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Predicting Syndromes of Amnesia from a Composite Holographic Associative Recall/ Recognition Model (CHARM)

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The composite holographic associative recall/recognition model (CHARM) is used to predict the amnesia syndromes that are expected under conditions of discrete lesions to different components of the model. The components that are needed to allow recognition, recall, and rehearsal are: (1) perceptual/lexical processing and pattern identification, (2) consciousness or working memory, (3) association formation, (4) composite storage, (5) novelty monitoring and control, and (6) retrieval. Deficits in each of these components will have specific effects on memory, generating characteristic profiles of performance. Comparison of the profiles exhibited by patients to the component-based profiles predicted by the model identify the component impaired in a given patient, and connect the memory impairments to the underlying infarcted brain structures. The model, thus, relates the memory tasks to the particular memory components that allow enactment of those tasks, and shows how the dysfunction of particular components produces specific impairments.

INTRODUCTION

Mayes and Downes (this issue) point out that: ''Most hypotheses about the functional deficits in amnesia have tended to assume that the syndrome is unitary and has a single underlying functional deficit'' (p.4). This article challenges that view, and proposes, instead, that different syndromes of amnesia can be produced as a result of the breakdown of any one of several components of memory, each of which will produce a characteristic profile indicative of the locus of dysfunction. The many different unitary factors that have been proposed as the locus of amnesia include encoding, retrieval, interference, storage, forgetting, type of information, failure of attention, and failure of deep

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processing, to give a few. The unitary cause position is undermined first of all by a striking lack of agreement over which factor is critical. It is further questioned by the fact that (a) different memory tasks differentially show impairments or the lack of impairments, (b) results from one lab often fail to replicate those of other labs, (c) amnesics with different lesions show different patterns of impairments, and (d) there appears to be general agreement that normal memory itself is not unitary but instead requires different components to function. It thus seems reasonable to explore the alternative view of human amnesia as a multifaceted phenomenon whose analysis must take account of the component operations and their interaction.

To enable a more detailed understanding of the necessary components and their function, I turn, here, to a formal model of human memory—CHARM (composite holographic associative recall model)—which has been shown to account for a broad sweep of human memory data (Metcalfe, 1990, 1991, 1993a,b, 1994; Metcalfe Eich, 1982, 1985). My working assumption is that each of the components needed in the formal model, to allow it to enact the tasks that normal humans can perform, is anatomically discrete (though connected to other components and interactive) and could break down separately. In this paper, I outline the implications of dysfunction in each of the components in terms of which tasks are expected to show impairment, and also in terms of the reasons for those impairments. To the extent that diseases and infarcts in humans affect components in a correlated manner, rather than individually, the analysis given here will be oversimplified. Although the localisation of components is a topic of great interest and importance, the formal model itself does not stand or fall on the assignment of components to anatomical locations.

AN OUTLINE OF CHARM

The basic components of the CHARM model are shown in Fig. 1. The model assumes that *sensory, perceptual, lexical processing, and identification* of events have already occurred before events are at the level of processing necessary for encoding, storage, and retrieval in the episodic memory system which is deeply embedded in the cognitive system. Formally, the events in the model are represented as multidimensional vectors of features that may vary in their similarity to one another, and over which attentional focusing may highlight some features and not others. Two such items, at the *level of consciousness (or, in working memory)*, may be *associated with one another by the operation of convolution*, which weaves all of the features into a complex new associative vector. Multiple associations are stored by being superimposed or added into a *composite memory trace*, which is itself a vector. The weighting on the association being entered into the composite trace is determined by a *monitoring and control circuit* that calculates novelty or familiarity and adjusts the weighting of the incoming event as an inverse function of its similarity to the

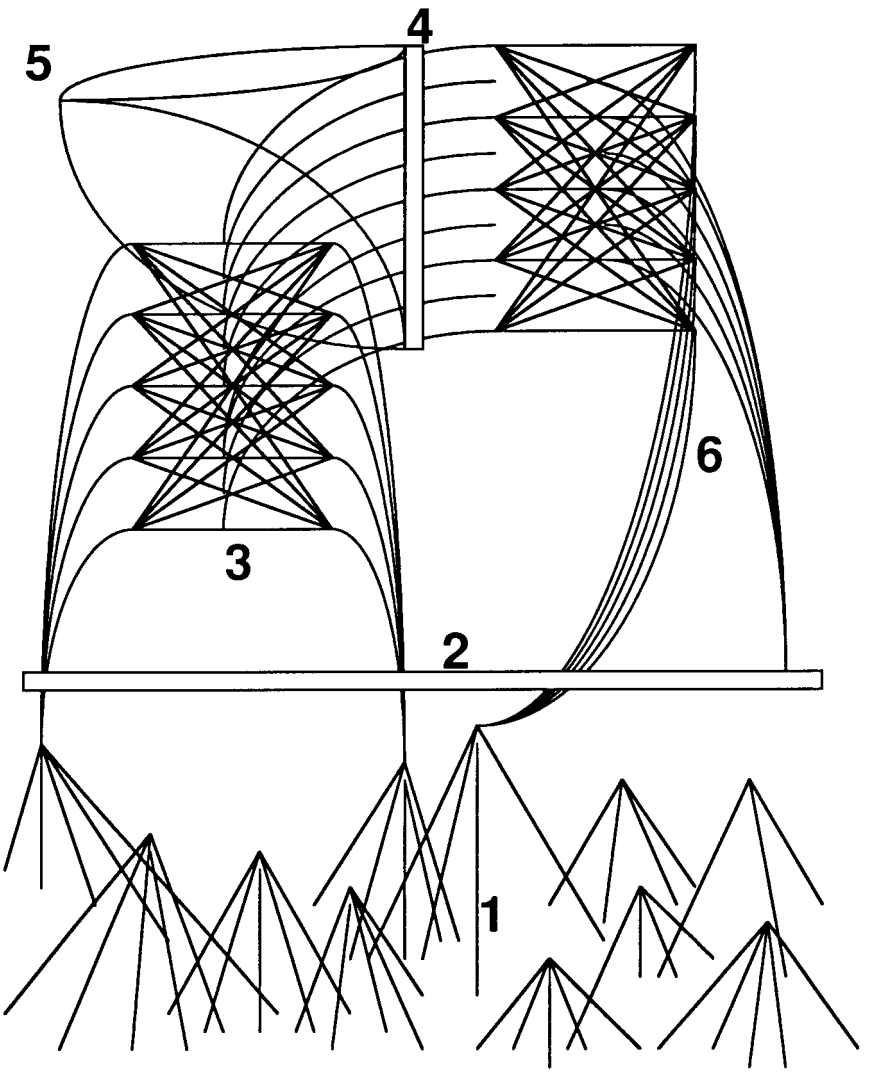

FIG. 1. An overview of CHARM. The components are (1) the perceptual system and lexicon, (2) working memory or consciousness, (3) association formation by convolution, (4) the composite memory trace, (5) novelty monitoring and control, and (6) retrieval by correlation.

composite trace. At time of test the retrieval cue, itself a vector of features that is available to consciousness, is *correlated* with the composite memory trace resulting in a retrieved vector, which is also at the level of consciousness. This retrieved item may be systematically distorted and is always noisy. Therefore the retrieved item must be *identified* (again in the lexicon or perceptual processor) if a discrete word is to be the response, or some other decision, such as a recognition judgement, must be made. The implications of selective impairment in each of these components is given in summary form in Table 1 and discussed in the sections that follow.

TABLE 1
CHARM Model Predictions

Component Damaged	*Impaired*	*Spared*
Perceptual and Lexical processing		
	deficit not labelled amnesia	
Working Memory		
Mild to Moderate Impairment	span	cued recall
		free recall
		rehearsal (elaborative and rote)
		priming
		categorisation
		recognition
		normal forgetting
		novelty response (Von Restorff)
		release from PI
		primacy
		spacing effects
		feeling of knowing judgements
Severe Impairment	span	
	cued recall	recognition
	free recall	priming
	elaborative rehearsal	
	forgetting rate	
Association formation (convolution)		
Mild to Moderate	graceful degradation of everything given under severe impairment	
Severe Impairment	cued recall	span
	free recall	priming
	elaborative rehearsal	categorisation
	binding tasks, within items	familiarity-based recognition
	forgetting rate	novelty response
	new association ''priming''	release from PI
		primacy
		spacing effects
		feeling of knowing judgements
	anterograde amnesia	retrograde amnesia

Composite Memory Trace		
	degraded performance on all episodic memory functions	span
		priming
	both retrograde and anterograde amnesia	categorisation
		familiarity-based recognition
		novelty response
		release from PI
		primacy
		spacing effects
		feeling of knowing judgements
Novelty Monitoring and Control		
	feeling of knowing judgements	span
	release from PI	priming
	novelty response: Von Restorff effect & P300	recognition
	spacing effects	cued recall
	exacerbated interference	
	primacy	
	habituation	
	memory tasks enhanced by clustering and categorisation	
Retrieval	impaired performance on all episodic memory functions	span
		priming
		fluency-based recognition
	both retrograde and anterograde amnesia	

Perceptual Identification, Lexical and Response Processing

Normal Functioning

It is assumed that events that impinge upon the senses are perceptually analysed prior to entry into the episodic memory system. Such events may be characterised as patterns of features, or as vectors, and if these events are words they activate a representation in a pre-episodic mental lexicon. Further processing in the episodic memory system, then, is predicted upon perceptual analysis. Plasticity may occur in this pre-episodic system—it is not assumed that only the episodic memory system exhibits plasticity. Hence, people can learn skills, concepts, categorisations, and show adaptive responding or learning and conditioning to a variety of stimuli that may never enter into the episodic memory system. This model, then, is consistent with the views of other theorists (Mishkin & Petri, 1984; Moscovitch, 1982, 1992; O'Keefe & Nadel, 1978,

Schacter, 1987; Squire, 1992; Tulving, 1985) who distinguish among a core episodic system and other more peripheral systems that underlie skill effects, priming effects, comprehension, motor and linguistic competence, and learning.

Mayes and Downes (this issue) note that amnesics show sparing of intelligence as measured by the Wechsler Adult Intelligence Scale. Presumably the tasks on this test (aside from those on the memory scale) can be enacted by the perceptual/motor/lexical system that exists outside the episodic memory system proper. A well-established phenomenon in the amnesia literature is that repetition and associative priming measured by lexical decision, fragment completion, homophone spelling, free association, and word identification tasks are spared even though recognition and recall—episodic memory tasks—are impaired (Buschke, 1965, 1968, 1984; Gardner, Boller, Morienes, & Butters, 1973; Graf, Shimamura, & Squire, 1985; Graf, Squire, & Mandler, 1984; Jacoby & Witherspoon, 1982; Moscovitch, 1982; Schacter, 1987; Shimamura, 1986). This result is consistent with the structure of the model in which it is necessary to go through the perceptual system to get to the episodic system. Even if the episodic system were damaged, the perceptual system and the lexicon feeding into it could support the priming effects and the effects of skill learning.

Memorial Consequences of Impairment of Functioning

If the perceptual system, the lexicon, or the output system were damaged in isolation, the syndrome would not be labelled amnesia, but rather aphasia, agnosia, anomia, prosopagnosia, dyslexia, cortical blindness or deafness, and so on, depending on the nature and location of the deficit. Impairments in these systems will have consequences for later remembrances of the events that occurred, of course.

Consciousness/Working Memory

Normal Functioning

If items are to be associated with one another (allowing later retrieval) then they must be capable of existing in a working memory buffer—available to consciousness—such that they can participate in the associative operation. Without a temporal store, holding *at least two* mental items, the episodic memory system could not function. In fact, it appears that human working memory can hold more than the minimal two items. Furthermore, retrieval, in the model, brings back into consciousness or working memory representations of past events.

It is assumed that a particular level of representation, which I have called the ''item'' level (Metcalfe & Murdock, 1981), is needed for phenomenological awareness. If representations are not yet transformed to this form, or if they are further transformed beyond this form, they are not consciously interpretable. An

analogy may be useful. We can only hear sounds within a certain bandwidth. But, if it serves our purposes, sounds can be transformed so that we can no longer hear them, by operations such as frequency modulation. Radio waves are hearable sounds that have been so transformed for transmission through the air. With the help of a radio receiver these signals can be reconverted into a form that is again available to our hearing sense. Similarly, it is proposed that there are forms of active neural representations that are not available to our conscious sense (e.g. the patterns that exist on the retina or at the level of the cochlea, or in memory proper), and a form of representation that is available to consciousness. This level of representation exists after lexical and perceptual representation and processing, and before association formation and storage in episodic memory. Items at this level of representation can exist in working memory, and focal attention may be directed to them. This level of representation is a necessary but not sufficient condition for entry into the episodic memory system.

The anatomical location of working memory is under investigation. Goldman-Rakic (1987) has demonstrated that particular neurons in the frontal cortex of monkeys fired selectively when the stimulus was not present but was being ''held in mind''. Other researchers have implicated the occipital and parietal lobes in related functions, so working memory may or may not be restricted to frontal cortex. Baddeley (1986, 1994), Shallice (1993), and others (McGlynn & Schacter, 1989; Stuss & Benson, 1986) have suggested that the frontal lobes may be implicated in the central executive and control functions of working memory, as well as in the function of holding items in mind.

Memorial Consequences of Impairment of Functioning

Attentional Disorder. An impairment in working memory, as is sometimes seen in Alzheimer's, Korsakoff, and frontal lobe patients, might not always be labelled amnesia, but could rather be diagnosed as an attentional or a motivational deficit.

Impaired Span With Normal Long-term Memory. Measurement of a patient's memory span provides a method of assessing the integrity of the working memory component. The CHARM model is resilient to very restricted working-memory capacity limitations—an impairment in this system will only show up as an impairment in the remainder of the episodic system if the deficit is so severe that the person cannot hold two items in working memory. If span sinks below two, the model predicts that the person will be unable to form inter-item associations. In the absence of inter-item associations, rehearsal will be impaired, although the system may be able to enact rote repetition of one item. Note, however, that a patient could show considerable deviation from normal working memory span and still be well above the level necessary for adequate associative processing, rehearsal, and retrieval from the core episodic memory

system, accounting for the sometimes-observed finding of a working or short-term memory impairment in the absence of a long-term memory deficit (Shallice & Warrington, 1970). An alternate explanation given by Baddeley and Hitch (1974) is that working memory has three components, and could rely on alternative components given impairments. Although it is likely that working memory is complex and may have separable components, we may also have overestimated the requirements for entry of associations into long-term episodic memory, as suggested by CHARM.

The deficits that follow refer to implications of severe restriction of working memory, below the minimum requirement of two items. As long as the capacity is not restricted below two items, all of these functions may be normal, even in the presence of abnormally low span measures.

Impaired Associative Recall but Unimpaired Recognition. If a person cannot maintain two items in consciousness he or she will be unable to enact inter-item associations. Under such conditions, retrieval-based recognition memory, which depends only on the association of one item with itself, may be intact, but cued recall, which depends on inter-item association, will be impaired. Hirst et al. (1986) and Hirst, Johnson, Phelps, and Volpe (1988) found this result with amnesic patients (cf. Squire et al., 1990).

Impaired Elaborative Rehearsal. Rehearsal is a process requiring explanation because the to-be-rehearsed items are not themselves available to consciousness. Therefore, in the model, rehearsal, like recall, entails retrieval. Rehearsal depends on the two items in consciousness first being associated. Then one of the items that is available to consciousness is used as a retrieval cue (see Metcalfe & Murdock, 1981). This conscious item is correlated with the composite memory trace to retrieve a representation (of the associated item) that is itself in a form available to consciousness and which can be identified by the lexicon and used as a retrieval cue to bring a different item (with which it was associated) into consciousness. One item retrieves the other until a new item enters consciousness from the senses rather than from memory. That new item is associated with whatever is being rehearsed, and is then itself used as a retrieval cue, to continue the process of rehearsal.

This simple rehearsal loop produces patterns of data like those found in studies of overt rehearsal (e.g. Rundus & Atkinson, 1970; Murdock & Metcalfe, 1978). People rehearse items in a backward graded function in which, in any given rehearsal interval, they say the last item most frequently, the next to last item next most frequently, and so on. The rehearsal loop in the model predicts that one would mostly think about or rehearse events from the close past, but occasionally rehearse events from the distant past (depending on their connections or associations to the present). Once people are rehearsing events

from the more distant past, they would tend to rehearse events that were associated with them for a period of time before once again being pulled into more ongoing present thoughts.

If working memory is so severely limited that only a single item can be maintained, then elaborative rehearsal, which is based on associative recall, cannot occur and only a single item will be repeated. Cermak, Naus, and Reale (1976) studied the overt rehearsal patterns of Korsakoff patients. They found the patterns of data produced by these patients to be abnormal—the Korsakoff patients tended only to rehearse the last word that was presented, whereas normals rehearsed a number of different words together, during any particular rehearsal interval.

Rate of Forgetting. If rehearsal causes the rehearsed events to be stored in the composite trace (see Bjork, 1988; Rundus & Atkinson, 1970, for evidence bearing on the idea that retrieval of an event, and not just its initial study presentation, is causal in that event's later memorability) then the psychological process of rehearsal may correspond to the construct of consolidation used in the neuroscience literature. Furthermore, if elaborative rehearsal allows events from the non-immediate past to be re-entered into the composite trace, then the rate of forgetting will be related to the extent to which the person is able to enact this kind of rehearsal. If rehearsal is prevented or impaired, the forgetting curve will be steeper than if rehearsal is normal. One would, therefore, expect that patients who have a severe impairment in working memory (to the extent that they cannot hold two items and consequently cannot associate and rehearse normally) will secondarily exhibit abnormally precipitous forgetting.

Free Recall. When normals are asked to free recall, their recall protocols are reminiscent of their rehearsal protocols—they recall a variety of items from the end of the list immediately—apparently chaining from one item to another through an associative network (see Metcalfe & Murdock, 1981), and then shift to the start of the list, chaining again through associated items. According to Cermak et al. (1976), Korsakoff patients' free recall is severely limited, consisting primarily of the last item presented—much as would be expected if they were unable to form the inter-item associations necessary for such chaining (either because the associative operation is impaired or because of severe restrictions of working memory). Indeed, all tasks that require memory for more than one item will be impaired if there is such a severe deficit in working memory.

Association Formation or Binding

Normal Functioning

In the model, two items, represented as vectors (at the level of consciousness), may be associated by the operation of convolution, whereby

all of the features within the two items are bound to one another. The association is a complex interactive combination of all the features of one item with all the features of the other. This transformation renders the association consciously uninterpretable, and retrieval is needed to return either item to a conscious form. Inter-item associations are needed to allow one item to provide a retrieval cue for another item; autoassociations underlie recognition memory, allowing an item to retrieve a representation of itself.

Memorial Consequences of Impairment of Functioning

Because all of the elements within and across items are interwoven by the operation of convolution, this model is a distributed-memory model, and will exhibit graceful degradation of information with impairment or lesion to this component. Only if the entire association component is completely lesioned will the association or binding function fail entirely.

Anterograde But Little Retrograde Amnesia. By some views of human memory, particularly those asserting that remembering consists of redoing the same operations enacted in encoding, the fact that amnesics frequently show anterograde but not retrograde amnesia is puzzling. The dissociation is, however, consistent with the componential model in which associative encoding is one kind of operation (convolution) and is assumed to take place in a different brain location from storage and from retrieval, which is a different kind of operation (correlation). By this view, once events have been associated and stored in the composite memory trace, damage to the area in which the association formation is conducted (presumably the hippocampus) will largely spare the memory for the events that have already been associated and stored, although new associations may not be laid down. Thus, as in the case of HM (Scoville & Milner, 1957) or RB (Zola-Morgan, Squire, & Amaral, 1986), in which the hippocampal damage occurred with an acute onset, new associations and explicit memories tended no longer to be formed after the damage—resulting in anterograde amnesia in the absence of notable retrograde amnesia.

There is one caveat to the claim that with damage to the association formation component, there will be little if any retrograde amnesia. Rehearsal may mediate the storage process of events after the time of nominal acquisition. In so far as rehearsal is needed to allow memories to be consolidated, and rehearsal is impaired when the convolution or association formation component is damaged, it follows that there should be *some* retrograde amnesia for events that were shown prior to the amnesia-inducing trauma because they will receive less rehearsal than they normally would.

Relational Memory. If association formation were selectively impaired, then memory tasks that require the establishment of new relations should be

impaired, as is frequently seen in the data. Thus, the tasks of cued recall, retrieval-based recognition, free recall, and rehearsal should all show impairment. Indeed, all tasks that require the generation of more than one item (at a delay that allows that working memory is not impacting on performance), or that depend on having bound the elements within an event together (as in retrieval-based recognition), should show impairment if the association operation were impaired.

Spared Item But Not New-association Priming. The model predicts that complete obliteration of the association formation operation should still allow spared item priming, via the lexicon and perceptual systems, but, because association formation between events represented beyond the level of consciousness (although not the simple associations underlying classically or operantly conditioned responses) is thought to occur only in the convolution component, patients with complete inability to form new associations should show impaired priming of new associations. Graf and Schacter (1985) presented subjects with unrelated pairs, such as WINDOW–REASON, either embedded in a meaningful sentence or simply as pairs. Subjects were then given a stem completion task, either with the same context (WINDOW–REA____) or a different context (OFFICER–REA____). Both normals and amnesics benefited from the list context, in contradiction to what the model would, at first blush, predict. However, this priming may have resulted because of feedback from a *partially* intact associative component. As the association formation operation in the model is distributed, partial damage is possible and the result would be ''graceful degradation'' of new association ''priming''. Schacter and Graf (1986) reported that the associative effect only occurred for *mildly* amnesic patients, and was not found with severely amnesic subjects. Similarly, Shimamura and Squire (1989) found *no* associative priming effect with patients who showed severe amnesia. Graf and Schacter's (1985) original results do point to feedback from the episodic system onto the lexical system in normals and mildly amnesic patients, as do other results such as subtle differences in the temporal course of priming effects in normals and severe amnesics.

Recognition Memory. Recognition memory responses, in the model, can stem from two sources. First, the normal recognition procedure in the model involves retrieval from the composite memory trace. The recognition probe is correlated with the composite trace, and then the item that is retrieved is matched to the probe. If it matches, above a certain criterion, then the model calls that item old. However, recognition could, under some circumstances, also be enacted by assessing the fluency of primed items in the lexicon or perceptual system (see Jacoby, Kelley, & Dywan, 1989, and also see Mandler, 1980). The former kind of recognition should suffer from impairment to the associative operation; the later kind should be unaffected.

Mishkin and Murray (1994) have reported that monkeys with surgically induced bilateral hippocampal lesions were able to enact an object-recognition task. Assuming that the hippocampus is the site of association formation or binding, it would appear that one would have to account for this result by recourse to the possibility that recognition in their task was based on feature fluency rather than on event retrieval. Indeed, nothing in their task forced the use of associative coding or binding information. The mere familiarity with or fluency of the parts of the objects would have been sufficient to allow the monkeys to distinguish the old probes from the new probes. In contrast, Kroll et al. (1996) used recognition tasks in which all of the elements or features within a probe, in the critical condition, were old, having occurred in different events at time of study. For example, subjects saw a list of faces at time of study. At test some of the probe faces consisted of the eyes and nose of one face inserted into the background of another face (see Reinitz, Lammers, & Cochran, 1992). On the basis only of familiarity or fluency of the parts, these faces (and other similarly constructed stimuli) should have been called old, as all of the parts had been seen in the previously studied list. To determine that these conjunction events were new, the subject needed to be able to access what information had co-occurred in the previous list—a kind of knowledge that is derivable, in CHARM, only as a result of the associative operation of convolution. Normal subjects were more able to correctly reject these conjunctions than were patients with hippocampal damage, who tended incorrectly to accept them as old. Interestingly, on the items that were actually old as compared to items that were entirely new, the patients performed normally—a result consistent with those of Mishkin and Murray (1994).

Finally, it has been shown (Metcalfe Eich, 1985) that similarity effects due to the study context in recognition memory, and levels of processing effects in recognition memory, are attributable, in CHARM, to the associative operation. Thus, these should fail to show up with extreme damage to the associative operation (even though noncontextual recognition might be spared).

Spared Classification. To the extent that classification performance does not depend on within-event binding, the model predicts that classification performance may be spared despite damage to the system underlying association formation. I have conducted extensive computer simulations of the model on this paradigm, and in general they show that the binding operation in the model results in hyperspecificity. Thus, with impairment to the associative operation, the model predicts not a failure of classification, but rather good generalisation to categorically similar events. Knowlton and Squire (1994) have shown effects of spared classification in amnesic patients.

Composite Memory Trace

Normal Functioning

Multiple associations are stored, in CHARM, by being superimposed or added into a composite memory trace. Because of the use of the operations of convolution and correlation, the CHARM model is said to be ''holographic''. In physical holograms, if some part of the film is destroyed the stored images may still be reconstructed, but the reconstruction will be less clear than it would have been had the entire film been used in the reconstruction. Similarly, in the model, if part of the composite memory trace were ablated, the remaining part of the composite memory trace would allow reconstruction (albeit degraded) of entire memories.

Memorial Consequences of Impairment of Functioning

If brain damage resulted in partial trace loss and nothing else, then, according to CHARM, the trace would still support at least some recall, recognition, cued recall, and so on, although memory would be degraded. It is extremely rare that a patient is unable to remember anything from his or her past. To my knowledge, there is only one patient who may be an exception to this general rule. That patient, KC, is reported to be unable to retrieve any episodic information about any events that occurred in his life, either before or after the motorcycle accident that was the cause of his amnesia (Tulving, Hayman, & MacDonald, 1991; Tulving, Schacter, McLaughlin, & Moscovitch, 1988), such as might occur with total trace loss. However, KC's lesion is both extremely extensive and diffuse, suggesting that caution needs to be exercised in interpreting his deficit.

Novelty Monitoring and Control

Normal Functioning

A novelty monitoring and control circuit is needed and employed in CHARM (Metcalfe, 1993b). This circuit assesses the novelty of the incoming association with respect to the pre-existing composite trace, and computes a global familiarity value. The value is assumed to provide the informational basis for feeding-of-knowing judgements. To stabilise the composite memory trace, the monitoring/control circuit damps down the input into the trace as an inverse function of the computed novelty/familiarity value. Novel events are given high weightings in memory; familiar events are given low weightings. This circuit solves an inherent problem in the basic memory system. By so doing, it produces an adaptive memory system that is responsive to novelty and relatively unresponsive to already learned events. Impairment of this circuit should give rise to a characteristic novelty-based deficit syndrome outlined later. The

thumbprint of this amnesic syndrome shows up with Korsakoff and sometimes frontal lobe patients.

Memorial Consequences of Impairment of Functioning

Feeling of Knowing. It has been argued that familiarity-based feeling of knowing judgements are based on the computation given by the novelty or familiarity of the cue (Metcalfe, 1993a,b, 1994). A number of experiments have shown that the feeling-of-knowing judgement is affected by manipulations that alter the cue familiarity, as predicted by the model (Metcalfe, Schwartz, & Joaquim, 1993; Reder & Ritter, 1992; Schwartz & Metcalfe, 1992) but not by manipulations that alter the retrievability of the target itself. Converging evidence for this proposal has been given by Metcalfe (1993b). Shimamura and Squire (1986) have shown that Korsakoff patients show abnormal patterns on the feeling-of-knowing task. Other amnesic patients do not show impairment on this task. Janowsky, Shimamura, and Squire (1989) have shown that frontal patients (who may have novelty-circuit impairment without damage to other parts of the memory system) also show selective impairment (although not as severe as the Korsakoff patients) on the feeling-of-knowing task.

Release From PI. In the release from proactive inhibition paradigm, subjects are presented with several trials of to-be-remembered triads from a single category. Performance decreases with each successive triad. This is expected as an offshoot of the familiarity monitor because the trace is becoming more and more like the triads with each successive presentation, and so familiarity of the incoming item to the trace is increasing, and the weighting on successive triads will decrease. When the materials are shifted to a new category, subjects' performance increases. In the model this occurs because the different-category triad is novel with respect to the already encoded events in the trace, and hence is assigned a high weighting. Several studies have shown that Korsakoff amnesics have abnormal release from proactive inhibition (Cermak, Butters, & Moreines, 1974; Squire, 1982; Winocur, Kinsbourne, & Moscovitch, 1981). Squire's study is of particular interest because other amnesics do not show this impairment. Some studies suggest frontal involvement (see Moscovitch, 1982; cf. Petrides, 1995), although studies with frontal patients have sometimes been equivocal (perhaps because the precise locale of this circuit within the frontal lobes is unknown).

Von Restorff Effects and P300s. Von Restorff effects provide another straightforward example of a memory effect attributable to the novelty monitoring system. A list of words from one category is presented to the subject. One of the words, however, is from a different category. That different word is remembered better than would have been the case had the item in its

serial position been from the background category. In the model, the von Restorff effect occurs because the novelty monitor assigns the novel word a high weighting. An event related potential deflection called the P300 (or sometimes the ''late positivity'', Picton, 1993; Sutton, Braren, Zubin, & John, 1965) is related to von Restorff effects (Fabiani, Gratton, Chiarenza, & Donchin, 1990; Fabiani, Karis, & Donchin, 1986). It is plausible to suppose that it, too, is ascribable to the novelty monitoring circuit (Metcalfe, 1993a). In particular, the circuit must match the incoming event to the trace (presumably in the hippocampus), assess its novelty (presumably a frontal function), and feed back a signal to modulate the weighting of the association in the trace. This latter step may relate to the synchronous firing in the hippocampus that is responsible for the P300 deflection. Disturbance anywhere in this circuit should impact on the P300 (see Metcalfe, 1993a). Knight (1984) showed that frontal lobe patients exhibit an impairment on the P3A (and early component related to the P300)—indicating that this event related potential may rely on the adequate functioning of a circuit that monitors the state of the hippocampus, computes this state, and then feeds back a signal to control the amplification of what is currently being registered in the hippocampus.

Spacing Effects. Some spacing effects (Metcalfe, 1993a) have been attributed to the novelty monitoring circuit. If a word is repeated immediately, the second repetition is highly similar to the trace at that moment (by virtue of the strong presence from the first repetition of the critical item in the trace) and so its weighting will be less than if it is repeated after some spacing (when its similarity to the trace will be decreased because the trace has changed with the intervening items). After some time has passed, a function of the sum of the initial weightings (which favours the spaced condition) will be apparent in memory performance. However, if the test is immediate, the first presentation of the word will still be heavily weighted in the trace (because of its recency) and will have a considerable impact on memory performance. With immediate test the recency of the first presentation will dominate, and so massed rather than spaced items will seem to be stronger. The differential weighting as a function of trace similarity is needed, in the model, to account for these effects. Impairment of the novelty monitoring circuit, selectively, should therefore impact on spacing effects: they should be less apparent for Korsakoff patients than for other amnesic patients. I know of no studies investigating spacing effects with amnesics.

Interference Effects. In CHARM simulations of the release from proactive interference paradigm (Metcalfe, 1993b), a difference in the pattern of intrusions from previous lists, which depends on whether or not the novelty monitor was used, can be observed. When the novelty monitor was used in the simulations, the responses tended to issue primarily from the last-presented list, whereas

when the novelty monitor was disengaged, there were more intrusions from previous lists. The idea that frontal damage results in an increase in interference, or an inability to gate information, is often proposed as an explanation of memory impairments in these patients (Knight, Scabini, & Woods, 1989; Shimamura, 1994). Shimamura et al. (1995) found selective disruption in frontal lobe patients in an AB–AC test and in an AB–ABr test, both of which were designed to measure interference.

Primacy. The primacy effect is probably due, at least in part, to the fact that people rehearse the first words in a list more than they do other words in a list (Murdock & Metcalfe, 1978; Rundus & Atkinson, 1970). However, primacy shows up even when the presentation rate is so fast that people are unable to rehearse at all. It is postulated that this second factor in the primacy effect may be attributable to the novelty monitor. The first item in a list is always different from the mental activity that preceded it, by virtue of being the first to-be-remembered item. We would expect it, then, to receive a novelty boost. Abnormal primacy effects, with frontal damage, have been reported by Petrides (1995).

Habituation. The novelty monitor not only provides a boost in weighting for novel items, but it also decreases the weighting on items that are similar to or identical with those that have come before—that is, it is responsible for habituation effects. The failure to habituate appropriately may be responsible for many of the abnormalities seen in Korsakoff and frontal patients. Hypervigilance is characteristic of some of these patients—everything seems novel. The reverse syndrome of apathy is also commonly observed—nothing seems novel. Both of these may be attributable to a failure to appreciate that a stimulus is either novel, and should be attended, or not novel, and hence may be safely ignored.

Retrieval

Normal Functioning

When a retrieval cue is given, it is perceptually analysed and used to retrieve a representation from the composite trace. The retrieval operation of correlation is the inverse operation to convolution. The item that is retrieved from memory is in a form available to phenomenological awareness. Interestingly, these retrieved items are noisy, distorted, and degraded. Retrieval has the characteristic, in this model, of being redintegrative: a part of an event is sufficient to retrieve the whole. Similarity effects fall out automatically. Furthermore, everything that was associated with a particular retrieval cue will be retrieved by that cue—resulting in the superimposed retrieval of multiple events that allows the model to account for such diverse phenomena as episodic

classification learning and generalisation (Metcalfe Eich, 1982), interference in the A–B A–C paradigm (Metcalfe Eich, 1982), and the effects of misleading suggestions on eyewitness testimony (Metcalfe, 1991).

Memorial Consequences of Impairment of Functioning

If there were a retrieval deficit, all episodic memories would be affected. The patient would experience both retrograde and anterograde amnesia, and all episodic function would be lost. Squire, Knowlton, and Musen (1993) report a number of examples of patients who exhibit the combined syndrome of both retrograde and anterograde amnesia. As mentioned earlier, KC is the only patient for whom the claim is made that he can recollect no episodic events from his past, and, as with the interpretation that this implies that he has complete trace loss, the interpretation that this means he has a complete retrieval failure needs to be viewed with extreme caution because of the extent and complexity of his brain damage. All non-episodic functions could be spared, as could working memory functions, in the face of severe retrieval deficits.

CONCLUSION

If this analysis is correct in the assumption that different amnesic syndromes result from a complex interaction of the requirements of particular tasks with the memory components that are needed to perform them, then we search in vain for a unitary cause of amnesia. The analysis of the memory tasks by models that can actually perform those tasks, and the isolation of the processes that are needed in their performance, appears to be necessary to further our understanding of human amnesia. Computational models, such as CHARM, allow the specific delineation of profiles of impairments that can be expected when particular components are impaired. The CHARM model, itself, was not initially designed to address the causes of human amnesia, but rather, was intended more simply to be a model of how normal people represent mental items or events, associate those items in episodic memory, store the associations, and then later, how they retrieve an item from memory when given a cue. Its use, in designating the patterns of impairments that are expected with breakdown of each of the major components of memory, provides a means to begin to disentangle and reconcile the perplexing results found in the amnesia literature, and, in particular, the seeming lack of replicability and stability. Such apparent variability is expected if multiple syndromes—each different in its underlying cause and in its manifestations—have been inappropriately conceptualised as if they comprised a unitary entity.

At a theoretical level CHARM permits a fine-grain differentiation of specific syndromes of human amnesia that allows us to articulate in considerable detail the various deficits that could occur under ''ideal'' conditions in which the underlying components were lesioned clearly and selectively. Empirically, of

course, in many if not most patients, such discrete effects are unlikely. Thus, even the somewhat complex pure syndromes predicted by discrete failure of selective memory components, as given in this article, may be found only rarely, and the prospect remains that further empirical investigation may more frequently reveal mixed and partial cases.

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