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Psychological Time and Memory Systems of the Brain

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Abstract Psychological time involves four partially dissociable memory systems of the brain. Procedural memory, subserved mainly by the cerebellum, controls timing of learned movements. Semantic memory, subserved by several areas of the cerebral cortex, concerns linguistic and conceptual information, presumably including general knowledge concerning time. Working memory, which involves the functioning of the dorsolateral prefrontal cortex, maintains a representation of current temporal contextual information. Episodic memory, which requires the functioning of the hippocampus and other medial temporal lobe structures, is necessary to encode long-term memory for personal experiences, including temporal information about them.

People continually engage in one or more of several different kinds of temporal activities, including controlling movement timing, expressing general temporal knowledge, representing present events, and remembering past durations (Block, 1979, 1990a). Tulving (1972, 1985, 1991) distinguished several memory systems, each differing in "its brain mechanisms, [the] type of information it handles, and the principles of its operations" (Tulving, 1991, p. 10). At least four partially dissociable memory systems are involved to greater or lesser extents in different temporal experiences, judgments, and behaviors.[1] These memory systems are: (1)

[1] I will not discuss the sensory processes involved in experiencing and judging simultaneity and successiveness of stimuli presented for brief durations and at very short intervals (Block and Patterson, in press; Carr, 1993). These processes may involve a fifth memory system, which Tulving (1991) called the *perceptual representation system.*

procedural memory, which contains information vital to the performance of learned movements and relatively automatic procedures; (2) semantic memory, which processes information about concepts, facts, linguistic expressions, and so on; (3) working, or short-term, memory, which contains highly accessible information about present (or very recently past) events; and (4) episodic memory, which contains information about past personal experiences, including recency, order, and duration information.

Evidence from neuropsychological, psychopharmacological, and cognitive neuroscience research suggests that anatomically and functionally separate, yet interconnected, brain areas subserve the functioning of these different memory systems that are involved in time-related tasks. Figure 5.1 shows the location of some brain areas or structures that are critically involved in psychological time. Some of the factors that influence different temporal functions (Block, 1989) may reflect the workings of neural networks, or information-processing modules, in these and other anatomically related areas. Each brain structure normally interacts directly with several other structures and indirectly with the rest of the brain. Although the present review does not describe these multiple interconnections, the interested reader may find neuroanatomical details in several sources (Kolb and Wishaw, 1990; Kandel, Schwartz, and Jessell, 1991).

The present article reviews evidence and theories on these memory systems and the brain structures that are critically involved in them. (Other recent reviews that focus on many of the issues described herein include those by Weiskrantz [1987], Melges [1989], and Kosslyn [1992].) I present a particular view on several unresolved issues, but I also discuss lingering theoretical disagreements. The review focuses mainly on evidence from studies of brain damage, neurotransmitters and drug effects, and physiological recording. In interpreting the findings from any source of evidence concerning brain function, caution must be exercised. Localized damage to a particular brain region or structure may not influence performance on a particular task, or it may produce impaired performance. This kind of evidence suggests that the damaged region or structure either does or does not participate in the interacting network of brain activity underlying task performance. Because the brain functions as a system containing highly interconnected structures, or modules, damage to any particular region or structure can also affect functioning of other regions or structures. If damage to a particular structure leads to impaired performance on a task, it may only be concluded that the structure is a critical component of the information processing required for task performance. If damage to a structure does not lead to impaired performance, then either the structure is not normally involved in performance of that particular task or some other brain structures can be deployed to perform the task. Similar caution must be exercised in interpreting studies of neurotransmitters and drug effects. A particular brain region normally contains several neurotransmitters, each of which is also distributed throughout various interconnecting brain regions. Most physiological recording techniques have a more basic source of limitation. For technical reasons that need not concern us here, these methods all suffer from limitations in ability to localize events, either

in brain space (e.g., event-related potentials) or in time (e.g., positron emission tomography [PET]).

Although one may legitimately question any particular source of evidence, using any particular methodology, converging evidence from studies using different methodologies suggests a reasonably coherent account. The present account discusses certain brain regions and structures that are most critically involved in psychological time. Future studies will need to specify more precisely the exact information processing that occurs in each region or structure in order to subserve performance of psychological functions related to time or timing.

Procedural Memory

In some temporal tasks, such as those in which a person must control the sequencing and production of movements, the supplementary motor area, premotor cortex, primary motor cortex, and related cortical areas subserve the planning and control of movement timing (Ghez, 1991b; Kosslyn, 1992; Vidal, Bonnet, and Macar, 1992). It appears that as movements become more well learned, control of movement sequencing and timing shifts to the cerebellum (see Figure 5.1). In particular, the lateral portion of the cerebellum (the so-called *cerebrocerebellum)* subserves procedural memory for timing relative intervals in highly automatized movement sequences. At present, it is unclear whether the cerebellum stores these procedural memories or whether other structures that are intimately connected with the cerebellum store the information (Leiner, Leiner, and Dow, 1991). The lateral cerebellum may then transmit the information on movement sequencing and timing to the premotor and motor cortical areas for execution. In addition, the spinocerebellum (i.e., the intermediate cerebellar hemisphere) controls some movements, and it also apparently corrects or adjusts movement sequences in progress (for a review, see Ghez, [1991a]). The extent to which the cerebral system or the cerebellar system primarily controls movement sequence timing may depend on the timing requirements of the task and the degree of automaticity of the motor program involved.

Evidence that the lateral cerebellum plays a critical role in movement timing which is dissociable from the role of the semantic, working, and episodic memory systems comes mostly from studies of brain damage. Lateral cerebellar damage does not affect other memory functions, but it produces deficits in timing movement execution and perhaps also in performing temporally predictive computations in other behavioral, perceptual, and cognitive situations (Ivry, Keele, and Diener, 1988; Ivry and Keele, 1989; Leiner et al., 1991). In contrast, damage to specialized cerebral structures that produce various types of amnesia does not impair procedural memory functions such as movement timing (Mayes, 1988).

Semantic Memory

Several areas of both cerebral cortices, but especially of the left hemisphere, subserve language. These include two classical areas: Broca's area, which is located
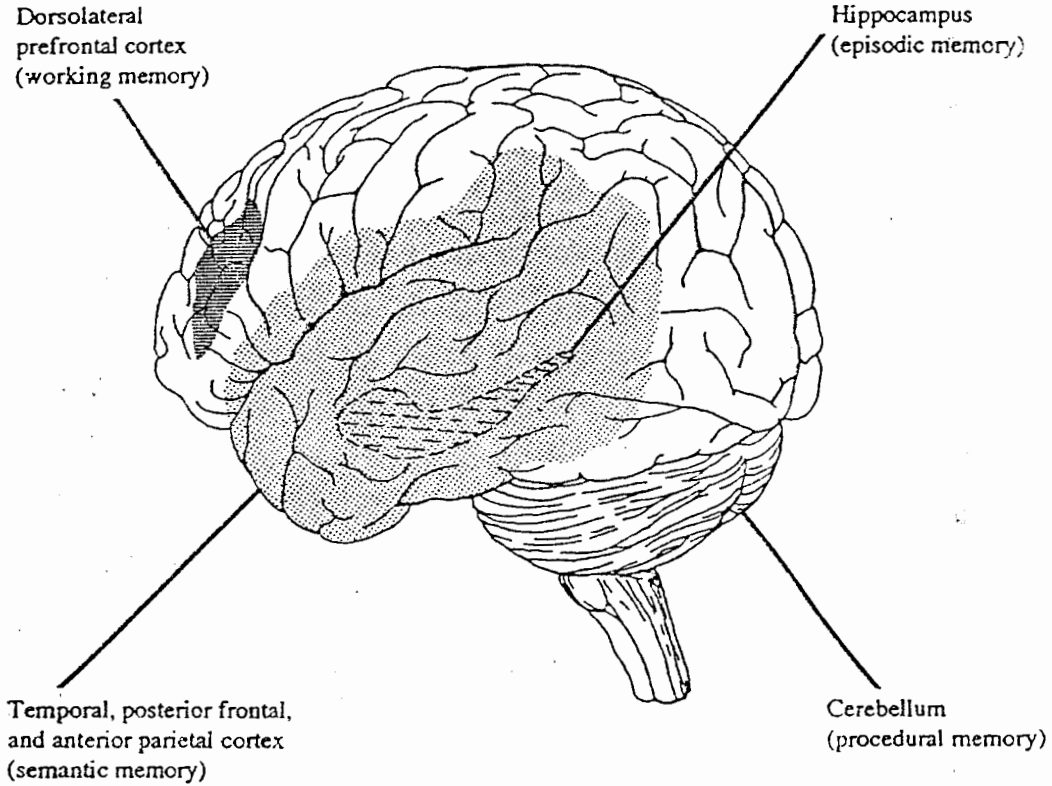
Dorsolateral
prefrontal cortex
(working memory)

Hippocampus
(episodic memory)

Temporal, posterior frontal,
and anterior parietal cortex
(semantic memory)

Cerebellum
(procedural memory)

Figure 5.1. Lateral view of the human brain (left hemisphere) showing the approximate location of several areas that subserve aspects of psychological time. See text for further explanation, and see other treatments (e.g., Kandel, Schwartz, and Jessell, 1991; Kolb and Wishaw, 1990) for additional neuroanatomical details. Clockwise from lower right: (1) The cerebellum, which is involved in procedural memory, is a complex subcortical structure. (2) Widespread areas of the temporal cortex, posterior inferior frontal cortex, and anterior inferior partietal cortex, especially of the left hemisphere, subserve semantic memory; corresponding areas of the right hemisphere are apparently not as heavily involved in semantic memory. (3) The dorsolateral prefrontal cortex of both cerebral hemispheres subserves working memory. (4) The hippocampus, which along with nearby structures is critically involved in episodic memory, is a subcortical structure underlying the temporal cortex of the left and right hemispheres in the approximate location depicted here. Only the left-hemisphere half of the hippocampus is depicted.

in the left inferior frontal lobe and is involved in linguistic production; and Wernicke's area, which is located in the left superior posterior temporal lobe and is involved in linguistic comprehension. It is now clear that many nearby cortical areas subserve various aspects of semantic memory, including widespread areas of the temporal, parietal, and frontal cortices (see Figure 5.1).

Studies of people with brain damage suggest that different memory systems may be selectively impaired, producing different kinds of amnesia (Mayes, 1988). I will call two main kinds *semantic amnesia* and *episodic amnesia* (Nielsen, 1958). Damage to any of several regions in the left cerebral hemisphere typically produces

semantic amnesia, which may be quite specific (Weiskrantz, 1987). For example, a man known as M.D. suffered cerebral damage as a result of stroke (Hart, Berndt, and Caramazza, 1985). Afterwards, he had considerable difficulty naming fruits and vegetables, such as a visually presented apple. He could easily name other kinds of objects, however, and he could categorize pictures of fruits and vegetables that he could not name. Studies of other patients reveal that parts of both cerebral hemispheres subserve linguistic and conceptual knowledge in other domains, such as about color (Damasio, Damasio, Tranel, and Brandt, 1990; Damasio and Damasio, 1992).

Studies using PET scanning reveal that when normal people perform certain semantic-processing tasks, neural activity increases in the posterior left-hemisphere frontal lobe (Posner, Petersen, Fox, and Raichle, 1988). However, Tulving (1989) reported preliminary evidence suggesting that regional cerebral blood flow (another presumed indicator of neural activity) is relatively greater in posterior regions of the cerebral cortex during retrieval of impersonal semantic information and is relatively greater in anterior regions during recollection of personally experienced episodes. The apparent discrepancy between these two sets of findings may be attributable to task differences, especially since several brain regions apparently subserve different aspects of semantic memory.

As involved in psychological time, the semantic system contains information about temporal units, concepts, and linguistic expressions. For example, people know that time seems to pass slowly during a boring experience (Block, Saggau, and Nickol, 1983–1984). Semantic memory also enables a person to use temporal metaphors, such as "the river of time" (Jackson and Michon, 1992; Michon, 1990). The semantic system, along with the working and episodic memory systems, also apparently mediates temporal perspective, or one's conceptualization of past, present, and future (Williams, Medwedeff, and Haban, 1989; see below). Research has not yet identified the specific cortical areas and processes that subserve linguistic and conceptual knowledge about time, but they are probably similar to those involved in other domains of knowledge such as color.

Working Memory

Evidence on the working memory system and its role in psychological time comes from several sources. Here I discuss mainly studies of brain damage, neurotransmitters and drug effects, and electrophysiological recording.

Brain Damage

Patients with frontal lobe damage usually show little or no impairment in remembering that a particular event occurred. Those with damage in the more anterior areas of the frontal lobe, the prefrontal cortex, however, have difficulty performing tasks

that require more explicit use of temporal information. For example, Milner and her colleagues found that these patients show serious impairment in judging which of two remembered events occurred more recently (Milner, 1971, 1974, 1982; Petrides and Milner, 1982; Milner, McAndrews, and Leonard, 1990). Their comparative studies of several patients with damage to various areas of the prefrontal cortex suggests that this impairment of temporal memory occurs mainly if there is damage to the dorsolateral prefrontal cortex, specifically in and around Broadmann's area 46 (see Figure 5.1).

Milner and her colleagues also found that encoding external temporal-order information more heavily involves the right prefrontal cortex, whereas encoding internal temporal-order information more heavily involves the left prefrontal cortex (see Milner, 1982). More recently, McAndrews and Milner (1991) found that frontal-lobe patients show normal accuracy in judging the relative recency of two objects that they actively manipulated. One possibility is that the procedural-memory system encodes temporal order information for actions, and if the prefrontal cortex is damaged this information is sufficient to mediate memory for temporal order of specific actions.

Damage in the anterior regions of the left hemisphere (but not of the right hemisphere) frontal cortex also leads to impaired planning in novel situations (Luria, 1966; Milner, 1982; Shallice, 1982, 1988). Damage in these regions does not impair the execution of well-learned, routine plans, perhaps because this function is subserved by the cerebellum rather than by the prefrontal cortex.

Neurotransmitters and Drug Effects

The neurotransmitter dopamine is found throughout the prefrontal cortex, and a growing body of evidence suggests that one type of dopamine receptor site (D1) plays a critical role in the efficiency of working memory (Sawaguchi and Goldman-Rakic, 1991; Goldman-Rakic, 1992). Some drugs that influence prospective temporal judgments may do so because they influence D1 dopamine receptors in the prefrontal cortex. Dopamine agonists tend to lengthen prospective duration experience (i.e., they increase the subjective time, rate), whereas dopamine antagonists tend to shorten prospective duration experience (Hicks, 1992).

Schizophrenics show various abnormalities in temporal judgment (for reviews, see Johnston [1960], Mo [1990]). Researchers increasingly view psychopathologies like schizophrenia as biological disturbances of specific neurotransmitter systems. Cohen and Servan-Schreiber (1992) reviewed evidence suggesting that schizophrenia involves a reduction of dopamine effects in the prefrontal cortex, resulting in an inability to form and maintain contextual representations. Weinberger (1987) suggested that schizophrenia involves two separate disturbances in dopaminergic pathways, increased activity in the mesolimbic dopaminergic system and decreased activity in the prefrontal cortex. The precise nature of the deficit in schizophrenia remains unclear, but it apparently involves dopaminergic transmission and the prefrontal cortex.

Electrophysiological Recording

The first electrophysiological evidence that temporal integration of behavior involves the prefrontal cortex arose from the discovery of the contigent–negative variation (CNV) (Walter, Cooper, Aldridge, McCallum, and Winter, 1964). The CNV is a relatively slow surface-negative electrical potential recorded by scalp electrodes. It occurs in the interval between two successive events, beginning about 300 ms after the presentation of the first. It appears if a person anticipates attending to a significant event, such as a temporal interval, a stimulus, or perhaps a movement (Macar and Vitton, 1979). Contingent-negative variation is prominent over the prefrontal cortex, in which it may originate (Borda, 1970; Loveless and Sanford, 1974; Rohrbaugh, Syndulko, and Lindsley, 1976). In duration–judgment experiments, CNV appears when one stimulus starts a duration and another stimulus ends it. Other evidence suggests that CNV amplitude is directly related to prospective duration judgments (McAdam, 1966). If subjects know that they must estimate duration, and perhaps in other situations as well, "CNV may be an index of a phase of information processing aimed at constituting internal time bases congruent with a particular situation" (Macar and Vitton, 1979, p. 226). Stated somewhat differently, CNV may be "an electrophysiological concomitant of attention to time" (Hicks, Gualtieri, Mayo, and Perez-Reyes, 1984, p. 235). Drugs such as barbiturates that retard activity in the prefrontal cortex decrease both the CNV amplitude and prospective temporal judgments (Hicks, 1992; Hicks et al., 1984); it is possible that they do so specifically because they affect the prefrontal cortex.

Single-neuron recording in the dorsolateral prefrontal cortex reveals that some neurons remain active between the time a stimulus disappears and the time a response is allowed (Kubota and Niki, 1971; Fuster, 1973, 1980, 1985a,b; Niki, 1974; Goldman-Rakic, 1987; Goldman-Rakic, Funahashi, and Bruce, 1990). Because these neurons show sustained activity over short time periods, they could possibly serve as internal clocks, or short-duration timers. Cognitive psychological evidence also suggests that the working memory system subserves short-term memory for an event, on the scale of several seconds (Baddeley, 1986). Goldman-Rakic (1987) proposed that the prefrontal cortex contains several working memory centers, each dedicated to a different information-processing domain. Attending to time may involve one or more of these centers. Contingent-negative variation may therefore frequently reflect the activity of attending to time, subserved in part by the dorsolateral prefrontal cortex.

Working Memory and Psychological Time: Conclusions

Psychological time, especially constructing and maintaining a temporally and contextually defined present, critically depends on the dorsolateral prefrontal cortex. This area apparently processes temporal contextual information, thereby enabling a person to remember the order of recent events and to prepare or plan for future

events (Fuster, 1984). In other words, the prefrontal cortex is a critical component involved in strategic and organizational control of behavior across time, which is why Moscovitch and Winocur (1992a,b) recently proposed calling this system *working with memory* rather than simply *working memory*. There is some lingering controversy about whether the prefrontal cortex processes both temporal and spatial contextual information. Schacter (1987) proposed that the prefrontal cortex is involved in both temporal and spatial context. Lewis (1989) argued that the prefrontal cortex processes temporal contextual information but that the hippocampus (see below) plays a more critical role in processing spatial contextual information.

Both older theories (e.g., James, 1890) and recent research (Block, 1992) distinguishes between prospective and retrospective duration judgment. The working memory system of the prefrontal cortex appears mainly to subserve prospective temporal judgment, or the experience of time in passing. Short-duration judgments, as well as subsequent recency and temporal-order judgments, presumably require the functioning of this system. Milner et al. (1990) proposed two hypotheses on how the prefrontal cortex may subserve temporal-order encoding: (1) "If the frontal lobes parse and organize the temporal contexts of events, one outcome of such operations could be thought of as a direct encoding of temporal tags for events in memory" (p. 991), and (2) the frontal lobes "develop appropriate encoding and retrieval strategies for the reconstruction of temporal order" (p. 992). Although they favored the second hypothesis, the first hypothesis is also tenable, and the two functions are not necessarily mutually exclusive.

Episodic Memory

Evidence on the episodic-memory system and its role in psychological time comes from several sources. Here I discuss studies of brain damage, neurotransmitters, and drug effects, and electrophysiological recording.

Brain Damage

The medial temporal lobe of the brain contains several structures, underlying the temporal lobes but apparently also involving parts of the temporal cortex, that are needed to form explicit long-term episodic memories (Squire, 1987, 1992; Squire and Zola-Morgan, 1991). The hippocampus apparently is the most essential structure (see Figure 5.1), and in the interest of brevity I will occasionally use the term *hippocampus* to refer collectively to these several structures. Damage to the hippocampus produces the most common type of amnesia, called anterograde amnesia, which is characterized by a severe impairment in the episodic memory system: A person permanently loses the ability to encode new personal experiences so that they may be explicitly retrieved at a later time. Hippocampal damage largely spares the other memory systems; a temporal-lobe patient typically can use existing motor

skills and learn new ones (procedural memory), can use existing general knowledge and acquire new knowledge (semantic memory), and can display normal short-term memory for recent events (working memory).

Several patients have received operations involving the hippocampus, usually in attempts to relieve epilepsy or remove tumors. Scoville and Milner (1957) tested the memory of 10 such patients. The most well-studied patient, known as H.M., became amnesic following a bilateral medial temporal lobe resection that included the hippocampus. Richards (1973) studied H.M. on a task requiring reproduction of 1 to 300 s durations. H.M.'s reproductions were normal for durations less than 20 s. Thus, H.M. was able to maintain a working memory context for events in the psychological present, presumably because his prefrontal cortex was intact. However, for durations longer than about 20 s, his reproductions were abnormally short: "one hour to us is like 3 minutes to H.M.; one day is like 15 minutes; and one year is equivalent to 3 hours for H.M." (Richards, 1973, p. 281). The most likely explanation is that damage to H.M.'s hippocampus produced a condition in which he cannot permanently encode personal experiences so that they may be explicitly retrieved later. H.M. described his condition as "like waking from a dream" (Milner, Corkin, and Teuber, 1968, p. 217), suggesting a state of consciousness in which present events pervade consciousness and (postoperative) past events do not exist. Kinsbourne and Hicks (1990) reported that Korsakoff patients show a similar, although less dramatic, deficit.

The most common explanation for this type of amnesia is that damage to the hippocampus (and possibly also related medial temporal-lobe structures) impairs the encoding of new episodic memories. There are two main sources of controversy about this conclusion (for useful discussions, see Mayes [1988], Shallice [1988]). First, in addition to anterograde amnesia, or a deficit in encoding new episodic memories, H.M. and many other temporal-lobe patients also show retrograde amnesia. Retrograde amnesia involves a deficit in retrieving episodic memories that were encoded before the hippocampal damage. Most temporal-lobe patients display retrograde amnesia for events that occurred during the several months or, at most, several years before the operation. But Zola-Morgan, Squire, and Amaral (1986) reported the case of R.B., who developed memory impairment following an ischemic episode. Memory testing revealed that R.B. showed extensive and typical anterograde amnesia but little, if any, retrograde amnesia. They subsequently conducted a thorough histological examination of his brain, which revealed a circumscribed bilateral lesion involving the CA1 field of the hippocampus. Thus, damage restricted to only a small portion of the hippocampus is sufficient to produce anterograde amnesia without appreciable retrograde amnesia. It seems that the hippocampus is, therefore, necessary for encoding new episodic memories. The hippocampus and related medial temporal-lobe structures do not directly store episodic memories; instead, storage of them probably occurs throughout all cortical regions that are active in specific information-processing tasks.

A second source of controversy involves semantic memory and the episodic-semantic distinction. In the past, neuropsychologists typically thought that hippocampal damage also impairs semantic memory encoding, and some still do (Squire,

1992). The recent case of K.C. may question this conclusion (Tulving, Hayman, and MacDonald, 1991). K.C. experienced profound episodic (anterograde) amnesia following an automobile accident that damaged several areas, mostly in the left hemisphere, including the left medial temporal lobe and part of the right medial temporal lobe. In spite of this damage, Tulving et al. successfully taught K.C. a large number of three-word sentences, such as "reporter sent review" and "student withdrew innuendo." K.C. implicitly remembered much of this semantic information up to a year later, even though he had no episodic memory for the experience: He could not remember anything about the circumstances in which the learning had occurred. Just as the hippocampus is not required for retrieving existing knowledge, it is apparently not essential for encoding new knowledge (semantic memories).

McAndrews and Milner (1991) presented temporal-lobe amnesic patients with a series of stimuli and then tested their memory by presenting test stimuli in pairs and asking them to judge which of the two occurred more recently. When these amnesic patients were able to remember both stimuli, they performed normally on the recency-judgment task. Thus, although patients with medial temporal-lobe damage show deficits in encoding new episodic memories, when they are able to acquire and explicitly retrieve an episodic memory, they usually can remember temporal contextual information (such as approximately when they experienced the events). As noted earlier, patients with frontal-lobe damage show an opposite kind of performance: impaired memory for temporal information but normal memory for event information per se. This double dissociation suggests that the hippocampus and the prefrontal cortex perform separate but interrelated functions: As the hippocampus encodes information about the *content* of an episode, the prefrontal cortex may supply it with information about the *context* of the episode (for a slightly different view, see Moscovitch and Umilta [1992]).

Neurotransmitters and Drug Effects

Several neurotransmitters are found in the hippocampus, including acetylcholine, glutamate, and NMDA. Evidence suggests that any drug which interferes with acetylcholine-based neurotransmission in the hippocampus will influence retrospective duration judgments. Hicks (1992) reported that acetylcholine antagonists shorten the remembered duration of a time period. In addition, patients suffering from Alzheimer's disease frequently show severely impaired memory for personal experiences. They typically have damage in several brain areas, including the hippocampus. Their brains also show decreased acetylcholine synthesis (Khan, 1986), which would impair hippocampal neurotransmission. Although researchers have not adequately studied their temporal judgments, one would expect Alzheimer's patients to make abnormal retrospective duration judgments for recent events that they can remember.

Electrophysiological Recording

People maintain dynamic internal models of the environment, and events that deviate from the current model require that it be updated. If a person is attending to the performance of some task, presenting a relatively unexpected, but task-relevant stimulus will trigger a positive event-related potential (i.e., a time-locked voltage shift reflecting a change in brain activity), which begins about 300 ms after stimulus onset. Early research (e.g., Halgren, Squires, Wilson, Rohrbaugh, Babb, and Crandall, 1980) suggested that the medial temporal lobe (i.e., hippocampus and related structures) may generate at least some portion of P300. Polich and Squire (1993), however, found that an intact hippocampus is not required for P300 to occur. Activity in several brain regions, including areas of the frontal and parietal cortex, may contain P300-generators that summate to produce the scalp-recorded P300 amplitude (Johnson, 1993). Because P300 is larger following a novel stimulus than an expected one, it may reflect a process of schema- or context-updating (Donchin and Coles, 1988). Thus, P300 may reflect contextual information processing required for the formation of new episodic memories, which then critically requires the hippocampus. Alternatively, P300 may be generated, at least in part, by "an ancillary monitoring and gain-control system that assesses and controls modulation of the basic hippocampal memory system as a function of novelty of the incoming events" (Metcalfe, 1993, p. 333). In Metcalfe's view, firing of hippocampal neurons ordinarily contributes to P300 but is not the sole cause of it.

Episodic Memory and Psychological Time: Conclusions

The prefrontal cortex and the hippocampus are fairly directly connected and play a conjoint role in the processing of working memory and episodic information (Goldman-Rakic, Selemon, and Schwartz, 1984; Goldman-Rakic, 1987; Olton, 1989). As discussed earlier, the working memory system of the prefrontal cortex apparently generates encodings of information concerning temporal context. This information, which is perhaps in the form of time-of-occurrence of event relative to other events (Hintzman, Summers, and Block, 1975) is critically important for the episodic system. Schacter (1989) proposed "that remembering of temporal order constitutes one component of episodic memory, subserved by the frontal regions, and that remembering of recently presented items constitutes another component of episodic memory, likely subserved by the medial temporal regions" (p. 704).

To the extent that retrospective duration, order, recency, and other similar temporal judgments rely on event information no longer represented in the working memory system, they require the hippocampus for the permanent encoding of events. Temporal memory judgments concerning past events depend on retrieving encoded contextual changes, including changes in process context, environmental context, emotional context, and other contextual associations (Block, 1982, 1990b, 1992; Block and Reed, 1978). Encoding these contextual changes relies heavily on

the hippocampus, perhaps using temporal context information generated and supplied by the prefrontal cortex.

Summary and Conclusions

Table 5.1 summarizes four memory systems of the brain that subserve psychological time. Any single technique to study the brain necessarily provides limited information. Converging evidence from multiple sources suggests these conclusions about the four systems:

Table 5.1
Memory Systems of the Brain and Associated Characteristics

Memory System	Type of Information	Time-Related Behavior/Judgment	Major Brain Area(s)
Procedural	Movement	Movement Timing, Motor Skills	Cerebellum; Cortical Motor Areas (supplementary motor, premotor cortex, and motor cortex)
Semantic	Factual (linguistic)	Temporal Concepts	Mainly Left Temporal, Parietal, and Frontal Cortex
Working	Temporal Contextual	Prospective Timing; Recency and Order Judgment	Dorsolateral Prefrontal Cortex
Episodic	Personal Experience	Retrospective Duration Judgment	Hippocampus; Various Cortical Areas

1. Procedural memory critically involves the cerebellum, although some structures in the cerebral cortex (e.g., the supplementary motor cortex) are also involved in controlling certain kinds of movement timing.
2. Semantic memory involves widespread areas of the left-hemisphere temporal, parietal, and frontal lobes in understanding and expressing temporal facts and concepts.
3. Working memory involves the dorsolateral prefrontal cortex in constructing an ongoing temporal context; this contextual information is used in tasks such as prospective duration timing, order judgment, and recency judgment.
4. The permanent encoding of episodic memories requires intact functioning of the hippocampus and other medial temporal-lobe structures, apparently working in conjunction with temporal contextual information supplied by the prefrontal cortex; retrospective duration judgments and other long-term temporal memory judgments depend on this episodic information.

References

Baddley, A. D. (1986), *Working Memory*. London: Oxford University Press.

Block, R. A. (1979), Time and consciousness. In: *Aspects of Consciousness*, Vol. 1, ed. G. Underwood & R. Stevens. London: Academic Press, pp. 179–217.

———— (1982), Temporal judgments and contextual change. *J. Experiment. Psychol.: Learn., Mem., & Cog.*, 8:530–544.

———— (1989), A contextualistic view of time and mind. In: Time and Mind: Interdisciplinary Issues, *The Study of Time*, Vol. 6, ed. J. T. Fraser. Madison, CT: International Universities Press, pp. 61–79.

———— ed. (1990a), *Cognitive Models of Psychological Time*. Hillsdale, NJ: Lawrence Erlbaum Associates.

———— (1990b), Models of psychological time. In: *Cognitive Models of Psychological Time*, ed. R. A. Block. Hillsdale, NJ: Lawrence Erlbaum Associates, pp. 1–35.

———— (1992), Prospective and retrospective duration judgment: The role of information processing and memory. In: *Time, Action and Cognition: Towards Bridging the Gap*, ed. F. Macar, V. Pouthas, & W. J. Friedman. Dordrecht, Netherlands: Kluwer Academic, pp. 141–152.

———— Patterson, R. (in press), Simultaneity, successiveness, and temporal-order judgments. In: *Encyclopedia of Time*, ed. S. L. Macey. Hamden, CT: Garland.

———— Reed, M. A. (1978), Remembered duration: Evidence for a contextual-change hypothesis. *J. Experiment. Psychol.: Hum., Learn., & Mem.*, 4:656–665.

———— Saggau, J. L., & Nickol, L. H. (1983–1984), Temporal Inventory on Meaning and Experience: A structure of time. *Imagin., Cog., & Personal.*, 3:203–225.

Borda, R. P. (1970), The effect of altered drive states on the contingent negative variation (CNV) in rhesus monkeys. *Electroencephalogr. & Clin. Neurophysiol.*, 29:173–180.

Carr, C. E. (1993), Processing of temporal information in the brain. *Ann. Rev. Neurosci.*, 16:223–243.

Cohen, J. D., & Servan-Schreiber, D. (1992), Context, cortex, and dopamine: A connectionist approach to behavior and biology in schizophrenia. *Psychol. Rev.*, 99:45–77.

Damasio, A. R., & Damasio, H. (1992), Brain and language. *Sci. Amer.*, 267:89–95.

———— ———— Tranel, D., & Brandt, J. P. (1990), Neural regionalization of knowledge access: Preliminary evidence. *Cold Spring Harbor Symp. on Quant. Biol.*, 55:1039–1047.

Donchin, E., & Coles, M. G. H. (1988), Is the P300 component a manifestation of context updating? *Behav. & Brain Sci.*, 11:357–373.

Fuster, J. M. (1973), Unit activity in prefrontal cortex during delayed-response performance: Neuronal correlates of transient memory. *J. Neurophysiol.*, 36:61–78.

———— (1980), *The Prefrontal Cortex*. New York: Raven Press.

———— (1984), Behavioral electrophysiology of the prefrontal cortex. *Trends Neurosci.*, 7:408–414.

———— (1985a), The prefrontal cortex and temporal integration. In: *Cerebral Cortex*, ed. A. Peters & E. G. Jones. New York: Plenum Press, pp. 151–177.

———— (1985b), The prefrontal cortex, mediator of cross-temporal contingencies. *Hum. Neurobiol.*, 4:169–179.

Ghez, C. (1991a), The cerebellum. In: *Principles of Neural Science*, 3rd ed., ed. E. R. Kandel, J. H. Schwartz, & T. M. Jessell. New York: Elsevier Science, pp. 626–646.

———— (1991b), Voluntary movement. In: *Principles of Neural Science*, 3rd ed., ed. E. R. Kandel, J. H. Schwartz, & T. M. Jessell. New York: Elsevier Science, pp. 609–625.

Goldman-Rakic, P. S. (1987), Circuitry of primate prefrontal cortex and regulation of behavior by representational memory. In: *Handbook of Physiology: The Nervous System. V*, ed. F. Plum. Bethesda, MD: American Physiological Society, pp. 373–417.

———— (1992), Working memory and the mind. *Sci. Amer.*, 267:111–117.

———— Funahashi, S., & Bruce, C. J. (1990), Neocortical memory circuits. *Cold Spring Harbor Symp. on Quant. Biol.*, 55:1025–1038.

———— Selemon, L. D., & Schwartz, M. L. (1984), Dual pathways connecting the dorsolateral prefrontal cortex with the hippocampal formation and parahippocampal cortex in the rhesus monkey. *Neurosci.*, 12:719–743.

Halgren, E., Squires, N., Wilson, C., Rohrbaugh, J., Babb, T., & Crandall, P. (1980), Endogenous potentials in the human hippocampal formation and amygdala by infrequent events. *Science*, 210:803–805.

Hart, J., Berndt, R. S., & Caramazza, A. (1985), Category-specific naming deficit following cerebral infarction. *Nature*, 116:439–440.

Hicks, R. E. (1992), Prospective and retrospective judgments of time: A neurobehavioral analysis. In: *Time, Action and Cognition: Towards Bridging the Gap*, ed. F. Macar, V. Pouthas, & W. J. Friedman. Dordrecht, Netherlands: Kluver Academic, pp. 97–108.

———— Gualtieri, T., Mayo, J. P., & Perez-Reyes, M. (1984), Cannabis, atropine, and temporal information processing. *Neuropsychobiol.*, 12:229–237.

Hintzman, D. L., Summers, J. J., & Block, R. A. (1975), Spacing judgments as an index of study-phase retrieval. *J. Exp. Psychol.: Hum., Learn., Mem.*, 1:31–40.

Ivry, R. B., & Keele, S. W. (1989), Timing functions of the cerebellum. *J. Cog. Neurosci.*, 1:136–152.

———— Keele, S. W., & Diener, H. C. (1988), Dissociation of the lateral and medial cerebellum in movement timing and movement execution. *Exp. Brain Res.*, 73:167–180.

Jackson, J. L., & Michon, J. A. (1992), Verisimilar and metaphorical representations of time. In: *Time, Action and Cognition: Towards Bridging the Gap*, ed. F. Macar, V. Pouthas, & W. J. Friedman. Dordrecht, Netherlands: Kluver Academic, pp. 349–360.

James, W. (1890), *The Principles of Psychology*, Vol. 1. New York: Henry Holt.

Johnson, R., Jr. (1993), On the neural generators of the P300 component of the event-related potential. *Psychophysiol.*, 30:90–97.

Johnston, H. M. (1960), A comparison of time estimation of schizophrenic patients with that of normal individuals. *Psychol. Bull.*, 57:213–236.

Kandel, E. R., Schwartz, J. H., & Jessell, T. M., eds. (1991), *Principles of Neural Science*, 3rd ed. New York: Elsevier Science.

Khan, A. U. (1986), *Clinical Disorders of Memory*. New York: Plenum Press.

Kinsbourne, M., & Hicks, R. E. (1990), The extended present: Evidence from time estimation by amnesics and normals. In: *Neuropsychological Impairments of Short-Term Memory*, ed. G. Vallar & T. Shallice. Cambridge, U.K.: Cambridge University Press, pp. 319–330.

Kolb, B., & Wishaw, I. Q. (1990), *Fundamentals of Human Neuropsychology*, 3rd ed. New York: W. H. Freeman.

Kosslyn, S. M. (1992), *Wet Mind: The New Cognitive Neuroscience*. New York: Free Press.

Kubota, K., & Niki, H. (1971), Prefrontal cortical unit activity and delayed alternation performance in monkeys. *J. Neurophysiol.*, 34:337–347.

Leiner, H. C., Leiner, A. L., & Dow, R. S. (1991), The human cerebro-cerebellar system: Its computing, cognitive, and language skills. *Behav. Brain Res.*, 44:113–128.

Lewis, R. S. (1989), Remembering and the prefrontal cortex. *Psychobiol.*, 17:102–107.

Loveless, N. E., & Sanford, A. J. (1974), Slow potential correlates of preparatory set. *Biol. Psychol.*, 1:303–314.

Luria, A. R. (1966), *Higher Cortical Function in Man*. London: Tavistock.

Macar, F., & Vitton, N. (1979), Contingent negative variation and accuracy of time estimation: A study on cats. *Electroenceph. & Clin. Neurophys.*, 47:213–228.

Mayes, A. R. (1988), *Human Organic Memory Disorders*. Cambridge, U.K.: Cambridge University Press.

McAdam, H. W. (1966), Slow potential changes recorded from human brain during learning of a temporal interval. *Psychonomic Sci.*, 6:435–436.

McAndrews, M. P., & Milner, B. (1991), The frontal cortex and memory for temporal order. *Neuropsycholog.*, 29:849–860.

Melges, F. T. (1989), Disorders of time and the brain in severe mental illness. In: Time and Mind: Interdisciplinary Issues, *The Study of Time*, Vol. 6, ed. J. T. Fraser. Madison, CT: International Universities Press, pp. 99–119.

Metcalfe, J. (1993), Monitoring and gain control in an episodic memory model: Relation to the P300 event-related potential. In: *Theories of Memory*, ed. A. F. Collins, S. E. Gathercole, M. A. Conway, & P. E. Morris. Hillsdale, NJ: Lawrence Erlbaum Associates, pp. 327–353.

Michon, J. A. (1990), Implicit and explicit representations of time. In: *Cognitive Models of Psychological Time*, ed. R. A. Block. Hillsdale, NJ: Lawrence Erlbaum Associates, pp. 37–58.

Milner, B. (1971), Interhemispheric differences in the localization of psychological processes in man. *Brit. Med. Bull.*, 27:272–277.

—— (1974), Hemispheric specialization: Scope and limits. In: *The Neurosciences: Third Study Program*, ed. F. O. Schmitt & F. G. Worden. Cambridge, MA: MIT Press, pp. 75–89.

—— (1982), Some cognitive effects of frontal lobe lesions in man. In: *The Neuropsychology of Cognitive Function*, ed. D. E. Broadbent & L. Weiskrantz. London: Royal Society, pp. 211–226.

—— Corkin, S., & Teuber, H. L. (1968), Further analysis of the hippocampal amnesic syndrome: 14-year follow-up study of H.M. *Neuropsycholog.*, 6:215–234.

—— McAndrews, M. P., & Leonard, G. (1990), Frontal lobes and memory for the temporal order of recent events. *Cold Spring Harbor Symp. Quant. Biol.*, 55:987–994.

Mo, S. S. (1990), Time reversal in human cognition: Search for a temporal theory of insanity. In: *Cognitive Models of Psychological Time*, ed. R. A. Block. Hillsdale, NJ: Lawrence Erlbaum Associates, pp. 241–254.

Moscovitch, M., & Umilta, C. (1991), Conscious and nonconscious aspects of memory: A neuropsychological framework of modules and central systems. In: *Perspectives on Cognitive Neuroscience*, ed. R. G. Lister & H. J. Weingartner. New York: Oxford University Press, pp. 229–266.

—— Winocur, G. (1992a), Frontal lobes and memory. In: *The Encyclopedia of Learning and Memory*, ed. L. R. Squire. Hillsdale, NJ: Lawrence Erlbaum Associates, pp. 182–187.

—— —— (1992b), The neuropsychology of memory and aging. In: *Handbook of Aging and Cognition*, ed. F. I. M. Craik. Hillsdale, NJ: Lawrence Erlbaum Associates, pp. 315–371.

Nielsen, J. M. (1958), *Memory and Amnesia*. Los Angeles: San Lucas Press.

Niki, H. (1974), Differential activity of prefrontal units during right and left delayed response trials. *Brain Res.*, 70:346–349.

Olton, D. S. (1989), Inferring psychological dissociations from experimental dissociations: The temporal context of episodic memory. In: *Varieties of Memory and Consciousness: Essays in Honour of Endel Tulving*, ed. H. L. Roediger III & F. I. M. Craik. Hillsdale, NJ: Lawrence Erlbaum Associates, pp. 161–177.

Petrides, M., & Milner, B. (1982), Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. *Neuropsycholog.*, 20:249–262.

Polich, J., & Squire, L. R. (1993), P300 from amnesic patients with bilateral hippocampal lesions. *Electroencephalogr. Clin. Neurophysiol.*, 86:408–417.

Posner, M. I., Petersen, S. E., Fox, P. T., & Raichle, M. E. (1988), Localization of cognitive operations in the human brain. *Science*, 240:1627–1631.

Richards, W. (1973), Time reproductions by H.M. *Acta Psycholog.*, 37:279–282.

Rohrbaugh, J. W., Syndulko, K., & Lindsley, D. B. (1976), Brain wave components of the contingent negative variation in humans. *Science*, 191:1055–1057.

Sawaguchi, T., & Goldman-Rakic, P. S. (1991), D1 dopamine receptors in the prefrontal cortex: Involvement in working memory. *Science*, 251:947–950.

Schacter, D. L. (1987), Memory, amnesia, and frontal lobe dysfunction. *Psychobiol.*, 15:21–36.

—— (1989), Memory. In: *Foundations of Cognitive Science*, ed. M. I. Posner. Cambridge, MA: MIT Press, pp. 683–725.

Scoville, W. B., & Milner, B. (1957), Loss of recent memory after bilateral hippocampal lesions. *J. Neurolog. & Neurosurg. Psychiat.*, 20:11–21.

Shallice, T. (1982), Specific impairments of planning. *Philosoph. Trans. Royal Soc. London*, B298:199–209.

—— (1988), *From Neuropsychology to Mental Structure*. New York: Oxford University Press.

Squire, L. R. (1987), *Memory and Brain*. New York: Oxford University Press.

—— (1992), Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychol. Rev.*, 99:195–231.

—— Zola-Morgan, S. (1991), The medial temporal lobe memory system. *Science*, 253:1380–1386.

Tulving, E. (1972), Episodic and semantic memory. In: *Organization of Memory*, ed. E. Tulving & W. Donaldson. New York: Academic Press, pp. 381–403.

———— (1985), How many memory systems are there? *Amer. Psychologist*, 40:385–398.

———— (1989), Memory: Performance, knowledge, and experience. *Europ. J. Cog. Psychol.*, 1:3–26.

———— (1991), Concepts of human memory. In: *Memory: Organization and Locus of Change*, ed. L. R. Squire, N. M. Weinberger, G. Lynch, & J. L. McGaugh. New York: Oxford University Press, pp. 3–32.

———— Hayman, C. A. G., & MacDonald, C. A. (1991), Long-lasting perceptual priming and semantic learning in amnesia: A case experiment. *J. Experiment. Psychol.: Learn., Mem., & Cog.*, 17:595–617.

Vidal, F., Bonnet, M., & Macar, F. (1992), Can duration be a relevant dimension of motor programs? In: *Time, Action and Cognition: Towards Bridging the Gap*, ed. F. Macar, V. Pouthas, & W. J. Friedman. Dordrecht, Netherlands: Kluwer Academic, pp. 263–273.

Walter, W. G., Cooper, R., Aldridge, V. J., McCallum, W. C., & Winter, A. L. (1964), Contingent negative variation: An electric sign of sensori-motor association and expectancy in the human brain. *Nature*, 203:380–384.

Weinberger, D. R. (1987), Implications of normal brain development for the pathogenesis of schizophrenia. *Arch. Gen. Psychiat.*, 44:660–669.

Weiskrantz, L. (1987), Neuroanatomy of memory and amnesia: A case for multiple memory systems. *Human Neurobiol.*, 6:93–105.

Williams, J. M., Medwedeff, C. H., & Haban, G. (1989), Memory disorder and subjective time estimation. *J. Clin. & Exp. Neuropsychol.*, 11:713–723.

Zola-Morgan, S., Squire, L. R., & Amaral, D. G. (1986), Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *J. Neurosci.*, 6:2950–2967.