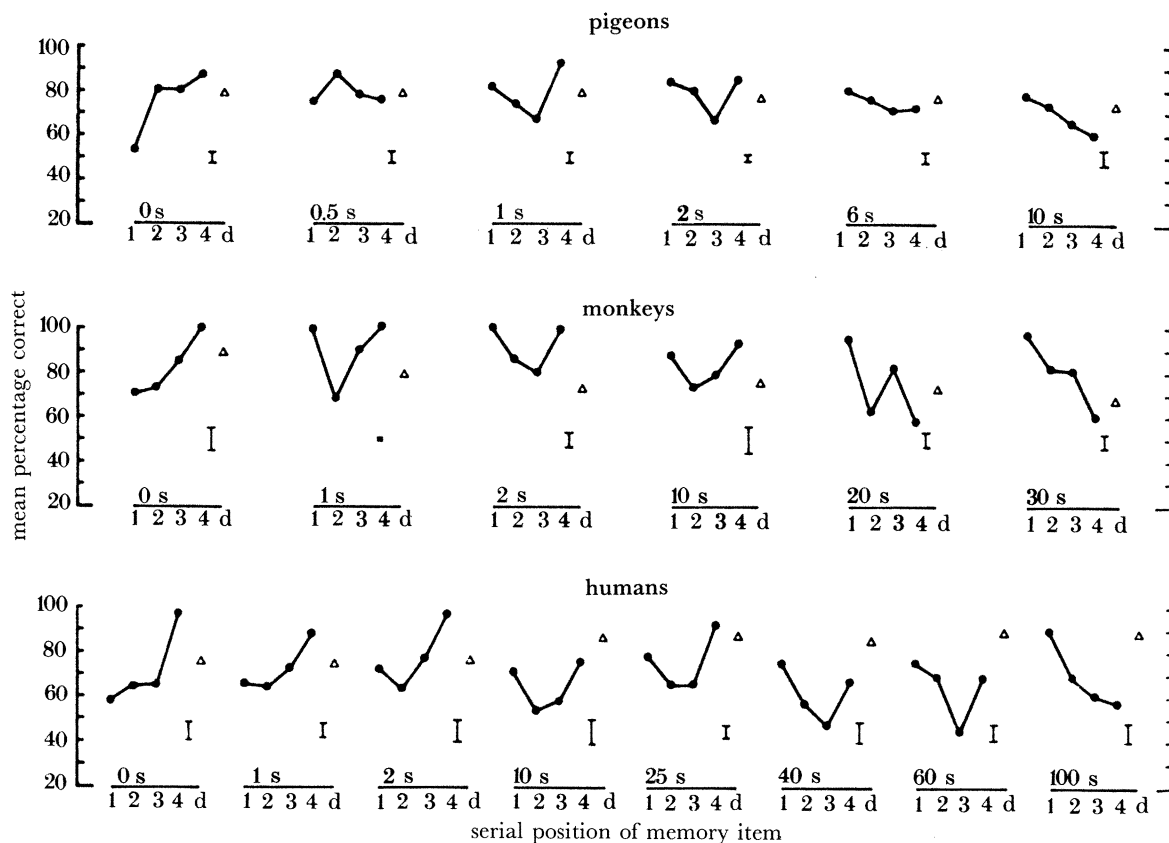


Figure 3. Performance as a function of delay and order of presentation in pigeons, monkeys, and humans. Note the decay of retention for the last item in the series as a function of increasing delay (the 'recency effect') and the preservation of retention for the first item ('primacy effect'). (From Wright *et al.* (1985) with permission.)

the animals had acquired the general rule, i.e. there was double dissociation.

How a short-term system, or process, is incorporated in an information processing flow is another matter which has occupied cognitive theorists greatly (see, for

example Baddeley (1986)). Here it is important to make two points in passing. First, if STM and LTM components can be doubly dissociated, thus challenging the ancient idea (still widely held) that the only route into LTM must be through STM. Secondly,

STM, as a designation, unfortunately is used in a variety of ways throughout a diverse set of phenomena, one of which is to refer to a hypothetical consolidation period, inferred from the retrograde amnesia caused by concussion and other traumas, or by pharmacological agents. There is no necessary connexion between that use, which itself raises all sorts of interesting but complex issues, and the one to which I refer.

The comparison of STM, of course, must be with long-term memory and here there *are* genuine sub-categories in terms of 'processing modularity', i.e. systems with qualitatively different demands and properties and neurobiological underpinnings. Again, one cannot be exhaustive, but I consider briefly four proposed long-term systems. First, knowledge memory systems, of which there are probably a number of different independent examples. Knowledge is, of course, a very general term, but the first level to which I refer is that of environmental knowledge; familiar objects, faces and places. Clinically there are patients who are agnostic for each of these categories (Warrington 1982, 1985). The pathology shows different loci (Damasio *et al.* 1989), and modern imaging techniques will no doubt continue to sharpen up this aspect. In the primate we have some idea of the cortical pathways that are critical for the formation of visual prototypes and their associated meanings, as well as for a dissociable route for spatial memory. Without going into these matters further, or the clinical distinction between stimulus categorizing and associative meaning, the point here is that agnostic patients need have no STM impairment, nor any problem in remembering individual events: a patient may say, 'I did not know what that picture [of a common object] was when you showed it to me yesterday, and I *still* do not know what it is!'

At quite another level, dissociable from this, we can consider simple and stable associations, as in classical conditioning. These, too, provide meanings in terms of valences, as in reward associates. We know that conditioning can be acquired by rats without any neocortex, and we shall hear (see Thompson, this symposium) that certain anatomical circuits in the cerebellum are critical for some aspects of conditioning. Some taxonomists would include associative learning under 'habits', referring to all tasks with slow incremental acquisition in which the associative rules linking S-S and S-R are unchanged, as in discrimination learning. But I think the important point is not whether learning is slow and incremental – it might be accomplished in one trial – but the stability of the relationship that has to be acquired. I prefer the more cumbersome term of simple, stable associations.

Another long-term system, not always separated from those we have just been considering, involves the acquisition of skills. Skills, in turn, may be cognitive, as in learning arithmetical skills, or perceptual-motor, as in learning to ride a bicycle, or to read in a mirror. They are sometimes lumped together but almost certainly they will be fractionated. These are sometimes called 'procedural' memories. Whatever designation one uses, it is now abundantly clear that the capacity to acquire skills can be perfectly intact in patients

whose memory for unique events is so severely impoverished as to make constant custodial care essential.

It is such patients, with the amnesic syndrome, who perhaps have been most thoroughly studied neuropsychologically and by cognitive psychologists (see, for example Cermak (1981); Schacter *et al.* (1988)). These patients do not acknowledge memory of an event a minute or two after it has occurred, nor do they normally behave overtly as though they have retained such information. As we have seen, their short-term memory need not be impaired at all; it might even become supernormal. But even though these patients will deny the outcome verbally, classical conditioning can be acquired and retained over long periods (Weiskrantz & Warrington 1979) (figure 4). There is evidence of good learning of visual discriminations, e.g. consistently favouring a particular stimulus, which in training had a coin under it, in preference to an unrewarded one (Frith *et al.* 1990). The patients can learn new motor and cognitive skills (figure 5), again without any acknowledgement of 'memory' for having done so (Corkin 1968; Brooks & Baddeley 1976; Cohen & Squire 1980), and are sensitive to semantic and phenemonic rules that apply across a range of items to be primed (Winocur & Weiskrantz 1976). Whether they can learn new semantic knowledge is still not clear – if they did they would not acknowledge it – but they do retain semantic knowledge acquired premorbidly and there is some indication that they can learn new associations based on priming (Schacter & Graf 1986). We know a lot about the general anatomical underpinnings of this debilitating and stable defect. They involve medial temporal and related diencephalic structures. The point is here that the amnesic syndrome can be dissociated both neuropsychologically and anatomically (Weiskrantz 1987) from other memory disorders and capacities.

There is one other category that seems to differ in essential regards from all others we have discussed so far, this is priming facilitation that occurs through repetition. No new association or information is added but the probability of performance is altered merely through the occurrence of an event or response (Jacoby 1983; Scarborough *et al.* 1977; Tulving 1983). It is typically tested by speed of reaction time in, for example, lexical decision tasks, or in the probability of correct identification of complete words or pictures from partial cues (figure 6). The facilitation may be long lasting, up to weeks or even months, without the subject necessarily being aware of it, and decays at a different rate from recognition memory (Tulving *et al.* 1982). It was long ago shown to be intact (figure 7) in severely amnesic patients (Warrington & Weiskrantz 1968, 1974) when explicit recognition was no better than chance, and is highly resistant to destruction by specific brain lesions (Shimamura 1986), given intact sensory input channels, although a recent report has shown that it can be affected by a benzodiazepine drug that may be assumed to have fairly widespread effects through the cerebrum (Brown *et al.* 1989). The evidence suggests that priming may well be a feature of neuronal pools along any route in the nervous system

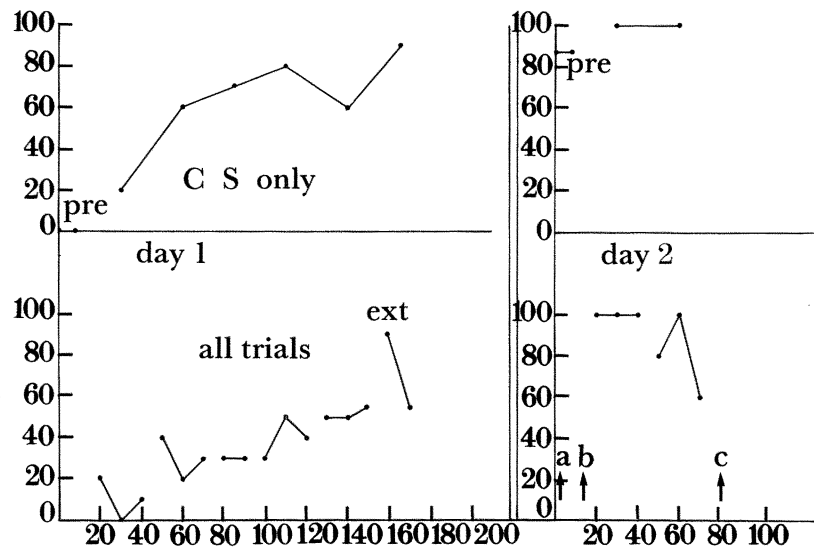


Figure 4. Eye-lid conditioning in an amnesic subject (Korsakoff psychosis). Percentage of responses in top panel are for 'probe' trials with the conditioned stimulus (light plus tone combination) in which no air puff (the unconditioned stimulus) was delivered. Breaks in the lower panel denote short rest periods, during which interviews were recorded (ext, extinction trials). No evidence of acknowledged memory was obtained in the interviews. (From Weiskrantz & Warrington (1979) with permission.)

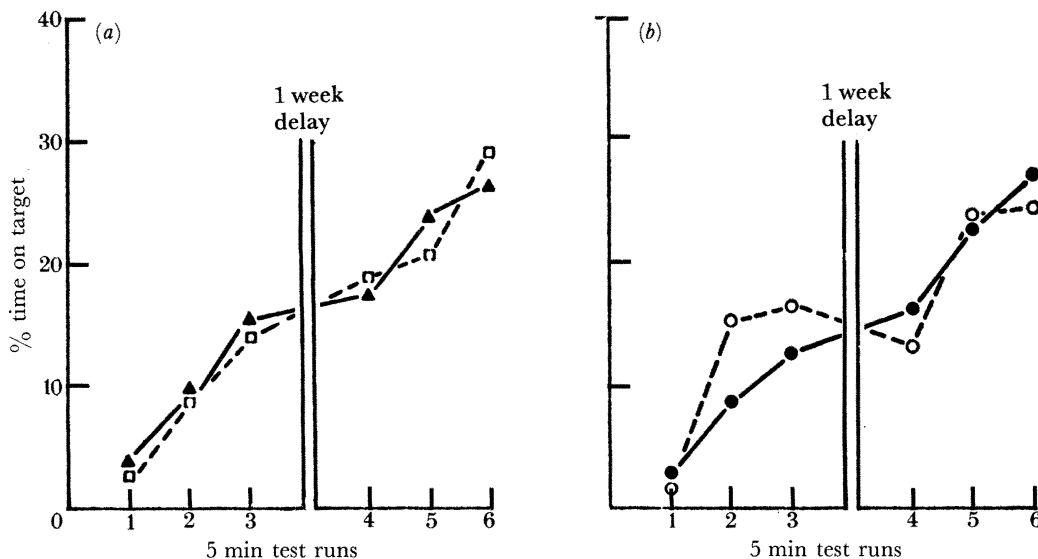


Figure 5. Normal learning and retention of motor skill tasks by amnesic and control patients. Despite good retention of the skill no acknowledged memory for the task was manifest by the amnesic subjects; (a) (▲), encephalitics; (□), control; (b) (●), Korsakoffs; (○), controls. (From Brooks & Baddeley (1976) with permission.)

involved in sensory or motor performance or higher order processing, perhaps with the critical neuronal phenomena occurring at ganglionic junctions having a minimal critical mass. Phenomena such as 'kindling' (Majkowski 1981), seen in the amygdala, or 'long-term potentiation' (Teiler & Discenna 1987), studied principally in the hippocampus but also found elsewhere in the nervous system (both of which are examples of long-term changes in sensitivity as a result of stimulation of underlying structures), may be related to the neuronal underpinnings of priming.

These examples of different putative memory systems are based on dissociations, which are to the neuropsychologist what the dissecting scalpel is to the anatomist. Evidence for independent systems comes from double dissociations and not merely double, but multiple dissociations, have been observed between

STM, knowledge, skills, simple associations, and episodic memory. However, it has been clear for some time that double dissociations are not infallible for inferring independent systems (Weiskrantz 1968); they can, in principle, be found from treatments acting within a single system. Shallice (1988) recently reviewed and analysed the logic of how such factors as different task demands can give rise to misleading or empty double dissociations. Cognitive psychologists have also had fun generating their own examples by manipulating task variables and instructions and some, as a result, have grown shy of the whole approach (see, for example Baddeley (1986); Roediger *et al.* (1989)). They have, for good reasons, found it difficult to find sharp indicators of each of the systems argued to be independent. In the face of logical inconclusiveness, over-abundance and complexity of dissociations (much

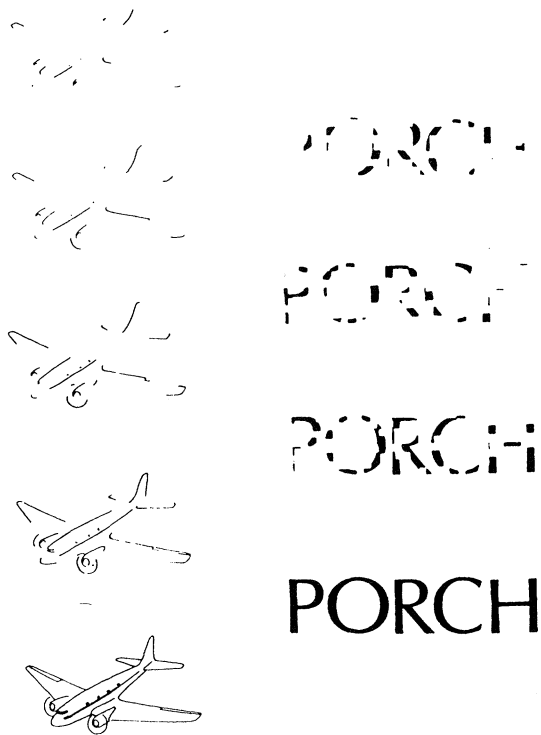


Figure 6. Example of fragmented material used to show priming in amnesic subjects. After presentation of more complete exemplars subjects can identify items with less complete (more fragmented) stimuli. (From Warrington & Weiskrantz (1968) with permission.)

of it self-generated in cognitive psychology) they have retreated to more global information processing characterizations. In its most extreme form this assumes that there is just one system that can be driven by different inputs and at different levels and possesses memory as a feature of its operating characteristics. This argument suggests that higher animals possess one large information-processing network, with independent inputs, within which enduring changes take place (at the nodes, say, or within hypothetical layers of cells) that lead to what might be called memory as weightings within the whole system change, as in PDP networks.

Indeed, it has been claimed that double dissociations can be found even in PDP networks, by hypothetical lesioning of different parts of the network (Shallice 1988). How pleased Lashley would be, as it was the discovery of neuropsychological double dissociations that certainly fired the retreat from mass action theory.

The problem remains of how to deal with a possible surfeit of minor dissociations, or the logical inconclusiveness of even the major ones. There are a few logical points to make and one important strategic point. The logical points are these: I follow Shallice in distinguishing not only types of dissociations, double or single, but in their strength and purity. He concludes that 'lesions within a properly distributed memory system will *not* give rise to a classical or strong dissociation'. Indeed, a feature of distributed memory systems is that small damage in any region would have widespread consequences throughout the system;

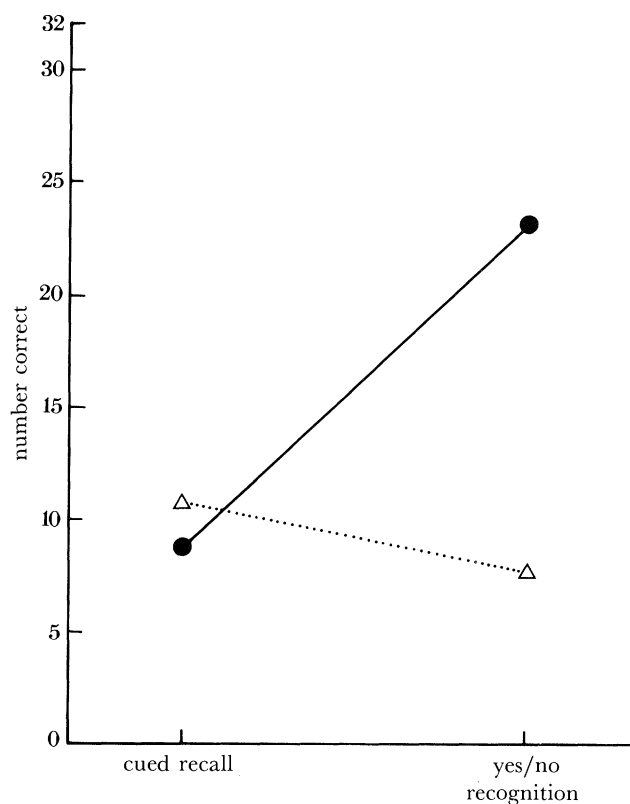


Figure 7. Normal performance by amnesic subjects in identifying 'primed' words after a 4-min interval when retention was tested by cueing with the initial 3 letters and impaired yes/no recognition of other words from the same list; ( $\Delta$ ), amnesics; ( $\bullet$ ), control. (From Warrington & Weiskrantz (1974) with permission.)

Marr (1976) has pointed out the advantages for computation systems of division into independent subparts. Shallice's 'weak' dissociations (what I have called 'relative double dissociations', Weiskrantz (1989)), are abundant and often part of the cognitive noise that keep cognitive theorists so busily occupied. The dissociations we have considered here are strong, what I have called 'absolute' double dissociations.

More realistically, perhaps, a network processing advocate might go so far as to say that humans are a *set* of independent information processing networks each of which behaves as a distributed system. For different forms of representational knowledge model this seems feasible. But, of course, it is already a concession to our present position and in a way that blunts Occam's Razor to the point where hairs are no longer cut, let alone split.

A second point is that because double dissociations are not logically sufficient, they are nevertheless logically necessary for demonstrations of independence. Logical insufficiency is not the same as pragmatic impotence and, while it is self-evident, it still seems necessary to repeat that when a double dissociation is found it is perfectly consistent with one assumption of independence. I say this because I suspect that useful and strong inferences about independence may be missed by those who feel uncomfortable with logical uncertainty. I suspect that some contemporary cognitive theorists have, by their own unease, failed to allow themselves to accept such important and now universally accepted dissociations as that, say, between scotopic and photopic visual systems, because they could not readily find a stimulus that did not affect both putative systems, because they would decline to look at something as irrelevant as hardware (i.e. the morphology of the receptors) and because trivial double dissociations could be too easily generated (for example between after-images of red and pink).

A third point is also obvious, although it too bears repeating. This is that in the study of whole animals, at least of organisms of moderate complexity, there is no such entity as a 'pure learning or memory task'. It does not exist, nor will it ever. In reading through cognitive psychology texts it is obvious how much confusion this simple consideration injects into their experimental analysis, especially as more and more clever variations of related tasks are generated. A so-called STM task also inevitably involves long-term event memory, plus knowledge, motivation, vigilance, and so forth, so it is no wonder that complex shifts in outcome occur with even slight experimental manipulations. Many theorists seem to become obsessed with trying to explain the variance rather than the main effects. It is also one place where correlative neuroscientific information (about affected neural pathways for example) might be helpful in trying to understand the basis of 'trend' or 'relative' dissociations in relation to 'strong' or 'absolute' dissociations for example.

Given a number of double dissociations, none logically leading to an inescapable conclusion, what strategy could be employed and what criteria sought to produce solid inferences about independent systems? Here is where pragmatics rather than logical syllogisms

are relevant and the key lies in conviction and economy emerging from converging evidence and argument. Inferences about multiple memory systems can refer to considerations external to the closed world of experimental cognitive psychology, on the one hand and raw neuropsychological dissociations, on the other. There are two such external considerations. The first concerns evolutionary constraints and advantages, the second, neuroscience.

The evolutionary analysis has been put forcefully by Sherry & Schacter (1987). They argue from comparative evidence that multiple memory systems can be inferred when functional demands between different retention tasks are incompatible, i.e. when 'systems are specialized to such a degree that the functional problem each system handles cannot be handled by another system'. They analyse, as exemplars, different requirements of memory for song, imprinting and memory for spatial locations in birds, and the distinction between incremental habit formation and memory for unique episodes in primates. They conclude that there exist cases of 'memory with restrictions on what is admitted to memory, restricted periods during which this can occur, or long periods of retention without additional opportunities for acquisition or rehearsal'. Such specialized systems evolve when the capacities of any system are incompatible with a new environmental demand. Their characterization both of knowledge and of skills, for example, is in terms of 'preservation of *invariances* across episodes'. While a system concerned with skills must, of course, have detailed information about episodes to extract invariance between them, 'the critical point is that this detail is not retained after the invariant features have been extracted'. In contrast, the event-memory system is designed 'to preserve *variance* across episodes, rather than invariance. When an animal remembers the location of stored food, or when and where a predator was encountered, a system is needed that can store and later provide access to a wealth of features that are uniquely characteristic of each episode. A system that specialized in the preservation of invariance would not be particularly helpful in such a situation'.

Cognitive processes, like systems, can be functionally incompatible, in precisely the way that Sherry & Schacter use the term, and require to be constrained in just the same way as the dissociations we have already considered. A processing approach based, for example, on the distinction between 'data driven' and 'concept driven' modes of processing, may be useful in helping to characterize the defining functional characteristics of putative memory systems and need not be logically incompatible with them.

Functional evolutionary arguments, based on the detailed analysis of animal behaviour in real life environments, thus both supplement and constrain the admission of dissociations between classes of memory tasks. A second appeal to outside evidence is to neuroscience. It is of the nature of neuropsychological evidence for dissociations that not only are memory tasks dissociated, but neurological conditions or treatments are also dissociated. This evidence suggests that different locations within the nervous system are



associated with or critical for the maintenance of different capacities. Localization is not the name of the game, but it *is* an extremely useful starting point, and the systems which are doubly dissociated from each other considered here do each have their characteristic anatomical underpinnings: for stable associative conditioning, the cerebellum; for event memory, the medial temporal-diencephalic limbic and frontal lobe structures; for knowledge systems and representations, the temporal and parietal neocortex; for many perceptual-motor skills, possibly either cerebellum or basal ganglia or both. As priming cannot readily be doubly dissociated from other phenomena and is widely immune to neurological insult, mediating structures would appear to be widely distributed. I have reviewed some of the anatomical evidence elsewhere (Weiskrantz 1987) and recently there has been some interesting correlative evidence from a blood-flow study of a functional difference between episodic and semantic information in human subjects during silent reminiscing, reported by Tulving (Tulving 1989).

Localization is sometimes dismissed by cognitive neuropsychologists as mere modern phrenology or as mere hardware. But localization is just the start. This discussion meeting will contain examples of more refined physiological, anatomical, and neurochemical analysis of specific systems. This evidence is cumulative and accumulating quite rapidly. Some cognitive neuropsychologists are parasitic upon neurology in that they use the evidence from neurological disorders but they often seem unwilling to become acquainted with possible underlying mechanisms associated with the revealed dissociations, claiming that this is mere hardware. It leads to a kind of candy floss neuropsychology, brightly labelled, complexly reticulated, full of growth but shifting in substance. The neuroscientific underpinnings are not merely insubstantial hardware (see Weiskrantz (1968)). The study of neural mechanism itself both constrains and suggests functional organisation and properties. This can be seen clearly in the visual domain, for example, in terms of its modular organisation; the same considerations apply in principle to all other systems. It is time that we abandoned self-imposed agnosia based on a model of aneural man.

So converging evidence from neuropsychology, animal behaviour, and neuroscience all suggest multiple, parallel, potentially independent but normally interacting memory systems. For the generation of inferences about multiple systems, it is clear that wide-angled neuropsychology (what perhaps used to be called physiological psychology) lies at the centre of any attempt to make inferences about multiple systems because it extends laterally into the analysis of tasks and all their settings in one direction and to the assimilation of physiological, anatomical and neurochemical evidence in the other.

There is another more restrained strategy that may seem different from the one I have outlined, which is to focus on some particular class of learning phenomenon, for example imprinting or classical conditioning, and to work out the relevant neural mechanisms in detail. This is proving very rewarding, as may be seen

elsewhere in this symposium. But one might ask, 'the neural mechanisms of *what*? To be certain of the boundaries of the class under consideration it is necessary to show whether it applies to other learned behaviour. Multiple memory assumptions suggest what contrasts might be pursued. Horn and Bateson show the power of this functional dissection very well in their contrast between imprinting, operating conditioning and discrimination learning (Horn 1985, 1988). Without such functional dissections, the same that lead to inferences based on double dissociations, boundaries remain unclear.

All memory systems, of course, share one underlying requirement, i.e. that some process be made to bridge a time-gap, (and this requirement is not just restricted to memory systems). No matter how many systems there are in memory they are all contained in organized sets of neurons and neurons are just neurons. It may be that all the systems we have considered share just a few possible underlying cellular processes that allow this to happen. It seems unlikely that the same cellular mechanisms will account for everything from STM, to representations in knowledge and priming, but I do agree that one can do a lot with sensitization and increased synaptic conductivity alone. Whatever the cellular mechanisms they will not independently address questions of organisation and capacity at the systems level; it is equally necessary to consider cellular mechanisms to account for the richness and diversity of forms of memory as it is to reduce everything to the cellular level. If there are independent multiple memory systems it follows that none is simply the composite of a set – no matter how large – of any other. Therefore, when cellular mechanisms are studied in the context of, say, alpha conditioning, delayed non-matching from sample, or whatever, it cannot be assumed that these mechanisms can necessarily be generalized.

In conclusion, the illustrative material reviewed here supports the general thesis that memory, like perception, is not holistic and that separate components can be subtracted without damaging the remainder. The anatomical details suggest that some memory systems involve virtually all levels of the nervous system, others are highly specific and relatively confined, with a spread of distribution between these extremes. Particular memory systems are confined to particular species. It would be rash to suggest that vision could ever be completely understood by studying the ear and its connections, similarly it may be just as unwise to study memory as though it were comprised of a single system or a single mechanism.

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