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A Disorder of Executive Function and Its Role in Language Processing

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Abstract

R. Martin and colleagues have proposed separate stores for the maintenance of phonological and semantic information in short-term memory. Evidence from patients with aphasia has shown that damage to these separable buffers has specific consequences for language comprehension and production, suggesting an interdependence between language and memory systems. This article discusses recent research on aphasic patients with limited-capacity short-term memories (STMs) and reviews evidence suggesting that deficits in retaining semantic information in STM may be caused by a disorder in the executive control process of inhibition, specific to verbal representations. In contrast, a phonological STM deficit may be due to overly rapid decay. In semantic STM deficits, it is hypothesized that the inhibitory deficit produces difficulty inhibiting irrelevant verbal representations, which may lead to excessive interference. In turn, the excessive interference associated with semantic STM deficits has implications for single-word and sentence processing, and it may be the source of the reduced STM capacity shown by these patients.

Keywords

Semantic short-term memory; executive function; aphasia

Short-term Memory and Language Processing

Recent “language-based” models of short-term memory (STM) have been proposed in response to patterns of memory and language impairments seen in patients with aphasia.^{1–3} These models have departed from traditional models of short-term and working memory; traditional models propose the verbal contents of STM to be purely phonological.⁴ In contrast, language-based models of STM emphasize a role for both phonological and lexical-semantic representations in STM. The language-based models assume a close relationship among language processing, language production, and memory. That is, these models assume that language processes such as comprehension and production activate phonological and lexical-semantic representations that are maintained in verbal STM.³

The model proposed by R. Martin et al.³ assumes there are separate stores for maintaining phonological and semantic representations. One source of neuropsychological evidence concerning the separation of the phonological and semantic stores comes from the study of

aphasic patients who demonstrate selective deficits in maintaining these representations. The most striking evidence comes from patients who show good single word processing in comprehension and production but who have very reduced STM spans, in the range of one to three items. Their reduced span is not a result of articulation difficulties because the patients are also impaired on probe tasks, which require the retention of information without output. Evidence for the phonological store comes from patients who do not show the standard phonological effects on STM performance. Normally, spans are smaller for both phonologically similar words (relative to dissimilar words) and long words (relative to shorter words). Phonological STM patients, however, show neither a disadvantage for phonological similarity nor a disadvantage for word length (Table 1). Word spans are substantially better than nonword spans, suggesting that these patients receive the typical benefit of lexical-semantic information from real words over nonwords. Additionally, these patients' spans are better for visually presented material, relative to auditorily presented material, because this type of input relies less on phonological retention. Critically, these patients perform better on a category probe task, compared with a rhyme probe task.⁵ In these probe tasks, a list of words is heard, followed by a probe word. In the rhyme probe task, subjects judge whether the probe word rhymes with one of the previously presented words. Correct responding requires retaining the phonological representations of the list items. In the category probe task, in contrast, subjects judge whether the probe word is in the same category as one of the previously presented words. Thus the category probe task requires the retention of semantic information. Accordingly, patients with a deficit in retaining phonological information show smaller rhyme probe spans, relative to category probe spans (Table 1).

Patient EA demonstrates a striking deficit in retaining phonological information. Like other patients in the literature with this type of deficit,⁶ she sustained damage to her left temporoparietal region, including the supramarginal gyrus.^{5,7,8} EA's spontaneous speech is relatively fluent. She shows single-word comprehension above the mean for control subjects.⁵ Nonetheless, EA's auditory span is only 1.5 items; her visual span is only 2.5 items. (Normal aged-matched controls typically have spans between 4 and 6 items.) In Martin et al.'s⁸ study, EA did not show the standard phonological effects on span measures, and her performance on the category probe task was better than the rhyme probe task. Also, she showed substantially better word than nonword recall. These data suggested that her ability to retain phonological representations was severely restricted, but she showed a better ability to retain semantic representations. Because her lexical-semantic STM was intact, her comprehension of sentences was relatively normal because she was able to extract semantic information from speech input and maintain it while integrating the meanings from different words in the sentence. Interestingly, EA's narrative speech showed normal features in terms of speech rate and sentence complexity. One might assume a phonological buffer is used in planning speech output.⁹ If so, then EA's normal speech production provides evidence for a separation between a buffer for maintaining input phonology (involved in perception) and one for maintaining output phonology (involved in production) (see R. Martin et al.³ for discussion). EA has a deficit in the input buffer but a preservation of the output buffer.

In contrast to deficits affecting the phonological store, neuropsychological evidence also suggests that patients can have deficits in maintaining lexical-semantic information in STM.

Patient ML provides a striking example of a patient who has a deficit retaining lexical-semantic information in STM. He has a frontal/parietal lesion (although the area of interest, as discussed later, appears to be the left inferior gyrus^{5,10–12}) and produces nonfluent, non-agrammatic speech. His semantic abilities, as assessed by single-word and production tasks, are preserved. His visual span is reduced to 1.5 items; his auditory span is 2.5 items. Although ML showed standard phonological effects in list recall, there was essentially no difference between his span for word items and nonword items. Additionally, his performance on the rhyme probe task was better than the category probe task (Table 1). As discussed later, this deficit in retaining semantic information causes difficulties in sentence comprehension and production. The dissociation between inability to retain one type of information (either phonological or semantic) in STM suggests separate stores in STM. Thus R. Martin et al.³ have proposed separate phonological and lexical-semantic stores, or buffers, in STM (Figure 1). In their model, verbal information is retrieved from long-term knowledge structures and actively maintained in the appropriate STM buffers. Phonological and semantic buffers provide a natural way to account for the dissociations seen in patients with different types of STM deficits. Specifically, R. Martin and colleagues have proposed three buffers; impairments accessing or storing information in these buffers produces restricted STM capacities. The semantic buffer is involved in the short-term retention of lexical-semantic information; this buffer is damaged in patient ML. In contrast, the input-phonological buffer is involved in retaining phonological segments for language perception; patient EA's deficit resulted from damage to this buffer. Similarly, the output-phonological buffer is involved in retaining phonological segments for speech production (see R. Martin et al.³ for patient data supporting the separation of input and output phonology). These deficits are not caused by damage to long-term knowledge stores because all patients studied by R. Martin and colleagues have intact semantic knowledge (assessed through picture-naming tasks, picture-matching tasks, attribute judgment tasks). Instead, the source of restricted STM capacity is attributed to either specific deficits to a STM buffer or deficits accessing information from a STM buffer. Further evidence in support of the separation between the buffers comes from evidence suggesting that these different types of information may be maintained in separate areas of the brain.¹³

Sources of Restricted STM Capacity

R. Martin and colleagues have investigated the consequences of restricted STM capacities on language processing and production.^{7,8,14} Because successful sentence comprehension requires the retention of lexical/semantic information but not the retention of verbatim phonological information, patients with phonological STM deficits tend to have mostly preserved sentence comprehension.¹ In contrast, sentence comprehension is impaired in patients with semantic STM deficits; reduced lexical/semantic STM spans do not allow these patients to retain all of the lexical/semantic information needed for comprehension.

¹There is considerable controversy over the extent to which a phonological STM deficit has any consequences for comprehension. Although Vallar and Baddeley³⁶ demonstrated that their patient PV had difficulty with long, semantically reversible sentences, studies with other such patients have found good comprehension for these sentence types and for other sentences with complex syntactic structures.³⁷

On the production side, patients with phonological STM deficits are impaired in verbatim repetition because they are unable to actively maintain the necessary phonological segments. Nonetheless, these patients show preserved phrase and sentence production: they are able to plan the lexical/semantic information necessary for speaking. Patients with semantic STM deficits, however, show impaired phrase and sentence production due to an inability to manipulate and retain lexical/semantic information.^{15,16} Thus damage to individual STM buffers has specific consequences for language processing and production.

Role of Executive Processing (Control Functions) in STM/Semantic Processing

Models of STM took a new direction with Baddeley's⁴ multicomponent model of working memory. Working memory was proposed as a theoretical expansion on STM; it includes not only the temporary storage of information (STM) but also the ability to actively manipulate information being stored. Baddeley's⁴ model proposes two stores for retaining different types of information. The visuospatial sketchpad is involved in storing visual and spatial information; the phonological loop is involved in rehearsing and storing verbal information. Operating over these subsystems of working memory is the central executive; this component is involved in control processes such as attentional control over particular types of information (e.g., paying attention to one thing while ignoring something else) and manipulating information in working memory. Although not well understood, the central executive is thought to be a supervisory system involved in focusing, dividing, and switching attention.¹⁷ Using the language-based model of STM proposed by R. Martin et al.,³ the phonological loop proposed in Baddeley's model of working memory^{4,18} can be reconceptualized as a verbal STM containing more than just purely phonological information. Instead, this implementation of verbal STM contains the three STM buffers discussed earlier: the lexical-semantic buffer, input phonology buffer, and output phonology buffer. Information in each STM component is controlled and accessed by the central executive, or executive control processes.

Several researchers have attempted to specify the control processes performed by the central executive component of working memory. For example, Miyake et al.¹⁷ suggest that the central executive is involved in (at least) three specific processes. First, the central executive inhibits irrelevant information from entering or interfering with the contents of working memory. Second, the central executive switches attention, such as shifting between various tasks. Third, the central executive updates the contents of working memory by discarding the no-longer relevant information and revising the information currently being held in memory. Many researchers have tried to determine the neural substrates involved in these executive processes (see Collette, Hogge, et al.¹⁹ or Collette, Van der Linden, et al.²⁰ for a review). Despite mixed results, executive processes appear to involve a network of frontal and parietal regions.¹⁹

Research has suggested that at least some verbal working memory control functions are localized in the left frontal cortex. For example, researchers have proposed that regions in the left frontal lobe are involved in the retrieval of semantic information,^{21,22} the selection of competing semantic information,^{23,24} the maintenance or retention of semantic information,^{13,25} and inhibition used in verbal working memory tasks.²⁶

Evidence for Inhibition Deficits in Patients with Semantic STM Deficits

Evidence from patients with impaired semantic STM suggests that deficits in retaining semantic information may be caused by a failure to inhibit irrelevant representations. That is, interference patterns in semantic STM (discussed in the next paragraph and the section “Consequences of Inhibition Deficit for Production and Comprehension”) might be explained by a failure in inhibitory executive control processes to act on verbal STM. Interestingly, the same left frontal regions that are damaged in semantic STM moderate some verbal inhibition processes. This suggests that deficits in inhibition may be found for patients with semantic STM deficits but not for patients with phonological STM deficits whose damage is in more posterior temporoparietal areas.

One source of evidence that semantic STM patients fail to inhibit irrelevant representations comes from intrusions made during serial list recall.¹² Semantic STM patients made intrusions from previous lists, even several trials after the word had been presented. That is, the patient might recall the word “rug” on one list and then intrude that word when recalling a later list. For this item to be recalled a second time, without being presented as a list item a second time, the item must retain its activation in STM. It is proposed that this now-irrelevant item was not inhibited, thus allowing it to intrude in recall on later trials. However, this pattern of intrusions is inconsistent with theories of overly rapid decay because an object that has decayed from STM – and is therefore no longer active – would not be produced as an intrusion. Critically, these intrusions are produced by patients with semantic STM deficits but not by patients with phonological STM deficits.

Similarly, interference patterns can be seen in phrase production.¹⁵ When the semantic relatedness between items in the phrases is manipulated, patients with semantic STM deficits show exaggerated naming latencies. For example, when naming two semantically related pictures (e.g., “hat and coat”), as opposed to two unrelated pictures (e.g., “hat and ball”), semantic STM patients show an interference effect (related to unrelated) ~300 to 500 milliseconds (ms) greater than controls. A patient with a phonological deficit did not show this exaggerated effect. This suggests that the semantic STM buffer supports phrasal planning in speech production, in addition to semantic retention.

Consequence of Inhibition Deficit for Production and Comprehension

Inhibition Deficit in Semantic STM Patient ML: Relation to STM

Whereas early research discussed overly rapid decay of semantic representations in STM as the source of semantic STM deficits,^{2,12} more recent research has suggested a critical role of interference.¹¹ Along similar lines, some researchers have proposed that the control process of inhibition plays a critical role in working memory. Specifically, Hasher et al.²⁷ suggest that poor inhibitory control causes irrelevant information to interfere with relevant information, and this interference has consequences for many cognitive processes.

Jonides et al.^{26,28} have related inhibition to Brodmann’s Area 45, which is one area damaged in patients with semantic STM deficits. Jonides and colleagues used neuroimaging techniques to investigate neural activations during a letter-probe version of the recent negatives task. In this task, subjects receive a list of letters, followed by a probe letter. Their

task is to indicate whether the probe letter was in the list. The critical comparison is between the trials in which the probe letter was not in the previous list (the “no,” or negative trials); these are referred to as the recent negative trials and the nonrecent negative trials. In the recent negative trials, the probe item in list $n+1$ is presented as a list item in trial n (but not in trial $n+1$). In the nonrecent negative trials, the probe item in a later list (list $n+3$, for example) is presented as a list item in trial n (but is not presented in any trials in between). The time taken to indicate whether or not the probe item was in the most recently presented list is longer when the item was more recently in a list (recent negatives) versus when the item was less recently in a list (nonrecent negatives). The difference between these two conditions (recent and nonrecent negatives) is attributed to proactive interference from the previous list in the recent negative trials, relative to the nonrecent negative trials. That is, this difference is attributed to the need to inhibit the tendency to make a “yes” response to a probe that was recently presented as a list item. Importantly, neuroimaging results demonstrate greater activation in the Brodmann’s Area 45, in the left inferior frontal gyrus, on the recent negative trials compared with the nonrecent negative trials. This suggests that this inferior frontal area is involved in the inhibition of irrelevant information.

To examine the interference effects in patients with semantic STM deficits, semantic patient ML was tested on a word version of the recent negatives task.¹¹ This task used a small set of 16 words that were repeated throughout the course of the experiment; 3 words were always followed by a probe word. Relative to controls, who showed a 91 ms interference effect (recent to nonrecent negatives) in reaction times, semantic STM patient ML showed an exaggerated interference effect of 731 ms; he also showed exaggerated interference in error rates. In error data, the same patterns have been replicated in semantic patient BQ, whereas phonological patient KV shows interference effects in errors that are more similar to controls.²⁹

Hamilton and R. Martin¹¹ also found exaggerated interference effects in the standard Stroop task. In this task, subjects are asked to name the ink color of a word while ignoring the word itself. In the neutral condition, a string of X’s (XXXX) is written in colored ink and the color of the ink is named. In the congruent condition, the color of the ink matches the written word (BLACK, written in black ink). In the incongruent condition, the color of the ink is different from the written word (BLUE, written in black ink). Interference is measured by comparing the incongruent and neutral conditions. In this task, semantic patient ML showed exaggerated interference effects (969 ms) compared with controls (197 ms). Although phonological patient KV showed an interference effect larger than controls, this effect was not nearly as exaggerated as the effect shown by patient ML. Interestingly, these exaggerated interference effects were not found on nonverbal inhibition tasks.¹¹ ML showed normal performance on a nonverbal, spatial Stroop task and also on an antisaccade task. (The antisaccade task requires subjects to ignore a visual cue presented to one side of the screen and instead attend to the opposite side of the screen.) Thus Hamilton and R. Martin¹¹ suggest a separation between inhibition abilities in the verbal and nonverbal domains; inhibition in the verbal domain may be selectively affected in patients with semantic STM deficits.

Inhibition Deficit in Semantic STM Patients: Relation to Word Production

In addition to the STM tasks already discussed, deficits in inhibitory processes are also found in tasks involving word production, suggesting that the failure to inhibit irrelevant information has consequences for language production.^{30,31} Semantic STM patients ML and BQ were tested on the picture-word interference task (reported in Biegler et al.²⁹). In this task, single pictures are presented with superimposed words, which are either related or unrelated to the picture. To perform the task, subjects are asked to ignore the distractor word and name the picture as quickly as possible. Despite normal performance on single picture naming (without distractors), semantic STM patients ML and BQ show exaggerated interference effects in reaction times (naming latencies) and errors, relative to age-matched controls. This suggests that these semantic STM patients have more difficulty than normal inhibiting the simultaneously presented distractor. Behaviorally, this leads to increases in naming latencies and error rates when two pictures are semantically related versus unrelated; semantically related items compete more than unrelated items. Patients with a deficit in inhibition are unable to inhibit, or suppress, this stronger competing (related) response.

Again, despite normal single picture naming accuracy, semantic STM patients also show exaggerated interference effects when the same pictures must be named repeatedly across several trials. In the semantically blocked naming task,^{32,33} a small set of pictures is repeatedly named over multiple presentations. Groups of pictures are either semantically related or unrelated. For control subjects, the naming latencies are longer in related sets relative to unrelated sets. Researchers have proposed that this effect is produced by overactivation in the conceptual/semantic system. That is, because items are repeatedly named, their representations remain active and compete for output. Competition is greater in the semantically related sets than the unrelated sets because it is more difficult to choose among semantically related items.

To examine the interference effects in patients with semantic and phonological STM deficits, Biegler et al.²⁹ tested semantic patient ML and phonological patient KV on the semantically blocked naming task. Related blocks were made up of repeated presentations of items from a single category, appearing successively in the same block for four cycles; mixed blocks were made up of repeated presentations of items from multiple categories (semantically unrelated), also appearing successively in the same block for four cycles. Results from elderly controls and fluent, phonological patient KV replicated previous findings in the literature;³³ phonological patient KV showed naming patterns very similar to controls. Semantic patients ML and BQ, however, showed an exaggerated interference effect that greatly increased over repeated naming cycles, suggesting greater competition among semantically related items compared with unrelated items. This pattern can be accounted for by assuming an impaired inhibition of competitors; semantically related competitors provide more competition to be selected for output, and semantic patient ML is unable to resolve this competition due to his inability to inhibit lexical-semantic representations.

Inhibition Deficit in Semantic STM Patients: Relation to Sentence Comprehension

Failures to inhibit irrelevant representations also have consequences for language comprehension, as can be seen in the resolution of homophone ambiguity. Gernsbacher and Faust³⁴ used meaning-fit judgments to measure the time course of inhibition in meaning resolution of homophone sentences. In this task, subjects heard a sentence with an ambiguous homophone; the sentence “He dug with a spade” will be used as an example. Subjects judged whether or not a probe word, presented after the sentence containing the ambiguous word, fit with the meaning of the sentence. This probe word was either presented immediately (100 ms after the sentence) or after a delay (1000 ms after the sentence). In related (yes) trials, the probe word was related to the meaning of the sentence (e.g., the probe word “garden” is related to digging with a spade). In the homophone (no) condition, the probe word was related to the homophone meaning not implied by the homophone (e.g., the probe word “ace” is related to the alternate meaning of spade). In the unrelated (no) condition, the probe word was not related to the sentence (e.g., the probe word “lid” is not related to the example sentence). Gernsbacher and Faust³⁴ compared good and poor comprehenders to determine whether there is a relationship between comprehension and the ability to inhibit irrelevant word meanings –specifically, the inappropriate meaning of the homophone.

Interference effects were measured by comparing reaction times to determine meaning-fit of the probe word in the related and unrelated conditions. Both good and poor comprehenders showed interference effects when the probe item was tested immediately. However, only the poor comprehenders continued to show interference effects when the probe word was presented after a delay. Gernsbacher and Faust³⁴ suggested that good comprehenders efficiently suppress the inappropriate homophone meaning, given some time. Poor comprehenders, in contrast, are less able to suppress the inappropriate homophone meaning after a delay, which produces interference effects in the meaning-fit judgment. Consequently, the authors suggested that inhibition mechanisms are important for language comprehension.

Martin et al.³⁵ tested these homophone ambiguity interference effects in semantic patient ML and phonological patient JJ, using auditory presentations of the material used by Gernsbacher and Faust.³⁴ Although his effect was larger, phonological patient JJ showed the same reaction time and error patterns as controls: greater interference when the probe word was presented immediately, and reduced interference when the probe word was presented after a delay. Semantic patient ML, however, showed the opposite pattern. In both reaction times and error rates, semantic patient ML showed interference effects that were larger than controls when the probe word was presented immediately. Instead of showing reduced interference at the long delay, however, ML showed greater interference. This suggests that unlike controls and phonological patient JJ, semantic patient ML was unable to inhibit the inappropriate homophone meaning, leading to greater interference at the time of probe meaning-fit judgments.

Conclusions

As suggested by the many studies discussed here, recent research with aphasic STM patients suggests an inhibition deficit may underlie the semantic STM deficit seen in some aphasic patients with left frontal damage. More specifically, it has been suggested that this inhibition deficit is specific to verbal materials because it was not found to generalize to nonverbal materials.¹¹ This suggests a separation between verbal and nonverbal inhibition processes: lesions to left inferior frontal areas can selectively damage verbal inhibition.

Patients with deficits in semantic STM typically produce nonfluent speech and have impaired comprehension. Thus deficits in verbal inhibition, and its function in STM, have implications for both language production and comprehension. On the production side, the failure to inhibit irrelevant representations may serve as the source of nonfluent production. Competition among uninhibited competitors may cause slower retrieval or selection, leading to slow, labored production. Similarly, the inability to resolve ambiguity may impair comprehension at lexical and/or syntactic levels of representation. If a sentence contains ambiguity, various word or sentence representations may compete for activation; this competition occurs because inappropriate meanings are not easily inhibited.

The suggestion that the control process of inhibition is involved in working memory fits nicely with the model of working memory presented by Baddeley.^{4,18} It is possible that inhibition is (at least) one component of the central executive, and this inhibition component functions to allow fluent production and intact comprehension in verbal working memory.

Finally, the relationship between inhibition and STM needs to be assessed further and with a wider range of patients. Is semantic STM necessarily associated with an inhibition deficit? Patients with phonological STM deficits appear to show normal inhibition/interference effects; what is the source of their STM deficit? Future research should examine the role of other proposed executive control processes¹⁷ in working memory. Do the shifting and/or updating executive functions have specific functional roles in working memory? Does a deficit in one of these processes create specific patterns of impairments, as seen with inhibition? One way to approach these questions is with neuroimaging techniques, investigating relationships between inhibition tasks (such as the recent negatives task) and language tasks (such as picture-word interference). Are the same areas activated when interference resolution is necessary? These are clearly important questions that will reveal more about the mechanisms of the central executive component of working memory, in addition to the executive processes involved in accessing and manipulating information in working memory.

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Learning Outcomes

As a result of this activity, the reader will be able to (1) describe the difference between traditional models of working memory and language-based models of short-term memory, (2) identify the components of verbal short-term memory and the patterns of performance that characterize deficits in these components, and (3) identify the neural structures involved in inhibition and identify the relation between the inhibitory component of executive function and language processing.

Figure 1.
Model of short-term memory proposed by R. Martin et al (1999).

Table 1

Performance of Semantic and Phonological STM Patients on Word and Nonword Serial Recall Tasks and Category and Rhyme Probe Tasks

Phonological STM Deficit

Visual > auditory

Failure to show standard phonological effects

Nonword span very poor (substantially better word span)

Category probe > rhyme probe

Semantic STM Deficit

Auditory > visual

Show standard phonological effects

Word span = nonword span

Rhyme probe > category probe