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Models of Memory and the Understanding of Memory Disorders

Daniel L. Schacter, Alfred W. Kaszniak, and John F. Kihlstrom

University of Arizona
Tucson, Arizona

In 1882, Theodule Ribot published one of the now classic treatises of 19th century psychology, *Diseases of Memory*. In addition to describing and integrating a large number of clinical studies of memory problems, Ribot also argued forcefully that these observations should not be viewed merely as "a collection of amusing anecdotes" (1, p. 10). Instead, he contended that the phenomena encountered in cases of memory disorders are "regulated by certain laws which constitute the very basis of memory, and from which its mechanism is easily laid bare" (1, p. 10). Ribot went on to suggest various ways in which the study of memory deficits could provide important insights into the nature of normal memory processes. Unfortunately, as pointed out by Schacter and Tulving (2), subsequent studies of memory pathology made little or no impact on experimental and theoretical analyses of normal memory function for most of the 100 years following the publication of Ribot's work; likewise, clinical observations of memory deficits were relatively uninfluenced by the techniques and ideas of experimental psychology for much of the same period. Schacter and Tulving have delineated some of the negative consequences attributable to the gulf that separated the study of normal and abnormal memory for nearly a century.

During the 1970s, however, there were signs that the gap between the two research areas had begun to narrow, and on that basis Schacter and Tulving (2, p. 2) predicted the coming of a "golden age" characterized by much more extensive and fruitful interactions between students of normal and abnormal

memory than had existed previously. As Shimamura (3) points out, a number of developments during the past several years suggest that the "golden age" is upon us: empirical phenomena observed in memory-disordered patients have heavily influenced theorizing about normal memory; research concerning memory deficits has made extensive use of paradigms and theories from experimental psychology; and studies of memory-impaired populations have appeared with increasing regularity in the pages of mainstream experimental and cognitive journals.

In view of these encouraging recent developments, it seems appropriate and even necessary for a clinically oriented volume to include a chapter that focuses on the relation between models of normal memory and the understanding of memory disorders. The main purpose of our contribution is to summarize briefly some of the ways in which the analysis of memory disorders is currently being influenced by—as well as contributing to—contemporary thinking about normal memory. To accomplish this objective, we shall consider research concerning three different populations in which memory deficits are observed: amnesic patients, demented patients, and the normal elderly. Since it is not practically feasible to discuss all theoretical aspects of memory disorders in these populations, we limit ourselves to considering several distinctions between different *types* or *forms* of memory that have influenced thinking about memory disorders: the distinctions between primary and secondary memory (4,5), episodic and semantic memory (6,7), and implicit and explicit memory (8,9), respectively. The main reason for such a focus, as opposed to other sorts of models, is that they have been central to various debates and discussions about the nature of memory disorders in recent years. It should be noted, however, that a number of well-articulated and reasonably precise formal models of memory do exist (10-14). However, there has been little if any attempt to apply these models to clinical populations (for an exception, see Ref. 15). Although we think that formal modelling of memory disorders is a potentially valuable enterprise—and would even predict that this will constitute one of the next major theoretical trends—we restrict the present focus to ideas that have already played a role in shaping contemporary thinking about the nature of memory disorders.

THE AMNESIC SYNDROME

The amnesic syndrome is perhaps the most striking of all memory disorders, insofar as a profound inability to remember recent events occurs against a background of relatively intact cognitive, linguistic, and perceptual abilities (for review, see Refs. 16-18). Though observed as a consequence of various types of brain injury and disease, amnesia is usually attributable to damage in either the medial temporal and diencephalic brain regions (19,20). A good

deal of research and theorizing during the past two decades has focused on distinguishing between preserved and impaired memory processes in amnesic patients.

PRIMARY AND SECONDARY MEMORY

One of the great debates in experimental psychology during the 1960s concerned the distinction between short-term and long-term memory. According to the modal model (cf., Refs. 5,21), it is necessary to draw a sharp distinction between two different memory stores or systems: a *short-term store* that is characterized by limited capacity, exclusive reliance on acoustic codes, and extremely rapid decay; and a *long-term store* that is characterized by unlimited capacity, reliance on semantic codes, and a slower rate of forgetting. Although the modal model was worked out in impressive quantitative detail and received experimental support, serious conceptual and empirical problems with this view were delineated during the 1970s—most notably by Craik and Lockhart (22), resulting in what Crowder (23) called the demise of the concept of short-term memory. Despite their rejection of the modal model, however, even Craik and Lockhart recognized the need to preserve some sort of distinction between immediate and delayed retention. *Primary memory* refers to the processes that support immediate retention, whereas *secondary memory* refers to processes that support retention across delays (4).

The distinction between primary and secondary memory fits nicely with, and receives empirical support from, studies of amnesic patients. One of the most consistently observed features of the amnesic syndrome is that even the most profoundly amnesic patients exhibit normal immediate retention of various kinds of information, as assessed by such tasks as digit span (e.g., 24, 25). If primary memory is equated with immediate retention, there can be little doubt that amnesic patients possess intact primary memory. Moreover, the primary/secondary distinction also receives support from studies of patients who exhibit normal long-term retention together with severely impaired immediate memory (e.g., 26). Controversy has arisen, however, concerning the ability of amnesic patients to retain information across relatively brief delays (i.e., 3-30 seconds) under conditions in which rehearsal is prevented, as in the classic Brown-Peterson short-term forgetting paradigm. Although normal forgetting by amnesic patients in this paradigm has been observed (24,27), impaired performance has also been reported (e.g., 28,29). The reasons for these discrepant findings are still not entirely clear (for discussion, see Refs. 16,27,30). Nevertheless, they indicate that any global statements about amnesic patients' ability to remember information across brief delays must be regarded cautiously. We can conclude unequivocally that primary memory is intact in amnesic patients only so long as "primary memory" is identified with immediate retention.

The most extensively investigated theoretical account of primary memory is found in the *working memory* model developed by Baddeley and his colleagues (see Ref. 31). According to Baddeley, working memory consists of three main components: a limited-capacity *central executive* that is involved in selection and control functions; the *articulatory loop*, a "slave subsystem" of the central executive that allows for temporary storage of up to three items of speech-based information; and the *visuospatial scratchpad*, which provides temporary storage of nonverbal information. Baddeley's group has reported a variety of elegant experiments using dual-task methodology that have supported the working memory model by teasing apart and delineating properties of the various subsystems. The model has been applied successfully to some memory-disordered populations (31), but as yet has not been systematically evaluated with respect to the amnesic syndrome. Research within the working memory framework might help to clarify further the nature of primary memory abilities of amnesic patients.

EPISODIC AND SEMANTIC MEMORY

According to Tulving (6), episodic memory entails recollection of specific autobiographical events that are unique to an individual and are defined by particular spatial and temporal contexts, whereas semantic memory involves general knowledge of the world; facts, vocabulary, rules, and the like that is common to many individuals. Although Tulving (6) initially put forward the episodic/semantic distinction as a heuristic device, he later took the stronger position that episodic and semantic memory represent distinct and dissociable memory systems (7). A good deal of controversy still exists concerning this latter, theoretically based version of the distinction (cf., Refs. 32,33).

With respect to the amnesic syndrome, the episodic/semantic distinction appears initially to provide a compelling account of patients' preserved and impaired abilities (29). After all, one of the most striking features of amnesia is the coexistence of a severe inability to remember recent events (episodic memory) with a normal ability to retrieve general knowledge and vocabulary (semantic memory). However, as pointed out by Huppert and Piercy (34) and Zola-Morgan et al. (35), this pattern of performance can be equally well described as an impairment in new learning together with intact access to old, premorbid knowledge acquired long before the onset of amnesia; that is, the distinction between episodic and semantic memory is confounded with the distinction between new and old learning. Thus, the critical questions for an episodic/semantic account of amnesia concern the status of new semantic learning (which should be intact) and old episodic memories (which should be impaired).

Consider first the question of whether amnesic patients can acquire new semantic knowledge, as would be expected if the semantic memory system is entirely preserved. On one hand, it is clear that *some* acquisition of new semantic knowledge occurs in densely amnesic patients. Thus, for example, Kinsborne and Wood (29) reported that Korsakoff amnesics learned and retained a new mathematical rule despite their impaired episodic memory. Schacter, Harbluk, and McLachlan (36) found that an etiologically mixed group of amnesic patients retained some fictitious facts about familiar and unfamiliar people, despite their inability to remember when and where they acquired the facts (see also Ref. 37). Glisky, Schacter, and Tulving (38-40) demonstrated that head-injured and other amnesic patients could learn, and retain across delays of up to nine months, new computer-related vocabulary as well as various complex computer commands and programming rules; even though some patients had no recollection that they had ever worked on a computer (see also Refs. 41,42). These results, as well as other similar reports (cf., 43,44), lend support to the episodic/semantic account. On the other hand, however, amnesic patients' semantic learning in the foregoing studies was consistently and sometimes severely impaired relative to the performance of control subjects; moreover, failure to observe any new semantic learning in amnesia has also been reported (e.g., Ref. 45). These studies thus do not provide strong support for the existence of a spared semantic memory system (for further discussion, see Refs. 2,19,44,46-48).

Studies concerning the status of old, premorbid episodic memories are also somewhat equivocal. Kinsbourne and Wood (29) claimed that amnesic patients could not retrieve any memories of autobiographical incidents in response to word cues (49), in contrast to their normal ability to retrieve old vocabulary and factual knowledge. However, Zola-Morgan et al. (35) reported that amnesic patients were no more impaired in gaining access to old episodic than old semantic memories. In a single-case study, Butters and Cermak (50) reported deficits in access to both premorbid episodic and semantic memories, although the episodic deficit appeared to be rather more severe than the semantic deficit. Tulving et al. (51) described a patient who showed excellent retention of factual knowledge that was acquired at a particular job he had performed prior to the onset of amnesia, yet could not recollect a single incident that occurred during the entire period that he performed the job. The data thus suggest the possibility of an episodic/semantic dissociation within the domain of premorbid knowledge, but the overall picture is still somewhat muddy and a good deal more pertinent evidence needs to be collected. Part of the problem here is that the criteria for distinguishing between episodic and semantic memories are not always stated explicity (for discussion see Refs. 46,51,52), nor is it entirely straightforward to determine

what constitutes an "episodic" task and what constitutes a "semantic" task. These kinds of issues will have to be resolved in future attempts to evaluate the utility of the episodic/semantic distinction as an account of amnesia.

IMPLICIT AND EXPLICIT MEMORY

In traditional investigations of episodic memory, subjects initially study target materials and are then tested with recall and recognition tasks that require them to deliberately think back to the study episode and retrieve target information. During the past several years, however, experimental psychologists have assessed memory in a rather different way. Instead of instructing subjects to try to remember previously studied information, they are simply required to perform a task, such as completing a word fragment or identifying a word from a brief perceptual exposure; memory is inferred when task performance is facilitated by prior study of target materials. Graf and Schacter (8,9) used the term *explicit memory* to refer to conscious recollection of recent events on recall and recognition tests, and the term *implicit memory* to refer to facilitations of performance on completion, identification, and other such tests that do not require conscious or intentional recollection of a specific prior episode. Graf and Schacter emphasized that the implicit/explicit dichotomy is a descriptive distinction that does not imply the existence of two separate systems underlying implicit and explicit memory, respectively (for further discussion of definitional and conceptual issues surrounding the implicit/explicit distinction, see Refs. 9,53,54).

The major reason for advancing an implicit/explicit distinction stems from empirical observations of dissociations between performance of recall and recognition tests on the one hand, and completion, identification, and similar tasks on the other. Studies of normal subjects have revealed that a number of experimental variables, including level and type of study processing, retention interval, and study/test modality shifts, have different and even opposite effects on tasks that tap implicit and explicit memory (e.g., 9,55-62). Equally importantly, neuropsychological investigations have shown that amnesic patients show intact performance on various implicit memory tests that do not require conscious recollection of a previous episode. A number of studies have shown that amnesic patients can acquire various kinds of perceptual/motor skills in a normal or near normal manner, despite their inability to remember explicitly the episodes in which they acquired the skills (e.g., 63-66). It has also been established that amnesic patients show normal priming effects on such implicit memory tasks as word completion (67,68), free association (69,70), and category instance production (71,72), as well as various other implicit tests (for review, see 9,44,73).

A number of theoretical proposals have been put forward to account for dissociations between implicit and explicit memory in amnesia. It has been suggested, for example, that intact perceptual and motor skill learning can be attributed to a spared *procedural* memory system that entails on-line modification of processing operations, and that is distinct from a *declarative* system that represents the outcomes of particular processing operations (e.g., 19,74). With respect to priming effects, some investigators have argued that amnesics' intact performance can be attributed to an automatic and temporary activation of pre-existing semantic memory representations (e.g., 46,47, 67,75), whereas others have suggested that priming may reflect the influence of newly created episodic representations that are inaccessible to conscious remembering (e.g., 8,76-78). A related proposal has been put forward recently by Schacter (79), who suggested that many implicit memory phenomena in normal and amnesic subjects can be attributed to the activity of *perceptual representation systems*—processors that represent domain-specific information about the form and structure of words and objects (cf., 80,81), but do not store and retrieve the kinds of information that are necessary for explicit remembering of episodes. Perceptual representation systems are typically unimpaired in amnesic patients, and thus could underly at least some of the implicit memory phenomena that have been observed (see Schacter, 79, for further discussion).

Although the present chapter does not allow us to explore fully the complex issues surrounding implicit/explicit dissociations in amnesic patients, it should be emphasized that this is one area of investigation in which studies of normal and abnormal memory have been, and will likely continue to be, tightly linked to one another. Indeed, the implicit/explicit distinction (unlike the primary/secondary and episodic/semantic distinctions) was directly motivated by empirical studies of amnesic patients.

MEMORY AND DEMENTIA

According to DSMIII-R, impairment of memory is an essential feature of dementia (82). During the past 15 years, there has been increasing research interest in the nature of memory deficit in various dementing illnesses (e.g., Alzheimer's disease, Huntington's disease). The largest body of this research has focused upon Alzheimer's disease (AD), the most prevalent cause of dementia among older adults (for comprehensive reviews, see Refs. 83-85). Longitudinal psychometric studies of AD patients (86,87) have supported clinical impressions that memory is impaired very early in the course of AD, and deteriorates progressively. We will limit our present discussion to those studies of AD patients relevant to the distinctions between primary/secondary,

episodic/semantic, and implicit/explicit memory. It should be noted that conclusions drawn from these studies may not generalize to other dementing illnesses, such as Huntington's disease (e.g., 88).

PRIMARY AND SECONDARY MEMORY

In contrast to amnesic syndrome patients, AD patients show impairment of primary memory, as reflected in impairment on digit, word, and block span tasks, the Brown-Peterson short-term forgetting paradigm, and the recency component of the serial-position curve in list-learning tasks (for review see Refs. 89,90). Although digit span may be normal or only minimally reduced early in the course of AD, it becomes clearly compromised as the disease progresses (86,91). Measures thought to reflect secondary memory, in comparison with those of primary memory, show more severe impairment throughout the course of AD (see Ref. 89).

There have been several attempts to account theoretically for the primary memory deficit of AD patients. Wilson et al. (92) employed a verbal free recall paradigm, using the scoring method of Tulving and Colotla (93) to define primary and secondary memory components. In this scoring method, items recalled with less than seven items between presentation and recall are identified as representing primary memory, and the rest as secondary memory. AD patients, relative to matched healthy controls, showed primary memory impairment, with the size of this impairment increasing linearly with greater numbers of items between presentation and recall. The secondary memory score showed an even greater difference between the groups. Further, although the primary and secondary memory scores were independent in the healthy controls, they were significantly correlated in the AD patients. Finally, Wilson et al. observed a lack of proactive interference effects for the AD patient group, as indicated by no decline in free recall across four consecutive list presentations and by fewer prior list item intrusions than healthy controls (cf. Ref. 94). On the basis of these observations, Wilson et al. (92) proposed that both the primary and secondary memory deficits of AD patients are at least partially the result of initial processing and encoding failure, perhaps reflecting attentional deficit. Martin et al. (95) have suggested a similar explanation of primary and secondary memory deficits in AD.

More recently, Morris and Baddeley (90), using Baddeley's (31) working memory model as a theoretical framework, have argued for impairment in central executive control processes as a cause of the primary memory deficit in AD. In contrast to Wilson et al. (92), Morris and Baddeley propose that this central executive impairment has its major effect on the manipulation and maintenance of information, rather than on its initial encoding. This conclusion is based upon several lines of evidence. First, the documented reduc-

tion of digit span in AD does not appear due to impairment in the articulatory loop system. Two subsystems are hypothesized to comprise the articulatory loop system, a phonological store and an articulatory rehearsal mechanism (31). Integrity of the phonological store in AD is inferred from the observation that phonological similarity reduces memory span for letters to the same extent in AD patients as in normal subjects (96), despite the AD patients' moderate overall reduction in span. Integrity of the articulatory rehearsal mechanism in AD is inferred from demonstrations of a normal effect of word length (longer words are presumed to take longer to be recycled through the articulatory loop, leading to slower and less effective rehearsal), a normal rate of articulating a random list of visually presented digits (and hence presumably of subvocal rehearsal), and normal suppression of word and letter memory span by concurrent articulation of irrelevant material (96-99). Second, impairment in the central executive component of working memory is inferred from demonstrations of disproportionate AD patient impairment in performance of various dual tasks (97,100).

Wilson et al. (92), on the basis of their observation that primary and secondary memory scores were independent in healthy controls but were significantly correlated in the AD patients, suggested that the secondary memory impairment of AD may be at least partially attributable to their primary memory deficit. This raises the question of whether all of the memory impairment of AD might be due to a single factor (e.g., impairment of the central executive component of working memory). Recently, Becker (101) presented evidence in support of dissociable contributions of both working memory and secondary memory deficits in AD. The performance of AD patients on tests related to working memory/central executive dysfunction could be statistically dissociated from that on tests related to secondary memory. Further, Becker described individual AD cases for whom the difference between scores on tasks related to these two memory domains was large and in different directions.

In summary, recent research has provided evidence for dissociable contributions of both primary and secondary memory deficit to the progressive memory impairment of AD. Further, studies of AD patients that have employed the working memory model have provided support for the dissociability of hypothesized components of this model. Becker (101) suggests that the secondary memory deficit of AD may be due to the perihippocampal damage that serves to functionally disconnect much of hippocampus and cortex (102,103). Becker further suggests that impairment of the central executive component of working memory may be attributable to pathology of the frontal lobes or their afferent connections in AD (104). An important direction for future research will involve the testing of such hypotheses, particu-

larly through the use of concurrent neuropsychological and regional brain metabolic measures within longitudinal research designs (e.g., 105).

EPISODIC AND SEMANTIC MEMORY

Much research has shown mildly demented AD patients to be comparable to amnesic syndrome (e.g., Korsakoff's) patients in their impairment on episodic memory tasks, such as recall of text passages (e.g., 106). However, unlike amnesic patients, AD patients also show impairment in the recall of previously acquired semantic knowledge. For example, on confrontation naming tasks, AD patients are impaired relative to healthy controls, with this impairment accounted for mostly by semantic errors (e.g., 107). Further, ability to retrieve items from within a given semantic category (e.g., animals) is progressively impaired in AD (87). AD patients produce fewer correct responses than do healthy controls, from fewer subcategories, and produce fewer responses per category (108,109). AD patients also are impaired in their memory for remote public events and public figures (110,111), information that is likely represented as semantic rather than episodic knowledge (46).

Several investigations have provided evidence consistent with the interpretation that semantic memory deficit contributes to the episodic memory impairment of AD. Inadequate semantic encoding of information has been suggested by such observations as the failure of AD patients to show the expected rare word advantage in verbal recognition memory (112), or to benefit from procedures designed to enhance elaborative semantic processing (113,114) and facilitate semantic organization (115) in episodic memory tasks. Significant intercorrelations have been found between episodic (e.g., free recall, selective reminding) and semantic (e.g., category generative naming) memory tasks for AD patients, but not for Korsakoff's amnesics, although both patient groups showed equally profound episodic memory impairment (116).

Although there is consensus that AD patients are impaired on tasks requiring retrieval from semantic memory, there is disagreement concerning the question of whether AD patients have an impairment in the representational structure of semantic memory (see Ref. 117 for a discussion of semantic memory models), or only in those processes necessary for its access (for review see Refs. 83,118). It has been argued (e.g., 119) that tasks such as category generative naming place heavy demands upon effortful processing, and that questions about the structural integrity of semantic memory in AD are more appropriately addressed by tasks involving automatic, implicit activation of lexical or semantic memory. Studies employing lexical and semantic priming paradigms will be discussed in the following section. Even among those studies relying upon explicit tasks, there remains disagreement. For example, some investigators (e.g., 108,120,121) have concluded that general categorical in-

formation (e.g., item membership in a superordinate semantic category) remains intact early in the course of AD, while ability to differentiate among items or attributes within a semantic category is impaired. These conclusions have been inferred on the basis of patterns of confrontation and generative naming errors, as well as AD patients' ability to select objects belonging to a specified functional category. Other investigators (122,123) have concluded that representation of the semantic attributes of concepts is intact in AD, based upon the performance of tasks requiring patients to determine whether various attributes (e.g., physical features, functions, actions) were related to a given concept.

In summary, there is general agreement that AD patients are impaired in their performance on tasks requiring episodic memory for recent experience, as well as on tasks requiring explicit retrieval of previously acquired semantic knowledge. Further, in AD patients, semantic memory deficit appears to contribute to the severity of their episodic memory impairment. Disagreement remains, however, concerning the question of whether the representational structure of semantic memory is disturbed in AD, or only those processes necessary for explicitly accessing semantic memory are impaired. An important task for future research is to resolve this controversy. It is possible that differences in dementia severity, or in AD patient sample heterogeneity, might contribute to apparently contradictory findings. Impairment of performance on semantic memory tasks is progressive over the course of AD, and various semantic memory tasks do not reveal equivalent impairment across levels of dementia severity (87,107). Further, the existence of AD patients with unusually severe linguistic/semantic deficits (and hemispherically asymmetric cerebral hypometabolism) early in their disease course have been documented (124,125). The presence of such "linguistic/semantic deficit" AD patients may contribute to variability in the results of prior studies, as they have been shown to demonstrate preserved semantic knowledge on a superordinate and category level but not at the level of object attributes (126).

Another possible contributor to the variability of conclusions within this literature may lie in the demands of the experimental tasks. As Nebes and Brady (123) point out, patients in the Martin and Fedio (108) study were asked explicit questions concerning properties of pictured objects (e.g., Is it used for cutting?), and thus had to search the semantic fields of concepts for particular attributes. In contrast, both Nebes and Brady, and Grober, et al. (122), AD patients were required to indicate whether given attributes were related to particular concepts, and thus had only to recognize that some association exists between the concept and attribute. It remains for future studies to contrast such differing experimental approaches within the same patient sample.

EXPLICIT AND IMPLICIT MEMORY

All of the research involving AD patients described above have used explict memory tasks, and have documented marked impairment for most tasks. As already mentioned, another approach to the question of whether the structure of semantic memory is impaired in AD has been through the use of implicit memory tasks. Unfortunately, this approach has also failed to settle the controversy. Several investigators (119,127,128) have used semantic priming and semantic category decision tasks in studies of AD patients. They have concluded that the network of associations existing between semantic concepts and attributes remains intact in AD, provided that the patient's use and retrieval of this information is guided by the stimulus context. Other investigators (129-131) have used lexical decision, word-stem completion, and word-association priming tasks. These investigators have found AD patients impaired on these implicit memory tasks, and concluded that conceptual relationships within semantic memory are disrupted. At present, the reasons for these discrepancies between studies are unclear. As with the studies reviewed in the episodic and semantic memory section above, possibilities would appear to include both sample differences as well as differences in experimental methodology, and remain to be determined in future studies.

Studies investigating other implicit memory phenomena in AD patients have also recently begun to appear. Relatively intact motor-skill learning (132,133) has been demonstrated in AD patients. Implicit learning of a repeating sequence of digits (as indicated by a serial reaction time task) was also found for many, although not all, AD patients studied by Knopman and Nissen (134). Those patients who failed to show implicit learning of the sequence were similar to learners in age and overall dementia severity, but scored lower on some tasks of nonverbal reasoning.

In summary, it appears that at least some AD patients are able to demonstrate relatively intact performance on certain implicit memory tasks. Future research will need to clarify the characteristics of both AD patients and tasks that result in preserved implicit memory performance. Such research is of both theoretical and practical importance, since the potential exists for designing interventions and management strategies based upon preserved domains of implicit learning, as has already been done with amnesic patients (e.g., 41,42).

MEMORY AND NORMAL AGING

In addition to the memory loss associated with Alzheimer's disease and other dementias, even normally healthy elders claim to have difficulty learning new information and remembering recent events. Although some complaints

about memory function may be related to depression, objective psychometric studies do indicate considerable but selective age-related impairments in memory function (for representative comprehensive reviews see Refs. 135-147). The selectivity of these impairments can be organized in terms of the three heuristic distinctions among forms of memory considered in the preceding sections.

PRIMARY AND SECONDARY MEMORY

There is almost universal agreement that normal aging has little or no deleterious effect on the operation of primary memory, or on the sensory information stores that hold information at a very early stage of processing (143, 148). For example, there are minimal differences in forward digit span, rate of forgetting in the Brown-Peterson paradigm, or the recency component of the serial-position curve. There are age effects on backwards digit span, however. This finding suggests an age-related deficit in working memory, in which the subject must actively manipulate and transform the material (140), presumably reflecting an underlying age-related difficulty with the controlled deployment of attention. By contrast, there is overwhelming evidence of age-related impairments in secondary memory (e.g., 136,137). Prima facie evidence for a specific age-related deficit in long-term memory comes from differences between young and old in single-trial free recall, and particularly in the primacy portion of the serial-position curve. The extent of the age deficit depends, of course, on the manner in which secondary memory is assessed. The modal finding in the literature is that the aged perform least well on tasks involving free recall, and best on tasks involving recognition (147). For example, Craik and McDowd (149) engaged subjects in a concurrent reaction-time task during tests of cued recall and recognition. They found no age-related differences in recognition (as measured by the signal-detection measure d'), but a substantial deficit in cued recall.

EPISODIC AND SEMANTIC MEMORY

Primary and secondary memory are both reflections of episodic memory, in that they tap the ability of the person to remember, after shorter or longer intervals of time, events that occurred in a specific spatiotemporal context. Thus, the aged clearly show an impairment in episodic memory, especially over long retention intervals and when retrieval cues are relatively impoverished; that is, free recall from secondary memory. By contrast, most evidence indicates that context-free semantic memory remains relatively intact in the healthy aged. For example, it has long been known that performance on "crystallized intelligence" tests involving vocabulary and general information—which might be called semantic memory in its purest form—shows rela-

tively little decrement, and may actually *increase* with age—presumably because age provides more opportunities to acquire this sort of information (143,150,151). Similarly, young and old subjects show the same magnitude of priming effect in a category verification task (139). However, response latencies in such tasks, as well as word fluency in general, do decrease with age; again perhaps as a result of a general age-related slowing of cognitive functions.

As indicated earlier, however, a clear distinction must be made between the type of memory, episodic or semantic, and the age of the memory, distant or recent. Most tests of semantic memory involve information learned while the subject was young, while most tests of episodic memory involve events that occurred quite recently. There is surprisingly little research available on the comparative abilities of young and old subjects to acquire wholly new vocabulary or world knowledge. On the other hand, there is fairly good evidence that the elderly have difficulty retrieving both remote and recent personal recollections (152,153). Unfortunately, in these studies the age of the subject is confounded with retention interval. Thus, when asked to recognize high-school classmates, 70 year olds tend to do worse than 50 or 30 year olds. But it should be noted that the 70 year olds are being asked to retrieve memories from 55 years ago, while 50 year olds are being asked to retrieve memories that are only 35 years old. At present, we do not know whether the elderly are more forgetful of remote memories when the retention interval has been held constant.

EXPLICIT AND IMPLICIT MEMORY

Research comparing explicit and implicit memory in the elderly is at a very early stage, but there is already some evidence that implicit memory is relatively spared among the normal aged. For example, Light et al. (154) asked old and young subjects to study a list of target words, followed by an explicit test of yes/no recognition and an implicit test of word fragment completion. Elderly subjects showed poor recognition accuracy compared with younger subjects, especially after one week; however, there were no significant age-related effects on word fragment completion. Similar results have been obtained by Light and Singh (155), and by others using a variety of paradigms (e.g., 156,157).

Because explicit recollection is mediated by retrieval of the context in which the target event occurred, the dissociation between explicit and implicit memory observed in older adults suggests that contextual information may be relatively vulnerable to encoding and/or retrieval difficulties. In fact, the available evidence indicates that the elderly show impairments in processing at least three forms of contextual information: temporal context, spatial

context, and external source of information (e.g., 136). For example, the elderly appear to be disadvantaged in remembering both the particular list in which an item was presented (e.g., 158), and the spatial location in which list items (both verbal and pictorial) are presented (e.g., 159,160), even when the items themselves are correctly recognized as belonging to a previously presented list, and even under intentional study conditions. With respect to source, the elderly have difficulty remembering the gender of the voice in which list items had been read (161), which of two experimenters provided them with new factual information (162), or whether a word had been presented visually or orally (163). Interestingly, however, the elderly appear to have little or no difficulty distinguishing between externally and internally generated list items (e.g., 163).

CONCLUDING COMMENTS

Although there was a time when theorizing about normal memory function and studying clinical memory disorders were independent enterprises, even our rather brief consideration of the literature confirms that this is clearly no longer the case. Ideas developed in the study of normal memory have become an almost ubiquitous component of clinical investigations concerning memory disorders observed in amnesia, dementia, and normal aging. The time when studies of memory disorders consisted solely of administering a theoretical test batteries or clincial protocols appears to be behind us. This development bodes well for both the clinical study of memory impairments, and the experimental study of normal memory. We have little doubt that if he were alive today, Theodule Ribot would have warmly applauded the emergence of the kind of studies that he had called for over a century ago.

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Edited by

Takehiko Yanagihara

Ronald C. Petersen

Mayo Clinic and Mayo Foundation
Rochester, Minnesota

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