objects, as a result of recent encounters with them. Nondeclarative memory also affords the ability to exhibit shifts in preferences and judgments after exposure to novel material, and the ability to change gradually one's response to the external world as the result of conditioning. These findings provide strong evidence that memory is not a unitary mental function, but a collection of different abilities.

The study of retrograde amnesia, i.e., the loss of memory learned prior to the onset of amnesia, provides additional clues about the foundation and neurological organization of memory. The brain system damaged in amnesia is essential for the formation and storage of declarative memory and for its retrieval during a lengthy period of consolidation and reorganization. As time passes, the role of this system in memory diminishes, and a more permanent memory gradually develops elsewhere, probably in neocortex. These conclusions are supported by quantitative studies of retrograde amnesia in memory-impaired patients and more recently by prospective studies of retrograde amnesia in monkeys and rodents.

Finally, cumulative and systematic work in monkeys has identified structures and connections important for memory function. The findings to date suggest that the hippocampal formation and the closely related perirhinal and parahippocampal cortices comprise the medial temporal lobe memory system. Stereotaxic lesions of the hippocampal region (involving hippocampus proper, dentate gyrus, and the subicular complex) produce only a modest level of memory impairment. A similar level of memory impairment is found after global ischemia, which produces readily detectable damage in the CA1 region of the hippocampus and in the hilar region of the dentate gyrus. Larger lesions that include the entorhinal, parahippocampal, and perirhinal cortices produce more severe memory impairment. Importantly, the perirhinal and parahippocampal cortex, and possibly other cortical areas projecting to entorhinal cortex, do not serve simply as a connecting route to the hippocampus but themselves contribute to memory functions. The amygdala does not appear to be an essential component of this memory system, although it has been implicated in other kinds of cognitive functions, e.g., the formation of associations between a stimulus and its affective component.

11. Recovery of Memory after Traumatic Brain Injury

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Memory recovers in stages after traumatic brain injury (TBI). The acute stage of memory recovery has been labeled posttraumatic amnesia (PTA).

The measurement of termination of PTA is important, since time of discharge from hospital often depends on this measure as a criterion. Moreover, the duration of PTA has been considered to be a sensitive predictor of eventual outcome. The operational definition of what constitutes PTA, however, remains controversial. A prospective study of hospitalized patients with varying degrees of severity of traumatic brain injury was conducted to investigate the cognitive operations maximally impaired during this acute recovery stage. Measures of simple and complex attention, recall and recognition memory scores, and the Galveston Orientation and Amnesia Test were administered on a regular basis until the patients obtained perfect free recall of three words after a 24-hr delay (arbitrary criterion for PTA termination). The results indicated the following: for memory measures, encoding recovered before retrieval; retrieval recovery occurred later than orientation measures. Simple attention (count forward by 1) recovered first. There was a logical correspondence in the recovery of attentional and memory measures, with attentional measures improving first. For the mild to moderately impaired patients defined by the Glasgow Coma Scale, complex attention measures recovered before PTA termination. This suggests that the essence of PTA may not be a real "amnesia," but a confusional state affecting memory. In patients with severe TBI, there may be both a confusional state and overlying more severe cognitive deficits including memory dysfunction. The defined measures of memory and attention were useful in subdividing patients into subcategories other than the severity levels provided by the Glasgow Coma Scale, subcategories which may provide superior prediction of outcome. In later stages of recovery, memory deficits are common. Different aspects of memory impairment can be dissociated after one year or more of recovery. Susceptibility to interference was common. Difficulties in organizational strategies have been reported by some researchers, but not all. Automatic memory was less affected, while controlled memory was impaired. The dissociation of these disturbances in memory at different stages of recovery can be helpful in prediction of outcome, management of the patient, and elaboration of rehabilitation and treatment strategies.

12. What are "Memory Disorders" Disorders of?

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A hundred years ago memory was a simple and well-understood faculty of the brain/mind, and it was easy to talk and write about it and its pathology with authority. Thanks to all the research that has been done since that time

memory today is no more simple nor is it well understood. Indeed, it has become a very broad umbrella term, one that covers many different assemblies of large varieties of different processes. Thus "memory" is a term like "health," and references to "memory disorders" are no more informative than references to "health problems." This is why much greater specificity in dealing with memory and memory disorders is necessary.

This specificity can be described in terms of four dimensions of memory: (i) organization of memory: kinds of memory, or memory systems (Schacter & Tulving, 1994); (ii) operations of memory: processes such as encoding and retrieval and their interaction (Tulving, 1983); (iii) neurocognitive states of memory, such as "retrieval mode" (Tulving, 1983); and (iv) behavior, cognition, and conscious awareness of memory (Tulving, 1989). In this lecture I provide illustrative examples of recent progress in the understanding of these dimensions of memory.

Recent brain imaging research with healthy subjects has provided support for the distinction between episodic and semantic memory and has shown that the right frontal cortical regions play an important role in retrieval of episodic memory information (Fletcher et al., 1995; Nyberg et al., 1996). Clinical studies of isolated retrograde amnesia in brain-damaged patients have corroborated this notion (Calabrese et al., 1996; Levine et al., in press; Markowitsch, 1995). Other brain imaging studies have suggested that the frontal lobes exert their influence on episodic memory by their role in the establishment and maintenance of a special neurocognitive state referred to as the episodic "retrieval mode," one of the general, or "task-specific," necessary conditions of successful retrieval (Kapur et al., 1995; Nyberg et al., 1995; Schacter et al., 1996). The efficacy of psychometric assessment of episodic memory abilities of patients at risk for Alzheimer's diseases and other neurodegenerative conditions can be greatly enhanced by using tests constructed to reflect what is known about the interaction between the processes of encoding and retrieval (Buschke et al., 1997), and by using methods designed to distinguish between different kinds of conscious awareness involved in remembering past events (Barba, in press).

Empirical and theoretical research has benefited greatly from multidisciplinary approaches to the study of memory. Future research on memory disorders and practice in treating them will succeed to the extent that they incorporate the findings about the nature and processes of memory that this research has generated.

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13. Cortical Mechanisms for Memory: Automatic versus Voluntary Processes

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Physiological studies in monkeys have demonstrated that plastic neuronal changes are induced in the cortex during the storage and expression of memories of visual stimuli. In memory demanding tasks, such as delayed matching-to-sample, three commonly observed neuronal mechanisms are repetition suppression, enhancement, and delay activity (Desimone, 1996). In repetition suppression, repeated experience with the same visual stimulus leads to reduced responses of visual neurons. Such suppressive effects occur both in the short-term, over the course of seconds, and in the long-term, over the course of hours, days, and perhaps even permanently. Repetition suppression appears to be an automatic mechanism that is an intrinsic property of visual cortical areas, such as the inferior temporal cortex. In contrast to repetition suppression, is neuronal enhancement, the responses of visual neurons are increased for visual stimuli that have behavioral relevance. Delay activity