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Toward a Cognitive Neuropsychology of Awareness: Implicit Knowledge and Anosognosia*

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ABSTRACT

Although a systematic cognitive neuropsychology of awareness has not yet emerged, a number of phenomena reported in the literature provide an empirical basis for developing it. The present discussion focusses on two such phenomena: *implicit knowledge,* which refers to knowledge that is expressed in task performance unintentionally and with little phenomenal awareness; and anosognosia, which refers to unawareness of neuropsychological deficits. Two types of theoretical accounts of these phenomena are discussed. A *first order* theoretical account entails postulating damage to, or disconnection of, a system or process that generates awareness across multiple domains. A *second-order* account does not postulate disruption of a cross-domain awareness mechanism, but instead appeals to difficulties in gaining access to particular kinds of domain-specific information that are associated with aware expressions of knowledge in individual domains. Instances of first- and second-order accounts are illustrated with examples from studies of memory and amnesia. The relation between implicit knowledge and anosognosia is also discussed.

Understanding the nature of phenomenal awareness constitutes one of the oldest and deepest mysteries in all of psychology or neuroscience. It is perhaps a commonplace but nevertheless profoundly puzzling observation that human beings do not simply "process information", as suggested by a computer metaphor of the mind. Information is also somehow represented to the processor, so that one has the subjectively compelling experience that "I perceive", "I understand", or "I remember", as pointed out eloquently by William James nearly 100 years ago. As far as we know or can guess, even the most advanced computer lacks the capacity to represent information to itself in

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such a way as to experience the sort of phenomenal awareness that is virtually a ubiquitous part of everyday human experience.

What kind of contribution has neuropsychology made to the understanding of phenomenal awareness? Even the most generous reading of the clinical and experimental literature that constitutes a century's worth of neuropsychological research leads to the conclusion that the contribution has been rather modest. In contrast to the intensive interest in, and important lessons that have been learned from, neuropsychological studies of language, memory, perception, reading, and other cognitive functions, a coherent neuropsychology of awareness has yet to emerge. Of course, one can discern pockets of neuropsychological interest in the phenomenon of awareness even from a cursory survey of modern research; the study of, and controversy about, so-called split-brain or commissurotomy patients is probably the best known example. Although the study of such patients has provided intriguing clues concerning the nature of awareness, appropriate patients are rare and some of the phenomena are still disputed. Thus, despite this and other pockets of relevant research, it seems safe to conclude that programmatic neuropsychological study of and theorizing about the nature of awareness remains elusive.

The main purpose of the present discussion is to delineate some possibly useful directions and ideas for neuropsychological research concerning awareness. Two types of phenomena observed in brain-damaged patients will be described -- implicit knowledge and anosognosia -- that involve disturbances of awareness. I believe that both phenomena have important implications for understanding the nature of phenomenal awareness and will attempt to delineate some of them. Second, two classes of theoretical explanations that can be applied to such awareness disturbances will be distinguished, which I will refer to, respectively, as *first-order* and *second-order* theoretical explanations. It is necessary to indicate briefly what is meant by these terms before moving on to consider the phenomena of interest.

By a first-order theoretical account of an awareness disturbance, I refer to an explanatory attempt that places the locus of the disturbance at the level of a hypothetical mechanism or mechanisms presumed to underly, or be in some way directly responsible for, the experience of phenomenal awareness itself. Thus, for example, if one took the position that phenomenal awareness is associated with the activity of a particular neuropsychological system or process (e.g., Dimond, 1976) a first-order theoretical account of an awareness disturbance would invoke a deficit at the level of the hypothetical awareness system or perhaps postulate a disconnection of that system from other perceptual or cognitive systems. Similarly, if one subscribed to the idea that phenomenal awareness is some sort of higher order emergent property that reflects the conjoint activity of numerous underlying systems (e.g., Kinsbourne, 1988; Sperry, 1984), a first-order account of an awareness disturbance would postulate a high level organizational deficit that interferes with the expression of this emergent property.

In contrast, a second-order account of an awareness disturbance does not appeal directly to a deficit in the processes that are presumed to be responsible for generating awareness across multiple domains. Rather, it appeals to an inability to gain access to a certain kind of domain-specific information that is normally associated with, or provides a basis for, an experience of awareness within a particular domain. A second-order account of an awareness disturbance is not inconsistent with the idea that some sort of general purpose, cross-domain awareness mechanisms exist; it merely indicates that one need not appeal to disruption of such mechanisms every time an awareness deficit is observed. The difference between first-order and second order theoretical explanations will be illustrated more concretely in relation to particular examples of implicit knowledge and anosognosia.

Some additional key terms also need to be defined. It would of course be presumptuous to claim that one has an adequate definition of a term such as "awareness" or "consciousness". A very simple definition that I have found useful, however, was put forward by Dimond (1976), who defined awareness as "the running span of subjective experience." The terms awareness and consciousness will be used in this sense -- to refer to an ongoing representation of specific mental activities -- rather than to refer to generalized states of arousal or alertness such as sleep, waking, or coma. The term "implicit knowledge" refers to knowledge that is expressed in task performance unintentionally and with little or no phenomenal awareness (e.g., Schacter, 1987a,b; Schacter, McAndrews, & Moscovitch, 1988). The term "anosognosia" will be used in the traditional sense to refer to diminished awareness of the existence of a neuropsychological deficit itself. The former phenomenon refers to patients who are unaware of *knowledge* that they in fact possess, whereas the latter involves patients who are unaware of their *deficit.* I will consider later how unawareness of knowledge and unawareness of deficit are related to one another.

IMPLICIT KNOWLEDGE IN NEUROPSYCHOLOGICAL SYNDROMES

Throughout most of the history of neuropsychological research, patients' deficits have been defined in terms of their poor performance on tests that tap explicit knowledge within a particular domain. Thus, a patient who cannot recollect recent events when queried explicitly about them is characterized as *amnesic;* a patient who cannot read a familiar word when asked to do so explicitly is called *alexic;* a patient who cannot recognize a well-known face when explicitly asked if it seems familiar is labelled *prosopagnosic;* and so on. For many years, little if any attention was paid to the possibility that patients might possess, and be able to express under appropriate conditions, implicit knowledge within the domain in which they lack explicit knowledge. However, during the past decade or two, and particularly in recent years, all that has

begun to change. So much so that in a recent chapter McAndrews, Moscovitch, and I (Schacter et al., 1988) were able to pull together evidence from a number of neuropsychological syndromes that, according to our view, is characterized by a common feature: It provides suggestive and in some cases compelling evidence for the existence of implicit knowledge despite patients' serious deficits on standard tests of explicit knowledge. Let us consider briefly evidence from just a few of the syndromes discussed in the Schacter et al. chapter.

Consider first data concerning the amnesic syndrome. It is now well known that despite their inability to explicitly remember recent events, amnesic patients show intact implicit memory in a variety of situations. In fact, the existence of these dissociations was one of the main reasons why Graf and Schacter (1985; Schacter, 1987b) put forward the distinction between implicit and explicit memory. Thus, for example, Milner, Corkin and their colleagues demonstrated many years ago that the well-known amnesic patient H.M. could acquire gradually across trials and sessions various perceptual and motor skills, even though he lacked any explicit memory for the episodes in which he acquired the skills (e.g., Milner, Corkin, & Teuber, 1968). Similar demonstrations of intact skill learning despite impaired explicit memory have been since reported by many others (e.g., Brooks & Baddeley, 1976; Cohen & Squire, 1980; Kinsbourne & Wood, 1975; Moscovitch, 1982). Along these same lines, Glisky, Schacter, and Tulving (1986a,b) were able to show that, with months of practice, amnesic patients could acquire and retain complex knowledge and skills needed to program and interact with a microcomputer - even though, when queried at the beginning of a learning session, some of these patients consistently failed to remember explicitly that they had ever worked on a microcomputer.

In addition to this evidence that amnesic patients can show implicit memory for skills and knowledge acquired gradually across many learning trials, a substantial literature now exists that demonstrates clearly that amnesic patients show intact repetition priming effects following a single exposure to a target stimulus. Repetition priming refers to the facilitatory effect of exposure to a target stimulus on subsequent processing of that stimulus on an implicit test that does not require intentional recollection of any prior encounter with the stimulus, such as word stem- and fragment completion (e.g., Graf, Squire, & Mandler, 1984; Graf & Schacter, 1985; Schacter & Graf, 1986; Warrington & Weiskrantz, 1968, 1974), word identification (e.g., Cermak, Talbot, Chandler, & Wolbarst, 1985; Jacoby & Dallas, 1981), and lexical decision (e.g., Moscovitch, 1982; Scarborough, Gerard, & Cortese, 1979). Studies of normal subjects have shown that priming effects on implicit memory tests can be sharply dissociated from recall and recognition performance (for review and discussion, see Richardson-Klavehn & Bjork, 1988; Schacter, 1987a,b). More importantly for the present purposes, we now know that even densely amnesic patients show normal priming effects on various kinds of implicit memory tests, as established initially in the classic studies of Warrington and Weiskrantz

(1968, 1974). Since that time, intact priming has been demonstrated in various patients on a wide variety of implicit memory tests (e.g., Cermak et al., 1985; Graf et al., 1984; Graf & Schacter, 1985; Moscovitch, 1982; Moscovitch, Winocur, & McLachlan, 1986; Schacter, 1985; Schacter & Graf, 1986; Shimamura & Squire, 1984).

These studies of implicit memory constitute just one example of preserved implicit knowledge in neuropsychological syndromes, and I will just briefly illustrate several others that are discussed at length in the Schacter et al. (1988) chapter. Consider first the syndrome of prosopagnosia, in which patients typically report no familiarity with the faces of family, relatives, and friends. However, research reported by Bauer (1984) and also by Tranel and Damasio (1985), using psychophysiological indices, has established that prosopagnosic patients possess some implicit knowledge of facial familiarity. For example, Tranel and Damasio found that a severely prosopagnosic patient showed larger skin conductance responses to familiar than to unfamiliar faces, even though none of the faces seemed familiar to the patient. In an important series of studies that have used more analytical behavioral techniques, deHaan, Young, and Newcombe (1987; Young & deHaan, 1988) have reported data that support and extend the psychophysiological findings. One of their patients was entirely unable to distinguish explicitly between familiar and unfamiliar faces. However, on a matching task that required same-different judgments about two simultaneously exposed faces, this patient, like control subjects, was faster to repond when a judgment was made about familiar than unfamiliar faces, thereby demonstrating some access to facial familiarity information. In addition, the patient was subject to interference from familiar faces - even though he did not recognize them explicitly - on a Stroop-like naming task, and also showed priming effects that required implicit though not explicit access to facial familiarity information.

Similar results have been obtained with alexic patients, who are unable to read visually presented words unless they resort to a letter-by-letter decoding strategy. Studies by Shallice and Saffran (1986), as well as by Landis, Regard, and Serrant (1980) and by Coslett (1986), have shown that, when words are presented at extremely brief tachistoscopic exposures that prevent letter-by-letter decoding, alexic patients can make above-chance lexical decisions, semantic categorizations, and other judgments about words that they cannot identify explicitly. Finally, no discussion of implicit knowledge in neuropsychological syndromes would be complete without mention of the phenomenon of blindsight studied extensively by Weiskrantz (1986) and others. Patients with lesions to striate cortex typically lack normal conscious visual experiences within their scotoma. Nevertheless, when required to "guess" about the location and other attributes of presented stimuli, such patients can make above-chance forced-choice judgments regarding stimuli that they cannot "see". Even though some aspects of the blindsight phenomenon are controversial (Campion, Latto, & Smith, 1983), there are solid reasons to

believe that these patients can show some implicit knowledge of stimuli that are not represented fully in conscious visual experience.

These examples of implicit knowledge in different syndromes are not exhaustive; the list can be expanded to include Broca's and Wernicke's aphasics, neglect patients, and split-brain patients among others (see Schacter et al., 1988, for discussion). But what are we to make of these counterintuitive and sometimes startling observations? And how does the distinction between first-order and second-order theoretical accounts of awareness disturbances apply to these phenomena? To bring these issues into sharper focus, let us consider briefly a descriptive model that I have advanced recently to accommodate implicit/explicit dissociations, referred to by the acronym DICE (Dissociable Interactions and Conscious Experience).

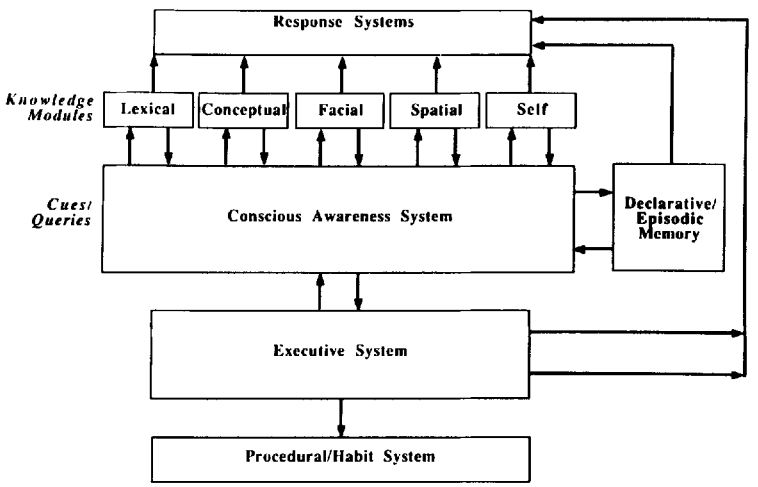

Fig. 1. A schematic depiction of DICE. Declarative/episodic memory subserves remembering of recent events and information; knowledge modules represent various kinds of nonepisodic information; the procedural/habit system is involved in perceptual/motor skill learning. Phenomenal awareness of specific types of information depends on intact connections between the conscious awareness system and individual knowledge modules or declarative/episodic memory. The procedural/habit system does not have any connections with the conscious awareness system. The conscious awareness system serves as the gateway to the executive system, which is involved in initiation of voluntary activities.

A basic idea motivating the DICE model, previously articulated by Baars (1983), Dimond (1976), Gazzaniga (1985), Johnson-Laird (1983) Marcel (1983), and others, is that the processes that mediate conscious identification and recognition --that is, phenomenal awareness in different domains --should be sharply distinguished from modular systems that operate on linguistic,

perceptual, and other kinds of information. Accordingly, the model takes as a starting point the idea that conscious experiences of perceiving, knowing, and remembering require the activation of what I have referred to as a *Conscious Awareness System* (or CAS for short) that normally interacts with, but can become disconnected from, modular-level processors (see Figure 1). Activation at the modular level will produce a change in performance or behavior such as a priming effect, but is not sufficient by itself to produce awareness of the activated information; an interaction with CAS is necessary for this to occur. Of course, simply postulating the existence of a mechanism such as CAS does not in any sense "solve" the awareness problem. It is merely a convenient shorthand for representing the idea that phenomenal awareness requires processing beyond the modular level.

Because this model was put forward primarily to address issues concerning the relation between memory and phenomenal awareness, let us consider it from that perspective. I have suggested that CAS can be activated by the outputs of two general types of memory structures: a declarative or episodic memory system that represents newly acquired time and place information about recent experiences, or various knowledge "modules" - processors that represent nonepisodic knowledge of various kinds, such as lexical, conceptual, spatial, and so forth. When CAS is activated by outputs of the episodic system, the result is a conscious memory for a recent event; when CAS is activated by output from a knowledge module, the result is a conscious experience of knowing a particular bit of information. (Note that I do not use the term "module" in strict conformity with all the criteria for modules that were proposed by Fodor [1983]; see Moscovitch and Umiltà [in press] for an overview and discussion). The model also postulates that acquisition of skills is mediated by a procedural or habit memory system of a kind discussed by Mishkin, Malamut, and Bachevalier (1984) and Squire (1987) that does not have an input connection to CAS, thus reflecting the idea that one cannot become aware of procedural knowledge.

Within the context of this model, a first-order theoretical account of dissociations between implicit and explicit knowledge would place the locus of damage somewhere at or close to the level of CAS -- the mechanism that is directly responsible for "generating" awareness in some as yet unspecified and poorly understood way. However, it is clear that simply postulating damage to CAS itself does not provide an adequate theoretical account. This is because damage to CAS would be expected to result in a global or generalized disturbance of awareness in all domains. However, the failures of awareness observed in amnesia and the other neuropsychological syndromes discussed above are in many cases rather *specific* -- that is, patients do not ordinarily have serious difficulty gaining conscious or explicit access to information outside the domain of their particular impairment. For example, amnesic patients do not have difficulties explicitly reading words like alexic patients do, and alexic patients do not have difficulties consciously recognizing faces like prosopagnosic patients do.

However, a first-order theoretical account that is consistent with the domain specificity of implicit knowledge could go as follows: perhaps implicit/explicit dissociations are attributable to a *selective disconnection* between the output of a particular module or system and CAS -- that is, modular outputs may fail to gain access to the awareness system. In the model, activation of CAS represents just one output route from an individual module; it is also possible for knowledge to be expressed through verbal or motor response systems that do not entail activation of CAS (Figure 1). When modular outputs affect response systems without corresponding activation of CAS, knowledge is expressed implicitly, without any phenomenal awareness or subjective experience of knowing, remembering, or perceiving.

Is it necessary to invoke such a first-order theoretical account to accommodate any or all of the various implicit/explicit dissociations that we have discussed? To answer this question, we must consider the possibilities for developing an adequate second-order account of these dissociations. This problem is best illustrated by considering the phenomena of implicit and explicit memory. There is virtually unanimous agreement that the most striking deficit of amnesic patients is their inability to express a fully aware or conscious re-experiencing of a recent event. To begin formulating a second-order theoretical account of implicit memory, we can begin by asking what kind of mnemonic information must be accessible to support an aware "re-experiencing" of an event. Many cognitive psychologists and neuropsychologists would agree that access to place and time information about the global context of an event is critical. Suppose, for example, that a subject encountered a familiar word (e.g., table) on a study list, and was later given a word stem (e.g., tab) with instructions to complete it with the first word that comes to mind. Assume further that he or she completed the stem with the word "table", but did not have access to any time/place information about the context in which the word was presented. The resulting experience would likely be very much like that of the implicit memory exhibited by the amnesic patient: A word pops to mind because it formed part of a recent event, but there is no conscious or aware re-experiencing of that event at the time of retrieval. Suppose further, however, that the subject is reminded of some salient detail of the original encoding context. Assuming that the subject is not amnesic, the contextual cue may trigger a full-blown re-experiencing of the episode in which the word "table" was studied. In this example, then, one could offer a reasonable *second-order* account of the implicit nature of the expressed memory by appealing to the inaccessibility of contextual information in episodic memory; it is not necessary to put forward a first-order account that appeals to some sort of breakdown in, or disconnection of, a mechanism such as CAS.

Let us apply this line of reasoning to the model that we have been discussing: Do the various implicit memory phenomena that have been considered demand a first-order theoretical account, or is a second-order account satisfactory? Clearly, the phenomenon of preserved skill learning can be easily accommo-

dated by a second-order account. One simply needs to assume that the procedural/habit system does not encode information about global context, and that amnesic patients have impairments in the episodic system that handles contextual information. It thus follows that implicit expressions of procedural knowledge can be handled by arguing that amnesic patients are unable to gain access to the sort of contextual information about when and where a skill was acquired that is necessary to support explicit remembering or re-experiencing of the acquisition episode.

A second-order account can also be applied to the phenomenon of intact word priming in amnesia, along the lines suggested above. Within the framework of the model, all one has to do is assume that word- priming effects are produced by activation of pre-existing lexical nodes in a knowledge module: If automatic activation of pre-existing representations occurs normally in amnesic patients, but the episodic system that handles contextual information is defective, then it follows naturally that one should observe implicit memory for a recently studied word without any explicit recollection of the prior occurrence of the word. The issue becomes rather more complex, however, when we ask about priming effects and implicit memory for newly acquired information that cannot be attributed to automatic activation of a pre-existing lexical or other memory unit.

The whole question of whether amnesic patients exhibit priming effects for newly acquired information is very much under debate; perhaps the fairest summary of the literature is that some amnesic patients show implicit memory for newly acquired information in some paradigms, but many do not; relevant work has been reported by Cermak and colleagues (Cermak et al., 1985, Cermak, Blackford, O'Connor, & Bleich, 1988; Cermak, Bleich, & Blackford, 1988) Gabrielli, Milberg, Keane, and Corkin (in press), Gordon (1988), Graf and Schacter (1985), Moscovitch et al. (1986); Schacter (1985), Schacter and Graf (1986), and Shimamura and Squire (1989), among others. For the present purposes, I will discuss briefly two phenomena studied in our laboratory to highlight the conceptual issues of interest. Consider first an experiment by McAndrews, Glisky, and Schacter (1987). Amnesic patients and control subjects were shown novel sentences that failed to make sense in the absence of a critical disambiguating word (e.g., *The notes were sour because the seams split;* see Auble & Franks, 1979). The subjects were given a minute to try to think of the critical word; if they could not think of it on their own, the experimenter provided it (e.g., *bagpipe*). The ambiguous sentence frames were then represented at various retention intervals ranging from 1 minute to 1 week. The critical finding was that even severely amnesic patients showed a priming effect on this task - that is, they came up with the critical disambiguating word significantly more often on the second exposure of the sentence frame than on the first - at all retention intervals, including 1 day and 1 week delays. Yet these same patients did not explicitly remember having seen the sentences previously when given a yes/no recognition test. With respect to the previous discussion, it

is difficult to argue that this priming effect reflects the transient activation of some preexisting memory node, since the sentences are novel constructions and the priming persists over a 1 week delay.

Does this phenomenon thus demand a first-order theoretical interpretation? That is, does it indicate that episodic memory is relatively intact in these amnesic patients, with the outputs of the episodic system disconnected from CAS? Probably not. One could just as easily argue that the phenomenon represents some sort of relatively permanent *restructuring* of preexisting semantic knowledge; after all, the individual words that constitute each sentence do have preexisting semantic and lexical representations. By this view, the reason that severely amnesic patients do not explicitly remember the prior occurrence of the sentences is that episodic memory is defective and thus the contextual information necessary to support an aware re-experiencing of the prior occurrence of the sentences is simply not available at the time of test.

Let us take the argument one step further and suppose that we are able to demonstrate that amnesic patients show intact implicit memory for entirely novel information that does not in any sense have a preexisting memory representation. In such a situation, any priming effect that is observed would have to be attributed to the formation of some sort of novel memory representation. Would we then be in a position to argue that a first-order theoretical interpretation of the results is demanded in which it is postulated that a relatively intact episodic memory system is disconnected from the awareness system? To illustrate the issue, I will summarize a new implicit memory paradigm being investigated in my laboratory with collaboration from Lynn Cooper and Suzanne Delaney (see Schacter, Cooper, & Delaney, (in press) for details). Although we have not yet applied the paradigm to amnesic patients, our results to date with normal subjects highlight issues that are critical to answering the question that I have just posed.

The main purpose of these studies was to determine whether we could observe evidence of implicit memory for a prior exposure to novel three dimensional objects that have no preexisting representation in memory. In our experiments, subjects studied line drawings that represent unfamiliar and rather unusual three dimensional constructions such as those displayed in Figure 2. In the first experiment, subjects studied the drawings in two different ways. One group of subjects was induced to encode information about the *global structure* of each object by deciding whether the object faces primarily to the left or to the right. A second group of subjects was induced to encode information about the *local features* of each object by deciding whether it has more horizontal or vertical lines. After completing these encoding tasks, half of the subjects in each group were given an explicit memory test -- a standard yes/no recognition test in which they were shown studied and nonstudied drawings and indicated whether or not they remembered seeing them previously. The other half of the subjects were given an implicit memory test. To assess implicit memory for these unfamiliar objects, we designed an *object decision test*. Although subjects are

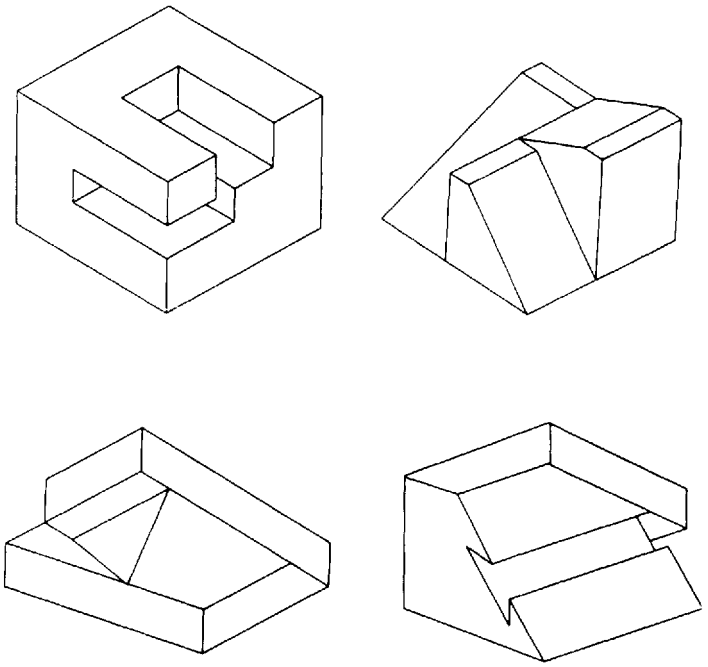

Fig. 2. Possible and impossible objects used in experiments by Schacter, Cooper, and Delaney (in press). The drawings in the upper row depict *possible* objects that could exist in three-dimensional form; the drawings in the lower row depict *impossible* objects that contain structural violations that would prohibit them from actually existing in three-dimensional form.

not informed of it at the time of encoding, half of the line drawings that they studied were possible objects -- their surfaces and edges are connected in such a way that they could potentially exist in three-dimensional space. The other half were *impossible* objects -- they contain subtle surface, edge, or contour violations that would prohibit them from actually existing in the three-dimensional world. We gave subjects brief, 100 ms exposures to drawings of studied and nonstudied possible and impossible objects; their task was to decide whether each object is possible or impossible. The object decision task can be thought of as an implicit memory test in the sense that it does not make explicit reference to, or require conscious recollection of, any specific previous encounter with a presented object. If, therefore, subjects are more accurate in making object decisions about studied than nonstudied objects, there would be some evidence of implicit memory for these unfamiliar, three-dimensional objects. More specifically, we reasoned that performing the object decision test requires analysis of the global structure of the object; subjects must gain access to information about global structural relations in order to decide whether an

object is possible or impossible. According to theoretical notions such as transfer appropriate processing (e.g., Roediger, Weldon, & Challis, 1989), it follows that prior encoding of information about global object structure -- but not local object features -- should produce priming or implicit memory on a subsequent object decision task.

Table 1

Object Decision Performance as a Function of Study Task, Test Order, and Item Type (Schacter, Cooper, & Delaney, in press)

Encoding Condition/Test Order						
	Left/Right			Horizontal/Vertical		
Item Type	First	Second	*M*	First	Second	*M*
Studied	.81	.81	.81	.72	.63	.67
Nonstudied	.63	.71	.67	.64	.64	.64
M	.72	.76		.68	.64	

Note. Each number in the table reflects the proportion of possible objects classified correctly on the object decision test.

The results of our first experiment, displayed in Table 1, were consistent with this hypothesis. Only the data for the possible objects are shown, because we failed to find priming of impossible objects in any of our experiments (see Schacter et al., in press). The data in the table depict object decision performance as a function of the left/right or horizontal/vertical encoding task and as a function of whether or not an item was studied. In addition, the subjects who were given the recognition test were also given an object decision test after it. Thus, we could examine object decision performance as a function of whether it was given as the first test or as the second test, following the recognition task.

The data indicate that, for both the first and second tests, object decision performance was significantly more accurate for studied than nonstudied drawings following the left/right study task, but there was no significant difference between studied and nonstudied objects following the horizontal/vertical study task.Thus, we found that implicit memory for unfamiliar objects depends on encoding of and access to some sort of global structural description of an object. Consistent with this idea, performance on the object decision task did not differ as a function of whether it was given first or whether it was given second, after the recognition task. This means that the appearance of studied and nonstudied objects on the recognition test did not facilitate subsequent object decision performance; in other words, deciding whether an object is old or new, at least under these test conditions, does not appear to involve the sort of structural encoding that is needed to produce implicit memory on an object decision test.

While it thus seems safe to conclude that implicit memory for unfamiliar objects is observed only following a highly specific form of structural encoding, the recognition data indicated that explicit memory for the objects was

comparable following the left/right and horizontal/vertical study tasks; performance in the two conditions did not differ significantly (see Schacter et al., in press). These results indicate that implicit and explicit memory for unfamiliar objects can be dissociated experimentally, and also suggest that the two types of memory are based on different kinds of underlying representations. Further evidence in support of these ideas was provided by a second experiment in which one group of subjects engaged in an elaborative encoding task that required them to think of a real-world object that each drawing reminded them of most. We hypothesized that such a task would require subjects to achieve a meaningful interpretation of the object by relating it to preexisting semantic knowledge. Based on many previous demonstrations that explicit memory is enhanced by semantic elaboration, we reasoned that this elaborative study task should enhance recognition performance relative to the left/right encoding task used in the first experiment. However, since the elaborative task does not involve specific *structural* encoding of the objects, it should not lead to more accurate object decision performance than the left/right task. The results were consistent with this expectation, and in fact revealed a dramatic dissociation between recognition and object decision performance. On the recognition test, elaborative encoding led to more accurate explicit memory than did left/right encoding (Table 2). In striking contrast, the object decision data, also displayed in Table 2, indicated that no priming was observed following the elaborative task, whereas significant priming was observed following the left/right task, in replication of Experiment 1. This dissociation is particularly impressive because there are very few studies in which an experimental manipulation that improves explicit memory also impairs implicit memory.

Table 2

Object Decision and Recognition Performance as a Function of Study Task and Item Type (Schacter, Cooper, & Delaney, in press)

Encoding Condition	Object Decision Test		Recognition Test	
Item Type	Left/Right	Elaborative	Left/Right	Elaborative
Studied	.78	.76	.69	.88
Nonstudied	.66	.73	.26	.19

Note. For the object decision test, each number reflects the proportion of studied or nonstudied possible objects classified correctly. For the recognition test, the first row indicates the proportion of studied possible objects called "old" (hit rate) and the second row indicates the proportion of nonstudied possible objects called "old" (false alarm rate).

Whatever one is to make of these results, and we have discussed their theoretical implications in detail elsewhere (Schacter, in press-a; Schacter et al., in press), the question germane to the present concerns is how these experiments

are related to the problem of distinguishing between first- and second- order interpretations of implicit memory effects. The data show clearly that implicit memory for entirely novel objects can be observed and dissociated sharply from explicit memory. Suppose that in addition we find that densely amnesic patients show intact object decision priming despite poor explicit recognition memory performance (we are presently investigating this issue). Since implicit memory in this paradigm is necessarily based on some sort of newly established memory representation of the object - as noted earlier, these objects were constructed such that they have no preexisting memory representations - it is tempting to conclude that such a result would indicate that episodic memory is in fact intact in some amnesic patients, but can only be expressed implicitly because the outputs of the episodic system are disconnected from the awareness system. However tempting such a conclusion may seem, the data do not *demand* such a first-order theoretical interpretation; let me suggest why. Our experiments demonstrate clearly that object decision priming relies on encoding of a highly specific structural description of an unfamiliar object. We have argued further on various grounds that representation and retrieval of this kind of information is handled by a structural description system of the kind discussed by Riddoch and Humphreys (1987a, 1987b; see also Warrington, 1982) in their work on object agnosia, a system that is held to be distinct from the episodic system that supports explicit memory for recent events. I have suggested further that the structural description system is one of several presementic *perceptual representation systems* that play a key role in implicit memory (see Schacter, in press-a). If this interpretation is correct (and even if it is not), one could offer a second order account of whatever object decision priming is observed in amnesic patients that holds that the structural description system is intact and supports priming whereas the episodic memory system is impaired; no direct appeal to a damaged or disconnected awareness system would be necessary.

This observation is somewhat unsettling: If a priming effect that reflects the establishment of an entirely novel memory representation does not demand a first-order theoretical interpretation, what kind of result is necessary? The answer, I think, is that one would need to show preserved implicit memory for the very global contextual attributes that are normally accessed explicitly and provide the underlying informational basis for an aware re-experiencing of a prior episode. If it were possible to demonstrate intact implicit memory for spatial, environmental, temporal, and other aspects of the global context or setting of an episode in amnesic patients who cannot remember these contextual attributes explicitly, then there would be strong grounds for advancing a first-order theoretical interpretation that postulates an intact episodic memory system whose outputs are disconnected from awareness. As Schacter et al. (1988) concluded in their review of implicit knowledge in neuropsychological syndromes, such evidence has not yet been produced.

The distinction between first- and second-order accounts of implicit/explicit dissociations can also be applied to the various other examples of implicit knowledge in neuropsychological syndromes discussed earlier. One can ask in all cases whether awareness mechanisms that operate across domains are implicated directly in the observed phenomena, or whether patients suffer from an inability to process or retrieve a particular type of domain-specific information that normally supports an explicit or aware expression of knowledge within the domain of their impairment, be it language, perception, reading, and so on. Although it is beyond the scope of the present discussion to consider all pertinent phenomena in any detail, it is worth noting that perhaps the strongest evidence in favor of a first-order theoretical interpretation is provided by the work of Young, deHann and their colleagues (deHaan et al., 1987; Young & deHaan, 1988) on implicit knowledge in prosopagnosia. They have provided a good deal of evidence that certain aspects of facial familiarity are processed normally by such patients, with the output of what they call face recognition units disconnected from awareness. Future research and theorizing on implicit knowledge in neuropsychological syndromes could well benefit from taking account of the distinction between first- and second-order theoretical intepretations of the critical phenomena.

ANOSOGNOSIA AND UNAWARENESS OF DEFICIT

Let us now turn to the second phenomenon that I believe is crucial to the development of a cognitive neuropsychology of awareness -- anosognosia or unawareness of deficit. This phenomenon was described initially by von Monakow in the 19th century, and given the name anosognosia by Babinski in 1914, meaning lack of knowledge of disease. The phenomenon is one of the most compelling in all of neuropsychology: hemiplegic patients may deny that there is anything wrong with a frankly paralyzed limb; Anton's syndrome patients are often unaware of their blindness and believe that they can see; and some densely amnesic patients may claim that their memory is entirely normal. A rather extensive clinical neurological literature concerning the phenomenon evolved during the 1930s and 1940s, culminating in Weinstein and Kahn's (1955) classic monograph, *Denial of illness.* Although the number of published studies of the phenomenon declined over the next 20-30 years, there has been a recent re-awakening of interest in it. For example, Bisiach and his colleagues have published several important papers on anosognosia for hemiplegia and hemianopia that have delineated possibly important theoretical consequences of the phenomenon and have established some new empirical facts about it (Bisiach, Voller, Perani, Papagae, & Berti, 1986; Bisiach, in press). Prigatano and collaborators have done some pioneering studies on attempted remediation of awareness of deficits in head-injured patients with various kinds of deficits

(Prigatano, 1986). Stuss and Benson (1986) have emphasized and discussed the role of the frontal lobes in monitoring and awareness of deficits. These and other developments are discussed in a comprehensive review paper on unawareness of deficits by McGlynn and Schacter (1989), and in chapters contained in a volume edited by Prigatano and Schacter (in press).

Before proceeding further, it should be emphasized that, when I refer to anosognosia or unawareness of deficit, I assume that there is more to the phenomenon than simple defensive or motivated denial of disability like that observed in non-brain-damaged patients. Although defensive denial no doubt plays a role in some cases of anosognosia, there are a variety of good reasons to believe that it is not the entire story, as discussed by Bisiach (in press), Bisiach et al. (1986), McGlynn and Kaszniak (in press), McGlynn and Schacter (1989), and Prigatano (1986).

In order to facilitate comparisons with the earlier discussion of implicit memory in amnesia, I will focus primarily on unawareness of memory deficit. McGlynn and Schacter (1989) noted a striking convergence of clinical, questionnaire, and experimental studies on the following point: Amnesic patients who are characterized by signs of frontal-lobe pathology generally exhibit diminished awareness of their memory problems, whereas amnesics whose neuropathology is restricted to medial temporal regions exhibit relatively intact insight into their memory disorder (see also Schacter, in press-b; Schacter, Glisky, & McGlynn, in press). The main question for the present purposes is whether unawareness of memory deficit in amnesic patients with frontal-lobe signs should be given a first- or second-order theoretical interpretation: Is unawareness directly attributable to a breakdown in, or disconnection of, an awareness system that operates across multiple domains? Or does it reflect instead a dysfunction of some other domain-specific cognitive or memory process, with unawareness being an indirect or secondary consequence of this?

Let us begin by considering the possibilities for a second-order account of unawareness of memory deficit. Perhaps the most obvious candidate for a second-order account ascribes the memory deficit itself a causal role in producing unawareness: Maybe anosognosic amnesic patients simply cannot remember that they have a memory problem. The difficulty with this idea, however, stems from the aforementioned fact that severely amnesic patients with restricted medial temporal damage generally exhibit awareness of their deficits. Although various kinds of evidence reviewed by McGlynn and Schacter support this contention, let me just provide a couple of compelling clinical examples. In Rose and Symonds' (1960) paper on global amnesia consequent to encephalitis, one densely amnesic patient commented that "There's nothing wrong with me physically but mentally things as they happen don't seem to impress themselves on my mind (p.195)", and another stated that "It appears to me that my memory is distant and I do not seem to be able to know anything very recent (p.200)."

These and other sources of evidence indicate that memory impairment alone is likely not a sufficient condition for producing unawareness of memory deficit; something else must be involved. A severe memory deficit, however, may help to *sustain* unawareness. This point is illustrated by an anecdote from a naturalistic case study published several years ago (Schacter, 1983) in which I observed the memory performance of a profoundly amnesic patient during two rounds of golf. This patient, who was then in the early stages of Alzheimer's disease, exhibited only dim awareness of his memory problem. After completing play on a particular hole, we left the green and walked several feet to the next tee. I then asked the patient whether he could tell me where he had just hit his putts on the previous green. He failed to remember what he had done, and in a shocked tone, claimed that his memory must be "a complete mess" in order to fail such an apparently simple memory test. But in response to a question about the state of his memory that I posed several minutes later, when he could no longer recollect this incident, he indicated that he only had a slight problem with his memory that was nothing to be concerned about. The patient's amnesia apparently sustained his unawareness of it. Another second-order account that is related but not identical to the previous one emerges from consideration of the question of how an amnesic patient ever becomes aware that he or she does have a serious memory impairment. One possibility is that the patient gradually learns about the deficit over time, as a function of extensive feedback concerning memory failure in the real world (Schacter, in press-b). We know that many amnesic patients can acquire knowledge and skills gradually through repetition. Perhaps frontal-lobe pathology interferes with this ability, such that some amnesic patients do not "learn" about their deficit. There are, however, two problems with this idea. First, in our own research, we have found that patients who are unaware of their deficits nevertheless show good incremental learning of complex knowledge and skills (e.g., Glisky et al., 1986a,b). Second, patients with transient global amnesia, generally thought to be attributable to some form of medial temporal dysfunction, appear to be aware of their memory loss immediately after it occurs, even when there has been no opportunity for gradual or repetitive learning about it. Thus, for example, Evans (1966) described a patient who had a sudden onset transient amnesia while taking a bath: "His first words were 'Am I going mad? I can't remember anything." Many similar observations have been reported in the literature on transient global amnesia (see McGlynn & Schacter, 1989). It thus seems unlikely that unawareness of memory deficit is attributable in any simple way to a learning deficit.

Another approach to developing a second-order account of unawareness of memory deficit would be to seize on the observed relation with frontal-lobe damage, and ask whether disruptions to any of the various functions normally associated with frontal functions could produce unawareness. For instance, it has been well-established by numerous investigators that patients with frontal impairments have serious problems inhibiting strong though inappropriate

response tendencies, and that they perform poorly on tasks that require making temporal discriminations and gaining access to temporal information (for discussion, see Butters & Miliotis, 1985; Milner, Petrides, & Smith, 1985; Moscovitch, 1982; Schacter, 1987c; Squire, 1987; Stuss & Benson, 1986). It is not difficult to imagine how such deficits could be involved in unawareness. If, for example, an amnesic patient with frontal damage were queried about the state of his or her memory, the "strongest" response available may pertain to the patient's *premorbid* memory function; unless this response is inhibited, the patient would report that memory function is unimpaired. If the patient is also unable to gain access to temporal information regarding the appropriateness of this response -- that is, that the response that has come to mind concerns the past and not the present -- then unawareness of memory deficit would be an inevitable result. I do not know of any empirical research that has systematically tested this idea, but it seems worth exploring.

In addition to their role in response inhibition and temporal memory, the frontal lobes have been linked directly to such functions as monitoring and awareness of complex mental activity, particularly by Stuss and Benson (1986). Accordingly, it is possible that frontal-lobe dysfunction could provide the basis for formulating a first-order account of unawareness of memory deficits in which a frontally based monitoring system is itself disrupted or perhaps disconnected from the memory system. Thus, for example, when memory fails in a transient global amnesia patient -- who likely has intact frontal functions and hence an intact monitoring system -- this failure can be appropriately monitored on-line, thus producing immediate awareness of deficit. A patient with extensive frontal damage, however, may not spontaneously engage in, or even be capable of, on-line monitoring of memory failure. An interesting though little-investigated question concerns whether such patients are capable of engaging in on-line monitoring of other response failures. If the monitoring defect were highly specific and restricted to memory, it would suggest a disconnection between a frontally based monitoring system and a memory system; a more global unawareness would suggest damage to the monitoring system itself.

Although it is beyond the present scope to examine anosognosia in other neuropsychological syndromes, it seems reasonable to suggest that the distinction between first- and second-order theoretical accounts could serve as a useful heuristic device when thinking about the kinds of models and ideas that can best explain these manifestations of unawareness.

RELATION BETWEEN IMPLICIT KNOWLEDGE AND ANOSOGNOSIA

To conclude the discussion, let us consider briefly the relation between the kinds of unawareness observed in cases of implicit knowledge and anosognosia. Are these phenomena simply different manifestations of disturbances in the same

underlying mechanism or mechanisms? Or are they attributable to impairments of distinct mechanisms? In the absence of an adequate theory of either implicit knowledge or anosognosia, this question cannot be answered definitively. Nevertheless, there exist some empirical grounds, at least within the domain of memory impairment, to argue that different mechanisms are responsible for unawareness of knowledge and deficit, respectively (see Schacter, in press-b, for more extensive discussion). First, preserved implicit memory, in the form of skill learning and priming effects expressed without any awareness of remembering, has been observed in densely amnesic patients with no known signs of frontal-lobe pathology. In contrast, unawareness of deficit is almost always associated with additional frontal-lobe signs.

A second reason for doubting that unawareness of knowledge and deficit are manifestations of the same underlying deficit stems from observations concerning a profoundly amnesic head-injured patient, K.C., who has been studied extensively by several investigators (see Tulving, Schacter, McLachlan, & Moscovitch, 1988). K.C. exhibits striking dissociations between implicit and explicit memory in a variety of situations: he shows robust priming effects on standard stem completion and free association tasks (Schacter, 1985; Schacter & Graf, 1986), as well as long-term priming on the sentence puzzle task that was described earlier (McAndrews et al., 1987). In addition, K.C. learned and retained complex new knowledge and skills concerning the operation and programming of a microcomputer -- even though each time he sat down anew to perform the computer task, he was unaware that he had ever worked on a computer (Glisky et al., 1986a and b; Glisky & Schacter, 1988). Yet K.C. appears to exhibit substantial awareness of his memory disorder in a variety of situations (see Schacter, in press-b). Evidence for awareness is perhaps somewhat perplexing in light of the preceding discussion, because K.C. has massive left frontal damage. Right frontal regions are relatively spared, however, and this may contribute to his relatively preserved awareness of deficit. In any case, observations concerning K.C. suggest that unawareness of deficit and knowledge can be dissociated.

Returning to the theoretical model that was presented earlier (Figure 1), it is perhaps not surprising that such a dissociation can be observed. In the model, the executive system is held to be responsible for intentional retrieval and ongoing monitoring of complex internal activities; the executive system is also held to be frontally based. As discussed earlier, however, dissociations between implicit and explicit knowledge are attributed to either a damaged episodic system together with intact access to knowledge modules and the procedural/habit system, or to an intact episodic system that is disconnected from the awareness system. Thus, the executive system is not held to be critically involved in implicit/explicit memory dissociations. What this means is that the locus of the disruption that produces unawareness of memory deficit is different from the loci of the various disturbances that may be associated with implicit memory effects. Accordingly, the model would not lead one to expect a close relation

between unawareness of memory deficit and implicit memory.

The model does suggest, however, one set of circumstances in which a relation between the two phenomena should be observed. Under normal conditions, CAS takes as input the highly activated outputs of various modules; weakly activated outputs do not gain access to the awareness system. Suppose that conditions exist under which CAS cas be selectively disconnected from a damaged module. The awareness system would then no longer receive those highly activated outputs that "alert" it to the state of the module's activity. Thus, with respect to CAS, the disconnected module would be in perpetual state of resting or baseline activity, no different from any other normally functioning module that is in a low state of activation. As McGlynn and Schacter (1989) have suggested, unawareness of deficit could result from such a disconnection, because information about a module's damaged condition would not be available to the patient consciously. With respect to implicit knowledge, this idea predicts that patients in whom implicit/explicit dissociations demand a first-order explanation -- that is, the dissociation can be attributed at least in part to a disconnection between CAS and a particular module -- should also exhibit poor awareness of their deficit. Unfortunately, as discussed earlier, unequivocal evidence in support of such a first-order interpretation of preserved implicit knowledge is not yet available, so it is difficult to assess the validity of this hypothesis. Perhaps it could be useful, however, in guiding further research.

Despite the enormous conceptual and empirical difficulties in attempting to study rigorously an issue as thorny and even ephemeral as that of the nature of phenomenal awareness, there are grounds for expressing optimism concerning the prospects for a cognitive neuropsychological approach. Cognitive neuropsychology has made impressive strides during the past decade, particularly in the areas of language, memory, object recognition, and reading. By bringing to bear on the problem of awareness some of the analytic tools developed in these and other sectors of research, perhaps we will be in a position to make some modest contributions toward understanding on one of the enduring mysteries of the human mind.

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