

SPECIAL ISSUE

THE NEUROPSYCHOLOGY OF AUTOBIOGRAPHICAL
MEMORY

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ABSTRACT

This special issue of *Cortex* focuses on the relative contribution of different neural networks to memory and the interaction of 'core' memory processes with other cognitive processes. In this article, we examine both. Specifically, we identify cognitive processes other than encoding and retrieval that are thought to be involved in memory; we then examine the consequences of damage to brain regions that support these processes. This approach forces a consideration of the roles of brain regions outside of the frontal, medial-temporal, and diencephalic regions that form a central part of neurobiological theories of memory. Certain kinds of damage to visual cortex or lateral temporal cortex produced impairments of visual imagery or semantic memory; these patterns of impairment are associated with a unique pattern of amnesia that was distinctly different from the pattern associated with medial-temporal trauma. On the other hand, damage to language regions, auditory cortex, or parietal cortex produced impairments of language, auditory imagery, or spatial imagery; however, these impairments were not associated with amnesia. Therefore, a full model of autobiographical memory must consider cognitive processes that are not generally considered 'core processes,' as well as the brain regions upon which these processes depend.

Key words: anterograde amnesia, retrograde amnesia, perceptual disorders, temporal lobe, visual cortex, memory

INTRODUCTION

Over the last few years, we have been developing a multi-system model of memory by combining our basic understanding of neuropsychology and neuroanatomy with behavioral studies. This development began with a study of oral traditions (Rubin, 1995), and was then extended to autobiographical memory (Rubin, 1998; Rubin and Greenberg, 1998, in press). Researchers disagree about the precise meaning of "autobiographical memory"; for example, some view it as a form of episodic memory (Kopelman and N. Kapur, 2001; Rubin, 1998), while others use different definitions of these terms and thereby arrive at the opposite view (Conway, 2001). Still others prefer the term "recollective memories" (Brewer, 1995). When we use the term "autobiographical memory," we refer to memories that have several properties. First, like episodic memory, autobiographical memory "receives and stores information about temporally dated episodes or events and temporal-spatial relations among them" (Tulving, 1983, p. 21). Second, such memories involve something more than the mere retrieval of stored data: the person remembering

the memory must be conscious of the prior conscious experience, a self-reflective mental state that Tulving terms *autonoetic consciousness* (Tulving, 1985). As with many of the other cognitive processes we will discuss, autonoetic consciousness is not sufficient for autobiographical memory (it plays a role in other processes, such as prospective memory (Wheeler, Stuss, and Tulving, 1997)) but it is a necessary feature: many philosophical accounts (Brewer, 1995) and the reports of some amnesics (e. g. Crovitz, 1986; O'Connor et al., 1992) suggest that an autobiographical memory should be accompanied by a sense of reliving as well as the belief that the remembered event actually occurred. We define an *autobiographical memory* as a memory of a personally experienced event that comes with a sense of recollection or reliving. *Autobiographical facts* (Brewer, 1995), on the other hand, are bits of personally relevant information that are retrieved without this sense of reliving.

Most theories of memory claim that memory requires an interaction between medial temporal lobes, frontal lobes, and the rest of the cortex (Conway and Pleydell-Pearce, 2000; Damasio, 1989; Fuster, 1995; Kopelman, 2000; Kopelman and N. Kapur, 2001; Mayes and Roberts, 2001; Markowitsch, 2000; McClelland et al., 1995; McDonald et al., 1999; Murre, 1999; Murre et al., 2001; Shastri, 2002; Squire, 1992). The implications of this claim have rarely been investigated, however. In particular, most major theories do not dissect memory storage into modality-specific components. Conway and Pleydell-Pearce (2000), for example, state that memories are stored in an “undifferentiated pool” called “event-specific knowledge” (Conway and Pleydell-Pearce, 2000, Figure 1, p. 265; also see Conway, 1992, 1995a and Conway and Rubin, 1993 for further discussion of this idea). Kopelman’s model provides a detailed analysis of executive and emotional systems that play a role in memory, but devotes only a single module to storage (Kopelman, 2000, Figure 6, p. 608; Kopelman and N. Kapur, 2001, p. 1417). Along the same lines, McClelland and colleagues (1995, Figure 14, p. 444), Squire (1992), Murre (1999, Figure 1, p. 269), and Markowitsch (2000) all focus on medial temporal regions and devote little attention to posterior neocortical storage sites. McDonald and colleagues (1999) mention the hippocampus, the amygdala, the frontal lobes, the thalamus, and the basal ganglia – in fact, almost every region *besides* the posterior neocortex. Even theories that do mention memory in the neocortex do not address the relative contributions of several different cortical areas to memory in general (with the exception of studies addressing anterior and lateral temporal regions, which we review later in the paper). We are not claiming that we know little about the roles of these neocortical regions – in fact, the contrary is generally true – but rather that any such knowledge has rarely found its way into neurobiological theories of *memory and autobiographical memory* specifically. Nor are we embarking on a rehash of current theories or a simple assignment of a function to a region; instead, we are attempting to ask more detailed questions: given what we know about autobiographical memory on the behavioral level, which brain regions should be involved? Most importantly, what are the particular contributions of each of these regions to autobiographical memory, and what happens when they are damaged or destroyed?

We attempt to answer these questions by reviewing relevant neuropsychological case studies. We organize the paper around five interacting cognitive processes that have been identified in the behavioral data as important components of autobiographical memory: explicit memory, imagery (in visual, auditory, and other modalities as well as multi-modal spatial imagery), language, narrative, and emotion. After reviewing the importance of these behavioral processes, we identify the brain regions on which they depend; then, we search the neuropsychological literature to discover how autobiographical memory changes after impairments of these processes and damage to these regions. Some of these components will turn out to be relatively unimportant; in other cases, neuropsychological evidence will force the addition of a component to the overall model.

We selected these processes for several reasons. First, neuropsychological studies have shown double dissociations between pairs of these processes. Second, these neuropsychological data, when coupled with evidence from functional imaging and neuroanatomical studies, demonstrate that these processes have distinct neurological substrates. Third, the literature on individual differences demonstrates that linguistic and imagery abilities vary along similar lines (Carroll, 1993). Fourth, perhaps for these reasons, they are also treated as distinct cognitive processes in the behavioral literature (and are even given separate sections in many textbooks and separate names in common language). While these processes can certainly be divided further, evidence on all levels indicates that they can be treated as separate and discrete behaviorally and neuropsychologically.

These processes and brain regions might seem excessively broad; however, several factors compel a broad analysis. First, behavioral research has largely dealt with broadly defined cognitive processes; therefore, if this investigation is to have clear relevance to the behavioral data, it must operate at a similar level. Second, the neuropsychological literature does not necessarily report subtle disorders, especially if they occur in a patient with another interesting disorder; therefore, relevant data are often absent. Third, this investigation is in its early stages, and an examination of broadly defined processes and brain regions is most likely to detect interesting results. We will see if the most severe impairments will have any effect on autobiographical memory; if they do, we can try to uncover the roles of the subcomponents of these cognitive processes by studying the effects of circumscribed impairments.

Throughout this paper, we make claims about where parts of memories are stored – a point that requires some explanation. Psychology uses two opposing metaphors of memory. The first claims that memories are stored at encoding and later retrieved. The second maintains that the mind/brain changes with experience, so that it will respond differently when exposed to stimuli in the future. Under the latter metaphor, a memory is not stored; rather, the system that perceives and acts is changed. Although the latter metaphor fits better with our general approach, we tried to write the paper so it could be understood in terms of either metaphor, because we have found that readers who think in terms of one metaphor often find the other extremely difficult to understand. When we say, for example, that visual aspects of an autobiographical memory are stored in the visual system and not the medial temporal or frontal lobes, under the first

metaphor we really mean the following longer and more awkward statement: “It is possible to damage parts of the visual system and remove visual information that is not available outside the visual system.” Under the second metaphor, we really mean “The visual system processes visual information, not all of which is shared with non-visual areas. Thus, when the visual system is damaged, the system that would respond to a cue with more detailed visual information and analysis is no longer available.” Our claim is that most visual information is stored and/or processed solely within the visual system, and so can be lost with damage to just that system. We will make similar claims for other sensory modalities, emotion, language, and narrative. In the last two cases, terms like “analysis” or “processing” may seem better to the reader than “storage,” but it must be processing that is particular to a specific autobiographical memory that reflects past experience. We note, though, that while the brain is largely modular (Fodor, 1983), some visual information, or binding or indexing of that information, is stored in the medial temporal or frontal lobes. There must be such information outside the visual system if there is to be both modularity and integrated autobiographical memories.

We base our approach on four well-supported observations:

1. Almost all (and perhaps all) cognition is affected by past experiences. Searching for a single neural location of memory is a fool’s errand. Memory is stored everywhere, and at every level of analysis (Fuster, 1995; Toth and Hunt, 1999). Autobiographical memories – which consist of multi-modal stimuli, extend over time, and are organized along narrative and emotional dimensions – are probably distributed throughout the brain. Neuropsychologists have long maintained that complex cognitive processes are localized not in specific brain nuclei, but in the coactivation of diffuse neuroanatomical substrates (Lashley, 1950/1960; Luria, 1966; Penfield and Perot, 1963). Thus, we are not proposing a new idea; instead, we are gathering the evidence needed to apply this idea to autobiographical memory.

2. Much of the brain is organized by sensory modality; we know at least as much about these areas as any other areas – and more about the cognition they subserve. These sensory areas qualify as *modules*: their processing is quick, obligatory, and cognitively impenetrable, and their output is shallow. Moreover, they are domain-specific – damage to one module does not affect cognition in another module (see Fodor, 1983 for a discussion of modularity and Moscovitch, 1992 for an application of modularity theory to the neuropsychology of memory).

3. Medial temporal and frontal structures are required for the recall of specific events, but the rest of the brain – especially the posterior cortices that process and store the sensory components of autobiographical memory – contributes something.

4. Because consciousness is such a difficult topic, and is defined in so many different ways by so many different people, we restrict our consideration of it to the phenomenological report that people often *recollect* – that is, they relive their past states of consciousness when they recall an event. We include this observation because many psychologists and many philosophers of mind consider it a defining feature of autobiographical memory (Brewer, 1986, 1995; Rubin and Greenberg, 1998; Wheeler et al., 1997).

EXPLICIT MEMORY

We provide only a brief summary of the main points of agreement in the field, which we will call the “consensus theory.” This theory makes three general claims:

1. *The medial temporal lobes form links between areas of the brain that are active at the same time.* According to the consensus theory, a given experience activates multiple regions in sensory and association cortex: visual stimuli activate visual cortex, auditory stimuli activate auditory cortex, and so on. The MTL then encodes the experience by binding together these disparate brain regions, thus forming conscious associations among stimuli that are presented at the same time (Squire, 1992). The retrieval of recent memories is thought to require the MTL as well as areas of sensory and association cortex. Other potential roles remain controversial; some theorists (e. g. Nadel, 1995; Nadel and Moscovitch, 1997, 1998) claim that all episodic memories require the MTL regardless of their age, but others (Squire, 1992) maintain that older memories are “consolidated” and can be retrieved without the MTL. These disagreements are not relevant to our arguments, which only require the consensus claim that the MTL binds together coactivated brain regions and does not contain the memory in its entirety.

2. *The frontal lobes search for relevant information and inhibit irrelevant information.* How does the brain select one particular association from multiple associations that have been stored at many different times? Substantial evidence indicates that the frontal lobes are involved in such selection (Nyberg et al., 1996a). For example, neuroimaging studies have shown that the left prefrontal cortex may help organize information for later recall (Fletcher et al., 1998). The right frontal lobe is generally thought to initiate ‘retrieval mode’ (Wheeler et al., 1997). One line of evidence suggests that the right frontal lobe is involved in retrieval attempt – even if the attempt is unsuccessful – via its interaction with posterior cortices (S. Kapur et al., 1995), and other research proposes that it is involved in error-checking. Thus, two regions – the frontal lobes and the medial temporal lobes – coordinate memory encoding and retrieval (Moscovitch, 1992). This dual-systems idea appears in cognitive theories as well; similar distinctions are made in the behavioral literature on post-traumatic stress disorder (Brewin et al., 1996) and childhood memory (Pillemer and White, 1989).

3. *These brain regions do not actually store the basic information that comprises our autobiographical memories, but rather coordinate the storage of these data in posterior cortical modules.* All major theories agree that the medial temporal and frontal lobes do not themselves provide long-term storage of most sensory data (though they may store connections among modules). These regions coordinate activation among widespread areas of cortex and produce a pattern of activation similar to that present during the original experience. Neocortical regions do seem to be able to store information on their own, particularly after many trials, but the extent of this ability remains unclear (McClelland et al., 1995).

Other Relevant Brain Regions

Other brain regions have been implicated in memory, including the thalamus (e. g. Hodges and McCarthy, 1993; Miller et al., 2001), and the fornix, medial forebrain, and temporal stem (Easton and Gaffan, 2001; Gaffan et al., 2001). In addition, research on *focal retrograde amnesia* (N. Kapur, 1999; Wheeler and McMillan, 2001) has suggested that the anterior inferior temporal lobes play an important role in memory (Eslinger et al., 1996; Eslinger, 1998; Tanaka et al., 1999). One theory suggests that these regions, not the MTL, may coordinate the firing among disparate sensory cortices that underlies retrieval (N. Kapur, 1997, 1999; N. Kapur et al., 1992; Yoneda et al., 1992). The theory we will present is neutral with respect to these regions.

A Real-World Example

An example will help clarify the consensus theory. The smell of hamburgers on a grill might lead to activation in olfactory areas of cortex, which, via MTL mediation, would activate a pattern of firing in visual cortex that represented a friend one saw at a recent cookout, as well as other memories. This activity in visual cortex might in turn stimulate new activity in auditory cortex that represented the sound of a conversation one had with that friend; it would also stimulate irrelevant information from other episodes that involved this particular friend. The frontal lobes work to inhibit information that is not part of one particular memory. This cascade of activation continues, with visual, olfactory, auditory, and other cortical areas stimulating other sensory components associated with other parts of the memory while feeding back upon and helping to maintain the original pattern of firing in olfactory cortex. The activity would also stimulate regions dedicated to language, narrative, and emotion that would organize and impart emotional tone to the memory. For these non-sensory areas, the consensus model is less clear; they can be viewed as processing areas with minimal storage of episodic information, or they can be seen as processing and storage areas. The recall of an autobiographical memory occurs over one measurable time period, not in one brain location; it is located in time but distributed in space.

With the basic framework described, we now turn to the roles of the brain regions other than those emphasized in the consensus theory.

THE ROLE OF THE REST OF THE BRAIN: A CONSENSUS VIEW EXTENDED

With few exceptions, investigations of the neural basis of human memory go this far and no further. Although the current models acknowledge the importance of the areas of posterior cortex that actually store the data that make up a memory, they rarely explore the consequences of damage to those regions (but see Rubin and Greenberg, 1998, and Conway and Fthenaki, 2000). All modern theories of amnesia explicitly state that the perceptual and conceptual information that is the basis of autobiographical memories is not stored primarily

in the medial temporal, diencephalic, or frontal regions, but rather in posterior cortices. Under this consensus view, removing these regions and modules must remove some of the basic information that comprises memories and should therefore have some noticeable effect on autobiographical memory. Again, these claims are straightforward, but have rarely been tested or even stated explicitly; we endeavor to do so here. Even neuroimaging approaches largely overlook this issue; although they may mention activation of posterior cortices (e. g. Conway et al., 1999; S. Kapur et al., 1995; Nyberg et al., 1995, 1996a, 2000a, b; Taylor et al., 1998), they rarely discuss such activations in detail (but see Conway et al., 2001, for a study that explicitly set out to examine activation in these regions).

A cognitive impairment can have two possible effects on autobiographical memory:

1. *The memory impairment will be limited to the cognitive process affected by the impairment.* Within this category, there are multiple possibilities. First, perhaps the damage will entirely destroy sensory data; for example, patients with an impairment of auditory imagery might experience memories without sound, much as though they were watching a movie with the sound turned off. Alternatively, the damage might preserve data but destroy the ability to interpret it; for example, a patient with prosopagnosia might still be able to see and describe people in his or her memories but be unable to identify them. When stored information is lost, this is the minimum possible effect, although it may remain undetected if the information is rarely used.

2. *The impairment will result in a global impairment of autobiographical memory.* Perhaps the damaged system is so important to autobiographical memory that its loss would be catastrophic. All modern theories evoke some form of a coactivation mechanism that at recollection has a pattern of firing similar to the pattern that occurred during the original event. Removing a large portion of that circuit could impair such patterns of firing. An impaired ability to retrieve data from posterior cortices could therefore result in global amnesia for autobiographical events, not just a simple loss of data within individual memories.

VISUAL OBJECT IMAGERY

Behavioral Data

In general, visual imagery facilitates recall. Many mnemonic devices, whether they come from ancient Greece or popular books on memory improvement, focus on the generation of visual images (Paivio, 1971; Yates, 1966); likewise, some notable mnemonists rely heavily on visual imagery (such as Luria's (1968) patient S.). The clearest, most salient, and most compelling autobiographical memories are "flashbulb" memories (R. Brown and Kulik, 1977), which the rememberer perceives as permanent, fixed pictures of important events (Conway, 1995; Neisser, 1982; Winograd and Neisser, 1992). These memories involve vivid visual images, even if such images were not deliberately stored during encoding (Rubin and Kozin, 1984). In fact, Brewer (1986, 1995) proposed that

the retrieval of a visual image is the defining feature of autobiographical memory – that it distinguishes events that are remembered from data that are simply known. All these observations suggest that an impairment of visual imagery might well result in an impairment of autobiographical memory.

Neuropsychological Data

Optic blindness, whether congenital or acquired, does not produce any significant memory impairment. At encoding, the blind use tactile, spatial, and auditory information to compensate for their lack of visual input (Tinti et al., 1999). Studies of imagery in the congenitally blind find that it is difficult to show any deficit on tasks involving visual imagery when tactile or verbal instead of visual input is used (De Beni and Cornoldi, 1988; Kerr, 1983). Even when deficits do exist, they are relatively mild; patients do not perform at floor (Vecchi, 1998). For patients with acquired blindness, anterograde memories – that is, the memories that were stored after they became blind – contain little if any visual information. Retrograde memories make use of stored visual data, which can be retrieved without difficulty since visual brain regions are intact (although if the memory is retrieved repeatedly, it is also possible that visual data are recoded into other modalities). Optically blind patients do not really fall into either of our categories of deficit, because they suffer from a peripheral sensory impairment rather than an impairment of a cognitive process; still, it is worth noting that they seem to be able to compensate for their deficits without significant impairment of autobiographical memory beyond the loss of visual information in anterograde memories (see Ogden and Barker, 2001, for an examination of this issue).

Visual imagery appears to rely upon the same posterior cortical regions as visual perception (Thompson and Kosslyn, 2000). What happens to people who make use of visual imagery throughout their lives, then suffer some trauma to the areas of posterior cortex that subserve it? On the surface, visual agnosics seem to be one such group of patients. Visual agnosics cannot indicate the name or the function of visually presented objects (for a review, see Farah, 1990). These patients would appear to suffer from a deficit of visual imagery; however, some of these patients can still access visual memories through other modalities; the patient known as HJA, for example, could draw pictures on verbal command and his autobiographical memories appeared to be intact (Humphreys and Riddoch, 1987). Although information remains scanty, visual agnosics who can still retrieve images appear to be at the first level of impairment.

What about patients who cannot access visual information through any modality? In earlier work (Rubin and Greenberg, 1998), we sought a set of such patients. We were aided in this task by Farah's (1984) neuropsychological investigation of visual imagery. Based on Kosslyn's (1980) detailed theory of visual imagery, she developed a component model of visual imagery, including a long-term visual memory for the store of visual images. She proposed that patients with a long-term visual memory deficit would meet three criteria. First, the patient must be able to copy line drawings, thereby indicating that their perceptual abilities are intact. Second, the patient must be unable to recognize

objects by sight, defined as an inability to indicate their names and their functions. Third, the patient must be unable to draw objects from memory, describe their visual properties from memory, or detect a visual image of them upon introspection. The first two criteria identify the patient as a visual agnostic; the third criterion demonstrates that the deficit arises from impaired access to long-term visual memory rather than a difficulty generating, manipulating, or interpreting images (Farah, 1984). We found 11 cases that met these three criteria (Albert et al., 1975; Beyn and Knyazeva, 1962; J. Brown and Chobor, 1995; Gomori and Hawryluk, 1984; O'Connor et al., 1992; Ogden, 1993; Ratcliff and Newcombe, 1982; Shuttleworth et al., 1982; Taylor and Warrington, 1971; Trojano and Grossi, 1992; Wapner et al., 1978). Our results were striking: as shown in Appendix A, all 11 of these patients suffered from amnesia. We called this syndrome *visual memory-deficit amnesia* (VMDA), meaning a form of amnesia that resulted from a deficit of visual memory. Although medial-temporal damage may account for some of the memory loss in some of these cases (5 of 11 patients had some sign of MTL trauma), the patterns of the patients' deficits suggest otherwise. Of the 7 cases that described their patients' memory deficits in detail, 5 suffered from severe retrograde amnesia with more moderate anterograde deficits. Moreover, the temporal gradient was absent in the 4 cases in which it was described (see Rubin and Greenberg, 1998 for another examination of these cases; for additional reviews, see Conway and Fthenaki, 2000, and Wheeler and McMillan, 2001). In contrast, MTL amnesics generally suffer from an anterograde deficit that is more severe than the retrograde and a temporally graded retrograde amnesia that spares older memories (but see Conway and Fthenaki, 2000, and Nadel and Moscovitch, 1997, 1998 for an opposing view). Of the three levels of severity and nature of loss described earlier, the 11 patients with visual memory loss all had the second of retrograde autobiographical memory loss – a loss that spread well beyond a visual deficit to all aspects of autobiographical memory.

Our investigation was hampered by the lack of detail provided by many of the reports. Although patients often present with many deficits, neuropsychologists tend to focus on an examination of one of these to make their reports clearer and more concise. Thus, most researchers were concerned with either agnosia or amnesia, not the relation between the two; only three cases provided thorough investigations of both types of deficit.

The earliest of these is the case of LD (O'Connor et al., 1992), who suffered a bout of herpes simplex encephalitis at the age of 26. In the right hemisphere, she suffered damage to ventromedial frontal, parietal, and parieto-occipital regions, as well as extensive destruction of the temporal lobe. In the left hemisphere, the damage was relatively minor and was limited to the ventromedial frontal lobe, parahippocampal gyrus, and insula. After recovering from the acute phase of her illness, she presented with severe agnosia and amnesia. The pattern of brain damage suggests that she may suffer in part from the form of focal retrograde amnesia described by N. Kapur (1999), but her imagery deficits are also significant. She was able to copy simple figures, but performed poorly on tests of confrontational naming, could not recognize friends or family, and was unable to draw even simple figures from memory. Tests of

autobiographical memory revealed preserved anterograde memory with severe retrograde deficits. She was tested on a modified version of the Crovitz test that was divided into two conditions: in the constrained condition, she was instructed to retrieve memories only from before her illness; in the unconstrained condition, she could recall memories from any period in her life. In the latter condition, she was able to retrieve memories, but they seemed much vaguer and more general than those of the control subject. In the constrained condition, LD was unable to retrieve any retrograde autobiographical memories whatsoever. She did seem to be able to retrieve some retrograde semantic information, but it was unclear exactly how she was doing so; in some instances, she tried to make educated guesses (for example, she speculated that she first drove a car in high school) or volunteered that she had relearned the information after her illness. Thus, LD suffers from a severe long-term visual memory deficit, as defined by Farah's criteria. She suffers from severe retrograde amnesia, with some sparing of semantic information; she also exhibits relatively preserved anterograde memory, though her memories are more general than those of controls, perhaps because of her inability to use visual information.

Brown and Chobor (1995) reported a similar case. Their patient, MM, suffered a closed-head injury during a car accident; MRI revealed right frontal and left occipital damage with no sign of temporal-lobe trauma. She manifested visual agnosia, aphasia, and amnesia; she could copy drawings, but had difficulty with tests of picture completion, block design, and drawing from memory. Her agnosia apparently resolved in part over time, but she still experienced some aphasic difficulties (for example, she still had trouble recalling the word for a particular object, but regained the ability to describe the object's purpose). Her memory deficits are severe as well; she has significant anterograde deficits, but is able to retain new information, particularly with repetition. She claimed to have few if any retrograde memories; she does appear to remember some songs, but does not seem to recall any specific episodes associated with them. Aside from these few fragments, she, like LD, claimed to have relearned all the retrograde information that she knew. Although the extent of MM's recovery is not entirely clear – it would be interesting to know, for example, if her agnosia and amnesia improved in parallel – her cognitive impairments are similar to those of LD, and she does not have damage to the anterior temporal regions associated with focal retrograde amnesia.

The clearest case is that of MH (Ogden, 1993). After a closed-head injury, MH presented with visual agnosia, prosopagnosia, achromatopsia, and profound amnesia. MRI scans revealed bilateral occipital damage extending from the gray matter into the white matter; there was no evidence of damage to the temporal lobes, although occipitotemporal and occipitoparietal connections appeared to be severed. He could copy drawings of animals, but could not recognize what he had copied; he could draw some simple figures from memory, but refused to attempt more complicated drawings. He also had difficulty recognizing objects by sight, correctly identifying only 8 of 30 objects; nonetheless, he could recognize all these objects by touch. Moreover, he reports that he does not dream and cannot generate visual images. His retrograde memory deficits were severe; on the Autobiographical Memory Interview (AMI), he retrieved 0 events

for the early-adult period (the normal range is 7-9), and 2 for the childhood period (the normal range is 6-9). His anterograde memory is better, though still abnormal; he retrieved 4 postmorbidity events on the AMI (the normal range is again 7-9). MH also seemed to have a high level of emotional, olfactory, auditory, and tactile data in his anterograde memories, and he too remembers songs he learned before his accident. The severity of his anterograde amnesia suggests that no other sensory modality can take on the role of visual memory.

All these results follow from the consensus view that the MTL and frontal lobes are processing areas that bind sensory and other information stored elsewhere in the brain at encoding, and that these other areas are processing areas for their specific modality but also serve to store information in that modality. Damage to the MTL will cause marked anterograde amnesia, because it will impair all future encoding; memories encoded prior to the damage may not be as impaired, because the MTL's role in retrieval, especially with the passage of time, may be decreased. In contrast, if much of the store of information is lost, retrograde amnesia will be severe and anterograde amnesia would be moderate if other modalities were available to store information using an intact MTL/frontal system; there would be no reason to suspect a temporal gradient different from normal forgetting.

CONCLUSIONS

The neuropsychological data support the claim that visual imagery plays a key role in autobiographical memory. In particular, they demonstrate that one component of visual imagery – long-term visual memory – is necessary for intact autobiographical memory. Patients who suffer trauma that impairs this component also lose much of the information that constitutes autobiographical memory. This loss can impair memory in several ways. First, visual stimuli will no longer be able to cue memories. Second, even if a nonvisual stimulus were used as a cue, large portions of the memory would be missing. The loss in these patients extends beyond a simple loss of data, however; these patients do not report a normal number of memories that simply lack visual information. Rather, the inability to access long-term visual memories impedes the coactivation of neural circuits required for recall. According to the consensus theory, data in one sensory store stimulate data in another sensory store in a lengthy chain of activation. Visual memory comprises so many vital links in the chain that its destruction entirely prevents the recall of the memory. In terms of the example above, destruction of visual memory would not only prevent the retrieval of the image of the friend, but would prevent the retrieval of the conversation as well. On the other hand, an inability to understand the meaning of objects or images, without a loss of visual memory, does not by itself produce amnesia. We speculate that these patients – who comprise the majority of visual agnosics – would report that they could see items and people in their “mind’s eye,” but that they could not identify them.

It might be suggested that the VMDA patients actually suffer from a disconnection between visual imagery and semantics, rather than the destruction

of stored visual images. Along the same lines, it might be proposed that these patients have lost semantic information about objects, not necessarily visual information *per se*. To both these questions, we can only respond that these patients do not seem to be able to use visual imagery in any way at all. They cannot generate any visual images (whether spontaneously or on request) and, if asked, they often report that they do not dream. Whether or not this is “semantic memory” depends on one’s definition of *semantic*. Under one definition – one that comes from linguistics – the term carries connotations of “meaning” and “knowledge.” If we use this definition, then HJA, a visual agnostic, has lost semantic information about objects, but still has visual imagery, as shown by his preserved ability to draw flowers from memory. Patients who meet Farah’s third criterion, on the other hand, have lost not only this semantic information but also the images themselves. If we use Tulving’s definition of semantic memory instead of the linguistic definition, then both the meanings of images and the images themselves are part of semantic memory. However we choose to define the term, it seems clear that these patients do not have a general, modality-independent semantic memory impairment, because they can access semantic information about objects using tactile and auditory data; rather, under this definition, these patients suffer from a semantic memory impairment limited to the visual modality.

The neuropsychological deficits we describe may only serve to identify individuals with one type of relevant damage. Other damage that severely inhibits coactivation in visual cortex may also result in severe impairments of autobiographical memory. In our search of the literature, we found seven patients who suffered from amnesia coupled with other forms of imagery loss. Three of these patients could not copy objects but met the other criteria for long-term visual memory loss, and may have suffered from some perceptual or optical impairments combined with a long-term visual memory deficit (Boyle and Nielsen, 1954; D. N. Levine, 1978; Shuttleworth et al., 1982). One patient, who also had medial temporal damage, fit our criteria; however, he recovered from visual memory loss but not amnesia (Schnider et al., 1992). The other three patients suffered from amnesia and other forms of visual imagery deficits (Arbuse, 1947; Grossi et al., 1986; Hunkin et al., 1995).

Hunkin and colleagues’ case report is the most extensive of these. Their patient PH suffered a closed-head injury in a motorcycle accident; like MH, PH’s damage did not appear to extend beyond bilateral occipital regions. PH reportedly had some trouble learning new visual material, but had only minor difficulty recognizing famous faces and had no sign of agnosia; his ability to draw from retrograde memory was not assessed. Thus, he does not meet the criteria we presented above. His memory impairments did generally follow the pattern of deficits we identified in other cases; he suffered from a dense retrograde amnesia with a relatively mild anterograde impairment that allowed him to relearn some of the information he had lost. His retrograde amnesia was temporally graded, unlike that of the three patients described above; on the AMI, he was able to recall only one incident from childhood, but a normal seven events from the young-adult period that immediately preceded his accident. Although PH does not meet our criteria for VMDA, his case may

illustrate another way in which visual deficits can affect autobiographical memory.

Visual imagery can be subdivided into many components, both on a behavioral and neural level (e.g. Kosslyn, 1994). These subcomponents have received relatively little attention in the autobiographical memory literature, and patients with impairments of these subcomponents have not undergone detailed autobiographical memory testing in the neuropsychological literature. Further research along both lines would help clarify the role of different types of visual imagery in autobiographical memory.

MULTI-MODAL SPATIAL IMAGERY

Behavioral and Neuroanatomical Data

Substantial research has shown that spatial and object imagery are dissociable on both behavioral and neuroanatomical levels (Rubin, 1995; Mishkin et al., 1993) and the role of both types of imagery in autobiographical memory can be further divided (Cornoldi et al., 1989). On a behavioral level, spatial imagery plays roles in many mnemonic strategies; the method of loci, for example, involves the placement of images of to-be-remembered items in a familiar mental landscape. On a neurobiological level, spatial and object imagery have different neurological substrates: spatial imagery is processed in the dorsal visual stream, which passes through the occipital and parietal lobes; object imagery is processed in the ventral visual stream, which extends from the occipital lobes through the inferior temporal cortex (Mishkin et al., 1983). Also, spatial information is not necessarily visual; it involves the incorporation of data from multiple sensory modalities (Andersen, 1999; Stein, 1992). Thus, all available evidence prompts a separate examination of visual and spatial imagery.

Neuropsychological Data

Damage to the MTL, particularly the right MTL, results in spatial deficits (O'Keefe and Nadel, 1978; Morris et al., 1999; Smith and Milner, 1981); however, unilateral right MTL damage does not produce profound global amnesia (Barr et al., 1990). In this case, our approach has identified a region involved in the "consensus theory" of explicit memory. We wish to focus on regions, processes, and disorders that have not previously been explored in the memory literature; moreover, the role of the hippocampus in spatial cognition has been described quite thoroughly (O'Keefe and Nadel, 1978; Morris et al., 1999). Because the same structure is involved in explicit memory and spatial imagery, we cannot discern whether any memory effects are caused by a direct loss to the explicit memory system itself or are mediated by a loss in spatial processing. Therefore we will not discuss this syndrome further aside from a brief mention in the Conclusions.

Patients with damage to the inferior right parietal lobe often suffer from hemineglect (N. Burgess et al., 1999). Patients with this disorder tend to ignore the left half of space (the precise definition of hemineglect remains a matter of

dispute, but this general description will suffice for our purposes). The disorder affects memory as well as perception (Gaffan and Hornak, 1999). For example, when hemineglect patients were asked to visualize an avenue in Milan, they tended to omit structures on the left side of the street; after they were asked to imagine turning 180 degrees, they described the buildings they had previously omitted, but now left out the structures they had previously described (Bisiach and Luzzatti, 1978). Thus, although hemineglect is associated with some retrieval deficits, it does not induce profound global amnesia. Moreover, this syndrome does not involve destruction of stored memories; information inaccessible in some circumstances may be accessible in others. The memory deficit in hemineglect therefore falls into our first category – a deficit limited to the impaired cognitive process.

Damage to the superior parietal lobe often causes astereognosia, a form of tactile agnosia characterized by an inability to determine the shape of objects by touch. Some authors have characterized this disorder as one of spatial imagery (Stein, 1992), while others describe it as a deficit of tactile perception. Whatever the proper classification may be, existing reports (Beauvois et al., 1978; Bottini et al., 1995; Caselli, 1991; Chang et al., 1992; Endo et al., 1992; Feinberg et al., 1986; Frenay and Endtz, 1975; Manguiere and Isnard, 1995; Nakamura et al., 1998; Reed and Caselli, 1994; Reed et al., 1996) have not uncovered any signs of amnesia in these patients.

Bilateral damage to parieto-occipital regions often produces Balint's syndrome, a condition in which patients suffer from simultanagnosia, oculomotor apraxia, and optic ataxia (Balint, 1909/1995; Mendez and Cherrier, 1998; Rafal, 1997). This condition sometimes presents as an early symptom of neurodegenerative diseases such as Alzheimer's (Cogan, 1985; Mendez et al., 1990; Victoroff et al., 1994). From this data alone, we might suspect that spatial deficits underlie the memory deficits in Alzheimer's. In this condition, though, the spatial deficits appear early in the course of disease, but memory and insight are generally preserved until later in the course of the disease, suggesting that the two may be independent (Victoroff et al., 1994). Moreover, a patient who suffered Balint's syndrome after bilateral strokes did not have any memory deficits (Baylis et al., 1994; Baylis and Baylis, 2001; Kim and Robertson, 2001; Wojciulik and Kanwisher, 1998).

Conclusions

We found no relation between amnesia and the spatial deficits that arise from posterior cortical lesions. However, we were unable to locate any reports of patients whose parietal lesions destroyed stored information; therefore, we cannot rule out the possibility that such trauma could cause severe amnesia. Nor can we rule out a central role for spatial imagery in autobiographical memory: as we noted before, the MTL plays major roles in spatial memory and spatial behavior. In fact, some researchers suggest that the MTL encodes the spatiotemporal context of experiences (O'Keefe and Nadel, 1978) – the sort of information that distinguishes autobiographical memories from autobiographical facts (Brewer, 1995; Tulving, 1995). As we said at the beginning of this section,

we will leave this debate to others. We only add the observation that autobiographical memory does not appear to require the parietal lobe's spatial functions; as best we can tell, it falls into our first category of deficit.

AUDITORY IMAGERY

Behavioral Data

Memory research in general has devoted substantial time to the study of visual imagery; likewise, philosophical and introspective accounts of autobiographical memory assign it a central role. Auditory imagery has received much less attention (but see Reisberg, 1992). In studies conducted in our laboratory, though, subjects frequently report that their memories contain auditory imagery (Rubin et al., under review).

Neuropsychological Data

As with blindness, deafness does not appear to be associated with any impairment of autobiographical memory; once again, we must turn to patients who cannot access imagery through any modality. The few available theories of auditory imagery assume that visual imagery is organized like auditory imagery (Intons-Peterson, 1992); so, if an impairment of long-term visual memory is associated with amnesia, it seems reasonable to begin our review of auditory imagery by searching for patients with deficits of long-term auditory memory. Once again, agnosics are the most likely group of patients to suffer such a deficit. As with many neuropsychological syndromes, auditory agnosia has been subdivided into many different classification schemes, but overall these patients can be grouped into three distinct categories. In *verbal auditory agnosia*, sometimes called *pure word deafness* (e.g. Goldstein, 1974), patients cannot comprehend or produce spoken language, but can understand and produce written speech without difficulty. In *nonverbal auditory agnosia*, patients can understand and produce speech, but cannot comprehend environmental sounds (like the ring of a telephone or the sound of an ambulance siren; Buchtel and Stewart, 1989). *Complete auditory agnosics* suffer from a combination of both deficits (Engelien et al., 1995).

We searched the auditory agnosia literature for patients who had a deficit of auditory memory; we used criteria that were analogous to those proposed by Farah (1984) for the study of visual imagery. First, the patient must not be deaf, as revealed by a test of audiometric thresholds or localization abilities¹. Second, verbal auditory agnosics had to be unable to comprehend speech but able to

¹ A more nearly analogous criterion would require that patients retain the ability to repeat words or sounds. We did not use this standard for two reasons. First, many researchers did not test their patients' repetition abilities (especially their ability to repeat nonverbal sounds), although these abilities were generally poor when they were in fact tested. Second, the auditory and visual tasks differ substantially. When a visual agnostic tries to copy the Rey-Osterreith figure, for example, the drawing is present the whole time, and the patient can look back and forth as many times as necessary. When an auditory agnostic tries to repeat a word, however, the word is presented for a moment and is then gone – a far more difficult task.

comprehend writing; nonverbal auditory agnosics had to be unable to comprehend environmental sounds; and complete auditory agnosics had to manifest a combination of these deficits. Third, verbal auditory agnosics had to be unable to speak. (We found no cases in which nonverbal or complete auditory agnosics were asked to produce environmental sounds from memory, so we assumed that these patients met the third criterion. As we shall see, this assumption makes very little difference.)

A search of the literature yielded 16 cases of auditory agnosia that met these criteria. Of these, 4 were nonverbal auditory agnosics (Albert et al., 1972; De La Sayette et al., 1994; Habib et al., 1995; Taniwaki et al., 2000), 8 were verbal auditory agnosics (Buchman et al., 1986, cases 1 and 2; Coslett et al., 1984; Klein and Harper, 1956; Mendez and Rosenberg, 1991; Otsuki et al., 1998; E. Wang et al., 2000; Papathanasiou, 1998), and 4 were complete auditory agnosics (Garde and Cowey, 2000; Kaga et al., 2000; Michel et al., 1980; Rosati et al., 1982). Amnesia was not noted in any of these 17 case reports.

We began this examination by searching for deficits of auditory memory, but other forms of auditory imagery impairment could be associated with memory deficits. We therefore eliminated our third criterion, thereby broadening our search and adding another 27 cases of auditory agnosia (Albert and Bear, 1957; Baddeley and Wilson, 1993; Buchtel and Stewart, 1989; Chocholle et al., 1975; Clarke et al., 2000; Engelien et al., 1995; Fujii et al., 1990; Fung et al., 2000; Gazzaniga et al., 1974; Godefroy et al., 1995; Goldstein et al., 1975; Jerger et al., 1972; Kazui et al., 1990; Lambert et al., 1989; Metz-Lutz and Dahl, 1984; Motomura et al., 1986; Nové-Josserand et al., 1998; Oppenheimer and Newcombe, 1978; Reinhold, 1950; Roberts et al., 1987; Saffran et al., 1976; Spreen et al., 1965; Takahashi et al., 1992; von Stockert, 1982; Wohlfart et al., 1952). These cases included patients who had verbal auditory agnosia but could still speak; these patients are analogous to HJA, the aforementioned visual agnosic who could draw flowers. Only one of these patients had amnesia; this patient suffered from acute anterograde deficits that soon resolved (Goldstein et al., 1975).

Conclusions

The results from the study of auditory agnosia suggest that visual and auditory imagery play different roles in autobiographical memory. There are several possible explanations for this result. First, perhaps the introspections of the philosophers and the data of psychologists are accurate – perhaps we rely on vision more than audition, so that the disruption of auditory memory removes little information and therefore has little if any effect on coactivation. Second, perhaps auditory imagery *is* as important on the behavioral level; perhaps it is instead organized differently on the neurobiological level. Earlier, we proposed that visual imagery plays a central role in the neural circuits that underlie autobiographical memory. Perhaps auditory imagery is peripheral; perhaps auditory images are not typically connected to other stored data, meaning that there is often little or nothing “downstream” from auditory imagery. Therefore, destruction of auditory cortex and removal of auditory imagery would have little

effect on coactivation. These possibilities – along with our general claims about the importance of stored information – could be tested in part by examining memories that depend heavily on auditory imagery, such as a memory of a concert or a conversation in the dark. If the amount, but not the nature, of the information is important, then destruction of auditory cortex should prevent the retrieval of these memories. If, on the other hand, our speculations about the organization of auditory information are correct, then the nonauditory aspects of these memories should remain.

Third, as stated earlier, these patients may differ in important ways from the VMDA patients. Many researchers did not test their patients' abilities to repeat sounds or words. Also, although verbal auditory agnosics could neither speak nor comprehend language, nonverbal auditory agnosics were not asked to produce environmental sounds from memory, meaning that they may not be as impaired as the VMDA patients were; thus, it is hard to make clear comparisons between the existing cases of auditory and visual impairments. Moreover, on the neurological level, the etiologies of the syndromes may differ in critical ways. It is relatively easy to damage both occipital lobes, as they are located next to each other and can be damaged by a single closed-head injury. However, it is relatively difficult to suffer damage to both auditory cortices, as they are on opposite sides of the brain. Most reported patients with bilateral damage to auditory cortex have suffered two independent strokes, and the patient may have partially compensated for the first stroke by the time the second stroke happened. Firmer conclusions require more thorough testing. Nonetheless, we have found that impairments of auditory imagery are not associated with global autobiographical amnesia; rather, patients with auditory agnosias fall into the first category of deficit.

OTHER FORMS OF IMAGERY

Behavioral Data

In healthy participants, odors tend to evoke memories that have rarely been retrieved or discussed (Rubin et al., 1984) and a taste was the stimulus for perhaps the most famous autobiographical memory ever recorded (Proust's (1934) memory of the petite Madeleine). Olfactory, gustatory, and tactile imagery may also play prominent roles in traumatic memories (Kline and Rausch, 1985).

Neuropsychological Data

Although older adults frequently lose olfactory, gustatory, and tactile sensitivity, these disorders are generally thought to result from peripheral impairments; their problems are therefore analogous to those of the blind or the deaf. Central impairments of tactile perception are more common; however, as we noted in our section on spatial imagery, existing reports have not reported any signs of amnesia in tactile agnosics or astereognosics.

Conclusions

There are relatively little behavioral and neuropsychological data on smell, taste, and touch. Some of the relevant neuropsychological syndromes, particularly anosmia, are thought to be widely underdiagnosed and underreported (Callahan and Hinkebein, 1999). There were no reports of syndromes similar to VMDA. Perhaps these syndromes do exist, but the sensory impairments were not detected; alternatively, these stores of sensory data may play a smaller role in autobiographical memory. According to the theory we propose, the loss of a little-used store of imagery would presumably affect relatively few memories.

Nonetheless, the available behavioral data suggest several interesting lines of investigation. If smell does play an unusually prominent role in traumatic memories, then PTSD patients who subsequently become anosmic might find that their traumatic memories have lost intensity. More generally, anosmia might induce relatively greater memory impairment in any population of subjects whose memories depend heavily on smell (cooks, gardeners, and the like). On one level, this proposal seems banal; we might appear to be saying that smell probably forms an important part of memories in which smell plays a prominent role. In reality, though, we are asking questions about individual differences in memories and organization: to what extent can the network be organized differently? Can olfaction take a central role, like the role of visual imagery above, or is it limited by neurobiological constraints to a peripheral role?

The structure of the olfactory system makes it a particularly interesting candidate for further study. There are direct connections from the olfactory apparatus to the amygdala, and olfaction is the only sense that is not routed through the thalamus before it is sent to cortex (de Olmos, 1990; Turner et al., 1980). Although such organizational differences need not be apparent on a behavioral level, it is nonetheless possible that the role of olfaction differs from that of other sensory modalities.

LANGUAGE

Behavioral Data

So far, we have focused on imagery; however, many other cognitive processes play important roles in autobiographical memory, and language is one of the most prominent. (We separate language from auditory imagery because most of the auditory agnosics listed above were able to communicate, usually through written language.) Classically, language is divided into phonetics, syntax, and semantics. One could easily claim that phonetics and syntax should have little effect on memory of any kind; they might mainly help us communicate ideas that are actually stored in some unspecified nonlinguistic form. On the other hand, a long tradition in psychology and philosophy claims that we often (or always) think in words, that we talk to ourselves, and that this inner speech is an essential part of consciousness (Damasio, 1994; Ericsson and Simon, 1993; Skinner, 1974). In this view, conscious and explicit memory requires language. There are stronger arguments for the importance of semantics. Semantics, both at

the level of words and syntax and at the level of phrases and sentences, have traditionally been seen as ways of interpreting and storing information about the world, both at a personal and a cultural level (Schrauf, 2000), as in Pavlov's second signal system (Popov and Rokhlin, n. d.). That is, from almost any perspective, language is a central aspect of our memory for events.

Neuropsychological Data

Language impairments, or aphasias, are a common consequent of brain injury and have been subdivided into dozens of categories (see Goodglass, 1993 for examples). A detailed analysis of these classifications is not useful here because, with only one exception – semantic dementia – language impairments are not associated with impairments of autobiographical memory.

In fact, we were able to find two autobiographies written by Broca's aphasics (Luria, 1972; Wulf, 1979). Wulf's narrative begins with her memories of her stroke, then continues with her perspective on her impairment six years later; she repeatedly states that her speech impairment far outweighs any intellectual difficulties she may suffer (Wulf, 1979). Luria's patient Zasetsky produces thousands of pages of fluid, emotional narrative describing his "shattered world" and his inability to use language in everyday life: "When a person has a serious head wound ... he no longer understands or recognizes the meaning of words right away and cannot think of many words when he tries to speak or think" (Luria, 1972, p. 75).² Even conduction aphasics, who may lose "inner speech," do not typically manifest any major memory deficits; for example, E.B., a conduction aphasic, remembers his first day at the hospital, and describes his sensations upon waking: "Began to see white and hear voices and hospital sounds and then could see nurse and young doctor above me, talking to me, asking me my name. Felt stunned, numb, and weak all over. Tried to move but nothing to move, tried to talk but could only make slurred sounds" (D.N. Levine et al., 1982, p. 394).

Conclusions

Although aphasia can cause severe, obvious, and extensive changes to affected patients' speech and comprehension, it does not necessarily prevent these patients from producing well-structured autobiographical memories (at least, not if they are given a long time and a choice of modalities in which to record the memory). With two exceptions (to which we return in a moment), we found no reports of patients whose language loss resulted in autobiographical amnesia. Thus, the loss of language in aphasia does not globally disrupt the encoding and retrieval of autobiographical memories; rather, patients who take a long time to produce written or spoken language take a long time to produce

² Luria does state that Zasetsky has amnesia; however, it is odd to classify a patient as an amnesic when he can write an autobiography that describes the details of his life both before and after his accident – including peritraumatic details, which are frequently lost in head-injured patients. An amnesic's autobiography would presumably look more like that of Clive Wearing (Wilson and Wearing, 1995), who repeatedly enters variations of the same sentence into his diary.

memories. Aphasia appears at most to be at our first level of severity: a loss restricted to the particular system that is damaged. Even this impairment may not be complete; the autobiographies of both Zasetsky and Wulf contain reports of conversations. This conclusion comes with one caveat: we were unable to find any total aphasics who could not engage in any kind of verbal communication to any degree. An investigation of such patients – say, by asking them to draw or order pictures – would permit firmer conclusions.

In our review of this literature, however, we excluded two important conditions. *Semantic dementia* appears to be more general than a language deficit, has a neural basis different from most forms of aphasia, and has different effects on autobiographical memory. *Narrative* loss is not usually considered as aphasia – that is, as a form of language loss. Although aphasics have difficulty with phonetics, grammar, or semantics, they are relatively unimpaired on tasks that ask them to use narrative skills. When aphasics do perform poorly on such tasks, they tend to make errors of quantity, not quality (Freedman-Stern et al., 1984; Ulatowska et al., 1983). Their deficits tend to involve the loss of information: first, their stories are less complex than those of healthy subjects; second, they include less information overall (Berko-Gleason et al., 1980; Ulatowska et al., 1981), though occasionally this effect is not significant (Ernest-Baron et al., 1987). This dissociation between narrative and linguistic abilities prompts a separate examination of narrative (to which we will return later in the paper).

SEMANTIC MEMORY

A disorder known as semantic dementia has noteworthy effects on autobiographical memory (Hodges et al., 1992; Snowden et al., 1989, 1994, 1995; see Hodges and Graham, 2001, for a review). Patients with this disorder suffer from a progressive anomia and loss of word comprehension, although their phonological and syntactical abilities are generally spared (Hodges et al., 1992); specifically, they suffer from a progressive loss of knowledge about the world (Graham, 1999). These patients manifest widespread damage to temporal neocortex, although medial temporal structures generally appear intact, at least in the early stages of the disease (Graham and Hodges, 1997; Harasty et al., 1996).

Although early investigations suggested that semantic dementia spares episodic memory (Hodges et al., 1992), more recent work shows that the disorder results in amnesia (Graham and Hodges, 1997). Afflicted patients manifest a pattern of deficit opposite from that of most other amnesics; as is the case with normal forgetting, recent memories are recalled better than older memories, although semantic dementia patients remember far less than controls do (Graham and Hodges, 1997; Hodges and Graham, 1998; Nestor et al., 2002; Snowden et al., 1995; though see Moscovitch and Nadel, 1999, and Westmacott et al., 2001 for a different view). The existence of a temporal gradient remains a matter of debate; although testing with the AMI does show a temporal gradient, tests with other measures do not (Nestor et al., 2002). On the AMI, patients with semantic dementia performed near the level of controls when asked to retrieve recent memories, but performed more poorly than an Alzheimer's

group when asked to retrieve early memories. A more detailed investigation of a single patient using the Galton-Crovitz task yielded a similar result: the patient produced mainly superficial and impoverished memories, and most of the detailed, temporally located memories came from the 2 years prior to testing. Although these patients clearly have a memory deficit in terms of the amount of information they can recall, it is not clear whether their rate of memory loss differs from that of controls, because the control participants generally perform at ceiling (Moscovitch and Nadel, 1999; though see Graham and Hodges, 1997, for an exception). To measure the rate of decline properly, recall at the most recent time period should be equated (Rubin and Wenzel, 1996); this was not the case for the patient studied by Graham and Hodges (1997).

Recent investigations have raised some fascinating possibilities about the interaction between semantic and episodic memory. The interpretation in the previous paragraphs suggests that episodic memory requires unimpaired semantic memory – a claim that accords with the theory presented here, which states that autobiographical memory requires not only the brain regions that encode and retrieve data but also those regions that actually store the data. Some studies report the converse, however – namely, that recent autobiographical experiences can help preserve related semantic information (Graham et al., 1999; Snowden et al., 1999). Clinical observation of semantic dementia patients reveals that they retain some information about objects they encounter on a regular basis. For example, one patient identified a clothespin as a device used to seal cereal packets, which was its typical use in her household; she did not know its usual function (Snowden et al., 1999). Another patient, known as KE, knew the function of her own teakettle and was able to make tea with it without difficulty (Snowden et al., 1999). Graham and colleagues (1999) counter that the preserved data is not semantic, but merely semantic-like. They note that while KE knows the function of her own teakettle, she does not or cannot generalize to other similar objects; that is, she may have relearned information about her teakettle, but she does not seem to know anything about teakettles in general. Similar problems with generalization of semantic knowledge appear to be quite common in semantic dementia (Graham et al., 2000; Snowden et al., 1995). This behavior is quite normal in one sense: in most people, new episodic experiences allow for new semantic learning, and the first patient we reviewed behaves exactly like a healthy person who had only seen clothespins used to close cereal packets. The available evidence suggests that these new semantic (or semantic-like) memories are transient and fade within a relatively short time, even with extensive practice (Graham et al., 1999).

Investigations into semantic dementia have prompted a reexamination of the relation between semantic and episodic memory. Tulving's original proposal claimed that the acquisition of new semantic memories required an intact episodic memory – that semantic memories are in fact episodic memories that have been separated from their original context (Tulving, 1972). A more recent version of his model claims that episodic memory requires input from semantic memory (Tulving, 1995). The available evidence instead suggests that semantic and episodic memory are interdependent and interactive (Snowden et al., 1999; also see Shastri, 2002 for another view of the relation between episodic and

semantic memory). In semantic dementia, autobiographical experience creates or reactivates semantic knowledge. This semantic knowledge can then be used in new episodic memories. This knowledge will decay over time, however, resulting in a loss of the data that comprise associated autobiographical memories and consequent amnesia.

Conclusions

On both a behavioral and neurobiological level, the deficits in semantic dementia patients generally parallel those in VMDA. VMDA patients can copy a drawing of an object, even if they cannot identify it (Rubin and Greenberg, 1998); semantic dementia patients can repeat words, even if they no longer know what those words mean (Hodges et al., 1992). In semantic dementia, the presentation of a word no longer calls to mind its meaning; VMDA patients suffer from the same disability, only with objects instead of words. Both disorders stem from neocortical pathology in the absence of medial-temporal trauma; in both disorders, this damage results in the loss of information crucial to autobiographical memory; and in both disorders, the patients suffer retrograde amnesia with relatively mild anterograde deficits. Murre and colleagues (2001) propose that the pattern of memory loss in semantic dementia stems from the nature of the interaction between the medial temporal lobe and the neocortex. According to Murre's Trace-Link model, memories are stored in disparate areas of neocortex, and are bound by the MTL; ultimately, they are consolidated into the neocortex and no longer depend on the MTL. In semantic dementia, temporal-lobe atrophy prevents or impairs neocortical consolidation (or at least severely impaired), so memories always depend on the MTL. The MTL has limited capacity, though, so over time these memories are overwritten and lost.

It is worth taking a moment to clarify further the behavioral differences between the two syndromes, as semantic dementia patients may appear visually agnostic on casual examination. VMDA patients and semantic-dementia patients are both unable to recognize objects or draw them from memory in response to a verbal command. When asked about a teakettle that is not present, VMDA patients will be unable to draw it, but will be able to state that one uses it to make tea, that one fills it with water and puts it on the stove, and so on; a semantic-dementia patient, on the other hand, would have no idea what a kettle is. For example, a semantic-dementia patient reported by Srinivas and colleagues (Srinivas et al., 1997) can copy a drawing of a carrot and can reproduce that drawing from memory a few minutes later, but cannot draw a carrot from memory on verbal command; VMDA patients, however, would by definition be unable to perform such a task. Moreover, VMDA patients can sometimes deduce the identity of an object by describing its visual properties to himself and guessing from the description (Ogden, 1993). There are no reports of semantic dementia patients who can accomplish the same feat.

An observation by Moscovitch and Nadel (1999) suggests another possible difference between semantic dementia and VMDA, and thus between the role of visual and semantic information. In VMDA, the loss of visual information causes a loss of autobiographical memories that does not depend on whether the

stimuli used to cue the memory was visual or verbal. Moscovitch and Nadel mention a patient with semantic dementia who could not provide autobiographical memories to word cues but who could provide them to visual cues (see Westmacott et al., 2001 for further investigations of this patient). Thus, for this patient (but not for the patients with VMDA), coactivation circuits could be maintained for at least some autobiographical memories when the initial cuing was not through the damaged system. It is not yet clear whether this difference is a matter of the severity of the damage or a fundamental difference between the role of semantic memory and the role of vision in autobiographical memory. Because Westmacott and colleagues' patient could talk about the memories cued with pictures, however, it follows that non-linguistic stimuli could activate stored semantic information.

The progressive nature of semantic dementia may also play a role in the differences between the two syndromes. As the disorder slowly advances, patients may be able to compensate for their deficits to a certain extent by placing greater reliance on semantic information that they can still recall – information that is presumably stored in undamaged neocortical areas. They may also come to rely on stored perceptual information, even if they have lost the meaning of that information; semantic-dementia patients retain the ability to recognize objects and faces if the stimuli are perceptually identical at encoding and test, although atrophy of the right temporal lobe may impair this ability (Graham et al., 2000; Simons et al., 2001). VMDA, however, generally stems from faster etiologies such as stroke or head injury; victims would not have a chance to engage in such compensation, so all premorbid memories would be equally affected. Although we were able to find two cases of semantic-dementia-like syndromes arising from a focal event (De Renzi et al., 1987; N. Kapur et al., 1994), these cases shed little light on this issue; one case (De Renzi et al., 1987) reported that their patient had no autobiographical memory deficit, while the other (N. Kapur et al., 1994) reported that their patient had a moderate impairment of retrograde memory and a mild anterograde deficit. Finally, only a few studies have examined episodic memory in these disorders, and the overall clinical picture may change substantially as more patients undergo testing.

NARRATIVE

Behavioral Data

We can trace the use and study of narrative at least back to Aristotle. A more recent description comes from Schank and Abelson's (1995) view of stories, which stresses their goal-directed nature. Kintsch and Van Dijk (1975) note that a coherent narrative requires judgments of empathy, relevance, and theme. Narrative is generally considered a part of language (Rubin, 1995, 1998; Rubin and Greenberg, in press), and a deviation from this categorization requires justification. Here, the separation was forced by evidence from the neuropsychological literature, which reveals a double dissociation between language and narrative impairments. Moreover, most researchers who have examined the form and content of autobiographical memory have focused on

narrative structure; in fact, most prior claims for the importance of language were actually claims for the importance of narrative. Robinson (1981, 1995) incorporated theories of narrative from linguistics and folklore into cognitive psychology. Barclay (1986, 1995; Barclay and Smith, 1992) studied the “conversational nature of remembering” (Barclay and Smith, 1992), which is produced by the schematic nature of autobiographical memory and its relation to the rememberer’s culture. Fitzgerald (1986, 1988, 1992) uses concepts like “narrative thought” and “self-narratives” to account for autobiographical memory and its changes with mood and age. Schank and Abelson (1995, p. 1) claim that “the content of story memories depends on whether and how they are told to others, and these reconstituted memories form the basis of the individual’s *remembered self*.” Along the same lines, McAdams (2001) views life narratives as fundamental components of identity.

Narrative structure also establishes a major form of organization in autobiographical memory, providing temporal and goal structure. Autobiographical memories are usually recorded as narrative; they are told to another person and to oneself. Inclusions and exclusions depend in part on the narrative structures used. If the narrative structure omits some information, it is less likely to be remembered. For example, Brown and Kulik (1977) observed that reports of flashbulb memories tend to have canonical categories, such as the place, ongoing event, informant, affect in others, affect in self, and aftermath. Neisser (1982) countered that these may not be properties of flashbulb memories at all, but rather properties of the narrative genre used to report any news. Thus, Neisser claimed that these autobiographical memories are shaped by narrative conventions of the culture.

Finally, Habermas and Bluck (2000) use the term “autobiographical reasoning” to define the process by which autobiographical memories are combined into a coherent life story and related to the current self. They identify four components of autobiographical reasoning: *temporal coherence*, which involves the sequencing of events in time; *causal coherence*, which serves to explain both life events and changes in the narrator’s personality; *thematic coherence*, which involves an analysis of common themes among many different memories; and the *cultural concept of biography*, the cultural mores that dictate the events that are incorporated into a life story (see Q. Wang, 2001; Q. Wang and Leichtman, 2000, for studies of cultural differences in autobiographical remembering).

Neuropsychological Data

Agnosia and aphasia are neuropsychologically defined syndromes and their neuropsychological properties and neural basis has been well studied. In contrast, narrative loss is not, unless one equates it with dementia. There is no generally accepted definition of narrative in the neuropsychological literature and patients are not classified into a narrative deficit group in neuropsychological studies. Because of the early state of studying narrative using neuropsychology, we start by searching for the brain regions that are responsible for narrative processes.

Several investigations of narrative impairments attribute problems with narrative tasks to frontal-lobe or right-hemisphere damage (at least in part). Right-hemisphere patients manifest a series of deficits that reveal difficulty comprehending speech at the discourse level; though they retain the ability to speak “with appropriate phonology and syntax” (Goodglass, 1993, p.147), they perform poorly on the sorts of judgments that narrative tasks require (but see Brookshire and Nicholas, 1984 for a conflicting view). These patients had difficulty understanding and completing jokes, and had trouble identifying the mood of a story and completing it appropriately (Huber and Gleber, 1982). Right-hemisphere patients also manifest a substantial impairment of their ability to organize and reorganize data. Hough (1990) showed that right-hemisphere patients had significant difficulty reproducing narratives when the theme was withheld to the end; left-hemisphere patients and healthy controls performed at ceiling levels, because either they were able to divine the theme or they could hold the story information in memory, reorganizing it after the theme was presented.

In short, right-hemisphere patients frequently lose their ability to appreciate context, presuppositions, affective tone, and theme. Given the importance of these abilities to autobiographical memory, we might expect that their loss would induce at least partial amnesia, but the studies of right-hemisphere patients do not report such deficits. The absence of such reports is significant in itself; if amnesia existed, it would almost certainly have been reported. However, these patients frequently confabulate, producing lengthy and detailed autobiographical memories, some of which could not have occurred. Although the neurobiological structures that contain the data that comprise their memories might be intact, these patients might not be able to access the data that make up a unique episode.

Other important data come from patients with frontal-lobe damage. Although some evidence indicates that right ventral frontal damage results in dense retrograde amnesia, these deficits may result from disruption of white-matter tracts that connect the frontal lobes to the MTL (Calabrese et al., 1996; Costello et al., 1998; B. Levine et al., 1998). Damage to the frontal lobes, particularly the right ventral frontal regions, more often results in confabulation (Baddeley and Wilson, 1986; Moscovitch, 1989, 1992; Moscovitch and Melo, 1997; Schacter et al., 1998). Moscovitch (1989) has proposed a definition of confabulation that is adapted in part from Talland (1965). According to this definition, confabulations are (a) autobiographical; (b) inaccurate; (c) based upon actual memories, including memories of past thoughts (which may be inaccurately recalled as actual events); (d) cobbled together from data from disparate times; (e) uttered without any awareness of their inaccuracy.³ There is currently a debate about the purpose behind confabulations; some researchers believe that patients generally confabulate to cover gaps in their memories or to rationalize implausible statements that they have already made (Conway and Tacchi, 1996); other researchers believe that most confabulations are essentially purposeless, and that a small subset of confabulated statements, called “secondary confabulations,”

³ Like other authors (e.g. P. Burgess and Shallice, 1996), we are ignoring fantastic confabulations, which are bizarre statements that seem to have no bearing whatsoever to actual events. It is worth noting, though, that fantastic confabulators are “on task” – that is, they are attempting to recall their memories, and (as best as anyone can tell) they actually believe the wildly implausible stories they concoct.

may serve as rationalizations (Moscovitch, 1989). Confabulations appear to be based on fragments of real experiences (Moscovitch, 1989, 1992; Schnider et al., 1996; Schnider and Ptak, 1999); however, confabulating patients are disoriented in space and in time (Schnider et al., 1996; Schnider and Ptak, 1999) and are particularly susceptible to proactive interference. On recognition tasks, they have a higher false-positive rate than MTL amnesics or healthy controls and incorrectly respond to items that were targets in previous tests (Schnider and Ptak, 1999). As criterion (e) suggests, the frontal lobes are involved in source monitoring (Johnson et al., 1997); these patients often incorrectly identify the source of a particular piece of information and fail to realize that they have uttered incorrect or inconsistent statements until an experimenter calls attention to their error.

Although one study reports a temporal gradient in confabulation (Dalla Barba et al., 1998), most studies show that it affects memories from the entire lifespan (Moscovitch, 1989). This result suggests that the frontal lobes are involved in all memories. Nonetheless, the evidence from MTL damage, in which patients cannot store any new memories, shows that the frontal lobes by themselves cannot form adequate links among brain regions. Most theorists accordingly hold that the frontal lobes are responsible for inhibiting irrelevant information, not for stimulating relevant information.

Confabulation is generally considered a retrieval problem; although patients can store and retrieve data, they cannot retrieve them in the proper order or at the proper time. The nature of this retrieval deficit should, however, be distinguished from the retrieval deficit induced by MTL damage. Patients with MTL damage lose the ability to link one stimulus to another and may lose previously created links as well. MTL amnesics cannot consciously retrieve the data that comprise their memories, although they may show evidence of implicit memory. A confabulating frontal-lobe patient, on the other hand, can retrieve data – but not the correct data. Without the frontal lobes, retrieval must depend on the strengths of the medial-temporal connections; data from many different memories are retrieved, and confabulation results (Moscovitch, 1992).

These patients appear to be impaired on some (but not all) of Habermas and Bluck's components of autobiographical reasoning. First, confabulators clearly have difficulty preserving the *temporal sequence* of their autobiographical memories, as they may combine statements from the past and the present when they are talking about their current lives; for example, they may state that they got married four months ago and then claim that they have four children over the age of 20 (Moscovitch, 1989). Because of this tendency, their autobiographical memories may contain inaccurate or implausible *causal sequences*. Confabulators generally seem untroubled by the absence of causal coherence in their narratives; however, if their attention is called to their errors, they may engage in secondary confabulations in an attempt to restore this missing causal structure. Thus, they may still be able to weave together a possible (if implausible and inaccurate) story from the discontinuous memories they have retrieved. It is unclear whether the *thematic coherence* of their narratives is affected, although one account suggests that they are striving for such coherence (Conway and Fthenaki, 2000); however, if confabulators

generate inconsistent, erroneous, disorganized memories, it will likely be difficult for them to identify common themes among those elements.

With the exception of semantic dementia, the loss of language leads to a minimal loss of autobiographical memory when compared with what would be expected from the behavioral literature; in contrast, when semantic information is lost, autobiographical memory is impaired. The loss of narrative remains an open question.

EMOTION

Behavioral Data

Emotional events are generally more memorable than neutral events (see Christianson and Safer, 1995 for a review), and strongly emotional memories have been claimed to be indelible (Brewin et al., 1996). Emotions are an important component of vivid or flashbulb memories (Brown and Kulik, 1977), but strong emotion does not necessarily facilitate overall recall, as a subject may fixate on emotional details of an experience to the exclusion of other details (Christianson and Safer, 1995); moreover, the facilitatory effect may be different for positive and negative events (e.g. Ochsner, 2000). Such general findings aside, the assessment of the role of emotion in memory is a daunting task. Although several studies have addressed some of these issues, the results have been unclear; investigations of mood-dependent memory, for example, have yielded conflicting results (see Eich et al., 1994, for a review). Although emotion is clearly an important component of autobiographical memories, it is also one of the most complicated.

Neuropsychological Data

Although depression and other psychiatric syndromes have clear effects on memory (Williams, 1995), the neurobiological substrate of these disorders is unclear and is at best diffuse (e. g. Byrum et al., 1999). We will therefore limit our examination to those cases in which specific neurological damage results in some alteration of emotional processing.

Several lines of research have demonstrated that the amygdala plays an important role in emotional memory (Adolphs et al., 2000; Gallagher and Chiba, 1996; LaBar et al., 1998; Markowitsch et al., 1994; for a review, see Cahill and McGaugh, 1998), perhaps via its involvement with glucocorticoids and epinephrine (McGaugh et al., 1996). Damage to the amygdala does not cause amnesia but can reduce the likelihood that a given event will be recalled (Markowitsch et al., 1994). In healthy controls, emotional words or details are recalled more readily than nonemotional stimuli. Amygdala damage sometimes eliminates this effect (Cahill et al., 1995), but not always (Phelps et al., 1997); this effect may only occur in patients who had amygdala damage during development (Gallagher and Chiba, 1996). Other studies suggest that amygdala damage eliminates the facilitatory effect of arousal, but does not alter the effect of emotional valence (Phelps et al., 1998). In fact, there is a double dissociation

between the effects of hippocampal and amygdaloid damage: medial-temporal amnesics still show enhanced recall for emotional stimuli (Hamann et al., 1997), but amygdala patients do not differ from healthy controls on recall of nonemotional stimuli (Adolphs et al., 1997). Finally, although the amygdala plays a role in the encoding of emotional stimuli, little is known of its role during retrieval; one study reports left amygdala activation during the retrieval of emotional memories (Dolan et al., 2000), while another reports right amygdala activity during autobiographical memory retrieval in general (Fink et al., 1996).

Conclusions

Although the amygdala is not necessary for the encoding, storage, and retrieval of autobiographical memories, it does have a modulatory effect that alters the likelihood that a memory will be retrieved. Its mechanism of action in encoding is unclear; the amygdala is connected to the frontal lobe, the MTL, and various areas of sensory cortex (de Olmos, 1990), and could exert its influence through any of these connections. Like the hippocampus, the left and right amygdalae may serve different roles, with the left specialized for language and the right specialized for visual imagery (Markowitsch, 1999). Furthermore, its involvement in retrieval is unclear. Few patients have damage localized to the amygdala, and many of these suffered damage throughout development – an etiology that substantially complicates the picture. Moreover, recent research suggests that emotion itself relies on distributed neural systems, with different emotions subserved by different (though overlapping) brain networks (Damasio et al., 2000). For now, the neurobiology of emotion – and its involvement in autobiographical memory – remains uncertain.

SUMMARY

This review of relevant neuropsychological case studies has led to several important modifications and additions to both behavioral and neurobiological theories of autobiographical memory:

1. *The brain regions subserving visual memory form a crucial part of the coactivated networks that underlie autobiographical memory.* Although the behavioral research had previously identified visual imagery as important, the neurobiological research had not. The available data support the claims of the behavioral research; furthermore, they demonstrate that long-term visual memory is the vital component. Destruction of large areas of occipital cortex is associated with an impairment of visual memory and profound retrograde amnesia. The striking evidence from the VMDA patients suggests that visual memory has central importance in most people. Our current knowledge does not permit a comparison of different imagery abilities or different regions of visual cortex.

2. *Most impairments of linguistic or auditory abilities have no major effect on memory.* Although some of these patients might manifest subtle

impairments of autobiographical memory, they clearly do not have profound autobiographical amnesia. This result suggests that for autobiographical memory, the language that is lost in aphasia is not a mode of storage, but is rather an output system used to interpret memories and communicate them to others.

3. *Semantic memory plays a vital role in autobiographical memory.* A profound loss of semantic information is correlated with a severe impairment of autobiographical memory. In these patients, new autobiographical memories are encoded in part by incorporating the semantic memories that are intact at the time. As the disease continues, those semantic memories are lost, as are the data that comprise the autobiographical memories, and the memories themselves are thereby destroyed. Although some modality-specific semantic information seems to be stored in sensory cortices, evidence from semantic dementia suggests that lateral temporal regions may store semantic information as well. Although researchers of semantic dementia have proposed that this brain region stores the information that comprises autobiographical memories, there is no reason to assume that there is only one region that performs such storage. In accord with the first point, we would instead propose that this region is only one of several important areas of the brain.

4. *Narrative reasoning and language are dissociable cognitive processes with separate neural substrates.* As described above, the process of narrative reasoning allows for the identification of relevant information and the organization of that information into a coherent form. Disruption of linguistic abilities results from left-hemisphere damage; disruption of narrative abilities results from prefrontal damage, and disruption of this ability results in disorganized autobiographical memories.

5. These results cannot be explained by taking a simplistic reading of Damasio (1989) and positing a dichotomy between primary sensory cortices and other brain regions. Roughly equivalent impairments of visual and auditory imagery produce different memory deficits, as do impairments of semantic memory and language. Nor can we explain these results by positing a dichotomy between brain damage that removes stored information and damage that impairs other functions (Rubin and Greenberg, 1998); auditory cortex damage and visual cortex damage can both remove stored information, but they have different effects on memory. We could claim that the damage must remove “important” information, but this begs the question of what “important” means in this context. Here the behavioral data and philosophical observations can provide some assistance, as both demonstrate that vision is more important than audition.

FUTURE DIRECTIONS

As suggested in the introduction, the next step in this investigation should focus on detailed studies of more specific cognitive impairments. These studies could use a variety of methods, including neuropsychology, structural imaging, and functional imaging; we deal with each of these in turn.

First, as noted before, several important questions remain unanswered. What role do specific types of imagery play in autobiographical memory? Can we find auditory agnosics who are analogous to our VMDA patients? What role do other sensory modalities – particularly olfaction – play in autobiographical memory? What role does semantic memory play in autobiographical memory? Can minor damage to multiple cortical regions cause amnesia (Evans et al., 1996)? The neuropsychological method can address each of these questions; the most useful studies would focus on the detailed pattern of impairments manifested by each case.

The neuropsychological approach is inevitably messy, and investigations based solely on this evidence face several problems (Weldon, 1999). Functional imaging experiments are thus an important complement to neuropsychological case studies. As Cabeza and Nyberg (2000) note, it is difficult for neuropsychological research to distinguish between an encoding and a retrieval deficit, particularly in a progressive disorder, while functional neuroimaging allows for the easy separation of these two tasks. As we suggested in the Introduction, studies of autobiographical memory should not look for activation in a specific area of the brain, but should rather attempt to describe the pattern and sequence of activation throughout the entire brain (e.g. Fink et al., 1996). Some work along these lines has already begun using structural equation modeling (e.g. Cabeza et al., 1997a, b; Nyberg et al., 1996b). Intriguingly, some studies have reported activation in posterior cortical regions, particularly during ephory (S. Kapur et al., 1995; Nyberg et al., 1995, 2000a, b; Wheeler et al., 2000; Conway et al., 2001; see Cabeza and Nyberg, 2000 for a review) although at least two studies have failed to find such activation (Roland and Gulyas, 1995; Allan et al., 2000).

Another possible approach would involve structural imaging. This method, which permits the calculation of precise volumes of brain structures, allows for precise estimates of cerebral atrophy (Byrum et al., 1996). Behavioral changes can then be correlated with brain changes to determine which structures are responsible for a given behavior. When used in this manner, structural neuroimaging is a more sensitive variant of the neuropsychological method; instead of investigating the behavioral effects of a sudden trauma that causes widespread damage, it explores the effects of progressive changes that result in more subtle deficits. This approach has been used with some success in studies of semantic dementia (Mummery et al., 2000) and memory in general (Fama et al., 2001).

Each method – be it neuropsychology, structural imaging, or functional imaging – has its deficits; a complete investigation will combine all these methods. Whatever the method used, this review suggests that a broader focus – one that encompasses brain regions and cognitive processes not generally seen as important to memory – will expand and strengthen our understanding of autobiographical memory.

Acknowledgements. We would like to thank Martin Conway, Mark Wheeler and two anonymous reviewers for helpful comments on the manuscript. This work was supported in part by NIA grant R01 AG16340 and a McDonnell-Pew Cognitive Neuroscience Program Individual Grant, both awarded to DCR.

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APPENDIX A
 All 11 cases of long-term-visual memory loss found in the literature

Reference	Etiology	Damage ¹	Amnesia	RA > AA ²	Gradient ³
J. Brown and Chohor (1995)	Closed head injury	b. occipital, r. frontal.	Yes	Yes	No
O'Connor et al. (1992)	Encephalitis	r. occipital, parietal, frontal, ~ medial and anterior temporal, inferotemporal	Yes	Yes	No
Ogden (1993)	Closed head injury	b. occipital.	Yes	Yes	No
Trojano and Grossi (1992)	Closed head injury	b. occipital, frontal, temp, ~ medial temporal, ~ inferotemporal	Yes	Yes	No
Ratcliff and Newcombe (1982)	Encephalitis	b. occipital., parietal., medial temporal, inferotemporal	Yes	Yes	?
Albert et al. (1975)	Cerebrovascular accident	b. occipital.	Yes	No	Yes
Gomori and Hawryluk (1984)	Cyst	b. occipital, inferotemporal	Yes	No	Yes
Beyn and Knyazeva (1962)	Cerebrovascular accident	?	Yes	?	?
Shuttleworth et al. (1982, case 2)	Closed head injury	b. occipital, ~ medial temporal	Yes	?	?
Taylor and Warrington (1971)	Atrophy	b. occipital, ~inferotemporal	Yes	?	?
Wapner et al. (1978)	Cerebrovascular accident	l. occipital, temporal, ~ medial temporal	Yes	?	?

The 11 cases listed met the three criteria for visual memory deficit. The five cases with RA > AA are listed first.

¹ ? = case study provides no information. ~ = possible. l. = left. r. = right, b. = bilateral.

² RA > AA = is the retrograde amnesia reported as more severe than the anterograde amnesia.

³ gradient = is there a temporal gradient.

APPENDIX B

All 16 cases of long-term auditory memory loss found in the literature

Reference	Etiology	Damage	Amnesia	Agnosia Type
Albert et al. (1972)	stroke	b. posterior	No	Nonverbal
Fujii et al. (1990)	stroke	r. temporal	No	Nonverbal
Habib et al. (1995)	stroke	b. temporal, insula	No	Nonverbal
Taniwaki et al. (2000)	stroke	b. putamen, temporal	No	Nonverbal
Buchman et al. (1986) case 1	stroke	l. temporal, r. parietal	No	Verbal
Buchman et al. (1986) case 2	stroke	b. temporal	No	Verbal
Coslett et al. (1984)	~stroke	b. temporal, r. frontal	No	Verbal
Klein and Harper (1956)	stroke	?	No	Verbal
Mendez and Rosenberg (1991)	?	l. temporal	No	Verbal
Otsuki et al. (1998)	?	l. temporal	No	Verbal
Papathanasiou et al. (1998)	stroke	?	No	Verbal
Wang et al. (2000)	stroke	l. temporal, frontal, parietal	No	Verbal
Garde and Cowey (2000)	hyponatremia	b. temporal	No	Complete
Kaga et al. (2000)	stroke	b. temporal, l. frontal, parietal	No	Complete
Michel et al. (1980)	?	b. temporal, r. parietal	No	Complete
Rosati et al. (1982)	stroke	b. temporal	No	Complete

? = case study provides no information. ~ = possible. l. = left. r. = right, b. = bilateral.