Long-term memory disorders: measurement and modeling
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Although semantic dementia is primarily characterized by deficits in semantic memory, episodic memory is also impaired. Patients show poor recall of old autobiographical and semantic memories, with better retrieval of recent experiences; they can form new memories, and normal performance on pictorial recognition memory has been demonstrated. As these abnormalities in episodic memory are virtually a mirror image of those seen in the amnesic syndromes, semantic dementia poses a challenge to extant models of remote memory and amnesia. In this chapter, it is shown that the TraceLink model can reproduce some of the principal findings on episodic memory in semantic dementia. A loss of nodes and connections within the trace system, which can be identified with the temporal neocortical memory storage sites implicated in semantic dementia, simulates without further assumptions the findings reported above.

6.1 INTRODUCTION

To accommodate conflicting and complex findings from amnesia (see chapter 2), a number of researchers have proposed that medial temporal lobe structures (i.e., the hippocampus, subiculum, parahippocampal gyrus and entorhinal cortex) play a temporary, time-limited role in the acquisition of human long-term memories (Graham & Hodges, 1997; Milner, 1989; Murre, 1996; Squire & Alvarez, 1995; Squire et al., 1984), though others disagree (Nadel & Moscovitch, 1997). In this line of thinking, the hippocampal complex is necessary for the retrieval of recently experienced events, but is not involved in the retrieval of older episodic and semantic memories. By contrast, regions of the neocortex are thought to be the more permanent repository of memory (Squire & Alvarez, 1995). This theory provides an explanation for why patients with damage to the hippocampal complex show a temporally-graded loss of memory, with recent memories being affected more severely than older memories.

The verbal theory described above has inspired several neuroanatomically-informed computational models of human memory. A number of researchers have shown that lesioning of the hippocampus in these models results in a temporal gradient in memory retrieval similar to that seen in amnesic patients with hippocampal damage (Alvarez & Squire, 1994; McClelland et al., 1995; Murre, 1996), while the previous chapter has shown how other experimental data related to amnesia can be modeled. This approach to the study of human long-term memory allows neuropsychological theories to be tested in detail, and results in the generation of new hypotheses which can be investigated in brain-damaged subjects.

This chapter is based on work done in co-operation with Jaap Murre, and also reported in Meeter & Murre (in press).
One condition that has so far not been investigated with these models, is semantic dementia, also called progressive fluent aphasia (Hodges, Patterson, Oxbury, & Funnell, 1992) and the temporal variant of frontotemporal dementia (Edwards Lee et al., 1997). Semantic dementia is associated with non-Alzheimer degenerative pathology of especially the inferolateral temporal neocortex, with relative sparing (at least in the early stages) of medial temporal regions (Hodges, Garrard, & Patterson, 1998; Mummyery et al., 2000); though some damage in the left hippocampus is evident in certain patients (Galton et al., 2001). Patients typically show a progressive deterioration in their semantic knowledge, and yet seem to possess relatively preserved day-to-day (episodic) memory. Semantic dementia provides, therefore, a unique opportunity to investigate the organization of human long-term memory.

Semantic dementia is primarily a disease in which semantic memory is compromised. Patients are unable to name previously familiar objects, people, and places, and show poor language comprehension. They show deficits on verbally-based semantic memory tests such as category fluency and picture naming, but also on non-verbal tests of semantic memory: they have difficulty matching animal and object sounds to pictures of the animal or object (Bozeat et al., 2000), and may have difficulties handling previously familiar objects (Hodges et al., 1998).

Episodic memory is relatively spared in semantic dementia. Nevertheless, it is not normal. Murre, Graham and Hodges (2001) have recently discussed semantic dementia in the context of neuroanatomically-based computational models of long-term memory, and suggested several characteristics of semantic dementia that these models could be expected to simulate appropriately. Three of the particularly salient ones were related to episodic memory.

1. Relative sparing of recent versus remote memories

Patients with semantic dementia show some retrograde amnesia. The amnesia is far more pronounced for the distant past than for recent periods. This has been demonstrated in both the autobiographical (Graham & Hodges, 1997; Murre et al., 2001; Snowden, Griffiths, & Neary, 1996) and public knowledge domains (Hodges & Graham, 1998; Snowden et al., 1996). These results stand in contrast to the Ribot gradient typically found in patients with an amnesic syndrome, in which recent memories are preferentially affected and remote memories are relatively spared.

Direct comparisons of semantic dementia with amnesia have generally been restricted to patients with Alzheimer's disease. Though Alzheimer's patients show pathology in almost all brain regions, the hippocampus is disproportionately affected in the early stages (Braak & Braak, 1991), and mild to moderate Alzheimer's patients have many characteristics in common with patients with discrete hippocampal damage (Deweer et al., 2001). Figure 18a shows one such comparison of as group of semantic dementia patients with normal controls and patients with Alzheimer's disease on a test of remote autobiographical memory (Graham & Hodges, 1997). While patients with Alzheimer's disease, like other amnestic groups (Gade & Morteson, 1990; Kopelman, 1989; Reed & Squire, 1998), were especially impaired for the most recent period, semantic dementia patients showed relatively preserved recent memory, and were impaired on remote memories.
2. Preservation of new learning, as measured by recognition memory, early in the disease

One of the consistent findings in semantic dementia is that patients, even those with severe semantic memory deficits, are still able to acquire new episodic memories. By contrast, an episodic learning deficit (anterograde amnesia) is the most prominent symptom of amnesic disorders. One semantic dementia patient, for example, who had forgotten the names of vegetables, was able to relearn to name them with the help of short descriptions (Funnell, 1995). Group studies show that patients with semantic dementia can perform normally on a three-alternatives forced choice test of visual recognition memory (Graham, Becker, & Hodges, 1997; Graham, Patterson, & Hodges, 1999; Graham, Simons, Pratt, Patterson, & Hodges, 2000, see Figure 19a). Though on the Rey complex figure their performance was not normal, it was far less impaired than that of a group of patients with presumed early Alzheimer disease. The capacity for new learning becomes compromised, however, as the disease progresses (Graham, Patterson, & Hodges, 1999).

3. Increased long-term forgetting of newly learned material

Murre (1996, 1997) has predicted, on the basis of an analytical review of the TraceLink model, that patients with semantic dementia might experience increased forgetting. Some evidence for this prediction came both from a recent semantic dementia case study (Graham, Patterson, Pratt, & Hodges, 1999). It has also been found in cases with lateral temporal lobe lesions other than through semantic dementia (Kapur et al., 1996). In two case studies (Graham, Patterson, Pratt et al., 1999; Graham, Patterson, Pratt, & Hodges,
2001), a patient with semantic dementia was able to relearn, via repeated training sessions, forgotten exemplars for categories, so as to perform normally on category fluency tests. Once the training sessions ceased, however, the exemplars were quickly forgotten. This is the opposite pattern of that found in amnesic patients, who once they have acquired memories, tend to show forgetting similar to that of normal controls (Hupper & Piercy, 1978; Kopelman, 1985).

One can conclude that in the realm of episodic memory, semantic dementia has many characteristics opposite to those seen in amnesia (Graham, Patterson, & Hodges, 1999; Hodges, 1995; Hodges et al., 1998; Hodges et al., 1992; Snowden, Goulding, & Neary, 1989). Semantic dementia patients remain capable of new learning, and their retrograde amnesia tends to spare recent memories. This syndrome, therefore, offers a challenge to computational models of human memory. Only if they are able to simulate the neuropsychological phenomena observed in semantic dementia as well as in amnesia, can they be regarded as viable models of long-term memory. Though Murre et al. (2001) suggest on the basis of verbal arguments that connectionist models of remote memory and amnesia would be able to account for the data reviewed above, this has so far not been shown. The aim of the current chapter is therefore to investigate whether one computational model of amnesia, TraceLink (see previous chapter), can simulate the findings.

In the TraceLink model, a natural implementation of semantic dementia would be a loss of trace elements and of trace-trace connections (i.e., cortico-cortical connections). This mimics the atrophy of temporal neocortex seen in semantic dementia (Galton et al., 2001; Hodges et al., 1998; Mummery et al., 2000). At the onset of the disease, loss of trace-trace connections dominates, as the chains connecting distant regions of the cortex (Abeles Chains) will be more vulnerable than the functional units in the neocortex (i.e., hypercolumns or ensembles). New memories can still be encoded, as the link system and modulatory system are still fully operational. The transition to stages 3

![Figure 19](image-url)  

*Figure 19:* (a.) Episodic learning in normal controls (Ctrl), patients with semantic dementia (SD), and patients with probable early Alzheimer’s disease (AD). Plotted is performance, corrected for guessing, on 3-alternative forced choice recognition of 40 pictures (adapted from Graham et al., 2001). (b.) Raw performance (proportion of recalled pattern nodes) on patterns 9, 10 and 11 immediately after acquisition. In the semantic dementia (SD) and amnesia conditions, these are the patterns acquired right after the lesion. *Intact* refers to the control simulation with the intact model.
and 4 via consolidation (see previous chapter) is severely impaired, however, because there are not enough trace-trace connections left to form supporting networks at the trace level. The system will show (i) a diffuse but possibly extensive loss of existing well-consolidated memories, (ii) preservation of the formation of episodic memories through the link system, (iii) strong interference of new over old episodic memories, because of the limited capacity of the link system. This will cause learned episodes to be forgotten faster than in the intact model. If a memory is not rehearsed regularly it will be lost from the link system. This behavior is very similar to that observed in patients with semantic dementia. In the next section, these explanations will be further explored in a series of simulations.

6.2 SIMULATING SEMANTIC DEMENTIA

Three simulations of semantic dementia were carried out, addressing the three properties of episodic memory in semantic dementia discussed above:

- relative sparing of recent versus remote memories;
- intact new learning;
- accelerated forgetting of newly learned material;

Moreover, a fourth exploratory simulation was done to investigate the behavior of the model in simulated implicit learning. In each of these simulations, the behavior of the intact model was compared to an ‘amnesic’ model (with link deactivation) and to a ‘semantic dementia’ model in which lesions to trace were made.

Simulation 1: remote memory gradient

Method In the first simulation, the retention curve was simulated under three conditions: normal memory, retrograde amnesia, and semantic dementia. In all three conditions, a list of 15 patterns was learned by the model, with consolidation periods interspersed between pattern acquisitions. Then the model was tested three times (since no learning occurred during the tests, preceding tests do not alter the results of subsequent tests; the three tests are therefore independent, and can be considered as within-subject comparisons). It was first tested in intact state for the control condition. Then, the link layer was deactivated, and the model was tested again for the amnesia condition.

To simulate a state of progressed semantic dementia, trace nodes and connections within the trace layer were lesioned. Our standard lesion was 80% of all connections within the trace layer and 10% of trace nodes (in simulation 3 other lesion sizes will be explored). The model was tested a third and final time in this state in the semantic dementia condition. This completed the simulation.

Aside from the new testing condition (the semantic dementia condition), no changes were made compared with previous simulations of amnesia (see chapter 5). No assumptions were added, nor were there any new parameters besides those involved in the semantic dementia manipulation.

Results and discussion In the control condition, the model shows the power law forgetting curve found in many studies of human memory (Anderson & Schooler, 1991; Rubin & Wenzel, 1996; Wixted & Ebbesen, 1991; see Figure 18b, inset).
Constructors of retrograde amnesia tests usually attempt to hold performance constant across decades for normal controls (Hodges, 1995; Mayes et al., 1994). As a consequence of this, memories for different time periods are not equivalent in original learning strength. The inequality of the items for different time periods can best be simulated by calculating the performance of the abnormal groups as a fraction of the performance of the normal controls. For better comparison with data using retrograde amnesia tests, we therefore report the results of the amnesia and semantic dementia as a fraction of the performance in the control condition. As can be seen in Figure 18b, deactivating the link layer in the retrograde amnesia condition generates a Ribot curve: recent patterns are lost, but remote ones were still available after deactivation of the link layer. In the semantic dementia condition, however, exactly the opposite occurs: the most recent patterns are still available, while old patterns are severely degraded by the lesion of the trace-trace connections. This amounts to a reverse pattern of retrograde amnesia similar to the one observed in semantic dementia patients (see Figure 18a for comparison).

Simulation 2: new learning and forgetting

Method To test whether learning would remain intact in a state of simulated semantic dementia, we compared learning in the intact model with learning in a condition where a lesion to trace system connectivity had been made. In the semantic dementia condition, a lesion of again 80% of all trace-trace connections and 10% of trace nodes was made after the acquisition of 8 patterns. Subsequently, 8 more patterns were presented to the model in its lesioned state. In the control condition, all 16 patterns were learned by an intact model. In both simulations, the model learned the patterns by going through the alternation of acquisition and consolidation as explained above.

Results and discussion We analyzed both new learning, and the forgetting of patterns in the model. To measure new learning, performance on newly acquired patterns was tested immediately after acquisition. In Figure 19b, retrieval of the first three patterns acquired after the

![Figure 20: The retention curve of pattern 10 in both the control condition and the semantic dementia conditions. For both curves, performance immediately after acquisition is set to 1, and then retention of pattern 10 is measured after acquisition of subsequent patterns. The x-axis gives the number of interfering, newly acquired patterns. For example, at "1" the performance on pattern 10 is given after pattern 11 has been acquired.](image)
lesion is plotted for the three conditions. The semantic dementia lesion did not hamper the ability of the model to encode new patterns, though new learning was much diminished in the amnesia condition.

To investigate forgetting in the model, we plotted how well the patterns acquired after the lesion (patterns 9, 10, 11 etc.) could be recalled after acquisition of more patterns. Figure 20 plots, as an example, how well pattern 10 can be retrieved after acquisition of the pattern itself, and after 1 pattern more is acquired, two patterns more, etc. To test whether forgetting was faster after a lesion of trace connectivity, we fitted power functions through all observed forgetting curves (i.e., those for patterns 9 to 15). Then we compared the exponent, which determines the rate of decay of a power forgetting function, of the best fitting power function for the forgetting curves of all patterns. For all seven patterns, the exponent was larger -and therefore forgetting faster- in the semantic dementia condition than in the control condition: the exponent for the control curves ranged from -0.36 to -0.039, while it ranged from -0.42 to -0.45 for the semantic dementia curves (p<0.01 with a simple binomial test of significance).

The simulation shows that the model was still capable of storing new patterns in the semantic dementia condition. Though acquisition was not worse than in the control condition, patterns were lost more rapidly in the semantic dementia condition. This is analogous to what is found in patients with semantic dementia, where intact episodic learning is combined with speeded forgetting (Graham, Patterson, Pratt et al., 1999).

In the TraceLink model, these results follow naturally from the assumption that there is a fast-learning temporary repository (the link system) from where patterns are consolidated into a more permanent store (the trace system). After the lesion of trace system connectivity with which we model temporal neocortex atrophy lesions, the model is still able to store patterns through the link system. Episodic learning is therefore intact. However, patterns are lost fairly rapidly in the link system, and as patterns can only be consolidated in a rudimentary form if connections within the trace system are lesioned, patterns cannot build up a more permanent representation in that system.

**Simulation 3: varying the lesion size**

In the previous simulations, the semantic dementia condition consisted of a lesion of 80% of trace-trace connections, and 10% of trace nodes. To investigate whether this particular choice of lesion influenced the results, we redid simulations 1 and 2 with a large variation in lesion sizes. This also offers an opportunity to investigate how TraceLink would predict amnesia to progress with progression of temporal lobe lesions.

**Method** The simulations 1 (remote memory) and 2 (new learning) were repeated, with the semantic dementia lesion varying in size. The percentage of connections lesioned varied between 0 and 100%, the number of trace nodes lesioned between 0 and 40%.

**Results and discussion** Figure 21 shows how retrograde amnesia, the gradient in retrograde amnesia, and new episodic learning depend on the size of the trace lesion. As our measure of retrograde amnesia, we summed performance on all patterns stored before the lesion and divided them by the same performance in the control condition. We then divided relative performance on the most recent patterns, by relative performance on the remote memories (both were first divided by performance in the control simulation). This gave us an index of the gradient: a value of 1 indicates that all memories were equally affected, while lower values indicate a reverse gradient. Our measure of new learning, finally, was the same as that in the previous simulation: performance on the first three patterns learned after the lesion.
Retrograde amnesia (Figure 21a) became progressively worse with increasing lesions of trace nodes and trace-trace connections. Its gradient also became steeper with more lesioned trace nodes, and especially with more lost connections (Figure 21b). New learning, on the other hand, held up very well to lesions of connections, but suffered strongly from lesions of trace nodes (Figure 21c). One way to interpret these results is that broad lesions (loss of connectivity) are responsible for the reverse gradient in remote memory, while localized lesions (loss of nodes) causes a loss of new learning capacity. If progression of the disease can be seen as a slow movement from the upper left corner in the plots of Figure 21 (no lesion) to the lower right corner (many nodes and connections lost), then the present results would predict that the gradient in remote memory comes early. With larger lesions, the steepness of the reverse gradient reaches a plateau, and remote and recent memories degrade at an equal rate. The capacity for new learning is initially spared, but becomes compromised with more sustained lesions, in which trace nodes are lost. Indeed, episodic learning becomes compromised later in the disease (Graham, Patterson, & Hodges, 1999).

Simulation 4: implicit memory

In the previous chapter, we simulated intact implicit learning in amnesia. In accordance with a large body of theoretical ideas and empirical data that implicates neocortical processing areas in implicit memory and its independence of medial temporal lobe regions (Gabrieli, 1998; Gabrieli et al., 1995; Keane et al., 1995; Knowlton & Squire, 1993; Knowlton et al., 1994; Schacter, 1992), implicit learning was modeled as involving only the trace system. With this assumption, intact implicit learning in amnesia could be modeled. In our fourth simulation, we investigate whether implicit learning is intact in simulated semantic dementia.

Method In the simulation of implicit memory, 15 patterns were learned by the intact model as in all other simulations. This simulated the background learning (existing memories) of a subject who comes into an experiment. In the semantic dementia and

Figure 21: (a). Retrograde amnesia in the semantic dementia condition for varying lesion sizes (performance on stored patterns in the semantic dementia condition as a proportion of performance in the control condition). (b). Gradient of retrograde amnesia in semantic dementia, measured as retrograde amnesia for remote patterns (patterns 1 to 4) divided by retrograde amnesia for recent patterns (patterns 11 to 14). For ungraded amnesia this number is 1, for amnesia with a reverse gradient it is lower than 1. The lower the number, the steeper the reverse gradient. (c) New learning, as measured by raw average performance on the first three patterns learned after the lesion (same as in figure 2).
amnesia conditions, one of the standard lesions was then made (either 80% trace-trace connections and 10% of trace nodes, or the link layer). Two randomly selected patterns were given an extra, simulated implicit learning trial. In this trial, only the trace portion of the pattern was activated. Learning occurred within the trace layer with the normal trace learning parameter, without involvement of either the link layer or the modulatory system. The model was tested both before the implicit learning trials and after, so that the effect of one implicit memory trial could be assessed.

Results and discussion Replicating results in the previous chapter, the implicit learning trial resulted in improved performance for the control and amnesia conditions. In the semantic dementia condition, performance also improved after an implicit memory trial, but markedly less so than in the other conditions (see Figure 22b). TraceLink thus predicts a priming deficit in semantic dementia for those types of implicit learning that involve the lateral temporal lobe.

There is some evidence for this prediction. Several studies have pointed to deficits in conceptual priming. Tyler and Moss (Tyler & Moss, 1998) followed one patient longitudinally. In the first testing session, associative priming for perceptual and functional properties was intact, though no priming for category relations could be observed. Eleven months later, primes could only activate their functional properties, and at the final session no priming at all was observed. Both Moss, Tyler, Hodges and Patterson (Moss, Tyler, Hodges, & Patterson, 1995) and Nakamura, Nakanishi, Hamanaka, Nakaaki, and Yoshida (Nakamura, Nakanishi, Hamanaka, Nakaaki, & Yoshida, 2000) found impaired conceptual priming in patients with semantic dementia. Nakamura et al. (2000) did not find any evidence of conceptual priming in semantic dementia patients at all (see Figure 22a). Conceptual priming is thus clearly compromised in semantic dementia. However, it must be noted that the semantic

![Figure 22](image-url)
priming measures used in both studies are different from the repetition priming simulated in TraceLink.

Comparison of panel a and panel b of Figure 22 shows both divergence and convergence. Nakamura et al. (2000) use as their priming measure the difference in reaction time between primed and unprimed items. As the model does not generate reaction times, our measure is an increase in retrieval. TraceLink predicts some conceptual priming in semantic dementia; this is contrary to the findings of Nakamura et al. (2000) but consistent with those of the single patient study by Moss et al. (1995). Strikingly, TraceLink produces more priming in the amnesia condition than in the control condition. This is mirrored by Nakamura et al. (2000)'s finding of higher priming in Alzheimer patients than in their controls. The reason in both cases is similar: a low baseline. In the model, performance in the amnesia condition is so low that one simulated implicit learning trial strongly boosts retrieval. In Nakamura et al. (2000), Alzheimer patients were more than twice as slow as normal controls for both primed and unprimed words, leaving more room for large absolute priming effects.

Our simulation does not distinguish between kinds of priming. Though conceptual priming is clearly compromised, perceptual priming may be intact in semantic dementia (Srinavas, Breedin, Coslett, & Saffran, 1997). This is consistent with a proposal by Graham and Hodges (1999), who suggest that episodic learning of new information in semantic dementia may occur via perceptual pathways. These are not implemented in the current model.

6.3 GENERAL DISCUSSION

Though TraceLink was originally developed as a model for amnesic syndromes (Murre, 1996), in this chapter it was demonstrated that the model, without any additional assumptions or additional parameters, can reproduce some of the principal characteristics of episodic memory in semantic dementia. A lesion of trace nodes and trace-trace connections, modeling atrophy of primarily temporal neocortex, was shown to produce:

- a relative sparing of more recent memories in retrograde amnesia;
- intact new learning of patterns;
- speeded forgetting of newly acquired memories.
- impaired priming, consistent with conceptual priming deficits found in semantic dementia

From our third simulation, some predictions can be generated about how the disease progresses. Our results predict that a graded loss of remote memories comes earlier in the disease (when most loss involves connectivity), with a broad retrograde amnesia and a loss of learning capacity following with more extensive damage.

These findings were simulated on a qualitative rather than quantitative level. However, no new assumptions needed to be made to extend the model from amnesia to semantic dementia, and no parameter needed tuning (the direction of the effects was independent of the size of the semantic dementia lesion). Other models of amnesia, notably those of Alvarez and Squire (1994) and McClelland et al. (1995), would probably also be able to simulate some -if not all- of the findings. This is so because the main assumption that enables TraceLink to simulate them, namely consolidation of memories to a neocortical
memory store, is shared by all three models. However, the Alvarez and Squire model is probably too small to produce the effects on gradients and of new learning (as only 2 patterns can be stored in the model), and the McClelland et al. model does not implement the hippocampal memory system (see Murre et al., 2001, for a more thorough discussion of the similarities and differences between these models).

A limitation of the current model is that it does not incorporate the more basic, visual systems that probably play a role in intact episodic learning, and intact perceptual priming. Primary and secondary sensory cortices seem to be the locus of perceptual priming effects (Cabeza & Nyberg, 2000; Gabrieli et al., 1995; Keane et al., 1995), while episodic encoding and retrieval of visual stimuli often causes activation in occipital, visual cortex (Cabeza & Nyberg, 2000). Primary and secondary sensory cortices have also been suggested to play an important role in intact learning in semantic dementia. Graham et al. (2000) state that intact learning seems primarily perceptual in nature, as recognition performance of semantic dementia patients was in their experiment only normal with perceptually identical stimuli. It was not normal when at test subjects were confronted with perceptually different stimuli that portrayed the same object as during study. Therefore, they suggested, direct connections from these structures to the hippocampal memory system, bypassing the semantic system in the temporal lobe, might be responsible for intact learning of visual stimuli.

However, perceptual learning may be better than nonperceptual learning not because of direct connections between perceptual areas and the hippocampal system, but because a damaged temporal lobe may be able to sustain storage and retrieval longer for materials for which it is just the gateway than for materials for which it is the ultimate store (such as vocabulary). In this context, it is important to note that even with perceptually different stimuli, performance for semantic dementia patients was much better than that of Alzheimer’s patients in Graham et al.’s study, and that a direct comparison of visual and verbal learning in semantic dementia patients has never been done. It is thus too early to decide whether intact episodic learning in semantic dementia requires new connections. Either way, including of sensory areas that remain intact in semantic dementia into TraceLink would probably increase its explanatory power, for example by offering a way to simulate different forms of priming, and account for their dissociation in semantic dementia.

Though the model is thus far from complete, it can be concluded that findings from semantic dementia offer support for the view that memory consolidation in humans is dependent upon interactions between the hippocampal complex and the neocortex (Alvarez & Squire, 1994; Marr, 1971; McClelland et al., 1995; Milner, 1989; Squire & Alvarez, 1995). This idea, though by now so common as to be called the “Standard View” of retrograde amnesia (Nadel & Moscovitch, 1997), is still controversial. Nadel, Moscovitch and coworkers (Nadel & Moscovitch, 1997; Nadel et al., 2000) have developed a competing view, the multiple-trace theory, which assumes that the hippocampus is always necessary for episodic memory retrieval. After complete lesions of the hippocampus, Nadel and Moscovitch (1997) claim, the typical pattern is not a Ribot gradient, but ungraded retrograde amnesia spanning the whole lifetime of the patient. Temporal gradients, when they occur, are explained as stemming from partial lesions, which are more likely to lead to the loss of recent than remote memories in their model.

With regard to semantic dementia, some discussion has also ensued as to whether it genuinely offers support for the “Standard View” or not (Graham, Patterson, Pratt et al., 1999; Moscovitch & Nadel, 1999). Moscovitch and Nadel (1999) have claimed that it
does not, on the grounds that the data pattern is different from what is commonly assumed. Unlike TraceLink, the multiple-trace theory would predict, that semantic dementia patients do not have a different remote memory curve than normal controls: though the performance on retrograde amnesia tests may be lower for patients, remote memories should not be affected any more than recent memories. Indeed, they claim that this is the case. The gradients reported in the literature could be the result of a greater difficulty of old versus new items on the tests, which is masked in the performance of normal controls by ceiling effects (Nadel et al., 2000). Moreover, retrograde amnesia may be overestimated because of a reliance on verbal cueing. Visual cueing of memories might, they claim, lead to better recall of remote memories. This they support with observations from one patient with semantic dementia, who, with help of visual cues, could be made to remember his war time memories of more than half a century ago (Nadel et al., 2000).

However, not in all tests of remote memory controls perform close to ceiling. Hodges and Graham (1999) found evidence for a better performance of semantic dementia patients for recent versus remote memories on the identification of famous names, even though normal controls performed off the ceiling and were not better for recent than for remote periods. Though an assessment of remote memory with visual cues has yet to take place, we would argue that the evidence so far has been on the side of the reality of the reverse pattern in retrograde amnesia. As the pattern in the retrograde amnesia seen in semantic dementia can clearly differentiate between the Standard View espoused here and the multiple-trace theory, more research is certainly warranted.

If a reverse pattern is indeed present, it supports the assumptions underlying TraceLink. Together with the findings from semantic dementia, the simulations presented here make a stronger case for the view that memories are, with help of the hippocampal memory system, consolidated into the neocortex, and that temporal neocortex atrophy blocks this consolidation process.