Chapter 23

SEMANTIC MEMORY IMPAIRMENTS

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The term *semantic memory* refers to our general knowledge of the objects, people, and events of the world.[1] The facts that Paris is the capital of France, birds have feathers, and a desk is a piece of furniture are examples of semantic memory. More particular knowledge, tied to an individual's personal experience, is considered *episodic memory* rather than semantic memory. Examples of the latter include the facts that you bought this book at a certain store or ate a certain food for breakfast this morning. Neurologic disease and damage can affect semantic memory disproportionately. In this chapter the different forms of semantic memory impairment are reviewed, with attention to their etiologies, major behavioral features, and implications for the neural substrates and functional organization of semantic memory in the normal brain.

GENERALIZED IMPAIRMENT OF SEMANTIC MEMORY

Warrington[2] first documented a pattern of preserved and impaired performance indicative of semantic memory impairment in a series of three patients suffering from progressive degenerative brain disease. Her subjects were relatively preserved on most measures of language and cognitive function but did poorly on tasks dependent on semantic memory, including confrontation naming, word-picture matching, and a verification task in which subjects were shown pictures or words and asked questions such as "Is it a bird?" or "Is it heavy?" In subsequent years a number of similar cases were reported, and the term *semantic dementia* was coined in the context of one such report.[3] Hodges and coworkers[4] presented a wide-ranging study of five new cases of semantic dementia, reviewed the literature, and drew a number of useful generalizations concerning the condition. A summary of their conclusions is presented here.

Semantic dementia may present initially as a language disorder whose most prominent feature is vocabulary loss, both expressive and receptive. Naming is minimally aided by phonemic cues, and naming errors tend to share a semantic relation with the correct name (e.g., *violin* for *accordion,* or *animal* for *fox*). In production, category fluency is severely impaired, and word definitions are impoverished or wrong. Such patients have sometimes been described as having a fluent form of primary progressive aphasia (see Chaps. 14 and 15), but additional language testing and nonverbal semantic memory testing suggest that the underlying impairment is one of semantic memory knowledge rather than language. Syntax and phonology tend to be preserved, whereas entirely pictorial tasks that depend on knowledge of the depicted objects, such as sorting together semantically related objects or distinguishing real from imaginary objects, are failed. Although the formal assessment of episodic memory is difficult because of the loss of knowledge of word and picture meanings, Hodges and coworkers[4] observe that at least some patients show significant preservation of autobiographical

memories and practical day-to-day memory. The neuropathologic changes in semantic dementia are focused in the temporal lobes, often affecting the left more than the right. A small number of brains have come to autopsy with Pick's disease.

Another degenerative condition affecting semantic memory is Alzheimer disease[5–10] (AD), although semantic memory is just one of many aspects of cognition impaired in AD, and initially some cases may present with only episodic memory impairment. To the extent that semantic memory is impaired in AD, pathologic changes in temporal cortex are responsible.

In sum, semantic memory is at least partially dissociable from other forms of memory, language, and cognition, generally as a result of degenerative diseases. It appears to depend on temporal cortex, with some degree lateralization to the left suggested.

SELECTIVE IMPAIRMENTS OF SEMANTIC MEMORY

In addition to the generalized impairments of semantic memory described above, particular aspects of semantic memory can be disproportionately impaired. These disorders are potentially informative about the internal organization of semantic memory in the brain, although their proper interpretation and even their existence have been issues of controversy.

Category-Specific Semantic Memory Impairment

In some cases it appears that knowledge from certain semantic categories is disproportionately impaired, suggesting that the neural bases of semantic memory are subdivided by semantic category. Category-specific semantic memory impairments are sometimes confused with category-specific impairments in name retrieval and visual recognition. The "fruit and vegetable" impairment observed in two cases[11,12] affects naming only; the face-specificity of prosopagnosia (see Chap. 7) affects visual recognition only. In contrast, category-specific semantic memory impairments are manifest in all tasks that require knowledge of the object, whether they involve vision, language, or other modalities of stimulus and response. The most common category-specific semantic memory impairment affects knowledge of living things.

The first report of impaired knowledge of living things was made by Warrington and Shallice,[13] who described three patients who had survived herpes encephalitis. Although the patients were impaired across the board at tasks such as picture naming and defining words, they were dramatically worse when the pictures or words represented animals and plants than when they represented artifacts. In subsequent years numerous other reports appeared of similar cases, generally suffering damage to temporal cortex from herpes encephalitis, closed head injury or, less frequently, cerebrovascular or degenerative disease. Category-specific disorders of semantic memory are distinct from the disorders described in the previous section, despite the implication of temporal brain regions in both, as neither semantic dementia[4] nor Alzheimer disease[14] routinely affect knowledge of living things more than nonliving.

The idea that certain brain regions are specialized for representing knowledge about living things has naturally aroused some skepticism and prompted a search for alternative explanations of apparently impaired knowledge of living things. The simplest alternative explanation is that the impairment is an artifact of the greater difficulty of retrieving knowledge about living things. It has been suggested that when difficulty is equated across living and nonliving test items, the selectivity of the semantic memory impairment disappears.[15,16] However, the selectivity has also been shown to be reliable in two cases when multiple measures of difficulty are accounted for,[17] and the null results in other controlled studies are likely due to insufficient statistical power, as our reliable findings disappeared when we reduced our data set to the size of the other studies' data sets.[18]

Cases of impaired knowledge of nonliving things with relatively spared knowledge of living

things are rarer but have also been described.[19–23] The lesions in these cases are confined to the left hemisphere. A precise intrahemispheric localization is not possible, as the lesions are typically large and relatively few cases have been reported, although the left temporal region again seems involved.[23] These patients provide the other half of a double dissociation with impaired knowledge of living things, thus adding further support to the hypothesis that category-specific semantic memory impairments are not simply due to the differential difficulty of particular categories.

Building on the hypothesis of Allport,[24] that semantic memory is subdivided into different sensorimotor modalities (e.g., visual knowledge, tactile knowledge, and motor knowledge; see Fig. 23-1 for an illustration of this idea), Warrington and Shallice[13] proposed a different kind of alternative explanation for category-specific knowledge deficits. They suggested that living and nonliving things may differ from one another in their reliance on knowledge from different sensorimotor modalities, with living things being known predominantly by their visual and other sensory attributes. Impaired knowledge of living things could result from an impairment of visual knowledge. Similarly, nonliving things might be known predominantly by their function, an abstract form of motoric representation, and impaired knowledge of nonliving things could result from an impairment of functional knowledge. This interpretation has the advantage of parsimony, in that it invokes a type of organization already known to exist in the brain—modality-specific organization—rather than invoking an organization based on semantic categories such as aliveness. A computer simulation of semantic memory and its impairments has shown that a modality-specific organization can account for category-specific impairments, even the finding that functional knowledge of living things is impaired after visual semantic damage.[25] The latter finding is explained by the need for a certain "critical mass" of associated knowledge to help activate collaterally any one part of a distributed representation; if most of the representation of living things is visual and visual knowledge is damaged, then the remaining functional knowledge cannot be activated.

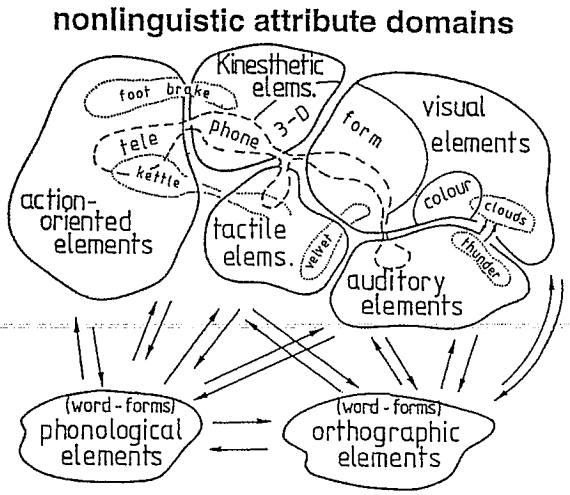

Figure 23-1
A modality-specific organization for semantic memory. Rather than hypothesize a store of knowledge in the brain separate from the various sensorimotor modalities used in perception and action, semantic memory is hypothesized to consist of representations in sensorimotor systems themselves. (From Allport,[24] with permission.)

Modality-Specific Semantic Memory Impairment

There is a second way in which the phrase *modality-specific semantic memory* has been used in neuropsychology, and that is for components of semantic memory that are accessed *through* a particular input or output modality. According to this usage, visual semantics refers not to semantic knowledge of the visual appearance of objects but to the semantic knowledge of appearance, function, and so on that is accessed when an object is seen. Whether semantic memory has a modality-specific organization in this sense is not clear, although such an organization has been hypothesized for purposes of explaining "optic aphasia."

Optic aphasia is a puzzling disorder, consisting of an impairment in naming visually pre-

sented stimuli in the face of relatively preserved naming of nonvisual stimuli and relatively preserved nonverbal demonstrations of visual recognition. It seems reasonable to assume that visual confrontation naming requires three major stages of processing: vision, semantics, and lexical retrieval. That is, it requires seeing the object clearly enough to access semantic knowledge of it, and using that semantic knowledge of what the object is to retrieve its name. Paradoxically, the preserved nonvisual naming and nonverbal recognition performance of optic aphasics seem to exonerate all three stages.

A variety of attempts have been made to explain how an anomia could exist for visual stimuli only, although none has been thoroughly tested or gained wide support (see Ref. 26 for a review). One of these accounts invokes a modality-specific semantic memory system in which visual semantics (i.e., the semantic knowledge accessed by visual inputs) has been disconnected from verbal semantics (i.e., the semantic knowledge necessary to access a verbal output).[27,28] This hypothesis was formulated to explain the major features of optic aphasia and is successful in so doing, although converging evidence from other sources is now desirable.

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