

Review

Implicit knowledge: New perspectives on unconscious processes

Daniel L. Schacter

Department of Psychology, Harvard University, Cambridge, MA 02138

ABSTRACT Recent evidence from cognitive science and neuroscience indicates that brain-damaged patients and normal subjects can exhibit nonconscious or implicit knowledge of stimuli that they fail to recollect consciously or perceive explicitly. Dissociations between implicit and explicit knowledge, which have been observed across a variety of domains, tasks, and materials, raise fundamental questions about the nature of perception, memory, and consciousness. This article provides a selective review of relevant evidence and considers such phenomena as priming and implicit memory in amnesic patients and normal subjects, perception without awareness and “blindsight” in patients with damage to visual cortex, and nonconscious recognition of familiar faces in patients with facial-recognition deficits (prosopagnosia). A variety of theoretical approaches to implicit/explicit dissociations are considered. One view is that all of the various dissociations can be attributed to disruption or disconnection of a common mechanism underlying conscious experience; an alternative possibility is that each dissociation requires a separate explanation in terms of domain-specific processes and systems. More generally, it is concluded that rather than reflecting the operation of affectively charged unconscious processes of the kind invoked by psychodynamic or Freudian theorists, dissociations between implicit and explicit knowledge are a natural consequence of the ordinary computations of the brain.

Consider the following two clinical scenarios. In the first, a patient with memory problems is shown a list of familiar words and several minutes later is unable to remember any of the list items when asked to recollect them; indeed, he denies that a list of words had been presented. But when he is required to perform an incidental test that does not require conscious recollection of the list, the patient’s performance indicates perfectly normal retention of the previously studied words. In the second scenario, a patient with perceptual problems is exposed to a bright visual stimulus and claims to see nothing. Yet when asked to “guess” in which of two locations the stimulus appeared, the patient performs well above the chance level, indicating that she has in some sense “seen”—

despite the absence of conscious experience—the target stimulus.

The foregoing scenarios may seem surprising and even bizarre: How can a patient exhibit memory without remembering or perception without perceiving? It is tempting to suggest that the patients suffer from psychiatric problems or, perhaps, are engaging in outright deception of the examiner. On the contrary, however, these two scenarios represent examples of what have become almost commonplace observations in the neuropsychological laboratory and are often referred to as dissociations between explicit and implicit knowledge (1). *Explicit* knowledge refers to knowledge that is expressed as conscious experience and that people are aware that they possess; the everyday uses of such terms as “seeing” and “remembering” refer to explicit knowledge. *Implicit* knowledge, by contrast, refers to knowledge that is revealed in task performance without any corresponding phenomenal awareness; implicit knowledge is often expressed unintentionally and tapped indirectly. Far from reflecting psychiatric symptoms or dissimulation, dissociations between explicit and implicit knowledge are providing important new insights into the fundamental nature of perception, memory, and conscious experience.

The terms explicit and implicit knowledge are quite similar in meaning to conscious and unconscious knowledge, and the two sets of terms can be used interchangeably. However, traditional conceptions of unconscious knowledge have been tied closely to Freudian and other psychodynamic constructs such as repression, drive, conflict, and the like. As this article should make evident, these concepts have little relevance to the kinds of phenomena that have been the subject of recent neuropsychological and cognitive studies. Because the classical notion of the “unconscious” is so closely linked to psychodynamic ideas, it seems prudent to use terminology that is not similarly burdened (for more extended discussion, see ref. 2).

The article provides a selective overview of research that has documented and explored dissociations between explicit and implicit knowledge. It will focus primarily on explicit/implicit dissociations in patients with memory disorders

and perceptual disorders, although similar phenomena that have been documented in other patient populations will also be noted. In addition, some attention will be paid to analogous dissociations that have been produced in cognitive studies of normal, non-brain-damaged subjects. The article will conclude by surveying theoretical accounts of the various dissociations and by considering whether these diverse phenomena depend on similar underlying mechanisms. Taken together, the neuropsychological and cognitive evidence suggests that, rather than reflecting the operation of affectively charged psychodynamic processes, many implicit or unconscious expressions of knowledge occur as a relatively routine consequence of the ordinary computations of the brain.

Memory Disorders

The most extensively studied neurological disorder of memory is known as the *amnesic syndrome*, which occurs as a consequence of various kinds of pathological conditions (e.g., stroke, encephalitis, anoxia) that produce damage to medial temporal and diencephalic brain regions (3, 4). Amnesic patients are characterized by a marked inability to remember recent experiences together with normal perception and intelligence. Their memory disorders are evident on a variety of explicit memory tests, including free recall, where patients attempt to retrieve recently presented items without the aid of experimenter-provided cues; cued recall, where various cues or hints are provided to assist recollection; and recognition, where previously studied items are presented together with new items, and subjects indicate which item they recollect from the study list.

Despite their severe impairments in explicitly remembering recently presented information, it has been established beyond dispute that amnesic patients can show intact implicit memory for aspects of the same information. One of the most intensively studied implicit memory phenomena in amnesia is known as repetition or direct *priming*: the facilitated ability to identify, or make judgments about, target stimuli as a consequence of a recent exposure to them (5). In a typical priming experiment, subjects

are shown a list of familiar words and are later given an apparently unrelated test that does not require explicit memory for the study list. For example, on a stem completion test, subjects would be given three-letter word beginnings (e.g., T-A-B—) and asked to complete them with the first word that comes to mind. Half of the stems could be completed with words that had appeared previously on the study list (e.g., TABLE), and the other half could not be completed with words from the study list. Priming is said to occur when subjects provide the target completion more frequently to stems that represent studied words than to stems that represent nonstudied words. Early studies by Warrington and Weiskrantz (6) indicated that amnesic patients show what we would now call normal priming effects on the stem-completion task despite poor explicit memory, although Warrington and Weiskrantz did not refer to the phenomenon as priming. Subsequent experiments confirmed and extended the finding of normal stem-completion priming in amnesia and also revealed that amnesics exhibit normal priming effects on a variety of other implicit memory tasks (refs. 7–12; for review, see ref. 13).

Recent research has extended further the boundaries of the priming phenomenon. In early experiments, the target items presented for study were well-learned materials, such as familiar words, that exist in memory before the experiment. Thus, it was possible to argue that priming effects in amnesic patients reflect the temporary activation of preexisting memory representations (e.g., refs. 7 and 14). However, it has now been established that amnesic patients can show intact priming for novel information that does not have a preexisting memory representation, including pseudowords (e.g., “numdy”; refs. 15 and 16) and nonverbal materials, such as novel objects or patterns (17–20). It has also been shown that priming effects in amnesia can be quite long-lived, lasting across retention intervals of days, weeks, or months (21–23).

Research on priming effects in amnesic patients has been complemented by a large and ever-increasing literature on normal subjects, indicating that priming can be dissociated sharply from explicit memory (for reviews, see refs. 24–26). One particularly important finding is that priming effects on various implicit memory tests are relatively unaffected by manipulations of how subjects encode target materials during study-list presentation. For example, when subjects are induced to process the semantic attributes of words at the time of study (e.g., make judgments about a word's meaning), their recollection of the word on subsequent explicit memory tests is generally much

higher than when they are induced to process nonsemantic physical features of the target words (e.g., count the number of vowels in a word). But the magnitude of priming effects is similar following the two kinds of study tasks (20, 27, 28). Moreover, the priming effect appears to be modality-specific: It is reduced by study-to-test changes in visual or auditory modality of presentation (28, 29). Under certain circumstances, priming is even reduced by study-to-test changes in the particular type font or case in which a word appears (30, 31) or the voice in which a word is spoken (32). These kinds of observations, taken together with the amnesia data, have led to a vigorous debate concerning the psychological and neurophysiological processes and systems that subserve priming and explicit memory, respectively (cf. refs. 3, 25, and 33).

Priming is not, however, the only example of preserved implicit memory in amnesic patients. It has been known since the classic studies of Milner and Corkin and their colleagues (34) that amnesic patients can acquire new motor skills across numerous training sessions, and it is now clear that they can gradually acquire perceptual and cognitive skills as well (cf. refs. 35–37). Amnesics have also exhibited normal implicit learning of the rules of an artificial grammar (38) and a complex spatio-temporal sequence (39) and have proved capable of acquiring classically conditioned responses (40, 41). Severely amnesic patients have even been able to learn (although not at a normal rate) the complex knowledge and skills needed to operate and program a computer and have exhibited robust retention of such knowledge over delays of 5–9 mo (42, 43)—despite little or no explicit memory for their learning experiences.

Some of this work on implicit memory for complex knowledge and skills has a parallel in—and, in fact, was inspired by—studies of normal subjects. For example, Reber has reported a series of studies over the past 25 yr (refs. 44 and 45) that have provided evidence for implicit learning of grammatical rules: After exposure to a list of consonant strings that are ordered according to the complex rules of an artificial grammar, subjects can later distinguish novel grammatical strings from novel nongrammatical strings, even though the subjects are unable to articulate the nature of the rule (for a critique, see ref. 46). Similarly, Lewicki and colleagues (47) have provided evidence that subjects can learn complex patterns and contingencies despite poor explicit knowledge of them.

Taken together, the data from amnesic patients and normal subjects indicate clearly that memory for various kinds of experiences can be expressed indepen-

dently from, and in the absence of, conscious recollection of those experiences.

Perceptual Disorders

At about the same time that early evidence was accumulating on implicit memory in amnesic patients, there were reports of a puzzling and, in some respects, analogous phenomenon in patients with disturbances of visual perception. Initially documented by Poppel, Held, and Frost (48), the phenomenon was referred to as “blindsight” by Weiskrantz and colleagues (49), in reference to the seemingly paradoxical nature of the visual behavior exhibited by certain patients with lesions to primary (striate) visual cortex. Such patients appeared to be “blind” in the sense that they denied seeing a stimulus presented in certain parts of the visual field. But when asked to guess about the location or other attributes of the same stimulus, the patients exhibited “sight” in the sense that their guessing performance was well above chance and sometimes nearly perfect.

The experimental paradigm used most frequently to demonstrate blindsight involves requiring patients to localize a stimulus presented in the blind field, either by pointing to or reaching toward the target location or by making a verbal response. Blindsight can also be exhibited when patients are asked to make visual discriminations about other kinds of targets by guessing, including simple figures (e.g., X vs. O) and line orientations (e.g., horizontal vs. vertical). However, Perenin and Jeannerod (50) failed to find evidence for pattern discrimination, and Weiskrantz (51) has suggested that orientation discrimination is more fully preserved than form discrimination in blindsight patients. Finally, blindsight has also been demonstrated by using experimental paradigms in which information presented in the blind field influences patients' perceptions of, and responses to, information presented in the sighted field (e.g., ref. 52).

One of the most intriguing questions about blindsight concerns exactly what patients experience when they respond accurately to stimuli presented in the blind field and how these “perceptions” differ from those of conscious visual experience. Although it is most often reported that patients claim to see nothing and are merely guessing, it has also been reported that some patients occasionally describe a rather primitive visual awareness of a target. For example, a patient studied by Weiskrantz (51) claimed to sense “a definite pinpoint of light” but when probed further insisted that it did not “actually look like a light [but] . . . nothing at all” (ref. 51, p. 378). It has also been reported that with extensive practice and training, patients can

develop a heightened awareness of stimuli in their blind fields (51, 53). In view of this evidence, some critics have suggested that rather than representing implicit or nonconscious perception of target stimuli, blindsight can be attributed to cautious responding on the basis of degraded (but conscious) vision that is produced by scattered light (54). However, such explanations have difficulty accommodating various kinds of evidence (for discussion, see refs. 1 and 55).

Recent research has revealed implicit perceptual knowledge in other kinds of brain-damaged patients that is, in some respects, similar to that observed in blindsight. One particularly striking example comes from the study of *visual form agnosia*, a disorder in which patients have difficulty perceiving and recognizing virtually all kinds of visual objects. Goodale, Milner, Jakobson, and Carey (56) described a patient who was severely impaired in making judgments about the width of three-dimensional objects but made entirely normal motor adjustments of hand position as she reached toward a target object; the positioning of her finger-thumb grip varied directly as a function of the object's width despite her impairment of conscious perception.

There has also been a good deal of recent research on implicit knowledge in patients with *prosopagnosia*—the impaired ability to recognize familiar faces, usually because of bilateral lesions to occipito-temporal cortex. Although such patients typically deny any familiarity with faces that ordinarily would be well known to them (e.g., a spouse or relative), there is now considerable evidence that they possess implicit knowledge of those faces. Early evidence was provided in a psychophysiological study by Bauer (57), in which a prosopagnosic patient viewed a familiar face and at the same time listened to the experimenter read a series of names; one belonged to the face and the others did not. Despite failing to recognize the face explicitly, the patient showed a maximal skin conductance response to the correct name. Tranel and Damasio (58) replicated and extended this phenomenon using a different paradigm for eliciting skin-conductance responses to familiar faces that their patient failed to recognize explicitly.

Young and De Haan and their colleagues have provided a systematic series of studies using behavioral measures to demonstrate and explore implicit facial familiarity in prosopagnosic patients (for review, see ref. 59). For example, they reported the case of a patient who performed at chance levels when required to choose which of two faces (one famous, one unknown) was familiar. However, when given a matching task in which subjects judged whether two simultane-

ously exposed faces were the same or different, the patient—just like normal control subjects—responded more quickly when the two faces were famous than when they were unknown. Similarly, they also found that the patient was slower to learn a name-face pairing when a familiar face was paired with an incorrect name than when it was paired with a correct name, even though he claimed that none of the faces were familiar; and presentation of a famous face that was unfamiliar to the patient speeded up his ability to make judgments about verbal information associated with the face (e.g., seeing a photo of Prince Charles facilitated his response to the name Princess Diana). Interestingly, Young and colleagues have reported another case in which the patient failed to show evidence for implicit knowledge of unrecognized faces (see also ref. 60).

The data from blindsight, visual form agnosia, and prosopagnosia indicate clearly that conditions exist in which some patients can show implicit knowledge of visual stimuli that they fail either to perceive or to recognize explicitly. Cognitive research with normal, non-brain-damaged subjects has long been concerned with the possible existence of "perception without awareness" or "subliminal perception". Early research in this area was fraught with methodological difficulties and marked by controversy (for reviews, see refs. 2 and 61). Although some of the confusion still persists in contemporary work, a number of findings and ideas have emerged during the past decade that have helped to clarify the sense in which, and extent to which, perception without awareness can be said to exist.

In a typical paradigm for studying perception without awareness, two kinds of evidence are provided: (i) explicit or direct measures that are used to document subjects' failure to perceive a stimulus consciously and (ii) implicit or indirect measures that reveal an impact of the undetected stimulus on some aspect of performance. For example, in a semantic priming paradigm, the subject may claim that he or she fails to detect the presence of the word chair when it is flashed briefly and obscured by a visual mask; but the subject will nevertheless be faster to identify or make a judgment about the related word table when it is presented immediately after the word chair than when it is presented after a semantically unrelated word (e.g., ref. 62). The existence of a semantic priming effect suggests that subjects have, indeed, registered some features of the target stimulus despite the apparent absence of conscious perception.

Why has research of this kind been dogged by controversy? As Merikle (ref. 63, p. 792) has stated, the controversy

"... has centered on the issue, What constitutes an adequate behavioral measure of conscious perceptual experience? Depending upon one's answer to this question, the evidence for perception without awareness is either overwhelming or nonexistent." Cheesman and Merikle (64) proposed a useful distinction between *subjective* and *objective* measures of conscious perception. Subjective measures typically involve a person's verbal statement that he or she does not detect the presence of a target stimulus, as in the foregoing example. Objective measures, by contrast, typically involve such tasks as forced-choice judgments in which subjects must choose between the presented stimulus and a nonpresented alternative, even when they feel that they are just guessing. As Merikle (63) points out, if one accepts as valid subjective measures of conscious perception, then the evidence for perception without awareness is strong (e.g., there is evidence for semantic priming from stimuli that subjects claim that they do not see); if one insists on an objective measure, however, then the evidence is weak or nonexistent (e.g., there is little evidence of semantic priming from stimuli about which subjects are unable to make accurate forced-choice discriminations).

Although the intricacies of this debate are beyond the purview of the present article, it is worth noting that the distinction between objective and subjective measures of conscious perception has implications for our understanding of neuropsychological phenomena, such as blindsight. In the blindsight literature, failures of conscious perception are typically inferred from subjective measures; patients claim that they do not see a target stimulus. Moreover, evidence for *implicit* knowledge or *nonconscious* perception is frequently inferred from above-chance performance on a forced-choice test in which the patient claims to be guessing—precisely the kind of test that some would refer to as an objective measure of *conscious* perception! To make matters even more complex, chance-level performance on forced-choice tests has been taken as evidence for an absence of conscious perception in some studies of implicit or covert recognition in prosopagnosia (59). Merikle (63) has delineated several reasons why it probably makes sense to accept data from subjective measures as evidence for failure of conscious perception. Nevertheless, it seems clear that careful attention must be paid to possible differences in underlying mechanisms when implicit knowledge is inferred from failures on subjective or objective measure of conscious perception, respectively.

Additional Neuropsychological Evidence for Implicit Knowledge

Although most of the work on implicit knowledge in neuropsychological syndromes has involved disorders of memory and perception, similar kinds of evidence have been gleaned from patients with a variety of neuropsychological deficits. Milberg and Blumstein and their colleagues (65), for example, have studied aphasic patients who exhibit severe deficits on explicit tests of language comprehension and yet show robust semantic priming effects for words that they fail to understand explicitly. Tyler (66) has described other kinds of aphasic patients who are unable to make explicit judgments about the meaning and grammaticality of sentences. But the performance of these patients on a target-monitoring task was disrupted by semantic and grammatical violations, and the pattern of disruption was similar to that observed in normal control subjects. Evidence for implicit knowledge has also been seen in studies of patients with reading disorders, who appear able to make judgments about properties of words that they cannot identify consciously (e.g., ref. 67). And observations suggestive of implicit knowledge have been reported in patients who exhibit spatial neglect, associative agnosia, and unawareness of deficit (for review, see ref. 1).

Theories and Mechanisms

The seemingly ubiquitous evidence for preserved implicit knowledge despite impaired explicit knowledge across a variety of patient groups, experimental tasks, and knowledge domains is compelling. Moreover, the converging evidence in several instances from studies of non-brain-damaged subjects indicates that the basic phenomenon is characteristic of normal cognitive function and is not some sort of exotic curiosity that occurs only in pathological conditions. What are we to make of these striking and counterintuitive phenomena? Although current theoretical understanding of them is rather modest, several different approaches can be distinguished.

The "family resemblance" among the various implicit/explicit dissociations across a variety of conditions has suggested to some that it is appropriate to seek a common explanation for them. For instance, Schacter *et al.* (1) speculated that a common mechanism may underlie conscious experiences of perceiving, knowing, and remembering—a high-level system that takes as its input the extensively processed output of perceptual and semantic representation systems and that must be activated for phenomenal awareness in different domains (see also ref. 68). They suggested further

that this mechanism can become selectively disconnected from individual brain modules that process and represent particular kinds of information. If such modules continue to function relatively normally, then the information on which they operate could affect performance and behavior implicitly, without any corresponding phenomenal awareness. A disconnection account of this kind seems to fit well with the neuropsychological data because patients do not suffer from generalized impairments of conscious experience; their problems with explicit knowledge are domain specific (see also ref. 55). One difficulty with this approach, however, is that it implies the existence of a "consciousness module" despite the paucity of experimental evidence for such a module. Another approach to a common explanation for a variety of implicit/explicit dissociations has been put forward by Edelman (69), who suggested that they may be attributable to selective dysfunctions of *re-entrant loops*—connections among brain regions that, when activated, are ordinarily responsible for particular kinds of conscious experiences of perceiving, knowing, and remembering. This approach represents a parsimonious attempt to accommodate numerous implicit/explicit dissociations without postulating a consciousness module, but there is, as yet, no direct empirical support for it.

In contrast to these attempts to develop a single account for a variety of phenomena, other researchers have focused on individual implicit/explicit dissociations. For example, a number of investigators have suggested that priming, skill learning, and other manifestations of implicit memory reflect the activity of memory systems that are spared in amnesic patients. These systems can function independently of the memory system that ordinarily supports explicit memory, depends on the integrity of the hippocampus and related structures, and is impaired in amnesia (cf. refs. 3, 4, 35, and 70). To illustrate, it has been suggested that priming depends on changes in early-stage perceptual representation systems that preserve information about the form and structure, but not the meaning and associative properties, of words and objects (e.g., refs. 5, 33, 70, and 71). Neuropsychological and neuroimaging evidence indicates that such systems depend on posterior cortical structures (cf. refs. 33 and 72), which is consistent with the proposal that they can function normally in amnesic patients. Although experience-induced changes in perceptual representation systems can provide a basis for facilitated identification of degraded words and objects, they do not provide access to the kind of contextual and associative information that is important for conscious recollection and that

appears to depend on the hippocampus and related structures. Thus, by this view priming and explicit remembering depend on different underlying memory systems.

An important observation supporting this kind of multiple memory systems account is that amnesic patients typically exhibit *normal* levels of implicit memory despite severely impaired explicit memory; accordingly, it makes sense to postulate that independent brain systems support the two forms of memory. By contrast, in the other neuropsychological syndromes discussed in this article, patients typically do not exhibit entirely normal performance on tasks that tap implicit knowledge, so it is more difficult to argue that independent brain systems underlie explicit and implicit knowledge (for discussion of the "multiple visual systems" approach to blindsight, see refs. 55, 56, and 73). For example, Wallace and Farah (74) have suggested that in some cases of prosopagnosia, residual implicit knowledge may be a natural consequence of impairment to the facial processing system that normally supports explicit knowledge. They noted that in simulations of prosopagnosia with a neural network, when "lesions" are made to a part of the network that supports facial recognition, the network still shows some residual ability to "perform" tasks analogous to those used to demonstrate implicit facial knowledge in prosopagnosic patients. This kind of observation is consistent with the idea that when patients exhibit some, but not normal, levels of implicit knowledge, the effect may be attributable to the impaired functioning of a damaged system that normally supports explicit knowledge.

Because research in this area is still in its infancy, it is too early to state confidently whether a unified theoretical account of different implicit/explicit dissociations will be possible or whether it will be necessary to construct separate domain-specific theories for each particular kind of dissociation. Current evidence does suggest, however, that demonstrations of fully intact implicit memory in amnesic patients probably demand a different kind of explanation than do demonstrations of residual (but not normal) implicit knowledge in blindsight, prosopagnosia, and other syndromes.

Whatever the ultimate theoretical account of implicit/explicit dissociations, the fact that these phenomena can be observed in normal subjects as well as neurological populations indicates that they are not exotic or unusual symptoms that represent pathological consequences of brain damage. Nor are these dissociations intimately intertwined with emotional conflicts or psychodynamic processes (e.g., repression) that were crucial to postulation of the Freudian uncon-

scious (e.g., ref. 75). Rather, dissociations between implicit and explicit knowledge seem to arise as a natural consequence of the functional architecture of the brain and reflect the activity of computations that are routinely performed during the course of perceiving, recognizing, and remembering. A major challenge for future research is to understand more deeply the properties of the architecture and the nature of the computations responsible for dissociations between implicit and explicit knowledge.

I thank Dana Osowiecki for help with preparation of the manuscript. The article was supported by Grant RO1 MH45938-01A3 from the National Institute of Mental Health.

1. Schacter, D. L., McAndrews, M. P. & Moscovitch, M. (1988) in *Thought Without Language*, ed. Weiskrantz, L. (Clarendon, Oxford), pp. 242-278.
2. Greenwald, A. G. (1992) *Am. Psychol.* **47**, 766-779.
3. Squire, L. R. (1992) *Psychol. Rev.* **99**, 195-231.
4. Weiskrantz, L. (1985) in *Memory Systems of the Human Brain: Animal and Human Cognitive Processes*, eds. Weinberger, N. M., McGaugh, J. L. & Lynch, G. (Guilford, New York), pp. 380-415.
5. Tulving, E. & Schacter, D. L. (1990) *Science* **247**, 301-306.
6. Warrington, E. K. & Weiskrantz, L. (1974) *Neuropsychologia* **12**, 419-428.
7. Graf, P., Squire, L. R. & Mandler, G. (1984) *J. Exp. Psychol. Learn. Mem. Cognit.* **10**, 164-178.
8. Cermak, L. S., Talbot, N., Chandler, K. & Wolbarst, L. R. (1985) *Neuropsychologia* **23**, 615-622.
9. Jacoby, L. L. & Witherspoon, D. (1982) *Can. J. Psychol.* **36**, 300-324.
10. Moscovitch, M. (1982) in *Human Memory and Amnesia*, ed. Cermak, L. S. (Erlbaum, Hillsdale, NJ), pp. 337-370.
11. Shimamura, A. P. & Squire, L. R. (1984) *J. Exp. Psychol. Gen.* **113**, 556-570.
12. Schacter, D. L. (1985) *Ann. N.Y. Acad. Sci.* **444**, 44-53.
13. Shimamura, A. P. (1986) *Q. J. Exp. Psychol.* **38A**, 619-644.
14. Rozin, P. (1976) in *Neural Mechanisms of Learning and Memory*, eds. Rosenzweig, M. R. & Bennett, E. L. (MIT Press, Cambridge, MA), pp. 3-48.
15. Cermak, L. S., Bleich, R. P. & Blackford, M. (1988) *Brain Cognit.* **7**, 145-156.
16. Haist, F., Musen, G. & Squire, L. R. (1991) *Psychobiology* **19**, 275-285.
17. Gabrieli, J. D. E., Milberg, W., Keane, M. M. & Corkin, S. (1990) *Neuropsychologia* **28**, 417-428.
18. Musen, G. & Squire, L. R. (1992) *Mem. Cognit.* **20**, 441-448.
19. Schacter, D. L., Cooper, L. A., Tharan, M. & Rubens, A. B. (1991) *J. Cognit. Neurosci.* **3**, 118-131.
20. Bowers, J. S. & Schacter, D. L., in *Implicit Memory: New Directions in Cognition, Neuropsychology, and Development*, eds. Graf, P. & Masson, M. E. J. (Academic, New York), in press.
21. Cave, C. B. & Squire, L. R. (1992) *J. Exp. Psychol. Learn. Mem. Cognit.* **18**, 509-520.
22. MacAndrews, M. P., Glisky, E. L. & Schacter, D. L. (1987) *Neuropsychologia* **25**, 497-506.
23. Tulving, E., Hayman, C. A. G. & MacDonald, C. (1991) *J. Exp. Psychol. Learn. Mem. Cognit.* **17**, 595-617.
24. Richardson-Klavehn, A. & Bjork, R. A. (1988) *Annu. Rev. Psychol.* **36**, 475-543.
25. Roediger, H. L., III (1990) *Am. Psychol.* **45**, 1043-1056.
26. Schacter, D. L. (1987) *J. Exp. Psychol. Learn. Mem. Cognit.* **13**, 501-518.
27. Graf, P., Mandler, G. & Haden, P. (1982) *Science* **218**, 1243-1244.
28. Jacoby, L. L. & Dallas, M. (1981) *J. Exp. Psychol. Gen.* **110**, 306-340.
29. Roediger, H. L. & Blaxton, T. A. (1987) *Mem. Cognit.* **15**, 379-388.
30. Graf, P. & Ryan, L. (1990) *J. Exp. Psychol. Learn. Mem. Cognit.* **16**, 978-992.
31. Marsolek, C. J., Kosslyn, S. M. & Squire, L. R. (1992) *J. Exp. Psychol. Learn. Mem. Cognit.* **18**, 492-508.
32. Schacter, D. L. & Church, B. (1992) *J. Exp. Psychol. Learn. Mem. Cognit.* **18**, 915-930.
33. Schacter, D. L. (1992) *Am. Psychol.* **47**, 559-569.
34. Milner, B., Corkin, S. & Teuber, H. L. (1968) *Neuropsychologia* **6**, 215-234.
35. Cohen, N. J. & Squire, L. R. (1980) *Science* **210**, 207-210.
36. Saint-Cyr, J. A., Taylor, A. E. & Lang, A. E. (1988) *Brain* **111**, 941-959.
37. Squire, L. R. & Zola-Morgan, M. (1990) *Psychobiology* **18**, 109-117.
38. Knowlton, B. J., Ramus, S. J. & Squire, L. R. (1992) *Psychol. Sci.* **3**, 172-179.
39. Nissen, M. J. & Bullemer, P. (1987) *Cognit. Psychol.* **19**, 1-32.
40. Daum, I., Channon, S. & Canavar, A. (1989) *J. Neurol. Neurosurg. Psychiat.* **52**, 47-51.
41. Weiskrantz, L. & Warrington, E. K. (1979) *Neuropsychologia* **17**, 187-194.
42. Glisky, E. L. & Schacter, D. L. (1988) *Neuropsychologia* **26**, 173-178.
43. Glisky, E. L. & Schacter, D. L. (1989) *Neuropsychologia* **27**, 107-120.
44. Reber, A. S. (1967) *J. Verb. Learn. Verb. Behav.* **6**, 855-863.
45. Reber, A. S. (1989) *J. Exp. Psychol. Gen.* **118**, 219-235.
46. Dulaney, D. E., Carlson, R. A. & Dewey, G. I. (1984) *J. Exp. Psychol. Gen.* **113**, 541-555.
47. Lewicki, P., Hill, T. & Czyzewska, M. (1992) *Am. Psychol.* **47**, 796-801.
48. Poppel, E., Held, R. & Frost, D. (1973) *Nature (London)* **243**, 2295-2296.
49. Weiskrantz, L., Warrington, E. K., Sanders, M. D. & Marshall, J. (1974) *Brain* **97**, 709-728.
50. Perenin, M. T. & Jeannerod, M. (1978) *Neuropsychologia* **16**, 1-13.
51. Weiskrantz, L. (1980) *Q. J. Exp. Psychol.* **32**, 365-386.
52. Torjussen, T. (1978) *Neuropsychologia* **16**, 15-21.
53. Zihl, J. (1980) *Neuropsychologia* **18**, 71-77.
54. Campion, J., Latt, R. & Smith, Y. M. (1983) *Behav. Brain Sci.* **6**, 423-486.
55. Weiskrantz, L. (1986) *Blindsight* (Clarendon, Oxford).
56. Goodale, M. A., Milner, A. D., Jakobson, L. S. & Carey, D. P. (1991) *Nature (London)* **349**, 154-156.
57. Bauer, R. M. (1984) *Neuropsychologia* **22**, 457-469.
58. Tranel, D. & Damasio, A. R. (1985) *Science* **228**, 1453-1454.
59. Young, A. W. & De Haan, E. H. F. (1992) in *The Neuropsychology of Consciousness*, eds. Milner, A. D. & Rugg, M. D. (Academic, San Diego), pp. 69-90.
60. Humphreys, G. W., Troscianko, T., Riddoch, M. J., Boucart, M., Donnelly, N. & Harding, G. F. A. (1992) in *The Neuropsychology of Consciousness*, eds. Milner, A. D. & Rugg, M. D. (Academic, San Diego), pp. 39-68.
61. Holender, D. (1986) *Behav. Brain Sci.* **9**, 1-23.
62. Marcel, A. J. (1983) *Cognit. Psychol.* **15**, 197-237.
63. Merikle, P. M. (1992) *Am. Psychol.* **47**, 792-795.
64. Cheesman, J. & Merikle, P. M. (1986) *Can. J. Psychol.* **40**, 343-367.
65. Milberg, W. & Blumstein, S. E. (1981) *Brain Lang.* **14**, 371-385.
66. Tyler, L. K. (1992) in *The Neuropsychology of Consciousness*, eds. Milner, A. D. & Rugg, M. D. (Academic, San Diego), pp. 159-178.
67. Shallice, T. & Saffran, E. M. (1986) *Cognit. Neuropsychol.* **3**, 429-458.
68. Schacter, D. L. (1989) in *Varieties of Memory and Consciousness*, eds. Roediger, H. L., III & Craik, F. I. M. (Erlbaum, Hillsdale, NJ), pp. 355-389.
69. Edelman, G. (1991) *Bright Air, Brilliant Fire: The Matter of Mind* (Basic Books, New York).
70. Schacter, D. L. (1992) *J. Cognit. Neurosci.* **4**, 244-256.
71. Schacter, D. L. (1990) *Ann. N.Y. Acad. Sci.* **608**, 543-571.
72. Squire, L. R., Zola-Morgan, M., Milner, A. D., Petersen, S. E., Videen, T. O. & Raichle, M. E. (1992) *Proc. Natl. Acad. Sci. USA* **89**, 1837-1841.
73. Cowey, A. & Stoerig, P. (1992) in *The Neuropsychology of Consciousness*, eds. Milner, A. D. & Rugg, M. D. (Academic, San Diego), pp. 11-37.
74. Wallace, M. A. & Farah, M. J. (1992) *J. Cognit. Neurosci.* **4**, 150-154.
75. Kihlstrom, J. (1987) *Science* **237**, 1445-1452.